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Case Report

Rash, Fever, and a Diagnostic Puzzle: Recognizing MIS-C Among Mimicking Illnesses in a Child After COVID-19 Exposure — A Case Report

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ABSTRACT

Multisystem inflammatory syndrome in children (MIS-C) is a delayed hyperinflammatory condition following SARS-CoV-2 infection that can mimic Kawasaki disease, sepsis, and toxic shock. A previously healthy 9-year-old boy presented on illness day 5 with fever (102.9°F), non-pruritic maculopapular rash, bilateral ankle pain with refusal to walk, and exertional dyspnea. Initial findings included room-air oxygen saturation of 87% with tachypnea (respiratory rate in the 40s), requiring supplemental oxygen via oxymask (2–3 L/min); ICU-level care was not required. Laboratory evaluation revealed markedly elevated C-reactive protein (25.16 mg/dL; reference ≤0.90), erythrocyte sedimentation rate (50 mm/hr; reference 0–15), transaminitis, direct hyperbilirubinemia, positive rapid streptococcal antigen, and anti-streptolysin O titer 1,650 IU/mL (reference <200). Chest radiography showed hypoinflated lungs with prominent vasculature but no consolidation. Echocardiography revealed elevated right ventricular systolic pressure (approximately half to two-thirds systemic) with preserved biventricular function and no coronary artery abnormalities. Despite azithromycin, ceftriaxone, and piperacillin-tazobactam, inflammatory markers and hepatic function worsened. On admission day 4, rheumatology elicited a history of COVID-19 exposure approximately 2 weeks earlier. Additional testing showed elevated D-dimer (6,048 ng/mL FEU; reference 215–499), ferritin (471.8 ng/mL; reference 16–300), lactate dehydrogenase (480 U/L; reference 110–260), and B-type natriuretic peptide (156 pg/mL). Intravenous methylprednisolone 1 mg/kg every 12 hours led to rapid defervescence, respiratory improvement, and laboratory normalization. Anti-nucleocapsid SARS-CoV-2 antibodies later returned positive, consistent with prior natural infection. This case highlights how concurrent streptococcal carriage and hepatic dysfunction may obscure MIS-C and underscores the importance of exposure history, inflammatory testing, and early corticosteroid therapy.

1. Introduction

Multisystem inflammatory syndrome in children (MIS-C) is an uncommon but potentially life-threatening complication of coronavirus disease 2019 that typically occurs several weeks after acute infection or exposure [1]. Affected children often present with prominent cardiovascular, gastrointestinal, and mucocutaneous manifestations that may resemble Kawasaki disease, septic shock, or toxic shock syndrome, making early recognition challenging [1, 2].

Because MIS-C presentations overlap with common pediatric infections and inflammatory conditions, diagnosis requires careful clinical assessment, targeted laboratory evaluation, and a high index of suspicion in the appropriate epidemiologic context [1, 3]. This report describes a child with fever, rash, and multiorgan involvement whose course initially suggested bacterial and viral etiologies but ultimately fulfilled established criteria for MIS-C after COVID-19 exposure was identified and serologic testing was obtained.

2. Case Presentation

A nine-year-old male with no significant past medical history presented with fever, maculopapular rash, bilateral ankle pain, and refusal to walk. One week prior to presentation, he developed a dry cough, chest pain, headache, back pain, and fever. He was evaluated at an urgent care clinic, where influenza and acute COVID-19 tests were negative, and was prescribed amoxicillin for presumed bacterial infection.

Despite antibiotic therapy, fever persisted, and he developed a non-pruritic rash involving both lower and upper extremities with vomiting, exertional dyspnea, and progressive difficulty walking, prompting emergency department evaluation. There was no history of trauma or recent travel other than a trip to Mexico three months earlier, and he had not received a COVID-19 vaccination.

On admission, he was febrile to 102°F, tachycardic, tachypneic (respiratory rate 40s), and hypoxemic (87% on room air), which improved to 95% on supplemental oxygen (2-3 liters/minute). ICU-level care was not required.

