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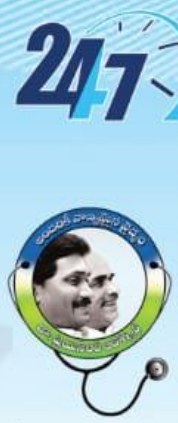

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DEPARTMENT OF PAEDIATRICS, ANDHRA HOSPITALS, VIJAYAWADA
E-JOURNAL

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Foreword

Greetings from the Andhra Hospitals!

For the past two decades, we are delivering excellence in terms of quality health care in the field of Paediatrics including Paediatric and Neonatal intensive care, Paediatric cardiac intensive care and other allied Paediatric sub-specialities.We take pride in introducing ECMO in pediatric specialty for the first time in the state of Andhra Pradesh. In this context, we convey our heartfelt thanks to you for your continued support and encouragement which played a pivotal role in our success.

As a token of our commitment to contribute to continuous medical education, we are introducing this monthly E-journal to showcase important clinical guidelines, recent advances in paediatric sub-specialties, interesting case reports, image quiz, OSCE scenarios etc, gathered from our patient database.

We hope this endeavor would prove to be useful to practicing paediatricians, intensivists, neonatologists and post-graduate students.

Please send your valuable feedback and suggestions to ***maramkp@gmail.com***.

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Multisystem inflammatory syndrome related to COVID-19 in children

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Introduction:

The recent pandemic caused by coronavirus disease-2019 (COVID-19) continues to inflict significant morbidity and mortality. National statistics from countries in Asia, Europe, and North America show that paediatric cases account for 2·1–7·8% of confirmed COVID-19 cases. (1) Unlike adults, the vast majority of children with COVID-19 have mild symptoms.(2) But recently a subset of children manifested with severe inflammation, multi-organ dysfunction needing ICU admission. In the past 3 months, there have been increasing reports from Europe, North America, Asia, and Latin America describing children and adolescents with COVID-19 associated multisystem inflammatory conditions, which seem to develop after the infection rather than during the acute stage of COVID-19.(1) The clinical features of these paediatric cases are both similar and distinct from other well described inflammatory syndromes in children, including Kawasaki disease (KD), Kawasaki disease shock syndrome (KDSS), and toxic shock syndrome (TSS). (1) This COVID-19 associated multisystem inflammatory syndrome in children and adolescents is referred to interchangeably as paediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2 (PIMS-TS) or multisystem inflammatory syndrome in children (MIS-C) associated with COVID-19, and herein is referred to as MIS-C.(1) Much remains unknown regarding the epidemiology, pathogenesis, clinical spectrum, and long-term outcomes of MIS-C. In this Review, we critically appraise and summarise the available evidence to provide insights into current clinical practice and implications for future research directions.

Case definitions and clinical spectrum:

Different terminology and case definitions for this COVID19-associated multisystem inflammatory phenotype in children are used depending on the country and region.

An internationally accepted case definition for MIS-C is still evolving. All the definitions require fever (though they differ with respect to duration), elevated inflammatory markers, at least two signs of multisystem involvement, evidence of SARS-CoV-2 infection or exposure, and exclusion of other potential causes.

Overlap has also been observed between the diagnostic criteria of KD, KDSS, and the newly emerged MIS-C. A large number of MIS-C cases present with Kawasaki-like clinical symptoms, and cardiac impairment and shock similar to KDSS. Gastrointestinal symptoms, hyponatremia, hypoalbuminemia, and intravenous immunoglobulin resistance are also common in KDSS and MIS-C.

