Acute pericarditis in a child during the recovery period of multisystem inflammatory syndrome

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Introduction

Coronavirus disease of 2019 (Covid-19) is considered the worst pandemic of the 21st century and led to approximately 7 million deaths worldwide[1]. Multisystem inflammatory syndrome in children (MIS-C) is a novel, life-threatening hyperinflammatory shock syndrome that is seen among children during the post-recovery phase of Covid-19[2]. Cardiac involvement forms one of the diagnostic criteria together with involvement of one or more other organ systems in making the diagnosis of MIS-C[3]. Common cardiac manifestations of MIS-C include cardiac arrhythmias, myocarditis, and coronary artery abnormalities[4]. However, the cardiac involvement in MIS-C is highly variable and the spectrum ranges from no cardiovascular involvement to sudden and severe cardiovascular compromise and shock[5].

Pericarditis during the acute phase of Covid-19 is very rare in children and there are only a few published case reports[6,7,8]. Pericarditis following recovery of Covid-19 is extremely rare and the first account of a paediatric patient with acute pericarditis following recovery of Covid-19 was published in 2021[9]. Having said this, the data regarding the frequency of pericardial disease in MIS-C is largely unknown[10]. The authors, herein, report an extremely rare presentation of acute pericarditis in a patient, as a post-recovery manifestation following initial complete recovery of MIS-C. The report supports the evidence base for the highly variable immune activation seen in severe acute respiratory syndrome coronavirus 2 (SAR-CoV-2) infection.

Case report

A 9-year-old boy presented with sudden onset retrosternal chest pain associated with a throat blocking sensation that lasted for one hour following dinner. The chest pain was tightening in character, radiated to the left upper arm and was associated with excessive sweating and nausea. There was no vomiting, palpitations or shortness of breath. The pain was not relieved by bending forwards, lying down and breathing worsened it, and it lasted for one hour before it gradually settled. There was no fever or respiratory symptoms. There was no significant past medical history including previous MIS-C or relevant family history, especially cardiac arrythmias.

On admission to hospital 2 hours after the onset of symptoms, the chest pain had settled. His blood pressure (BP) was 117/ 67 mmHg and the pulse was regular with a rate of 130 beats per minute. There were no cardiac murmurs. Abdomen was distended but soft and the other system examination was normal.

Two weeks back the child had been treated for MIS-C with intravenous immunoglobulin 2g/kg over 48 hours, intravenous methyl prednisolone 30 mg/kg for 5 days, and subcutaneous enoxaparin 1 mg/kg twice daily. He had been discharged with prednisolone tailing off regimen and aspirin 75 mg daily. 2D echocardiography, electrocardiogram (ECG) and troponin I levels were normal at that time. During the second presentation, the child was taking aspirin 75 mg daily and oral prednisolone tailing off regimen as his regular medications.

During the initial presentation, the child had high grade fever for 4 days in association with arthralgia, myalgia, abdominal pain, vomiting and diarrhoea. SRS-CoV-2 polymerase chain reaction (PCR) was negative and immunoglobulin G (IgG) was positive. Physical examination revealed bilateral non-purulent conjunctivitis, and cracked lips, and with no cervical lymphadenopathy or rashes. There was no hepatosplenomegaly. Low BP (76/44 mmHg) was noted during management in the high dependency unit (HDU). Investigations revealed white blood cell (WBC) count: 20,300/cu mm (neutrophils 90%), haemoglobin (Hb): 10.3 g/dL, platelet count: 298,000/cu mm, serum sodium: 132 mEq/L, serum potassium: 3.4 mEq/L, international normalised ratio (INR): 1.2, activated partial thromboplastin time (APTT): 26.8 seconds, D-dimer: 3436 ng/mL, erythrocyte sedimentation rate (ESR): 73/1st hour, C-reactive protein (CRP): 179 mg/dL, lactate dehydrogenase: 420 U/L and serum ferritin: 800 ng/mL.

Because of this background history, an in-ward urgent ECG was done which revealed ST elevation (concave up) in II, III, AVF, V6 leads and PR elevation in AVR (Image 1).

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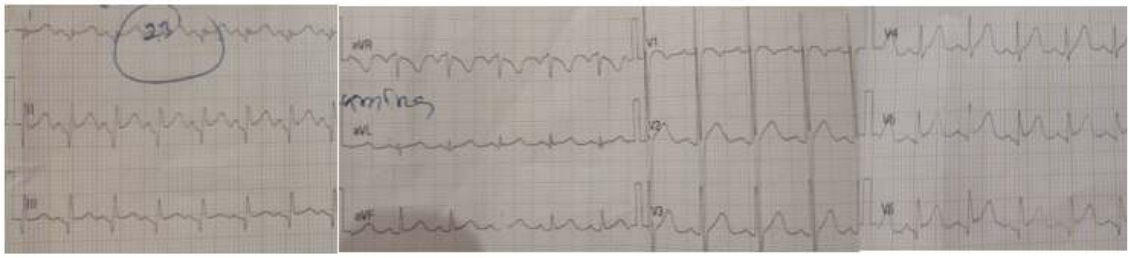

Image 1: ***Electrocardiogram findings at admission***

Whilst awaiting troponin I level and 2D echocardiography, he was commenced on subcutaneous enoxaparin. Troponin I was normal and 2D echocardiography showed evidence of acute pericarditis with an effusion. Chest x ray did not reveal a characteristic water bottle shaped cardiac shadow (Image 2).

