

HealthNews DIGEST DECEMBER 2023





Prostate Enlargement & Management



Cow's Milk Protein Allergy in Children 10

Acute Coronary Syndrome (ACS) -NSTEMI



Roux-en-Y Hepaticojejunostomy (RYHJ)



Dr. Sherbaz Bichu

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On behalf of Aster's leadership, I am pleased to welcome you to the 16th edition of the HealthNews Digest. Seeing the constant enthusiasm and commitment towards this initiative is heartening. It has resulted in a wide array of informative content for readers, providing valuable insights and knowledge that broadens our understanding of the medical sector.

The HealthNews Digest has evolved into an essential platform for Aster to maintain its commitment to clinical excellence and knowledge distribution. I am confident that the exceptional team of Aster doctors will continue to provide their expertise and devotion to this initiative, which will deepen our understanding of the medical field and contribute to shaping healthcare's future.

As we explore unique clinical cases and thought-provoking articles, we can significantly impact patients' lives and continue to raise the bar in clinical excellence.



Dr. Ramanathan V

Medical Director Aster Hospitals & Clinics, UAE

As the Medical Director for Aster Hospitals and Clinics, I am delighted to greet all our doctors as we commence the 16th edition of HealthNews. Witnessing their dedication and hard work in tackling complex and challenging cases and their commitment to finding solutions and improving patient outcomes is fascinating.

The medical field is constantly evolving, and it is through the efforts of our doctors that we are advancing medical knowledge and fostering innovation; our tenacity and unwavering resolve in the face of demanding medical challenges exemplify the essence of healthcare excellence.

I enthusiastically applaud the encouragement to continue producing outstanding achievements in medical science and contribute to subsequent HealthNews Digest editions.

Dengue Myositis

An unusual presentation of Dengue with Severe Myositis treated successfully at Aster Cedars Hospital and Clinic, Jebel Ali

PRESENTATION

- 27 year old male
- No medical history of Diabetes Mellitus / Hypertension / Dyslipidemia
- No family history of medical illness
- Admitted with:
 - Fever and Diarrhea for 2 days
 - Weakness in both lower limbs for 2 days
 - No previous history of similar complaints

FINDINGS

During Examination:

- GCS 15/15, higher mental function normal
- No pallor/icterus
- No petechial rashes
- Normal cranial nerves
- Normal upper limb examination
- Lower limbs: Power reduced 1/5 bilaterally, reflexes reduced in both lower limbs
- Normal sensation, planter flexor
- Tenderness in both calves

Tests	On admission	After 3 days
Hb	15.7	15
Platelets	186	164
Creatine Phosphokinase (CPK)	>22000	8300
Potassium	3.37	3.7
TSH	3.6	
Dengue NS1	Positive	
Urine Myoglobin	Positive	









TREATMENT

Dengue fever was managed conservatively as per the guidelines. The patient was treated with IV fluids and supportive care. His urine output and vitals were monitored daily. Gradually, his power improved, and his total CPK was reduced.

DIAGNOSIS

The patient was diagnosed with Dengue fever with myositis. Surprisingly, the patient's platelet counts were normal during the hospital stay. Conservative management was done. Gradually, his power improved; 5/5 on both limbs and reduced CPK levels.

DISCUSSION

Viral infections leading to myositis have been described widely in literature, but very few reports are associated with dengue infection (1). It has been postulated that myositis can be because of the interaction of host cells and viruses in various ways after a viral infection, like dengue. It can occur because specific receptors on particular organs stress the reason for specific organ involvement. The probable mechanisms of dengue myositis can be because of the invasion of muscle cells by the virus directly and the generation of toxins inside the muscle cells. Various myotoxins have been seen, like tumour necrosis factor (TNF) and interferon–(IFN)– γ (2).

A total of 34 studies (3) of dengue-associated myositis were compiled, and it was reported that dengue-associated myositis is common in the younger age group (range: 32-56 years; mean: 24.6 years). Most affected patients were male, with the ratio of male to female being 26:8. Onset of weakness varies from 3 to 36 days (mean: 9.4 days). Muscle weakness is frequently accompanied by muscular pain. Serum CPK is often markedly elevated (mean: 10,558 IU/L; range: 162–117,200 IU/L). In the majority of patients, there was spontaneous and complete recovery (mean: 7 days). Occasionally, corticosteroids were used.

