

MIS-C with Aneurysmal Coronary Dilation: A Case Study

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Abstract

COVID-19 related multisystem inflammatory syndrome in children (MIS-C) can present with cardiac manifestations, including coronary dilation and aneurysms. There is gray zone between Kawasaki and Kawasaki like illness with MIS-C in covid infection, with different pattern of coronary dilation [5]. The study describes 2 cases, 1st one is 3-year-old patient with giant coronary aneurysms associated with COVID-19 related MIS-C, including the treatment with intravenous immunoglobulin, corticosteroids, and aspirin. 2nd case is 13 years old girl with neglected type one truncus arteriosus presented with giant coronary aneurysms associated with COVID-19 related MIS-C and first presentation of diabetics ketoacidosis, including the treatment with intravenous immunoglobulin and insulin infusion [10]. This study emphasizes the awareness of early detection and aggressive management of MIS-C to prevent potentially life-threatening consequences.

Keywords: *Coronary Artery Aneurysms; COVID-19; MIS-C; Pediatrics; Diabetic Ketoacidosis*

Introduction

Cardiovascular disease associated with COVID-19 infection usually due to myocardial damage during initial viral infection of respiratory system and subsequent multisystemic inflammatory syndrome in children (MIS-C) which occur in less than 1% of affected cases [10].

Cardiovascular complications of severe SARS-CoV-2 range from peri-myocarditis occasional, heart rhythm disorders, with or without pulmonary hypertension. Transient coronary dilation is common while appearance of giant coronary aneurysms is less likely [10].

In congenital heart disease patient sever complications and deterioration specially in fragile and decompensated cases [7].

MIS-C occurs between 2 and 6 weeks after the acute onset of SARS-CoV-2 infection, sometimes in a previously asymptomatic patient and consists of a systemic hyperinflammatory status having symptoms similar to septic shock or Kawasaki disease [10].

It is assumed to be a delayed immune response to COVID-19, which can lead to severe cardiovascular involvement [5].

The death rate in MIS-C patients is 2%. Myocardial infarction in children is extremely rare, with most suspected cases being in fact cases of fulminant myocarditis. Myocardial infarction is secondary to acute coronary thrombosis [10].

The predisposition for the appearance of a thrombus depends on several factors: alteration of the vascular endothelium, modification of blood coagulation parameters by the appearance of thrombophilia, and hemodynamic changes of the blood vessel (stasis, turbulence).

These conditions are fulfilled point by point in MIS-C with aneurysmal coronary dilation, similar to those that appear in children with classical Kawasaki disease. Kawasaki disease (KD) is a medium-sized systemic vasculitis with predilection of coronary arteries, predominantly in children < 5 years of age [14].

Given the impact of COVID-19 infection on the incidence of new-onset diabetes, there also remain puzzles that need to be solved. In epidemiology, the characteristics of children with new-onset diabetes after infection are predominantly derived from single centers or case reports. However, there is a pressing need for evidence showing the rising incidence of diabetes and DKA across multiple centers [8].

Numerous studies have examined the incidence of type 1 diabetes (T1D) in children before and after the COVID-19 pandemic in found that in addition to directly affecting human islets, COVID-19 can also induce hyperglycemia by infecting other tissues [1].