	MIS-C associated with COVID-19	PIMS-TS	MIS-C associated with COVID-19	Complete Kawasaki disease	Incomplete Kawasaki disease	Kawasaki disease shock syndrome
Organisation or publication	WHO[6]	Royal College of Pediatrics and Child Health[39]	US Centers for Disease Control and Prevention[37]	American Heart Association[40]	American Heart Association[40]	Kanegaye et al,[41]
Age	0-19 years	Child (age not specified)	<21 years	Child (age not specified)	Child (age not specified)	Child (age not specified)
Inflammation	Fever and elevated inflammatory markers for 3 days or more	Fever and elevated inflammatory markers	Fever and elevated inflammatory markers	Fever lasting 5 days or more*	Fever lasting 5 days or more*	Fever
Main features	Two of the following: (A) rash or bilateral non-purulent conjunctivitis or mucocutaneous inflammation signs (oral, hands, or feet); (B) hypotension or shock; (C) features of myocardial dysfunction, pericarditis, valvulitis, or coronary abnormalities (including echocardiogram findings or elevated troponin or N-terminal pro B-type natriuretic peptide); (D) evidence of coagulopathy (elevated prothrombin time, partial thromboplastin time, and elevated D-dimers); and (E) acute gastrointestinal problems (diarrhoea, vomiting, or abdominal pain)	Single or multiple organ dysfunction (shock or respiratory, renal, gastrointestinal, or neurological disorder; additional features (appendix 6 pp 3–4)	Clinically severe illness requiring hospitalisation; and multisystem (two or more) organ involvement (cardiac, renal, respiratory, haematological, gastrointestinal, dermatological, or neurological)	Four or more principal clinical features: (A) erythema and cracking of lips, strawberry tongue or oral and pharyngeal mucosa; (B) bilateral bulbar conjunctival injection without exudate; (C) rash; (D) erythema and oedema of the hands and feet in acute phase and periungual desquamation in subacute phase; and (E) cervical lymphadenopathy	Two or three principal clinical features or a positive echocardiogram	Kawasaki disease-like clinical features and any of the following causing initiation of volume expansion, vasoactive agents, or transfer to the intensive care unit: systolic hypotension based on age, or a decrease in systolic blood pressure from baseline by 20% or more, or clinical signs of poor perfusion
Exclusion	Other microbial cause of inflammation	Any other microbial cause	Other plausible alternative diagnoses	–	–	Other microbial cause
SARS-CoV-2 status	Positive RT-PCR, antigen test, or serology; or any contact with patients with COVID-19	RT-PCR positive or negative	Positive RT-PCR, serology, or antigen test; or COVID-19 exposure within the past 4 weeks before symptom onset	–	–	–

MIS-C=multisystem inflammatory syndrome in children. PIMS-TS=paediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2. SARS-CoV-2=severe acute respiratory syndrome coronavirus 2. *In the presence of four or more principal clinical features, particularly when redness and swelling of the hands and feet are present, the diagnosis of Kawasaki disease can be made with only 4 days of fever.

Table 1: Preliminary case definitions for MIS-C

Parameter	MISC	KD
Age	Older children and adolescents	Infants and young children
GIT complaints	More common	Less common
LV dysfunction	More common	Less common
Shock	More common	Less common
Elevated ferritin and D-dimer	More common	Less common
Hyponatremia	More common	Less common
Platelet count	Low normal or Low	High normal or elevated
Need of adjunctive steroid treatment	Common	Less

Table 2: Differentiating features of MIS-C from Kawasaki disease (KD).(3)

Pathophysiology:

The pathophysiology of MIS-C is not well understood. It has been suggested that the syndrome results from an abnormal immune response to the virus, with some clinical similarities to KD, macrophage activation syndrome, and cytokine release syndrome. However, based on the available studies, MIS-C appears to have an immunophenotype that is distinct from KD and MAS. The mechanisms by which SARS-CoV-2 triggers the abnormal immune response are unknown. A post infectious process is suggested, based on the timing of the rise of these cases relative to the peak of COVID-19 cases in communities.(1)

Antibodies might enhance disease by increasing viral entry into cells. Alternative mechanisms include antibody or T-cell-mediated cell damage or activation of inflammation. Antibodies or T cells attack cells expressing viral antigens or attack host antigens which cross-react or mimic viral antigens. The low rate of virus detection in MIS-C would favour this second mechanism rather than the classic antibody-dependent enhancement.(1)

Clinical manifestations:

Presenting symptoms — In the available case series, clinical presentations were similar, including (1,3–7):

- Persistent fevers (median duration four to six days) – 100 percent
- Gastrointestinal symptoms (abdominal pain, vomiting, diarrhea) – 60 to 100 percent
- Rash – 45 to 76 percent
- Conjunctivitis – 30 to 81 percent
- Mucous membrane involvement – 27 to 76 percent
- Neurocognitive symptoms (headache, lethargy, confusion) – 29 to 58 percent
- Respiratory symptoms – 21 to 65 percent

● Sore throat – 10 to 16 percent
● Myalgia – 8 to 17 percent
● Swollen hands/feet – 9 to 16 percent
● Lymphadenopathy – 6 to 16 percent

Most patients present with three to five days of fever, though fewer days of fever have been reported. In the largest series, which included 186 patients, 10 percent had three days of fever, 12 percent had four days, and 78 percent had ≥5 days.