This report has relevance to all primary care physicians as it emphasizes that myositis can be one of the manifestations of dengue. It can be managed well per dengue national management guidelines, and corticosteroids can be used occasionally. And patients usually recover completely without any residual weakness.

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Understanding and Managing Prostate Enlargement

Optimal Approaches for Treating and Managing Enlarged Prostate



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INTRODUCTION

Prostate enlargement is characterized by the excessive proliferation of smooth muscle, glandular epithelial tissue, and connective tissue within the prostate gland, leading to its enlargement (1). The prevalence of prostate enlargement rises with age, ranging from 50%-60% of men in their 60s to 80%-90% in older age (1,2). This age-related increase corresponds to a higher likelihood of experiencing uncomfortable urinary symptoms (3). The primary driver of prostatic growth is dihydrotestosterone (DHT), a metabolite of testosterone produced within prostate cells through the conversion of testosterone by the enzyme 5-alpha reductase (1). Certain factors such as metabolic disorders, obesity, and genetic predisposition elevate the risk of prostate enlargement (2). The diagnosis typically involves a comprehensive approach, including symptom assessment, patient history, and physical examination (3). While managing prostate enlargement, both pharmacotherapy and surgical interventions are considered, with the choice depending on the severity of the disease and its impact on the patient (2).

This article discusses the approach and strategy for the treatment and management of prostate enlargement.

DIAGNOSIS OF PROSTATE ENLARGEMENT

Diagnosis of prostate enlargement involves a detailed assessment of the medical history and a physical examination of the patient, specifically a digital rectal examination (DRE) (3). The medical history should include a thorough evaluation of symptoms, such as frequent urination, urgency, hesitancy, urinary tract infections, etc (3). This includes a bedside urine dipstick analysis, post-void residual volume measurement, and the International Prostate Symptom Score (IPSS) to measure the severity of these urinary symptoms (4). Additional diagnostic tests such as blood tests, urinalysis, prostate-specific antigen (PSA) measurement, ultrasound imaging, uroflow studies to assess urine flow, and cystoscopy may be considered based on the patient's medical history and individual needs (2,3). These tests help provide a more thorough evaluation and help in the management of enlarged prostate (2).







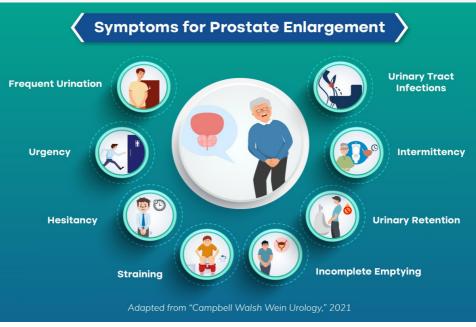


Figure 1: Manifestations of Prostate Enlargement (3)

TREATMENT AND MANAGEMENT

The recent guidelines from both American and European Urology Associations highlight a range of strategies for effectively managing prostate enlargement (5). These approaches encompass behavioral modifications as well as pharmacological and surgical interventions (6).

INITIAL MANAGEMENT

Introducing lifestyle adjustments can help in the management of enlarged prostate and can be integrated into the care plan for patients experiencing mild symptoms (1). These modifications include reducing alcohol and caffeine intake, limiting fluid intake before bedtime to alleviate nocturia symptoms, and adhering to a scheduled voiding routine (4).

PHARMACOTHERAPY

ALPHA-BLOCKERS

The prostate stromal smooth muscle and bladder neck contain alpha 1-adrenoreceptors (4). Alpha 1-receptor blocker causes stromal smooth muscle relaxation, which addresses the dynamic component detrusor smooth muscle of the urinary tract, and in-turn improves flow (4). There are five main alpha-blockers: terazosin and doxazosin from the second generation of blockers, and tamsulosin, alfuzosin, and silodosin from the third generation (1).





Finasteride (5 mg once a day) and dutasteride are alpha-reductase inhibitors that function by preventing the conversion of testosterone into dihydrotestosterone (DHT) (7). 5-ARI monotherapy should be used for patients with prostate volume >30cc on imaging (1). They primarily target the non-changing element of prostate by causing a decrease in prostate size, and while optimum efficiency is normally attained within six months, it typically takes several weeks to notice any significant improvement (4). This therapy can result in a 25% reduction in prostate volume and a reduction in serum PSA levels of roughly 50% (2).

