LETTER TO EDITOR

Acute acalculous cholecystitis following the bite of Indian saw-scaled viper

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Full Text

Sir,

A 27-years-old man was bitten on the left foot by a saw-scaled viper. Twelve hours later he developed local pain, swelling and hematuria. On admission to the hospital 24 hours later, he had anuria and jaundice. He had tachycardia, normal blood pressure and normal systemic examination. Laboratory tests revealed blood urea nitrogen 78 mg/dL, s. creatinine 6.0 mg%, s. potassium 5.8 mmol/L, total leucocyte count (TLC) 21 x 10^9/L, platelet count 3 x 10^9 sub/L, activated plasma thromboplastin time 95.3 seconds (control 29.4 seconds), INR 2.8, fibrin degradation products > 80 mg/ml, fibrinogen 130.5 mg/dl and serum bilirubin 6.8 mg% (direct bilirubin 1.6 mg%). Ultrasonography showed enlarged kidneys with normal liver, gallbladder and biliary system. He received 10 vials of polyvalent anti-snake venom (Haffkine Institute, Mumbai), fresh frozen plasma, platelet transfusions and intermittent hemodialysis. Over the next 5 days, coagulation parameters, bilirubin and TLC normalized. Six days after admission, the patient developed severe right hypochondriac pain, high-grade fever and a palpable, tender gallbladder lump. Ultrasonography revealed distended gallbladder with thickened wall (4 mm) and rim of pericholecystic fluid [Figure 1] with no gallstones. Total bilirubin increased to 3.8 mg%, direct was 1.9 mg%, SGOT (AST) 181 IU/L, SGPT (ALT) 313 IU/L, alkaline phosphatase 359 IU/L and TLC increased to 27.3 x 10^9/L. Blood culture grew E. coli sensitive to cefoperazone and urine culture was sterile. 99m-Technetium mebrofenin scintigraphy revealed hepatomegaly, a normal biliary tract and non-visualization of the gallbladder, confirming the diagnosis of acute cholecystitis. Patient was given cefaperazone and metronidazole. After initial improvement in serum bilirubin and hepatic enzymes, the patient developed septic shock and eventually succumbed to multiple organ dysfunction 11 days after snakebite. Post-mortem examination revealed a grossly distended, non-perforated gallbladder. Histopathology confirmed acute cholecystitis.

Viper venom contains phospholipase A2, which produces local inflammation and tissue necrosis and hemorrhagin, which causes endothelial damage.[1] Activation of platelets and coagulation cascade and primary fibrinolysis is caused by hemorrhagin, Echicetin (a heterodimeric C-type lectin), Echistatin (an RGD-disintegrin) and metalloproteases like ecarin and carinactivase A.[2] This causes disseminated intravascular coagulation (DIC) with hematuria, epistaxis, bleeding gums and gastrointestinal bleeding. Acute renal failure occurs in 20% of cases due to ischemic tubular necrosis and a direct nephrotoxic effect of the venom. Phospholipase A2 also induces myocardial damage.[1] However, acalculous cholecystitis has not been reported following viper bite.

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Acute acalculous cholecystitis occurs in 1.5% of critically ill patients due to stasis of bile and ischemia of the gallbladder wall from hypotension and microcirculatory thrombosis.6 Predisposing conditions include sepsis, severe falciparum malaria, total parenteral alimentation and positive pressure ventilation;[3] our patient had none of these. The exact mechanism of acalculous cholecystitis in our patient is not clear. When radiolabelled venom is administered by parenteral injection, a large proportion of it is excreted by the hepatobiliary route.[4] Phospholipase A2, which causes local inflammation and necrosis, may play an important role in the pathogenesis by converting phospholipids in bile into toxic fatty acids and lysolecithin.[5] Microcirculatory thrombosis secondary to DIC may also have contributed by causing gallbladder ischemia.

References

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