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Case Report

The lurking potential of tangential forces: A case of an arteriovenous shunt developed by percutaneous coronary intervention for the septal branch

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KEYWORDS
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Summary We describe the case of a 59-year-old male. His first percutaneous coronary intervention (PCI) using a bare metal stent was performed for a 90% stenosis in the mid portion of the left anterior descending artery (LAD). However, we performed re-PCI because in-stent restenosis developed during a chronic stage. After the first dilatation of the restenotic lesion, using a cutting balloon, the stenosis at the ostium of the septal branch, which takes off from the stent strut, became exacerbated. Therefore, after selective guidewire insertion to the septal branch, we performed balloon inflation. Unfortunately, a coronary dissection and perforation developed in the septal branch and a coronary arteriovenous shunt was also formed. Additional inflation for in-stent restenosis with a perfusion balloon provided successful occlusion of the ostium of the septal branch and the shunt flow disappeared. After careful re-selection of a guide wire into the septal branch, the perforated portion was then dilated using a small-sized conventional balloon. Finally, reperfusion of the septal branch was accomplished without any angiographic sign of coronary dissection, perforation or shunt. We herein report a rare case of coronary arteriovenous shunt formation due to the dissection and perforation of a coronary artery.

Introduction

Although a perforation of a coronary artery rarely develops during percutaneous coronary intervention (PCI), it can sometimes be fatal due to critical complications such as cardiac tamponade [1]. We herein present a rare case of a coronary arteriovenous shunt associated with a coronary perforation during PCI.

Case report

A 59-year-old male with symptoms of chest pain during exertion and drinking was admitted to our hospital. His coronary risk factors were smoking and diabetes mellitus. Coronary

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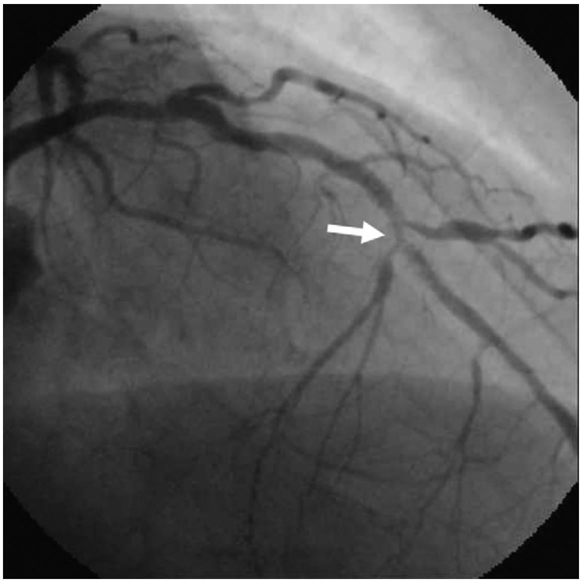

Figure 1 Coronary angiogram revealed in-stent restenosis in mid portion of left anterior descending artery (indicated by the arrow).

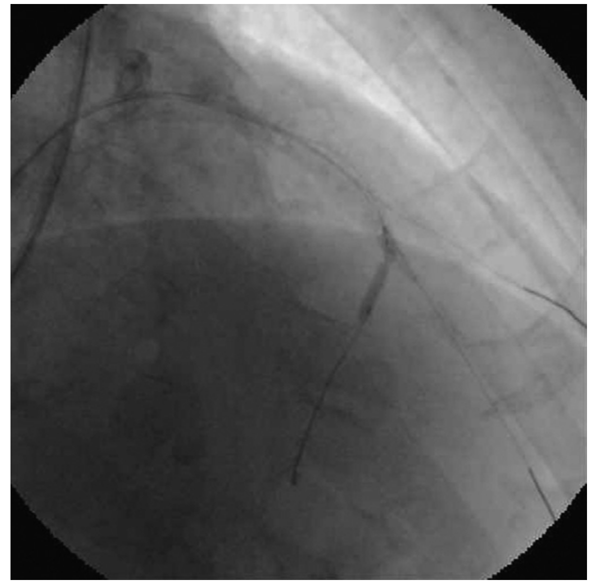

Figure 2 The ostium of the septal branch was dilated after the guidewire selection into the septal branch through the stent strut.

