INTRODUCTION

Coronary artery disease is the major cause of mortality and morbidity worldwide. Approximately 3.8 million men and 3.4 million women die of the disease each year. Reperfusion after acute coronary event, either by thrombolytic therapy or by primary percutaneous coronary intervention, helps to reduce the size of the myocardial infarct and prevents necrosis by restoring the blood flow to the ischemic myocardium. However, restoration of blood flow is not always beneficial and does not always guarantee myocardial salvage. It can lead to a phenomenon called “reperfusion injury”. This is thought to be due to the production of free oxygen radicals following re-oxygenation of the ischemic myocardial tissue. In most cases, the reperfusion injury is minor with no clinical effects but full-blown myocardial infarction, along with its complications, can occur.

In this case, disobliteration of infarct-related coronary artery resulted in ventricular septal defect secondary to reperfusion injury.

CASE REPORT

A man aged 63 years was admitted to the hospital after having sustained an inferior myocardial infarction. During the admission, his peak Creatinine Kinase (CK) rise was 2300 IU/l. He was treated with Tissue Plasminogen Activator (TPA) given via accelerated regimen and concurrently intravenous nitrates and heparin. He continued to have unstable angina over the ensuing 2 days without resolution of the ECG changes. Risk factors included hypercholesterolaemia, hypertension and a strong family history. He remained haemodynamically stable throughout.

He was referred for urgent coronary angiography which showed complete obstruction of the right coronary artery with retrograde filling from the left system which was not obstructed (Figure 1a). Disobliteration and stenting of the right coronary artery was performed with good angiographic results (Figure 1b). Distal flow was normal (TIMI grade 3) immediately following recanalisation. Six hours after the procedure, he developed chest pain, and exhibited cardiogenic shock (tachycardia at 122/minute and blood pressure of 96/60 mmHg). He was noted to have a new pansystolic murmur with rapidly progressive congestive cardiac failure and renal dysfunction. This was associated with a rise of troponin-I of 5.5 µg/L 8 hours after stenting from a pre-stenting value of 1.9 µg/L (Figure 2). A transthoracic echocardiogram confirmed a mid septal ventricular septal defect measuring 1.6 cm and hyperdynamic ventricles with dilatation of the right ventricle.

ABSTRACT

Reperfusion injury is thought to occur during coronary recanalisation but rarely produces clinically significant effects other than arrhythmia. We report an unusual case of Ventricular Septal Defect (VSD) developing after successful disobliteration of the right coronary artery. In this case clinical, electrocardiographic and biochemical evidence of myocardial injury developed 6 hours after successful percutaneous recanalization of the infarct related artery. A rapidly developing VSD soon became apparent necessitating surgical intervention to repair the defect. Unfortunately the patient died soon after surgery.

Key words: Reperfusion. Myocardial infarction. Ventricular septal defect. Coronary artery disobliteration.

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Figure 1(a): RCA: pre-stenting, (b): RCA: post-stenting.
Despite mechanical and pharmacological support the patient's condition continued to deteriorate and then he was referred for surgery. At surgery, a large (4x3 cm) ventricular septal defect was closed with a PTFE patch (Figure 3). He came off by-pass on a high dose of inotropic support but unfortunately died of intractable biventricular failure. Postmortem showed patent right coronary artery with no evidence of acute thrombosis but necrotic myocardium of the septum around the patch.

DISCUSSION

Tennant and Wiggers in 1935 described the arrhythmias associated with the reintroduction of previously obliterated coronary blood flow. Almost 36% of patients undergoing primary angioplasty will experience reperfusion injury. Reperfusion injury has three distinct components: reperfusion arrhythmias, myocardial stunning and lethal myocyte injury. Intra-coronary thrombolysis and primary coronary angioplasty are associated with reperfusion arrhythmias. Grines et al., demonstrated in a randomized clinical study, that ventricular fibrillation was significantly more common in the primary coronary angioplasty group as compared to the intravenous thrombolytic treatment group. Although severe arrhythmias are not common after reperfusion injury, they are associated with significant mortality if they do occur. Myocardial stunning, on the other hand, is a reversible phenomenon with very little clinical significance. Bolli et al. and Kloner stated, in their clinical review, that myocardial stunning may actually delay the recovery from cardiogenic shock after myocardial infarction. The third component of reperfusion injury is lethal myocyte injury. Its clinical relevance is, however, debatable. It is thought to be due to the destruction of reversibly damaged myocardial cells by reperfusion.

The pathogenesis of reperfusion injury is still unclear and various etiological factors have been implicated. These factors include: tissue damage caused by free oxygen radical, complement system activation, calcium ion influx, overstimulation of neural pathways and cytokine-chemokine induction and secretion via toll-like receptor pathway.

Ventricular septal defect, as a complication of myocardial infarction, is a well known phenomenon. It occurs in approximately 1% of survivors of myocardial infarction. However, VSD occurring after successful disobliteration of a completely occluded coronary artery has not yet been reported in medical literature. The time interval between infarction and the onset of VSD varies from a few hours to 15 days. Mortality is highest for those in cardiogenic shock and for inferiorly located VSD after right coronary occlusion.

Controversy still exists as to whether the ultimate survival or death of myocardial tissue is dependent solely on events during ischaemia, reperfusion injury, no-reflow phenomenon or all. Several studies, have demonstrated that angiographically successful recanalization of the infarct related artery does not necessarily guarantee myocardial salvage in patients. In this case the patient developed ischaemic chest pain, increasing ST segment elevation and the steep rise in troponin-I after disobliteration of the RCA suggest that reperfusion injury may have contributed to the development of the VSD. Disobliteration of the right coronary artery may have liberated free radicals thereby accelerating death in vulnerable ischaemic myocytes. VSD may have developed irrespective of any intervention, as is the natural history of myocardial infarction.

This case illustrates that a good angiographic result, following disobliteration of a coronary artery, may not protect from the consequences of coronary artery occlusion. It also suggests that myocyte injury, due to reperfusion phenomenon, may be precipitated by this treatment.

REFERENCES


Ventricular septal defect following disobliteration of right coronary artery