PHOSPHODIESTERASE INHIBITORS (PDE5I)

In addition to their well-known use in erectile dysfunction (ED), PDE5I are now approved for daily usage for enlarged prostate (7). These inhibitors works by preventing phosphodiesterase from converting cGMP to GMP (7). PDE 4, 5, and 11 are found in the prostate, and cross-reactivity from PDE5I causes vasodilation and an improvement in Lower Urinary Tract Symptoms (LUTS) (7). Tadalafil has been demonstrated to improve quality of life (QoL) and can be used for men with both LUTS and ED (1).

COMBINATION THERAPY

Several extensive clinical trials have consistently demonstrated the advantages of utilizing combination therapy to manage LUTS associated with prostate enlargement (7). This combined approach is particularly effective in individuals with enlarged prostates, as the alpha blocker relaxes the smooth muscle of the prostate, leading to enhanced therapeutic benefits (4). Combination therapy of 5-ARI with alpha-blocker is considered for LUTS with prostate volume >30cc (1). Anticholinergics alone or with alpha-blocker for moderate to severe storage LUTS and beta-3-agonists with alpha-blocker for moderate to severe storage LUTS (1).

SURGICAL THERAPIES

If patient's urinary symptoms persist despite medicinal treatment, minimally invasive surgical procedures may be considered (2,5).

TRANSURETHRAL RESECTION SURGERY (TURP)

The purpose of TURP is to debulk the prostate to create an appropriate route for urine flow (2). Clinicians can use both monopolar and bipolar approach for TURP as a treatment option for enlarged prostate (5). This is accomplished by employing diathermy to generate a high-frequency current that permits tissue cutting (2). An appropriate route for urine flow can be created by resecting all blocking prostatic tissue (2). Bipolar diathermy has mostly replaced monopolar diathermy procedures for TURP, providing additional benefits such as saline resection and a lower risk of TUR syndrome (2).

HOLMIUM LASER ENUCLEATION OF THE PROSTATE (HoLEP)

HoLEP is frequently thought of as the endoscopic equivalent of open prostatectomy (OP) (5). It works along the plane between the adenoma and the surgical capsule (8). This approach is responsible for the remarkable success in eliminating a large amount of prostate tissue (8).





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A study that compared the outcomes of HoLEP for prostates of different sizes (less than 75 mL, between 75 mL and 125 mL, and bigger than 125 mL) discovered no significant differences in the requirement for blood transfusions or rates of incontinence among these groups (8). This strong data supports the efficacy of HoLEP, which can effectively treat prostate adenomas of any size (8).

UROLIFT

The UroLift/Prostatic Urethral Lift (PUL) is an endoscopic procedure designed to alleviate bladder outlet obstruction caused by prostate-related issues (7). PUL should be considered for LUTS/BPH patients with prostate volume between 30cc and 80cc (5). It creates an unobstructed passage through the urethra, extending from the bladder neck to the tip of the prostate (7). This procedure utilizes a rigid cystoscope to strategically place small, permanent UroLift Implants within the anterior and lateral regions of the prostatic capsule, effectively establishing a wider, open lumen within the prostatic urethra which leads to reduced risk of bleeding in patients and expands the channel within the prostatic urethra and improving LUTS (7). Studies have demonstrated various benefits, including the potential for day-case surgery, preservation of sexual function, as well as enhancements in symptom scores (IPSS) and urine flow rates (QMax) (2).

• WATER VAPOUR THERMAL THERAPY (WVTT)

WVTT injects sterile water vapor into the prostate adenoma, disrupting prostate cells and causing cell death (7). The technique is minimally invasive, with shorter recovery time and fewer side effects, providing relief from the symptoms like frequent urination, urgency, and nocturia (3). This treatment should be considered for patients with LUTS/BPH with a prostate volume of 30cc to 80cc (5). WVTT may also be offered as a treatment option to eligible patients who desire to preserve erectile and ejaculatory function (5).

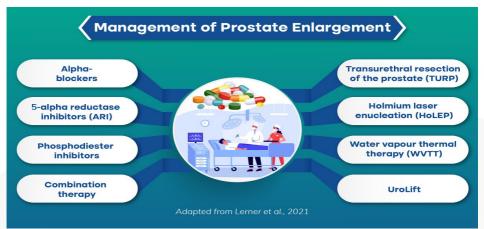


Figure 2: Strategies for Addressing Prostate Enlargement (1,5)







Key Highlights

- Prostate enlargement results in glandular growth, leading to urinary symptoms that significantly impact patient's quality of life (1).
- The diagnosis typically involves a comprehensive approach, including symptom assessment, patient history, and physical examination (3).
- The primary strategy for relieving symptoms include lifestyle adjustments, alpha-blockers, 5-alpha reductase inhibitors, and phosphodiesterase inhibitors (2,7).
- For persistent symptoms, surgical procedures such as TURP, HoLEP, UroLift, and WVTT can be considered for relieving urinary symptoms and obstruction (2,3,5).

