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CONTENTS

Predicting 30-day mortality in geriatric trauma: integration of GTOS and trauma-specific frailty index

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ABSTRACT

Background

Geriatric trauma patients exhibit higher mortality due to frailty, comorbidities, and low physiological reserves. Traditional trauma severity scales often underestimate this risk because they do not use frailty markers. The Geriatric Trauma Outcome Score (GTOS) and Trauma Specific Frailty Index (TSFI) may help improve mortality prediction in this population.

Objective

To evaluate and compare the predictive accuracy of GTOS and TSFI for in-hospital mortality among geriatric trauma patients.

Methods

A retrospective observational study was conducted at KIMSHEALTH, Trivandrum, over one year (October 1, 2024 – October 1, 2025). Patients aged ≥65 years presenting with traumatic injuries were included. GTOS and TSFI scores were calculated and their ability to predict 30-day mortality was assessed using ROC curves, sensitivity, and specificity.

Results

Among 198 patients, non-survivors had higher median GTOS (115) and TSFI (0.51) compared with survivors (GTOS: 81; TSFI: 0.29). GTOS demonstrated good discrimination (AUC: 0.80), while TSFI showed fair-to-good discrimination (AUC: 0.74). When both scores were combined in a single model, predictive accuracy improved further, with the model yielding the highest AUC, indicating that using GTOS and TSFI together provides added value for mortality prediction. Optimal cutoff values were identified as GTOS ≥103 and TSFI ≥0.44.

Conclusion

GTOS and TSFI are valuable and complementary predictors of 30-day mortality in geriatric trauma patients. GTOS effectively captures anatomical and physiological severity, whereas TSFI

reflects baseline frailty. Their combined use in a single model further enhances mortality prediction beyond each score alone and may assist clinicians in decision-making.

Introduction

The aging population has transformed the epidemiology of trauma worldwide. Older adults now represent one of the fastest-growing trauma subgroups and account for a disproportionate share of trauma-related deaths.

Conventional scoring systems, such as the Injury Severity Score (ISS) and the Trauma and Injury Severity Score (TRISS), were not designed specifically for geriatric physiology. Consequently, they may fail to identify high-risk patients who appear anatomically stable but have low functional reserve, as these scoring systems do not incorporate components to measure frailty.

The Geriatric Trauma Outcome Score (GTOS) uses age, ISS, and transfusion needs to measure physiological stress after trauma. The Trauma-Specific Frailty Index (TSFI) assesses baseline frailty using variables such as comorbidities and functional status. Frailty is an important predictor of poor outcomes in trauma, often outperforming chronological age as a marker of vulnerability.

Despite growing recognition of frailty-informed trauma care, comparative evidence evaluating GTOS and TSFI remains limited, particularly in resource-variable emergency settings such as those in India. This study, therefore, aims to evaluate the relative predictive accuracy of GTOS and TSFI for mortality among geriatric trauma patients and explore whether a combined approach enhances mortality prediction.

Methodology

Study design and setting

A retrospective observational study was conducted at KIMSHEALTH Emergency Department, Trivandrumfrom October 1, 2024, to October 1, 2025.

Patients aged 65 or older with traumatic injuries requiring emergency admission and complete GTOS/TSFI data were included. Patients with blunt or penetrating traumatic injuries requiring emergency department (ED) evaluation and hospital admission, with complete data for the calculation of GTOS and TSFI, were included.

Patients who were dead on arrival, transferred to other facilities after initial resuscitation without available follow up data, had isolated soft tissue injuries or burns, or had incomplete data required to calculate GTOS or TSFI were excluded.

Data collection

Data on demographics, mechanism of injury, vital signs, ISS, comorbidities, interventions, and 30-day mortality were collected. GTOS was calculated using the formula: Age + (2.5 × ISS) + 22 (if transfused within 24 hours).

TSFI scores were based on 15 frailty variables and ranged from 0 to 1.

Outcome

The primary outcome was to assess and compare how well GTOS and TSFI predicted 30-day mortality in geriatric trauma patients.

Secondary outcomes included evaluating the discriminatory performance of GTOS and TSFI using AUC (Area Under the Receiver Operating Characteristic Curve); and determining the incremental prognostic value of adding frailty to GTOS.

Statistical analysis

All statistical analyses were performed using SPSS Statistics. Continuous variables were summarized as mean ± standard deviation (SD) or median with interquartile range (IQR). The predictive accuracy of GTOS and TSFI for 30 day mortality was assessed using receiver operating characteristic (ROC) curve analysis. For each score, the area under the ROC curve (AUC) with 95% confidence intervals (CI) was calculated to quantify discriminatory ability.

Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were reported with 95% CIs.

To examine the incremental prognostic value of frailty, three logistic regression models were constructed with 30 day mortality as the dependent variable: Model 1 included GTOS alone, Model 2 included TSFI alone, and Model 3 included both GTOS and TSFI.

Adjusted odds ratios (OR) with 95% CIs were estimated for each predictor. All tests were two sided, and a p-value <0.05 was considered statistically significant.

Results

Patient characteristics

During the study period, 213 geriatric trauma patients presented to the emergency department; 198 met the inclusion criteria and were included in the analysis. The cohort had a mean age of approximately 75.6 ±

7.1 years, with a slight male predominance (56.6%).

The predominant mechanism of injury was ground-level falls (68.1%), followed by road traffic accidents (26.8%) and other mechanisms (5.1%). The most common comorbidities were hypertension (57.5%), diabetes mellitus (36.4%), and coronary artery disease.

Injury severity was largely mild to moderate, with a median Injury Severity Score (ISS) of 13, demonstrating a right-skewed distribution with relatively fewer patients sustaining critical injuries.

Among the 198 geriatric trauma patients included in the study, 28 died within 30 days of the index emergency department presentation, corresponding to a 30 day all cause mortality rate of 14.1%.

GTOS and TSFI distributions

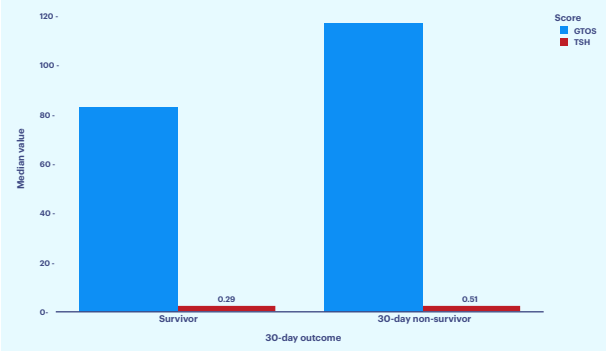

Figure 1: Distribution of median GTOS and TSFI according to 30 day survival status. Non survivors had higher GTOS (115 vs 81) and TSFI (0.51 vs 0.29) compared with survivors, indicating that greater injury severity and higher frailty burden are associated with increased 30 day mortality in geriatric trauma patients.

Mortality outcomes

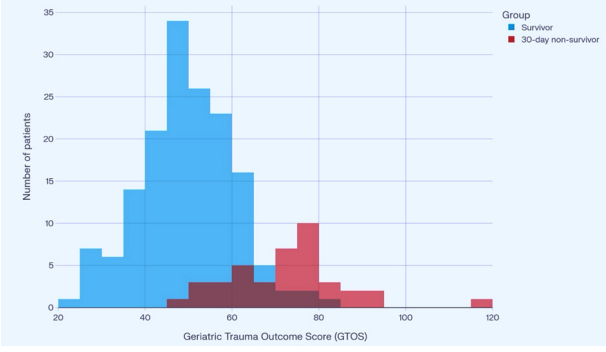

Figure 2: Overlaid histograms of GTOS stratified by 30 day survival status. Non survivors demonstrate a right shifted distribution with higher GTOS values compared with survivors, indicating that greater injury severity and frailty are both associated with an increased risk of 30 day mortality.

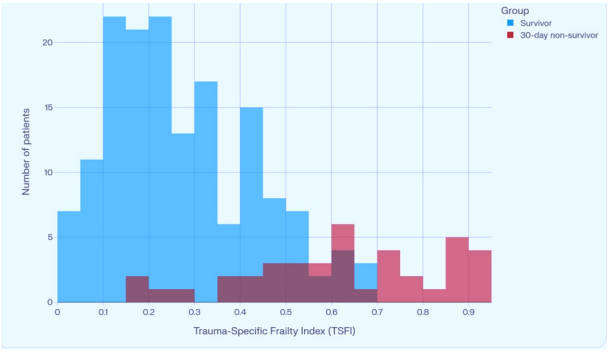

Figure 3: Overlaid histograms of TSFI by 30 day survival status. Non survivors exhibit a right shifted distribution with higher TSFI values compared with survivors, indicating that greater frailty is associated with an increased risk of 30 day mortality, reinforcing the association between biological vulnerability and adverse outcomes in geriatric trauma patients.

Discriminatory performance of GTOS and TSFI

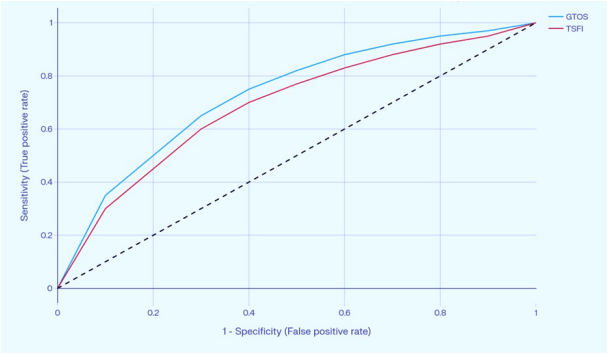

Figure 4: Receiver operating characteristic curve analysis demonstrated strong predictive capability for both scoring systems: GTOS showed strong discrimination with an area under the curve (AUC) of 0.88, while TSFI demonstrated fair-to-good discrimination with an AUC of 0.79.

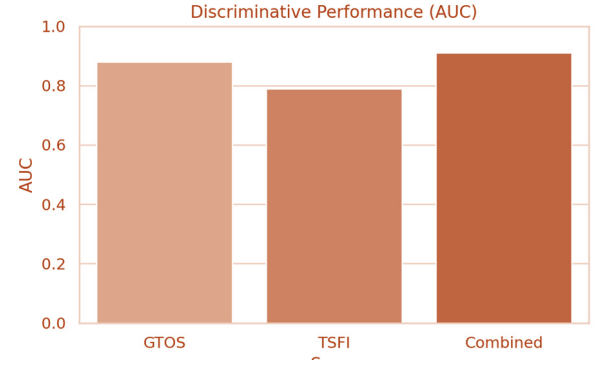

Figure 5:Combined model integrating GTOS with TSFI achieved an AUC of 0.91, indicating excellent prognostic performance and suggesting meaningful incremental value from frailty assessment.

Diagnostic thresholds

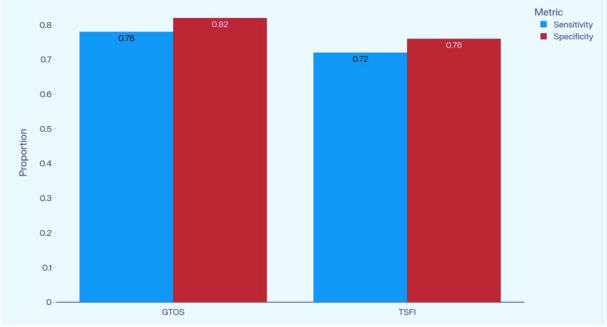

Figure 6: Optimal cutoffs were identified for GTOS ≥103 (Sensitivity 85%, Specificity 76%), demonstrating strong ability to identify high risk patients while maintaining acceptable specificity. A TSFI threshold of ≥0.44 yielded Sensitivity 69% and Specificity 71%.

Discussion

This retrospective cohort study highlights the complementary roles of GTOS and TSFI in predicting 30-day mortality among geriatric trauma patients. These findings support the concept that structural injury burden and pre existing physiological vulnerability are complementary domains that jointly determine short term outcomes in older trauma patients.[1,2] (Figure 1).

Our results are consistent with prior work validating GTOS as a geriatric-specific mortality prediction tool. Contemporary reviews of trauma scoring in older adults, therefore, highlight GTOS as one of the most practical tools for early risk stratification in geriatric trauma.[3] The original GTOS derivation study and subsequent external validations have repeatedly demonstrated good to excellent discrimination for in hospital or 30 day mortality, with AUCs often ≥0.80 and optimal cut offs around 100.[4-7]

A recent national analysis further confirmed that GTOS performs robustly across a large, heterogeneous trauma population and can match or exceed the predictive accuracy of more complex models while remaining simple to calculate at the bedside.[8] In this context, our finding that GTOS alone demonstrated good discriminatory performance and was substantially higher in non survivors reinforces its utility in daily practice (Figure 2).

Frailty assessment adds another important dimension to this study. Numerous studies have demonstrated that frailty, regardless of the instrument used, is independently associated with increased mortality, complications, prolonged hospitalisation, and adverse discharge disposition after trauma, often outperforming chronological age.[9-11] The Trauma Specific Frailty Index (TSFI) was designed to operationalise frailty in injured older adults and has been externally validated as an independent predictor of poor in hospital and post discharge outcomes[12-14] (Figure 3).

The key contribution of our study is the demonstration of incremental prognostic value when TSFI is combined with GTOS. The higher AUC observed for the GTOS+TSFI model compared with either score alone suggests that frailty captures vulnerability not fully reflected in anatomical and physiological injury metrics (Figures 4 and 5). These findings mirror those from multicentre studies showing that frailty indices and trauma severity scores provide independent information for predicting mortality, intensive care use, and other adverse outcomes in older trauma patients.[15]

From a clinical perspective, these results support a dual assessment strategy in which frailty screening is applied alongside routine trauma scoring to refine triage decisions, identify patients requiring early geriatric or palliative input, and guide realistic goals of care discussions with patients and families.[3,16] (Figure 6).

Our results are also aligned with evolving guideline recommendations. The 2023 World Society of Emergency Surgery guidelines on the management of trauma in elderly and frail patients recommend the use of GTOS to estimate mortality risk and advocate routine frailty assessment to identify high risk individuals.[16] Similarly, the American College of Surgeons best practice guidelines emphasize structured geriatric assessment, including frailty evaluation, as a cornerstone of modern geriatric trauma care.[16] By showing that both GTOS and TSFI were significantly elevated in 30 day non survivors and that their combination improved discriminatory performance, our study provides real world support for these recommendations in a resource limited setting.

This study has limitations. It is a single centre study with a relatively modest sample size, which may limit generalizability and contribute to wider confidence intervals around AUC estimates compared with large registry based analyses. The analysis focused on 30 day mortality and did not evaluate long term functional outcomes or quality of life, which are increasingly recognized as important endpoints in geriatric trauma research. TSFI was measured only once in the emergency department; dynamic changes in frailty status and the impact of acute illness were not captured. Finally, residual confounding remains a possiblity, as in any observational study.

Despite these limitations, this study represents the first comparative evaluation of GTOS and TSFI in an Indian emergency department with a comprehensive retrospective dataset. These findings provide contemporary evidence that incorporating frailty assessment alongside GTOS enhances short-term mortality prediction in elderly trauma patients. Future multicentre studies should validate optimal combined cut offs and compare the performance of GTOS+TSFI with emerging geriatric-specific scores such as GERtality and GTOS II, with attention not only to survival but also functional recovery and patient centred outcomes.

Conclusion

GTOS and TSFI are valuable tools for predicting 30-day mortality in geriatric trauma patients, and their combined use offers a more comprehensive assessment by capturing both anatomical injury severity and baseline frailty. Incorporating these scores into clinical practice may improve risk stratification and support more informed management decisions in this vulnerable population.

Geriatric trauma challenges clinicians to look beyond the injury and evaluate the patient in totality. These findings suggest that the most accurate predictions emerge when anatomical severity is interpreted through the lens of biological resilience. Integrating frailty assessment into early trauma evaluation may therefore represent an important advancement in modern emergency care for older adults, shifting practice from reactive management toward anticipatory, risk-informed intervention.

Funding

This research received no external funding.

Conflict of interest

The authors declare that they have no conflicts of interest related to this study.

Patient consent

For this study, patient data were collected from hospital records. No identifiable patient information has been included. Individual patient consent was waived due to the study's retrospective design.

Ethics committee approval

The study protocol was approved by the Institutional Ethics Committee of KIMSHEALTH, Trivandrum. Due to the study's retrospective nature, informed consent was waived.

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Clinical profile, triggers, and disability assessment in pediatric migraine: A tertiary care experience from south India

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ABSTRACT

Background

Migraine is a common neurological disorder in children and adolescents and contributes significantly to school absenteeism and reduced quality of life. Pediatric migraine often presents differently from adult migraine and may be influenced by various lifestyle and environmental triggers. This study aimed to evaluate the clinical characteristics, associated triggers, and migraine-related disability in children with migraine.

Methods

This prospective observational study was conducted in the Pediatric Neurology Outpatient Department of a tertiary care center in Kerala over 15 months (May 2023 to August 2024). Children aged 5–15 years diagnosed with migraine according to the International Headache Society (IHS) criteria were included. Patients with a normal systemic and neurological examination, experiencing 2–8 attacks per month, and not on prophylactic therapy for the previous four months were recruited. Clinical characteristics, associated symptoms, and potential triggers were recorded using a migraine diary. Migraine severity was assessed using the Visual Analog Scale (VAS), and disability was evaluated using the Pediatric Migraine Disability Assessment Score (PedMIDAS). Patients were counseled regarding lifestyle modification and followed for three months.

Results

Among the 100 children studied, 66% were aged 5–10 years, and 59% were boys. Bilateral headaches were observed in 68% of children, predominantly bifrontal. A positive family history of migraine was present in 80% of cases, and aura was reported in 17%. Photophobia was noted in 92% and phonophobia in 50%. Common triggers included cocoa-containing foods (51.9%), fast foods (30.7%), increased screen time (20.1%), and stress (18.8%). Based on PedMIDAS scores, 68% had mild disability, and 29% had little or no disability. Lifestyle modification and biobehavioral therapy resulted in improvement in 61% of cases, while the remaining children received nutraceuticals such as magnesium

or prophylactic medications including Flunarizine, amitriptyline, and beta-blockers. After three months of follow-up, 98% of children had mild or no migraine-related disability.

Conclusion

Pediatric migraine is commonly associated with identifiable dietary and lifestyle triggers. Early identification of triggers and implementation of lifestyle modifications can significantly reduce migraine-related disability in children.

Introduction

Pediatric migraine is increasing day by day due to changes in lifestyle. Approximately 50% of school-aged children experience headaches, with migraine accounting for a significant proportion. This study evaluates clinical characteristics, identifies triggers, and investigates the impact of lifestyle modifications to reduce migraine attacks in children.

Migraine is a neurological condition characterized by episodic attacks of moderate to severe headache, often accompanied by nausea, vomiting, and sensitivity to light or sound, lasting from a few hours to several days.[1,2] While migraines are common in adults, they also affect about 11% of children and adolescents.[3] In children, migraines often present differently from those in adults, typically with shorter duration and bilateral pain, frequently in the forehead.[4]

Children with frequent or severe migraines (PedMIDAS score > 20) may require preventive therapy, such as medication, supplements, or behavioral interventions, to lessen the frequency and intensity of episodes.[5]

Methodology

This was a prospective observational study conducted over 15 months, from May 2023 to August 2024. Children aged 5 to 15 years who attended the Paediatric Neurology Outpatient clinic and were diagnosed with 'migraine' according to the criteria of the International Headache Society (IHS) were included in the study.

Children meeting the IHS diagnostic criteria for migraines, who had normal systemic and neurological exams, experienced two to eight attacks per month, and had not received prophylactic medication in the previous four months, were included. Children currently on prophylactic medications or who did not provide consent were excluded.

All patients meeting the inclusion criteria were recruited after obtaining informed consent. Each participant was issued a migraine diary and instructed on how to record the number and duration of attacks. The severity of migraine attacks was recorded using the visual analogue scale (VAS) and the Pediatric Migraine Disability Assessment Score (PedMIDAS).

The decision to initiate antimigraine medications or magnesium depended solely on the judgment of the primary consultant. PedMIDAS scores were evaluated for all participants who met the inclusion criteria, and they were counseled on avoiding migraine triggers and on lifestyle modifications, such as practicing good sleep hygiene, avoiding chocolate and fast food, and minimizing screen time.

Patients were followed for 3 months (and advised to review at the end of one month and three months). During follow-up visits, migraine diaries were checked for compliance with prescribed medication. PedMIDAS scores were reassessed at the end of three months. Abortive therapy was advised if acute episodes had occurred during the study period. The calculated sample size for the study was 183.[6]

Data collected through Google Forms were analysed using SPSS version 16.0, with continuous variables reported as mean ± standard deviation (SD), and categorical variables presented as frequencies and percentages. Qualitative associations between various groups were assessed using the chi-square test or Fisher's exact test. Student's t-test was used, and a p-value of less than 0.05 was considered statistically significant.

Results

In our study on pediatric migraine, 66% of the children were between five and 10 years of age, while 34% were between 10 and 15 years, with a mean age of 9.24 years. Boys (59%) outnumbered girls (41%) in the study population. Among the 100 children included in the study, 68 experienced bilateral headaches, mainly bifrontal headaches (86%), while 31 reported unilateral headaches.

A strong family history of migraine was noted in 80% of children. Only 17% of the children reported aura, mostly in the form of visual disturbances such as scotoma and halos. Severity averaged 5.6 on a visual scale. Photophobia was present in 92% of children, and phonophobia was reported in 50% cases. Gastrointestinal issues included nausea, vomiting, abdominal pain (18.18%), and constipation (24.49%), with seven of 18 having infantile colic. Allergic rhinitis was observed in 30% of children. Food-related triggers impacted 33.96%, with cocoa (51.9%), fast food (30.71%), and MSG (6.3%) being the most common triggers. Other reported triggers included sleep deprivation (18.8%), prolonged screen

time (20.1%; median screen time of four hours per day), stress (18.8%), weather changes, travel, and poor sleep hygiene (26.2%).

Among girls aged 10–15 years, 12 had reached menarche, and five experienced catamenial migraines. Sleep provided relief from headaches in 83% of cases, and vomiting relieved headaches in 58%. Analgesics were used by 38% of the children, most commonly paracetamol. General examination findings were normal in 68% of children. However, 22 children were overweight, and 10 were underweight. MRI was performed in 27 cases and revealed abnormalities in two cases: one child had aT2 Flair hyperintensity and had an arachnoid cyst.

Based on PedMIDAS scores, 68% of children had mild disability (score 11–30), while 29% had little or no disability (score 0–10). Only 3% had moderate disability (score 31–50), and no children had severe disability (score >50).

Biobehavioral therapy alone led to improvement in 61% of cases, while the remaining children received nutraceuticals such as magnesium or prophylactic medications including Flunarizine (13 children), amitriptyline (10 children), and beta-blockers (nine children) (Figure 1). Two children receiving Flunarizine experienced significant weight gain. No adverse effects were observed in the amitriptyline or beta-blocker groups.

