Myocardial infarction following sternal surgery

RK Aggarwal, WL Morrison

Summary
We report a case of myocardial infarction in a 32-year-old man undergoing sternal surgery. Thrombotic occlusion of the right coronary artery with no underlying atheromatous disease was demonstrated angiographically and successfully treated with intracoronary thrombolysis.

Keywords: myocardial infarction, sternal surgery

Myocardial infarction following blunt chest trauma is a recognised phenomenon. We describe a case of myocardial infarction secondary to angiographically proven thrombotic occlusion of a normal right coronary artery in a patient undergoing surgical debridement of a chronic sternal sinus. To our knowledge, this is the first report describing spontaneous coronary thrombosis following the trauma of sternal surgery.

Case report
A 32-year-old man was admitted for surgical debridement of a chronically discharging sternal sinus. Eight months earlier, he had undergone repair of a proximal ascending aortic dissection using a collagen-impregnated dacron tube graft. At that time he was found to have untreated hypertension. Following uneventful surgical debridement of the sternal sinus, he developed profound bradycardia and chest pain during recovery from general anaesthesia. Twelve-lead electrocardiogram demonstrated acute inferior wall myocardial infarction with complete atroventricular block. Coronary angiography demonstrated an occluded dominant right coronary artery with thrombus along its entire length (figure 1); the left coronary artery was normal. He was given 25 mg of recombinant tissue-type plasminogen activator (rtPA), infused over 10 minutes directly into the right coronary artery, which resulted in almost total lysis of the thrombus after 60 minutes. No underlying obstructive lesion or intimal tear was seen on repeat angiography (figure 2). His clinical course thereafter was uncomplicated. The right coronary artery was shown to be patent at repeat angiography a year later.

Summary points
- thrombotic coronary artery occlusion in the absence of underlying atheromatous disease may cause myocardial infarction following non-penetrating chest trauma
- early coronary arteriography and thrombolysis should be considered in patients presenting with features of acute myocardial infarction, even after relatively modest blunt chest trauma

The Cardiothoracic Centre, Liverpool L14 3PE, UK
RK Aggarwal
WL Morrison

Correspondence to
Dr RK Aggarwal, Division of Cardiology, Faculty of Medicine, University of Leicester, Clinical Sciences Wing, Glenfield General Hospital, Leicester LE3 9QP, UK

Accepted 13 November 1995

Figure 1 Right coronary arteriogram showing thrombus (filling defects indicated by arrows) along its entire length

Figure 2 Right coronary arteriogram 1 hour after infusion of intracoronary rtPA, showing successful lysis of the thrombus
Discussion

Direct damage to coronary arteries (intimal tears, atheromatous plaque fissuring or coronary artery rupture) can occur following non-penetrating chest trauma. In general, the trauma is of severe magnitude; reports in the literature most commonly describe cases involved in road traffic accidents. In most cases underlying atheromatous coronary artery disease is presumed to be present, since angiographic imaging is generally not available. Where no atheromatous disease is evident, arterial occlusion resulting from intimal tears has been described. Cases where angiography is undertaken immediately after initial presentation appear to be rare. This case is unusual in that the chest trauma was of considerably smaller magnitude than is usually the case and occurred in a 'controlled' operative situation. Furthermore, coronary arteriography was undertaken within 30 minutes of the onset of symptoms and revealed extensive thrombus within the right coronary artery.

Thrombus formation in otherwise normal coronary arteries secondary to catecholamine-mediated spasm is a rarely described phenomenon, but is unlikely to have been a factor in our patient since no electrocardiographic ST segment changes were apparent during the course of his general anaesthesia. Microscopic intimal tears, not visible on angiography, may have precipitated thrombus formation in this case. The relatively low magnitude of chest trauma suggests that this phenomenon may be a contributory factor in some cases of perioperative myocardial infarction following cardiothoracic surgery. Thus in patients presenting with electrocardiographic features of acute myocardial infarction following blunt chest trauma (including 'controlled' surgical trauma) early coronary angiography should be considered.

Primary Sjogren's syndrome, ulcerative colitis and selective IgA deficiency

A Steuer, DJ McCrea, CB Colaco

Summary

A 24-year-old man with primary Sjogren's syndrome presented with xerophthalmia, xerostomia, and marked parotid swelling. He had a previous history of selective IgA deficiency and ulcerative colitis treated with sulphasalazine. Immunosuppression and withdrawal of sulphasalazine resulted in rapid resolution of the parotitis and disappearance of autoantibodies. A possible role for sulphasalazine in the induction of autoimmunity in this case is discussed.

Keywords: Sjogren's syndrome, ulcerative colitis, IgA deficiency

Primary Sjogren's syndrome is a chronic autoimmune disease of unknown aetiology predominantly affecting women in middle age. Ulcerative colitis has been rarely reported in association with Sjogren's syndrome, however both conditions may occur more often in the presence of selective IgA deficiency. We report a patient with ulcerative colitis who developed Sjogren's syndrome in the presence of selective IgA deficiency whilst on long-term sulphasalazine treatment and suggest a role for the sulphasalazine in the development of autoimmunity.

Case report

An 18-year-old Asian man presented in April 1988 with a haemorrhagic colitis. Routine haematological and biochemical profiles were normal, though the erythrocyte sedimentation rate (ESR) was raised at 65 mm/h. Colonic biopsy confirmed the typical histological features of ulcerative colitis. The patient was treated with low-dose steroids which were withdrawn after six months and sulphasalazine 1 g bid. He maintained a reasonable remission with only occasional flares of active colitis.

In July 1994 he presented with swollen parotid, submandibular and lacrimal glands, widespread cervical lymphadenopathy, xerostomia, xerophthalmia and myalgia. Schirmer's test (strips for measuring tear production) was dry (right eye 4 mm, left 6 mm after five