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PRESENTATION 83 year old female

- Smoker for >40 years
- Medical history of type II Diabetes Mellitus and hypertension

Successful Management of Acute Coronary Syndrome (ACS) - NSTEMI with Acute Diastolic

Heart Failure in Octogenarian Patient at

- No family history of cardiac illness
- Presented with a history of chest pain, which was intermittent and typical for the past 3-4 days, radiating to the left hand.
- The patient also complained of difficulty in breathing for the past few months, which increased in the past 3-4 days and was associated with coughing, predominantly dry in nature.
- Admitted for further evaluation and management, including CAG (Coronary Artery Angiography).

FINDINGS

During Examination:

- Conscious and oriented
- Afebrile
- Moderate to severe dyspnea, NYHA class II- III
- BP: 190/110 mmHg
- PR: 80 bpm
- SpO2: 95-97% on 2 liters of oxygen
- CVS: S1, S2+, no murmurs heard
- RS: Bilateral crepitation heard, predominantly inspiratory, wheeze bilateral diffuse

CLINICAL DIAGNOSIS

Acute Diastolic Heart Failure, NYHA Class II-III, Left Ventricular Ejection Fraction (LVEF) 50%, secondary to accelerated Hypertension, Acute Coronary Syndrome-Non-ST-segment Elevation Myocardial Infarction (ACS-NSTEMI), Lower Respiratory Tract Infection (LRTI), Type 2 Diabetes Mellitus (T2DM), Hypertension.



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ACS-NSTEMI





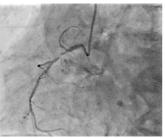






COURSE IN HOSPITAL

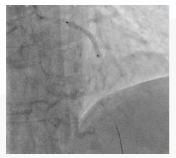
- The patient was admitted to the CCU.
- ECG had ST-T changes. BNP was elevated, and serial cardiac enzymes showed a significant rising trend; troponin at baseline was 20.11, raised to 491.90; CK-MB was 3.18, raised to 10.21.
- Echocardiogram showed adequate left ventricular systolic function: LVEF: 50%. Mid and apical anteroseptum, anterior wall, apical septum, and apex hypokinetic. Grade III diastolic dysfunction was observed. Trace tricuspid regurgitation, the estimated Pulmonary Arterial Systolic Pressure (PASP) was 35 mmHg.
- The patient received unfractionated heparin, Dual Antiplatelet Therapy (DAPT), statin, IV diuretics, trimetazidine, bronchodilator and steroid nebulization and other supportive medications.
- After clinical stabilization of heart failure, the patient still had intermittent angina with dynamic ECG changes in anterior leads; hence, she was taken up for CAG.
- CAG was done through the right radial route, which revealed Double Vessel Disease.
- She underwent PTCA to LAD. TIMI-III flow attained with good results.
- Pulmonology consultation was sought, given emphysematous changes in chest X-ray with lung Rhonchi, and orders were followed.



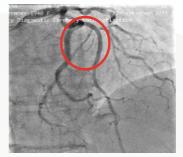
CAG showing RCA, non-dominant vessel with 90-95% Stenosis



CAG showing Left Coronary System, proximal LAD has focal 90-95% Stenosis



Stent deployment in proximal LAD



Final Angiogram showing well deployed LAD Stent with TIMI III Flow







POST PROCEDURE

The patient tolerated the procedures well with no peri-procedural complications. She became clinically better, was ambulated with no symptoms, medications were optimized, and she was discharged in a stable condition.

DISCUSSION

The clinical evidence for the treatment of Acute Coronary Syndrome (ACS) in older people is less robust than in patients younger than 75 years of age. The elderly have the highest incidence of cardiovascular disease and frequently present with ACS. This number can be expected to increase over time because society is aging. Older adults often sustain unfavorable outcomes from ACS because of atypical presentation and delay in recognition.