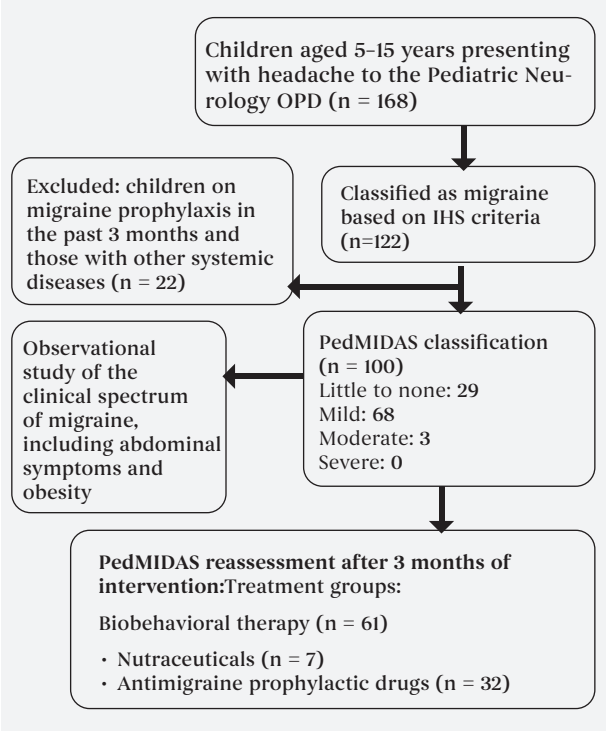

Figure 1: Flowchart of study design and patient selection

After three months, 98% of children had mild or no migraine-related disability. Statistical analysis showed a significant reduction in migraine frequency across all treatment groups, with the biobehavioral therapy group showing significant improvement ($p = 0.016$) and all other groups showing highly significant reductions ($p < 0.0001$).

Discussion

Our study provides valuable insights into childhood migraine, revealing distinct patterns in age distribution, gender prevalence, and headache characteristics. Among the children studied, 66% were between 5 and 10 years old, while 34% were in the 10 to 15-year-old group. Boys (59%) showed a higher prevalence compared to girls (41%), aligning with the American Migraine Prevalence and Prevention (AMPP) study, which reported that migraine is more common in boys before puberty but more frequent in girls during adolescence.[7] Stewart and colleagues found that migraine with aura was most common in boys at age 5 and girls at age 12, whereas migraine without aura peaked at ages 10 in boys and 14 in girls.[8]

Regarding headache characteristics, 68% of children experienced bilateral headaches, predominantly bifrontal, while 31% had unilateral headaches, a pattern typically seen in adults.

A strong hereditary link was observed, with 80% of children having a family history of migraine spanning two to three generations. Moreover, 17% experienced preceding aura, most commonly visual in nature (52.63%), including scotomas and gray halos; one child illustrated the aura on paper. Other aura symptoms included vestibular sensations such as rotational movement or a feeling of levitation, and sensory disturbances, such as 'ant-crawling' sensations on the hands. Al-Futaisi et al. reported aura in about one-third of migraine patients, usually manifesting as visual illusions known as fortification spectra, while 31% experienced numbness or tingling of the face or arms.[9]

In our study, 92% of children with migraines experienced photophobia, while phonophobia was reported in only 50%. This observation aligns with previous findings that photophobia is common in pediatric migraine, whereas phonophobia is less frequent.[10]

Gastrointestinal manifestations were also common, with nausea and vomiting being the most frequent symptoms, while 18.18% of children experienced abdominal pain, and 24.49% had constipation. Notably, seven of the 18 children with abdominal pain had a history of infantile colic. Additionally, allergic

rhinitis was present in 30% of children, contributing to poor sleep due to mouth breathing and thereby increasing headache susceptibility.

Dietary triggers frequently played a significant role, with chocolate, citrus fruits, nuts, ice cream, alcoholic beverages, coffee, and caffeine being the common migraine triggers.[11] Other compounds such as monosodium glutamate (MSG), histamine, sucralose, and gluten were also identified as potential triggers.[12] In this study, food-related triggers were identified in 33.96% of children, particularly cocoa-containing products (51.9%), fast food (30.71%), and aginomoto (6.3%), which were the most common triggers.

Sleep deprivation was another major factor (18.8%), while increased screen time, with a median of four hours per day, was reported as a trigger in 20.1% of cases. Stress was a contributing factor in 18.8% of children. Changes in weather and travel, particularly long school bus rides, affected some children, with seven reporting worsening symptoms following sun exposure.

The median migraine severity, assessed using the visual analog scale, was[5,6], confirming that pediatric migraines occur less frequently, are shorter in duration, are less severe, and are easier to manage than adult migraines. The most common relieving factors reported were sleep (83%), vomiting (58%), and analgesic use (38%).

Catamenial migraine has been reported to affect 35% to 54% of females.[13] In our study, catamenial migraine was reported in five girls (5/12). General examination findings were normal in 68% of the children. A study by Kjnik ST examined the impact of obesity on migraine severity in children and reported an obesity rate of 17% among children with migraine.[14] In comparison, our study found a slightly higher prevalence of overweight/obese children (22%).

Based on PedMIDAS scores, 68% of children had mild disability (score 11–30), while 29% had little or no disability (score 0–10). Only 3% had moderate disability (score 31–50), and no children had severe disability.

Management relied predominantly on biobehavioral therapy, which resulted in improvement in 61% of children. Nutraceutical supplementation with magnesium was used in 7% of cases. Prophylactic medications were prescribed to 32% of children, including flunarizine (13 children), amitriptyline (10 children), and beta-blockers (nine children).

Lifestyle modifications, particularly restricting cocoa-containing foods, significantly reduced migraine frequency, highlighting diet as a potential disease-

Paediatric migraine disability assessment (PedMIDAS)

The Pediatric Migraine Disability Assessment Score (PedMIDAS) is a validated questionnaire used to measure migraine-related disability in children over the previous three months. The score is calculated by summing the responses to the six questions below.

PedMIDAS questionnaire

1. How many full school days were missed in the last 3 months due to headaches? _____
2. How many partial days of school were missed in the last 3 months due to headaches (Do not include full days counted in Question 1)? _____
3. How many days in the last 3 months did you function at less than half your ability in school because of a headache? (Do not include days counted Questions 1 and 2)? _____
4. How many days were you unable to perform activities at home (e.g., chores, homework, etc.) due to a headache? _____
5. How many days did you not participate in other activities due to headaches (e.g., play, go out, sports, etc.)? _____
6. How many days did you participate in these activities but functioned at less than half your ability (Do not include days counted in Question 5)? _____

Scores:

Total PedMIDAS score _____

Headache frequency _____

Headache severity _____

PedMIDAS disability classification

PedMIDAS score	Disability grade	Interpretation
0-10	Grade I	Little to no disability
11-30	Grade II	Mild disability
31-50	Grade III	Moderate disability
>50	Grade IV	Severe disability

Interpretation: Higher PedMIDAS scores indicate greater disability related to migraine and may help guide decisions regarding preventive therapy and lifestyle modification.

How severe is your pain today ? Place a vertical mark on the line below to indicate how bad you feel your pain is today.

No pain |_____| **Very severe pain**

Figure 2: Visual Analog Scale (VAS) used for assessment of headache severity

Figure 3: For younger children who may have difficulty interpreting numerical pain scales, the Wong–Baker FACES Pain Rating Scale was used. This scale uses a series of facial expressions corresponding to increasing levels of pain intensity. Children were asked to select the face that best represented their pain level

modifying factor. After three months of treatment, 98 children transitioned to the 'little or no disability' category on the PedMIDAS scale, with only two children showing minimal improvement — one in the behavioral therapy group and one in the flunarizine group. Large clinical trials such as the CHAMP study have demonstrated that biobehavioral therapy and lifestyle modifications significantly reduce migraine frequency, with similar improvements observed among children receiving amitriptyline, topiramate, or placebo.

Additionally, an Indian study by Keerthana et al. reported comparable migraine disability scores between propranolol and placebo groups.[15]

A limitation of our study was the use of the PedMIDAS scores to assess migraine disability, which is subject to recall bias and subjective interpretation, particularly in children who may have difficulty accurately reporting symptoms over time.

Conclusion

The clinical profile of 100 children with migraine was analyzed in this study. The most common triggers identified were cocoa-containing foods (51.9%) and fast foods (30.7%). Lifestyle factors such as stress, poor sleep hygiene, and increased screen time also played an important role in triggering migraine attacks. A notable proportion of children (22%) were found to be overweight or obese. Lifestyle modification and trigger avoidance resulted in improvement in 61% of children.

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Impact of a nurse-led oral anticoagulation clinic on clinical outcomes and healthcare costs: A retrospective study

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ABSTRACT

This study evaluated the impact of a nurse-led anticoagulation clinic on clinical and economic outcomes of oral anticoagulant (OAC) therapy at a tertiary care hospital from 2018 to 2024. A retrospective analysis revealed that OAC-related complications, including thromboembolic and haemorrhagic events, were eliminated after the structured implementation of the nurse-led clinic. The annual cost burden from OAC complications declined from ₹3.2 million in 2018 to zero in 2024, marking a 100% reduction. These outcomes occurred even as patient volume increased significantly, highlighting the efficiency and scalability of the nurse-led model.

The findings are consistent with existing evidence that structured anticoagulation management and nurse–led interventions improve therapeutic safety, patient adherence, and healthcare efficiency. They also enhance cost-effectiveness in chronic disease management. This study underscores the importance of nurse autonomy, structured monitoring, and patient education in optimizing anticoagulation outcomes. It supports broader adoption of nurse-led clinics to manage high-risk populations.

Introduction

Safe management of patients receiving oral anticoagulant therapy (OAC) is essential for achieving optimal clinical outcomes. However, many patients undergoing valve replacement lack adequate understanding of their anticoagulant regimen, highlighting the need for targeted patient education.[1] Anticoagulant-related complications often result in hospitalizations and frequent consultations, imposing a substantial economic burden on patients and healthcare systems.

Medication adherence plays a key role in achieving optimal therapeutic outcomes. Standardized frameworks have been developed to better understand adherence behaviours in clinical practice[1]. Effective patient education is essential for improving adherence and minimizing complications.[2] Evidence-based guidelines emphasize the importance of structured monitoring and patient education in anticoagulation management to

maintain therapeutic International Normalized Ratio (INR) levels and prevent complications.[3]

The Nurse-Led Oral Anticoagulation Clinic (NL-OACC) at KIMSHEALTH, Trivandrum, addresses these challenges by providing comprehensive, cost-free follow-up care delivered by trained nurses. They also offer telephonic support, patient education, and proactive management of complications. This model emphasizes individualized, patient-centered care and structured education to enhance therapeutic safety.

This study evaluates the impact of the Nurse-Led Oral Anticoagulation Clinic (NL-OACC) in delivering cost-effective care for patients registered at KIMSHEALTH, Trivandrum.

Objectives

1. To provide structured education to patients on oral anticoagulant therapy.
2. To monitor laboratory values and promptly identify critical or panic values.
3. To identify causes of International Normalized Ratio (INR) variance among patients.
4. To reduce oral anticoagulation-related complications and associated treatment costs.
5. To decrease the frequency of clinic visits required to achieve target INR values, thereby reducing overall healthcare costs.
6. To minimize routine physician consultations for dose adjustments, reduce consultation expenses, travel-related inconvenience, and associated costs.

Methods

A retrospective review was conducted on patients registered in the NL-OACC from 2018 to 2023. The following interventions were implemented:

- Direct consultations and inpatient follow-up for patients receiving anticoagulation therapy registered with the nurse-led clinic.
- Structured patient education through counselling sessions, informational booklets, and alert cards.
- Systematic telephonic follow-up included weekly calls for new patients. Stable patients received monthly follow-ups. The clinic also offered 24/7 telephonic support.
- Proactive identification of INR variability and timely dose adjustments in collaboration with clinicians.
- Patient and family education on therapy, warning signs, and adherence.
- Maintenance of comprehensive clinical records to support monitoring and continuous quality improvement.
- Training of nursing staff in anticoagulation management, including escalation protocols.
- Evaluation of drug–food interactions in collaboration with clinical pharmacists and dieticians.

Results

A retrospective analysis from 2018 to 2024 showed a sharp drop in oral anticoagulation therapy (OAC) complications after the nurse-led anticoagulation clinic began. Table 1 shows that OAC-related complications dropped from 15 in 2018 to zero in 2024. This represents a 100% reduction. Both thromboembolic and hemorrhagic events fell to zero, demonstrating significant improvement in safety. The cost burden for OAC complications fell from ₹3.2 million in 2018 to zero in 2024, demonstrating their full elimination. These improvements happened even as the patient volume increased, indicating that the nurse-led model is efficient and scalable.

Year	Total patients	New patients	Complications (n)	Thromboembolic (n)	Hemorrhagic (n)	Estimated Cost (₹)
2018	-	126	15	3	12	3,200,000
2019	-	110	13	2	11	2,424,000
2020	1,482	132	5	2	3	1,452,000
2021	1,600	126	4	2	2	840,000
2022	2,100	140	3	1	2	266,000
2023	2,374	167	3	0	3	151,000

Table 1: Trends in oral anticoagulant (OAC)–related complications and cost before and after implementation of the nurse-led clinic (2018–2024)

Note: Cost estimates represent annual expenditure associated with managing OAC-related complications.

These findings highlight the effectiveness of structured nurse-led monitoring, patient education, and timely follow-up in reducing adverse events and improving the overall cost-effectiveness of anticoagulation management.

Discussion

This study demonstrates that a structured nurse-led anticoagulation clinic significantly improves both clinical outcomes and economic efficiency. Following full implementation of the clinic at KIMSHEALTH, OAC-related complications declined progressively, ultimately reaching zero by 2024. This finding is

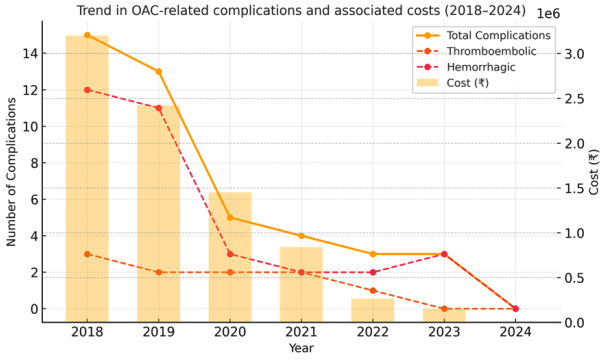

Figure 1: Trend in OAC-related complications and associated costs following implementation of the nurse-led clinic (2018–2024)

Note: This figure shows the trend in oral anticoagulant (OAC)-related complications and management costs over 7 years (2018–2024) following the implementation of a structured nurse-led anticoagulation clinic. The solid lines represent the number of complications, subdivided into thromboembolic and hemorrhagic events. The shaded bars show estimated annual costs of OAC-related adverse outcomes.. A progressive and sustained decline is evident in all categories of complications, with total, thromboembolic, and hemorrhagic events reduced to zero by 2024. Correspondingly, the annual cost burden fell from ₹3.2 million in 2018 to ₹0 in 2024, reflecting a 100% reduction. Despite a steady increase in patient volume, complication rates remained negligible. These findings highlight the effectiveness of nurse-led clinics in optimizing anticoagulation management through systematic patient monitoring, adherence education, and timely dose adjustment.

consistent with existing evidence showing that structured monitoring and patient education reduce adverse events and hospitalizations.[3,6]

Specialized anticoagulation clinics have consistently demonstrated superior anticoagulation control and reduced healthcare costs compared with usual care.[4] Structured monitoring, standardized protocols, and patient education are fundamental to improving the safety and effectiveness of OAC therapy.[3] Randomized trials have further shown that nurse-led anticoagulation management is comparable to physician-led care in terms of safety and clinical outcomes.[5,6]

The development of nurse-managed anticoagulation programs has contributed to improved International Normalized Ratio (INR) monitoring and more appropriate dose adjustment practices. These structured approaches are particularly important for high-risk populations, such as patients undergoing valve replacement, who often have inadequate knowledge of their anticoagulant therapy.[1]

Inadequate knowledge has been associated with reduced adherence and an increased risk of adverse events.[2] Educational interventions targeting patients receiving lifelong oral anticoagulant therapy following mechanical heart valve replacement have demonstrated improved adherence and better therapeutic INR control compared with routine education strategies.[7]

Medication adherence remains a critical determinant of treatment success in chronic therapies, and structured follow-up mechanisms significantly improve adherence and clinical outcomes.[1] Personalized, nurse-led educational interventions have been shown to significantly improve adherence to OAC therapy among patients with mechanical heart valve prostheses[8]. Furthermore, educational strategies delivered by healthcare professionals enhance patient knowledge and reduce medication-related complications[9]. Structured discharge education and multidisciplinary interventions have also been associated with improved patient understanding and reduced medication-related complications.[5]

Comparative evaluations have shown that anticoagulation clinics achieve better therapeutic control and lower healthcare costs than conventional physician-led models[8]. Nurse-led services, in particular, have shown equivalent safety and effectiveness while improving efficiency and reducing the need for frequent clinic visits.[6]

The findings of this study reinforce the effectiveness of nurse-led anticoagulation services in optimizing INR monitoring, facilitating timely dose adjustments, and enhancing patient engagement. Structured follow-up mechanisms, including telephonic support and proactive identification of INR variability, likely contributed to the observed reduction in complications and associated costs. These results align with previous studies demonstrating improved clinical outcomes and cost-effectiveness with nurse-managed anticoagulation programs.,[6,8]

From an operational perspective, the Nurse-Led Oral Anticoagulation Clinic (NL-OACC) at KIMSHEALTH demonstrated strong sustainability by effectively managing increasing patient volumes without compromising the quality of care. The elimination of complication-related costs further highlights the economic value of structured anticoagulation management programs.[8]

Complications and the associated economic burden steadily declined following the implementation of the clinic. The interventions coincided with improved patient monitoring and education, contributing to reduced adverse events, hospitalizations, and costs.

Significance in nursing

- Modern health systems emphasize not only clinical outcomes but also patient safety and education.
- Nurse-led specialty clinics empower patients and families, improve knowledge, and promote safer therapeutic practices.

- Integration of specialty clinic-based education into nursing in-service programs enhances nurses' competence and readiness to identify and prevent complications.
- Strengthening infrastructure for nurse-led clinics contributes to improved patient satisfaction and enhances institutional reputation.
- Maintaining a comprehensive database within the nurse-led oral anticoagulation clinic provides valuable reference material, creates research opportunities, and promotes quality improvement.

Recommendations

- Broader implementation of nurse-led clinics to ensure safe and cost-effective care delivery
- Integration of such clinics into strategic business units to expand institutional reach.
- Advanced training and credentialing of nurses support independent clinical decision-making.
- Adoption of systematic record-keeping to facilitate monitoring, research, and quality improvement.
- Expansion of the nurse-led model to preventive care clinics.

Conclusion

The Nurse-Led Oral Anticoagulation Clinic at KIMSHEALTH, Trivandrum, significantly reduced therapy-related complications and eliminated the financial burden associated with their management. Robust patient surveillance, structured education, and provision of zero-cost care played pivotal roles in enhancing patient safety and improving economic outcomes. These findings support the broader adoption of nurse-led clinics as an equitable and efficient care model.

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Pediatric appendicitis: integrating clinical assessment with an ultrasound-first imaging strategy

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ABSTRACT

Background

Acute appendicitis is the most common surgical emergency in children. Although imaging plays an important role in diagnosis, the overuse of computed tomography (CT) exposes children to avoidable radiation. This study evaluates a diagnostic approach that prioritizes clinical assessment with ultrasonography as the primary imaging modality and CT reserved for selected cases.

Methods

A retrospective observational study was conducted involving 143 children aged 3–15 years who underwent appendectomy for suspected appendicitis. We analyzed data on clinical presentation, laboratory parameters, ultrasonography findings, CT imaging (when performed), intraoperative findings, and outcomes.

Results

Abdominal pain was present in 99.3% of patients. Vomiting occurred in 76.2%. These findings highlight the predominance of clinical features in diagnosis. Ultrasonography was performed in 127 patients and was suggestive of appendicitis in 98 cases (77.1%). The diagnostic performance of ultrasonography showed a sensitivity of 77.4% and a high positive predictive value of 97.96%. CT scans were used selectively in 42 patients (29.4%), mainly for inconclusive ultrasound or diagnostic uncertainty. They were suggestive of appendicitis in all cases. Only three patients (2.1%) had a normal appendix on intraoperative evaluation. This indicates a low negative appendectomy rate.

Conclusion

The diagnosis of pediatric appendicitis remains primarily clinical. However, it can be especially challenging in children under five years of age. Ultrasonography is an effective first-line imaging tool with high positive predictive value. CT should be reserved for cases needing more diagnostic clarification. An ultrasound-first strategy reduces radiation exposure but does not compromise diagnostic accuracy.

Introduction

Acute abdominal pain in children has a broad differential diagnosis, including infectious, inflammatory, musculoskeletal, traumatic, gynecologic, and other etiologies. Among these, acute appendicitis is the most common surgical emergency in the pediatric population.[1] The overall lifetime risk of developing appendicitis is estimated to be approximately 8%, with incidence peaking during the adolescent years.[2] Acute appendicitis in preschool children is rare, accounting for less than 10% of pediatric appendicitis cases.[2,3]

Although appendicitis is uncommon in infants, perforated appendicitis has been seen even in premature neonates.[4] In some cases, perforation may be the final sign of an underlying illness, such as Hirschsprung disease, in neonates.[5] Despite advances in diagnosis and treatment, appendicitis continues to bring significant morbidity. Though rare, it still remains a potential cause of mortality.

Only about 60% of children present with classic symptoms of appendicitis, such as migratory right lower quadrant pain, fever, nausea, and vomiting.[6,7] In younger children, symptoms such as abdominal pain, vomiting, and diarrhoea may initially lead to a diagnosis of acute gastroenteritis in those later confirmed to have appendicitis.[3,6]

Imaging plays an important role in early diagnosis, with ultrasonography and computed tomography (CT) utilized depending on institutional expertise and regional practice patterns.[8]

Despite advances in clinical assessment, laboratory testing, and imaging, early diagnosis of appendicitis in children remains challenging. This is particularly true in younger age groups where presentations are often atypical. Delayed diagnosis increases the risk of complications and associated morbidity. Identifying reliable clinical and imaging predictors of disease severity is essential. This helps improve early diagnosis and optimize management strategies. The present study was undertaken to evaluate the clinical profile and imaging patterns in children with suspected appendicitis.

Materials and methods

This retrospective observational study was conducted at the Department of Pediatric Surgery at KIMSHEALTH, Thiruvananthapuram, Kerala, India. The study included children aged 3–15 years who underwent appendectomy for suspected acute appendicitis during the study period. Patients with incomplete clinical, laboratory, or imaging records were excluded.

Data were retrieved from medical records and operative registers. This included demographic details, duration of symptoms, presenting clinical features, and imaging findings (ultrasonography and CT, when performed). Intraoperative findings were also recorded.

Appendicitis was classified intraoperatively as uncomplicated (inflamed appendix without perforation, gangrene, abscess, or generalized peritonitis). It was considered complicated if there was perforation, gangrene, abscess, or pyoperitoneum. Ultrasonography was used as the first-line imaging modality, with CT performed selectively in cases with inconclusive ultrasonography or diagnostic uncertainty; MRI was not routinely used.

Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Categorical variables were presented as frequencies and percentages.

Results

Clinical profile

Table 1: Age distribution of study population

Age (years)	Number of patients	Percentage (%)
3-6	26	18.3
7-9	47	32.9
10-12	44	30.8
13-15	26	18.2
Total	143	100

A total of 143 patients were included in the analysis. The mean age at presentation was 9.53 ± 2.83 years, with a median age of 9 years. The age ranged from 3 to 15 years. The majority of patients were in the 7–9 years age group (32.9%), followed by the 10–12 years group (30.8%). The 3–6-year and 13–15-year groups each accounted for 18.2% of cases. The interquartile range was 7 to 12 years.

The mean duration of symptoms was 2.15 ± 1.87 days; with a median of 2 days. Symptom duration ranged from 1 to 14 days. The interquartile range was 1- 2 days, indicating that most patients presented early in the course of illness.