angiography revealed a significant stenosis in the left circumflex artery (LCx) and the mid portion of the left anterior descending artery (LAD). After the first PCI for the LCx, the second PCI for a 90% stenosis in the LAD was successfully performed with implantation of bare metal stents (Duraflex™ 3.0/8-mm and 3.0/25-mm, Goodman, Nagoya, Japan). A follow-up coronary angiography performed 6 months later, however, revealed a significant restenosis in the proximal stent site (Fig. 1) and a third PCI was performed for that lesion. Pre-procedure medications included 100 mg/day of aspirin and 200 mg/day of cilostazol. After the sheath insertion, 5000 units of heparin was intravenously administered, followed by continuous infusion at 400 units/h. A right radial artery approach was taken using a 5F-guiding catheter (Heartrail JL3.5™, Terumo, Tokyo, Japan). After two guide wires (Runthrough NS™, Terumo) were placed in the septal branch diverging from the stent placement and the 2nd diagonal branch, a third guide wire (Whisper™, Guidant, Santa Clara, CA, USA) was selected for the distal LAD. Then, the first dilatation of the culprit lesion was attempted using a cutting balloon (Cutting Balloon™ Ultra 2.5/10-mm, Boston Scientific, Maple Grove, MN, USA). However, the balloon did not advance to the stenosed lesion. After the removal of the two side branch guide wires, the balloon was then successfully advanced to the stenosed lesion and the lesion was dilated using the cutting balloon with nominal pressure. The stenosis at the ostium of the septal branch became exacerbated after dilatation, so the guide wire (Runthrough NS™) was again advanced through the stent strut and the septal branch was dilated using a conventional balloon (Maverick 2™ 2.25/20-mm, Boston Scientific) (Fig. 2). After 12 atm of balloon inflation was attempted due to indentation, which remained at the septal branch, the coronary flow suddenly became disrupted at the middle of the septal branch (Fig. 3). The angiogram performed after the removal of guide wires showed an expanded septal branch associated with the harsh blood flow running parallel to the LAD toward the cardiac base (Fig. 4). These angiographic findings showed the complication of an arteriovenous shunt due to septal dilatation. First of all, we administered 18 mg of protamine sulfate intravenously. Next, in order to occlude the ostium of the septal branch, a perfusion balloon (ACS Rx Esprit™, 2.5/20-mm, Guidant) was inflated at the proximal stent site for approximately 5 min. Thereafter, the septal branch was occluded from the ostium and the abnormal blood flow disappeared (Fig. 5). In addition, to ensure the closure of the perforated portion in the middle of the septal branch, which was possibly related to the formation of an arteriovenous

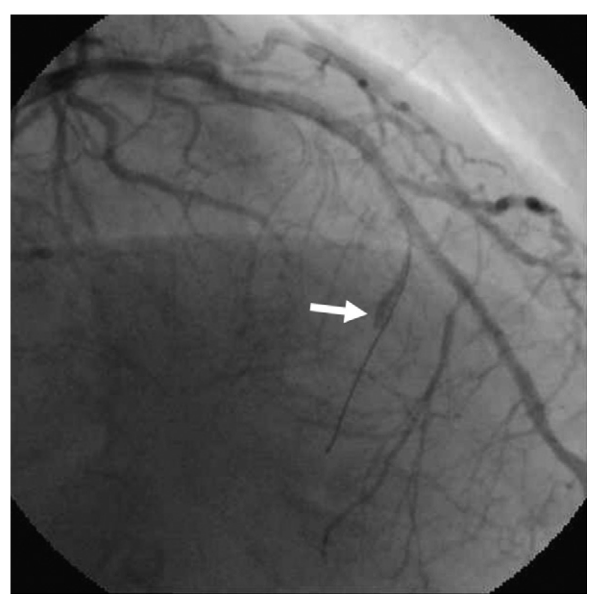

Figure 3 Coronary angiogram, immediately after the dilatation of the septal branch, showed the septal branch dissection with the disruption of blood flow from the same portion (indicated by the arrow).

