Acute Right Ventricular Failure After Successful Opening of Chronic Total Occlusion in Right Coronary Artery Caused by a Large Intramural Hematoma

Masataka Kawana, MD; Anson M. Lee, MD; David H. Liang, MD, PhD; Alan C. Yeung, MD

A 76-year-old man with history of critical aortic stenosis was referred for surgical aortic valve replacement. Two months prior, preoperative diagnostic angiogram showed 2-vessel coronary artery disease, including 80% stenosis in midsection of left circumflex (LCX) artery, and serial lesions in the right coronary artery (RCA) with chronic total occlusion (CTO) of the distal segment (Movie I in the Data Supplement; Figure 1A) and significant left to right collateralization. At the time of surgical aortic valve replacement, coronary artery bypass was attempted; however, his saphenous veins were found to be unsuitable to use for bypass. His target vessels were deeply buried into myocardial fat as well. It was the heart team's decision to bring him back for elective percutaneous coronary intervention (PCI). Unfortunately, 3 weeks after the aortic valve replacement surgery, the patient was admitted to medical service for urosepsis with mild elevation in troponin because of demand ischemia. His transthoracic echocardiogram showed normal left ventricle (LV) function with basal inferior and midseptal wall hypokinesis at this time. Given the elevated troponin and known need to perform complete revascularization, we proceeded to PCI of his 2-vessel disease, including the mid-LCX lesion and the CTO in the large RCA system after he recovered from infection.

Before the PCI, he was maintained on dual antiplatelet therapy with aspirin 81 mg and clopidogrel 75 mg. He was loaded with additional 300 mg of clopidogrel before the procedure. Dual femoral accesses with 6F sheaths were placed, and CTO lesion was treated via antegrade approach. One drug-eluting stent was placed in mid-LCX lesion, and the CTO of RCA was opened with 3 drug-eluting stent (Movie I in the Data Supplement; Figure 1B). CTO lesion length was 50 mm.

Total 12 000 U of unfractionated heparin was used, and highest activated clotting time recorded was 201; no other anticoagulants were used. The procedure was uncomplicated, and the plan was to continue the same dual antiplatelet therapy regimen with aspirin and clopidogrel.

Six hours after the procedure, he developed acute left shoulder and chest pain in the recovery unit. ECG showed ST-segment–elevation in inferolateral leads (Figure 1D). He was loaded with prasugrel and emergently taken back to the catheterization laboratory with assumed in-stent thrombosis. An intra-aortic balloon pump was placed from the right femoral artery before coronary angiography was performed from the right radial artery, which showed patent stents in RCA and LCX and no significant disease in left anterior descending artery (Movie II in the Data Supplement; Figure 1C). There was an acute obstruction of small posterior descending artery, which was treated with balloon angioplasty but the flow could not be restored distally. Limited transthoracic echocardiogram in the cath laboratory showed no evidence of pericardial effusion.

He was transferred to coronary care unit afterward, where he was found to have increasing inotrope requirement, continued chest pain unresponsive to narcotics, and a sharp rise in Troponin I level to >40.0 ng/mL raising concern for ongoing myocardial injury. Moreover, echocardiographic images taken from subcostal view showed a large mass measuring 3.5×6 cm along the space between epicardium and pericardium that resulted in severe compression of right ventricle (RV; Movie III in the Data Supplement; Figure 2, upper left). The mass extended to the LV septum and inferior wall but did not compromise LV systolic function. Serial transthoracic echocardiography demonstrated ongoing enlargement of the mass up to 4×13 cm in size (Movie IV in the Data Supplement; Figure 2, upper right). Fluid inside the mass was organizing, and there was color Doppler evidence of blood flow inside (Movie V in the Data Supplement; Figure 2, lower left). Meanwhile, his liver function and urine output worsened despite medical and mechanical support. He was thus taken to the operating room for hematoma evacuation.

After redo sternotomy and lysis of adhesions of the heart, he was found to have a large intramural hematoma extending from the acute margin of the heart around the apex and onto the LV side of the intraventricular septum (Movie VI in the Data Supplement; Figure 3). Pericardiectomy from phrenic to phrenic was performed to relieve any possible constriction on the RV. Transesophageal echocardiography demonstrated a return of the RV cavity. Because of the recent prasugrel loading and concern for continued space-occupying lesions of the intramural hematoma on the RV, his chest was kept open and was subsequently monitored in cardiac intensive care unit.

From the Division of Cardiovascular Medicine, Department of Medicine (M.K., D.H.L., A.C.Y.) and Department of Cardiothoracic Surgery (A.M.L.), Stanford University School of Medicine, CA.