Symptomatology

Abdominal pain was the most common presenting symptom, reported in 142 patients (99.3%). Vomiting was the second most common symptom, present in 109 patients (76.2%), followed by fever in 58 patients (40.6%). Loose stools were noted in 24 patients (16.8%),

while sepsis at presentation was rare, observed in 2 patients (1.4%).

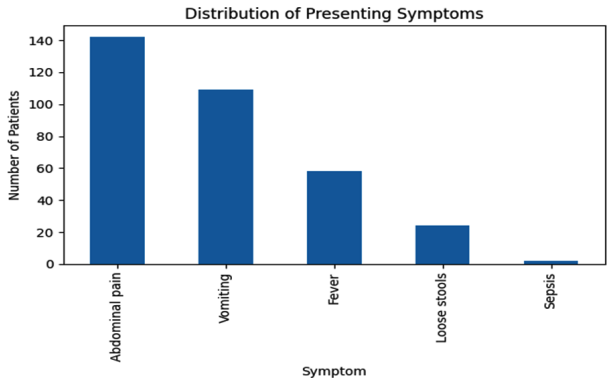

Figure 1: Bar chart showing distribution of presenting symptoms

Ultrasonography

Ultrasound was performed in 127 cases. In the remaining 16 cases, ultrasound was deferred, since there was a strong clinical suspicion of appendicitis. Among those who underwent ultrasonography, findings were suggestive of appendicitis in 98 patients (77.1%), while 29 patients (22.8%) had non-suggestive results. Overall, ultrasonography was suggestive of appendicitis in the majority of patients in this cohort.

Diagnostic performance of ultrasonography was as follows: sensitivity: 77.4 0%, specificity: 33.3%,positive predictive value (PPV): 97.96%,negative Predictive Value (NPV): 3.45%,and overall accuracy: 76.4%.

Most patients who did not undergo ultrasonography at our center had prior imaging at peripheral hospitals. These earlier studies were reportedly negative for appendicitis. In many of these cases, CT imaging was done later for further evaluation.

CT scan was performed and was suggestive of appendicitis in 42 patients (29.4%). CT imaging was selectively used in this cohort, primarily in patients requiring additional diagnostic clarification.

Discussion

Appendicitis is the most common surgical emergency in children. Eighty-six cases of appendicitis per 100,000 children are estimated to occur annually, and this number is increasing.[2] Acute appendicitis is a rapidly progressive condition that can lead to significant morbidity if not diagnosed and managed promptly.[9] Early and accurate diagnosis is essential to prevent septic complications, which are associated with advanced disease.[10,11]

In this cohort, most patients presented within two days of symptom onset. A one-day duration of symptoms was most common (65 patients, 45.5%), followed by two days (46 patients, 32.2%). Presentations after three days were less common, and the numbers decreased as the duration increased. Only isolated cases were seen as late as 10 and 14 days. These data suggest that early medical attention was sought, mainly within the first 48 hours of symptom onset.

Characteristic symptoms of appendicitis include vomiting (96%), fever (85%), and right lower quadrant abdominal pain (81%).[12,13] Overall, abdominal pain and vomiting were the predominant early clinical features; systemic manifestations such as fever were less frequent. Severe systemic manifestations, such as sepsis, were uncommon.

According to Turpin et al., abdominal ultrasound (US), CT, and MRI are all used in the evaluation of pediatric appendicitis.[14] However, at our institute, MRI is not routinely used. This is due to its higher cost, longer scan time, and frequent need for general anesthesia in children. These factors make MRI less practical compared to ultrasonography and CT.

In this study, ultrasonography demonstrated a high sensitivity and positive predictive value for diagnosing appendicitis. However, specificity and negative predictive value could not be reliably assessed due to the low number of negative appendicectomy cases. The low negative predictive value indicates that a non-suggestive ultrasound does not reliably exclude appendicitis. Thus, ultrasonography was highly reliable when positive but less reliable in excluding appendicitis when negative.

Although CT is widely used in many centers, its major limitation is exposure to ionizing radiation. Children are more sensitive to radiation than adults and have a longer life expectancy, so their cumulative risk of radiation-induced adverse effects is higher.[15]

In this cohort, CT findings were suggestive of appendicitis in all patients who underwent CT imaging. Therefore, the calculated diagnostic yield of 100% reflects dataset characteristics rather than true diagnostic performance and should be interpreted cautiously. The other diagnostic performance metrics, such as specificity and negative predictive value, could not be reliably determined. CT was predominantly used as a second-line imaging modality, after non-diagnostic or inconclusive ultrasound findings.

A low negative appendicectomy rate of 2.1% was observed in this cohort. Given the small number of cases, meaningful statistical correlation with laboratory or imaging parameters was not feasible. No consistent clinical or laboratory pattern was identified among these patients. The low negative

appendicectomy rate likely reflects careful patient selection based on combined clinical and imaging assessment.

Conclusion

The diagnosis of acute appendicitis in children continues to rely primarily on careful clinical evaluation. In this cohort, classical clinical features were present in the majority of patients, highlighting the central role of clinical suspicion in diagnosis. Ultrasonography proved to be a reliable first-line imaging modality with a high positive predictive value, supporting its routine use in the initial evaluation of suspected pediatric appendicitis.

Computed tomography was required only in a limited number of patients with inconclusive ultrasound findings or diagnostic uncertainty. The low negative appendicectomy rate observed in this study further supports the effectiveness of a clinical assessment–driven, ultrasound-first diagnostic approach.

Adopting such a strategy may help reduce unnecessary radiation exposure while maintaining diagnostic accuracy in children with suspected appendicitis. The ultrasound has now become the gold standard investigation in the diagnosis of acute appendicitis in children.

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Long-term outcomes of biopsy-proven lupus nephritis: A 5-year retrospective cohort study

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- *end-stage renal disease*
- *immunosuppressive therapy*
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- *retrospective cohort*

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ABSTRACT

Aim: To perform a retrospective analysis evaluating the long-term outcomes of patients with lupus nephritis treated at our centre.

Methods: This retrospective observational study included all patients with biopsy-proven lupus nephritis diagnosed at our hospital between 2012 and 2018, with follow-up data available through 2023, ensuring a minimum follow-up of 5 years.

Results: The average age of the study population (n=55) was 37.12 years. The average age at diagnosis of lupus nephritis was 30.24 years.

All 55 patients underwent renal biopsy. Among them, 26 patients had Class IV lupus nephritis, 6 had Class III, 8 had Class V, and 4 had Class VI. Two patients each had Class I, Class II disease, while the remaining patients had mixed classes.

Proliferative lupus nephritis was treated with corticosteroids in combination with intravenous cyclophosphamide (Euro-Lupus protocol) or mycophenolate.

Four patients conceived during the study: one had a successful pregnancy without flare, one developed preeclampsia with lupus flare, one had a medical termination, and one had a lupus flare with worsening renal function.

Five patients died during follow-up. Eleven patients developed chronic kidney disease (CKD), of which four progressed to end-stage renal disease (ESRD). Among these ESRD patients, two underwent renal transplant and currently have good graft function.

Conclusion: The overall survival rate was 90.9%, indicating a favorable long-term outcome in lupus nephritis at our centre. Outcomes are influenced by adherence to immunosuppressive therapy, careful monitoring, and prevention of treatment default. Pregnancy-related morbidity may be reduced with appropriate pre-pregnancy counselling and management.

Introduction

Lupus nephritis is a form of glomerulonephritis and is among the most common complications of systemic lupus erythematosus (SLE). It affects approximately 35-60% of patients with SLE.[1] As our understanding of lupus nephritis grows, it is now evident that optimal management depends on rapid and effective control of renal disease, followed by sustained remission using appropriate immunosuppressive agents to prevent relapses.

Long-term outcomes have traditionally been considered suboptimal, with chronic kidney disease (CKD) rates of nearly 50% in studies from Asia.[2] However, we hypothesize that good initial treatment and prevention of disease flares could result in good long-term outcomes. Therefore, we reviewed patients with lupus nephritis who had at least 5 years of follow-up to evaluate long-term outcomes.

Aim

To determine long-term outcomes in patients with biopsy-proven lupus nephritis in terms of response to therapy, disease flare, mortality, treatment-related complications, and overall outcomes.

Methods

This retrospective observational study was conducted among all patients diagnosed with biopsy-proven lupus nephritis at KIMSHEALTH, Trivandrum, between 2012 and 2018, who were available for follow-up for at least 5 years after that diagnosis.

The clinical details, the biopsy findings, and the treatment protocol were extracted from hospital records. Outcomes assessed included complete or partial remission, progression to CKD, mortality, and pregnancy outcomes within the cohort.

Results

Data were available for 55 out of the 66 biopsied patients; the remaining patients were lost to follow-up. The average age of the study population(n=55) was 37.12 years, and the average age at which lupus nephritis was diagnosed was 30.24 years. At the time of biopsy, 45 patients were on treatment for SLE; four had stopped treatment on their own; one was on homeopathic treatment; one was on ayurvedic treatment; and four were diagnosed with SLE during nephrology evaluation Baseline characteristics are summarized in Table 1. All patients had proteinuria, and most had evidence of urinary deposits, particularly microhematuria.

Age at diagnosis	30.24 years
Males vs females	8 vs 47 ratio approximate ratio 1:6
Hypertension	17 (30.9%)
Edema	16(29.1)
Proteinuria	55(100%)
Microhematuria	45(81.8%)
RENAL BIOPSY FINDINGS	
Class I	2
Class II	2
Class III	6
Class IV	26
Class V	8
Class VI	4
Mixed classes	4/5 (4) 4/6 (2) 3/5 (1)

Table 1

Renal biopsy was performed in all 55 patients. Class IV lupus nephritis was the most common histological pattern (47%), followed by Class III and Class V lesions. These findings were consistent with two other studies from the region. Notably, despite evidence of proteinuria, 2 patients had only Class I lesions, and 2 had Class II disease (Table 2).

Patients with class III, IV, and V lupus nephritis were treated with intravenous cyclophosphamide and steroids according to the Euro-Llupus protocol[5] or with mycophenolate and steroids. Among the cohort, 45 patients (82%) adhered to prescribed therapy, 6 (10.9%) defaulted, and 4 (7.2%) opted for alternative treatments.

REGIMEN	RENAL FUNCTION ON FOLLOW UP			**FISCHERS EXACT TEST**
	NORMAL	DERANGED	TOTAL	P VALUE O.O36
EUROLU-PUS	17	4	21	
MMF and IV methyl-predniso-lone	6	9	15	
MMF/Aza with oral steroids	14	5	19	

Table 2: Initial regimen and outcomes

Among patients with proliferative lupus nephritis (Class III/IV; n = 39), 26 (66.6%) achieved complete or partial remission. Four patients (10.2%) progressed to CKD Stage 4, three (7.6%) to CKD Stage 3, and six (15.4%) developed end-stage renal disease (ESRD). All patients who progressed to ESRD had either defaulted on therapy or pursued alternative treatments.

Among the 8 patients with membranous lupus nephropathy, 5 achieved complete remission (62.5%) and 1 maintained proteinuria with partial remission (12.5%). One progressed to ESRD (a treatment defaulter), and one patient died.

Overall, 5 deaths (9.1%) occurred in the cohort. Eleven patients developed CKD Stage III or higher, including 4 with ESRD. Of these, 2 patients underwent renal transplantation and currently have good graft function.

The Euro-Lupus protocol appeared to yield better outcomes compared to other treatment regimens (Table 2).

There were 4 pregnancies during the study period. One resulted in an uneventful full-term delivery, one was complicated by lupus flare with preterm delivery, one required medical termination of pregnancy due to early lupus flare and renal dysfunction, and one had worsening renal function despite a successful pregnancy outcome.

Discussion

Lupus nephritis is a severe manifestation of lupus that requires prompt induction therapy and long-term treatment to address renal inflammation, prevent immunological damage, and attain long-term suppression of lupus activity. In this cohort of patients followed for a minimum of 5 years, we observed that, when treated appropriately, both short- and long-term outcomes are favorable. However, treatment non-adherence and the use of alternative medical therapies emerged as important causes of treatment failure and poor outcomes.

Despite these challenges, we achieved an overall patient survival rate of 50 out of 55 patients (90.9%) at a minimum follow-up of 5 years.

Both survival and response rates in our cohort were marginally better than those reported in previous studies from India.

Our findings also suggest that the Euro-Lupus protocol using intravenous cyclophosphamide may be associated with better outcomes compared to other treatment regimens. However, this could be due to the small numbers and heterogeneity of patients at presentation in the other groups.

Consistent follow-up and strict adherence to therapy appear to be key determinants of favorable outcomes. Pregnancy in patients with lupus nephritis was associated with significant morbidity in our cohort. However, many of these complications might have been mitigated through better patient selection, preconception counseling, and planned pregnancies.

Emerging targeted therapies, including biologics such as belimumab and obinutuzumab, as well as complement inhibitors, offer promising avenues for improving outcomes in lupus nephritis. So, the prognosis for lupus seems good.[6,7,8]

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Spectrum of late gadolinium enhancement patterns in cardiac magnetic resonance imaging

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- *myocarditis*
- *cardiac sarcoidosis*

ABSTRACT

Late gadolinium enhancement (LGE) imaging is a fundamental technique in cardiac magnetic resonance imaging (CMR) for the detecti0n myocardial fibrosis, necrosis, and infiltration. The pattern and distribution of LGE provide valuable insights into the underlying etiology of myocardial injury and enable differentiation between ischemic and non-ischemic cardiomyopathies.

In this case series, we present a spectrum of LGE patterns observed in patients undergoing cardiac MRI, including ischemic cardiomyopathy, myocarditis, cardiac sarcoidosis, amyloidosis, dilated cardiomyopathy, and hypertrophic cardiomyopathy. Each condition exhibits characteristic enhancement patterns that facilitate accurate diagnosis, risk stratification, and clinical management.

Through illustrative cases, this article highlights the diagnostic value of recognizing specific LGE distributions and their correlation with distinct myocardial pathologies. An understanding of these patterns reinforces the role of cardiac MRI as a powerful, non-invasive modality for the evaluation of cardiomyopathies.

Introduction

Cardiac magnetic resonance (CMR) imaging has emerged as a powerful, non-invasive imaging modality for comprehensive evaluation of myocardial structure, function, and tissue characterization. Among the various techniques used in CMR, late gadolinium enhancement (LGE) imaging plays a central role in identifying myocardial fibrosis, scar formation, and infiltrative processes. LGE imaging is typically performed 10–15 minutes after administration of a gadolinium-based contrast agent using inversion-recovery gradient-echo sequences. In this technique, the signal from normal myocardium is nulled, allowing regions with increased extracellular volume — such as fibrosis, necrosis, or infiltration — to appear hyperintense. This approach provides a unique capability to delineate myocardial tissue abnormalities that may not be evident in conventional imaging modalities.[1]

One of the most significant advantages of LGE imaging is its

ability to differentiate ischemic from non-ischemic myocardial disease based on enhancement patterns. Ischemic cardiomyopathies typically demonstrate subendocardial or transmural enhancement conforming to a coronary artery distribution. In contrast, non-ischemic cardiomyopathies often exhibit mid-myocardial, subepicardial, or diffuse patterns that do not correspond to a vascular territory.[1]

Recognizing these characteristic patterns is essential for accurate diagnosis, prognosis assessment, and therapeutic decision-making. This case series presents a spectrum of LGE patterns observed across different myocardial pathologies and underscores the diagnostic value of LGE imaging in cardiac MRI.

Case presentation

Case 1: Ischemic cardiomyopathy

A patient with a history of coronary artery disease underwent cardiac MRI for evaluation of myocardial viability. Imaging revealed thinning of the anteroseptal, anterior, and anterolateral segments at the mid-ventricular level, as well as involvement of the septal and anterior segments at the apical level and the apex. Late gadolinium enhancement demonstrated transmural enhancement in these regions, consistent with a prior myocardial infarction in the left anterior descending (LAD) artery territory (Figure 1).

In another patient, cardiac MRI demonstrated a narrow-necked akinetic outpouching arising from the inferolateral wall of the basal to mid left ventricle. The wall of this outpouching showed LGE, consistent with a left ventricular pseudoaneurysm secondary to a previous infarction (Figure 2).

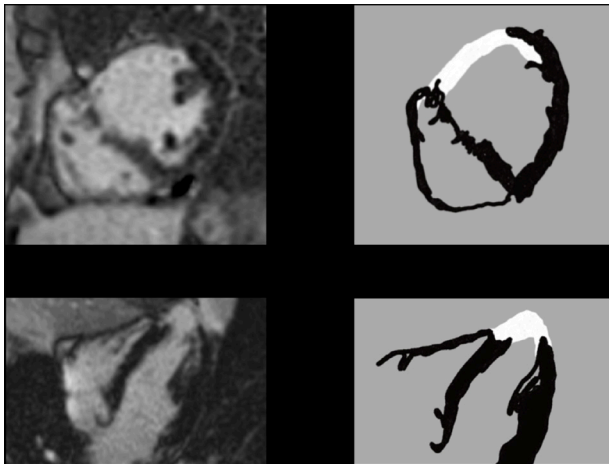

Figure 1: Thinning of the anteroseptal, anterior, and anterolateral segments at the mid-ventricular level; septal and anterior segments at the apical level and apex, with transmural LGE—transmural infarction in the LAD territory.

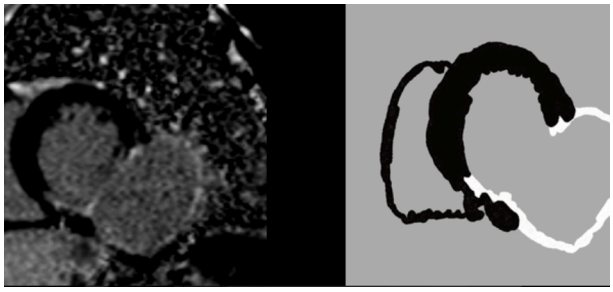

Figure 2: Akinetic outpouching from the inferolateral aspect of the basal and mid-ventricular wall of the left ventricle, with LGE of the walls of the pseudoaneurysm _ pseudoaneurysm from the lateral wall of the LV.

These findings illustrate the typical ischemic LGE pattern, in which enhancement begins in the subendocardium and may extend transmurally depending on the severity of myocardial injury.

Case 2: Myocarditis

A young patient presenting with chest pain and elevated cardiac enzymes underwent cardiac MRI for evaluation of suspected myocarditis. LGE imaging demonstrated nodular and coalescing areas of enhancement involving the septal and lateral segments of the apical left ventricle and the apex. The enhancement predominantly involved the subepicardial and mid-myocardial layers (Figure 3).

This pattern of LGE, characterized by subendocardial sparing and a lack of conformity to coronary arterial distribution, is typical of myocarditis. The presence of subepicardial or mid-wall enhancement in the lateral wall of the left ventricle is particularly suggestive of inflammatory myocardial injury.

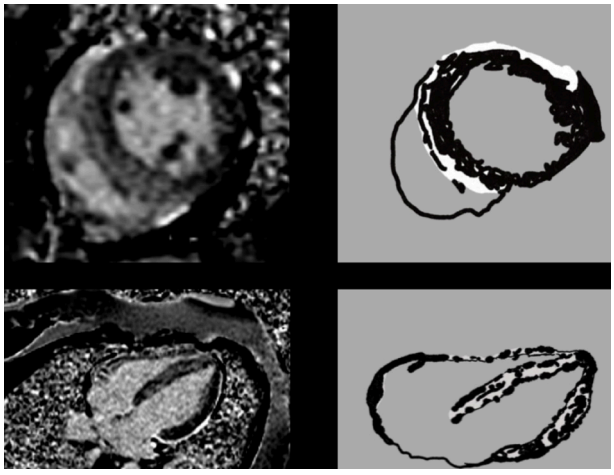

Figure 3: Linear mid-myocardial LGE in the interventricular septum and basal anterolateral left ventricular wall, with additional enhancement at the superior and inferior right ventricular insertion points, suggestive of acute myocarditis.

Case 3: Cardiac sarcoidosis

Cardiac MRI in a patient with systemic sarcoidosis revealed irregular areas of mid-wall LGE involving the interventricular septum and adjacent myocardial segments (Figure 4).

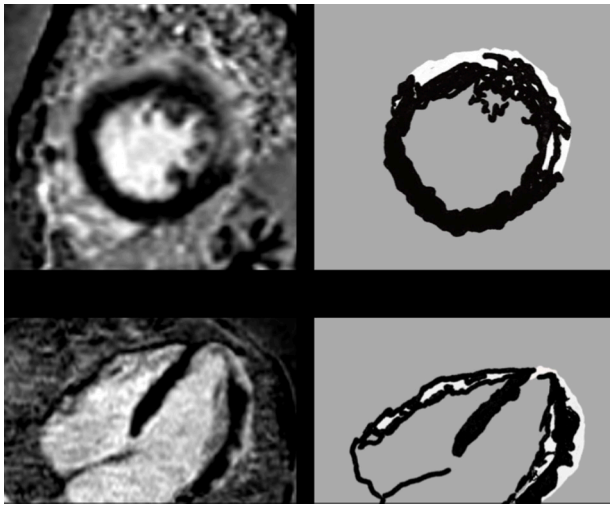

Figure 4: Nodular and coalescing areas of LGE involving the anterior and lateral segments of the apical left ventricle and the apex, predominantly involving the subepicardial and mid-myocardial layers, consistent with cardiac sarcoidosis. Irregular mid-wall LGE.

Cardiac sarcoidosis typically presents with patchy, multifocal LGE patterns, often involving the basal septum or lateral wall. The combination of structural abnormalities on CMR and metabolic activity on FDG PET imaging further supports the diagnosis.

Case 4: Cardiac amyloidosis

In another case, cardiac MRI demonstrated diffuse subendocardial enhancement involving the left ventricle, extending into the inner two-thirds of the myocardium. Additional enhancement was noted within the interatrial septum (Figure 5).

This diffuse, non-territorial subendocardial LGE pattern is characteristic of cardiac amyloidosis and reflects widespread amyloid deposition within the myocardial extracellular space.

Case 5: Dilated cardiomyopathy

A patient with dilated cardiomyopathy underwent cardiac MRI for further evaluation. LGE imaging demonstrated linear mid-myocardial enhancement involving the interventricular septum and free wall at the basal and mid-ventricular levels, with additional involvement of the lateral segment at the apical level (Figure 6).

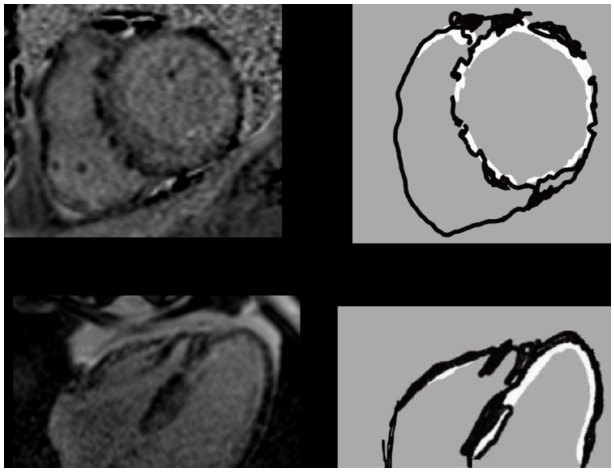

Figure 5: LGE images show diffuse non-territorial enhancement of the subendocardium up to the inner two-thirds of the myocardium. LGE is also noted in the interatrial septum _diffuse subendocardial enhancement (amyloidosis).

