

Remdesivir/methylprednisolone/convalescent-anti-SARS-CoV-2-plasma

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Elevated aminotransferase levels and off-label use: case report

An 18-year-old woman experienced elevated aminotransferase levels after receiving treatment with remdesivir for COVID-19 infection. Additionally, she received off label treatment with convalescent-anti-SARS-CoV-2-plasma and methylprednisolone for COVID-19 infection [*not all routes and dosages stated*].

The woman presented to the hospital after experiencing nausea, fever, epigastric pain, vomiting, diarrhoea, and shortness of breath for 3 days. Eventually, she underwent laboratory investigations and was diagnosed with COVID-19. She developed hyperbilirubinaemia, severe coagulopathy, and profound haemolytic anaemia with aminotransferase elevations. The following laboratory examination revealed: AST was 596 U/L and ALT was 37 U/L. She underwent several laboratory investigations and revealed hypoechoic hepatic echotexture and haemophagocytosis. She was hospitalised due to COVID-19 and intermittent haemodialysis (IHD) was started on day 4 with continuous renal replacement therapy on day 6. Renal and hepatic failure precluded remdesivir use. She received off-label treatment with 2 units of convalescent-anti-SARS-CoV-2-plasma [convalescent plasma] on days 5 and 6, and off-label IV methylprednisolone 125 mg/day. Consequently, she was also diagnosed with wilson's disease. Despite supportive treatment, her condition worsened and she fulfilled acute liver failure criteria for hepatic encephalopathy, coagulopathy, and lack of documented past liver illness. She had abnormal hepatic echotexture, and her spleen was mildly enlarged with no specific portal hypertension or cirrhosis observed. Despite administering lactulose and rifaximin, she had increasing hepatic encephalopathy on day 14. She received albumin dialysis as a possible bridge to transplantation. Eventually, on day 16, she was diagnosed with acute liver failure secondary to wilson's disease, but also atypical COVID-19-related liver injury was not completely excluded. She then received ceftriaxone, vancomycin, and anidulafungin. She underwent liver transplantation on day 17. Post-transplant revealed confluent hepatocyte necrosis, cirrhosis, and steatohepatitis. Postoperatively, she received immunosuppression treatment unspecified tapered steroid and basiliximab along with tacrolimus. Her mycophenolate mofetil treatment was halted because of COVID-19. Remdesivir 200mg per NG tube day 1, 100mg days 2–10, was started right after the transplant. Post transplantation, she underwent following laboratory examination including: WBC 18.1; haemoglobin 7.5; MCV 98.7; platelets 39; INR 2.8; fibrinogen 190; LDH 1563; total bilirubin 21.9; direct bilirubin 17.2; Na⁺ 146; K⁺ 4.4; HCO₃⁻ 23; creatinine 2.57; BUN 36; Ca²⁺ 8.5; glucose 136; AST 545; ALT 419; ALP 50; albumin 2.7; CK 2,529; ferritin 9,455; c-reactive protein 19.9; ammonia 125; Lactate 9.5. Post-transplant, her ALT level was found to be elevated and she tested positive for SARS-CoV-2 PCR. Eventually, a diagnosis of elevated aminotransferase levels was made [*time to reaction onset not stated*]. On day 27, she was extubated and on day 36 her treatment with mycophenolate mofetil was started. She was on continuous renal replacement therapy until day 37, when she was transferred to IHD and eventually restored renal function without IHD. Her transaminase levels are now steady but considerably increased: AST was 109 U/L and ALT was 173 U/L. She also developed rhabdomyolysis. Her treatment was started with prednisone due to suspected COVID-19-related myopathy. Eventually, she was diagnosed with myopathy. Later on, she was discharged and had no cognitive or neurologic abnormalities [*outcome not stated*].

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