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Correspondence to Alan C. Yeung, MD, 300 Pasteur Dr Rm A260, MC 5319, Stanford, CA 94305. E-mail ayeung@stanford.edu

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The chest was closed 2 days later, and subsequently he had an uncomplicated postoperative recovery. He was eventually discharged to a skilled nursing facility. Follow-up transthoracic echocardiography after 6 weeks showed resolution of the intramural hematoma, normal LV size and systolic function with estimated ejection fraction 66%, normal RV size with moderately reduced systolic function, with distal RV free wall adhered to the pericardium. (Movie VII in the Data Supplement; Figure 2, lower right).

Analysis of National Cardiovascular Data Registry has shown that PCI of CTO carries a higher procedural complication compared with non-CTO PCI (1.6% versus 0.8%).[1] Steinwender et al[2] reported a similar case of intramural RV hematoma after failed attempt to cross the CTO in RCA, resulting in pseudotamponade physiology causing refractory cardiac arrest. Unfortunately, this patient did not survive because of severe hypoxic brain injury secondary to prolonged resuscitation attempts.[2] Lee et al[3] reported a case of RV hematoma after balloon angioplasty of CTO at distal posterior descending artery and drug-eluting stent placement to posterolateral branch, which spontaneously drained into RV without intervention and the patient did well. In the present case, the initial PCI of LCX and RCA successfully revascularized all the lesions, and the repeat coronary angiography showed no clear evidence of coronary dissection or perforation that were extensively evaluated during the case. There was no incidence of extraluminal wire placement or myocardial staining during the initial PCI. The case itself was one of the least eventful cases, which was done in under 2 hours, including 3 RCA stents, 1 LCX stent, and fractional flow reserve measurement of ramus intermedius (0.84, not intervened). Nevertheless, it is speculated that there was a microperforation of RCA, which was exacerbated by the reloading of antiplatelet therapy and repeat heparinization, as well as possible further mechanical damage during the second angiogram, which may have contributed to rapidly enlarging hematoma. In the setting of recent surgery and pericardial adhesions and inflammation, this likely caused severe compression of RV resulting in RV failure and tamponade physiology in the absence of pericardial effusion. Prompt recognition and treatment for RV failure, discovery of RV hematoma, and timely surgical intervention with relief of constriction were all critical in this patient's care.

Disclosures

None.

References

1. Brilakis ES, Banerjee S, Karmpaliotis D, Lombardi WL, Tsai TT, Shunk KA, Kennedy KF, Spertus JA, Holmes DR Jr, Grantham JA. Procedural outcomes of chronic total occlusion percutaneous coronary intervention: a report from the NCDR (National Cardiovascular Data Registry). *JACC Cardiovasc Interv*. 2015;8:245–253. doi: 10.1016/j.jcin.2014.08.014.
2. Steinwender C, Hofmann R, Leisch F. Pseudo-pericardial tamponade after perforation of the right coronary artery. *Heart*. 2004;90:e36.
3. Lee YW, Chun KJ, Park YH, Kim JS, Kim J, Kim JH, Lee SG, Lee DI, Kim JH. Right ventricular wall hematoma secondary to percutaneous coronary intervention. *J Cardiovasc Ultrasound*. 2009;17:99–101. doi: 10.4250/jcu.2009.17.3.99.

KEY WORDS: cardiac tamponade ■ coronary artery disease ■ coronary occlusion ■ echocardiography ■ hematoma ■ percutaneous coronary intervention ■ ventricular dysfunction, right