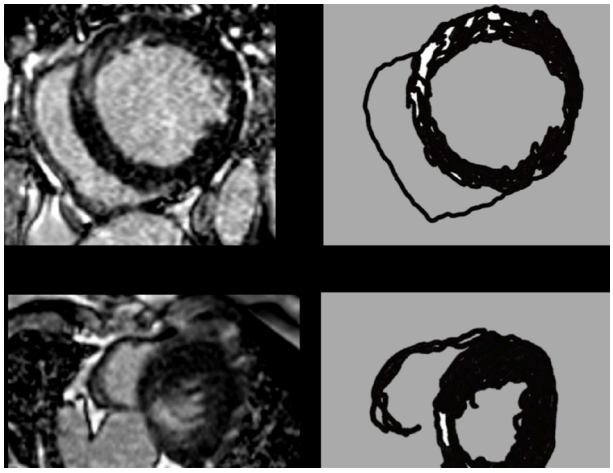

Figure 6: Linear mid-myocardial LGE involving the interventricular septum and free wall at the basal and mid-ventricular levels, with additional involvement of the inferior segment at the apical level _mid-wall LGE in DCM, a predictor of poor prognosis

Mid-wall LGE in dilated cardiomyopathy is a well-recognized imaging finding and has been associated with an increased risk of adverse cardiac outcomes, including arrhythmias and progressive heart failure.

Case 6: Hypertrophic cardiomyopathy

Cardiac MRI in a patient with hypertrophic cardiomyopathy demonstrated asymmetric hypertrophy of the anteroseptal and inferoseptal segments at the basal and mid-ventricular levels. LGE imaging showed multifocal and confluent areas of mid-myocardial enhancement within the hypertrophied septal myocardium (Figure 7).

The presence of LGE in hypertrophic cardiomyopathy represents myocardial fibrosis and has been associated

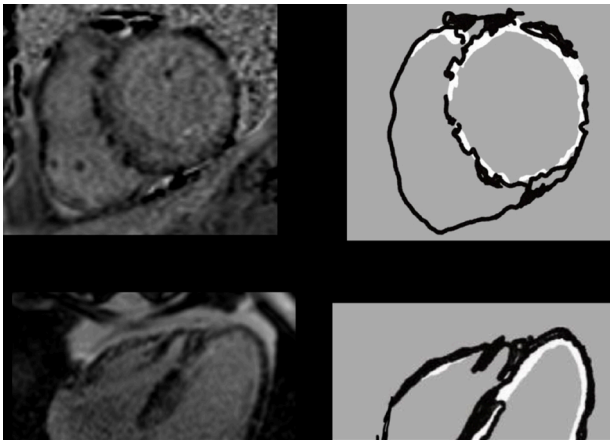

Figure 7: Asymmetric hypertrophy of the anteroseptal and inferoseptal segments at the basal and mid ventricular levels, with multifocal and confluent mid myocardial LGE / fibrosis in the hypertrophied septal myocardium- hypertrophic cardiomyopathy (HCM) with mid myocardial LGE.

with an increased risk of ventricular arrhythmias and sudden cardiac death.

Discussion

Late gadolinium enhancement (LGE) imaging has significantly advanced the evaluation of myocardial disease by enabling direct visualization of myocardial fibrosis and scarring. The technique relies on the differential distribution of gadolinium contrast between normal and diseased myocardium. Areas with expanded extracellular space retain gadolinium longer, appearing hyperintense on delayed imaging.[1]

One of the most important clinical applications of LGE imaging is its ability to distinguish ischemic from non-ischemic cardiomyopathies. In ischemic injury, myocardial necrosis begins in the subendocardium due to its higher metabolic demand and relative vulnerability to ischemia. As ischemia severity increases, the injury progresses transmurally. Consequently, LGE patterns in ischemic cardiomyopathy typically follow the distribution of coronary arteries and involve the subendocardial or transmural layers.[1]

In contrast, non-ischemic cardiomyopathies often demonstrate enhancement patterns that spare the subendocardium and involve the mid-myocardial or subepicardial layers. For example, myocarditis typically produces patchy subepicardial or mid-wall enhancement, especially in the lateral wall. Cardiac sarcoidosis often exhibits multifocal and irregular areas of LGE involving the septum or free wall.[2]

In infiltrative cardiomyopathies such as amyloidosis, the enhancement pattern tends to be diffuse and may involve the entire subendocardium or even extend transmurally. This pattern reflects extensive deposition of abnormal proteins within the myocardial interstitium.[2]

Dilated cardiomyopathy frequently demonstrates linear mid-wall enhancement in the interventricular septum, which is thought to represent replacement fibrosis. Several studies have shown that the presence of LGE in dilated cardiomyopathy is associated with adverse clinical outcomes and a higher risk of arrhythmias.[3]

Similarly, in hypertrophic cardiomyopathy, LGE corresponds to areas of myocardial fibrosis and may serve as an important marker for risk stratification. The extent of LGE has been correlated with an increased risk of sudden cardiac death.[4]

Therefore, accurate recognition and interpretation of these characteristic enhancement patterns is essential for clinicians and radiologists. LGE imaging not only aids in establishing the diagnosis but also provides prognostic information and can influence therapeutic decisions.[5]

Conclusion

Late gadolinium enhancement imaging is an essential component of cardiac MRI, providing critical insights into myocardial tissue characterization. Distinct LGE patterns help differentiate ischemic from non-ischemic cardiomyopathies and identify specific disease processes such as myocarditis, sarcoidosis, amyloidosis, dilated cardiomyopathy, and hypertrophic cardiomyopathy.

Recognition of these characteristic patterns enables accurate diagnosis, risk stratification, and improved clinical management. As cardiac MRI continues to evolve, LGE imaging will remain a cornerstone technique for evaluating myocardial injury and fibrosis, ultimately contributing to improved patient outcomes.

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Laparoscopic management of pediatric inguinal hernia: A single-center experience

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ABSTRACT

Aim: This study aims to document our experience in laparoscopic hernia repair in the pediatric population.

Methods: A total of 11 hernia repairs were performed in 8 children. Using standard three-port laparoscopy, the peritoneum at the open deep ring was dissected, the distal sac disconnected, and the ring was closed using a purse-string technique with intracorporeal suturing using 4-0 Vicryl sutures.

Results: Of the 8 children,3 were boys. In 3 of the children, a contralateral open deep inguinal ring with a patent processus vaginalis was found, and bilateral herniotomy was performed in the same sitting. The operative time ranged from 65 to 75 minutes for unilateral herniotomies, 90 to 100 minutes for bilateral cases, and 95 minutes for a unilateral complicated (obstructed) hernia repair.

Postoperative inguinoscrotal edema was not observed in any case except in one patient with an incarcerated, obstructed hernia. All cases are under follow-up, and no recurrences have been reported to date.

Conclusion : Laparoscopy is a safe and effective alternative to the conventional open approach for both elective and emergency herniotomies in children. It also has additional advantages, including minimal handling of the cord, the ability to address contralateral hernias in a single sitting, and the identification of Müllerian structures in girls with bilateral inguinal hernia.

Introduction

Inguinal hernia repair is one of the most commonly performed operations in pediatric practice.[1] The traditional 'open' inguinal herniotomy remains the gold standard of hernia repair in children worldwide. However, since the early 2000s, laparoscopic techniques have become increasingly common.[2,3] Laproscopy provides several potential advantages, including reduced post-operative pain, faster recovery, and importantly, the ability to visualize the contralateral deep inguinal ring and minimal handling of the spermatic cord structures.[4]

The techniques of laparoscopic inguinal hernia repair in children are not yet standardized, which may account for the variability in recurrence rates reported in the literature.[5,7]

Both intracorporeal and extracorporeal methods have been described.[8,9] The method involving incision of the peritoneum at the deep inguinal ring, disconnection of the distal sac, followed by the closure of the peritoneum at the deep ring, is said to have low recurrence rates, comparable to those of standard open herniotomy.[10]

Laparoscopic repair provides good visual exposure, facilitates assessment of the contralateral side, and minimizes the handling of cord structures, thereby reducing the risk of iatrogenic trauma.[11]

However, controversies regarding the use of laparoscopy persist due to potential damage to the intra-abdominal structures because of the breached peritoneal cavity, longer operative times, and higher costs.[12]

In this study, we adopt the laparoscopic approach of peritoneal incision at the deep inguinal ring, disconnection of the distal sac, and closure of the peritoneum using purse-string intracorporeal suturing.

Case presentations

Case 1

A 4-year-old boy presented with a right inguinal hernia that was detected one week prior to admission. Laparoscopic right herniotomy was performed. The left deep inguinal ring was found to be closed. The operative duration was approximately 65 minutes. The patient was discharged within 24 hours and remains well during follow-up.

Case 2

A 5-year-old girl presented with a right inguinal hernia that was noticed since infancy. Laparoscopic right herniotomy was performed with an operative duration of approximately 75 minutes. The patient was discharged on the same day, experienced no postoperative complications, and is well on follow-up.

Case 3

A 2-year-old female child with a diagnosis of Complete Androgen Insensitivity Syndrome (CAIS) presented with bilateral large inguinal hernias. Laparoscopic bilateral herniotomy was performed, enabling intraoperative confirmation of the absence of Müllerian structures while preserving the testes within the inguinal canal. The procedure lasted 100 minutes. Postoperative recovery was uneventful, and the patient was discharged the following day. The patient continues to do well on follow-up.

Case 4

A 4-year-old girl presented with a right inguinal hernia, with ultrasonography suggesting a bilateral inguinal hernia. Laparoscopy revealed bilateral patulous internal rings, and she underwent bilateral laparoscopic herniotomy with an operative time of 100 minutes. The presence of Müllerian structures was confirmed to rule out CAIS. She had an uneventful hospital stay of a day and remains well on follow-up.

Case 5

A 7-year-old girl presented with a left inguinal hernia noted since early childhood. She underwent laparoscopic left herniotomy with an operating time of 60 minutes. She had a smooth postoperative course and was discharged the same day. She is well on follow-up with no complications.

Case 6

An 8-year-old boy underwent laparoscopy for an undescended testis. The intra-abdominal left testis was found to be small, atrophic, and associated with a short vas deferens and a patulous left deep inguinal ring. Left orchiectomy with closure of the open deep ring was performed. The postoperative recovery was uneventful, and the patient was discharged the following day. The patient continues asymptomatic on follow-up.

Case 7

A 2-year 9-month-old male patient presented with signs of intestinal obstruction and an irreducible right inguinal hernia of one day's duration. Emergency laparoscopy allowed for the reduction of hernial contents under direct visualization, confirming viability. Herniotomy followed the standard technique

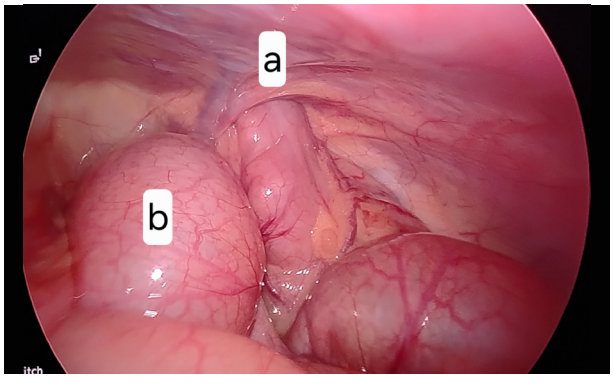

Figure 1: (a) The bowel herniating into the right deep ring; (b) Proximal dilated small bowel, suggestive of obstruction.

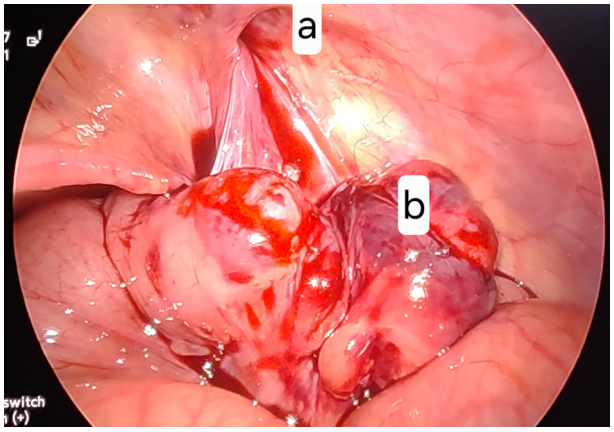

Figure 2: (a) Open right deep ring after reduction of contents; (b) Reduced small bowel showing intact viability.

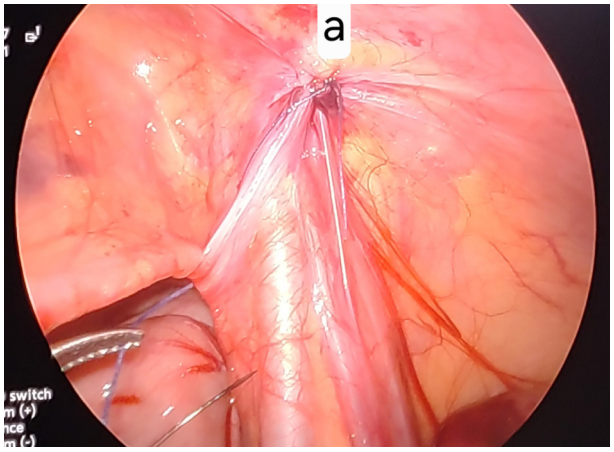

Figure 3: The closed deep ring after herniotomy.

of incision of the peritoneum and closure. Operative time was approximately 95 minutes. The postoperative recovery was uneventful except for minimal right inguinal edema. The patient was discharged on postoperative day four and is well on follow-up.

Case 8

An 8-year-old girl presented with left inguinal swelling of long-standing duration. Laparoscopic examination identified bilateral open internal rings. Laparoscopic bilateral herniotomy was performed in a single session, with an operative duration of 90 minutes. The recovery was uneventful, and the patient was discharged the following day. She remains well on follow-up.

Discussion

Surgical correction of inguinal hernias (IHs) is one of the most commonly performed procedures in pediatric surgery. The increasing adoption of laparoscopic inguinal hernia repair is attributable to its ability to identify and simultaneously manage a contralateral patent processus vaginalis.[13] In our series, we found that laparoscopy is a safe approach for children with inguinal hernia. It provides excellent visualisation, minimal handling of the vas deferens and other cord structures in boys, and aids in the examination of Müllerian structures in girls with bilateral inguinal hernia.

In our study, laparoscopy also aided in the identification of a contralateral patent processus vaginalis in 2 out of 8 children, which might have become symptomatic following conventional open herniotomy on the recognised side. Chong et al., in a retrospective review of 1,697 pediatric inguinal hernia cases, reported a significantly higher incidence of metachronous contralateral inguinal hernia following open repair (10.7 per 1000 person-years) compared to laparoscopic repair (3.4 per 1000 person-years). Furthermore, when compared with open surgery, the hazard ratio for requiring a second operation (for either recurrence or metachronous contralateral hernia) was 0.3 with the laparoscopic approach, indicating a substantially lower risk.[14]

Complete Androgen Insensitivity Syndrome (CAIS), also known as testicular feminization syndrome, is a rare disorder of sex development characterized by a typical female phenotype and a 46XY karyotype. Patients with CAIS commonly present with primary amenorrhea and may also have unilateral or bilateral inguinal hernias due to the presence of undescended testes within the inguinal canal.[15] Most studies suggest that orchiectomy is preferably performed in early adulthood or the post-pubertal period, to allow for the aromatization of testosterone to estrogen, which supports normal pubertal development.

In our series, two girls presented with bilateral inguinal hernias, and laparoscopy confirmed the presence of Müllerian structures in both, thereby ruling out CAIS. In contrast, in the known case of CAIS in our series, laparoscopy confirmed the absence of Müllerian structures and allowed bilateral herniotomy while preserving the testes to maintain hormonal support.[15,16]

Laparoscopic management of incarcerated or irreducible inguinal hernia in children is increasingly recognized as a safe and effective alternative to open repair. In such cases, open surgery can be technically challenging due to edema and distortion of the inguinal anatomy, increasing the risk of injury to the vas deferens and spermatic vessels.[17]

Laparoscopy avoids dissection through inflamed tissues and provides magnified visualization, enabling controlled reduction of the herniated contents and direct assessment of bowel viability.[18,19] In addition, laparoscopy allows inspection of the contralateral

internal ring and simultaneous repair of a patent processus vaginalis, thereby reducing the risk of metachronous hernia.

Kaya et al. demonstrated the feasibility of this approach in children with irreducible hernias following failed manual reduction, with favorable outcomes. Other studies and systematic reviews have similarly supported the safety and diagnostic advantages of laparoscopy in both elective and emergency pediatric inguinal hernia repair.[20,21]

In our series, the child presenting with an obstructed hernia was successfully managed laparoscopically on an emergency basis, with the advantages described above.

Conclusion

Laparoscopic herniotomy is a safe and effective alternative to the conventional open approach for the management of pediatric inguinal hernias in both elective and emergency settings. The technique offers excellent visualization of the internal ring, enabling precise closure while minimizing manipulation of the spermatic cord structures. It also enables assessment of hernia contents and bowel viability in complicated cases, such as incarcerated or obstructed hernias.

An additional advantage is the ability to inspect the contralateral internal inguinal ring and repair a patent processus vaginalis during the same procedure, thereby potentially preventing metachronous hernias and avoiding the need for a second anaesthetic exposure. Laparoscopy also helps in the identification of Müllerian structures in girls presenting with bilateral inguinal hernias, thereby helping rule out conditions such as Complete Androgen Insensitivity Syndrome (CAIS).

In our series, laparoscopic herniotomy was associated with acceptable operative times and favorable short-term outcomes. No recurrences were observed during follow-up. These findings support the feasibility and safety of laparoscopic herniotomy in children. With increasing surgical experience and standardization of techniques, laparoscopy is likely to become an important component of the management of pediatric inguinal hernias.

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Sternal fractures in blunt chest trauma: Diagnostic value of CT and clinical and radiological insights

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ABSTRACT

Background: Sternal fractures are relatively uncommon injuries following blunt chest trauma; however, they may be associated with significant intrathoracic complications.

Methods: We present a case series of three patients with sternal fractures resulting from blunt chest trauma. Clinical features, computed tomography (CT) findings, and management strategies were evaluated.

Results: CT imaging identified fracture patterns and associated complications, including retrosternal hematoma and haemothorax. One patient required surgical intervention due to hemodynamic instability, while the other two were managed conservatively with favourable outcomes.

Conclusion: CT is the imaging modality of choice for diagnosing sternal fractures and associated thoracic injuries. Early identification facilitates appropriate management and improves clinical outcomes.

Introduction

Sternal fractures represent a relatively small proportion of blunt thoracic injuries, with a reported incidence ranging from 3% to 8%. These injuries are most frequently associated with high-energy mechanisms, such as road traffic collisions and direct anterior chest trauma. Although isolated sternal fractures are generally associated with a favourable prognosis, their presence raises concern for concomitant intrathoracic injuries, including myocardial contusion, pulmonary contusion, and mediastinal hematoma.

Historically, lateral chest radiography was employed for diagnosis; however, its sensitivity is limited, particularly for nondisplaced fractures. Multidetector computed tomography (CT) has emerged as the preferred imaging modality due to its superior diagnostic accuracy and ability to detect associated thoracic injuries.

This case series aims to highlight the clinical presentation,

radiological features, and management strategies of sternal fractures diagnosed using CT imaging in the emergency setting.

Methods

This is a retrospective case series that includes three patients who presented to the emergency department with blunt chest trauma. All patients underwent CT imaging of the chest as part of their trauma evaluation. Clinical presentation, imaging findings, management approaches, and outcomes were reviewed.

Case 1

A 63-year-old male presented to the emergency department following a high-impact road traffic collision involving a four-wheeler. He reported severe anterior chest pain, rated 9/10 on the Numeric Rating Scale (NRS).

On initial assessment, the patient was hemodynamically unstable, with a blood pressure of 86/54 mmHg, a heart rate of 118 beats/min, a arespiratory rate of 26 breaths/min, and an oxygen saturation of 90% on room air. Physical examination revealed marked tenderness over the anterior chest wall and decreased breath sounds over the right hemithorax, without visible chest wall deformity. Electrocardiography (ECG) demonstrated sinus tachycardia without ischemic changes. Serum troponin levels were mildly elevated, suggestive of possible myocardial contusion.

Computed tomography (CT) of the chest demonstrated a sternal fracture associated with a large retrosternal hematoma measuring approximately 3.7 × 10.7 × 14.6 cm, along with a right-sided haemothorax (Figures 1 and 2). The patient underwent emergency intercostal drain placement, followed by surgical intervention, including evacuation of the retrosternal hematoma via sternotomy. Postoperatively, pain improved to 3/10 on the NRS, and the patient gradually recovered functionally, achieving independent ambulation at discharge.

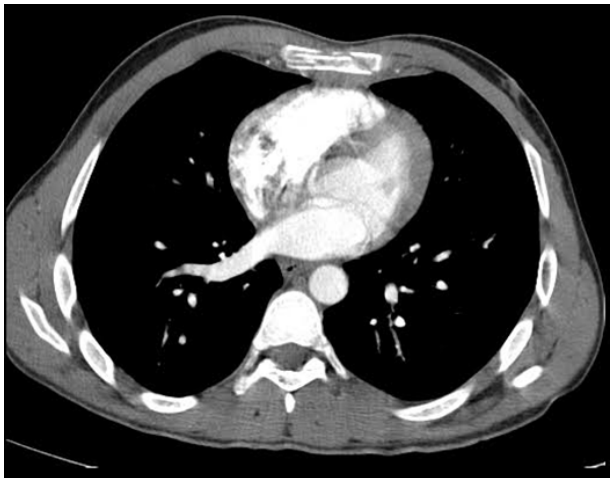

Figure 1: Axial contrast-enhanced CT image of the chest demonstrating a fracture of the manubrium sterni, characterized by cortical irregularity and surrounding soft tissue edema, consistent with acute traumatic injury

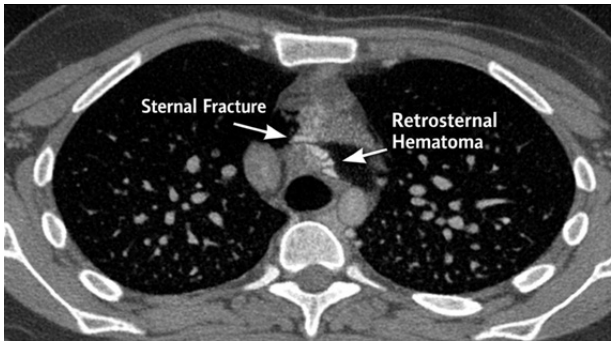

Figure 2: Axial CT image demonstrating a displaced fracture of the sternal body, characterized by cortical discontinuity and an associated hyperdense retrosternal collection, consistent with a hematoma.

Case 2

A 49-year-old male presented to the emergency department following a road traffic collision involving a car and a truck. He reported an acute onset of central chest pain, rated 6/10 on the NRS, immediately after the impact.

On initial assessment, the patient was hemodynamically stable, with a blood pressure of 120/70 mmHg, heart rate of 88 beats/min, respiratory rate of 18 breaths/min, and oxygen saturation of 98% on room air. Physical examination revealed localized tenderness over the sternum without swelling, deformity, or evidence of respiratory compromise. ECG showed a normal sinus rhythm. Serum troponin levels were within normal limits.

