Oesophageal dissection after thrombolytic treatment for myocardial infarction

F Jishi, C E Sissons, E J Silverstone, J F Coakley, F Fraser

Abstract

A 62 year old woman admitted with a history suggesting acute myocardial infarction had thrombolytic treatment with anisoylated plasminogen-streptokinase activator complex, which resulted in submucosal haemorrhage in the oesophagus; this caused dissection of the wall of the oesophagus and complete dysphagia. The haematoma resolved spontaneously, leaving behind a diverticulum, with reduced peristalsis and delayed emptying but no obstruction.

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Thrombolytic treatment is now an established part of treatment for acute myocardial infarction. Serious haemorrhagic sequelae, though rare, are a recognised complication of thrombolytic treatment. We describe a case of submucosal haemorrhage in the oesophagus that followed treatment with anisoylated plasminogen-streptokinase activator complex.

Case report

A 62 year old woman presented with crushing retrosternal chest pain radiating to the jaw, sweating, and dyspnoea lasting about two hours. She gave a two year history of symptoms suggesting angina pectoris. She had had no previous upper gastrointestinal symptoms and no other important medical problems.

Her ECG at admission showed Q waves with T wave inversion in leads 3 and AVF. On the basis of her history and ECG changes a provisional diagnosis of acute myocardial infarction was made and she was given a bolus of anisoylated plasminogen-streptokinase activator complex. She was also given soluble aspirin (162·5 mg).

The day after admission she started to complain of difficulty in swallowing and pain at the back of her throat. Local examination of the throat showed mild inflammation and few aphthous ulcers. Over the next two days the dysphagia gradually progressed until it became complete and the haemoglobin concentration fell from 12·9 to 10·4 g/dl.

She also complained of recurrent central chest pains, which were thought to be due to unstable angina and which responded to calcium antagonist and glyceryl trinitrate infusion. Serial ECGs showed persistent Q waves in the inferior leads with the ST and T wave changes of myocardial ischaemia, but serum cardiac enzymes were always normal.

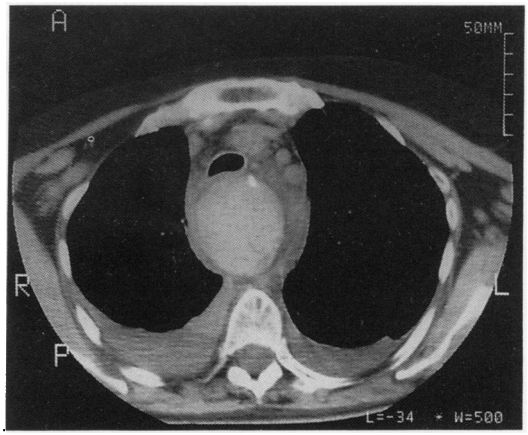

Figure 1 Computed tomogram of the upper thorax: grossly distended oesophagus with radio-opaque feeding tube anteriorly, compression of the trachea, and bilateral pleural effusions.

A barium swallow showed complete obstruction at the level of T3. The oesophagus tapered over a short segment and was irregular with mild proximal dilatation. In addition, there was a suggestion of a posterior mass projecting into the lumen.

At oesophagoscopy the cricopharyngeal area was found to be friable. Attempts to pass a flexible and a rigid oesophagoscope beyond this level were unsuccessful, as were attempts to pass a nasogastric tube. The following day a fine bore nasogastric tube was passed under radiographic control. Computed tomography of the neck and thorax showed the oesophagus to be massively distended from the diaphragmatic hiatus to the thoracic inlet, with a maximum diameter of 55 mm. The lumen was identified anteriorly by the feeding tube and a rim of gastrografin outlining the periphery of the mass. The mass was non-homogeneous with a density of up to 68 Hounsfield units, consistent with organising haematoma (figs 1 and 2). A diagnosis of oesophageal submucosal haemorrhage secondary to thrombolytic treatment was made.

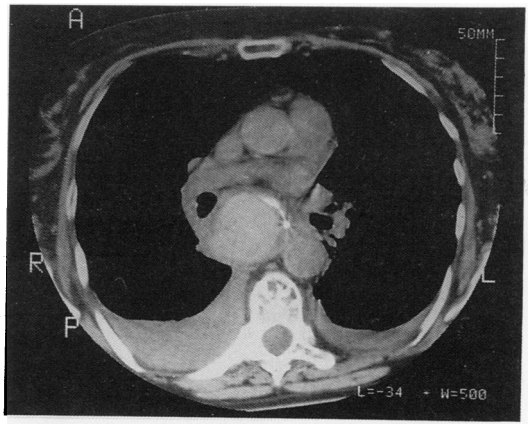

Figure 2 Computed tomogram of the thorax at the subcarinal level: feeding tube and gastrografin outline of the compressed oesophageal lumen.

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The patient was fed by a nasogastric tube for the next 12 days, after which she was able to swallow liquids. By the end of six weeks she was able to swallow soft food. Repeat computed tomography at this stage showed a dissection of the oesophageal mucosa with a false lumen and small residual haematoma.

Five months after the initial event the patient is able to eat and drink normally. A recent barium swallow shows generalised residual irregularity and poor peristalsis, but no obstruction or stricture. She continues to suffer from angina, however, and coronary angiography has shown occlusion of the right coronary artery. Coronary bypass surgery has been recommended.

Discussion

Thrombolytic treatment has been the most important recent advance in the management of myocardial infarction since the advent of defibrillation and is expected to save about 30 lives per 1000 patients treated.[1]

Minor bleeds (bleeding from the skin at venepuncture, gum bleeds, and microscopic haematuria), though common, are of little medical importance, and in fact can be taken as an indication of the effectiveness of the treatment. Major gastrointestinal, cerebral, or other haemorrhage, however, may have serious consequences. Fortunately, severe bleeding occurs in only 1·9% of all patients receiving thrombolytic treatment.[2] The incidence of gastrointestinal bleeding is only 0·8% higher in patients with myocardial infarction receiving thrombolytic treatment than in control patients.[2]

In our patient bleeding into the oesophageal submucosa caused dissection of the oesophagus. The bleeding may have been spontaneous or it may have been induced by the retching and vomiting that followed administration of diamorphine.

An alternative possibility is that the patient's initial presentation with chest pain was due to spontaneous oesophageal rupture, and that thrombolytic treatment aggravated her condition by causing severe bleeding. Such a possibility can be dismissed because the patient gave no history of dysphagia, vomiting, or retching before admission. The finding of an occluded right coronary artery at angiography also supports the diagnosis of myocardial infarction.

Although the benefits of thrombolytic treatment in myocardial infarction outweigh the risks of serious haemorrhage, clinicians must be aware of the potential for bleeding at unusual sites. To our knowledge acute dysphagia due to submucosal haemorrhage in the oesophagus has not been described before.

1 Second International Study of Infarct Survival (ISIS-2) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17 187 cases of suspected acute myocardial infarction. *Lancet* 1988;ii:349–60.

2 Yusuf S, Collins R, Peto R, Furberg G, Stampfer MJ, Goldhaber S, *et al*. Intravenous and intracoronary fibrinolytic therapy in acute myocardial infarction: overview of results on mortality, reinfarction and side effects from 33 randomized controlled trials. *Eur Heart J* 1985;6:556–85.