Kawasaki Disease (KD)	Incomplete (or Atypical) KD	Pediatric Multisystemic Inflammatory Syndrome (Required all 6 Criteria)
Fever, and 4/5 criteria: - Erythema and cracked lips, strawberry tongue, and/or erythema of the pharynx and oral mucosa - Bilateral bulbar conjunctival injection - Rash maculopapular, erythematous - Erythema and edema of the hands and feet in the acute phase or periungual desquamation in the sub-acute phase - Cervical lymph nodes \geq 1.5 cm	Children with: · Prolonged fever (\geq 5 days) · 2 - 3 criteria OR - Infants with prolonged fever (\geq 7 days without other explanation) - Compatible laboratory tests (3 of the 6 criteria) o Anemia o Thrombocytosis after the 7 th day of fever o Albumin level \leq 3 g/dL o Elevated ALT level o WBC \geq 15,000/mm ³ o Urine \geq 10 WBC/hpf - Compatible echocardiographic findings (any of the following) o Z score LAD or RCA \geq 2.5 o Coronary artery aneurysm o \geq 3 features from: - Decreased LV function - Pericardial effusion - Z score LAD 2–2.5 - Mitral regurgitation	Child 0 - 19 years - Fever \geq 3 days - Clinical signs of multisystem involvement (at least 2 of the following): o Rash/bilateral non-purulent conjunctivitis/mucocutaneous inflammation signs: oral, hands, or feet o Hypotension or shock o Features of myocardial dysfunction, pericarditis, valvulitis, coronary abnormalities (echo findings or troponin/NT proBNP) o Evidence of coagulopathy (prolonged prothrombin time, partial thromboplastin time, or elevated D-dimers) o Acute gastrointestinal symptoms (diarrhea, vomiting, abdominal pain) - Elevated markers of inflammation such as C reactive protein, procalcitonin, erythrocyte sedimentation rate. - No other obvious microbial cause of inflammation, including bacterial sepsis, staphylococcal/streptococcal toxic shock syndrome - Evidence of COVID-19 (RT PCR, antigen test, serology) or likely contact with patients with COVID-19

Table 1: Comparative view of the diagnostic criteria of Kawasaki disease, incomplete or atypical Kawasaki disease, and pediatric multisystemic inflammatory syndrome, with permission from Voicu., et al. [12-14]. COVID-19: Coronavirus 2019 Disease; LAD: Left Anterior Descending; NTproBNP: N-Terminal Pro Brain Type Natriuretic Peptide; RCA: Right Coronary Artery; RT PCR: Reverse Transcriptase-Polymerase Chain Reaction; WBC: White Blood Cells.

Clinical	Laboratory Criteria for SARS-CoV-2 Infection	Epidemiologic Linkage Criteria	Vital Records Criteria
<p>An illness characterized by all of the following, in the absence of a more likely alternative diagnosis*</p> <p>Subjective or documented fever (temperature $\geq 38.0^{\circ}\text{C}$)</p> <p>Clinical severity requiring hospitalization or resulting in death</p> <p>Evidence of systemic inflammation indicated by C-reactive protein ≥ 3.0 mg/dL (30 mg/L)</p> <p>New onset manifestations in at least two of the following categories:</p> <p>Cardiac involvement indicated by:</p> <p>Left ventricular ejection fraction $< 55\%$ OR Coronary artery dilatation, aneurysm, or ectasia, OR Troponin elevated above laboratory normal range, or indicated as elevated in a clinical note</p> <p>Mucocutaneous involvement indicated by:</p> <p>Rash, OR Inflammation of the oral mucosa (e.g., mucosal erythema or swelling, drying or fissuring of the lips, strawberry tongue), OR Conjunctivitis or conjunctival injection (redness of the eyes), OR Extremity findings (e.g. erythema [redness] or edema [swelling] of the hands or feet)</p> <p>Shock**</p> <p>Gastrointestinal involvement indicated by:</p> <p>Abdominal pain, OR Vomiting, OR Diarrhea</p> <p>Hematologic involvement indicated by:</p> <p>Platelet count $< 150,000$ cells/μL, OR Absolute lymphocyte count (ALC) $< 1,000$ cells/μL</p>	<p>Detection of SARS-CoV-2 RNA in a clinical specimen*** up to 60 days prior to or during hospitalization, or in a post-mortem specimen using a diagnostic molecular amplification test (e.g. polymerase chain reaction [PCR]), OR</p> <p>Detection of SARS-CoV-2 specific antigen in a clinical specimen*** up to 60 days prior to or during hospitalization, or in a post-mortem specimen, OR</p> <p>Detection of SARS-CoV-2 specific antibodies^ in serum, plasma, or whole blood associated with current illness resulting in or during hospitalization</p>	<p>Close contact ‡ with a confirmed or probable case of COVID-19 disease in the 60 days prior to hospitalization</p>	<p>A person whose death certificate lists MIS-C or multisystem inflammatory syndrome as an underlying cause of death or a significant condition contributing to death</p>

Table 2: CSTE/CDC case definition for MIS-C [11].