Gastrointestinal symptoms (abdominal pain, vomiting, diarrhoea) are particularly common and prominent, with the presentation in some children mimicking appendicitis. Some children have been noted to have terminal ileitis on abdominal imaging and/or colitis on colonoscopy.

Respiratory symptoms (tachypnoea, laboured breathing), when present, are most often due to severe shock. Cough is uncommon. Though some children require supplemental oxygen or positive pressure ventilation for cardiovascular stabilization, severe pulmonary involvement (eg, acute respiratory distress syndrome) is not a prominent feature.

Neurocognitive symptoms are common and may include headache, lethargy, confusion, or irritability. A minority of patients present with more severe neurologic manifestations, including encephalopathy, seizures, coma, meningoencephalitis, muscle weakness, and brainstem and/or cerebellar signs.

Clinical findings — Common clinical findings reported in the available case series include(1,3–5,7–10):

● Shock – 32 to 76 percent

- Criteria met for complete Kawasaki disease (KD) – 22 to 64 percent
- Myocardial dysfunction (by echocardiogram and/or elevated troponin or brain natriuretic peptide [BNP]) – 51 to 90 percent
- Arrhythmia – 12 percent
- Acute respiratory failure requiring noninvasive or invasive ventilation – 28 to 52 percent
- Acute kidney injury (most cases were mild) – 8 to 52 percent
- Serositis (small pleural, pericardial, and ascitic effusions) – 24 to 57 percent
- Hepatitis or hepatomegaly – 5 to 21 percent
- Encephalopathy, seizures, coma, or meningoencephalitis – 6 to 7 percent

Laboratory findings — Laboratory abnormalities noted in the available case series include (1, 3-10):

- Abnormal blood cell counts, including:
  - ✓ Lymphocytopenia – 80 to 95 percent
  - ✓ Neutrophilia – 68 to 90 percent
  - ✓ Mild anemia – 70 percent
  - ✓ Thrombocytopenia – 31 to 80 percent
- Elevated inflammatory markers, including:
  - ✓ C-reactive protein (CRP) – 90 to 100 percent
  - ✓ Erythrocyte sedimentation rate – 75 to 80 percent
  - ✓ D-dimer – 67 to 100 percent
  - ✓ Fibrinogen – 80 to 100 percent
  - ✓ Ferritin – 55 to 76 percent
  - ✓ Procalcitonin – 80 to 95 percent
  - ✓ Interleukin-6 (IL-6) – 80 to 100 percent
- Elevated cardiac markers:
  - ✓ Troponin – 50 to 90 percent
  - ✓ BNP or NT-pro-BNP – 73 to 90 percent

- Hypoalbuminemia – 48 to 95 percent
- Mildly elevated liver enzymes – 62 to 70 percent
- Elevated lactate dehydrogenase – 10 to 60 percent
- Hypertriglyceridemia – 70 percent

Laboratory markers of inflammation appear to correlate with severity of illness. For example, children who developed shock had higher CRP values, higher neutrophil counts, lower lymphocyte counts, and lower serum albumin compared with children without shock. In addition, children with shock more commonly had elevated cardiac markers.

Imaging findings — Findings on diagnostic imaging may include (3-10):

- Echocardiography – Echocardiographic findings may include depressed LV function and coronary artery (CA) abnormalities (including dilation or aneurysm), mitral valve regurgitation, and pericardial effusion. The frequency of cardiac involvement in MIS-C is uncertain. In three large case series, approximately 30 to 40 percent of children had depressed LV function and 8 to 19 percent had CA abnormalities. These reports included patients with severe MIS-C as well as milder cases. Case series including only severely affected patients reported considerably higher rates of depressed LV function (approximately 50 to 60 percent) and CA abnormalities (approximately 20 to 50 percent), but these estimates probably do not reflect the risk in the broader population.

