Letters to the Editor

Right-sided endocarditis in the non-drug addict

Sir,

The differential diagnosis of the underlying causes of right-sided endocarditis includes not only the non-neoplastic conditions listed by Naidoo but also the occasional case of colonic neoplasm complicated by streptococcus bovis bacteraemia. I concur that one sometimes has to institute full antibiotic therapy for infective endocarditis even when patients with right-sided vegetations have sterile blood cultures. This was my experience in the management of an 80 year old woman presenting with syncope and a systolic murmur, in whom two-dimensional transthoracic echocardiography showed a 1 cm diameter pulmonary valve vegetation which appeared to prolapse into the pulmonary artery. Three blood cultures were sterile, and the serological tests for chlamydia, Coxiella burnetii, and brucellosis were negative. Four weeks and 16 weeks, respectively, after presentation, the pulmonary valve vegetation remained unchanged, although the patient felt well and was afebrile, with a normal ESR. Like the patients reported by Naidoo, this woman had no history of intravenous drug abuse. Finally, notwithstanding the success of two-dimensional transthoracic echocardiography in imaging right-sided vegetations, transthocharic echocardiography has, nevertheless, been shown to have greater diagnostic sensitivity, as well as the ability to distinguish more successfully between right atrial anatomical artifacts and authentic space occupying lesions.

Acknowledgement

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References


Dystonia – a rare manifestation of carbamazepine toxicity

Sir,

Carbamazepine is widely used to treat seizure disorders, trigeminal neuralgia and, more recently, affective disorders. Side effects commonly encountered during its use include drowsiness, diplopia, blurring of vision, disturbances of equilibrium and, after prolonged use, abnormalities of liver functions. Dystonia is a distinctly uncommon manifestation associated with carbamazepine toxicity and has been reported only infrequently. We encountered a patient who presented with dystonia after ingesting a single toxic dose of carbamazepine.

A 13 year old female was brought to the medical emergency room 3 hours after she had developed altered sensorium and abnormal posturing of her trunk and extremities. She was not a known epileptic and there was no other significant present or past history. A detailed review of family history revealed that her grandmother, who was a member of the household, was taking carbamazepine for a major psychotic illness.

Examination revealed that the patient was comatose, responding only to painful stimuli. She had frequent dystonic movements involving her trunk and extremities. The cerebrospinal fluid, haemoglobin, serum biochemistry, urine, chest X-ray and electrocardiogram were normal.

The patient was given gastric lavage and supportive care. The next day her sensorium improved and a down-beat nystagmus became elicitable. By the third day, she had regained consciousness sufficiently to volunteer that she had swallowed eight of her grandmother's pills (each tablet containing 200 mg of carbamazepine).

Despite the widespread use of carbamazepine and many reports of its toxicity, there have been relatively few reports of dystonia occurring after its use. Some of these have been of dystonia associated with the use of carbamazepine in brain-damaged children. In others, the patient had received other drugs in addition to carbamazepine. We could find only a single case of deliberate carbamazepine poisoning resulting in dystonia in the English language literature. Though in most of these cases serum levels of carbamazepine were in the toxic range, in at least one case the blood level was within the accepted therapeutic range. There is no fixed relationship between the dose of the drug and its concentration in the serum.

Although in our patient the serum levels of carbamazepine could not be measured, the temporal relationship of ingestion of 1,600 mg of carbamazepine to the occurrence of dystonia and nystagmus, and the disappearance of these symptoms over the next 2 days establishes a causal relationship.

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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: plasmas bilirubin 133 IU/l (NR <17), aspartate aminotransferase 140 IU/l (NR 6–38), alanine amino transferase 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. Jaffé jaundice has been reported after streptokinase infusion for extensive deep venous thrombosis, settling when the infusion was discontinued, but recurring when rechallenged.

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. 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 plasmolysis, 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.

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.

References


Dystonia--a rare manifestation of carbamazepine toxicity.

P. Soman, S. Jain, V. Rajsekhar, S. Vineeta and B. K. Sharma

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