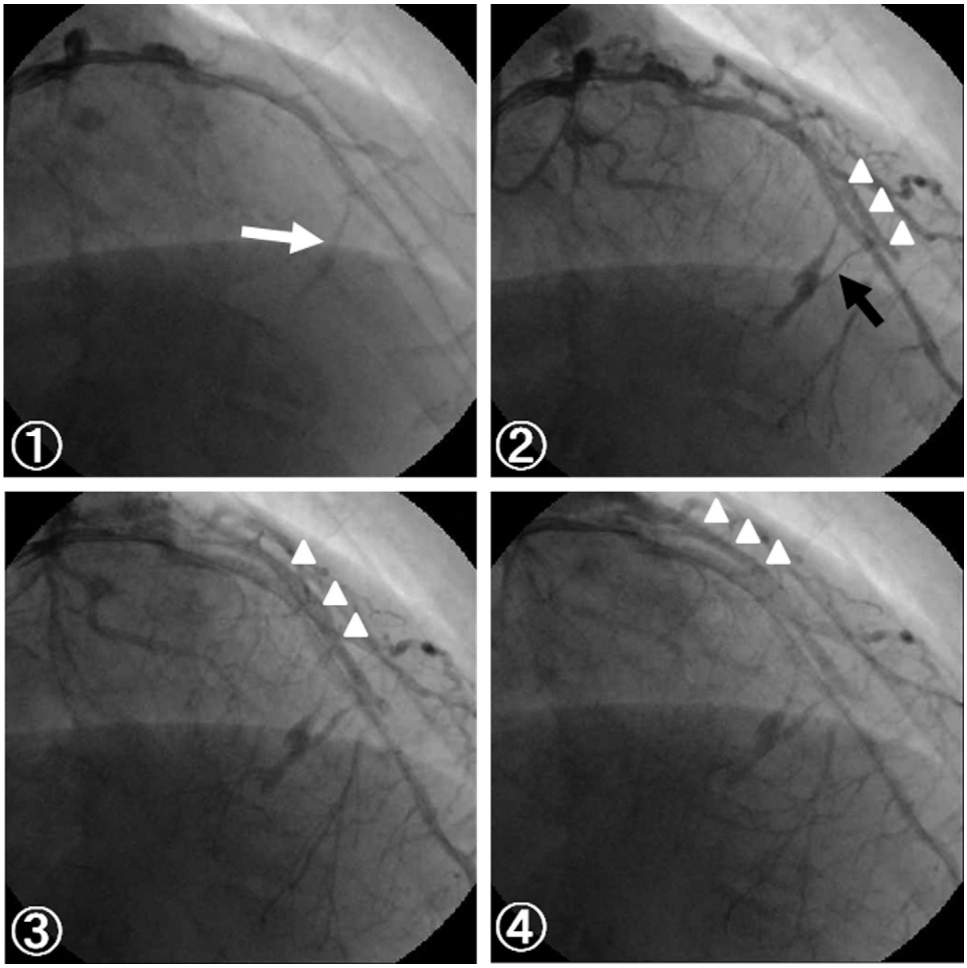

Figure 4 Serial images of a coronary angiogram (1 and 2) revealed the novel blood flow (black arrow) from the dissected portion of the septal branch (white arrow) to the left anterior descending artery. This flow followed a course into the vessel running parallel to the left anterior descending artery toward the proximal portion (3 and 4; arrow heads).