- Chest radiograph – Many patients had normal chest radiographs. Abnormal findings included small pleural effusions, patchy consolidations, focal consolidation, and atelectasis.

- Computed tomography (CT) of chest – Chest CT (when obtained) generally had findings similar to those on chest radiograph. A few patients had nodular ground-glass opacification.

- Abdominal imaging – Findings on abdominal ultrasound or CT included free fluid, ascites, and bowel and mesenteric inflammation including terminal ileitis, mesenteric adenopathy/adenitis, and pericholecystic edema.

EVALUATION:

Patients with suspected MIS-C should have laboratory studies performed to look for evidence of inflammation and to assess cardiac, renal, and hepatic function. Testing should also include polymerase chain reaction (PCR) and serology for SARS-CoV-2. In addition, patients should be assessed for other infectious or noninfectious conditions that may have a similar presentation.

Our approach outlined below is generally consistent with guidance published by the American College of Rheumatology and the American Academy of Pediatrics.
The initial laboratory evaluation of a child with suspected MIS-C depends on the presentation.

Mild illness	Moderate to severe
1. CBC with differential 2. CRP 3. Serum electrolytes and renal function tests	1. Complete blood count (CBC) with differential 2. C-reactive protein (CRP) and erythrocyte sedimentation rate (optional: procalcitonin)

If these results are abnormal, additional testing is performed.	3. Ferritin 4. Liver function tests 5. Serum electrolytes and renal function tests 6. Urinalysis 7. Lactate dehydrogenase 8. Coagulation studies (prothrombin time/international normalized ratio, activated partial thromboplastin time, D-dimer, fibrinogen) 9. Troponin I 10. Brain natriuretic peptide (BNP) or NT-pro-BNP 11. Cytokine panel (if available)

Inflammatory markers (CRP, erythrocyte sedimentation rate, procalcitonin, ferritin) are measured at the time of admission and then serially to monitor progression. Similarly, if cardiac markers (troponin and BNP) are elevated, they should be followed serially to monitor progression.

The clinician should also assess for other common causes of fever (eg, streptococcal pharyngitis, mononucleosis). While it does not definitively exclude MIS-C, identifying another source of fever makes the diagnosis of MIS-C less likely, particularly in an otherwise well-appearing child.

Additional testing for other pathogens may be warranted, depending on the geographic location and exposure history. This may include:

Scrub typhus IgM

Dengue NS1 and IgM

Leptospira serology

Testing for SARS-CoV-2 — All patients with suspected MIS-C should be tested for SARS-CoV-2, including both serology and RT-PCR on a nasopharyngeal swab.

As previously discussed, approximately 60 percent of patients have positive serology with negative PCR, and approximately 30 to 35 percent are positive on both tests.A minority of patients (approximately 5 to 10 percent) have negative results on both tests. In these cases, the diagnosis of MIS-C requires an epidemiologic link to SARS-CoV-2 (eg, exposure to an individual with known COVID-19 within the four weeks prior to the onset of symptoms).

Detection of other respiratory pathogens (eg, rhinovirus, influenza, respiratory syncytial virus) in nasopharyngeal specimens does not exclude COVID-19.

Cardiac testing — In addition to troponin and BNP/NT-pro-BNP levels, the cardiac evaluation of a patient with suspected MIS-C includes a 12-lead electrocardiogram (ECG) and echocardiography. Echocardiography is also recommended for children with documented SARS-CoV-2 who do not meet all criteria for MIS-C but who have either shock or features consistent with incomplete or complete Kawasaki disease (KD).

Children and adolescents with mild COVID-19 without signs of systemic inflammation are unlikely to have coronary artery (CA) changes or myocarditis. In such children, echocardiography is generally not necessary but may be considered if there are specific clinical concerns.

In children with MIS-C, baseline ECGs may be nonspecific, though arrhythmia and heart block have been described. Findings on initial echocardiography may include CA dilation, left ventricular (LV) systolic dysfunction, and pericardial effusion. The CA abnormalities can progress to aneurysm, including giant coronary aneurysms.