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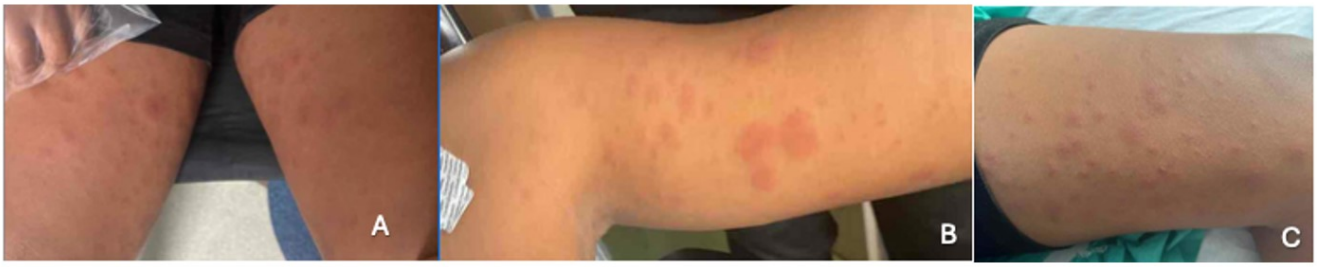

Figure 1: Maculopapular rash in multisystem inflammatory syndrome in children (MIS-C). (A) Erythematous maculopapular lesions over the anterior thighs. (B) Similar lesions over the upper extremity. (C) Involvement of the lateral thigh and hip region.

Chest radiography revealed hypoinflated lungs with prominent pulmonary vasculature but no consolidation, lobar infiltrate, or interstitial edema pattern, arguing against primary bacterial pneumonia or overt pulmonary edema as the predominant cause of hypoxemia.

Laboratory evaluation demonstrated elevated inflammatory markers (C-reactive protein 25.16, erythrocyte sedimentation rate 50), elevated liver enzymes (aspartate aminotransferase 85, alanine aminotransferase 75), and a normal complete blood count. Repeat COVID-19 and influenza tests were negative. A rapid antigen test for group A Streptococcus (GAS) was positive with an anti-streptolysin O (ASO) titer of 1,650 IU/mL (reference <200); however, he did not meet criteria for acute rheumatic fever, and there were no features of pharyngitis, exudative tonsillitis, or scarlatiniform rash.

Transthoracic echocardiography showed elevated right ventricular pressures (approximately half to two-thirds systemic) with preserved biventricular function and no coronary artery abnormalities. Examination confirmed a non-pruritic, non-confluent maculopapular rash over the thighs and lower extremities, without conjunctival injection, oral mucosal changes, lip erythema, or lymphadenopathy **(Figure 1)**.

Cardiology, infectious disease, and pulmonology services were consulted, and empiric therapy was initiated for community-acquired pneumonia, Mycoplasma infection, and possible viral myocarditis. Despite treatment with azithromycin, ceftriaxone, and piperacillin-tazobactam, respiratory status worsened, and direct hyperbilirubinemia with worsening transaminitis prompted gastroenterology consultation for suspected hepatitis.

Rheumatology was consulted for rash and arthralgia. Further history revealed recent close contact with a football teammate with COVID-19, raising suspicion for a post-infectious inflammatory process. On hospital day 7, a COVID-19 quantitative anti-nucleocapsid (anti-N) antibody and D-dimer were obtained. The D-dimer level was markedly elevated at 6,048 ng/mL FEU, consistent with a hyperinflammatory and prothrombotic state; it was interpreted as one supportive component of the overall clinical picture rather than a determinative finding. Based on the constellation of persistent fever, multiorgan involvement (cardiac, hepatic, respiratory, musculoskeletal), failure to improve with broad-spectrum antimicrobials, and the documented epidemiologic exposure history, the patient was clinically diagnosed as MIS-C. Intravenous corticosteroid therapy was initiated [1, 3], resulting in rapid defervescence, improved respiratory status, and a downward trend in liver enzymes and inflammatory markers. He was progressively weaned from supplemental oxygen to room air over 3 – 4 days, transitioned to an oral prednisone taper, and discharged home in stable condition on hospital day 6.