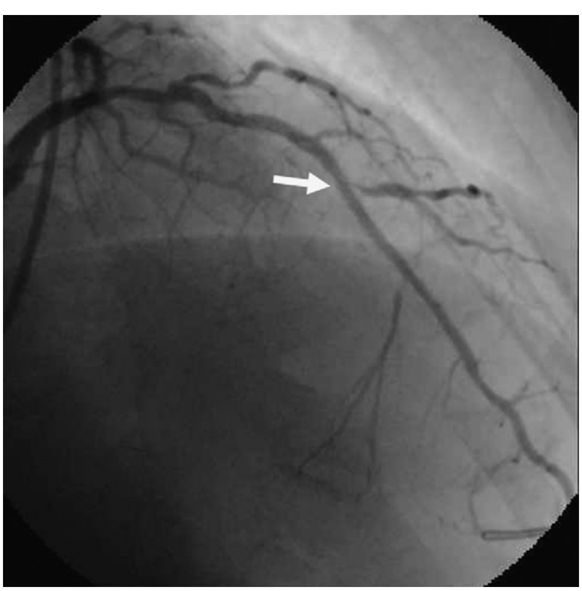

Figure 5 Coronary angiogram after the dilatation using the perfusion balloon revealed successful occlusion of the septal branch and disappearance of shunt flow (indicated by the arrow).

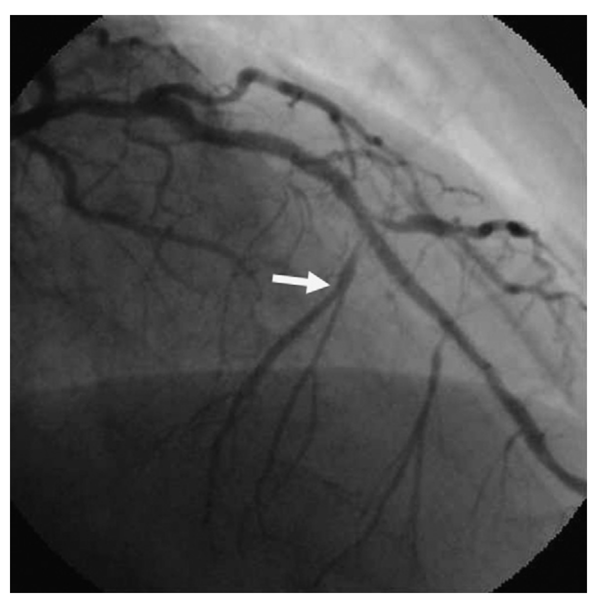

Figure 6 Final angiogram revealed successful reperfusion of the septal branch without coronary dissection, perforation and/or shunt (indicated by the arrow).

shunt, additional dilatation (at 4 atm) was performed using a small-sized balloon (Maverick 2TM, 1.5/20-mm) after a careful guide wire (Runthrough NSTM) re-selection to the septal branch. Finally, the reperfusion of the septal branch was accomplished without any angiographic sign of either coronary dissection, perforation, or arteriovenous shunt (Fig. 6).

Discussion

A coronary artery perforation is a relatively rare complication and the rate of occurrence is approximately 0.5%, according to previous reports [1—7]. With regard to classifications of coronary perforation, the classification reported by Ellis et al. has been widely used [1]. Namely, the perforations are divided into types 1, 2, and 3 and the existence of cavity filling, depending on the angiographic findings. Our case was classified as type 3 with cavity spilling. This group is reported to be very rare, comprising only 0.02% of all PCI patients and 5% of all perforation cases according to the report by Witzke et al. [2] or 0.01% of all cases that have undergone PCI and 2% of all cases of perforation according to the report by Fasseas et al. [3]. On the other hand, in Japan, Shirakabe et al. demonstrated that it accounts for 0.35% of all PCI cases and 17% of all perforations [6].

The main cause of coronary perforation is thought to be related to wire manipulation [2] during procedures. However, in this case, because we had no difficulty advancing the guide wire to the septal branch and noting that the perforation formed immediately after the balloon inflation in the septal branch, we believe that it is unlikely that guide wire manipulation was the cause. Regarding cases of perforation due to balloon dilatation, the main cause is reported to be the discrepancy between arterial diameter and balloon size [8]. The ratio of the balloon diameter to vascular diameter was significantly greater in the group with perforation than in the non-perforation group [1,9]. In this case, the balloon edge was carelessly advanced to a small septal branch and it caused a noticeable size difference.

