Cardiac tamponade after thrombolysis

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Summary: Thrombolysis has been very effective in reducing the morbidity and mortality from acute myocardial infarction. Serious adverse events are not uncommon, however. We describe a case in which a haemopericardium and tamponade developed in a patient with a history of recurrent idiopathic pericarditis and to whom streptokinase had been administered following a suspected myocardial infarction. The case highlights the need for caution in the administration of thrombolytics to patients with a documented history of pericarditis.

Introduction

Thrombolysis is the treatment of choice in acute myocardial infarction although serious adverse effects are well recognized and not uncommon.¹ Cerebrovascular accidents and gastrointestinal bleeding are the commoner life-threatening problems described. Little attention has been paid to another potentially fatal haemorrhagic complication, that of cardiac tamponade. We report a case of tamponade following the administration of streptokinase to a patient presenting with severe central chest pain.

Case report

A 42 year old woman with an established diagnosis of recurrent idiopathic pericarditis presented with further central chest pain one week after the withdrawal of non-steroidal anti-inflammatory drugs. On this occasion the patient's pain was reported as significantly more severe than during any previous episode. Radiation of the pain to the left shoulder was described.

Clinical examination was unremarkable. No pericardial rub was audible. Electrocardiography recorded a sinus tachycardia at a rate of 100 beats per minute with widespread concave ST segment elevation. Chest X-ray showed a heart of normal size and contour with clear lung fields. A small pericardial effusion was demonstrated by echocardiography.

A presumptive diagnosis of recurrence of pericarditis was made and the patient admitted for observation. Non-steroidal anti-inflammatory drugs were reintroduced. A cardiac enzyme series was ordered.

On the following morning the patient's pain became much more severe. She was dyspnoeic and nauseated. Though examination revealed no objective deterioration in the patient's clinical state her electrocardiogram revealed new T wave inversion in the anterior leads. Concerned that she was suffering a myocardial infarction, streptokinase was administered. Two hours later the patient became hypotensive. She had a tachycardia, an elevated jugular venous pressure and quiet heart sounds. Urgent echocardiography was arranged. A large pericardial effusion was demonstrated and the diagnosis of cardiac tamponade made. Pericardiocentesis with the drainage of several litres of haemorrhagic fluid followed.

With the correction of her streptokinase-induced clotting disorder the patient made a steady complete recovery. Cardiac enzyme assay results that were later available demonstrated no sequential rise suggesting that no ischaemic damage had in fact occurred.

The patient's electrocardiogram has since returned to normal. The patient herself remains well on a low dose of prednisolone.

Discussion

Of the haemorrhagic complications of thrombolysis for acute myocardial infarction, cerebrovascular and gastrointestinal problems feature most strongly in the literature.¹ Only recently have studies focused on the problem of pericardial effusion. One author reported that pericardial effusion as a side effect of thrombolysis *per se* is very rare and possibly no greater than that due to the process of infarction itself.² In another series approximately 1% of patients treated with thrombolytics for acute myocardial infarction suffered from haemorrhagic effusions significant enough to

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require pericardiocentesis. Those patients with large anterior myocardial infarcts and thrombolysed early appeared to be at greatest risk.³ Even in the absence of coronary disease patients may be at risk of developing life-threatening effusions if given thrombolytic drugs.⁴

Our case highlights how the dangers of throm-

bolysis can be exacerbated in the presence of pre-existing pericardial disease. We suggest that a recent history of pericarditis may be a contraindication to thrombolysis. If the differential diagnosis includes pericarditis then thrombolysis should only proceed with caution. A high level of suspicion for tamponade should be exercised.

References

1. ISIS 2, Second International Study of Infarct Survival: Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected myocardial infarction. *Lancet* 1988, **ii**: 349–360.
2. Belkin, R.N., Mark, D.B., Aronson, L., Szwed, H., Califf, R.M. & Kisslo, J. Pericardial effusion after intravenous recombinant tissue type plasminogen activator for acute myocardial infarction. *Am J Cardiol* 1991, **67**: 496–500.
3. Renkin, J., de Bruyne, B., Benit, E., Joris, J.M., Carlier, M. & Col, J. Cardiac tamponade early after thrombolysis for acute myocardial infarction: a rare but not reported haemorrhagic complication. *J Am Coll Cardiol* 1991, **17**: 280–285.
4. Blankenship, J.C. & Almquist, A.K. Cardiovascular complications of thrombolytic therapy in patients with a mistaken diagnosis of acute myocardial infarction. *J Am Coll Cardiol* 1989, **14**: 1579–1582.