In addition, elderly patients commonly do not receive optimal guideline-directed ACS treatment. Owing to their high baseline risk of ischemic complications, the elderly also fare worse even with optimal ACS treatment as they frequently have more complex coronary disease, more comorbidities, less cardiovascular reserve, and a higher risk of treatment complications. They are also subjected to a broader range of pharmacologic treatment. Treatment complications can be mitigated to some extent by meticulous dose adjustment of antithrombotic and adjunctive therapies.

Randomized clinical trials, on the other hand, have included substantially fewer elderly patients than clinicians encounter in real life (1). Thus, the basis of evidence forming the foundation of ACS treatment may not apply to a large number of patients, and clinicians need to extrapolate evidence to match their older patients' needs and preferences. Sixty percent of ACS hospitalizations occur in patients older than 65 years, and 85% of ACS mortality occurs in the Medicare population. Most deaths related to myocardial infarction occur in patients older than 65 years of age (2).

Age is not only a powerful risk factor for cardiovascular disease; it is also an independent risk factor for adverse outcomes after cardiovascular events, for complications of cardiovascular procedures and interventions, and for side effects of pharmacotherapy, particularly from antithrombotic therapies. The mortality rate after a first non-ST segment elevation myocardial infarction (non-STEMI) in very elderly patients is very high: with respect to 1-year outcomes, among patients who were 65–79, 80–84, 85–89, and at least 90 years old, mortality increased progressively from 13.3% to 23.6%, 33.6%, and 45.5%, respectively (3).

In addition, older patients generally have more complex cardiovascular disease, more comorbidities, and generally a more atypical clinical presentation. There is a greater prevalence of hypertension, congestive heart failure (CHF), atrial fibrillation, cerebrovascular disease, anemia, and renal insufficiency in older patients with ACS. Age also has important implications on pharmacokinetics and pharmacodynamics (4). Challenges in taking care of elderly patients with ACS include timely recognition, not withholding lifesaving therapies on the basis of age alone and respecting the patients' preferences and goals of care.





CONCLUSION

As the population continues to age, physicians will be confronted with an increasing number of elderly and very elderly patients presenting with ACS. While care needs to be individualized, age alone should never be the reason to withhold potentially lifesaving procedures and interventions. Elderly patients are at high risk for bleeding complications, but they are also at the highest risk for ischemic complications if less aggressive treatment strategies are pursued. So clinicians are tasked with meticulous risk stratification for ischemic risk and bleeding risk while taking into account assessment of frailty, quality of life, goals of care, and individual preferences. Early invasive protocols seem to be just as feasible in the elderly as in the general population.

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December 2023 14

Diagnosis and Management of Cow's Milk Protein Allergy in Children

Addressing Cow's Milk Protein Allergy in Children: Diagnostic Insight and Effective Management

INTRODUCTION

Cow's Milk Protein Allergy (CMPA) is an immune-mediated reaction to cow's milk proteins, primarily affecting infants and younger children (1). While CMPA is most common in infancy (2–3%), its prevalence decreases to less than 1% by 6 years of age (2). CMPA can be IgE-mediated, non-IgE-mediated, or mixed, leading to skin, respiratory, and gastrointestinal symptoms (2). Around 10-35% of children with CMPA also have soy allergy (1).

While most children eventually outgrow CMPA and develop tolerance, the rate varies based on the type of allergy (3). Limited awareness among paediatricians regarding CMPA contributes to misdiagnosis, incorrect dietary advice, and unnecessary expenses (4). Advances in comprehending CMPA's immune mechanisms have shifted dietary management towards active interventions to encourage tolerance development (5).

This article discusses the different diagnostic approaches and management strategies for CMPA in children. It emphasizes the need for more active interventions to encourage tolerance development and improve long-term outcomes for affected children.

CLASSIFICATION OF CMPA

CMPA can be classified into two main subtypes: IgE-mediated and non-IgE-mediated allergies, sometimes featuring mixed traits (2).

- **IgE-mediated allergies** prompt rapid symptoms, typically within hours, and may involve skin, gastrointestinal, and respiratory reactions (3).
- **Non-IgE-mediated allergies** show delayed symptoms and are further categorized into distinct conditions (6):
 - Food protein-induced enterocolitis syndrome (FPIES) triggers repetitive vomiting followed by diarrhea, often accompanied by lethargy and other symptoms (6).
 - Food protein-induced enteropathy (FPE) mimics celiac disease, leading to chronic diarrhea and malabsorption after cow's milk consumption (6).







