What is the cause of hypotension? A rare complication of percutaneous coronary intervention of a chronic total occlusion: a case report

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Background

In the last few years, complex techniques and advanced equipment became available to treat chronically occluded coronary arteries. Such procedures portend a series of possible complications that operators should be ready to quickly recognize and deal with.

Case summary

A 75-year-old lady with uncontrolled stable angina underwent percutaneous treatment of a chronically occluded right coronary artery. After balloon angioplasty and stenting, she developed a severe hypotension, refractory to fluid resuscitation and vasopressors. Computerized tomography scan demonstrated an intramural haematoma (IMH) of the right atrioventricular groove resulting in life-threatening pseudotamponade (or dry tamponade), as further confirmed by cardiac magnetic resonance imaging (MRI). The decision was for conservative management and haemodynamic support by intra-aortic balloon pump. Clinically, the patient improved and was discharged a few days later. Follow-up MRI confirmed resolution of the IMH.

Discussion

Severe hypotension during percutaneous treatment of chronically occluded coronary arteries may be related to various causes. Differential diagnosis is thus important in this setting and should include IMH, a rare but potentially fatal complication as it may cause compression of cardiac chambers and lead to pseudotamponade. A high index of suspicion is required to diagnose IMH but there are no clear guidelines for management of such cases.

Keywords

Pseudotamponade • Intramural haematoma (IMH) • Hypotension • Chronic total occlusion (CTO)

• Percutaneous coronary intervention (PCI) • Case report

Learning points

- Differential diagnosis of hypotension during percutaneous coronary intervention (PCI) of a chronic total occlusion (CTO).
- High index of suspicion about intramural haematoma as a possible complication of PCI of a CTO.

Introduction

Percutaneous coronary intervention (PCI) of chronic total occlusions $(CTO)^1$ is usually a complex procedure portending a series of possible complications that operators should be ready to quickly recognize and possibly treat. Complications can be classified as

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cardiac (either non-coronary such as aortic dissection, or coronary such as perforation, acute vessel closure, or equipment loss/entrapment) or extracardiac (radiation injury, vascular complications, contrast induced nephropathy).² The OPEN-CTO registry³ showed that, despite a technical success rate of 86% in expert centres, 7% of patients had major cardiovascular and cerebrovascular events. All of the deaths (nine patients) were associated with a complication: nine had a perforation requiring treatment, two of whom also experienced peri-procedural myocardial infarction.

Timeline

Time	Events
Day 0	Percutaneous coronary intervention to chronically occluded right coronary artery complicated by refractory hypotension → intra-aortic balloon pump (IABP) inserted.
Day 0 (2 h later)	Urgent computerized tomography scan incidentally showed intramural haematoma (IMH) of the atrioventricular groove, resulting in pseudotamponade.
Day 3	Patient normotensive \rightarrow IABP removed.
Day 10	Cardiac magnetic resonance imaging (MRI) confirmed diagnosis of IMH (improving). Patient discharged, haemodynamically stable.
7 months	Follow-up. Cardiac MRI showed almost resolved IMH. Patient normotensive and free from angina.

Case presentation

A 75-year-old lady presented with a history of treated hypertension and long-standing stable angina despite therapy with three antianginals uptitrated to maximum tolerated doses (bisoprolol 5 mg o.d., amlodipine 5 mg o.d., and isosorbide mononitrate 40 mg b.i.d.). Her electrocardiogram (ECG) showed sinus rhythm and no Q waves. Her transthoracic echocardiogram showed preserved left ventricular systolic function with mild hypokinesia of the inferior wall and no significant valvular disease. Her coronary angiogram showed a dominant right coronary artery (RCA) with CTO in proximal segment (Figure 1A; Supplementary material online, Angio 1), mild/moderate narrowing in proximal left anterior descending and moderate eccentric narrowing in proximal non-dominant left circumflex artery providing cross-filling to the occluded RCA through epicardial collaterals (Figure 1B; Supplementary material online, Angios 2-5). J-CTO score was 1. The left coronary lesions were thought unlikely to be flow limiting and after discussion with the patient, the interventional plan was to attempt a PCI to the occluded RCA with antegrade wire escalation strategy.