Observation

Case 1

A case of 3 years old boy presented to ER with respiratory distress grade IV, Father gives history of high-grade fever for one month duration following tonsillectomy and adenoidectomy. Repeated courses of antibiotics had been given by his treating physician with no improvement. Two days ago, child was distressed and chest x-ray done and diagnosed as pneumonia, another course of antibiotic given but the case deteriorates rapidly and father brought the child to ER. Past history of snoring specially during sleep and mouth breather so his doctor advice adenoidectomy as a radical cure of the problem. child has history of neonatal admission for 25 days in NICU on assisted ventilation and long-term oxygen support patient stabilized in ER and admitted on assisted ventilation, fluid support, laboratory investigation, and routine antibiotic given, chest CT requested and covid swab as well.

Clinical examination at presentation in our hospital revealed a febrile 3-year-old male (39.7°C , axillary) with a height of 93 cm, a weight of 15 kg, relatively bad general condition, pale, dehydrated, respiratory rate of 45/min, oxygen saturation at 90% in room air, systolic murmur II/VI, protohistoric third sound, soft systolic murmur in pulmonary area II/VI with accentuated second heart sound, BP at 90/50 mmHg, HR at 150/min, diuresis present, and disturbed consciousness. Complete blood count revealed 3.330 WBC/ mm^3 , Neutrophils 34.5% ($1.700/\text{mm}^3$), Lymphocytes 57.2% ($2.820/\text{mm}^3$), Monocytes 9% ($410/\text{mm}^3$), RBC $4.560.000/\text{m cL}$, Hb 7.2 g/dL, Hct 29.6%, and

Platelets 530.000/mm³. Serum electrolytes were mild hypokalemia and hypernatremia (Na⁺ 160 mmol/L, K⁺ 3.36 mmol/L, Ca²⁺ 1.26 mmol/L), Troponin T > 3.000 ng/mL (NV < 14), TGP: 40 IU/L, urea: 15 mg/dL, and creatinine: 0.40 mg/dL, CRP 96 IU/ml, serum ferritin 1000 µg/dl, D dimmer 6.00 and -ve Covid swab.

Electrocardiogram (ECG) showed sinus tachycardia rhythm, HR 150/mi. Chest Xray shows pulmonary consolidation minimal pleural effusion, mild cardiomegaly with pulmonary congestion. Chest CT supporting the x ray result. Echo study requested due to oxygen lability and unstable condition show depressed myocardial function FS is 27% EF 54% moderate mitral regurge, tricuspid regurge, pulmonary regurge mild aortic regurge. decrease TAPSI 1.2 tricuspid regurge is 18 mmHg, congested IVC, mild to moderate pulmonary hypertension, right side dilation, So the case diagnosed as most probably COVID-19 related multisystem inflammatory syndrome in children (MIS-C) in spite of covered -ve swab, (as all swabs are negative this season). But chest CT, clinical and laboratory finding supporting the diagnosis. Pulse steroid, anti-failure in the form of dobutamine 10 microgram/kg/minute and frusemide 4 mg/kg/day proper sedation and oxygenation with minimal handling. After 48-hour the child dramatically improved and transferred to intermediate unit for 24 hours, anti-failure measures changed to oral form and oral feeding initiated, but sudden deterioration of the case, chest x-ray shows white lung lesion suspected as aspiration. Patient ventilated again and 2nd echo requested. Echo study shows regression of valvular regurge, no more pulmonary hypertension, collapsed IVC with depressed myocardial function, minimal pericardial effusion, hugely dilated left main coronary with saccular giant aneurysm (z-score + 26) and a large (z-score +5.5) right coronary artery aneurysm. During this time there is leukocytosis 22.000 WBC/mm³, thrombocytopenia 75.000/mm³ with normal level of PT 12 seconds, PTT 35 seconds and INR 1.1. Updating the diagnosis as in MIS-C with aneurysmal coronary dilation. It was day 4 of pulse steroid, gamma globulin requested, and enoxaparin added to the previous management, aspirin postponed as there is GIT bleeding.



Figure 1: 2D-echocardiography long axis parasternal m- mode shows functional depression of left ventricle.