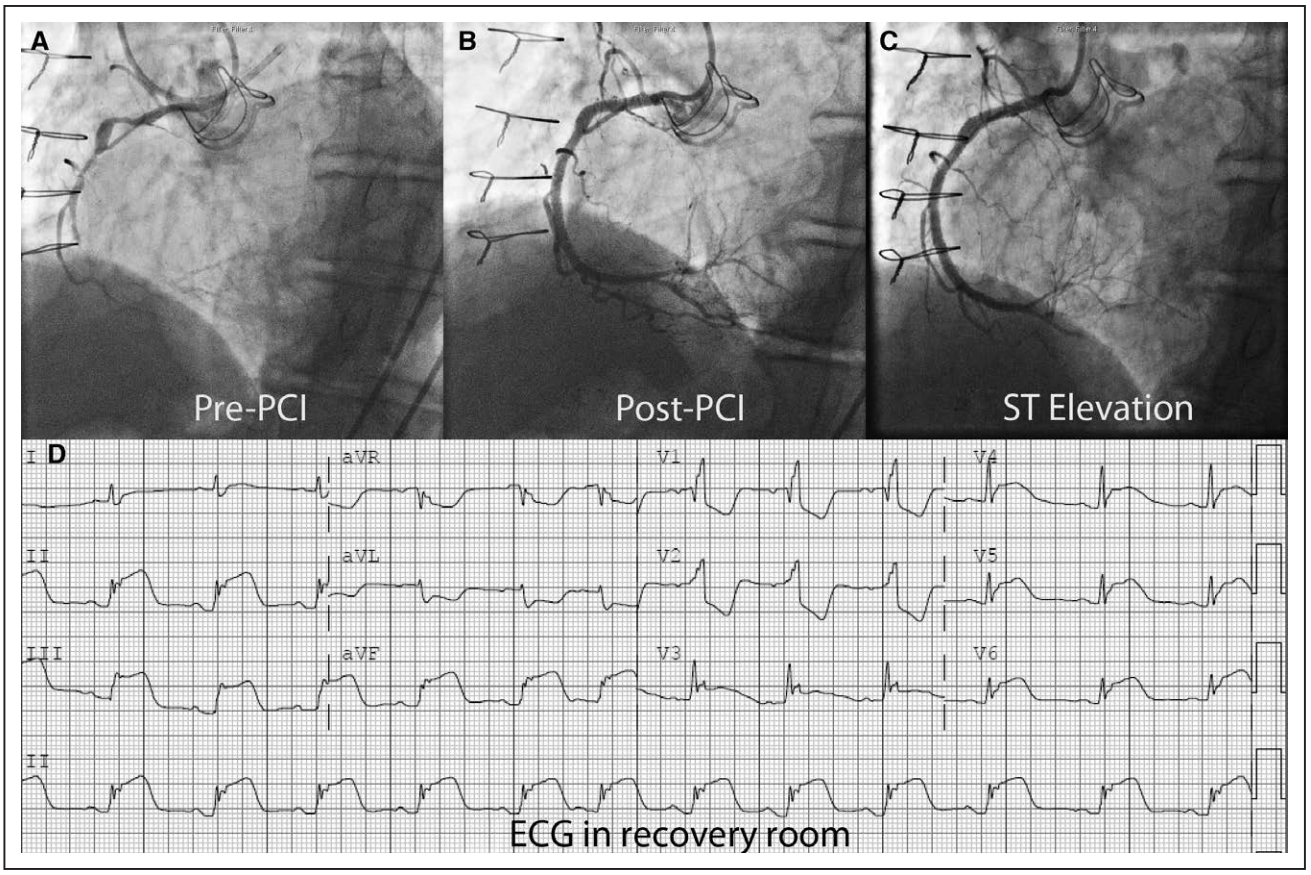

Figure 1. A, Right coronary artery (RCA) before the percutaneous coronary intervention (PCI) of serial high-grade lesions and chronic total occlusion (CTO). **B**, RCA immediately after the conclusion of PCI showing successful revascularization of CTO in RCA. **C**, Repeat coronary angiography after the chest pain and ST-elevation demonstrating patent stents in RCA and occlusion of small posterior descending artery (PDA). **D**, ECG obtained in recovery room showing inferolateral ST-elevation. The equipment used for the initial PCI includes (1) middle left circumflex: 6F EBU3.5 Guide, RunThrough Guidewire, predilated with Emerge Monorail RX 2.25×12 mm balloon, intervened with Synergy MR DES 2.25×20 mm, post-dilated with NC Trek RX 2.5×12 mm balloon and (2) RCA CTO: AL1 Guide, Asahi Corsair (2.6F distal 2.8F proximal×135 cm) Microcatheter, RunThrough Guidewire, predilated with Mini Trek 1.5×12 mm and Emerge Monorail RX 2.25×12 mm. Xience Alpine RX drug-eluting stent (DES) used: 2.5×38 mm in distal right coronary artery, 3.0×38 mm in middle right coronary artery and 3.5×23 mm in proximal right coronary artery. Post-dilated with NC Monorail 3.5×30 mm FG Emerge balloon.