● **Echocardiographic evaluation** – The echocardiographic evaluation includes the following:

- Quantitative assessment of LV size and systolic function (LV end-diastolic volume, ejection fraction)
- Qualitative assessment of right ventricular systolic function
- CA abnormalities (dilation or aneurysm)
- Assessment of valvar function
- Evaluation for the presence and size of pericardial effusion
- Evaluation for intracardiac thrombosis and/or pulmonary artery thrombosis, particularly apical thrombus in severe LV dysfunction
- Strain imaging and LV diastolic function (optional)
- CA assessment is based on Z-scores, with the same classification schema used in KD

● **Timing of follow-up echocardiography** – At our center, echocardiography is performed at the time of diagnosis, with follow-up examinations at the following intervals:

- In patients who initially have normal function and normal CA dimensions, follow-up echocardiogram is performed one to two weeks post-diagnosis to recheck CA size.
- In patients who have CA dilation/aneurysm on initial echocardiogram, echocardiography is repeated every two to three days until CA size is stable and then every one to two weeks for the next four to six weeks.
- For patients with systolic dysfunction/myocarditis and normal CAs on initial echocardiogram, the echocardiogram is repeated as clinically indicated, including repeat imaging of the CAs with each study.
- For patients who had evidence of CA involvement or systolic dysfunction/myocarditis in the acute phase, cardiac magnetic resonance

imaging can be considered at approximately two to six months after the acute illness to assess ventricular function and evaluate for edema, diffuse fibrosis, and scar by myocardial delayed enhancement.

DIFFERENTIAL DIAGNOSIS:

In children presenting with signs and symptoms consistent with MIS-C, the differential diagnosis is broad and includes other infectious and inflammatory conditions:

1. Bacterial sepsis .
2. Kawasaki disease (KD) .
3. Toxic shock syndrome.
4. Dengue and scrub typhus.
5. Appendicitis.
6. Other viral infections – Other viral pathogens that may manifest with multisystem involvement and/or myocarditis include Epstein-Barr virus, cytomegalovirus, adenovirus, and enteroviruses. These viruses rarely cause severe multisystem disease in immunocompetent children. Serology and polymerase chain reaction (PCR) testing can distinguish these from COVID-19-related MIS-C.
7. Hemophagocytic lymphohistiocytosis (HLH)/macrophage activation syndrome (MAS) –Most children with HLH/MAS are acutely ill with multiorgan involvement, cytopenias, liver function abnormalities, and neurologic symptoms.Cardiac and gastrointestinal involvement are less common, and neurologic symptoms are more prominent.
8. Systemic lupus erythematosus (SLE) – SLE can present with fulminant multisystem illness. Such patients generally have considerable kidney and central nervous system involvement, which are not common features of MIS-C.
9. Vasculitis.

Management:

1. Mainly supportive + Immunomodulation
2. Empiric antibiotics to be started and continued till blood/urine cultures are ready.
3. Clindamycin has to be added if TSS is suspected.
4. Fluid resuscitation and inotropes/vasopressors in shock.
5. Respiratory support if needed.

Immunomodulatory therapy:

1st line: Intravenous immunoglobulin(IVIG) 2gm/kg calculated according to ideal body weight.

2nd line: Intravenous Methylprednisolone (MPS) 10-30mg/kg/day, for children who remain unwell 24 hour after IVIG infusion, particularly if they have ongoing pyrexia.

3rd line: Biological therapy-Tocilizumab or Anakinra, for children who not responded to IVIG+ MPS.

This management pathway is based primarily on expert opinion and should be updated as new evidence emerges.

Indication for IVIG:

1. Features of kawasaki disease (complete or incomplete)
2. Evidence of coronary artery abnormality
3. Meeting criteria of toxic shock syndrome
4. Evidence of progressive disease
5. Extended duration of fever (>5days)

Indiaction of IVIG + MPS as 1st line therapy:

For life-threatening complications, such as myocarditis/ shock, and specifically, if a patient requires high dose or multiple inotropes and/or vasopressors.

Role of antivirals:

Remdesivir can be considered in children with MIS-C when PCR is positive and having severe illness not responding to IVIG and steroids.

