1. Introduction

Rhabdomyolysis is a syndrome caused by the rapid breakdown of damaged skeletal muscle, which results in the release of potentially toxic intracellular content into plasma. It is characterized by a triad of muscle weakness, myalgia, and abnormal blood tests in the context of other underlying problems. Most adult cases of rhabdomyolysis are due to abuse of illicit drugs or alcohol, muscular trauma, crush injuries, prolonged immobilization, excessive muscular activity, electrolyte abnormalities, and myotoxic effects of prescribed drugs such as statins and cocaine (1–3). Lamivudine-induced rhabdomyolysis is a rare adverse drug reaction and can be fatal if not recognized early. Various complications are associated with rhabdomyolysis, including hypovolaemia, compartment syndrome, arrhythmias and cardiac arrest, disseminated intravascular coagulation, hepatic dysfunction, acidosis, and myoglobinuric acute renal injury (2). Fatality, which has been reported at in-hospital rates as high as 59%, and myoglobinuric acute renal injury can be prevented by early fluid resuscitation (3, 4). Therefore, early identification of this syndrome is important. However, treatment may be complicated by the patient’s underlying co-morbidities, such as congestive cardiac failure or underlying chronic kidney disease. Lamivudine-induced rhabdomyolysis has been reported previously in other literatures (5), but to the best of our knowledge, it has not, to the best of our knowledge, been reported in Malaysia before (5, 6).

2. Case presentation

2.1. Clinical presentation

A 31-year-old Malay man, admitted to the cardiothoracic ward for an elective Bentall procedure, was referred to the medical team at day 4 of admission for worsening renal and hepatic profiles. Upon admission to the cardiothoracic ward, he appeared to be well within class II of the New York Heart Association (NYHA) Functional Classification. New York Heart Association (NYHA) functional class II. His vital signs were stable. Physical examination revealed a moderate pan-systolic murmur and a palpable liver edge, about 3 cm below the right costal margin. Other system examinations were unremarkable. Upon consultation with the hepatobiliary team, a provisional diagnosis of acute liver failure secondary to acute flare of hepatitis B infection was made and empirical antiviral treatment with 100 mg of oral Lamivudine daily was initiated. Three days after the initiation of Lamivudine, the patient developed myalgia. There was significant muscle tenderness and swelling of the upper and lower limbs, but he did not have tea-coloured urine or gross haematuria.

2.2. History

The patient had a background history of congenital valvular heart disease, thoracic aortic dissection, aneurysm, and aortic dissection, and recently diagnosed hepatitis B viral infection. An echocardiogram showed a congenital bicuspid aortic valve with severe aortic regurgitation, complicated by dilated cardiomyopathy.
2.3. Laboratory and imaging findings

Baseline blood investigations conducted upon admission showed mild hepatic and renal impairments with alanine transaminase (ALT), aspartate transaminase (AST), total serum bilirubin, urea, and creatinine levels of 361 U/L, 181 U/L, 53 µmol/L, 8.9 mmol/L and 127 µmol/L, respectively. On subsequent days of admission, his hepatic and renal function worsened, with ALT elevated up to ten-fold from the baseline value, urea of 28 mmol/L, and creatinine of 152 µmol/L. A full blood count revealed thrombocytopenia with a platelet count of 71 x 10^9/L; also, the coagulation profile was also deranged. Screenings for other common infectious diseases were negative, pending hepatitis B e-antigen and viral load. An ultrasound of the patient’s hepatobiliary system revealed normal findings with no focal attributes. Worsening of the liver function tests led to the provisional diagnosis of acute liver failure secondary to acute flare of hepatitis B infection.