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Streptokinase-induced jaundice

Sir,

Streptokinase is widely used to treat myocardial infarction. It can cause abnormal liver function but rarely jaundice. We describe the case of a patient who developed jaundice following streptokinase infusion for acute myocardial infarction.

A 43 year old male was admitted with an acute myocardial infarction. He took atenolol 50 mg daily for hypertension, smoked 20 cigarettes each day and drank 2 pints of beer a week. He had no history of gallstones, foreign travel, contact with hepatitis, previous blood transfusion or intravenous drug abuse. He received 300 mg aspirin and an infusion of 1.5 MU streptokinase. Pain and nausea were relieved by intravenous diamorphine and metoclopramide.

The following day he was jaundiced. Urinalysis showed blood and bilirubin. His full blood count remained stable, and plasma electrolytes and clotting screen were normal. Liver function tests (normal on admission) showed: plasma bilirubin 133 IU/l (NR < 17), aspartate aminotransferase 140 IU/l (NR 6–38), alanine aminotransferase 143 IU/l (NR 4–40) and alkaline phosphatase 117 IU/l (NR 30–130). Abdominal ultrasound showed no evidence of intra- or extrahepatic biliary dilatation or biliary calculi. Markers for hepatitis A and B, cytomegalovirus and Epstein–Barr virus were negative. The jaundice subsided and liver function tests returned to normal over the next 3 days. In the absence of any other explanation, the jaundice was attributed to streptokinase.

Four weeks later he was readmitted with chest pain and left ventricular failure. Serial electrocardiogram and cardiac enzymes confirmed extension of the original myocardial infarction. He did not receive further thrombolysis.

The Committee on Safety of Medicines has received 22 reports of liver complications with streptokinase (July 1963–April 1993): four with abnormal liver function, 15 with jaundice and four with hepatocellular damage. Yet, despite its widespread use in acute myocardial infarction, only three cases of jaundice have been reported in this context in the literature.^{1,2} Jaundice has been reported after streptokinase infusion for extensive deep venous thrombosis, settling when the infusion was discontinued, but recurring when rechallenged.³

The pathophysiology of streptokinase-induced jaundice is not known. Animal studies show a rise in liver

enzymes following infusion with streptokinase, thought to be due to proteases reaching liver tissue.⁴ Normally proteases are rapidly inactivated by plasma protein binding to alpha 2 plasmin and alpha 2 macroglobulin, before being metabolized by the liver. Streptokinase causes generalized fibrinolysis which may overwhelm this protective mechanism in susceptible individuals.

When this patient presented with further myocardial infarction, we considered additional thrombolytic treatment but were apprehensive about subsequent reaction. It is possible that alteplase, being clot specific, so causing less plasmin formation, could have been given with impunity.

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Massive rectal bleeding due to ileocaecal tuberculosis (conservative approach)

Sir,

Torrential bleeding due to ileocaecal tuberculosis is a rare manifestation. The traditional method of treatment has been surgical resection of the affected part. We report a case managed conservatively with complete recovery.

A 27 year old female was admitted with continuous fever and weakness for about 2½ months. On physical examination she was emaciated, febrile, pale and toxic. There was icterus, tachycardia and mild pallor. The liver was 1 cm below the subcostal arch, with a firm mass in the right iliac fossa. Next day she had 250 ml of reddish bleeding per rectum. Her general condition deteriorated and she continued to bleed for one week.

Her haemoglobin was 7.8 g/dl which dropped down to 4.5 g/dl; chest X-ray revealed a non-homogeneous opacity in the left upper zone consistent with active tuberculosis. Colonoscopy showed an oozing nodular,