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**Case Report**

# Pericardial Effusion as A Presentation of Multisystem Inflammatory Syndrome – Adult (MIS –A)

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**Abstract:** COVID-19 associated Multisystem inflammatory syndrome in adults (MIS-A) can present with varied cardiac manifestations like ST segment changes, wall motion abnormalities, arrythmias and valvular abnormalities. Although the CDC has released criteria for diagnosis in July 2020, there is still a lack of clarity due to the varied presentation of cases and overlap of symptoms with other common disease conditions. A delay in diagnosis due to time taken to rule out other conditions can delay appropriate treatment. We present a case of an adult male who presented with fever, chest discomfort and abdominal symptoms with a history of previous COVID-19 infection. His vitals were stable without any signs of cardiac failure. We tested for COVID-19 antigen, antibody, and inflammatory markers. ECG and CT-chest and abdomen showed ST-T changes and a significant pericardial effusion. Though there were no cardiac symptoms, he was monitored with serial Troponin T and transthoracic ECHO cardiography which helped us to pick up deterioration of cardiac function. An immediate coronary angiogram was done which was normal. He was treated with corticosteroids, diuretics and colchicine and with daily monitoring his cardiac function improved. The pathophysiology of MIS-A is currently not well understood and a pericardial effusion without any significant cardiac symptoms as a presentation of MIS-A has not been reported previously. A multi-disciplinary approach and a high index of suspicion was required along with vigilant monitoring in an intensive care setup to recognise and treat this patient who had a good recovery with supportive care.

**Keywords:** MIS-A, COVID-19, pericardial effusion, myocarditis, polyserositis, corticosteroids, colchicine.



## INTRODUCTION

Multisystem inflammatory syndrome in Adults (MIS-A) is an uncommon but serious complication of SARS-CoV-2 infection. It was first described in children and adolescents [1]. Since June 2020 there has also been published cases of MIS in adults. The Centre for Disease Control and Prevention (CDC) released a case series of 27 patients from the USA and UK of a similar syndrome seen in adults. (2) These patients had varied presentation but diagnostic criteria used by the CDC was:

1) A severe illness requiring hospitalization in a person aged ≥21 years
2) A positive test result for current or previous SARS-CoV-2 infection (nucleic acid, antigen, or antibody) during admission or in the previous 12 weeks
3) Severe dysfunction of one or more extrapulmonary organ systems (e.g., hypotension or shock, cardiac dysfunction, arterial or venous thrombosis or thromboembolism, or acute liver injury)
4) Laboratory evidence of severe inflammation (e.g., elevated CRP, ferritin, D-dimer, or interleukin-6)
5) Absence of severe respiratory illness

We present an interesting case of MIS-An associated with Covid-19 infection which should be suspected in certain cases where the CDC criteria is met, so that it can be diagnosed and appropriately managed.

## CASE REPORT

A 36-year-old male, with Body Mass Index (BMI) of 25 with no comorbidities presented to the

