

Cardiac Tamponade Caused by Angiographically Silent Coronary Artery PerforationKohei Hirobe¹Motohiro Fujita¹, Yuki Nakata¹, Naoki Serikawa¹, Eiji Shibahashi¹, Hisao Otsuki¹, Azumi Hamasaki², Hiroshi Niinami², Junichi Yamaguchi¹¹ Department of Cardiology, Tokyo Women's Medical University, Japan² Department of Cardiovascular Surgery, Tokyo Women's Medical University, Japan

Coronary artery perforation is a rare but potentially fatal complication of percutaneous coronary intervention (PCI). Distal wire perforation may remain angiographically occult. We report a case of cardiac tamponade caused by a suspected guidewire perforation without visible contrast extravasation.

A man in his 80s with hypertension, dyslipidemia, and a smoking history underwent PCI for worsening exertional angina. Coronary angiography showed a calcified lesion in the proximal-to-mid left anterior descending artery with an FFR of 0.76. Because occlusion of the second diagonal branch (D2) was a concern, a guidewire was placed in D2 for protection. Lesion preparation was performed using intravascular lithotripsy, followed by drug-eluting stent implantation. Final optical coherence tomography (OCT) confirmed good stent expansion. Immediately after OCT, the patient developed hypotension and ST-segment elevation in leads I and aVL. Repeat angiography showed no perforation, vessel occlusion, or other abnormality. His condition improved with low-dose noradrenaline, and he returned to the ward. However, 30 minutes later, he developed recurrent hypotension and cardiac arrest. Echocardiography revealed a large pericardial effusion. Emergency pericardiocentesis restored spontaneous circulation, but bloody drainage persisted. Repeat angiography, including selective and microcatheter contrast injection into D2, showed no definite extravasation. Because bleeding from the D2 territory was strongly suspected, D2 was occluded using autologous fat embolization. Nevertheless, drainage continued. Contrast-enhanced computed tomography showed a high-density area in the anterolateral myocardium, suggesting intramyocardial bleeding.

Surgical exploration revealed a hematoma on the myocardial surface in the D2 territory with continuous myocardial oozing. Hemostasis was achieved by surgical ligation. The postoperative course was uneventful, and the patient was discharged ambulatory on postoperative day 25.

The presumed mechanism was penetration of a small D2 branch by the protective guidewire at a myocardial bridge, followed by injury to an intramyocardial vein and gradual hematoma enlargement. Contrast injection during OCT may have increased hematoma pressure and transiently compressed the coronary circulation.

This case highlights that angiographically silent wire perforation can cause intramyocardial bleeding and delayed cardiac tamponade. Prompt echocardiography and early surgical exploration should be considered when unexplained hemodynamic deterioration persists despite the absence of angiographic extravasation.