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Right coronary artery spasms triggered by mechanical compression during off-pump coronary artery bypass grafting surgery

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Abstract
In this case report, we describe recurrent spasms of the right coronary artery with no apparent preexisting abnormal angiogram that caused second-degree atrioventricular (AV) block and inferior wall hypokinesis. These hemodynamic changes were induced by mechanical compression of the right coronary artery by a pericardium drain tube. From our experience, we should be aware that mechanical compression may trigger an exaggerated vasomotor response, leading to severe coronary artery spasms.

Key words OPCABG · Coronary artery spasm · Mechanical compression

Coronary artery spasm has been recognized as a possible cause of perioperative myocardial ischemia during and after coronary artery bypass grafting surgery and can be associated with circulatory collapse and death [1,2]. We report a rare case of spasms of the right coronary artery with no apparent preexisting abnormal angiogram in off-pump coronary artery bypass grafting (OPCABG) that were triggered by mechanical compression by a drain tube, inducing electrocardiographic and transesophageal echocardiographic changes. This case was successfully treated with replacement of the tip of this tube and intravenous diltiazem.

Case report

A 69-year-old man, 155 cm in height and 58 kg in body weight, with an abdominal aortic aneurysm was admitted for elective Y-graft replacement surgery. He sometimes presented with angina even at rest. Preoperative cardiac catheterization revealed normal features of the right coronary artery and 90% stenosis of the left anterior descending coronary artery (segment 7) and the left circumflex artery (segment 13). The left ventriculogram showed a normal ejection fraction of 67%. OPCABG surgery was determined to be performed in advance of Y-graft replacement surgery. Under general anesthesia with sevoflurane inhalation (end-tidal concentration of sevoflurane, 1.2%–2.5%) and IV fentanyl (22 $\mu g \cdot kg^{-1}$), a median sternotomy was performed, and the left internal thoracic artery (LITA) and the right gastroepiploic artery (GEA) were dissected and mobilized over their entire length. Heparin 100 $U \cdot kg^{-1}$ was administered intravenously 5 min before distal transection of the LITA, which was a very large conduit with excellent flow characteristics.

Isosorbid dinitrate (0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and nicorandil (0.5 $mg \cdot h^{-1}$) were infused intravenously throughout surgery, and noradrenaline (0.05–0.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was also infused to keep systolic arterial blood pressure at more than 100 mmHg during the graft anastomosis. The patient underwent double CABG: a LITA graft to the left descending coronary artery and a GEA graft to the left circumflex artery. The graft anastomosis was achieved uneventfully, and normal wall motion was assessed by transesophageal echocardiography (TEE) (ProSound SSD-5500SV; Aloka, Tokyo, Japan).

At closure of the sternum, blood pressure and heart rate fell immediately to 65/36 mmHg and 53 bpm, associated with ST-segment elevation over the inferior leads (Fig. 1). Subsequently, bradycardia progressed (HR, 33 bpm), and atrial pacing failure due to the second-degree atrioventricular (AV) block was diagnosed in the ECG (Fig. 2A). M mode on the transgastric view by TEE showed a severe hypokinesis on the inferior wall, and the fraction area change was calculated as about 28%. About 5 min before closure of the sternum, arterial blood gas analysis revealed a normal acid–base balance and electrolyte concentration (pH, 7.39; Pa_{CO_2}, 39.3 mmHg; Pa_{O_2}, 148.2 mmHg; Sa_{O_2}, 99.8%; Na^+,

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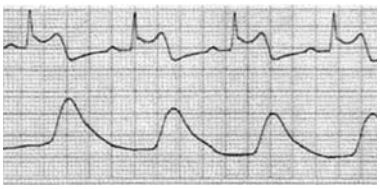

Fig. 1. Electrocardiogram in lead II and pulse wave recorded by pulse oxymeter at closure of the sternum. Heart rate fell immediately to 53 bpm associated with ST-segment elevation in leads II

