A case of coronary perforation induced by balloon dilatation after insufficient lesion modification with intravascular lithotripsy for in-stent calcified nodule

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An 86-year-old woman with a history of hypertension and chronic kidney disease on hemodialysis presented with chest pain at rest. Twelve years before, she had undergone percutaneous coronary intervention (PCI) for effort angina at another hospital, with Xience and Promus stents deployed in the RCA (#1?#2), and a Promus 3.0×28 mm stent implanted from the LMT (#5) to LCX (#11).

She was diagnosed as non-ST elevation myocardial infarction (NSTEMI), and urgent coronary angiography revealed 99% in-stent restenosis in LCX #11, which was identified as the culprit lesion. PCI was subsequently performed.

IVUS revealed a calcified nodule within the stent. Balloon dilation alone failed to achieve sufficient lumen expansion. While debulking was considered, intravascular lithotripsy (IVL) was selected due to the acute coronary syndrome (ACS) setting. IVL was attempted using a Shockwave C2 3.0×12 mm; however, balloon indentation persisted, and IVUS confirmed residual stenosis caused by the calcified nodule. Further dilation was attempted using an Aperta NSE 3.0×13 mm balloon.

During IVUS observation post dilation, the patient developed progressive hypotension and subsequent cardiopulmonary arrest. Coronary angiography revealed coronary artery perforation. Hemostasis was initially attempted with a Ryusei 3.0×20 mm perfusion balloon while initiating venoarterial extracorporeal membrane oxygenation (VA-ECMO) and pericardial drainage. After circulation stabilization, angiography confirmed the perforation site, which was on the opposite side of the calcification nodule observed in the IVUS. Hemostasis and revascularization were finally achieved by implanting a PK Papyrus 2.5 × 15 mm covered stent.

Although the patient developed post-resuscitation encephalopathy, her left ventricular ejection fraction (LVEF) was preserved. With rehabilitation, her functional status improved to the pre-event level, and she was transferred to another hospital for continued rehabilitation.

Discussion

Coronary artery perforation caused by balloon dilation for in-stent restenosis is a rare complication; however, a case of coronary artery perforation occurring inside the double-layered previous stents in-stent restenosis involving calcified lesions has been reported. Therefore, it has been proposed that calcified plaques should be debulked or IVL should be performed prior to dilation.

On the other hand, the efficacy of IVL for calcified lesions has been widely reported, and some studies have reported its efficacy even in cases of in-stent restenosis. However, in the present case, despite performing IVL on the calcified nodule within the stent, adequate expansion was not achieved, leading to coronary artery perforation during subsequent balloon dilation. Therefore, debulking for calcification volume reduction should be further considered.

In conclusion, it is important to note that even after IVL treatment for in-stent calcification, there is a risk of coronary artery perforation if the calcification nodules are not sufficiently modified.