CASE REPORT

Atheroembolism in an endomyocardial biopsy from a cardiac transplant recipient after coronary angioplasty

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Abstract

A case of a coronary atheroembolism after coronary angioplasty was detected by routine myocardial biopsy six years after cardiac transplant. The patient had had three balloon angioplasties within a five week interval for symptomatic accelerated coronary artery disease. Histological examination clearly identified atheromatous debris in a small intramyocardial artery with a secondary inflammatory response. This complication of angioplasty has been identified at necropsy in association with new myocardial infarction. It is unusual to identify this complication in a survivor, and in a case in which there was no clinical evidence of myocardial infarction. Myocardial biopsy samples a relatively small amount of myocardium, and this case suggests that coronary atheroembolism may not be an uncommon complication of angioplasty.

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A recent necropsy study showed that atheroembolism was common in small intramyocardial arteries after coronary interventions: its incidence in survivors is unknown.¹ To our knowledge, this is the first report of an intramyocardial artery atheroembolism found in an endomyocardial biopsy specimen from a surviving patient after percutaneous transluminal coronary angioplasty.

A 45 year old man underwent cardiac transplantation for ischaemic cardiomyopathy. He received the heart from a 20 year old woman, and managed well in the early phase post-transplant. He had important risk factors for transplant atherosclerosis, including hypercholesterolaemia, systemic hypertension, and clinical cytomegalovirus infection. Five years after transplant, exertional dyspnoea developed and coronary angiography showed complete occlusion of a large first diagonal branch and other areas of luminal irregularity, including a 60% obstruction of the mid left anterior descending coronary

artery. Five months later the patient sustained an inferior acute myocardial infarction complicated by right and left ventricular failure and post-infarction angina. Thallium scintigraphy showed reversible anterior ischaemia. Repeat coronary angiography showed significant progression of disease with a new 80% obstruction in the proximal right coronary artery, a complete obstruction at the origin of the posterior descending artery, progression to 95% stenosis of the mid left anterior descending lesion, and a new proximal left anterior descending lesion. One week later the left anterior descending lesions and the complete occlusion of the first diagonal were subjected to balloon angioplasty. The angina initially improved but recurred. Repeat angiography four weeks later showed occlusion caused by the lesion in the mid left anterior descending coronary artery. A large acute marginal branch of the right coronary artery penetrated into the interventricular groove, and the proximal right coronary artery lesion was dilated. Angina persisted and two weeks later a repeat balloon angioplasty of the lesion in the mid left anterior descending coronary artery was performed. This was followed by clinical improvement. Three and half weeks after the last balloon angioplasty the patient had a routine right ventricular biopsy. There was no evidence of rejection, however, an atheromatous embolus was seen in a small intramyocardial coronary artery (figure). Its age could not be established, but had been present long enough for an inflammatory reaction associated with the crystalline material to develop in the lesion.

Manipulations of atherosclerotic arteries may cause atheroembolism in different organs as a result of plaque rupture or dislodgment of atheromatous debris from the vessel surface. In a retrospective study atheroemboli were found in intramyocardial arteries in 19% of necropsy cases when coronary angiography was performed.² A recent report described coronary embolisation in 76% of patients after balloon angioplasty who died within three months of balloon angioplasty, this was associated with multiple interventions, intervention in the left anterior descending coronary artery, medial dissection, and plaque rupture or extrusion.¹ Clinically, the patients were more likely to develop new electro-

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Atheromatous embolus in small intramyocardial artery in an endomyocardial biopsy specimen. Combined Verhoeff's elastic and Masson's trichrome stain; magnification $\times 100$.

cardiographic changes, infarct extension, and new myocardial infarction than a control group who died of myocardial infarction with no previous coronary intervention.¹ Previous reports have also described embolic complications after balloon angioplasty of bypass grafts.³

To our knowledge, this is first report of coronary embolisation detected in an endomyocardial biopsy specimen from a surviving patient who had balloon angioplasty. This patient had multiple interventions for accelerated atherosclerosis after transplant. Although this disease is considered mostly a concentric and diffuse process, an important eccentric morphological component is often present, which may be related to superimposed atherosclerosis.⁴ Its clinical manifestations may include angina, myocardial infarction, and sudden death, and coronary interventions to treat these lesions have a similar risk of complications as conventional atherosclerotic plaques.⁵

In our case, the patient had accelerated progression of his coronary disease as judged by his last routine examination by coronary angiography and the occurrence of myocardial infarction five months later. After infarction severe eccentric disease of several coronary vessels was seen. This required

intervention because it caused angina. The patient underwent three balloon angioplasties within a five week interval. It is difficult to ascertain which intervention caused the embolus that was seen in the biopsy specimen. Judging by the coronary anatomy and possible location of the biopsy site, we believe that either the right coronary artery or left anterior descending artery was the source. The clinical consequences of coronary embolism depend both on size and number of emboli, and they include acute myocardial ischaemia or infarction, ventricular arrhythmias or sudden death.¹ Ventricular dysfunction also may be caused by myocyte damage secondary to the perivascular inflammation that is usually associated with atheromatous emboli.⁶ This patient had already presented with right and left ventricular failure after infarction and it is unclear whether ventricular function deteriorated after balloon angioplasty.

In summary, diagnosis of coronary embolisation after coronary interventions is difficult in surviving patients. However, its detection by endomyocardial biopsy, as reported here, which samples only a small amount of myocardium, suggests that, as indicated by previous necropsy series,¹ this is not an uncommon complication especially in patients who undergo several balloon angioplasty procedures. Its clinical relevance in survivors is unclear.

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