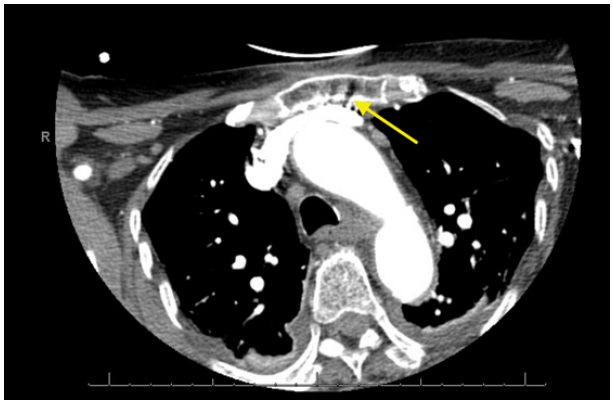

Figure 3: Axial CT image demonstrating a linear, undisplaced fracture of the sternal body, evident as a cortical discontinuity without displacement or associated intrathoracic injury.

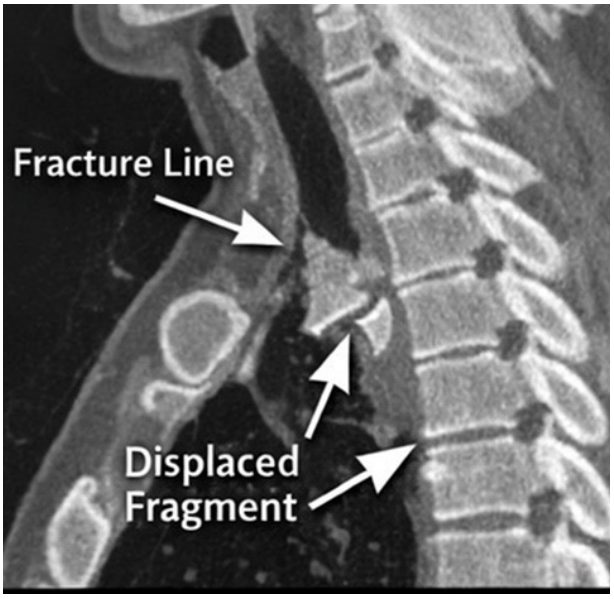

Figure 4: Sagittal reconstructed CT image demonstrating a displaced sternal fracture with posterior displacement of fracture fragments, clearly delineating fracture morphology.

CT imaging of the chest demonstrated a linear, undisplaced fracture of the sternal body, with no associated intrathoracic injury (Figures 3 and 4). The patient was managed conservatively with adequate analgesia and close clinical observation. Pain improved to 2/10 on NRS prior to discharge. He was able to perform activities of daily living without limitation and was discharged with outpatient follow-up.

Case 3

A 50-year-old male presented to the emergency department following a motorcycle accident in which he sustained blunt anterior chest trauma from the handlebars. He reported localized chest pain rated 7/10 on the NRS.

On initial evaluation, the patient was hemodynamically stable with normal vital parameters. Physical examination revealed tenderness over the upper sternum, without deformity or signs of respiratory distress. ECG demonstrated a normal sinus rhythm, and serum troponin levels were within normal range.

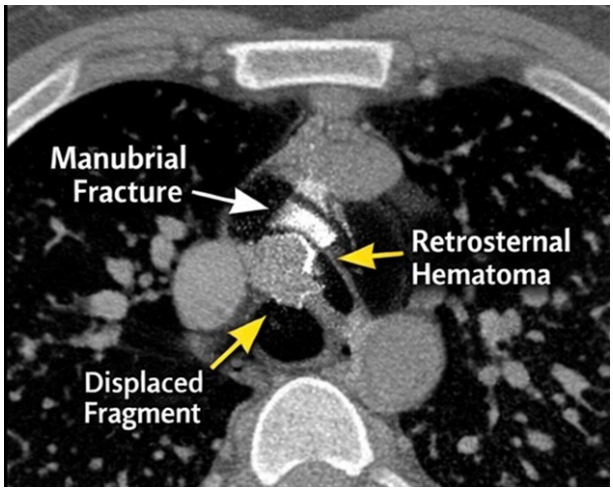

Figure 5: Axial CT image showing a fracture of the manubrium sterni, with adjacent soft tissue swelling and a small retrosternal hematoma.

Variable	Case 1	Case 2	Case 3
Age/Sex	63/M	49/M	50/M
Mechanism of injury	High-impact RTA (car collision)	RTA (car vs truck)	Motorcycle accident
Hemodynamic status	Unstable	Stable	Stable
Pain score (NRS)	9/10 3/10	6/10 2/10	7/10 2–3/10
ECG findings	Sinus tachycardia	Normal	Normal
Troponin levels	Mildly elevated	Normal	Normal
CT findings	Sternal fracture + large retrosternal hematoma + haemothorax	Undisplaced sternal body fracture	Manubrial fracture + small hematoma
Associated injuries	Haemothorax, mediastinal hematoma	None	Minimal (small retrosternal hematoma)
Management	Surgical (sternotomy + drainage)	Conservative	Conservative
Outcome	Stabilized, ambulatory at discharge	Full recovery	Full recovery
Functional status at discharge	Independent ambulation	Normal activities of daily living (ADLs)	Normal ADLs

CT imaging of the chest demonstrated a fracture involving the manubrium sterni, with an associated small retrosternal hematoma (Figure 5). The patient was managed conservatively with analgesia and observation. Pain improved to 2–3/10 on NRS, and he regained full functional capacity, including normal respiratory effort and mobility, prior to discharge.

Discussion

Sternal fractures are relatively uncommon injuries, accounting for approximately 3–8% of blunt thoracic trauma cases, and are most frequently associated with high-energy mechanisms such as road traffic collisions.[1]The increasing use of seat belts and airbags has led to a shift in injury patterns, with a relative rise in anterior chest wall injuries, including sternal fractures.[2] These injuries typically result from direct anterior chest impact or rapid deceleration forces causing flexion-compression of the thoracic cage.[3]

Clinically, patients with sternal fractures most commonly present with localized anterior chest pain and tenderness, often exacerbated by respiration or movement[4]. While isolated fractures generally have a favourable prognosis, their presence necessitates careful evaluation for associated injuries, particularly cardiac contusion, pulmonary contusion, and mediastinal hematoma.[5] Current evidence supports the use of electrocardiography and cardiac biomarkers in patients with suspected myocardial injury, as early detection is critical for preventing adverse outcomes.[6] The presence of elevated troponin levels in the first patient further highlights the importance of screening for blunt cardiac injury, as recommended in established trauma guidelines.[6]

Imaging plays a central role in diagnosis. Although lateral chest radiography was historically used, it has limited sensitivity, particularly for nondisplaced fractures.[7] Multidetector computed tomography (CT) has emerged as the gold standard imaging modality due to its superior sensitivity and ability to detect associated thoracic injuries.[8] CT imaging was essential in all cases in this series for accurately identifying fracture characteristics and associated injuries, reinforcing its established role in the evaluation of blunt chest trauma.[8] The ability to detect clinically significant findings, such as retrosternal hematoma, directly influenced management decisions in our series.

Recent advances, including three-dimensional CT reconstruction, have further enhanced the assessment of fracture morphology and displacement, aiding both diagnosis and surgical planning.[9]

A key associated finding is retrosternal hematoma, which may result from bleeding at the fracture site or injury to adjacent mediastinal structures.[10] While small hematomas are typically self-limiting, larger collections can lead to mediastinal compression and hemodynamic instability, necessitating urgent intervention.[11]

In our series, one patient required surgical evacuation of a large retrosternal hematoma, highlighting the importance of early recognition and timely management. This observation is supported by prior studies demonstrating that associated mediastinal injuries, particularly large hematomas or haemothorax, are key indicators for operative management.[11]

Management strategies for sternal fractures depend on fracture characteristics and associated injuries. Most isolated, nondisplaced fractures can be managed conservatively with adequate analgesia, respiratory support, and observation.[12] The cases presented in this series illustrate the spectrum of clinical presentations and management strategies for sternal fractures. While most patients can be managed conservatively, the presence of associated complications such as retrosternal hematoma or haemothorax may necessitate surgical intervention. These findings reinforce the pivotal role of CT imaging in guiding diagnosis and management decisions in the emergency setting.

The findings from this case series are consistent with existing literature regarding the clinical spectrum and management of sternal fractures. Two of the three patients in our series were hemodynamically stable with isolated, nondisplaced fractures and were successfully managed conservatively. This aligns with current evidence indicating that the majority of isolated sternal fractures can be treated non-operatively with adequate analgesia and observation.[12]

Pain scores improved significantly across all cases with appropriate management, emphasizing the role of effective analgesia in preventing respiratory complications such as hypoventilation and atelectasis. This is consistent with evidence underscoring pain control as a cornerstone of conservative treatment strategies.[12] Surgical fixation is increasingly being utilized in selected cases, particularly in patients with displaced or unstable fractures, persistent pain, or respiratory compromise.[13]

Recent studies have demonstrated improved outcomes with surgical fixation using titanium plating systems, including enhanced chest wall stability, reduced pain, and shorter hospital stays.[14] Furthermore, early operative intervention in appropriately selected patients may improve pulmonary function and reduce complications.[15]

A multidisciplinary approach is essential for optimal management, particularly in polytrauma patients. Collaboration among emergency physicians, trauma surgeons, radiologists, and cardiothoracic specialists facilitates early diagnosis and comprehensive care.[16] Standardized trauma protocols incorporating CT imaging and cardiac evaluation have been shown to improve detection of associated injuries and reduce morbidity.

Conclusion

Sternal fractures, although uncommon, are clinically significant injuries that require thorough evaluation for associated complications. CT imaging plays a central role in diagnosis and management planning. While most cases can be managed conservatively, early identification of complications, such as retrosternal hematoma, is critical to facilitate timely intervention and improve patient outcomes.

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A rare triad of idiopathic intracranial hypertension, partial empty sella, and narcolepsy due to hypothalamic-pituitary dysfunction

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- *excessive daytime sleepiness*
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ABSTRACT

Background

Type I narcolepsy is a rare sleep disorder characterised by hypocretin (orexin) deficiency. Secondary causes involving structural or functional hypothalamic–pituitary abnormalities are uncommon. In obese individuals, excessive daytime sleepiness (EDS) is frequently attributed to obstructive sleep apnoea (OSA), which may delay recognition of alternative central causes of hypersomnolence.

Case presentation

We report a 33-year-old woman with morbid obesity and multiple comorbidities, including hypothyroidism, systemic hypertension, and type 2 diabetes mellitus. She was previously diagnosed with OSA and was on continuous positive airway pressure (CPAP) therapy. Despite documented CPAP compliance, she reported persistent fatigue and EDS and presented following a syncopal episode at work.

Investigations revealed hypotension and low serum cortisol. A short synacthen test was consistent with central adrenal insufficiency. Magnetic resonance imaging (MRI) of the brain demonstrated a partial empty sella, posterior deviation of the pituitary stalk, and prominent perioptic subarachnoid spaces, findings suggestive of chronically raised intracranial pressure consistent with idiopathic intracranial hypertension (IIH). Cerebrospinal fluid (CSF) orexin-A level was markedly reduced at 7.5 pg/mL (normal >50 pg/mL), confirming type I narcolepsy.

The patient was treated with intravenous corticosteroids, followed by oral corticosteroids, alongside modafinil, fluoxetine, and semaglutide for weight reduction, with subsequent clinical improvement.

Conclusion

This case illustrates a rare but important triad of IIH, partial empty sella, and secondary type I narcolepsy arising from hypothalamic–pituitary dysfunction. Persistent EDS despite

adequate CPAP therapy warrants thorough evaluation for central causes of hypersomnolence. Chronically elevated intracranial pressure may impair both pituitary function and orexinergic neurons, and clinicians should maintain a high index of suspicion in obese patients with refractory somnolence.

Introduction

Type I narcolepsy is a rare sleep disorder characterised by a deficiency of the neuropeptide hypocretin (orexin), produced by a discrete population of neurons within the lateral hypothalamus. Most cases are idiopathic or autoimmune in aetiology, with secondary structural causes accounting for only a small minority.[1,2,5] Excessive daytime sleepiness is among the most disabling symptoms of narcolepsy and is also the cardinal feature of OSA, a condition highly prevalent in individuals with obesity. This overlap of symptoms frequently leads to misattribution of somnolence to OSA, thereby delaying identification of underlying central aetiologies.[3,7]

Idiopathic intracranial hypertension (IIH) is characterised by elevated intracranial pressure in the absence of an underlying structural cause and is strongly associated with obesity. Chronic elevation of intracranial pressure may lead to mechanical and vascular compromise of the hypothalamic–pituitary axis (HPA), resulting in central hypopituitarism and, in rare cases, disruption of orexin-producing neurons.

We report a rare case of secondary type I narcolepsy arising in the context of hypothalamic–pituitary dysfunction secondary to IIH with partial empty sella syndrome. This case underscores the importance of thorough evaluation in patients with persistent hypersomnolence despite apparently adequate OSA therapy.

Case presentation

Clinical history

A 33-year-old woman, employed as an information technology (IT) technician, presented following a sudden fall at her workplace, preceded by intermittent low-grade headaches and overwhelming daytime somnolence. She had morbid obesity (weight 139.6 kg) and a background of hypothyroidism, newly diagnosed systemic hypertension, and type 2 diabetes mellitus.

She had previously been diagnosed with OSA and commenced on CPAP therapy, with documented compliance. Despite this, she continued to experience persistent fatigue and EDS, leading to recurrent hospital attendances without meaningful symptomatic improvement.

Examination

On presentation, she was hypotensive with a blood pressure of 80/60 mmHg, heart rate 72 beats per minute (regular), and oxygen saturation 100% on room air. Systemic examination was otherwise unremarkable. No focal neurological deficits were identified. Fundoscopic examination revealed no papilledema.

Investigations

Initial investigations, including full blood count and inflammatory markers, were within normal limits. Serum cortisol was low, prompting further endocrine evaluation (Table 1).

A short synacthen test (SST) demonstrated a

Table 1: Summary of endocrine investigations

Investigation	Result	Reference Range	Interpretation
Short Synacthen Test (SST) — Cortisol Response			
Basal serum cortisol	4.8 mcg/dL	7–25 mcg/dL (morning)	Low — central adrenal insufficiency suspected
Cortisol at 30 min post-ACTH	15.5 mcg/dL	Peak ≥18–20 mcg/dL	Suboptimal peak — adrenal reserve preserved but inadequate stimulation
Cortisol at 60 min post-ACTH	20.0 mcg/dL	Peak ≥18–20 mcg/dL	Borderline — favours central rather than primary adrenal insufficiency
Anterior Pituitary Hormones			
Oestradiol (E2)	160 pg/mL	Follicular: 20–350 pg/mL	Within range (phase-dependent)
FSH (Follicle-Stimulating Hormone)	2.76 mIU/mL	Follicular: 3.5–12.5 mIU/mL	Low - normal - possible gonadotrophin deficiency
Prolactin	9.3 ng/mL	Female: 2–29 ng/mL	Normal
IGF-1 (Insulin-like Growth Factor-1)	1.5 ng/mL	Age-specific: 94–252 ng/mL (30–35 yrs)	Low - suggestive of GH deficiency

Abbreviations: ACTH = adrenocorticotropic hormone; FSH = follicle-stimulating hormone; GH = growth hormone; IGF-1 = insulin-like growth factor-1; SST = short synacthen test. † SST pattern: basal cortisol low with suboptimal but borderline peak response - consistent with central (secondary) adrenal insufficiency; adrenal reserve intact, implying deficient hypothalamic–pituitary CRH/ACTH drive.

suboptimal cortisol response, with basal serum cortisol of 4.8 mcg/dL, rising to 15.5 mcg/dL at 30 minutes and 20 mcg/dL at 60 minutes post-adrenocorticotrophic hormone (ACTH) administration, a pattern consistent with preserved adrenal reserve but favouring central adrenal insufficiency.

Further pituitary hormone evaluation revealed: oestradiol 160 pg/mL, follicle-stimulating hormone (FSH) 2.76 mIU/mL, prolactin 9.3 ng/mL, and insulin-like growth factor-1 (IGF-1) 1.5 ng/mL, the latter suggesting growth hormone deficiency.

MRI of the brain revealed partial empty sella with posterior deviation of the pituitary stalk, and prominent perioptic subarachnoid spaces — findings consistent with chronically elevated intracranial pressure in keeping with IIH. Video electroencephalography (EEG) was normal, effectively excluding seizure disorder.

CSF examination (Table 2) revealed glucose 62 mg/dL, chloride 125 mmol/L, and protein 26 mg/dL (turbidimetric method). The fluid was clear and colourless, with red blood cells 660 cells/mm³ and white blood cells 1 cell/mm³. CSF manometry pressure was 15 cm HO. Crucially, CSF orexin-A was markedly reduced at 7.5 pg/mL (reference: >50 pg/mL normal; <50 pg/mL consistent with type I narcolepsy), confirming the diagnosis.

Table 2: CSF analysis results

Parameter	Result	Reference Range	Clinical Comment
Macroscopic Appearance			
Colour	Colourless	Colourless	Normal
Appearance	Clear	Clear	Normal
Cell Count			
Red Blood Cells (RBC)	660 cells/mm³	0–5 cells/mm³	Elevated
White Blood Cells (WBC)	1 cell/mm³	0–5 cells/mm³	Normal — no pleocytosis
Lymphocytes	1 cell seen	Predominantly lymphocytes if any	Normal differential
Biochemistry			
Glucose	62 mg/dL	45–80 mg/dL (or >50% serum glucose)	Normal
Protein (turbidimetric)	26 mg/dL	15–45 mg/dL	Normal
Chloride	125 mmol/L	120–130 mmol/L	Normal
Biochemistry			
CSF Manometry (opening pressure)	15 cm HO	7–18 cm HO (lateral decubitus)	Upper normal range — measured at time of lumbar puncture post-treatment initiation
Orexin (Hypocretin) — Diagnostic Test			
CSF Orexin-A (Hypocretin-1)	**7.5 pg/mL**	**Normal: >50 pg/mL<50 pg/mL: Type I Narcolepsy**	**DIAGNOSTIC — severely reduced orexin confirms Type I Narcolepsy (secondary, due to hypothalamic–pituitary dysfunction)**

Abbreviations: CSF = cerebrospinal fluid; RBC = red blood cells; WBC = white blood cells. † Elevated RBC count is consistent with a traumatic lumbar puncture; this does not affect the orexin-A result. ‡ CSF orexin-A <110 pg/mL is highly sensitive and specific for Type I narcolepsy (Mignot et al.); levels <50 pg/mL are considered diagnostic. The value of 7.5 pg/mL in this patient represents severe orexinergic deficiency, confirming secondary Type I narcolepsy due to hypothalamic–pituitary dysfunction.

Management

The patient was treated with intravenous corticosteroids, followed by a transition to oral steroids, resulting in normalisation of blood pressure. For management of narcolepsy, modafinil (a CNS stimulant), fluoxetine, and methylphenidate (as required) were initiated. Given the well-established association between obesity and IIH, semaglutide was initiated alongside intensive lifestyle counselling and weight-reduction strategies.

Discussion

This case presents a rare and instructive triad: IIH leading to partial empty sella syndrome, secondary hypothalamic–pituitary dysfunction, and consequent type I narcolepsy — three conditions that are individually plausible yet, in this patient, causally interlinked.

OSA is the most common cause of EDS in individuals with obesity and is often the initial focus of clinical evaluation. However, persistence of somnolence and fatigue despite adequate CPAP therapy — as observed in this patient — must prompt systematic

re-evaluation for alternative central aetiologies, including hypopituitarism and narcolepsy.[1,7] Diagnostic delay due to attribution of symptoms solely to OSA has been well-documented and may lead to prolonged morbidity.

IIH is an established cause of pituitary dysfunction through chronic pressure-mediated injury to the hypothalamic–pituitary axis. The pathophysiological mechanisms include mechanical compression of the pituitary gland and stalk within the sella turcica, vascular compromise of the portal blood supply, and direct injury to hypothalamic nuclei — including the lateral hypothalamic neurons responsible for orexin synthesis.[2] In this patient, the MRI findings of partial empty sella with stalk deviation, in conjunction with low basal cortisol and a suboptimal short synacthen test (SST) response, reflect the extent of this structural damage.

Measurement of CSF orexin-A remains the gold-standard investigation for type I narcolepsy, with levels <110 pg/mL, and particularly <50 pg/mL, being highly specific for the diagnosis.[4,6] In this patient, an orexin-A level of 7.5 pg/mL confirmed severe orexinergic deficiency. Secondary narcolepsy due to structural hypothalamic damage — as in this case — is well-described but rare; reported causes include tumours, inflammatory lesions, trauma, and, as demonstrated here, chronic pressure effects from IIH.[2]

The management of this complex case required a multi-layered approach: addressing the central adrenal insufficiency with corticosteroid replacement, managing narcolepsy with wake-promoting agents, and targeting the underlying IIH-associated obesity with GLP-1 receptor agonist therapy. The use of semaglutide in this context is noteworthy, given emerging evidence that GLP-1 receptor agonists may reduce intracranial pressure in IIH through weight loss and potentially direct mechanisms.

This case underscores the need to consider rare but treatable secondary causes of hypersomnolence, particularly in patients with features of raised intracranial pressure, pituitary dysfunction, or unexplained endocrine abnormalities.

Conclusion

Secondary type I narcolepsy should be considered in patients with hypothalamic–pituitary abnormalities and unexplained EDS, especially when symptoms persist despite appropriate OSA therapy. Not all EDS in individuals with obesity can be attributed solely to OSA. Partial empty sella associated with chronically elevated intracranial pressure represents a rare but clinically important cause of combined endocrine and sleep dysfunction. A systematic, physiology-based diagnostic approach is essential to avoid prolonged morbidity in such complex cases.

Declarations

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Conflicts of interest: None declared.

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Giant infected bronchogenic cyst mimicking lung abscess in a young adult

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ABSTRACT

Bronchogenic cysts are uncommon congenital foregut malformations resulting from abnormal budding of the primitive tracheobronchial tree during embryogenesis. They are often asymptomatic but may present with complications such as infection, compression of adjacent mediastinal structures, or rupture.

We report the case of a 27-year-old male presenting with acute febrile illness and pleuritic chest pain, who was found to have a large intrathoracic cystic lesion with an air-fluid level in the left hemithorax on imaging. Radiological evaluation suggested a bronchogenic cyst complicated by secondary infection.

The patient was initially managed with broad-spectrum antibiotics and image-guided drainage followed by definitive surgical excision via mini-thoracotomy. Intraoperative findings confirmed a large multiloculated infected cyst occupying the basal segments of the left lower lobe.

This case highlights the importance of considering congenital cystic lung lesions in the differential diagnosis of cavitary pulmonary disease.

Introduction

Bronchogenic cysts arise from abnormal budding of the embryonic foregut during early tracheobronchial development.[1,2] They represent one of the most common congenital cystic lesions of the mediastinum and lung.[1,3] Histologically, they are lined by ciliated pseudostratified columnar respiratory epithelium and may contain cartilage, mucous glands, and smooth muscle within the cyst wall.[1,2]

Most bronchogenic cysts remain asymptomatic and are detected incidentally during imaging studies performed for unrelated indications.[1,4] However, complications such as infection, haemorrhage, compression of adjacent structures, or rupture can lead to significant clinical manifestations.[2,4,5] Infected bronchogenic cysts may radiologically resemble lung abscess, necrotising pneumonia, or other cavitary thoracic lesions, which

can delay definitive diagnosis.[1,3,6]

Case presentation

A 27-year-old male presented with high-grade fever of four days' duration, associated with productive cough, pleuritic chest pain in the left axillary region, and generalized myalgia. The patient had a prior history of a large intrathoracic cystic lesion measuring approximately 11-12 cm with an air-fluid level detected on CT imaging in February 2025.

