CASE REPORT

Transcatheter embolization by autologous blood clot is useful management for small side branch perforation due to percutaneous coronary intervention guide wire

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Summary  A 75-year-old man underwent PCI for a bifurcation lesion with 90% stenosis in segment 6 and 75% proximal stenosis in segment 9 of the left coronary artery. We implanted a Duraflex coronary™ stent into segment 6 and kissing balloon inflation for segments 6 and 9. Although these 2 lesions were adequately dilated, we noticed coronary perforation caused by the guide wire in a small branch of segment 9. We tried to repair the perforation using a small balloon and long inflation, but unfortunately the perforation was not improved. We attempted to occlude the small branch including the perforation site with an autologous blood clot via a wire microcatheter inserted into the small branch. The autologous blood clot was suspended in contrast media and saline. Using this procedure, the small branch of segment 9 was occluded completely and the perforated site was repaired. After the procedure, no significant CPK elevation was detected, and 6 months later, we confirmed that small branch embolization was improved and coronary flow was good. Autologous blood clot is useful to occlude and repair perforations in small side branches of the coronary artery without myocardial damage.

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Introduction  Coronary stenosis at bifurcations is one of the most challenging lesion subsets for percutaneous
coronary intervention (PCI) [1]. Drug-eluting stents have considerable potential for these lesions, using deployment strategies such as crush stents [2]. In such situations, there may be increasing deployment of the double-wire technique using a hydro-coated guide wire which can be easily passed or taken out even in complex coronary anatomy or side branches, or a stent strut. Oozing perforation of a vessel at a distal site by a deeply deployed wire is possible. We report a case that was difficult to salvage from oozing perforation caused by a hydro-coated guide wire during PCI for bifurcation, and in which the small branch perforation site was successfully occluded with an autologous blood clot.

**Case report**

A 75-year-old man presented with a 2-month history of chest pain on effort. He had a risk factor of smoking for coronary atherosclerosis. An electrocardiograph at rest was within normal limits. He had recurrent chest pain and underwent cardiac catheterization. Coronary angiography demonstrated 90% bifurcation stenosis in segment 6 and 75% proximal stenosis in segment 9 of the left coronary artery (Fig. 1A and B), and 75% stenosis in segment 1 of the right coronary artery (Fig. 1C and D). He began to take aspirin and ticlopidine but drug eruption from ticlopidine occurred, and it was changed to cilostazol, as opposed to clopidogrel, which is not covered by insurance in Japan. We have an informed consent on the complications and the emergent rescue strategies of the cardiac catheterization to the patients and his family, including the coronary perforation in all patients prior to the cardiac catheterization in writing. PCI was performed by the right femoral approach, a PCI wire crossed by Filder™ (St. Jude Medical, Inc., CA, USA) to segment 6 and Whisper MSTM (Guidant Inc., Indianapolis, USA) to segment 9. Duraflex™ stent (3.0 mm × 25 mm) (Goodman CO. LTD., Nagoya, Japan) was deployed over segment 6 inflated at 16 atm (Fig. 2A). Post-dilatation was performed with the kissing balloon technique using a noncompliant balloon (3.25 mm × 15 mm) for segment 6 and (2.0 mm × 15 mm) segment 9 (Fig. 2B).
Intravascular ultrasound demonstrated complete stent deployment (not shown). The immediate angiographic result appeared good, but on the last projection after the PCI wire was taken out, we noticed enhancement with oozing, which was a coronary perforation caused by the guide wire in a small branch of segment 9. The patient was hemodynamically stable. However, we made a sure of the presence of pericardial effusion caused by the oozing blood a little revealed on the ultrasound cardiography. The wire was immediately re-crossed and a 2.0 mm × 15 mm balloon was inflated with low pressure (0–1 atm) over 60 min. Activated clotting time was 124 s with weak heparin by protamin adjunction; however, the oozing perforation could not be repaired, even after over the 60 min long-inflation with neutralization of heparin (Fig. 2C). We attempted to occlude the small branch including the perforation site by other methods to repair the oozing perforation completely, because we noticed the increasing volume of the pericardial effusion revealed on the ultrasound cardiography. We made a uniform autologous blood clot by stirring blood gathered from the sheath after receiving special informed consents on using the autologous blood clot from the patient and his family orally (Fig. 2D). About 0.5 ml blood of this autologous clot was suspended in contrast media and saline and inserted into the small branch via a wire-supported microcatheter (Transit™, Cordis, FL, USA) (Fig. 2E). Using this procedure, the small diagonal branch was occluded completely and the perforated site was repaired (Fig. 2F). During 60 min observation there was no oozing contrast at cineangiography. Post-procedure laboratory assessment showed levels of creatine phosphokinase of 60 IU/L with 5 IU/L MB. During the hospital stay, the patient developed no pain and no electrocardiography or enzymatic alterations were observed. Two days after stent implantation, he was discharged from the hospital. The patient had no episode of chest pain for six months after PCI. He then underwent left ventriculography and repeated coronary angiography. Left ventriculography shows normal contraction without an infarcted area (Fig. 3A and B), and angiographic findings of the occluded branch showed normal flow without delay (Fig. 3C and D).
Figure 3 Left anterior oblique of left ventriculography 6 months after PCI shows normal wall motion without an infarcted area (end-diastolic: A, and end-systolic: B). Repeated coronary angiography showed excellent angiography and good flow of the diagonal branch (straight cranial projection: C, and left anterior oblique cranial projection: D).