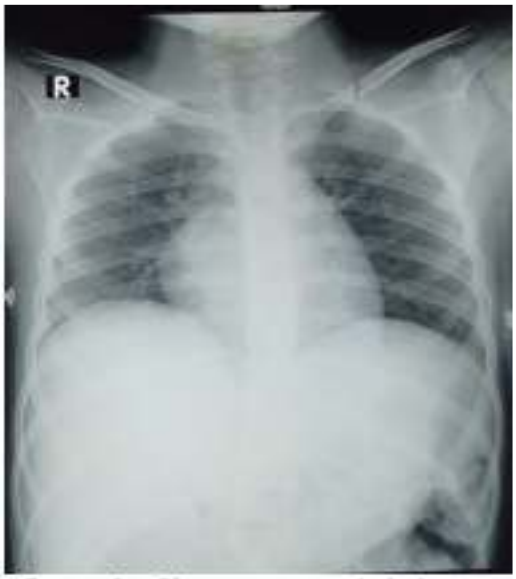

Image 2: ***Chest x-ray on admission***

Total WBC count was 21,200/cu mm (74% neutrophils). Inflammatory markers were elevated (ESR: 50 mm/1st hour, CRP: 19 mg/L). SARS-CoV-2 and Influenza A and B PCR were negative.

Diagnosis of acute pericarditis was made based on clinical history, characteristic ECG findings and echocardiography findings. Enoxaparin was subsequently discontinued. Treatment with aspirin was continued with close monitoring in the HDU. The child did not deteriorate or became symptomatic at any point except having persistent tachycardia of 120-135 bpm. ECGs were repeated 6-hourly during the first 48 hours following admission. After 48 hours, ECG changes gradually improved. The child was discharged on aspirin 75 mg for another 8 weeks duration and repeat 2D echocardiography was planned in two weeks' time. At the time of discharge, the ECG was normal (Image 3) and heart rate was within the normal range. Follow up 2D echocardiography performed in 2 weeks revealed normal findings.

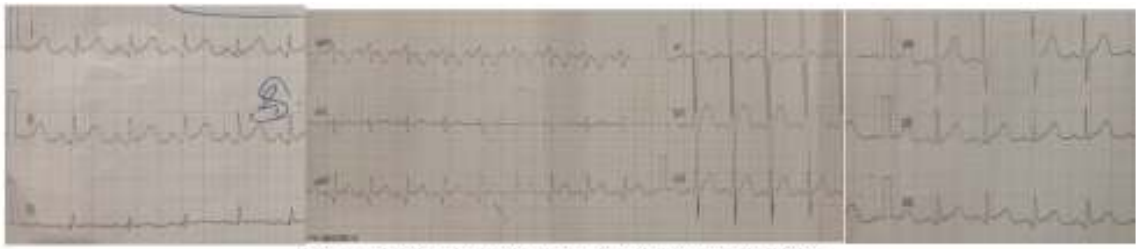

Image 3: ***Electrocardiogram findings at discharge***

Discussion

During the initial phase of the global pandemic of Covid-19, children were considered less commonly affected by SARS-COV-2[11]. However, following initial mild disease, some paediatric patients showed elevated inflammatory markers and cytokine storms characterised by multiorgan involvement and the condition was recognised as MIS-C. Management of MIS-C is based on expert consensus and include immune modulatory agents, anticoagulation and cardiac support[12].

Cardiac involvement is common in MIS-C and includes myo-pericardial inflammation, coronary artery dilatation and aneurysm and arrhythmias[13]. Pericarditis has been rarely reported during the acute phase of MIS-C[7]. This patient presented with acute pericarditis two weeks after the recovery of MIS-C and with no evidence of pericardial involvement during the acute phase of MIS-C.

SARS-CoV-2 is a cardiotropic virus and heart involvement can be highly variable based on the site of involvement and timing of the onset in relation to the

course of the disease and recovery[14]. Extensive literature survey did not reveal pericarditis during the recovery from MIS-C. However, there has been a report of pericarditis occurring in a teenager following initial complete recovery of Covid-19 infection[9]. The reported child presented with newer onset chest pain associated with echocardiographic evidence of pericardial effusion. In our child, the possible mechanisms for the causation of acute pericarditis include post-inflammatory immune-activation following either initial Covid-19 infection or recent MIS-C. A new-onset autoimmune disease possibly triggered by a recent incidental viral infection cannot be completely ruled out. However, the course of clinical events was not supportive for a recent viral infection and the only preceding clinical event was MIS-C for which the child was treated two-weeks before the current presentation.

As there is a wide spectrum of clinical presentation in MIS-C and long-term follow-up studies to assess the natural history of this disease process are still under evaluation, the authors highlight the importance of long-term follow up and monitoring of immune mediated complications including cardiac involvement in paediatric patients.

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