On admission, the patient was haemodynamically stable and maintaining adequate oxygen saturation on room air. Clinical examination revealed reduced breath sounds in the left infrascapular region, without significant respiratory distress. Laboratory investigations showed a total leukocyte count of 6700 cells/mm³ with neutrophilic predominance and markedly elevated C-reactive protein levels.

Management and clinical course

Initial management included intravenous ceftriaxone and doxycycline for presumed infectious pathology. An extensive infectious workup, including sputum studies and tuberculosis workup, was negative. Computed tomography of the thorax revealed a large intrathoracic cyst measuring approximately 11-12 cm with an air-fluid level, along with a second thin-walled cyst measuring approximately 2.5 cm.

Due to persistent fever spikes, antimicrobial therapy was escalated to piperacillin-tazobactam, linezolid, and azithromycin. Image-guided drainage was subsequently performed with insertion of a 12F pigtail catheter, yielding approximately 80 mL of purulent fluid.

Definitive surgical management was performed via left mini-thoracotomy through the fifth intercostal space. Intraoperative findings revealed a large multiloculated cyst occupying the basal segments of the left lower lobe, with an organized purulent collection and thickened parietal pleura adherent to the diaphragm.

The cyst wall was excised, and the involved segments were preserved with satisfactory postoperative lung expansion and no air leak.

Following the procedure, antibiotics were discontinued, and the patient was clinically monitored. Once the patient stabilized, he was discharged on postoperative day 7 on symptomatic medication.

Discussion

Bronchogenic cysts are developmental anomalies originating from abnormal foregut budding during early embryogenesis.1,2 They account for a significant proportion of congenital mediastinal cysts and may remain clinically silent until complicated by infection or compression.[2,5,6]

Secondary infection is a well-recognized complication and may present with features mimicking a lung abscess or necrotizing pneumonia.[1,4,6] Computed tomography is the imaging modality of choice for evaluating cystic thoracic lesions and identifying complications such as air-fluid levels, wall thickening, and surrounding inflammatory changes.[3,6]

Complete surgical excision is generally recommended for symptomatic lesions as it provides a definitive diagnosis and prevents recurrence and other complications.[1,4-7] Minimally invasive techniques are increasingly used; however, open thoracotomy may still be necessary in cases of large infected cysts or dense adhesions. [1,4,7]

Conclusion

Large infected bronchogenic cysts may radiologically mimic lung abscesses, potentially delaying definitive management. Early consideration of congenital cystic lesions is essential when large air-fluid cavities are identified in young patients without a clear infectious etiology.

Radiological findings

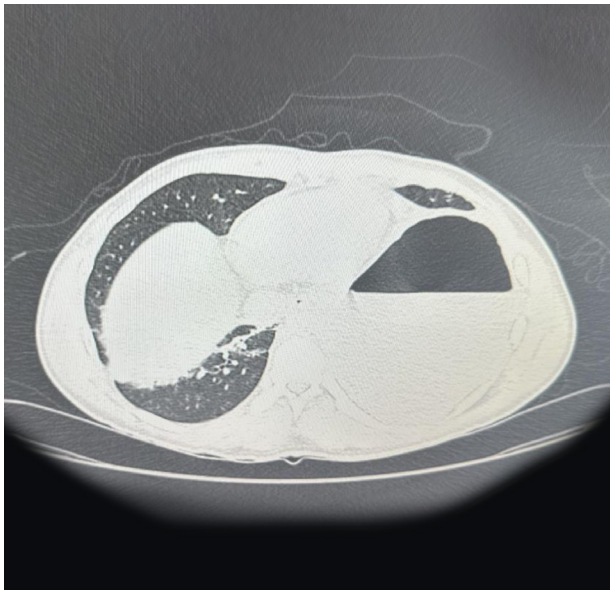

Figure 1a: CT thorax demonstrating a large cystic lesion with an air-fluid level.

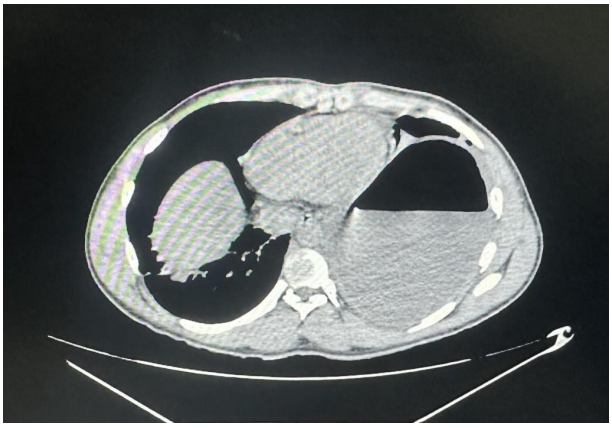

Figure 1b: CT thorax showing the intrathoracic cystic cavity.

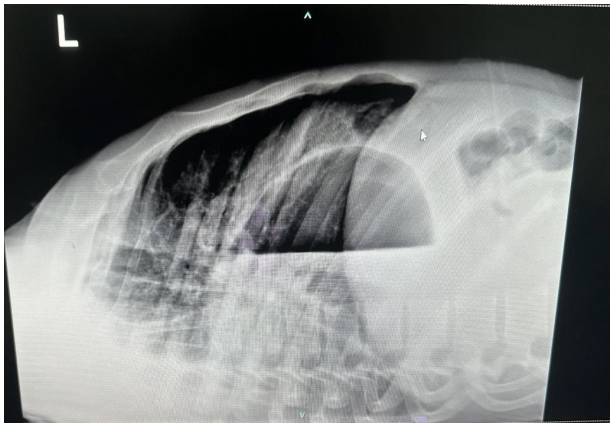

Figure 2: Lateral decubitus chest X-ray demonstrating the air-fluid level.

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Fixed food eruption with multiple food triggers following suspected drug reaction

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ABSTRACT

Fixed Food Eruption (FFE) is a rare hypersensitivity reaction characterized by recurrent erythematous or hyperpigmented lesions occurring at the same anatomical site following ingestion of a specific food allergen. It resembles Fixed Drug Eruption (FDE), a well-recognized cutaneous adverse reaction triggered by medications.

We report the case of a 21-year-old female who initially developed lesions suspected to be secondary to Naproxen-induced fixed drug eruption and later experienced recurrent pruritic lesions following ingestion of various foods. Allergy evaluation demonstrated sensitivity to multiple food items, including seafood, wheat, and spices. The patient showed improvement with avoidance of the identified allergens and antihistamine treatment.

This case highlights the importance of considering fixed food eruption in patients presenting with recurrent, fixed lesions associated with food intake.

Introduction

Fixed Food Eruption is a rare, localized hypersensitivity reaction characterized by the recurrence of lesions at the same anatomical site following ingestion of a specific food allergen. Clinically, the condition closely resembles Fixed Drug Eruption, which is more commonly associated with medications such as antibiotics and non-steroidal anti-inflammatory drugs, including naproxen.[1]

The lesions typically appear as well-defined erythematous macules or plaques that later heal with post-inflammatory hyperpigmentation. The underlying pathogenesis involves a type IV delayed hypersensitivity reaction mediated by intraepidermal CD8 memory T cells. These cells persist in previously affected skin and trigger inflammation upon re-exposure to the antigen.[2]

Although FDE is widely reported, fixed food eruption is rare and is likely under-recognized. A variety of foods, including seafood, nuts, cereals, and fruits, have been reported as potential triggers.[3] Because of the clinical similarity between drug-induced and food-

induced fixed eruptions, a detailed clinical history is crucial for accurate diagnosis.

We report a case of a young female who initially presented with lesions suggestive of fixed drug eruption and subsequently developed recurrent lesions following ingestion of specific foods, consistent with fixed food eruption.

Case presentation

A 21-year-old female presented with generalized itching and pruritic, hyperpigmented macules on the dorsum of the left hand, the forehead, above the upper lip, and below the lower lip.

She had two prior admissions to our hospital with similar complaints. During the first admission, about a month prior to the current presentation, she had developed comparable lesions, which were diagnosed as Fixed Drug Eruption, suspected to be secondary to naproxen intake.

During the second admission, which occurred about a week later, she presented with features of urticaria and anaphylaxis. But no definite trigger could be identified at that time. She was treated symptomatically and got better.

During the current admission, occurring a month after the initial reaction to naproxen, the patient developed similar skin lesions along with urticaria following the consumption of outside food, specifically parotta and chicken. She was admitted and treated symptomatically.

Dermatological examination revealed well-defined hyperpigmented macules on the dorsum of the left hand and the forehead (Figures 1 and 2).

Figure 1: Hyperpigmented lesion over the dorsum of the left hand demonstrating a well-circumscribed violaceous patch with post-inflammatory hyperpigmentation, typical of a healed lesion of Fixed Food Eruption

Figure 2: Hyperpigmented macule over the forehead, showing a well-defined violaceous-brown patch consistent with a healed lesion of Fixed Food Eruption or Fixed Drug Eruption.

Following the initial episode, the patient reported that the lesions became more pruritic after eating, although no specific dietary trigger was identified initially. In view of persistent symptoms, a food allergy panel was performed. The patient was found to be allergic to the following food items:

- Crab
- Shrimp
- Onion
- Mutton
- Garlic
- Sesame seeds
- Wheat

During the present admission, the patient was treated with intravenous hydrocortisone and oral fexofenadine (180 mg).

However, during her hospital stay, she developed episodes of itching, erythematous lesions, and aggravation of the previously existing hyperpigmented lesions, suggesting a hypersensitivity reaction to the administered medications.

Considering the possibility of a drug-related hypersensitivity reaction, intravenous hydrocortisone was discontinued. Following its withdrawal, the patient showed marked clinical improvement. Subsequently, she did not experience further reactions to food intake, except on exposure to identified allergens.

The patient improved clinically and was discharged

in stable condition with advice to avoid the identified allergenic foods and to follow up.

Discussion

Fixed Drug Eruption (FDE) is a well-recognized cutaneous adverse reaction characterized by the recurrence of lesions at the same site following re-exposure to the offending drug. Common causative agents include non-steroidal anti-inflammatory drugs, antibiotics, and anticonvulsants.[4]

In contrast, Fixed Food Eruption (FFE) is a relatively uncommon condition characterized by lesions that appear after ingestion of specific foods. The clinical features closely resemble those of fixed drug eruption, making diagnosis challenging in the absence of careful identification of dietary triggers.[3]

Distinguishing FFE from FDE can be difficult, as both conditions share similar clinical morphology and immunological mechanisms. Therefore, careful correlation with the dietary history and elimination of suspected allergenic foods is essential for establishing the diagnosis.

The pathogenesis of both conditions involves a localized delayed hypersensitivity reaction mediated by epidermal CD8 memory T cells. These cells persist in previously affected skin sites and,upon re-exposure to the antigen, trigger an inflammatory response.[2]

A wide range of foods, including seafood, nuts, fruits, cereals, and spices, have been reported as triggers of FEE. Cases reported in the literature include FFE due to mango, kiwi, nuts, and shellfish[3], For example, a rcent case reported from India described FEE following mango ingestion, confirmed by oral provocation testing.

Compared with previously reported cases, the present case is notable for the presence of multiple food sensitivities and a preceding history of drug-induced fixed eruption. This observation suggests that individuals predisposed to hypersensitivity reactions may develop both drug-and food-induced fixed eruptions.

Management primarily involves identifying and avoiding the offending allergen(s), along with symptomatic treatment with antihistamines such as fexofenadine. Corticosteroids may be considered in more severe cases.[4]

Conclusion

Fixed Food Eruption is a rare but important clinical entity that should be considered in patients presenting with recurrent localized lesions associated with food intake. Careful history-taking, identification of allergens, and avoidance of trigger foods remain the cornerstones of management.

This case highlights the possible coexistence of fixed drug eruption and fixed food eruption, underscoring the need to consider dietary triggers in patients with persistent or recurrent cutaneous eruptions.

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Intermammary pilonidal sinus mimicking acneiform lesions: A rare ectopic presentation

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ABSTRACT

Pilonidal sinus is a chronic inflammatory condition that most commonly occurs in the sacrococcygeal region. Its occurrence in other anatomical sites is uncommon and often leads to diagnostic confusion. Intermammary pilonidal sinus is an extremely rare entity, with only a limited number of cases reported in the literature.

We report the case of a 32-year-old female who presented with recurrent swelling and intermittent seropurulent discharge from the intermammary region for four months. She had previously been treated by multiple physicians and dermatologists for presumed acneiform lesions and sebaceous gland overactivity, given the presence of facial acne and the atypical location of the lesions.

Clinical examination, imaging, and subsequent surgical exploration revealed an intermammary pilonidal sinus. The sinus tract was excised en bloc, and histopathological examination confirmed the diagnosis of pilonidal sinus.

This case highlights the importance of considering intermammary pilonidal sinus in the differential diagnosis of chronic discharging lesions in the intermammary region, especially in patients with predisposing factors such as obesity, large pendulous breasts, and excessive sweating. Awareness of this rare condition is essential to avoid misdiagnosis and delays in treatment.

Introduction

Pilonidal sinus disease is a chronic inflammatory condition characterized by the presence of hair-containing sinus tracts within the subcutaneous tissue. The condition most commonly occurs in the sacrococcygeal region, particularly among young adults, with a higher prevalence in males.[1] The pathogenesis is generally considered to be acquired and involves the penetration of loose hair shafts into the skin, leading to chronic inflammation, foreign body reaction, and sinus tract formation.

Although the sacrococcygeal region accounts for the majority of cases, pilonidal sinuses have also been described in several

unusual locations, including the umbilicus, axilla, interdigital spaces of barbers, nose, suprapubic region, and breast region. Intermammary pilonidal sinus is an extremely rare presentation and is often overlooked because of its unusual location.[2]

Case presentation

A 32-year-old female presented with complaints of recurrent swelling and induration in the intermammary region associated with occasional seropurulent discharge for six months. She had previously consulted multiple physicians for the same complaint. As the patient also had facial acne, the lesion was initially treated by local general practitioners and dermatologists as acneiform lesions or sebaceous gland overactivity, and she received symptomatic treatment at different intervals. However, her symptoms persisted.

Due to the persistent nature of the condition, she presented to our outpatient department for further evaluation. On clinical examination, the patient was moderately built and nourished, with a recent history of weight gain. She reported excessive sweating and had large pendulous breasts, which resulted in increased friction and moisture in the intermammary cleft.

Local examination of the intermammary region revealed a healed scar with multiple pus points, associated with mild tenderness and minimal local rise in temperature. There was no history of fever or other systemic symptoms.

An ultrasound examination of the breast and intermammary region was performed. Imaging demonstrated a linear subcutaneous tract extending inferiorly in the midline, more towards the left, with multiple linear echogenic foci, suggestive of an intermammary pilonidal sinus. A small, well-defined cystic lesion with low-level internal echoes was also noted in the subcutaneous plane of the intermammary region.

Based on the clinical history, examination findings, and imaging features, a diagnosis of intermammary pilonidal sinus was made, and the patient was planned for surgical excision under general anesthesia.

Preoperative photographs were obtained (Figure 1). Intraoperatively, methylene blue dye was injected to delineate the sinus tract, which was then identified and excised en bloc (Figure 2). Hemostasis was secured, and the wound was closed in layers without a drain. The excised specimen was sent for histopathological examination.

Histopathological analysis shows hair follicle

Figure 1: Clinical photograph showing a sinus opening in the intermammary region.

Figure 2: Excised specimen demonstrating the sinus tract involving the skin and subcutaneous tissue.

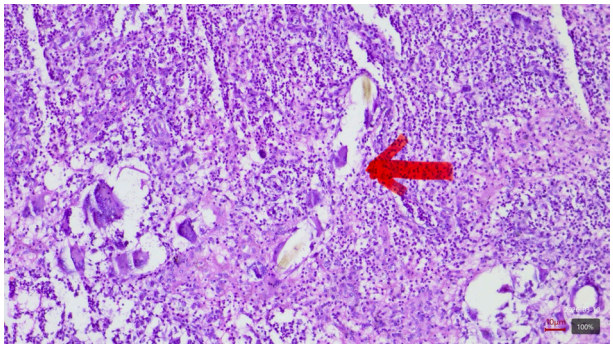

Figure 3: Histopathological examination of the excised specimen showing free hair shafts (arrow) within the surrounding inflammatory reaction.

entrapment and a sinus tract lined by granulation tissue, with a dense mixed inflammatory infiltrate composed of neutrophils, lymphocytes, and plasma cells. Proliferating small blood vessels were also seen. Focally entrapped keratin, numerous foamy macrophages, and a collection of multinucleated giant cells were also observed, consistent with pilonidal sinus (Figure 3).

Discussion

Pilonidal sinus disease is a chronic inflammatory condition characterized by the presence of a sinus tract containing hair shafts within the subcutaneous tissue. The condition most commonly occurs in the sacrococcygeal region, accounting for the vast majority of cases. It typically affects young adults between 15 and 40 years of age and has a reported male predominance with a male-to-female ratio of approximately 4:1.3 The overall incidence has been estimated at 26 cases per 100,000 population, although the exact incidence varies geographically and is influenced by occupational and lifestyle factors.

Although the sacrococcygeal region remains the classical site, ectopic pilonidal sinus disease has been reported in several unusual anatomical locations, including the umbilicus, axilla, interdigital spaces of barbers, suprapubic region, nose, scalp, and the breast region. Among these atypical sites, the intermammary pilonidal sinus in females is particularly rare, with only a limited number of cases reported in the literature. Due to its uncommon location and non-specific clinical presentation, the condition often poses a significant diagnostic challenge.

Pathogenesis

Historically, the pathogenesis of pilonidal sinus disease was believed to be congenital. However, current evidence strongly supports an acquired etiology. According to the widely accepted Karydakis theory,[4] loose hair fragments penetrate the skin due to friction and mechanical forces. These hair shafts act as foreign bodies, initiating a chronic inflammatory response that eventually leads to sinus tract formation.

Several predisposing factors[5] contribute to the development of pilonidal sinus disease, including:

- Obesity
- Excessive sweating
- Deep skin folds
- Friction between adjacent skin surfaces
- Local trauma or pressure
- Poor hygiene
- Presence of coarse hair

In the intermammary region, similar mechanisms may facilitate hair penetration. Women with large pendulous breasts often have a deep intermammary cleft, creating an environment characterized by increased friction, moisture, and skin maceration. These conditions promote the accumulation of loose hair and debris within the cleft. Repeated mechanical pressure and friction may allow hair fragments to penetrate the skin, leading to chronic inflammatory reaction and the formation of a sinus tract.

The patient described in this report had several predisposing factors, including recent weight gain, excessive sweating, and large pendulous breasts, all of which likely contributed to the development of the intermammary pilonidal sinus.

Diagnostic challenges

The diagnosis of intermammary pilonidal sinus can be difficult because its clinical features often resemble more common dermatological conditions. Patients typically present with pain, swelling, induration, or intermittent seropurulent discharge in the intermammary region. However, because this condition is rare, clinicians may initially attribute the lesion to other more common pathologies.

The differential diagnosis of intermammary lesions includes:

- Acne vulgaris
- Folliculitis
- Hidradenitis suppurativa
- Epidermal inclusion cyst
- Sebaceous cyst
- Chronic abscess
- Infected dermoid cyst

In the present case, the patient was initially treated for acneiform lesions or sebaceous gland overactivity, given the presence of facial acne and the superficial appearance of the lesions. This misinterpretation led to a delayed diagnosis, highlighting the importance of maintaining a high index of suspicion when evaluating persistent or recurrent discharging lesions in atypical anatomical locations.

Diagnostic approach

The diagnosis of pilonidal sinus in unusual locations requires careful clinical evaluation, supported by imaging, as needed.

A suggested diagnostic approach for suspected intermammary pilonidal sinus includes:

1. **Detailed clinical examination**
   Identification of sinus openings, chronic

discharge, induration, or healed scars in the intermammary cleft.

2. **Assessment of risk factors**

   Presence of obesity, pendulous breasts, excessive sweating, or deep skin folds.

3. **Imaging studies**

   Ultrasonography can help identify sinus tracts, hair fragments, and associated cystic lesions in superficial tissues.

4. **Definitive diagnosis by histopathology**

   Histological examination typically reveals a sinus tract lined with granulation tissue, hair shafts within the tract, chronic inflammatory infiltrates, and foreign-body giant-cell reactions.

In the present case, ultrasonography demonstrated a linear subcutaneous tract with echogenic foci, raising suspicion for a pilonidal sinus. The diagnosis was confirmed following surgical excision and histopathological examination, which revealed entrapped hair follicles, chronic inflammatory infiltrate, foamy macrophages, multinucleated giant cells, and keratin debris, consistent with pilonidal sinus disease.[6]

Management

The treatment of a pilonidal sinus depends on the extent and location of the disease. For ectopic pilonidal sinuses, complete surgical excision of the sinus tract remains the gold standard treatment.[7] Various surgical techniques have been described, including:

- Excision with primary closure
- Excision with secondary healing
- Flap reconstruction for complex or recurrent disease

For small, localized lesions, en bloc excision with primary closure often provides excellent results with low recurrence rates.

In this case, the sinus tract was delineated intraoperatively with methylene blue, facilitating accurate identification and complete excision. The wound was closed primarily without a drain. The patient had an uneventful postoperative recovery and no recurrence.

Clinical significance

The rarity of intermammary pilonidal sinus makes it an important diagnostic consideration in patients presenting with chronic discharging lesions in the intermammary region. Delayed diagnosis may lead to prolonged symptoms and repeated ineffective treatments.

Awareness of this unusual presentation among clinicians can improve early diagnosis and appropriate management. The present case adds to the limited literature on intermammary pilonidal sinus and highlights the importance of considering this rare entity in the differential diagnosis of chronic intermammary lesions.

Conclusion

Learning points

1. Intermammary pilonidal sinus is an extremely rare form of ectopic pilonidal disease, and its atypical location often leads to misdiagnosis and delayed treatment. A high index of suspicion is therefore required when evaluating persistent or recurrent discharging lesions in atypical anatomical locations.

2. Chronic discharging lesions in the intermammary cleft should raise suspicion for pilonidal sinus, particularly in patients with predisposing factors such as large pendulous breasts, obesity, excessive sweating, and deep intermammary clefts.

3. An intermammary pilonidal sinus may mimic acne, folliculitis, a sebaceous cyst, or hidradenitis suppurativa, making careful clinical examination essential.

4. Ultrasonography can help in delineating sinus tracts; however, histopathological examination remains the gold standard for confirming the diagnosis.

5. Complete surgical excision of the sinus tract with primary closure remains the definitive treatment, with excellent outcomes and low recurrence when adequately performed.

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Adult-onset acute disseminated encephalomyelitis following severe Salmonella gastroenteritis

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ABSTRACT

Background

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated demyelinating disease of the central nervous system that typically follows infection or vaccination. Although it is most commonly seen in children, adult presentations are increasingly recognized. Gastrointestinal infections are an uncommon but important trigger.

Case summary

A 35-year-old male presented with acute, severe gastroenteritis characterized by profuse watery diarrhea, vomiting, fever, and dehydration following ingestion of food outside the home. Subsequently, he developed multiorgan dysfunction, including prerenal acute kidney injury and hepatic dysfunction. Stool culture identified Salmonella species. He was therefore treated with intravenous ceftriaxone (2g once daily) and oral azithromycin (1g once daily).

During hospitalization, he developed acute neurological symptoms including dysarthria, ataxia, visual blurring, and postural instability. MRI of the brain demonstrated a transient splenial lesion in the corpus callosum, suggestive of a metabolic or inflammatory etiology. Cerebrospinal fluid analysis was unremarkable. In view of suspected ADEM, he was treated with high-dose intravenous methylprednisolone, resulting in significant clinical improvement.

