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

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 leukocyte count (TLC) 21 x 10⁹/L, platelet count 3 x 10⁹/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. Histopathology confirmed acute acalculous cholecystitis.

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. Predisposing conditions include sepsis, severe falciparum malaria, total parenteral alimentation and positive pressure ventilation; our patient had none of these. The exact mechanism of acalculous cholecystitis in our patient is not clear. When radio-labelled venom is administered by parenteral injection, a large proportion of it is excreted by the hepatobiliary route. 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 lyssolecithin. Microcirculatory thrombosis secondary to DIC may also have contributed by causing gallbladder ischemia.

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Viper venom contains phospholipase A₂, which produces local inflammation and tissue necrosis and hemorrhagia, which causes endothelial damage. Activation of platelets and coagulation cascade and primary fibrinolysis is caused by hemorrhagia, Echicetin (a heterodimeric C-type lectin), Echistatin (an RGD-disintegrin) and metalloproteases like ecarin and carinactivase A. 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. However, acalculous cholecystitis has not been reported following viper bite.

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**References**

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