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Exploring Multisystem Inflammatory Syndrome in a Pediatric Case Report

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ABSTRACT

Multisystem inflammatory syndrome in children (MIS-C) is a rare post-infectious hyperinflammatory disorder that follows a recent SARS-CoV-2 infection, characterized by systemic inflammation, fever, hypotension, and cardiac dysfunction. Patients with this disorder might also present with overlapping symptoms with Kawasaki disease (KD), toxic shock syndrome (TSS), and macrophage activation syndrome (MAS). The incidence of MIS-C is uncertain, it seems to be a rare pediatric complication of SARS-COV-2 infection. The Centers for Disease Control and Prevention (CDC) estimates that in 2023 the overall MIS-C incidence was 0.11 cases per million person-months. Traditional methods of SARS-COV-2 surveillance do not capture all COVID-19 cases because some are asymptomatic, undiagnosed, or not reported.

In our case, we want to describe the story of a 6-year-old male patient who suffered from the condition after unrecognized acute SARS-COV-2 infection.

Keywords: COVID-19; Kawasaki Disease; MIS-C; SARS-COV-2; Systemic Inflammation.

INTRODUCTION

MIS-C represents a rare yet severe complication linked to SARS-CoV-2, the causative agent of COVID-19. Predominantly affecting individuals under 21 years of age, particularly children and adolescents, it is distinguished by systemic inflammation involving multiple organ systems.[1] Typically, MIS-C symptoms emerge approximately two to six weeks following acute infection.[2] In our case, the presentation exhibited overlapping features with Kawasaki Disease.

CASE

A 6-year-old male was admitted to our hospital on the third day of illness presenting with a pruritic maculopapular eruption on his lower extremities, which subsequently spread to the trunk, upper extremities, and face, resulting in a confluent rash. The following day, he developed a high-grade fever (38.5-39°C), enlarged occipital lymph nodes, neck muscle pain, conjunctivitis, malaise, and periungual desquamation. His mother reported a history of multiple upper respiratory tract infections in the preceding months.

On admission, the patient appeared adynamic, unable to tolerate oral intake, with swollen eyelids, inflamed conjunctiva, and a purplish confluent, pruritic maculopapular rash with periungual desquamation, along with decreased skin elasticity. Physical examination revealed cervical, submandibular, and occipital non-tender lymphadenopathy, mucositis characterized by tongue and oral cavity mucosal redness, diminished heart sounds with a systolic murmur on both the apex and the base, decreased urine output, and enlarged liver and spleen (2-2.5cm and 2cm, respectively). The patient exhibited no bowel movement in the previous 24 hours, and while neck muscle pain was present, there were no signs of neck rigidity or other meningeal signs.

Laboratory investigations revealed significantly elevated inflammatory markers: CRP 171.0 mg/L, leukocytosis (14.2x10^9/L) with left shift to metamyelocytes (9%), bands (25%), lymphopenia (0.6x10^9/L), ESR 45mm/h, PCT 10 ng/ml, mild thrombocytopenia (151x10^9/L), elevated LDH (558 U/L), ferritin (311.7 mg/L), and hyponatremia (129.6 mmol/L). Coagulation parameters indicated coagulopathy: PT 20.3 sec, INR 1.26, and elevated D-dimer (4.59 mcg/ml). A rapid test for SARS-CoV-2, influenza A and B, and adenoviruses was negative. Ultrasound confirmed bilateral postauricular lymphadenopathy with decreased echogenicity, and cardiac ultrasound revealed pericardial effusion with 3-4mm separation.

Blood cultures were obtained, and the patient received intravenous broad-spectrum antibiotics and supportive treatment due to suspected bacterial infection. Despite treatment, the fever persisted, and pleural and peritoneal effusions developed. Renal function tests were normal, but hypoalbuminemia was noted (albumin 23.6 g/L). Given the clinical presentation and markedly elevated inflammatory markers, the SARS-CoV-2 antibody titer was measured (1:766 AU/ml), confirming the diagnosis of MIS-C. Treatment with IVIG 2g/kg, high-dose aspirin (50 mg/kg), and high-dose prednisolone (2mg/kg) was initiated. Blood cultures were negative. Following IVIG administration, the patient's condition improved, with resolution of fever and other

symptoms, and normalization of inflammatory markers. Subsequently, the patient was transitioned to low-dose aspirin (5mg/kg) and prednisolone tapering. A comprehensive follow-up plan was established, and the patient was discharged from the clinic.

DISCUSSION

In our study, the pediatric patient, who had not received vaccination against SARS-CoV-2 and had no prior exposure to the virus, exhibited symptoms resembling Kawasaki disease. Given the patient's age of 6 years, which falls outside the typical age range for Kawasaki disease, and considering the prevalence of SARS-CoV-2 infection, we conducted serological testing, which returned positive results. Based on these findings, the case fulfilled both the Centers for Disease Control and Prevention (CDC) and World Health Organization (WHO) criteria for Multisystem Inflammatory Syndrome in Children (MIS-C).

According to the CDC, MIS-C is diagnosed in individuals under 21 years old in the absence of a more likely alternative diagnosis, presenting with subjective or documented fever (>38.0°C), requiring hospitalization or resulting in death due to clinical severity, with C-reactive protein (CRP) levels exceeding 3.0 mg/dL, and new onset manifestations of more than two of the following categories: cardiac (e.g., coronary artery dilatation/aneurysm, decreased left ventricular ejection fraction (<55%), elevated troponin), shock, mucocutaneous (e.g., rash, oral mucosal inflammation, conjunctivitis), gastrointestinal (e.g., abdominal pain, vomiting, diarrhea), and hematologic changings like thrombocytopenia - PLT<150,000/µL, lymphopenia, ALC < 1000/µL, and any evidence of SARS-COV-2 exposure like: detection of SARS-CoV-2 RNA or antigen up to two months prior to or during hospitalization or in an autopsy specimen, OR serologic evidence associated with the current illness, OR close contact with a confirmed/probable COVID-19 case in the two months before hospitalization.[3]

Alternatively, according to the WHO criteria: “MIS-C is diagnosed in children and adolescents aged 0–19 years with a fever lasting more than 3 days and two or more of the following: symptoms of mucocutaneous inflammation (oral, hands or feet), rash, or bilateral non-purulent conjunctivitis, hypotension or shock, features of myocardial dysfunction, pericarditis, valvulitis, or coronary abnormalities (including echocardiographic findings or elevated Troponin/NT-pro BNP), evidence of coagulopathy (by PT, PTT, elevated d-Dimers), acute gastrointestinal problems (diarrhea, vomiting, or abdominal pain), elevated markers of inflammation such as ESR, C-reactive protein, or procalcitonin, and absence of other obvious microbial causes of inflammation.” [4]

Despite potential diagnostic challenges due to similarities with other conditions, adherence to these comprehensive criteria facilitates accurate diagnosis and enables timely initiation of appropriate treatment for affected children.

CONCLUSION

Despite the typically milder course of acute SARS-CoV-2 infection in children compared to adults, it's crucial to maintain vigilance for the virus's post-infectious complications. Decreased testing for SARS-CoV-2 in pediatric patients with minor upper respiratory infection-like symptoms who do not require hospitalization may inadvertently lead to missed diagnoses. Therefore, healthcare providers should remain cognizant of the potential for infection and its subsequent complications to promptly identify cases and administer appropriate treatment

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