World Academy of Science, Engineering and Technology
International Journal of Medical and Health Sciences
Vol:16, No:05, 2022

Post COVID-19 Multi-System Inflammatory Syndrome Masquerading as an Acute Abdomen

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Abstract : This paper describes a rare occurrence where a potentially fatal complication of COVID-19 infection (MIS-A) was misdiagnosed as an acute abdomen. As most patients with this syndrome present with fever and gastrointestinal symptoms, they may inadvertently fall under the care of the surgical unit. However, unusual imaging findings and a poor response to anti-microbial therapy should prompt clinicians to suspect a non-surgical etiology. More than half of MIS-A patients require ICU admission and vasopressor support. Prompt referral to a physician is key, as the cornerstone of treatment is IVIG and corticosteroid therapy. A 32 year old woman presented with right sided abdominal pain and fevers. She had also contracted COVID-19 two months earlier. Abdominal examination revealed generalised right sided tenderness. The patient had raised inflammatory markers, but other blood tests were unremarkable. CT scan revealed extensive lymphadenopathy along the ileocolic chain. The patient proved to be a diagnostic dilemma. She was reviewed by several surgical consultants and discussed with several inpatient teams. Although IV antibiotics were commenced, the right sided abdominal pain, and fevers persisted. Pan-culture returned negative. A mild cholestatic derangement developed. On day 5, the patient underwent preparation for colonoscopy to assess for a potential intraluminal etiology. The following day, the patient developed sinus tachycardia and hypotension that was refractory to fluid resuscitation. That patient was transferred to ICU and required vasopressor support. Repeat CT showed peri-portal edema and a thickened gallbladder wall. On re-examination, the patient was Murphy's sign positive. Biliary ultrasound was equivocal for cholecystitis. The patient was planned for diagnostic laparoscopy. The following morning, a marked rise in cardiac troponin was discovered, and a follow-up echocardiogram revealed moderate to severe global systolic dysfunction. The impression was post-COVID MIS with myocardial involvement. IVIG and Methylprednisolone infusions were commenced. The patient had a great response. Vasopressor support was weaned, and the patient was discharged from ICU. The patient continued to improve clinically with oral prednisolone, and was discharged on day 17. Although MIS following COVID-19 infection is well-described syndrome in children, only recently has it come to light that it can occur in adults. The exact incidence is unknown, but it is thought to be rare. A recent systematic review found only 221 cases of MIS-A, which could be included for analysis. Symptoms vary, but the most frequent include fever, gastrointestinal, and mucocutaneous. Many patients progress to multi-organ failure and require vasopressor support. 7% succumb to the illness. The pathophysiology of MIS is only partly understood. It shares similarities with Kawasaki disease, macrophage activation syndrome, and cytokine release syndrome. Importantly, by definition, the patient must have an absence of severe respiratory symptoms. It is thought to be due to a dysregulated immune response to the virus. Potential mechanisms include reduced levels of neutralising antibodies and autoreactive antibodies that promote inflammation. Further research into MIS-A is needed. Although rare, this potentially fatal syndrome should be considered in the unwell surgical patient who has recently contracted COVID-19 and poses a diagnostic dilemma.

Keywords : acute-abdomen, MIS, COVID-19, ICU
Conference Title : ICAGSO 2022 : International Conference on Advanced General Surgery and Oncology
Conference Location : Sydney, Australia
Conference Dates : May 16-17, 2022