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Case Report

A case of ventricular septal rupture associated with major septal branch occlusion after percutaneous coronary intervention

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ABSTRACT

A 67-year-old man underwent elective percutaneous coronary intervention (PCI) of the left anterior descending artery. The major septal branch became occluded during coronary stenting. The patient developed dyspnea 19 days later. Chest radiography revealed lung congestion and a pleural effusion. Transthoracic echocardiography revealed a basal ventricular septal rupture. Emergency coronary angiography did not reveal any in-stent restenosis, and the major septal branch remained occluded. Therefore, the patient underwent closure of the ventricular septal rupture. The postoperative period was uneventful, and he was discharged 29 days after the operation. Septal branch occlusion due to coronary stenting occasionally occurs during routine PCI for which recanalization is sometimes not attempted. However, this case demonstrates that occluded septal branches, although rare, may cause serious complications.

<Learning objective: Rupture of the ventricular septum, a complication of acute myocardial infarction, is usually observed in the setting of acute myocardial infarction associated with major coronary artery occlusion. However, ventricular septal rupture associated with side branch occlusion due to coronary stenting for stable angina pectoris is uncommon. Awareness of this rare complication is useful during routine percutaneous coronary intervention.>

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Introduction

Rupture of the ventricular septum sometimes occurs as a complication of acute myocardial infarction (AMI). Most patients require surgical intervention because the rupture site can expand abruptly, resulting in sudden hemodynamic collapse [1].

We usually observe this complication in the setting of AMI associated with major coronary artery occlusion [2]. However, ventricular septal rupture associated with side branch occlusion due to coronary stenting for stable angina pectoris is uncommon.

In this report, we describe a rare case of an elderly man who developed ventricular septal rupture due to major septal branch

* Corresponding author at: Department of Cardiology, Shizuoka City Shizuoka Hospital, Otemachi 10-93, Aoi-ku, Shizuoka City, Shizuoka 420-8630, Japan. Tel.: +81 54 253 3125: fax: +81 54 252 0010. occlusion during coronary stenting of the left anterior descending artery.

Case report

A 67-year-old man presented with jaw discomfort and a temporary loss of consciousness. He initially visited the neurology department and underwent brain computed tomography. Magnetic resonance imaging, carotid ultrasonography, and electroencephalography did not reveal any abnormalities. Therefore, the neurologist consulted our department.

We performed coronary computed tomography angiography to investigate ischemic heart disease since the patient was suspected of having multiple coronary vessel stenoses with calcification. Coronary angiography revealed severe segmental stenosis in the proximal right coronary artery (RCA) and diffuse stenotic lesions in the left anterior descending artery (LAD); no collateral flow to

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Fig. 1. Angiogram before coronary stenting. (A) Focal severe stenosis is visible in the right coronary artery. (B) A diffuse stenotic lesion is visible in the left anterior descending artery. The major septal branch originates from the stenotic lesion (white arrow). (C) Some minor septal branches are also seen (white arrows).

either the RCA or the LAD was observed (Fig. 1). The left ventricular wall motion was hyperkinetic; on left ventriculography, the ejection fraction was 71%. Since his syncope was suspected to be due to myocardial ischemia, we recommended coronary revascularization of the RCA and LAD. Therefore, he underwent percutaneous coronary intervention (PCI).

We first performed PCI of the LAD by inserting a 6-Fr JL3.5 guiding catheter via the right brachial artery to the left coronary artery and crossing the coronary wire through the long stenotic lesion of the LAD. Since we could not deliver an intravascular ultrasound catheter (View-It[®]; Terumo, Tokyo, Japan), we performed pre-dilatation using a 3.0-mm semi-compliant balloon (Ryujin plus[®]; Terumo). After dilatation, a LAD coronary dissection occurred. The intravascular ultrasound catheter passed the lesion and revealed calcification around the origin of the major septal branch. We then implanted everolimus-eluting stents (Promus Element plus[®] 3.0 mm × 32 mm; Boston Scientific, Natick, MA, USA) to the mid-LAD lesion and performed post-dilatation using a 3.0-mm non-compliant balloon at 26 atm (iBP22[®]; Kaneka, Osaka, Japan).

After dilatation, the major septal branch became occluded (Fig. 2), and electrocardiography (ECG) revealed an ST elevation at the V1–3 leads (Fig. 3). We were able to observe 3 septal branches prior to the PCI, but the largest septal branch in the middle was not visible after PCI (Figs. 1 and 2). After stenting, the patient developed chest pain that was resolved with the intravenous

administration of buprenorphine. Since the other 2 septal branches remained unaffected, we did not try to reopen the occluded septal branch. We stented the proximal LAD lesion using a Promus Element plus[®] 3.0 mm × 22 mm stent and were careful to not re-jail the occluded septal branch. After PCI, the patient had no symptoms, but ECG revealed persistent ST elevation in leads V1–3 and a change in the complete right bundle branch block (Fig. 3).

Levels of creatine kinase/creatine kinase-MB increased to 1170/ 110 IU/L, and transthoracic echocardiography revealed hypokinetic motion at the intraventricular septum after the procedure. The patient had no chest pain: therefore, he was discharged 3 days after PCI. However, he developed dyspnea and visited our hospital 19 days after undergoing PCI. ST elevation at the V1-3 leads showed improvement on ECG (Fig. 3) and chest radiography revealed lung congestion and a pleural effusion. Transthoracic echocardiography revealed basal ventricular septal rupture (Fig. 4); therefore, an emergency coronary angiography was performed, but it did not reveal in-stent restenosis (Fig. 2). The major septal branch remained occluded. The Op/Os ratio measured using a cardiac catheter was 3.75. We initiated intra-aortic balloon pumping support and consulted a cardiac surgeon. The patient then underwent closure of the ventricular septal rupture using a double patch and coronary artery bypass grafting to the RCA (Fig. 4). Intraoperative findings revealed that the rupture site was within the basal ventricular septum, which was usually perfused by a major septal branch.



Fig. 2. Angiograms during and after percutaneous coronary intervention (PCI). (A) The major septal branch was occluded after coronary stenting, but the minor septal branch was occluded after coronary stenting, but the minor septal branch was occluded.

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