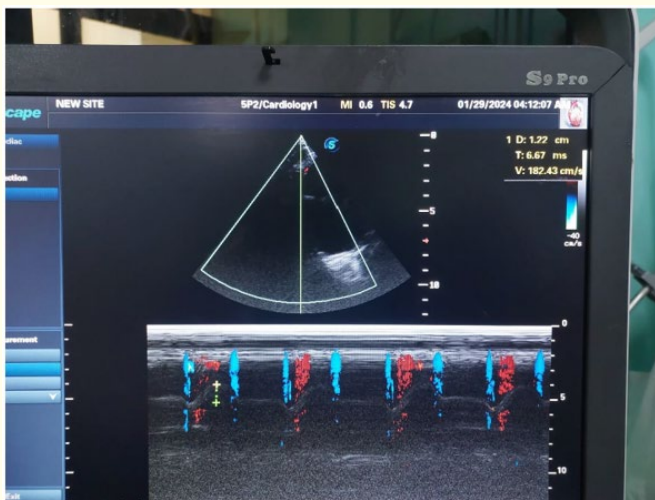


Figure 2: 2D-echocardiography 4 chamber view, M-MODE, shows decrease TAPSI.

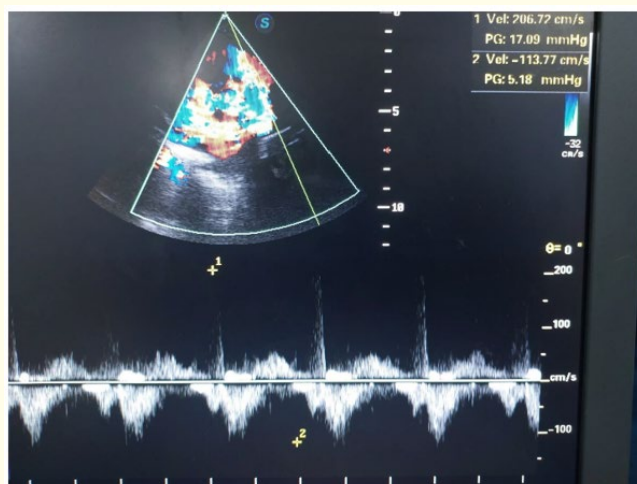


Figure 3: 2D-echocardiography short axis parasternal CW doppler shows pulmonary regurgitation.



Figure 4: 2D-echocardiography apical 4 chamber view shows dilated hyperilluminated both right and left coronaries.



Figure 5: 2D-echocardiography short axis parasternal view shows aneurysmal dilation of left main coronary.



Figure 6: 2D-echocardiography short axis parasternal view shows aneurysmal dilation of left main coronary and saccular dilation of rt coronary.



Video 1: Long axis parasternal mitral regurgite.



Video 2: Short axis parasternal view aneurysmal dilation of the left main coronary.

Case 2

A case of 13 years old girl presented to ER in bad general condition cachectic with Respiratory distress grade IV, dehydrated, severe hyperglycemia with acidotic breath, acetone 2 plus in urine. Father gives history of low-grade fever for 3 weeks duration in a known cardiac patient. Repeated courses of antibiotics had been given by her treating physician with no improvement. 4 days ago, child was distressed, persistent vomiting with abdominal pain and chest x-ray done and diagnosed as pneumonia and heart failure, another course of antibiotic and intensification of her ant failure pills but the case deteriorates rapidly and her random blood sugar is shooting with positive acetone in urine so referred to our hospital. Past history of uncorrected cyanotic congenital heart disease diagnosed at 6-month patient on regular Lasix and ACI family refusing open heart surgery. child has history of recent loss of weight, frequent micturition, polyphagia, polydipsia, vomiting patient stabilized in ER and admitted on assisted ventilation, fluid support, laboratory investigation, and routine antibiotic given, chest CT requested and covid swab as well.