At the 2-week follow-up visit, the anti-N SARS-CoV-2 antibody test was positive. Anti-N antibodies are generated exclusively by natural SARS-CoV-2 infection and are not induced by mRNA-based COVID-19 vaccination; in this unvaccinated patient with a compatible clinical and epidemiologic picture, this result is consistent with prior natural SARS-CoV-2 infection. The patient demonstrated complete clinical recovery with resolution of rash, arthralgia, and respiratory symptoms, and normalization of inflammatory markers.

Cardiac follow-up with repeat transthoracic echocardiography was performed at approximately 5 weeks post-discharge. The study demonstrated complete normalization of right ventricular systolic pressure, evidenced by normal septal motion and normal right ventricular systolic function.

3. Discussion

MIS-C is a post-infectious hyperinflammatory syndrome that typically develops several weeks after exposure to severe acute respiratory syndrome coronavirus 2, often in patients without documented acute infection [1–3]. It is characterized by persistent fever, elevated inflammatory markers, and involvement of at least two organ systems, with frequent cardiac, gastrointestinal, mucocutaneous, and hematologic manifestations [1, 4].

In this case, a positive streptococcal test, respiratory symptoms, and radiographic findings compatible with pneumonia initially favored common pediatric infections, and management focused on antimicrobial therapy. The differential diagnosis encompassed Kawasaki disease, toxic shock syndrome, septic shock, viral myocarditis, and Henoch-Schönlein purpura [1, 2, 4]. The evolving multisystem inflammatory picture, failure to improve with antibiotics, and subsequent recognition of COVID-19 exposure prompted reconsideration of the diagnosis and evaluation for MIS-C.

The positive GAS rapid antigen test and markedly elevated ASO titer (1,650 IU/mL) warranted careful adjudication between true streptococcal infection and asymptomatic colonization. Several features argued against GAS as the primary pathologic driver: there was no pharyngitis, exudative tonsillitis, or scarlatiniform rash to support active streptococcal disease; the patient remained hemodynamically stable without features of streptococcal toxic shock; blood cultures showed no growth; and, critically, the clinical trajectory worsened despite targeted ceftriaxone coverage until corticosteroid initiation. Elevated ASO titers may persist for weeks to months after prior infection and, therefore, cannot be used to diagnose current active infection in isolation [1]. Throat culture was not obtained – a rapid antigen test was used – and procalcitonin was not measured; these represent limitations of this case, as their absence

prevents definitive microbiologic distinction between carriage and true infection.

The markedly elevated D-dimer (6,048 ng/mL FEU) was consistent with the hyperinflammatory and prothrombotic state seen in MIS-C but was interpreted as one element of a broader clinical picture. The diagnosis and treatment decision rested on the overall constellation of persistent fever, multiorgan involvement, elevated right ventricular pressures, hepatobiliary dysfunction, failure to improve with antimicrobials, and exclusion of alternative primary diagnoses.

Current guidelines emphasize early recognition and prompt initiation of immunomodulatory therapy, most commonly intravenous immunoglobulin, corticosteroids, or a combination, alongside antithrombotic prophylaxis in selected cases [3, 4]. In this patient, intravenous corticosteroids alone were associated with rapid clinical and biochemical improvement, highlighting the potential effectiveness of timely steroid therapy in carefully selected patients.

4. Conclusion

This case highlights the need to maintain a high index of suspicion for multisystem inflammatory syndrome in children in patients who present with fever, rash, and multiorgan involvement following COVID-19 exposure, even when initial testing for acute infection is negative [1]. Early recognition of MIS-C and timely initiation of corticosteroid therapy in this child were associated with rapid clinical improvement and favorable short-term outcomes, emphasizing the importance of prompt diagnosis and management in similar pediatric presentations.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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Informed Consent

Verbal informed consent for publication was obtained by telephone from the patient's parents, and the consent was documented.

Large Language Model

None.

Author Contributions

TS and LK contributed to patient care, clinical data collection, manuscript drafting, and literature review. DN contributed to case conceptualization, diagnostic refinement, manuscript revision, and critical intellectual review. SU served as the supervising attending physician and contributed to overall clinical supervision, case oversight, diagnostic guidance, and final manuscript approval.

Data Availability

The data supporting the findings of this case report are not publicly available due to patient privacy considerations. De-identified data may be made available by the corresponding author upon reasonable request, subject to institutional privacy protocols.

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