Conclusion

ADEM can occur in adults after severe systemic infections, such as gastrointestinal sepsis. Recognizing neurological symptoms early and promptly beginning immunosuppressive therapy can lead to favorable outcomes.

Introduction

Acute Disseminated Encephalomyelitis is a monophasic inflammatory demyelinating disease affecting the brain and

spinal cord. It typically occurs 1-3 weeks after infection and is characterized by encephalopathy and multifocal neurological deficits. Adult-onset ADEM is rare and may pose diagnostic challenges, especially when accompanied by systemic illness.

Case presentation

A previously healthy 35-year-old male presented with a 3-day history of loose stools, vomiting, nausea, high-grade fever, and reduced urine output. Notably, these symptoms began after eating at a restaurant. A similar illness was reported also among others who had eaten at the same place.

Upon arrival at our institution, he was dehydrated and tachycardic (pulse rate:130bpm) with borderline hypotension. Systemic examination was otherwise unremarkable. Laboratory findings revealed elevated hemoglobin and packed cell volume (PCV) suggestive of hemoconcentration, acute kidney injury (serum creatinine: 2.5 mg/dL), hyperbilirubinemia, elevated liver enzymes, raised C-reactive protein (CRP), and mild hyponatremia. Stool culture grew Salmonella species. Ultrasound of the abdomen showed fatty liver, gallbladder sludge, and mild splenomegaly.

On the second day of hospitalization, the patient developed dysarthria, ataxia, visual blurring, and postural instability. Neurological examination showed right-sided cerebellar signs and scanning speech. There was no history of neurological illness or recent vaccination.

MRI of the brain demonstrated a small T2/FLAIR hyperintense lesion in the splenium of the corpus callosum consistent with a transient splenial lesion. Cerebrospinal fluid (CSF) analysis was normal.

Given the clinical context, a diagnosis of ADEM was considered.

Management and outcome

The patient was treated with intravenous methylprednisolone (1 g daily for three days) followed by oral steroid taper.

His neurological symptoms improved significantly after treatment, and he was discharged in stable condition.

Discussion

Acute Disseminated Encephalomyelitis (ADEM) is an immune-mediated inflammatory demyelinating disorder of the central nervous system characterized by a monophasic course and multifocal neurological deficits. Although it predominantly affects children,

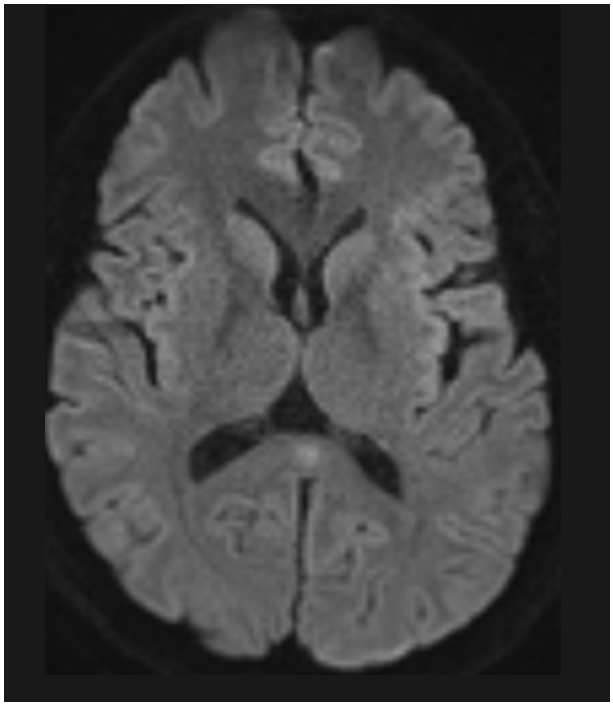

Figure 1: Diffusion weighted imaging

Figure 2: T2-weighted imaging

Figures 1 and 2: MRI of the brain demonstrates a small T2/FLAIR hyperintense lesion with patchy diffusion restriction in the splenium of the corpus callosum at the midline, consistent with a transient splenial lesion. In the given clinical context. This finding is likely secondary to metabolic imbalances in the clinical setting.

adult-onset ADEM is increasingly recognized in clinical practice and often presents diagnostic challenges due to overlapping features with infectious encephalopathy, metabolic disturbances, and multiple sclerosis.[1]

The pathogenesis of ADEM is believed to involve molecular mimicry, wherein infectious antigens trigger a cross-reactive autoimmune response against myelin proteins. While viral infections are classically implicated, bacterial pathogens have also been reported as triggers. Several case reports have described ADEM following enteric infections, including those caused by Salmonella species.

Kamei et al. reported a case of adult-onset ADEM following Salmonella enteritidis gastroenteritis, with neurological manifestations developing days after resolution of systemic symptoms and showing a favorable response to corticosteroid therapy.[2] Similarly, Ozkale et al. reported post-infectious ADEM associated with bacterial gastrointestinal infection, emphasizing the temporal relationship between systemic inflammation and subsequent demyelination.[3]

Neurological complications of Salmonella infection are variable; however, immune-mediated demyelination remains a rare but important differential diagnosis. In the present case, the onset of dysarthria, ataxia, and visual disturbances during recovery from severe gastrointestinal sepsis raised suspicion of a secondary inflammatory process rather than primary infectious involvement of the CNS. The normal cerebrospinal fluid findings further supported a para-infectious immune-mediated mechanism rather than direct bacterial invasion.

The presence of a transient splenial lesion in the corpus callosum adds another notable feature to this case. Such lesions are commonly associated with infections, metabolic derangements, and systemic inflammatory states. In this patient, the lesion likely reflects a reversible inflammatory process in the setting of systemic immune activation. The rapid clinical improvement following high-dose intravenous methylprednisolone further supports an immune-mediated etiology.

Conclusion

Although rare, adult-onset ADEM accounts for up to 30% of all ADEM cases and has an estimated annual incidence of 0.2–0.4 per 100,000 population. While a preceding infection is common, bacterial gastrointestinal infections such as Salmonella are uncommon but important triggers.

Neurological complications occur in a minority of patients with severe systemic Salmonella infection, with immune-mediated demyelination being uncommon. Nevertheless, early recognition is important, as timely initiation of corticosteroid therapy results in clinical improvement and radiological resolution in most cases of transient splenial lesions.

This case highlights the need for maintaining heightened clinical vigilance for immune-mediated neurological complications in adults who develop new focal deficits during or after severe gastrointestinal sepsis. Early neuroimaging and prompt immunosuppressive therapy can significantly improve outcomes and prevent long-term neurological disability.

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Glanzmann thrombasthenia in pregnancy complicated by refractory postpartum hemorrhage

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ABSTRACT

Background: Glanzmann thrombasthenia (GT) is a rare bleeding disease due to an autosomal recessive platelet function disorder.[1] It is caused by pathogenic variants in ITGA2B or ITGB3, resulting in defective glycoprotein IIb/IIIa–mediated platelet aggregation.[1] Pregnancy in women with GT carries a high risk of antepartum, intrapartum, and postpartum hemorrhage, making management extremely challenging and necessitating meticulous multidisciplinary planning and execution.[2] We report such a case.

Case: A 33-year-old multigravida (G2P1L1) presented at 37 weeks with childhood-onset GT (type 2B suspected). Her history included iron deficiency anemia, gestational diabetes mellitus controlled with oral agents, prior secondary postpartum hemorrhage after cesarean delivery for placental abruption, and varicella infection in early gestation. Throughout pregnancy, she had normal fetal growth, normal antenatal testing, and no bleeding episodes.

At 37 weeks, following multidisciplinary planning, an elective lower-segment cesarean section with sterilization was performed under subarachnoid block. Prophylactic uterotonics and tranexamic acid were administered, and perioperative platelet support was arranged, including an intraoperative transfusion of 1 unit of single-donor platelets (SDP).

A healthy term male neonate weighing 2.9 kg was delivered.

The initial postoperative period was uneventful. However, on postoperative day 2, the patient developed severe anemia with hemoglobin dropping from 8.7g/fL to 6.3g, hypotension, tachycardia, abdominal discomfort, and vaginal bleeding.

Two units of fresh single-donor platelets were transfused, but bleeding persisted due to anti-platelet antibodies from prior transfusions leading to rapid destruction of transfused platelets.

CT angiography demonstrated intrauterine and rectus sheath hematomas with active contrast extravasation. Pelvic angiography identified bleeding from the left inferior epigastric artery. Successful Gelfoam embolization was performed, along with pre-emptive embolization of bilateral uterine arteries and the right

inferior epigastric artery.

The patient required multiple transfusions (total: 3 SDP, 5 units of LDRBC, and 10 units of cryoprecipitate), along with tranexamic acid and antibiotics for postoperative fever. Due to persistent bleeding, the patient was given recombinant activated factor VIIa (rFVIIa, NovoSeven).

The calculated dose was 8100 mcg (90 mcg/kg for 90 kg body weight). An initial reduced dose of 400 mcg was given due to financial constraints, followed by the remaining dose after government financial assistance was secured. Subsequently, the patient showed clinical improvement.

Serial monitoring showed stabilization, without further major bleeding; hematuria and wound oozing were resolved. She was discharged hemodynamically stable with a hemoglobin of 8.1 g/dL after a total hospital stay of 12 days.

Conclusion: Despite the severe hemorrhagic risk in GT, a favorable maternal and neonatal outcome could be achieved in this case with early diagnosis, anticipatory multidisciplinary planning, availability of blood products, judicious use of antifibrinolytics and hemostatic agents, and timely interventional radiology procedure for refractory bleeding.[2,4,5]

Case presentation

Examination

A 33-year-old multigravida (G2P1L1) at 37 weeks of gestation presented for safe confinement. She was a known case of Glanzmann thrombasthenia diagnosed at 3 years of age (suspected Type 2B). She was not on regular medication but had a history of prior blood transfusions.

Her obstetric history included a previous full-term cesarean section for abruptio placentae in 2016, when she delivered a male baby weighing 3.8 kg. The postpartum period was complicated by secondary postpartum hemorrhage on day 9 following minor trauma, requiring suction evacuation and transfusion of two units of packed red blood cells.

The current pregnancy was conceived spontaneously and complicated by iron deficiency anemia, gestational diabetes mellitus on oral hypoglycemic agents, and varicella infection during early pregnancy.

On examination, she was afebrile with stable vital signs. Abdominal examination revealed a term relaxed uterus with fetal heart sounds present. The non-stress test was reactive.

Investigations

Initial laboratory investigations revealed hemoglobin of 10.9 g/dL and platelet count of 115,000/µL. Peripheral smear showed a normocytic normochromic picture with mild thrombocytopenia. The blood group was AB positive and the indirect Coombs test was negative.

Platelet aggregation studies performed in 2016 reportedly confirmed the diagnosis of Glanzmann thrombasthenia, though detailed records were unavailable.

Ultrasound performed at 36 weeks showed a single live intrauterine gestation with growth parameters above the 80th percentile and estimated fetal weight at the 59th percentile.

Postoperative CT angiography demonstrated intrauterine hematoma extending into the lower uterine segment and cervix, rectus sheath hematoma extending into the pelvic cavity, and contrast extravasation suggestive of active vascular leak.

Management

A medical board counseled the patient and family regarding delivery and postpartum management.[2]

The patient underwent an elective lower-segment cesarean section with sterilization at 37 weeks under subarachnoid block. A healthy male baby weighing 2.9 kg was delivered. The baby was normal. One unit of SDP was transfused intraoperatively along with tranexamic acid and uterotonics.

Postoperatively, the patient developed hypotension, tachycardia, and vaginal bleeding. An additional SDP transfusion was administered. CT angiography confirmed active bleeding, and pelvic angiography identified contrast leak from the left inferior epigastric artery feeders. An interventional radiologist performed timely Gelfoam embolization of the bleeding vessel along with pre-emptive embolization of bilateral uterine arteries and right inferior epigastric artery feeders.

During hospitalization, she received a total of 5 packed red blood cell transfusions,10 units of cryoprecipitate, 3 units of single donor platelets, and tranexamic acid. Recombinant activated factor VIIa (NovoSeven) 8100 mcg IV infusion was administered for persistent bleeding following which the bleeding gradually subsided and the patient stabilized.

Discussion

Glanzmann thrombasthenia is a rare platelet function disorder caused by defects in platelet integrin αIIbβ3,

which mediates platelet aggregation.[1] Patients with this disorder typically have normal platelet counts but impaired platelet aggregation, leading to mucocutaneous bleeding.

Pregnancy in women with GT carries a significant hemorrhagic risk, particularly during delivery and the postpartum period.[2] Management requires multidisciplinary planning, and availability of blood products, antifibrinolytic agents, platelet transfusions, and recombinant factor VIIa for refractory bleeding.[2,5] In the present case, all these modalities were used because bleeding was very severe.

Interventional radiology procedures, such as arterial embolization, are important life-saving interventions when conservative management fails.[4] In this case, prompt embolization successfully controlled bleeding and prevented further deterioration.

Previous reports have described variable severity, with some cases requiring aggressive therapy and necessitating the use of recombinant factor VIIa.[2,5] Some cases have been managed conservatively where the disease was only mild to moderate in severity.[3]

Limited data from India suggest that most reported cases of thrombasthenia during pregnancy involved mild to moderate disease managed with platelet transfusion alone, with only two reports of the use of recombinant factor VIIa (NovoSeven), where the bleeding was severe, as it was in the present case.

Conclusion

Glanzmann thrombasthenia in pregnancy is a rare but high-risk condition. Successful maternal and neonatal outcomes depend on early diagnosis, multidisciplinary management, adequate preparation with blood products, and timely use of advanced interventions such as arterial embolization and recombinant factor VIIa.[2,5]

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Extracorporeal cardiopulmonary resuscitation as a bridge to percutaneous coronary intervention in refractory asystolic cardiac arrest

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ABSTRACT

This case details the emergency management of a 43-year-old male with a history of coronary artery disease who presented with recurrent refractory cardiac arrest. Cardiopulmonary resuscitation (CPR) was subsequently escalated to Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO) during ongoing cardiopulmonary resuscitation (ECPR). This was followed by life-saving percutaneous coronary intervention (PCI).

Introduction

Refractory cardiac arrest remains one of the most formidable challenges in emergency cardiovascular medicine, with exceedingly high mortality when treated with conventional advanced cardiac life support (ACLS) alone. Although standard cardiopulmonary resuscitation (CPR) is the cornerstone of initial management, it often fails to achieve the critical threshold of systemic perfusion, typically maintaining only 20% to 30% of normal cardiac output required to sustain vital organ function during prolonged resuscitative efforts.[1] In the context of acute myocardial infarction (AMI), this limitation creates a lethal paradox: the patient requires definitive coronary intervention to survive, yet is often too hemodynamically unstable to undergo the procedure.

Extracorporeal cardiopulmonary resuscitation (ECPR) using veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has emerged as a transformative "bridge-to-decision" or "bridge-to-intervention" strategy. By rapidly providing full circulatory and respiratory support, ECPR interrupts the cycle of progressive global hypoxia and metabolic acidosis, establishing a stable "hemodynamic platform" for clinicians to address the underlying cause. [2,3.] Recent literature, including the landmark ECPR trials, suggests that in carefully selected patients with potentially reversible etiologies, early initiation of ECMO is associated with significantly improved survival and favorable neurological outcomes compared with standard ACLS.[3]

Despite increasing adoption, the logistical complexity of ECPR requires seamless multidisciplinary coordination between

emergency medicine, cardiology, and cardiothoracic surgery teams. The decision-making process is particularly nuanced in patients presenting with non-shockable rhythms, such as asystole, where prognosis has traditionally been poor.

This case report details the successful resuscitation of a 43-year-old male with recurrent, refractory asystolic cardiac arrest through emergency implementation of VA-ECMO. It highlights the critical role of ECPR in enabling life-saving percutaneous coronary intervention (PCI) and underscores the importance of timely escalation to mechanical circulatory support in "no-flow" or "low-flow" states of cardiac arrest.

Case presentation

A 43-year-old male, with a history of hypertension and coronary artery disease (non-ST elevation myocardial infarction in 2020),previously treated with percutaneous coronary intervention (PCI) to the left anterior descending (LAD) artery, presented to the emergency department with an acute onset of chest pain radiating to the left upper limb for one hour.

On arrival, he had ongoing chest pain with a heart rate of 70 beats /min, blood pressure of 140/100 mmHg, respiratory rate of 28 breaths/min, and oxygen saturation of 100% on 4L/min of oxygen. While the catheterization team was being activated and preparations were underway, the patient developed vomiting followed by bradycardia and progressive drowsiness, necessitating rapid sequence intubation by the anesthesia team.[5]

Shortly thereafter, the patient suffered a cardiac arrest. ECG telemetry revealed asystole, and cardiopulmonary resuscitation (CPR) was initiated in accordance with advanced cardiac life support (ACLS) protocols. Despite standard resuscitation efforts and a brief return of spontaneous circulation (ROSC) followed by temporary pacemaker (TPI) insertion for junctional bradycardia, the patient lapsed back into asystole.

Given the refractory nature of the arrest, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was considered, and the cardiothoracic and vascular surgery (CTVS) team was mobilized. VA-ECMO flow was initiated in the ICU.

Once hemodynamic stability was achieved with VA-ECMO, the patient was transferred to the Cath Lab for definitive management. Emergency percutaneous coronary intervention was performed to treat the culprit coronary lesion.

Discussion

The management of refractory cardiac arrest in the setting of acute myocardial infarction poses a significant clinical challenge, as standard CPR often fails to provide sufficient cardiac output to sustain vital organ function during prolonged resuscitation. In this case, the implementation of Extracorporeal Cardiopulmonary Resuscitation (ECPR) served as a critical bridge to definitive therapy.

By using a portable ECMO machine as an adjunct to standard CPR, the clinical team maintained systemic circulation while the patient was transferred to the Cath Lab for intervention. The VA-ECMO provided a stable "hemodynamic platform," allowing the interventional team to focus on the complex coronary anatomy without the immediate threat of hypoperfusion-induced brain injury or multi-organ failure.

Conclusion

The survival of this patient underscores the potential of extracorporeal cardiopulmonary resuscitation (ECPR) as a life-saving intervention when conventional advanced cardiac life support (ACLS) measures fail. A successful outcome in such high-acuity cases depends entirely on the prompt activation of a multidisciplinary "heart team," including cardiologists, cardiothoracic surgeons, cardiac anesthesiologists, and resuscitation teams. Quick initiation of ECPR provides a means to address the primary cardiac insult—in this case, via coronary intervention—while the ECMO circuit protects the brain and other end organs. This case highlights the importance of timely escalation from standard CPR to extracorporeal support as a key strategy in the modern management of refractory cardiac arrest.

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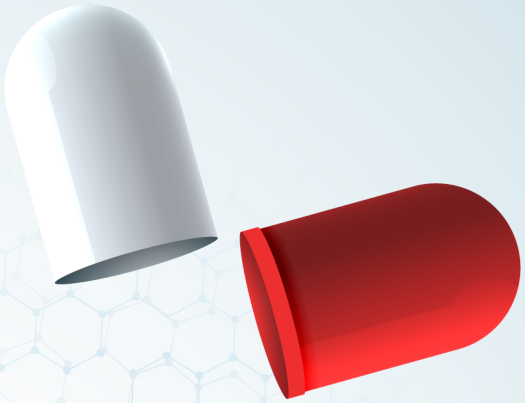
PHARMA UPDATES



Stay informed with the latest in medicines and treatment guidelines.

Dima Zeineddine
Clinical Pharmacist,
KIMSHEALTH Oman

Pharma Updates are brought to you by the Clinical Pharmacy Department, KIMSHEALTH Oman.

ISMP targeted medication safety best practices 2024-2025 (Part II)

To reduce the risk of medication errors, the Institute for Safe Medication Practices (ISMP) has developed Targeted Medication Safety Best Practices for Hospitals. These best practices aim to identify, inspire, and mobilize widespread adoption of consensus-based strategies addressing specific medication safety issues that continue to cause serious harm and fatalities in patients.

Here are some selected best practices, chosen for our readers:

Best Practice 8:

Administer all medication and hydration infusions using a programmable infusion pump with dose error-reduction systems.

Rationale: To ensure the use of dose error-reduction technology to prevent infusion-related medication errors, which can result in patient harm.

Best Practice 9:

Ensure that all appropriate antidotes, reversal agents, and rescue agents are readily available.

- Establish standardized protocols and/or coupled order sets that allow for the emergency administration of these agents.
- Ensure that directions for use and administration are readily accessible in all clinical areas where these agents may be required.
- Clearly identify which agents can be administered immediately in emergency situations to prevent patient harm.

Best Practice 13:

Eliminate injectable promethazine from the formulary.

Rationale: This best practice aims to eliminate the risk of serious tissue injury and amputations caused by inadvertent arterial injection or intravenous (IV) extravasation of injectable promethazine.

In 2023, the US Food and Drug Administration (FDA) began asking manufacturers to include administration recommendations in prescribing information and on carton and container labels (www.ismp.org/ext/1288). The FDA recommends deep intramuscular injection instead of IV administration.

Best Practice 14:

Proactively seek and utilize information about medication safety risks and errors that have occurred in other organizations, and take action to prevent similar errors.

Rationale: One of the most effective ways to prevent medication errors is to learn from incidents in other institutions and to use that information to identify potential risk points or practices within your organization to prevent similar errors.

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Why reporting side effects matters: Understanding pharmacovigilance

Medicines play a vital role in preventing and treating diseases, improving the quality of life, and saving lives. However, no medicine is entirely free of risks. Even after rigorous clinical trials, some side effects (also known as adverse drug reactions, ADRs) may only become apparent when a medicine is widely used by larger and more diverse populations. This is where pharmacovigilance becomes essential.

What is pharmacovigilance?Pharmacovigilance (PV) is defined by the World Health Organization (WHO) as the science and activities related to the detection, assessment, understanding, and prevention of adverse effects or other medicine-related problems.

Why was pharmacovigilance created?

Pharmacovigilance was developed to protect public health and improve patient safety. Historical events, such as the thalidomide tragedy in the 1960s — where a medication given to pregnant women caused severe birth defects — highlighted the need for robust post-marketing surveillance of medicines. In response, regulatory authorities such as the World Health Organization (WHO) and national drug agencies established pharmacovigilance systems to:

- Identify new or rare side effects not detected during clinical trials.
- Evaluate the severity and frequency of known adverse effects in larger populations.
- Take appropriate actions when risks outweigh benefits, such as updating product labels, issuing safety warnings, or withdrawing medicines from the market.

Why is reporting side effects important?

Reporting side effects of medicines—by healthcare professionals, patients, or manufacturers — is the cornerstone of pharmacovigilance. It is essential for:

1. **Early detection of risks**

   Individual reports of adverse reactions, especially of unexpected or severe reactions, may provide the first sign of a previously unknown problem with a medicine.

2. **Improving patient safety**

   Once side effects are reported and analyzed, regulators and pharmaceutical companies can update safety information, adjust dosages, or issue warnings to reduce harm.

3. **Supporting evidence-based decisions**

   Data generated from reports helps doctors and pharmacists make informed decisions about prescribing and dispensing medicines.

4. **Building public trust**

   Active monitoring and transparent responses to safety concerns strengthen public confidence in healthcare systems and medicines.

How to report side effects?

The health ministries of most countries collaborate with international organizations like the WHO and the Uppsala Monitoring Centre (UMC) to enhance pharmacovigilance efforts. Healthcare professionals, patients, and pharmaceutical companies are encouraged to report suspected adverse drug reactions and product quality issues to the designated agencies in their respective regions.

