Multiple drugs

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Hepatic steatosis: case report

A 14-year-old boy developed hepatic steatosis during treatment with aspirin, unspecified low-molecular-weight heparin, immune globulin, methylprednisolone and prednisone for multisystem inflammatory syndrome in children (MIS-C) [*not all routes stated*].

The boy was admitted to the Paediatric Emergency Department with compromised general status on 1 March 2021. He was found to be oriented and alert, febrile, tachycardic, hypotensive, saturating 100% on room air breathing 42 breaths per minute. Upon clinical examination, he was found to be dehydrated, with jaundiced skin, capillary refill 3–4s, cold extremities with feeble peripheral pulses, palmoplantar erythema and oedema, diffusely painful abdomen and palpable hepatosplenomegaly. He was in a condition of multiorgan failure: acute kidney injury, hepatosplenomegaly with increased inflammatory markers, particularly IL-6 and compromised haemodynamic status with reduction of left ventricular ejection fraction. Also, elevated values of ALT and AST were noted, and it indicated cholestasis. Considering the family history of SARS-CoV-2 infection and the positivity of SARS-CoV-2 IgG from serum, a diagnosis of MIS-C was made. His compromised haemodynamic status necessitated vasoactive support at the Cardiac Intensive Care Unit for the first 3–4 days of hospitalisation. He was treated with IV immune globulin [immunoglobulin] 2 g/kg over 48 hours and pulse methylprednisolone 10 mg/kg a bolus twice a day for 3 days and then progressively reduced. Owing to the elevated D-dimer, a prophylactic antithrombotic therapy was commenced with aspirin 3 mg/kg/day and unspecified low-molecular-weight heparin. During hospital course, an amelioration of his general conditions with return to clinical baseline and progressive normalisation of laboratory alterations, especially ALT and cholestatic indices, was observed. Abdominal ultrasonography was performed prior to discharge, and no abnormalities were observed. He was discharged at day 14 with indication to continue therapy with aspirin and oral prednisone, in progressive gap according to the tapering strategy. Following discharge, he was re-evaluated in a follow-up visit program, during tapering off of prednisone was started (until 1 mg/Kg/day). On 23 March 2021, at the first follow-up visit, he was found to be in good conditions, with no particular findings on the physical examination. The laboratory tests were normal besides the elevation of ALT. On the second follow-up visit, 30 days after the onset of MIS-C, he was completely asymptomatic but clinical assessment showed palpable hepatomegaly and further elevation of ALT on laboratory testing. Abdominal ultrasonography showed that hepatomegaly was secondary to hepatic steatosis, with absence of pre-existing obesity and signs of Non-alcoholic Fatty Liver Disease. Additionally, any other previous or existing inflammatory pathology was ruled out. The following follow-up visits, laboratory tests and abdominal ultrasonographies showed data indicating hepatic steatosis as well. It was considered that he developed hepatic steatosis secondary to aspirin, unspecified low-molecular-weight heparin, immune globulin, methylprednisolone and prednisone use.

A drug therapy was not introduced, but a lifestyle approach including dietary improvements and increasing physical activity were started. At the time of this report, the boy was in good general clinical condition, with an optimal recovery of cardiac and renal function and not on drug therapy, as steroid therapy was discontinued during the follow-up visit program. Nonetheless, he still had hepatic steatosis, which was also confirmed by abdominal ultrasonography performed during the most recent follow-up visit on 27 May 2021.

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