Antiplatelet and Anticoagulation therapy: Controversial in children

1. MIS-C children having kawasaki phenotype should receive aspirin according KD treatment guidelines i.e 30-50mg/kg/day till fever subsides then 3-5mg/kg/day.
2. All other children should receive low dose aspirin (3-5mg/kg/day) for a minimum of 6 weeks or till repeat ECHO shown normal coronaries.
3. Children with documented thrombosis, grossly dilated CAA or EF <35% should receive therapeutic anticoagulation with enoxaparin.
4. Children with high risk for thrombosis, elevated D-dimers and age above 12 yrs should receive prophylactic enoxaparin. (weak eveidence)

Key messages:

1. MISC is a novel condition that has emerged during the COVID-19 pandemic; ongoing research into its cause, disease course, and therapies that improve the outcomes of children with the condition is essential.

2. Children suspected of having MISC should undergo first line blood tests to determine if they meet the diagnostic criteria; subsequent tests to determine the severity of disease, exclude important differential diagnoses, and screen for cardiac involvement are recommended

3. A multidisciplinary team is an essential facet in the care of children with MISC and every child with suspected MISC should be discussed with a multidisciplinary team within 24 h of suspected diagnosis and when considering biological therapy

4. Therapeutic choices for MISC are dependent on the presenting phenotype (Kawasaki disease-like or non-specific presentation) and high-risk features or severity of disease; a step-wise pathway of intravenous immunoglobulin, followed by methylprednisolone and biological therapy is recommended for children.

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Image quiz

Dr Lalitha Sudha.Chigurupati

MRCPCH Trainee, Andhra Hospital

A) What is the diagnosis?

B) What is the mode of inheritance?

C) What is the management?

Answers:

A. Crouzon syndrome or cranial dysostosis.

B. Autosomal Dominant inheritance

C. Surgical management to relieve pressure on brain and to improve shape of the head.

Discussion:

Crouzon syndrome is a genetic disorder characterized by premature closure of cranial sutures. This prevents the skull from growing normally and affects the shape of head and face.The main cause for this is mutation in FGFR2 gene on chromosome 10. (Fibroblast growth factor receptor gene 2). It is inherited in autosomal dominant pattern.

Signs and Symptoms:

A defining character of crouzon syndrome is **craniosynostosis**, which results in abnormal head shape. This is present in combination on turricephaly, frontal bossing and trigonocephaly(fusion of metopic suture), brachycephaly(fusion of coronal suture), Dolichocephaly(fusion of saggital suture), Plagiocephaly(unilateral fusion of lambdoid and coronal suture)

Exophthalmos due to shallow eye sockets with vision problems

Hypertelorism

Psittichorhina(beak like nose)

Other facial characteristics include external strabismus and hypoplastic maxilla, which results in relative mandibular prognathism, which gives the effect of concave face.

Hypodontia and crowding of teeth due to maxillary hypoplasia.

Approximately 30% of children develop Hydrocephalus.

Sensorineural hearing loss is present in some children.

Investigations: Measuring the head circumference

X-ray of the skull

CT scan of the head

MRI scan.

Management:

Surgery is typically used to prevent closure of sutures of the skull. Strip Craniectomy or open vault surgery is usually done. Strip craniectomy is done in children less than 6 months old and following this a helmet is worn for several months. With out surgery blindness and intellectual disability are typical outcomes.

Reshaping the orbits and moving the orbital and maxillary bones forward correct the midfacial hypoplasia.

Following surgery these children tend to have a normal development and life span.