Clinical examination at presentation in our hospital revealed a febrile 13-year-old male (37.8°C, axillary) with a height of 154 cm, a weight of 27 kg, relatively bad general condition, cachectic, cyanotic, pale, dehydrated, respiratory rate of 30/min, oxygen saturation at 80% in room air, cardiomegaly apex shifted to 7th space outside midclavicular line, accentuated second heart sound, pan systolic murmur V/VI, gallop rhythm, harsh systolic murmur in aortic area IV/VI with, BP at 90/40 mmHg, HR at 150/min, diuresis present, and disturbed consciousness. ABG PH 7.1 MMOL, HCO₃ 6.3 MMO/L bicarbonate PCO₂ 22, lactate 4.3 MMOL complete blood count revealed 3.700 WBC/mm³, neutrophils 43% (1.700/mm³), lymphocytes 49% (2.820/mm³), monocytes 5% (410/mm³), RBC 5.000.000/m cL, Hb 9.2 g/dL, Hct 29.6%, and platelets 74.000/mm³. Serum electrolytes were hypokalemia (Na⁺ 142 mmol/L, K⁺ 2.36 mmol/L, Ca²⁺ 1.26 mmol/L), troponin T > 2.000 ng/mL (NV < 14), TGP: 48 IU/L, SGOT 2110 U/I, SGPT 1625 U/I, albumin 1.8/gdl, urea 99 mg/dL, and creatinine: 1.6 mg/dL, CRP 96 IU/ml, serum ferritin 780 µg/dl, D dimmer 4.00.RBS is 590 mg/dL, acetone in urine is 2 plus, HBA1C 8.5 and -ve Covid swab.

Electrocardiogram (ECG) showed sinus tachycardia rhythm, HR 110/min. Chest Xray shows pulmonary consolidation mod-pleural effusion, severe cardiomegaly with pulmonary congestion. Chest CT supporting the x ray result. Echo study requested to assess the cardiac anomaly FS is 28% EF 50%, severe mitral regurge, tricuspid regurge, severe aortic regurge. decrease TAPSI 1.00, tricuspid regurge is 25 mmHg, collapsed IVC, huge biventricular dilation, type one truncus arteriosus with aneurysmal coronary dilation. So, the case diagnosed as most probably COVID-19 related multisystem inflammatory syndrome in children (MIS-C) in spite of covid -ve swab, (as all swabs

are negative this season). But chest CT, clinical and laboratory finding supporting the diagnosis. Pulse steroid, anti-failure in the form of dobutamine 10 microgram/kg/minute and frusemide 6 mg/kg/day proper sedation and oxygenation, regular insulin infusion with minimal handling. the case diagnosed as in MIS-C with aneurysmal coronary dilation and diabetic ketoacidosis in a neglected case of type 1 truncus arteriosus, gamma globulin requested. After 36-hour the child dramatically improved and transferred to intermediate unit for 48 hours, anti-failure measures changed to oral form and oral feeding initiated, subcutaneous insulin when electrolyte, ABG, random blood sugar and vomiting controlled but sudden deterioration of the case, with sever hypoglycemia. PT was 92.3 sec, control is 12.4, conc 9 percent INR is 8.23. Patient ventilated again and 2nd echo requested. Echo study congested IVC with depressed myocardial function, mod pericardial effusion, aneurysmal dilatation of left main coronary (z-score + 15) and a large (z-score +25) hugely dilated right coronary artery with saccular giant aneurysm y aneurysm. During this time there is leukocytosis 33.000 WBC/mm³, thrombocytopenia 22.000/mm³. A case of MIS-C in post covid 19 patients with neglected congenital cyanotic heart disease (truncus arteriosus) and newly diagnosed diabetes mellitus. Unfortunately, the case died after 12 hours from sever cardiogenic shock.



Video 3: Apical 5 chamber view shows aneurysmal dilation of left coronary artery.



Video 4: Short axis parasternal chamber view shows aneurysmal dilation of right coronary artery.

Discussions

Shock, pericardial effusion, cardiac arrhythmias and coronary artery dilatation have been reported as the most common cardiovascular complication of post covid MIS-C. The majority of MIS-C associated coronary artery abnormalities take different forms ranging from mild coronary dilation which pass un-noticed with complete regression to giant coronary dilation with myocardium infarction and long-term complication [10].