Paediatrics (specialist) Aster Clinic, Al Quoz, Dubai







 Food protein-induced allergic proctocolitis (FPIAP) affects breastfed infants, causing inflammation in the rectum and colon, resulting in bloody, mucus-containing stools (6).



Figure 1: Symptoms related to IgE-mediated CMPA (3)



Figure 2: Symptoms related to non-IgE mediated CMPA (3)

DIAGNOSTIC MODALITIES OF CMPA

Diagnosing CMPA relies on a reliable history and thorough clinical examination (1). Other predisposing conditions like infective colitis, celiac disease, gastroesophageal reflux disease, and immune deficiency should be considered (4). The diagnostic process involves various methods:

1. DIAGNOSTIC ELIMINATION TRIAL

According to the most recent recommendations from the European Society of Paediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN), the initial approach to diagnosing CMPA involves excluding cow's milk proteins from the diet (2). This approach is designed to monitor if symptoms show improvement during the elimination diet (2). Following this, the verification of CMPA diagnosis includes conducting an oral challenge using cow's milk to determine whether symptoms return (2). If strict elimination fails to alleviate symptoms, the likelihood of CMPA diagnosis is uncertain (2).





2. ORAL FOOD CHALLENGE

The gold standard for confirming CMPA is the double-blind placebo-controlled food challenge, but its feasibility is limited by its lengthy duration and associated costs (2). Oral food challenges (OFCs) play a crucial role in diagnosing food allergies by accurately distinguishing between sensitization and clinical allergy (4). These challenges are primarily standardized for lgE-mediated reactions and are performed with medical oversight (4). In cases of severe anaphylaxis, immediate therapeutic elimination is prioritized (4).

3. OTHER METHODS:

- Specific IgE Antibodies to Cow's Milk: This test detects circulating antibodies against CMP (2). However, the IgE result doesn't confirm allergy or differentiate sensitization from clinical allergy (4). It is not effective for diagnosing non-IgE-mediated CMPA (2).
- Skin Prick Test: This assessment detects IgE-bound antibodies within the skin, making it valuable for diagnosing IgE-mediated conditions (2). However, a positive result alone doesn't necessarily confirm an allergy conclusively (2).
- **Component-Resolved Allergy Testing:** This technique employs allergen-specific IgE and IgG4 antibodies obtained from purified or recombinant allergens (6).
- **Basophil Activation Test (BAT):** BAT is an in vitro test for diagnosing IgE-mediated allergies, measuring basophil activation through CD63 and CD203c markers (6).

MANAGEMENT OF CMPA

The most prudent approach to CMPA management is strictly avoiding cow's milk for a specified duration (2). A delayed diagnosis can give rise to concerns such as failure to thrive, anaemia, and hypoproteinaemia (4). Nonetheless, an inaccurate diagnosis can result in unwarranted dietary restrictions, elevating the risk of issues like rickets, diminished bone mineralization, and economic strain (4). It is imperative to carefully select appropriate alternatives to fulfil nutritional requirements while avoiding cow's milk (4). Factors such as age, feeding pattern, allergy type, reaction severity, and clinical manifestations should be considered before recommending milk substitutes (4).

EXCLUSIVELY BREASTFED INFANTS

Exclusively breastfed infants are recommended to continue breastfeeding for at least 6 months while the mother refrains from consuming cow's milk and dairy products in her diet (2). After stopping cow's milk, it might take up to 72 hours for antigens to clear from breast milk (2). If symptoms persist after elimination, consideration should be given to other allergens or potential causes (2).

EXCLUSIVELY FORMULA-FED INFANTS

When dealing with infants relying solely on formula, it is crucial to choose suitable alternatives



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for managing CMPA (2). AAF and eHF are recommended to replace standard cow's milk-based formula (2). However, it's essential to exercise caution when considering soy-based options for infants under 6 months due to safety concerns and the possibility of cross-allergy, observed in 10-15% of CMPA cases (2).

INFANTS ON MIXED FEEDS

The recommended approach for infants younger than 6 months with mild to moderate reactions is to remove cow's milk protein from their diet (2). Breastfeeding should continue without requiring dietary elimination. In this group, eHF is the recommended option (2). For infants over 6 months of age with mild to moderate reactions, soy protein formula can be considered a replacement for eHF, considering financial factors (2). AAF should be prioritized if there is no improvement within 2 weeks or in severe or life-threatening conditions (2). In situations of uncertainty, AAF should be considered before ruling out CMPA, particularly for ill infants or those presenting severe symptoms (4).

