Acute Pancreatitis Mimicking Myocardial Infarction: Potential for Inadvertent Use of Thrombolytic Therapy

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SUMMARY: A case of acute pancreatitis mimicking myocardial infarction is presented and the potential for inadvertent use of thrombolytic therapy is discussed.

Introduction

In the recent past the management of a patient suspected of having had an acute myocardial infarction would have been largely conservative (1). Serial electro cardiograms (ECG) and cardiac enzyme estimations would have clarified the diagnosis over the ensuing few days. The advent of thrombolytic therapy has created pressure to make a more rapid and certain diagnosis (2) and to exclude mimicking conditions where the use of this therapy may be hazardous (3, 4, 5). Considerable emphasis has been placed on the inadvertent use of thrombolytics in pericarditis or aortic dissection (2, 4) but less attention has been given to the potential dangers to the patient with acute intra-abdominal pathology presenting with chest pain and ECG abnormalities.

Case Report

A 79-year-old female was admitted with a 5 hour history of severe retrosternal chest pain of sudden onset. The pain radiated through to the midscapular area and was associated with nausea and profuse sweating. There was no history of cardiac or abdominal pathology. Her only medication was thyroxine (0.1 mg/day). She was a lifelong non-smoker and drank 5 units of alcohol weekly. Her ECG showed sinus rhythm (rate 66/min), complete left bundle branch block and a P-R interval of 0.22 sec. Her chest X-ray was normal and there were no clinical features of pericarditis or dissection of the thoracic aorta. Despite a strong clinical diagnosis of acute myocardial infarction, abdominal examination revealed slight tenderness in the epigastrium on deep palpation. While streptokinase had been drawn up it was withheld pending surgical assessment. This prompted measurement of the serum amylase. On report of a serum amylase of >5,000 IU/l the clinical diagnosis was revised to acute pancreatitis and the patient managed conservatively. A gastrograffin swallow showed no leak from stomach or duodenum. The serum amylase fell progressively to 522 IU/l by day 5 and subsequent investigation revealed the presence of multiple gallstones.

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She underwent laproscopic cholecystectomy on day 13 and had an uneventful recovery. The ECG abnormalities did not change and the cardiac enzymes did not rise.

Discussion

In the presence of bundle branch block the initial diagnosis of acute myocardial infarction must be made on clinical grounds alone. Such patients sustain substantial benefit from prompt thrombolytic therapy, an effect magnified in the elderly (6). In the case presented only the presence of slight epigastric tenderness raised the possibility of intra-abdominal pathology and delayed streptokinase administration pending amylase estimation.

The one similar case in the literature tells of a patient given streptokinase with a diagnosis of an acute myocardial infarction. Unbeknown to the admitting physicians there was previously proven alcohol related pancreatitis with pseudocyst formation. Thrombolytic therapy was followed by fatal intra-abdominal and retroperitoneal haemorrhage (7).

Table 1
Causes of hyperamylasaemia which may mimic acute myocardial infarction

1.	Aortic aneurysm with dissection
2.	Pneumonia
3.	Pancreatitis
4.	Biliary tract disease
5.	Perforated gastric or duodenal ulcer

(Adapted from references 8 and 9)

Hyperamylasaemia is observed in a number of conditions that may present as 'myocardial infarction' (Table 1) (8, 9). In these conditions it is unlikely that thrombolytics would be helpful and could lead to severe haemorrhagic problems. The high frequency of ECG abnormalities observed in patients presenting with acute pancreatitis may also lead to diagnostic confusion (10).

This report suggests that a low threshold should exist for measuring serum amylase prior to the administration of thrombolytic agents in the presence of abdominal pain or previous history of pancreatic disease.

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