FDA initiates removal of 'black box' warnings from menopausal hormone replacement therapy products

Menopausal hormone therapies, also commonly referred to as hormone replacement therapy (HRT), are approved to provide relief from common menopause symptoms, such as hot flashes and night sweats, and symptoms due to changes to the vagina, vulva, and urinary tract caused by decreased estrogen.

In November 2025, the FDA announced plans to remove its long-standing 'black box' warnings from HRT products, because of which women may be under-utilizing many approved therapies that can alleviate menopause symptoms and improve health.

The FDA is initiating removal of the boxed warnings following a comprehensive review of the scientific literature, an expert panel in July, and a public comment period.

The agency is working with companies to update language in product labeling to remove references to risks of cardiovascular disease, breast cancer, and probable dementia. However, it is not seeking to remove the boxed warning for endometrial cancer for systemic estrogen-alone products.

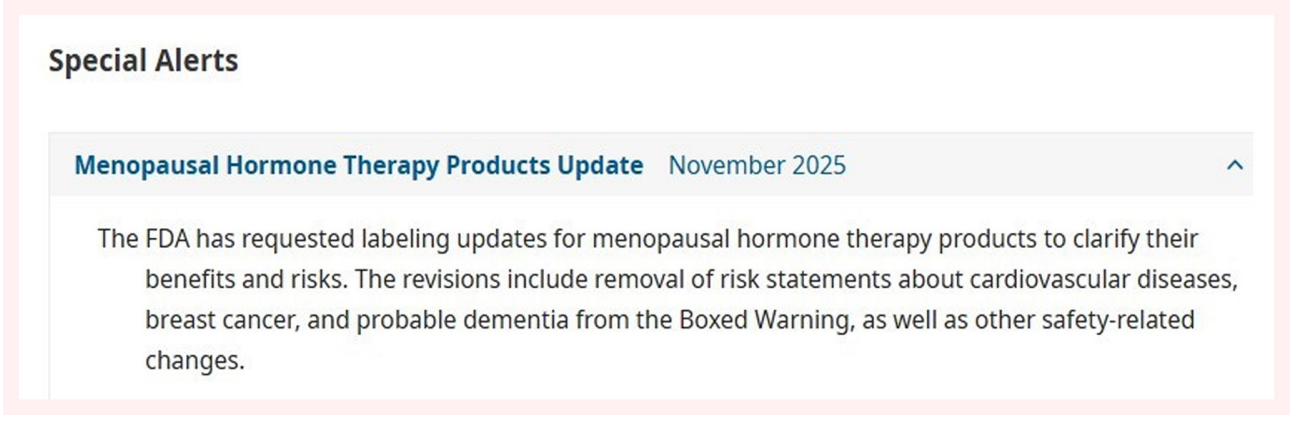

Figure 1: Special alert. Hormone therapy/UpToDate reference

Supportive evidence

Studies have provided evidence that starting HRT within ten years of the onset of menopause can have numerous benefits that for most women, outweigh potential risks. Benefits include a reduced risk of all-cause mortality and fractures. 50% reduction in heart attack risk, 64% reduction in cognitive decline, and 35% lower risk of Alzheimer's.

In addition, an analysis of 30 trials with 26,708 women participants found HRT was not associated with increased cancer mortality. In fact, women who start HRT before age 60 appear to have a decreased mortality risk.

Key information

The removal of a warning label, however, does

not imply that HRT is suitable for every woman or that it should be broadly prescribed without careful evaluation.

Treatment must remain individualized, considering age, symptom severity, personal and family risk profile, comorbidities, and patient preference.

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1 FDA news release, November 10, 2025. < https://www.fda.gov/news-events/press-announcements/hhs-advances-womens-health-removes-misleading-fda-warnings-hormone-replacement-therapy>

Do egg allergies still matter for vaccination decisions?

Health authorities and recent clinical evidence indicate that egg allergy should no longer be considered a routine concern when administering the most common vaccines, easing long-standing immunization precautions.

For vaccines such as influenza, measles, mumps, rubella (MMR), and rabies, experts have for several years advised that patients do not need to be screened for egg allergy prior to vaccination. Studies show that while these vaccines may contain trace amounts of egg protein, the levels are too low to trigger allergic reactions, even in individuals with known egg allergies.

Recent research has extended this reassurance to the yellow fever vaccine.

In the largest study conducted to date, 171 children with egg allergies, including nearly a quarter with a history of anaphylaxis, were safely vaccinated against yellow fever.

Participants underwent skin testing before vaccination, but no allergic reactions were observed regardless of test outcomes.

Despite these findings, current guidelines from the U.S. Centers for Disease Control and Prevention (CDC) and vaccine manufacturers continue to list the yellow fever vaccine as contraindicated for individuals with a history of severe hypersensitivity to egg proteins or related products.

However, milder or localized egg or feather allergies are not considered a barrier to vaccination and typically do not require prior skin testing.

Experts note that while new evidence is encouraging, additional data are still needed before official recommendations are fully updated.

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3 International Archives of Allergy and Immunology: https://karger.com/iaa/article-abstract/186/1/52/911605/No-Yellow-Fever-Vaccine-Reactions-in-IgE-Mediated?redirectedFrom=fulltext

Practical tips for management of polypharmacy

Polypharmacy, literally meaning "many medications," is commonly defined as the regular use of five or more medicines, including prescription, over-the-counter, traditional, or complementary products.

While sometimes necessary, polypharmacy can increase the risk of adverse drug reactions, drug–drug or drug–disease interactions, falls, functional decline, and poor adherence—especially among older adults and those with multiple chronic conditions.

The following tips outline practical approaches to managing polypharmacy and reducing its risks:

Verify actual medication use: Never assume that patients are taking medications exactly as prescribed.

Clarify prescription purpose: Ensure each medication has a clearly documented indication or medical condition for which it is intended.

Prioritize substitution over addition: Whenever appropriate, substitute medications rather than adding new agents to existing regimens.

Simplify medication regimens: Aim to limit dosing to once or twice daily where possible. Minimize overall "pill burden" while maintaining effective therapy.

Provide clear instructions: Ensure patients receive written guidance, including detailed dosing schedules.

Recognize and mitigate risks: Be aware of potential side effects, drug–drug interactions, and drug–disease interactions associated with prescribed medications.

Collaborate with clinical pharmacists: Engage in discussions with pharmacy colleagues in both community and hospital settings, particularly for complex or long-term regimens. They can assist with medication safety, identify potential interactions, recommend suitable formulations, and support patient understanding.

Monitor, review, and adjust regularly: Medication review is an ongoing process that requires a multidisciplinary approach and continuous patient monitoring.

Medication review tools

To help healthcare providers identify opportunities to improve medication management and optimize treatment plans, medication review tools are often used alongside clinical judgment and patient input to support patient-centered care.The choice of tool depends on the population being assessed and the goals of the review. Some commonly used tools are listed below (see Figure 3)

Examples of medication review tools for screening polypharmacy:

- Beers Criteria (AGS Beers List)
- STOPP/START Criteria
- Medication Appropriateness Index (MAI)
- NOR-FRAIL Tool (Fatigue, Resistance, Aerobic capacity, Illnesses, and Loss of weight)
- MedStopper Tool
- Anticholinergic Burden (ACB) Calculator

Summary

Practical management of polypharmacy relies on regular medication review, effective use of clinical tools, and close collaboration among healthcare professionals. With this approach, polypharmacy can be transformed from a risk into an opportunity for safer, more individualized patient care.

AWaRe classification of antibiotics, a tool to complement antibiotic stewardship policy

Antimicrobial resistance is a global threat and has been recognized as one of the top three major public health challenges by the World Health Organization (WHO).

Recent national data from Oman, for instance, have shown an increasing prevalence of multidrug-resistant Gram-negative bacteria such as Acinetobacter spp. (MDRA) and carbapenamase-producing Enterobacterales (CRE), as well as methicillin-resistant *Staphylococcus aureus* (MRSA). Additionally, enteric pathogens such as *Salmonella*,

resistant fungal pathogens such as *Candida auris*, and multidrug-resistant tuberculosis (MDR-TB) are of growing concern.

In response to this issue, the Sultanate of Oman has strengthened its efforts to combat antimicrobial resistance by implementing the Antimicrobial Stewardship (AMS) program and publishing a national policy on January 1, 2025.

This policy provides a comprehensive framework to support healthcare professionals and

stakeholders in optimizing antimicrobial use, and preserving their effectiveness for future

generations. It emphasizes evidence-based practices, enhanced infection prevention and control, and strengthening surveillance systems.

A key component of the national AMS program is the adoption of the AWaRe classification of antibiotics. This classification serves as an essential tool for monitoring antibiotic consumption, optimizing prescribing practices, and supporting national-level antibiotic stewardship efforts.

The AWaRe classification was developed by the World Health Organization (WHO) in 2017. It categorizes antibiotics into three groups, Access, Watch, and Reserve, based on their spectrum of activity and potential to develop resistance:

Access group

- Includes first-line antibiotics for the most common and serious infections.
- These antibiotics should be widely available, affordable, and of good quality.

Watch group

- Includes antibiotics with a higher resistance potential.
- Their use should be limited and carefully monitored. Should be prescribed sparingly to avoid resistance.

Reserve group

- Includes last-resort antibiotics for the treatment of multidrug-resistant infections.
- Their use should be reserved for specific cases to preserve their efficacy.

Access Group	Watch Group	Reserve Group
Amikacin	Azithromycin	Cefiderocol
Amoxicillin	Cefixime	Ceftazidime / Avibactam
Amoxicillin/ Clavulanate	Cefotaxime	Colistin
Ampicillin	Ceftazidime	Fosfomycin
Ampicillin/ Sulbactam	Ceftriaxone	Linezolid
Cefalexin	Cefuroxime	Meropenem*
Cefazolin	Cefepime	Meropenem/Vaborbactam
Clindamycin	Ciprofloxacin	Plazomicin
Cloxacillin	Clarithromycin	Polymyxin B
Flucloxacillin	Erythromycin	
Doxycycline	Levofloxacin	
Gentamicin	Moxifloxacin	
Metronidazole	Piperacillin / Tazobactam	
Nitrofurantoin	Vancomycin	
Penicillin	Ertapenem	
Trimethoprim/ Sulfamethoxazole	Streptomycin	

Table 1: Classification of antibiotics into Access, Watch, and Reserve groups as per the WHO AWaRe framework

(Note: Edited excerpts from KIMSHEALTH Pharma Updates: Issues 20 and 21, KIMSHEALTH, Oman.)

RADIOLOGY SPOTTERS

A case-based quiz on diagnostic imaging. Answers on Pages 75.

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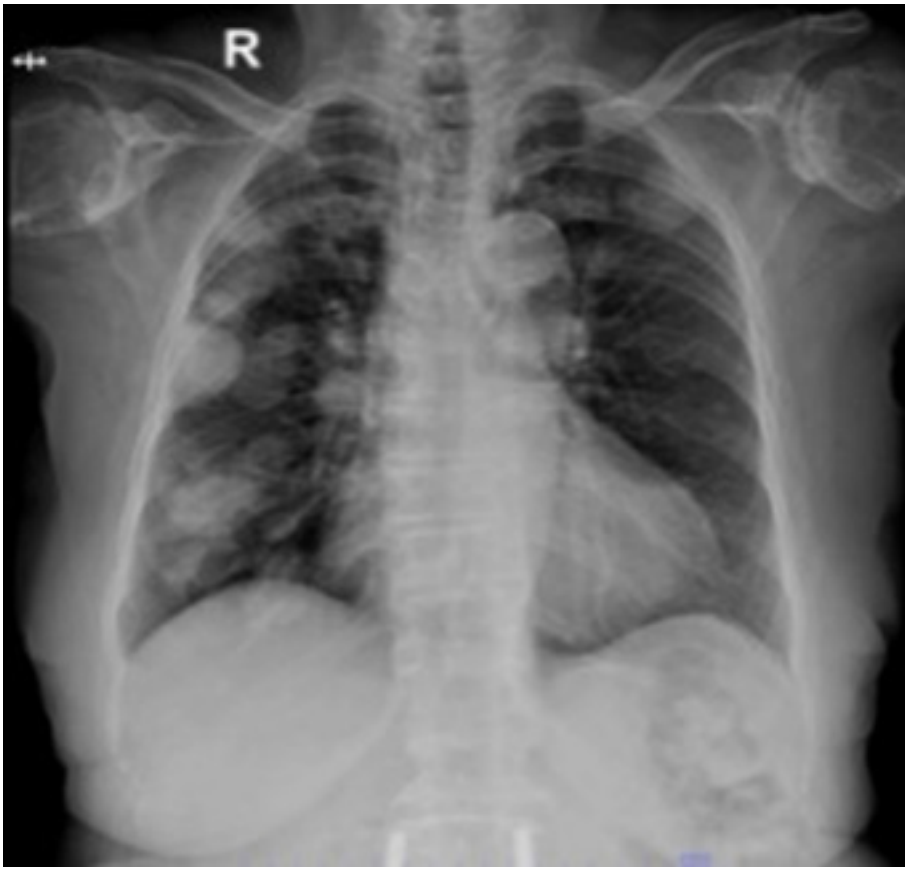
Department of Imaging & Interventional Radiology, KIMSHEALTH Hospital, Trivandrum.

CASE 1: A 10-year-old boy presents with swelling and tenderness over the proximal part of the left leg. X-ray of the left knee (AP view) and leg (lateral view) were obtained.

What is the diagnosis?

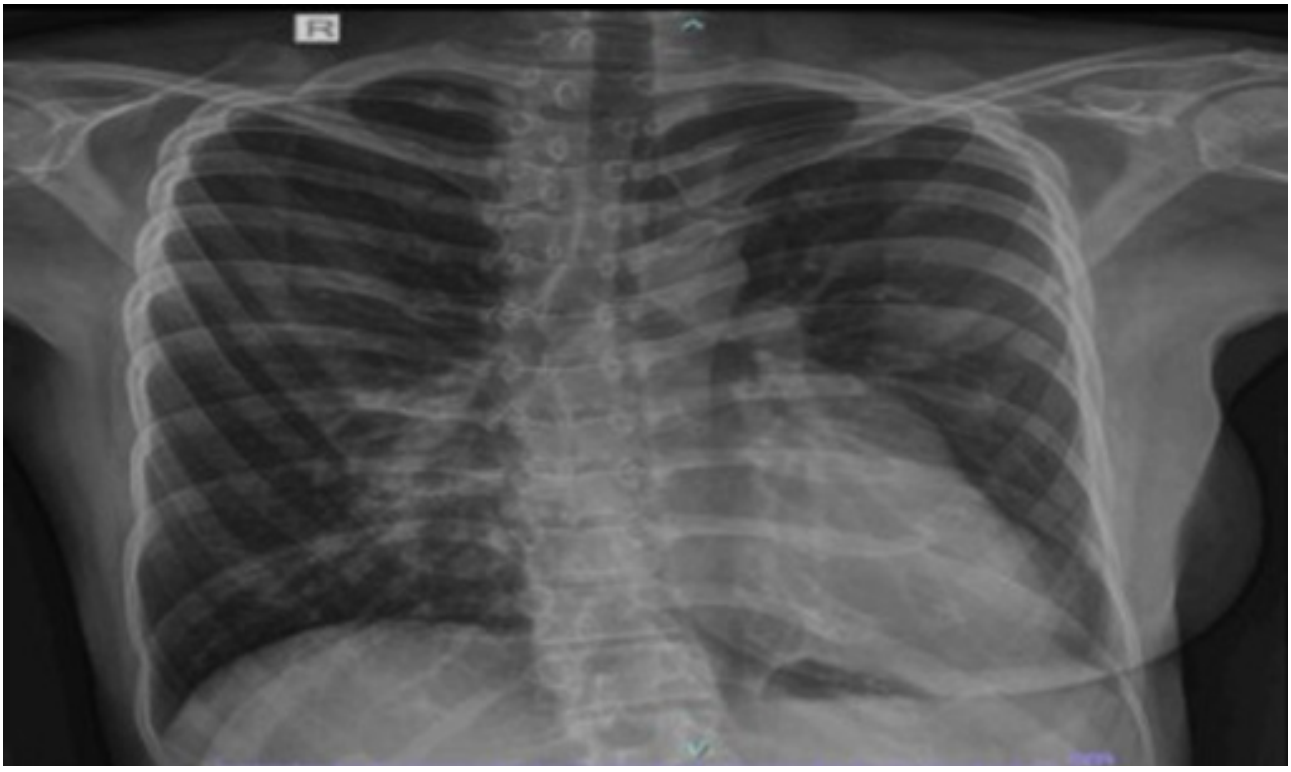


CASE 2: A known case of carcinoma of the urinary bladder on chemotherapy presents with respiratory distress. A chest X-ray (AP view) was obtained.

What is the diagnosis?

CASE 3: A 48-year-old male presents with a history of fall from height, followed by pain in the left shoulder. An X-ray of the left shoulder (AP view) was obtained.

What is the diagnosis?



CASE 4: A 31-year-old female, known case of pectus excavatum and congenital mitral valve prolapse, underwent a routine chest X-ray (AP view).

What is the diagnosis?

SCRAMBLE

Unscramble the letters below to reveal the correct medical term related to various specialties. Use the clues provided. Answers are on Page 76.

By Hilal SM

Department of Healthcare Promotions, KIMSHEALTH, Trivandrum.

ENDOCRINOLOGY

1 **Clue:** A rare, benign tumor of the adrenal medulla that causes excessive secretion of catecholamines leading to severe hypertension, headaches, palpitations, and intense sweating.

O Y C M H O E C A P T O R H M O

2 **Clue:** A tumor outside the pituitary gland that produces excessive hormone causing high cortisol levels, severe hypercortisolism, skin hyperpigmentation, high blood pressure, and muscle weakness.

I T C E C O P C T H A E Y S O N M D R

3 **Clue:** A genetic disorder associated with decreased reaction of body tissues to thyroid hormones.

R F E T F F E O D S N R E M Y O

4 **Clue:** An extremely rare, autosomal recessive disorder characterized by dark, thick skin, growth retardation, and dysmorphic facial features.

A R S N B O - H N L D N E L A M E D O S N E M Y R

5 **Clue:** A group of inherited genetic disorders that cause endocrine tumors. Common symptoms include high calcium levels, neck pain, or hormone imbalances.

T M I E L L U P C E O I R D N N E A N P E O A L I S

PEDIATRICS

6 **Clue:** A highly aggressive central nervous system tumor that often presents with headache, vomiting, seizures, ataxia, and cranial nerve palsies.

T R C I E A D P I M E L G L A T N E P O N I E O M A N L E M A

7 **Clue:** A genetic, neuromuscular disorder that causes progressive degeneration resulting in muscle weakness, atrophy, and difficulty with movement, swallowing, and breathing.

P I A L S N S M L C A R U U R O A P T Y H

8 **Clue:** An inherited neurodegenerative disorder leading to DNA repair failure. Progressive walking difficulties, balance issues, and dilated blood vessels in eyes/skin are early childhood symptoms.

A A I A X T N I E A G A L T S C E I A T

9 **Clue:** A rare, progressive, and chronic inflammatory neurological disease, causing severe, drug-resistant epilepsy, cognitive decline, and progressive hemiparesis primarily in children under 15 years.

U M S R E N S A S E D M Y S O N R

10 **Clue:** A polio-like neurological condition causing sudden muscle weakness, loss of reflexes, and limb paralysis due to spinal cord gray matter inflammation requiring immediate hospitalization.

U C T E A I F A L C D C Y S I E T I L M

UROLOGY

11 **Clue:** A rare genetic condition combining urinary incontinence, kidney damage, and an inverted facial expression where smiling appears as crying.

U A A F O I L R C Y E M R O S D N

12 **Clue:** A potentially life-threatening infection involving gas-forming bacteria within the bladder wall, occurring in diabetic, elderly females and presenting with abdominal pain, fever, and dysuria.

P A S E Y M H T U O S E M T S I S T I C Y

13 **Clue:** A condition considered one of the most severe birth defects, causing protruding internal organs, a non-fused pelvis, major spinal abnormalities, a shortened colon, and an imperforate anus.

A O C A L L C H T E Y O P X S R

14 **Clue:** An uncommon, pouch-like sac that develops along the female urethra, which can cause pelvic pain, frequent urinary tract infections, blood in urine, and incontinence.

L T U H R E A R R D U U C L T M V I E

15 **Clue:** A rare, inherited autosomal recessive disorder where an excess production of oxalate combines with calcium, leading to recurrent kidney stones, nephrocalcinosis, and often progressive renal failure.

M R Y A P R I I L U P H A Y A R O E R X

ONCOLOGY

16 **Clue:** A rare malignancy that develops in the lower uterine segment or cervix several decades after pregnancy and typically presents as abnormal vaginal bleeding.

E I D O L E P T H I I O S H L A R O I C T P T B O T R U M

17 **Clue:** An exceptionally rare and aggressive cancer primarily affecting infants and young children, originating in the lungs and pleura, and presenting with cough, shortness of breath, and fever.

M Y P E R O P L O L A U R U N T S A B O A L M

18 **Clue:** An aggressive soft tissue cancer, typically affecting young adults near joints in the limbs and causing slow-growing, painful, deep-seated masses.

V O I L N A S Y A A O M S C R

19 **Clue:** A rare tumor of the adrenal gland that causes overproduction of adrenaline and noradrenaline, leading to severely high blood pressure, headaches, palpitations, and excessive sweating.

O C H R P C O O E Y T O H A M M

20 **Clue:** A unique and aggressive liver tumor that occurs distinctively in young patients without underlying liver disease.

A A F L L M L I O R R B E C I M A C N R O A

RADIOLOGY SPOTTERS

Answers

CASE 1:

Diagnosis: Sessile osteochondroma

Imaging findings:

- A sessile bony outgrowth is seen arising from the posteromedial aspect of the proximal tibial metaphysis, projecting away from the adjacent epiphysis.

CASE 2:

Diagnosis: Pulmonary metastases

Imaging findings:

- Multiple, well-defined, rounded nodular opacities are seen in all zones of the right lung and in the upper zone of the left lung.

CASE 3:

Diagnosis: Left acromioclavicular (AC) joint dislocation

Imaging findings:

- There is loss of normal alignment between the distal clavicle and the acromion. The distal clavicle is displaced superiorly, with increased coracoclavicular distance.

CASE 4

Diagnosis: Poland syndrome

Imaging findings:

- Suggestive of hypoplasia/aplasia of the right pectoralis major muscle, with hyperlucency of the right hemithorax.

SCRAMBLE
Answers

ENDOCRINOLOGY

1. PHEOCHROMOCYTOMA
2. ECTOPIC ACTH SYNDROME
3. REFETOFF SYNDROME
4. RABSON-MENDENHALL SYNDROME
5. MULTIPLE ENDOCRINE NEOPLASIA

PEDIATRICS

6. PEDIATRIC LEPTOMENINGEAL MELANOMA
7. SPINAL MUSCULAR ATROPHY
8. ATAXIA-TELANGIECTASIA
9. RASMUSSEN SYNDROME
10. ACUTE FLACCID MYELITIS

UROLOGY

11. UROFACIAL SYNDROME
12. EMPHYSEMATOUS CYSTITIS
13. CLOACAL EXSTROPHY
14. URETHRAL DIVERTICULUM
15. PRIMARY HYPEROXALURIA

ONCOLOGY

16. EPITHELIOID TROPHOBLASTIC TUMOR
17. PLEUROPULMONARY BLASTOMA
18. SYNOVIAL SARCOMA
19. PHEOCHROMOCYTOMA
20. FIBROLAMELLAR CARCINOMA

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