hospital with a three-day history of abdominal pain, chest discomfort, diarrhoea and an episode of fever. He was tested positive for COVID-19 three weeks' prior and was on home quarantine. Patient did not give any history of respiratory symptoms during that period. On examination he was conscious, oriented, heart rate was 130/ min with a blood pressure of 130/80 mm of Hg. His respiratory rate was 16 / minute and saturation was 99% on room air. He had warm peripheries, no pedal oedema and no fever on presentation. COVID 19 RT PCR test was done which was reported positive. ECG showed T wave inversions in lead II, III and AVF. Trans thoracic ECHO revealed a moderate pericardial effusion with normal left ventricular function, with no signs of cardiac tamponade. Chest X-ray showed cardiomegaly with clear lung fields. His blood reports showed neutrophilia, lymphopenia, significantly elevated cardiac enzymes (troponin t, CK-MB and NT proBNP). His Inflammatory markers, CRP and D dimer were elevated whereas ferritin was within normal limits. As he had abdominal symptoms amylase and lipase were tested which were normal. He was shifted to COVID intensive care unit for further care. Since he had ECG changes with elevated cardiac enzymes and no regional wall motion abnormality, he was treated medically as myocarditis. An ultrasound abdomen showed mild ascites, gall bladder wall edema, minimal pleural effusion and fatty liver. Liver function test showed a mild transaminitis. A contrast CT thorax and abdomen was done which revealed a mild to moderate pleural effusion, moderate pericardial effusion and mild sub pleural atelectic changes with no evidence of pulmonary embolism. His Troponin T continued to rise on serial monitoring. Transthoracic ECHO was repeated after 24 hours which showed new onset regional wall motion abnormality with hypokinesia of basal inter ventricular septum and mid to basal inferior segments, Left Ventricular ejection fraction of 50% along with moderate pericardial effusion. With this finding, a coronary angiography was done on the same day which showed a normal study. Antiplatelets were discontinued with a diagnosis of Multisystem inflammatory syndrome in adults as per guidelines by CDC for MIS-A. Cardiac failure was treated with diuretics and beta blocker. He was started on steroids (methyl prednisolone 1mg/kg) along with colchicine 0.5mg bid. All supportive care and monitoring were continued. His clinical condition gradually improved over next 5 days with declining cardiac Troponin levels and NT proBNP. Repeat transthoracic ECHO was done which showed reduction in pericardial effusion with improved LV function. A COVID-19 antibody test was done which showed high titres of total antibody and IgG levels. Dengue virus NS-1, IgG and IgM antibodies, ANA, ANCA were done to rule out possible causes of polyserositis. He was discharged home after a week with RT- PCR negative for SARS-CoV-2.

**Table-1: Investigations**

| Test | Reference range | 6.1.21 | 7.1.21 | 8.1.21 | 9.1.21 | 10.1.21 | 11.1.21 |
|---|---|---|---|---|---|---|---|
| WBC count (thousand/µL) | 4.0-10.0 | 16.03 | 17.6 | 21.5 | 16.59 | 13.1 | 10.8 |
| Neutrophil count (thousand/µL) | 2.0-7.0 | 12.66 | 13.81 | 17.52 | 13.44 | 10.54 | 8.97 |
| Lymphocyte count (thousand/µL) | 1.0-3.0 | 2.03 | 2.27 | 2.04 | 1.69 | 1.43 | 1.21 |
| Haemoglobin (g/dl) | 13-17 | 15.1 | 13.9 | 13.6 | 13.4 | 13.2 | 13.2 |
| Platelet count (thousand/L) | 150-450 | 304 | 319 | 325 | 300 | 289 | 292 |
| Potassium (mmol/L) | 3.5-5.1 | 3.94 | 3.98 | 4.04 | 4.09 | 4.46 | 4.94 |
| Sodium (mmol/L) | 136-145 | 129 | 129 | 130 | 132 | 136 | 137 |
| Creatinine (mg/dl) | 0.7-1.2 | 0.97 | 1.05 | 1.04 | 1.04 | 0.94 | 0.81 |
| BUN (mg/dl) | Jun-20 | | 12 | 12 | 13 | | |
| C reactive protein (md/L) | 0-6 | 55.4 | | | | 21.8 | |
| D-dimer | <0.5 | 2.77 | | 3.22 | | 4.64 | |
| Ferritin (ng/ml) | 30-400 | 353 | | | | 335.9 | |
| Troponin T (pg/ml) | <14 | 460 | | 1114 | | 892 | 269 |
| | | 663 | | | | | |
| CK MB (ng/ml) | <3.61 | 8.24 | | | | | |
| NT-proBNP (pg/ml) | <125 | 10255 | | 13439 | | 13131 | 7168 |
| SGPT (U/L) | <41 | 77 | 96 | 102 | | 118 | 88 |
| SGOT (U/L) | <40 | 59 | 77 | 62 | | 40 | 29 |
| Triglycerides (mg/dl) | <150 | 230 | | | | 130 | |
| RT PCR | | Positive | | | | Positive | |
| ANTI-SARS-COV-2 TOTAL ANTIBODY | <1 | 94.32 | | | | | |
| SARS-COV-2 IGG ANTIBODY | <1.4 | 7.39 | | | | | |
| Transthoracic ECHO | | | | Hypokinesia of IVS and Inf segment LVEF: 45% | | Mild hypokinesia of IVS and Inf segment LVEF: 50% | Mild hypokinesia of IVS and Inf segment LVEF: 50% |
| Pericardial Effusion | | | | PE: 14mm | | PE:11mm | PE:11mm |