Villacis-Nunez., *et al.* reported 3 cases of giant coronary aneurysms secondary to MIS-C. MIS-C has been associated with cardiac complications in up to 87% of cases, manifesting as various degrees of myocardial dysfunction, pericardial effusion, arrhythmias, valvopathies, and/or coronary artery involvement. Coronary artery aneurysms and dilation, usually mild or moderately sized, occur in 6% to 24% of cases [12].

Hejazi., *et al.* described at least one patient whose medium coronary artery aneurysm persisted at 6 months of follow up. While MIS-C is widely accepted as a post-infectious inflammatory disorder, the exact mechanism causing aneurysms in the setting of MIS-C is unclear [4].

Grech., *et al.* published a case of atypical Kawasaki in a 9-month-old child with giant coronary artery aneurysms similar to our case in pre- COVID era [3].

Some have hypothesized that similar to Kawasaki disease (KD), inflammatory cytokines in circulation disrupt the arterial wall. As coronary abnormalities seen in MIS-C are generally relatively mild and resolve rapidly [6].

In a case study Mustafa., *et al.* found complete resolution of coronary artery aneurysms after 6 weeks and he hypothesized that the aneurysm formation in multisystem inflammatory syndrome in children is related to fever and circulating inflammatory mediators rather than disruption of the arterial wall [7].

Some have also speculated that coronary enlargement is a result of proinflammatory vasodilation instead [5].

Based on the shared clinical features with KD, current treatment for MIS-C includes anticoagulant, aspirin and IVIG with the addition of glucocorticoids for moderate to severe illness or inadequate response to IVIG. Infliximab, a tumor necrosis factor antagonist, recombinant Il-1 receptor antagonist, has also been associated with improvement of coronary artery aneurysms in MIS-C patients with severe illness or who failed to respond to IVIG [2].

Our first patient initially presented with symptoms that aligned more closely with acute COVID-19 infection, but by the time of hospital admission he met WHO and CDC criteria for MIS-C as well as criteria for incomplete KD. by his second echocardiography study he developed giant coronary artery aneurysms. IVIG is thought to prevent coronary artery aneurysm formation and progression in KD. Coronary artery aneurysm progress till one week after IVIG seen in this patient then regress. Still there is gray zone between Kawasaki and Kawasaki like illness with MIS-C in covid infection, so more studies are needed to study the pattern of coronary aneurysmal formation and the associated risk factor complicating the outcome [13].

In second case the patient was neglected cardiac case with fragile cardiac condition complicated with diabetes mellitus which aggravate the condition. Compared to the incidence of diabetes before the pandemic, there was a significant increase after the COVID-19 outbreak. Notably, DKA, a severe condition associated with diabetes, has become more frequent during the pandemic One of the most extensive international multicenter studies conducted to date was led by Niels H. Birkebaek. Drawing upon data from 13 national diabetes registries, the research revealed a significantly elevated observed prevalence of DKA at the time of T1D diagnosis compared to the predicted prevalence based on the previous decade's data [1]. in our case as a neglected cardiac case diabetic ketoacidosis complicate the

case and we don't know which of them suppress the child immunity diabetes or covid 19 infection. Another multicenter regional study conducted in the UK revealed a higher proportion of children presenting with DKA in the COVID-19 PCR-positive groups [8].

Conclusion

This patient's case highlights the difficult task facing clinicians in treating MIS-C patients who may present with uncountable mixtures of symptoms, clinical findings, and disease courses. Therefore, further researches which identifies patient characteristics or biomarkers associated with coronary artery aneurysm formation will be highly useful. Physicians would also benefit from further research on which MIS-C patients are at greatest risk of coronary artery aneurysms refractory to standard treatment as well as potential alternative treatment. During the COVID-19 pandemic, there has been an increase in the incidence of new-onset diabetes, particularly with a higher prevalence of Diabetic Ketoacidosis (DKA) and severe DKA. Both *in vivo* and *in vitro* basic research provide multiple mechanisms to elucidate the risk of diabetes following a COVID-19 infection. Considering COVID-19 as a type of virus, the pandemic has imparted valuable experiences and lessons. By applying these insights, we can significantly improve our prevention and treatment strategies for virus-induced new-onset diabetes.

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