With regard to forming an arteriovenous shunt, simultaneous perforation occurring both in the artery and the vein was considered to be essential. Regarding the mechanism of the simultaneous perforation, the first possibility was the artery and the vein were penetrated concurrently with a pointed tip of a guidewire [3,6]. However, perforation by a guide wire was considered to be unlikely in our case since guide wire manipulation in the septal branch was technically simple. In addition, neither a shunt flow nor any extravasation of contrast medium was detected in a test shot after guide wire placement. In this case, therefore we may suggest another possibility of shunt formation: if the artery and the vein are anatomically located very closely together, the surplus tangential force, for instance, by an inappropriate balloon/vessel ratio, which was 1.76 in our case, can cause the simultaneous perforation of artery and adjacent vein wall. This microscopic section including the septal branch from autopsy heart after paraffin fixation and hematoxylin—eosin staining shows the septal branch and the vein running parallel and very close together (Fig. 7).

With regard to the initial treatment, we believe that the administration of protamine to neutralize the effect of heparin was appropriate [8]. In order to close the shunt flow, balloon dilatation of the LAD with a perfusion balloon to occlude the ostium of the septal branch might be the simplest option, but the possibility of recanalization could not be fully ruled out. Recent reports have indicated the effectiveness of a covered stent for the treatment of coronary perforation [10]. Therefore, the implantation of a covered stent to cover the ostium of the septal branch for the purpose of occluding the shunt flow could be a possible strategy. Moreover, when the covered stent is deployed, while reperfusion for the branches would be virtually impossible, the occlusion of the 2nd diagonal branch from the ostium, resulting in the occurrence of myocardial infarction, becomes inevitable due to the anatomical relationship in our case.

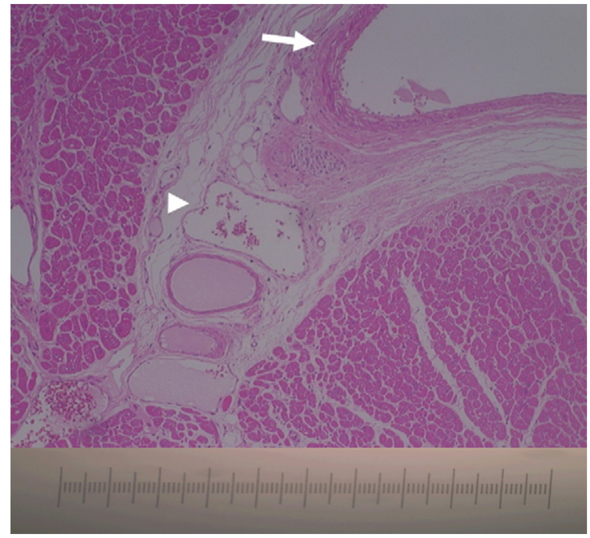

Figure 7 A microscopic section including the septal branch (indicated by the arrow) from autopsy heart: the vein (arrowhead) is located closely to the septal branch.

The clinical course in the cavity spilling group of class 3 was reported to be good [1,7]. Shirakabe et al. [6] have reported that only balloon inflation or neutralization of heparin was performed for successful closure. Therefore, the balloon inflation of the perforated lesion should have been attempted first, but advancing the guide wire into the perforated septal branch was considered to involve the risk of exacerbating the perforation. As a result, a prudent guide wire manipulation into the perforated septal branch was essential. The direct closure of the perforated vessel using thrombin [11], adipose tissue [12], Gelfoam [13], coils [14], polyvinyl alcohol [15], and clots [16] have all been reported as possible strategies for coronary artery perforation. However, these procedures were generally used for perforation of a peripheral coronary artery caused by a guide wire and are not suitable for the treatment of the intermediate portion of relatively large vessels such as in this case.

In our case, although only the ostium of the septal branch should have been dilated, the balloon was carelessly advanced into the small branch of the septal as well, thereby creating a size mismatch and thus causing a perforation and arteriovenous shunt. This case serves as a reminder that careless minor procedural mistakes can cause a serious complication.

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