Discussion

There have been few instances of guide wire perforation during or before PCI. Some investigators have reported that guide wire perforation occurred in 0.36%, limited to the distal vessel in 0.13%, and almost all angiographic appearances were epicardial staining without a jet of contrast extravagation, which necessitates only careful observation with repeated injections of contrast dye [3]. There were several reports described the usefulness of the subcutaneous tissue or autologous blood clotting for the treatment of the balloon-induced perforation of the main coronary vessel [4–6]. However, there is few report described on the treatment of the guide-wire injury as like the present case using a hydro-coated guide wire. Another report showed that microfibrillar collagen is useful to occlude and repair perforations in small side branches of the coronary artery [7], but it can not be achieved easily, and instead we used an autologous blood clot, because it could be obtained simply and was cost effective and safe. Autologous blood clots are already used in the treatment of traumatic organ hemorrhage or priapism necessitating wide embolization [8]; occlusion should be reversible in order to salvage tissues or target organs. Embolization with an autologous blood clot satisfies this requirement, but not with subcutaneous tissue, because of clot lysis and consequent vessel recanalization. Previous studies of transcatheter arterial chemoembolization with an autologous blood clot for unresectable hepatocellular carcinoma demonstrated that the clot did not collapse the hepatic arteries in the short-term follow-up period [9] and the ability of the clot to maintain the patency of hepatic arteries was reconfirmed in long-term follow-up [10]. The fact that clots did not damage the hepatic arteries is a striking advantage of this treatment option over the conventional use of gelfoam. These cases reflected our case in the feasibility of the method and ability to preserve myocardial viability. Generally, uniform blood clotting can be achieved by stirring, and when left alone, the clot is easily divided into a hard thrombus and fragile thrombus. Different from alcohol embolization such as percutaneous transluminal septal myocardial ablation, embolization by autologous blood clot may form complex coagulation and the appropriate quantity
is unclear; one strategy is to start with a small quantity. In our case, clot embolization was reversible with good flow 6 months later. The characteristics of clot embolization may help to avoid a wide range of myocardial infarction. Nonetheless, we also have to point out that autologous blood clot is an own product, but once it formed outside the body, which might cause the infection or other embolization, such as cerebral infarction. Therefore the attention that is enough for the use is necessary.

**Conclusion**

The use of an autologous blood clot in a small branch was effective for oozing distal coronary perforation caused by a PCI guide wire.

**References**