Case Report: A case of Massive Pericardial & Pleural effusion

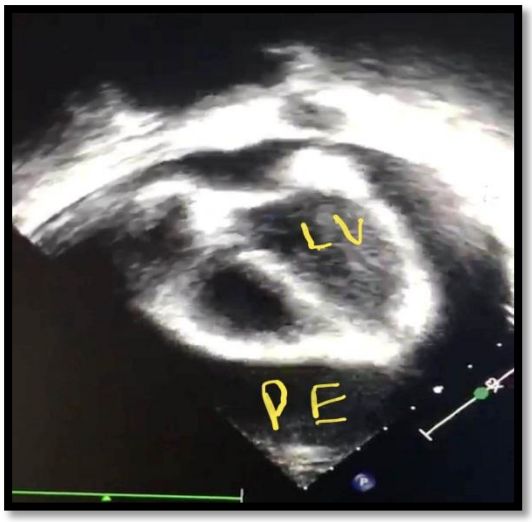
Dr Deepthi Varanasi

MRCPCH Trainee, Andhra Hospitals

Pericarditis is inflammation of the pericardium that envelopes the heart and normally contains a small amount of serous fluid. Though small amount of pericardial effusion is well tolerated and clinically silent, massive fluid accumulation may result in distension of pericardium and an impairment of cardiac filling, which is termed cardiac tamponade. Viral infection is probably the most common cause of pericarditis, although a third of the cases have unknown ethology and are termed idiopathic. Coxsackievirus, an RNA enterovirus, is an invasive pathogen that is capable of causing conditions ranging from mild respiratory and gastrointestinal illness to life- threatening myocarditis and pericarditis. Herein, we present a case of coxsackievirus infection causing pericarditis with cardiac tamponade and pleural effusion.

Case Report

An 11 years old female child, who was apparently well 6 days prior to admission, complained of sudden unset of diffuse abdominal pain, not responding to medication, followed by vomitings, cough and shortness of breath. She had non–bilious, non-projectile vomitings that subsided with medication. She then developed shortness of breath and non-productive cough 4 days prior to admission.



2D-ECHO showing massive pericardial effusion causing a tamponade effect

Haemorrhagic pericardial fluid

Further history revealed no recent infection of any family members, no un-usual food consumption, no weight loss, negative for history of contact with TB patients. Past medical history was significant for febrile seizures at 5 years of age. The child was taken to a hospital in her hometown where a 2D Echo revealed massive pericardial effusion for which she was referred to our hospital for further management.At the time of presentation, she was hemodynamically unstable with hypotension and tachycardia and moderate respiratory distress.

She was resuscitated as per standard PALS guidelines. An emergency 2D-ECHO demonstrated a massive pericardial effusion with cardiac tamponade along with massive bilateral pleural effusions. Emergency pericardiocentesis was performed which drained around 600 ml of hemorhagic fluid.She also had bilateral tube thoracostomy to drain pleural effusions.She had immediate stmptomatic relief following pericardiocentesis and thoracocentesis.

The child was further managed with Oxygen administration,IV antibiotics, Milrinone, steroids and Diuretics. To improve cardiac function, she was started on Milrinone and Lasix infusion. Suspecting viral pericarditis, IV

dexamethasone were started. Eventually, child had become clinically better and drain tubes were removed 6 days later. Extensive workup of pleural fluid was carried out which was negative for TB and bacterial etiology. However, work up for possible viral aetiology revealed a positive IgM titre for Coxsackie viral infection. She made a full recovery and was discharged after 9 days of hospitalisation.

Causes of Pericarditis:

- Viral infection – most common cause in paediatric group.
- Acute rheumatic fever
- Tuberculosis
- Bacterial infections – S.aureus, Streptococcus, Haemophilus influenza,
- Neisseria
- Post pericardiotomy syndrome (following cardiac surgery)
- Oncologic disease
- Uremia

Conclusion:

Pericarditis is a very uncommon condition in paediatric population and may often be overlooked during early stages of presentation. Early suspicion, prompt diagnosis and timely referral by the clinician plays a paramount role in preventing life threatening events.

Coxsackie Infections in Children:

These infections have different clinical spectrum and involved different age groups. Symptomatic disease is generally more common in young children.

1. Non – specific febrile illness – most common symptomatic manifestation which is difficult to clinically differentiate from condition like bacteremia, urinary tract infections and bacterial meningitis.

2. Hand – foot – and – mouth disease – one of the more distinctive rash syndromes most frequently caused by coxsackievirus group A.
3. Skin lesions occur more commonly on hands than feet, more common on dorsal surfaces, frequently occurring on palms and soles. These are tender, 3-7 mm lesions that resolve.
4. Herpangina – characterized by sudden onset of fever, sore throat, dysphagia and painful lesions in posterior pharynx.
5. Respiratory illness – sore throat and coryza are predominate manifestations.
6. Acute hemouhagic conjunctivitis.
7. Myocarditis and pericarditis.
8. Gastrointestinal illness – emesis, diarrhea and abdominal pain.