Spontaneous iliopsoas haemorrhage – an unusual complication of streptokinase therapy

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Summary: We report a case of spontaneous iliopsoas haemorrhage following intravenous streptokinase which serves to remind physicians of the potential dangers of this form of therapy.

Introduction

Thrombolytic drugs have made a major impact on the management of acute myocardial infarction in the last few years. Their use is associated with the most significant reduction in mortality since the introduction of coronary care units in the 1960s. However, if the benefits of this form of therapy are to be realized fully the dangers of its use must also be recognized. If thrombolytic drugs are used unwisely the risks of their use could soon outweigh the benefits. It must be recognized that thrombolytic drugs cause a generalized and profound hypocoagulable state. If they are given to patients with conditions other than acute myocardial infarction, disastrous consequences may ensue. Recent reports have highlighted the dangers of giving thrombolytic drugs to patients with chest pain who have not had a myocardial infarction but have had aortic dissection.1,2 We describe a patient in whom a spontaneous haemorrhage occurred into the iliopsoas muscle following intravenous streptokinase therapy for an acute myocardial infarction.

Case report

A 55 year old man presented with a 6-hour history of chest pain radiating to his left arm. He was sweating and distressed, but there were no abnormal findings on physical examination. An electrocardiograph showed an acute anterior myocardial infarction. He was given 5 mg diamorphine and 12.5 mg prochlorperazine intramuscularly together with 300 mg aspirin orally. Ten minutes later he developed ventricular fibrillation from which he was successfully cardioverted. An infusion of lignocaine was commenced and he was transferred to the coronary care unit where he was given 1.5 million units of streptokinase intravenously over 1 hour, along with 5,000 units calcium heparin subcutaneously and topical glyceryl trinitrate paste. Oral aspirin was continued and subcutaneous heparin 5,000 units 3 times daily.

Forty eight hours after admission a left subconjunctival haemorrhage was noted. He complained of pain in the left groin, and had restricted movement of the left leg. Examination revealed bruising over the lower anterior abdominal wall with a tender mass palpable in the left iliac fossa. There was a fixed flexion deformity of the left hip but neurological examination of both lower limbs was normal. An iliopsoas haemorrhage was suspected and considered spontaneous in the absence of previous trauma or undue exercise involving that muscle. Investigations showed a haemoglobin of 12.5 g/dl compared to 16.7 g/dl on initial presentation. A coagulation screen was normal suggesting reversal by 48 hours of the coagulopathy induced by streptokinase. Computer tomographic scan of the pelvis confirmed a left iliacus muscle bleed (Figure 1). Subcutaneous heparin was discontinued and the patient managed conservatively. Five days later full range of movement was still not possible at the left hip and he required daily physiotherapy to assist walking. He was discharged 9 days after admission on a daily dose of atenolol of 50 mg and aspirin 300 mg and was asymptomatic with full mobility on outpatient review one month later.

Discussion

Parenteral administration of streptokinase produces a profound systemic hypocoagulable state characterized by activation of plasmin and defibrination. Despite these marked changes haemorrhagic complications are rare. In the 2 major trials of streptokinase in acute myocardial infarction14

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Accepted: 25 April 1990
Nearly 15,000 patients were given intravenous streptokinase. Minor bleeds (for example oozing from puncture sites, microscopic haematuria) were reported in 3.6% of cases and major bleeds requiring blood transfusion in 0.5%. Bleeding complications were not more common in those taking aspirin but were significantly more frequent in patients who also received heparin by the subcutaneous or intravenous route. However, patient selection for these trials was strict. Serious haemorrhagic complications which do not require blood transfusion may also occur. There were 10 cases of catastrophic cerebral haemorrhage complicating streptokinase treatment in ISIS-2 which carried a high in-patient mortality of 90%. Haemorrhage into other sites including spleen, liver, and mediastinum has also been reported. Spontaneous iliopsoas haemorrhage has not been reported with streptokinase given for treatment for acute myocardial infarction but there is one case report of femoral neuropathy complicating streptokinase use for ergotism.

Bleeding into the iliopsoas muscle is uncommon except in patients with coagulopathies. Haemophiliacs may develop such haematomas and its occurrence during anticoagulation treatment has been recognized since 1966. The importance of this haemorrhage lies in the potential to cause a partial or complete femoral neuropathy. Clinical diagnosis can be supported by ultrasound or computed tomographic scanning as in our case. Management is usually conservative with a 75% chance of full recovery; however, decompressive surgery has been advocated and performed.

References

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doi: 10.1136/pgmj.66.780.862