Case report

Dissecting sub-epicardial hematoma—challenges to surgical management

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Abstract

A 67-year-old man developed a large dissecting sub-epicardial hematoma of his heart following a percutaneous coronary interventional procedure. While undergoing a dilatation of in-stent restenosis of a saphenous vein graft to the diagonal artery, he developed an anastomotic perforation that lead to a large sub-epicardial hematoma that sheared off all the epicardial vessels from the underlying myocardium. Emergent surgery was performed as he began to evolve a large myocardial infarction. Evacuation of the hematoma and gluing back of the epicardium was the only operation possible due to the complexity of the problem. Deteriorating hemodynamics post-operatively led to the placement of a percutaneous ventricular assist device despite which he succumbed. This case report is intended to alert the surgical community to such a rare complication of aggressive percutaneous intervention, difficulty in establishing an accurate diagnosis and repairing the sub-epicardial dissecting hematoma.

Keywords: Cardiac catheterization; Sub-epicardial hematoma

1. Introduction

We present our experience with this rare complication of percutaneous coronary intervention, focusing upon the surgical management and outcome.

2. Case report

The patient was a 67-year-old man with hypertension, diabetes and a long history of coronary artery disease. He underwent coronary artery bypass 10 years ago with saphenous vein grafts (SVG) to the diagonal, obtuse marginal and right coronary artery. He had undergone successful stenting of the SVG to diagonal 5 months ago.

He presented with recurrent angina and dyspnea. Catheterization showed occluded native circumflex, right coronary arteries, grafts to the right coronary artery and obtuse marginal. There was diffuse in-stent restenosis of the SVG to diagonal and 40% stenosis in the native left anterior descending artery (LAD). The in-stent restenosis was dilated successively. The final dilatation with a cutting balloon created an anastomotic perforation with extravasation of dye into a small confined space (Fig. 1A). The leak was controlled with balloon inflation and reversal of heparin to an activated clotting time (ACT) to 131 s.

Flow was sluggish but the extravasation was halted. Thrombosis in the body of the SVG, causing chest pain and bradycardia, was treated with catheter thrombectomy, heparin (ACT 248) and proximal stenting. Transthoracic echocardiogram (TTE) showed a collection along the lateral wall, a presumed contained pericardial hematoma without tamponade. Repeat angiography showed TIMI-2 flow, no leak and a patent graft (Fig. 1B). An intra-aortic balloon pump (IABP) was placed.

In the coronary care unit, a repeat TTE showed localized collection without expansion. For persistent EKG changes suggestive of anteroseptal ischemia beyond the territory of the diagonal, he underwent repeat angiography that showed filling of the proximal LAD and SVG to diagonal; in both, the flow was sluggish with no distal runoff. The leak remained sealed.
Emergent surgery was performed for evacuation of a presumed intra-pericardial hematoma. It was felt to be compressing the epicardial LAD distally as well as the diagonal beyond the anastomosis, creating ischemia by tamponade effect.

The heart was approached through a left thoracotomy and ante-phrenic pericardiotomy. There was no blood or clot in the pericardial space, which was loosely but diffusely fused to the heart by adhesions. The entire visible epicardial surface of the left ventricle was blue and taut. On incising the epicardium, a large sub-epicardial hematoma was evacuated. It had dissected the epicardium and the epicardial vessels of the underlying myocardium over a broad area. The perforating arteries and veins were sheared off at entry into the muscle and the epicardial ends were bleeding severely. No major arteries could be identified.

Digital exploration of the emptied hematoma cavity revealed extensive disconnection of the epicardial LAD from the underlying anterior interventricular myocardium precluding surgical revascularization. The sub-epicardial hematoma dissected posteriorly as well. The progression/expansion of the hematoma was fed by bleeding from avulsed arteries and veins.

The ischemic anterolateral wall could not be revascularized. Cautery could not achieve hemostasis. Biogluè® (Cryolife International, Kennesaw, GA) was applied to the inside of the evacuated hematoma cavity and the epicardium was ‘glued back’ on the myocardium.

He needed high dose inotropic pressor and intra-aortic balloon pump support. Inotropic support decreased to moderate levels in the first 48 h as he evolved a large myocardial infarction. Multi-organ dysfunction occurred. As he showed no further improvement in cardiac function from an ejection fraction of 10%, he was considered for ventricular assist device (VAD) support as an outside chance for bridge to transplant. It was felt that he would not survive at any traditional surgically implanted VAD procedure. He received a percutaneous LVAD (Tandem Heart®, Cardiac Assist Inc., Pittsburgh, PA) per the nonrandomized phase of a clinical trial. Despite adequate flow rates (3.5 l/min), he did not sustain significant recovery of organ function, including neurologic function. Progressive limb ischemia occurred despite antegrade leg perfusion and rhabdomyolysis added further renal insult. His family opted for withdrawal of care and he succumbed promptly.

A full autopsy was carried out. Examination of the heart revealed the gross findings of extensive sub-epicardial dissecting hematoma (Fig. 2A and B). The sectioned specimen revealed extensive hemorrhagic infarction in addition to the dissecting hematoma (Fig. 2C).

2. Comments

We report an unusual complication of percutaneous coronary angioplasty that produced severe and extensive infarction of the left ventricle due to a relentless, self-propagating sub-epicardial dissection of the heart.

Although the rate of perforations is low after routine angioplasty (0.2–0.6%), the risk of these perforations occurring after newer percutaneous intervention techniques is higher (0.5–3.0%) [1]. These perforations are graded on a scale of 1–3 [2]. Grades 1 and 2 perforations can usually be handled by reversal of anticoagulation and percutaneous techniques. Grade 3 perforations are described as at least
1 mm across and may cause significant bleeding resulting in cardiac tamponade and death without surgical intervention. However, a sub-epicardial dissection has not been included in these grades.

Rehders et al. [3] possibly reported the first case in 1993 where a sub-epicardial bleed from a leaking LAD produced significant anterior ischemia requiring emergency CABG—left internal mammary artery to LAD. Furushima et al. [4] reported a case in 1997 when direct surgical suture control was needed to fix a bleeding obtuse marginal artery following angioplasty. Their patient had a small sub-epicardial hematoma that was producing localized compression and ischemia. Misfield et al. [5] reported a similar case in 2001 wherein they needed to evacuate the sub-epicardial hematoma in order to relieve ischemia without any additional suture techniques. In all cases, combinations of echocardiographic and/or angiographic aids were used to diagnose the problem along with other monitoring devices.

Our case differs significantly from previously reported cases. A sub-epicardial hematoma was caused by a large tear in the SVG. This broad-based dissecting hematoma avulsed perforating myocardial coronary branches producing more ischemia and was self-propagating by more bleeding caused by the severed arteries. This lead to a large anterolateral infarct that led onto the cascade of events that caused his death. The pathology and the extensive nature of the infarction were such that no other modality of treatment was considered at that time. In hindsight, other therapeutic modalities for consideration would be immediate placement of a percutaneous LVAD or extra corporeal life support. The need for anticoagulation for these devices adds hemorrhagic risks to a patient already compromised by a bleeding complication.
References