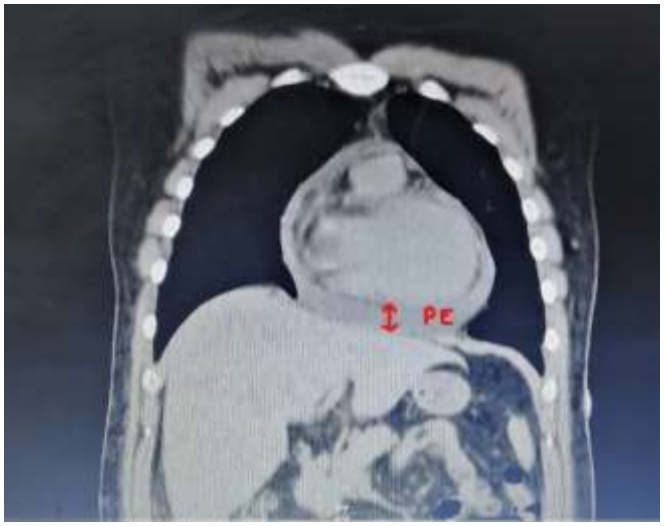


**Fig-1: CT scan showing pericardial effusion**

## DISCUSSION

Previously published data indicate that adult patients of all ages with current or previous SARS-CoV-2 infection can develop this multisystem inflammatory syndrome (MIS-A). The characteristic difference between hospitalized severe COVID 19 and this is the lack of severe hypoxia and respiratory symptoms [2].

Our patient was a healthy young male with no previous known risk factors for myocardial dysfunction. His clinical presentation was an episode of fever with predominantly abdominal symptoms. Due to history of recent COVID-19 infection and high community prevalence he was tested for COVID-19 antigen again. Without significant hypoxia or respiratory symptoms at presentation, this could have been missed unless we have a high index of suspicion.

His transthoracic ECHO at presentation did not show any regional wall motion abnormalities despite an increase in troponin levels and ECG changes. We continued monitoring of troponin levels on day 3 as well as did a repeat transthoracic ECHO. This helped us to pick up a new onset regional wall motion abnormality despite no change in symptoms. We did a coronary angiogram which showed normal study. This helped us to diagnose Myocarditis secondary to a multi-inflammatory syndrome secondary to COVID 19 infection.

The pathophysiology of MIS-A is currently not well understood. One of the theories proposed is persistent infection outside the respiratory tract. Other theories include endothelial damage, thromboinflammation and a dysregulated immune response [3]. An acute cardiogenic shock and myocarditis like picture has been described in adults following a COVID 19 infection. Zachary Most *et al*. has described a group of patients with positive tests for COVID 19 at presentation and features of fever, elevated enzymes, biventricular dysfunction, elevated troponins and ST-T wave changes. In the few cases where cardiac biopsy was done it showed a modest inflammatory lymphocytic myositis [4].

Due to the polyserositis and myocarditis present in our patient with a persistent pericardial effusion, we started him on IV methylprednisolone (1mg/kg/day). The treatment described for MIS has been inconsistent. In children diagnosed with MIS treatment that has been described includes IV corticosteroids, IV immunoglobulins, IL-6 inhibitors. (5) Similar treatment has been tried in adults. For persistent pericardial effusion we started him on colchicine. Colchicine has been shown to be effective in pericardial diseases by inhibiting WBC motility, degranulation and phagocytosis. It also inhibits IL-1 beta, and IL- 18 both of which have been recognized as playing a role in acute coronary syndrome and pericarditis [6].

His troponin, NT-Pro BNP and ventricular function improved over the course of the week. His abdominal symptoms settled and he was discharged after a negative RT PCR report.

MIS A has been shown to have a good recovery rate and low mortality when compared to MIS C. However, it requires a high index of suspicion, a multi-disciplinary team care with monitoring in an acute intensive care setup. As COVID-19 infections continue to persist in communities, it is important to continue to suspect MIS A in adults presenting with symptoms fitting the CDC criteria.

## CONCLUSION

Clinicians should be aware of MIS-A association with Covid-19. MIS-A may lead to life-threatening complications and death. This can be avoided by having a high index of suspicion and then

maintaining the appropriate approaches for managing MIS-A.

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