MILK LADDER

Cow's milk allergy will resolve in majority of the children (3). They should be reassessed at 6–12 monthly intervals from 1 year of life to check if cow's milk can be reintroduced into their diet (3). The reintroduction of cow's milk may be graded as per the 'milk ladder' with less allergenic forms offered initially (3). More allergenic forms are then introduced later as tolerated (3). Reintroduction can be performed at home or in a hospital-based on the severity of the reaction (3).

CONCLUSION

Diverse manifestations of CMPA, including IgE-mediated and non-IgE-mediated allergies, underscore the complexity of diagnosis and management (2). Advances in understanding the immune mechanisms driving CMPA have led to a more active approach to promoting tolerance development (6). A comprehensive evaluation is necessary to ensure an accurate diagnosis, involving diagnostic elimination trials, oral food challenges and various testing methods (2). Effective management strategies encompass strict avoidance of cow's milk and carefully considering suitable alternatives (2). The balance between addressing nutritional needs and allergen avoidance remains central to CMPA management (2).





Key Highlights

- Cow's Milk Protein Allergy is a common condition in infants and young children caused by an abnormal immune response to cow's milk proteins (1,2).
- The diagnosis of CMPA relies on an elimination diet with subsequent oral food challenge for clinical response assessment (1).
- The management of CMPA includes avoiding cow's milk, initiating breastfeeding, and considering options like eHF and AAF for infants (1).

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Roux-en-Y Hepaticojejunostomy (RYHJ)

Successful Management of Post Laparoscopic Cholecystectomy Bile Duct Injury and Complex Biliary Stricture at Aster Hospital, Mankhool

BACKGROUND HISTORY

- 27 year old female
- Medical history of:
 - Acute Calculous Cholecystitis
 - Underwent Laparoscopic Cholecystectomy at another hospital. As per the surgeon, surgery remained uneventful and showed:
 - Adhesions covering <50% of gallbladder
 - Distended Gallbladder with the inability to grasp with atraumatic laparoscopic forceps
 - Pericholecystic Fluid: Present
 - Cystic Duct: Normal
 - Stones in GB: Multiple
 - Liver: Multiple hemangiomas of 1-2 cm
- Post-operative USG abdomen showed no significant collection, and the common bile duct appeared normal in size. But, on follow-up after 5 days, LFT showed an increasing trend with high WBC.
- MRI study of the upper abdomen was done, which showed:
 - Right and left hepatic duct narrowing at the confluence.
 - Suboptimal visualization of common hepatic duct below the right and left hepatic duct confluence due to fluid collection in GB fossa.
 - Focal collection noted in GB fossa just below the confluence of the right and left hepatic ducts measuring 2.4 x 1.7 cm.
 - Free fluid collection noted around the liver surrounding spleen left hypochondrium inter bowel loops bilateral iliac fossa (R > L) and in the pelvis.
- In view of the same, a pigtail catheter under USG guidance was decided in the hepatorenal pouch. However, a drain was placed in the pelvis-dependent part due to inadequate space. The fluid drained was bilious (approx. 1.5 L total in 2 days).



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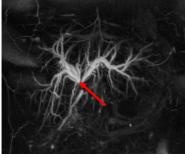


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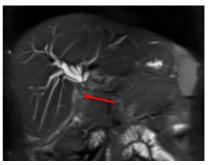


PRESENTATION

- Since the patient was having persistent symptoms of Epigastric and left hypochondrial pain due to inadequate drainage even after pigtail insertion, she was referred to a Surgical Gastroenterologist at Aster Hospital, Mankhool on POD10.
- She underwent Laparoscopic Lavage and drain placement in the sub-hepatic region.
- Initially, the drain output was approximately 250-400/Day Bilious, and as the patient was
 free of pain and sepsis, she was discharged with a drain in situ to follow up in OPD with a
 plan of RYHJ once stricture developed.
- She came for a follow-up after 4 weeks with minimal drain output and progressive jaundice.
- MRCP of abdomen was done, which showed:
 - Moderate central and peripheral bilobar intrahepatic biliary radicle dilatation was seen.
 - The confluence was not appropriately formed with only a common roof, but septum present between right and left hepatic duct suggesting a Type III B Benign Biliary Stricture.
 - The common hepatic duct/proximal CBD was poorly visualised for approximately 7-8 mm in length.
 - Postoperative drain was noted with the proximal tip at the gallbladder fossa just inferior and posterior to the primary confluence. Minimal free fluid was seen in the gallbladder fossa.
- Liver function tests were done, which showed: Total Bilirubin 7.61, SGOT/SGPT 374/826, GGT 880, and Alkaline Phosphatase 662.
- Given the biliary structure and rising bilirubin, she was taken up for Roux-en-Y Hepaticojejunostomy (HJ) under general anaesthesia.



