1. Introduction

Coronary artery spasm is defined as a reversible coronary stenosis that limits coronary blood flow. Per-operative coronary spasm is a potentially life-threatening complication of cardiac surgery. Most published cases occurred following coronary artery bypass surgery, its occurrence after valve replacement is uncommon [1, 2].

We report a case of right coronary artery vasospasm, with profound electrical and hemodynamic instability, after aortic valve replacement (AVR).

2. Case report

A 62-year-old woman with symptomatic severe aortic regurgitation (NYHA class III) was admitted for elective AVR. There was no history of angina pectoris or of any other important clinical problem in the past. Preoperative cardiac catheterization confirmed important aortic regurgitation with normal left ventricular function. Coronary angiography showed no lesions. Aortic valve was replaced by a 19-mm mechanical prosthesis (St Jude Medical); thoracic aorta cross-clamping lasted 60 min.

On admission at ICU, ST segment elevation occurred and was followed by transitory complete A-V block, pronounced hemodynamic instability and ventricular fibrillation. Percutaneous cardiopulmonary support was immediately started and an emergent transesophageal echocardiography (TEE) showed no signs of mechanical prostheses dysfunction or evidence of myocardial impairment. Since marked hemodynamic and electrical instability persisted, coronary angiography was done that showed a pronounced spasm of the distal portion of the dominant right coronary artery. Intravenous isosorbide dinitrate was promptly administered. Concomitantly, compression of the vessel wall by a chest drain tube was observed (Fig. 1a). The pericardial drainage tube was removed with relief of coronary vasospasm (Fig. 1b). Intra-aortic balloon counterpulsation was started and intravenous isosorbide dinitrate was maintained for 48 h. The remainder of the postoperative course was uneventful. There was no evidence of myocardial infarction (EKG, Enzymes). Pre discharge evaluation (TEE, coronary angiography) showed normal aortic prosthesis, left ventricular functions and coronary perfusion (Fig. 2). The patient was discharged on the 11th postoperative day under nitrates and calcium-channel blocker. Two-year follow-up evolved without angina or cardiovascular events.

3. Discussion

Coronary artery spasm is an abnormal transient contraction of a segment of an epicardial artery resulting in myocardial ischemia. The mechanism of spasm remains uncertain, but is considered to be multifactorial including the autonomic nervous system, platelet aggregation, vascular endothelium, among others. Endothelin and nitric oxide have been implicated in the control of vascular tone, and their activity seems to be impaired in the coronary arteries of patients who develop spasm. Recently, some genetic factors have been implicated [3].

Postoperative coronary arterial spasm has been associated to coronary artery trauma during surgical manipulation, compression by chest drain tubes, alkalosis, low body temperature and release of vasospastic factors by platelets damage during cardiopulmonary bypass [4]. The manifestations of spasm range from asymptomatic ST elevation to hypotension and circulatory collapse, related to arrhythmias or myocardial injury. Most cases have been reported in Japanese patients and during coronary artery bypass graft. Previous reports of postoperative coronary...
Fig. 1. Pronounced spasm of the distal portion of the dominant right coronary artery, apparently related to compression of the vessel wall by a chest drain tube (arrow) (a). Intracoronary isosorbide dinitrate was administered, the pericardial drainage tube removed, and coronary vasospasm was relieved (b).

In conclusion, this case shows that coronary spasm should be considered as a cause of unexplained electrical or hemodynamic instability after cardiac surgery and adequate attitudes should be promptly undertaken.

References


ICVTS on-line discussion A

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eComment: I read with great interest the article of Pinho et al. [1] in which they speculate that the coronary artery spasm should be considered as a cause of unexplained electrical or hemodynamic instability. Be that as it may, there are some physiological issues that cause one to pause regarding the findings in this case report.

Ischemic or myocardial preconditioning [IPC] is a phenomenon whereby brief intervals of sublethal ischemia either delay or reduce the extent of necrosis following a subsequent more prolonged episode of ischemia. Coronary vasospasm may be provoked by a supersensitivity of the vascular smooth muscle cells. In the vasculature, ATP-sensitive potassium [KATP] channels regulate vascular tone. The channel is composed of a pore-forming unit [Kir6.x] and a regulatory subunit, the sulfonylurea receptor [SUR-alternatively spliced form SUR2]. In the cardiovascular system, Kir6 and SUR2 constitute the major KATP channels of the heart [2]. There is evidence