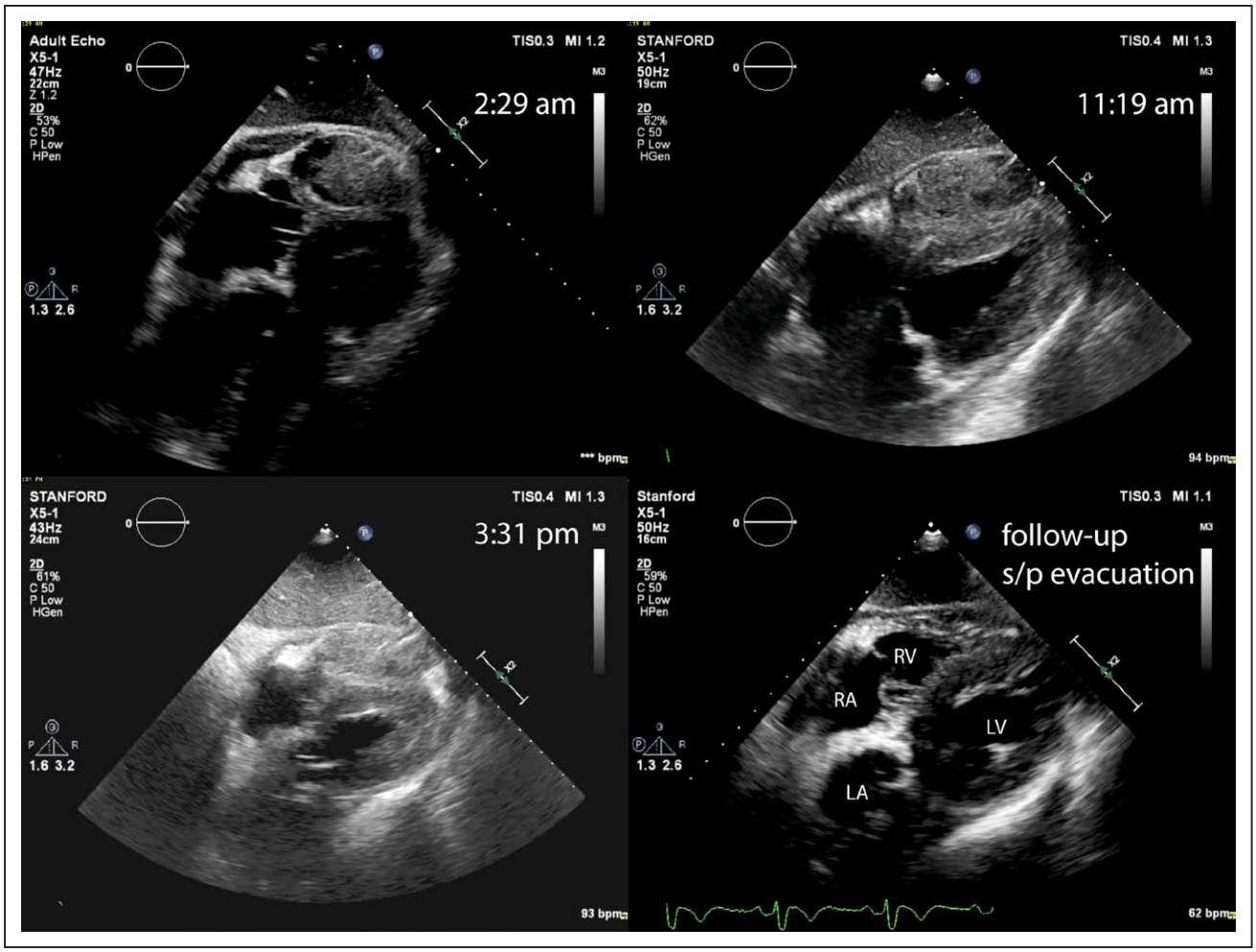

Figure 2. Time course of evolving intramural hematoma causing compression of right ventricle (RV). **Upper left**, Taken immediately after the repeat coronary angiography. The follow-up image (**lower right**) was taken 6 wk after the hematoma evacuation showing the disappearance of the mass with recovery of RV filling.

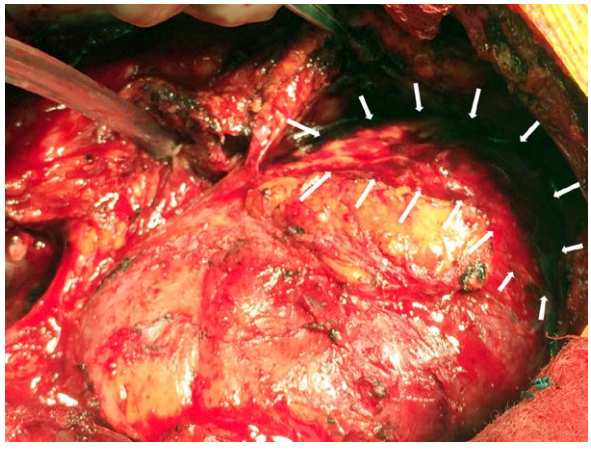

Figure 3. Intraoperative image showing a large bulging hematoma (depicted with arrows) on the diaphragmatic surface of the heart (**right**).

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