Structure at Confluence



Drain at Hilum

INTRA-OPERATIVE FINDINGS

- Dense adhesions present between the Omentum, Gall Bladder Fossa, and Liver.
- Drain tract was seen extending from abdominal wall to the hilum.
- Duodenum densely adherent to the hilum. Clips were seen in GB fossa.
- Stricture present involving the confluence with non-patent confluence.
- Right and left hepatic duct separated by a septum suggestive of Type IIIB BBS.

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SURGICAL PROCEDURE

- The patient was laid in supine position.
- Parts were painted and draped in a usual sterile manner.
- Subcostal incision was made, and the abdomen was opened in layers.
- Adhesiolysis was done, and omentum was separated from the liver.
- Drain was seen extending till hilum and was removed.
- Intra-op findings were identified, and the base of segment IV of the liver was dissected above to expose the confluence.
- Confluence was dissected and right and left hepatic ducts were identified.
- Septoplasty was done using PDS 4-0.
- ROUX limb was made using a 55 mm triple layer cartridge and brought out in retrocolic fashion through transverse mesocolon.
- End-to-side Hepaticojejunostomy (HJ) done of approximately 2.5 cm using PDS 4-0 sutures in an interrupted fashion.
- Jejunojejunostomy was done in two layers continuously using PDS 4-0 at approximately 60 cm distal to HJ.
- Lavage done and hemostasis achieved, and absence of bile leak was confirmed.
- 28F drain was placed. The sheath was closed with PDS No 1 loop.
- Skin closed with the stapler.



Forceps in Left Hepatic Duct



Forceps in Right Hepatic Duct



Forceps demonstrating Septoplasty between Right and Left Ducts



Anterior Preplaced Sutures

Roux Limb of Jejunum anastomosed to Hepatic Duct

POST PROCEDURE

Post-operatively, the patient was managed in HDU with epidural analgesia and strict monitoring. She was shifted to the ward on POD2. The oral diet was gradually advanced. The epidural catheter and drain were removed on POD3. She was tolerating a normal diet and remained haemodynamically stable throughout. LFT showed a decreasing trend, and jaundice subsided. She was discharged in a stable condition.





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FOLLOW UP

At the 3-month follow-up, the patient was found asymptomatic with normalised liver function tests and USG showing normal intrahepatic biliary radicles suggestive of successful repair.

DISCUSSION

latrogenic bile duct injury is a dreaded complication of cholecystectomy and has been estimated to occur in approximately 0.5 to 0.9 % of cases. The introduction of laparoscopic cholecystectomy in the 1990s and its widespread application since then has resulted in a marked increase in the incidence of BDIs. In addition, BDI associated with laparoscopic cholecystectomy tends to be more complex than after an open approach. Early diagnosis of BDI is essential for reducing morbidity and mortality. However, only 25%–30% of BDI are diagnosed intraoperatively.

The surgical biliary repair remains the mainstay treatment of BDI. The outcome of surgery greatly depends on the repair technique and the surgeon's experience. The result of immediate BDI repair has comparable long-term outcomes in expert hands compared to delayed repair. Adherence to the fundamental principles of bile duct repair with the correct surgical technique is vital for long-term success. The repair should be tension-free, between well-vascularised segments of duct and mucosa and drain all liver segments.

The important factors determining the success of biliary reconstruction include:

- The complete eradication of intra-abdominal infection
- Complete characterisation of the injury with cholangiography
- Use of the correct surgical technique and repair performed by an experienced biliary surgeon.

CONCLUSION

The present case emphasises the importance of early recognition of bile duct injury and its management at a specialised hepatobiliary centre. Apparently, a simple injury can turn out to be a major one, necessitating complex repair surgery. Management at an experienced centre, correct surgical technique and complete delineation of biliary anatomy before surgery are vital for long-term repair success.

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