Case reports

Postpartum myocardial infarction

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SUMMARY A 29 year old woman had a myocardial infarction three weeks post partum. Coronary angiography was performed six days later. No abnormalities were seen initially, but re-injection of the left coronary artery resulted in a dissection that extended through the anterior descending and circumflex branches and a reinfarction.

This case suggests that myocardial infarctions occurring in patients with angiographically normal coronary arteries may be caused by dissections that heal by the time of catheterisation.

Myocardial infarctions are uncommon in young women, and only a small percentage occur during pregnancy, labour, or soon after delivery. The incidence is estimated at one per 10 000 deliveries,¹ and a recent review noted only 75 published reports of such cases.² The number of reported myocardial infarctions occurring in the early postpartum period is considerably smaller.

We report the case of a myocardial infarction occurring post partum in a 29 year old woman. We believe that this may have resulted from spontaneous dissection of the left coronary artery.

Case report

A 29 year old woman was admitted to hospital after the sudden onset of substernal pain and pressure three weeks post partum. The initial electrocardiogram showed deep ST segment depression in the inferior and precordial leads, and serum concentrations of creatine kinase rose to 2910 IU/l with 12.8% MB isotype. The ST segment depression resolved, and T wave inversion was subsequently noted in leads I, aVL, and V2-V6. The patient had no heart failure or arrhythmias, but had a brief episode of chest pain two days after admission without any

increase in the titre of serum creatine kinase MB. An echocardiogram done at this time showed apical akinesis and the presence of a large apical thrombus. The patient was treated with intravenous heparin and oral diltiazem.

Coronary angiography was performed six days after the onset of chest pain. The right coronary artery was normal, and initial views of the left coronary system showed no abnormalities (fig a). After the fourth injection into the left coronary artery, however, the patient complained of chest pain. Retention of contrast agent in the left coronary artery adjacent to the ostium was noted, and a re-injection of contrast agent showed dissection of the left coronary artery extending to the proximal and middle portions of the left anterior descending artery and the proximal portion of the circumflex artery (fig b). The patient's electrocardiogram showed ST segment elevation in leads I, aVL, and V2-V6. The patient was treated with intravenous glyceryl trinitrate. On repeat re-injection, the left coronary artery and all obvious branches were seen to be patent, but the diameter of the vessel lumens appeared to be reduced, and a "double lumen" remained evident in the circumflex artery (fig c).

The patient was treated subsequently with oral diltiazem and isosorbide dinitrate. Heparin, which had been stopped before catheterisation, was not restarted. The patient's serum creatine kinase titre rose to 3817 IU/l with 23.6% MB isotype. After resolution of the pain over the next 24 hours, the

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Figure (a) Left coronary artery in shallow right anterior oblique projection. (b) Left coronary artery in caudal angulated right anterior oblique projection. Arrows indicate dissection of the anterior descending and circumflex arteries. (c) Left coronary artery in caudal angulated right anterior oblique projection.

patient recovered without any episodes of heart failure. She had an episode of chest pain two days after coronary angiography that was accompanied by ST segment elevation and a junctional rhythm. The pain was relieved by a sublingual glyceryl trinitrate, and no increase in serum creatine kinase titre was noted. An echocardiogram obtained one day later was not significantly different from that obtained before angiography. The patient was discharged nine days after angiography on diltiazem and isosorbide dinitrate, with warfarin added two weeks later. Six months after the infarction the patient required a heart transplant for progressive heart failure. Microscopic examination confirmed the presence of a dissection in the left anterior descending artery. This was not visible on gross examination.

The patient had no history of symptoms suggestive of coronary artery disease, and no risk factors were identified. All of her pregnancies and deliveries had been normal.

Discussion

Primary coronary artery dissections are rare. Since the first report in 1931,³ there have been about 60 reported cases⁴⁻⁶—about 80% of them in women and a quarter of these occurred during the third trimester of pregnancy or the early postpartum period.⁴ The diagnosis is usually made post mortem; only eight cases were diagnosed via coronary angiography and only nine patients survived.⁴⁻⁶

The aetiology of primary coronary artery dissections is unknown. Most commonly the left coronary artery, and more specifically its anterior descending branch, is affected. Dissection typically arises within 2 cm of the left coronary ostium.⁷ Haematomas are usually confined to the media without evidence of an intimal tear. Atherosclerosis of the affected vessel is generally slight or absent. Medial necrosis and infiltration of the adventitia with inflammatory cells consisting primarily of eosinophils were seen on

histopathological examination.^{8,9} In one case, collagen metabolism was abnormal.¹⁰ The association of primary coronary artery dissections with peripartum and postpartum states suggests that hormonal changes and haemodynamic stresses related to pregnancy, labour, and delivery may in some way lead to a loss of structural integrity of the blood vessel wall.

We describe a myocardial infarction three weeks post partum in a 29 year old woman whose coronary arteries initially appeared normal on angiography. Most of such infarctions have been attributed by default to spasm, embolism, or thrombosis with subsequent clot lysis. In our case, however, the demonstration of dissection of the left coronary artery during catheterisation raises the possibility that the infarction five days earlier was the result of a primary coronary dissection. We cannot be certain that this was true, but we suggest that the patient's original dissection had healed incompletely, and that dye injection caused a redissection in the weakened vessel wall.

Our experience with this case has led us to suspect that many myocardial infarctions in patients with angiographically normal coronary arteries resulted from dissections that have healed by the time of angiography. There are three reports of myocardial infarctions during pregnancy in which there was no evidence of vessel obstruction at coronary angiography several months later.¹¹⁻¹³ In one, however, a small aneurysm was noted at the origin of the left anterior descending artery,¹¹ and in the remaining two cases there was tapering of the left anterior descending artery and attenuation of its branches.^{12,13} These findings raise the suspicion of dissection in all of these cases. In addition, angiography may be unable to detect those coronary dissections that occur without evidence of intimal tear. In such cases, the intraluminal coronary pressure may be sufficient to maintain the patency of the affected vessel, with intramural displacement extending from the media toward the adventitia rather than the intima. A patient in whom an angiographic coronary obstruction was ascribed to atherosclerosis was found during planned bypass surgery to have an intramural haematoma in the affected vessel.⁴ Evacuation of the haematoma successfully relieved the obstruction.

Our case suggests that the timing of coronary angiography after peripartum or postpartum myocardial infarctions may be crucial. We performed angiography in the early recovery period because the patient had persistent pain, diffuse ST segment

changes, and no Q waves. These findings could have been the result of premature coronary atherosclerosis (possibly of the left main coronary artery), which made reinfarction likely. In this case early angiography itself resulted in a new dissection and reinfarction. One case cannot form the basis for a policy, but in view of the risk of this complication we wonder whether coronary angiography should be deferred for several weeks or months after uncomplicated peripartum and postpartum infarctions.

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