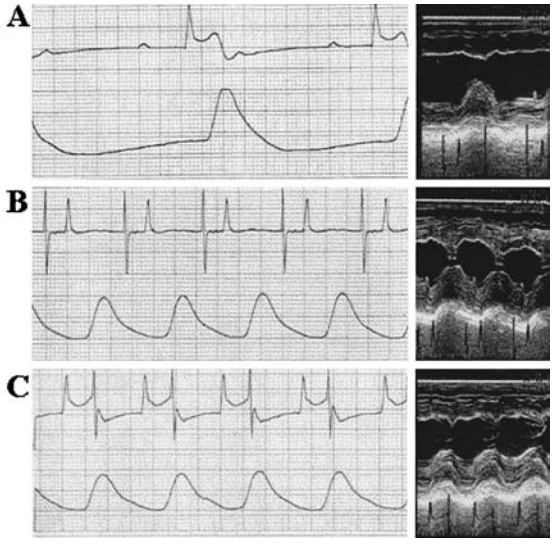

Fig. 2. Electrocardiograms in lead II and left ventricular wall motion in M mode by transesophageal echocardiography (TEE) transgastric view. **A** Bradycardia with second-degree atrioventricular (AV) block progressed (HR, 33 bpm) with severe hypokinesis on the inferior wall. **B** Rapid electrocardiographic improvement occurred with successful atrial pacing, and left ventricle wall motion recovered after reopening the sternum and intravenous infusion of diltiazem. **C** In spite of reopening the sternum, ST-segment elevation with first-degree AV block and severe hypokinesis on the inferior wall occurred

142 mEq/l; K^+, 4.6 mEq/l). According to those findings, we diagnosed spasms of the right coronary artery triggered by closure of the sternum. After reopening the sternum, ventricular pacing was performed to keep HR as 70 bpm, and 2.5 mg diltiazem was injected intravenously followed by continuous infusion ($2.5\,\mu g \cdot kg^{-1} \cdot min^{-1}$). Reopening the sternum and intravenous infusion of diltiazem effectively resolved the coronary artery spasms with rapid electrocardiographic improvement. Simultaneously, the left ventricle wall motion recovered, and atrial pacing started to work effectively (Fig. 2B). However, at 10 min after recovery of hemodynamical electrocardiographic changes, closure of sternum had induced ST-segment elevation over the inferior leads again. In spite of again reopening the sternum, this ST-segment elevation persisted and first-degree A-V block occured. A transgastric view by TEE showed severe hypokinesis on the inferior wall (Fig. 2C). Because hemodynamic and electrocardiographic changes could not be improved several minutes after the sternum was reopened, an additional 2.5 mg diltiazem was injected intravenously. Then, these electrocardiographic changes were resolved and the left ventricular wall motion improved dramatically.

The surgeons realized the possibility that the right coronary artery was mechanically compressed by a drainage tube (Thoracic LCV-UK catheter 5730–10 UE; Tyco Healthcare, Tokyo, Japan) into the pericardium space, and they placed the tip of this tube apart from the right coronary artery. Thereafter, closure of the sternum did not induce ST-segment elevation over the inferior leads. After completion of surgery, the patient was admitted into the intensive care unit. No elevation of creatine kinase MB levels was detected postoperatively, and the patient was extubated 18 h later. The patient's recovery was uneventful thereafter.

Discussion

Although sudden circulatory collapse during coronary artery bypass may be caused by several factors, previous reports have emphasized the importance of an early diagnosis of coronary spasm, which can be the cause of severe, life-threatening hypotension soon after coronary artery revascularization [3,4]. In our case, electrocardiographic and TEE findings consistently indicated segmental dysfunction in the area of the right coronary artery. Because a normal right coronary artery was identified by preoperative cardiac catheterization, a severe spasm of the right coronary artery was diagnosed. Indeed, intravenous diltiazem injection improved ST-segment elevation and severe hypokinesis over the inferior wall.

The factors responsible for the spasm include coronary artery trauma due to surgical manipulation or to compression by chest drain tubes, alkalotic blood pH, alpha-adrenergic stimulation, low body temperature, and release of vasospastic factors by platelets damaged during cardiopulmonary bypass [5]. The feature of our case was the spasms triggered by closure of the sternum and resolved by diltiazem injection. However, these spasms occurred whenever the sternum was closed and were resolved after replacement of a pericardium drain tube apart from the right coronary artery. Therefore, we failed to identify any of these factors in the pathogenesis of the spasm in our patient, except for an external compression to the right coronary artery by the pericardium drain tube.

Severe spasm with circulatory collapse after multivessel OPCABG surgery has been reported [6]. Vessels with endothelial injury and dysfunction are subject to coronary artery spasms [7]. Although coronary angiography in our case displayed a normal coronary artery, endothelial dysfunction that could not be detected by angiography may have be present. Recently, Trimboli et al. reported a case in which trauma caused by external traction and compression on the coronary arterial wall might induce diffuse coronary spasm, which was treated with nitroglycerin infusion for several days [8]. In our case, because a single intravenous administration of diltiazem dramatically resolved this coronary artery spasm induced by mechanical compression by a pericardium drain tube, it is unlikely that trauma of the coronary arterial wall might trigger this spasm.

In summary, we experienced recurrent spasms of the right coronary artery with no apparent preexisting abnormal angiogram, which were induced by mechanical compression by a pericardium drain tube. From our experience, we should be aware that mechanical compression may trigger an exaggerated vasomotor response, leading to severe coronary artery spasms.

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