The procedure was carried out using dual access (right radial 6 Fr and right femoral 8 Fr). A Voda Left 3.5 6 Fr was used to engage the left coronary system and a Judkins Right 48 Fr to engage the RCA. A soft, polymer-jacketed, tapered wire (Fielder XT) with a microcatheter (Corsair) back-up was unable to cross the occlusion. Therefore, it was switched to a stiffer polymer-jacketed wire (Gaia 2nd) which was able to cross the occlusion. The correct position in the distal true lumen was confirmed by contralateral injection. The wire was then exchanged out over the Corsair to a standard coronary wire (Sion Blue) and balloon angioplasty was performed with a 2.5×15 mm balloon in the proximal and mid-segment of RCA, with subsequent evidence of extensive dissection of the vessel (Figure 1C; Supplementary material online, Angio 6). Two overlapping drugeluting stents (DES) $(3.5 \times 36 \, \text{mm})$ and $3.5 \times 33 \, \text{mm}$) were deployed in proximal-mid vessel (Supplementary material online, Angio 7), following which the patient developed hypotension (60/40 mmHg). There was evidence of residual dissection in distal RCA and two further overlapping DES ($2.5 \times 19 \, \text{mm}$ and $2.5 \times 29 \, \text{mm}$) were deployed (Figure 1D; Supplementary material online, Angio 8). The patient remained hypotensive, refractory to fluid resuscitation and intravenous phenylephrine boluses, complaining of dizziness and with evidence of raised venous pressures. A pericardial tamponade was suspected and an urgent echocardiogram was performed (Supplementary material online, Echo still picture and Echo video). This showed no pericardial effusion in subcostal view. A careful review of the coronary angiogram did not show any obvious perforations in the RCA or catheter-related dissection in the left coronary system. In addition, no significant ST changes were noted on ECG during the procedure. A femoral angiogram ruled out any bleeding at the access site. Other possible differential diagnoses were nitrate effect (unlikely, due to the long time after last nitrate administration and not responsiveness to fluids and phenylephrine), vagal reaction (unlikely, as the heart rate was slightly raised and there was no responsiveness to fluids and phenylephrine), or anaphylaxis (unlikely, due to late presentation and not responsiveness to hydrocortisone and chlorpheniramine). As the hypotension persisted, an intra-aortic balloon pump (IABP) was inserted with an augmented systolic blood pressure of 100 mmHg. Urgent blood tests showed a drop in haemoglobin as compared to pre-procedure (from 13.2 to 11 g/dL). To rule out a retroperitoneal bleed, an urgent computerized tomography (CT) scan was performed. This did not show any bleeding at the femoral site but revealed a large ($36 \times 31 \, \text{mm}$) intramural haematoma (IMH) in the right atrioventricular groove (Figure 2) compressing the tricuspid valve annulus and restricting right ventricular filling. The final diagnosis was thus pseudotamponade (or dry tamponade). As the patient did not show any sign of further haemodynamic deterioration in the next few hours, a conservative management plan was adopted. After 3 days, she became normotensive (120/70 mmHg) and the IABP was removed. A cardiac magnetic resonance imaging (MRI) showed a localized IMH (Figure 3A; Supplementary material online, cMRI video 1), slightly smaller (28×23 mm) than that seen on the CT scan. She was successfully discharged once clinically and haemodynamically stable (on Day 10) with a plan to take Aspirin and Clopidogrel for 6 months. After 7 months, repeat cardiac MRI (Figure 3B; Supplementary material online, cMRI video 1) showed an improvement of the haematoma which had not fully resolved but was

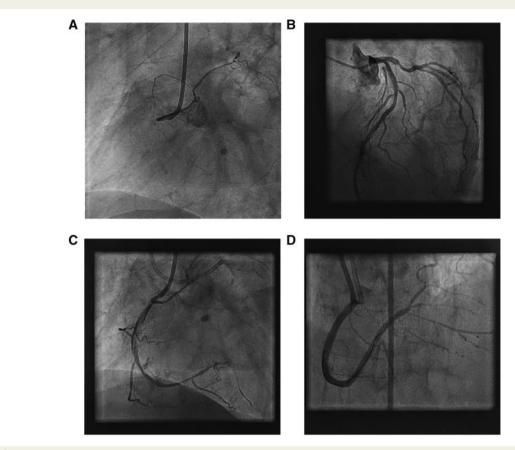


Figure 1 Coronary angiogram showing baseline right coronary artery (A) and left coronary system (B); extensive dissection after balloon angioplasty of right coronary artery (C) with final result of percutaneous coronary intervention (D).

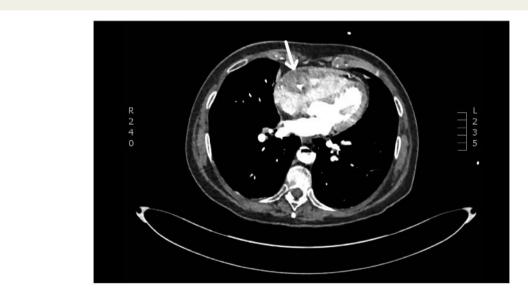
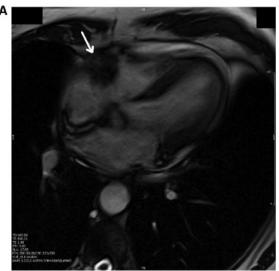


Figure 2 Computerized tomography scan showing intramural haematoma (white arrow) in right atrioventricular groove affecting tricuspid valve annulus.

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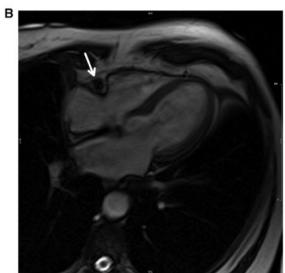


Figure 3 Cardiac magnetic resonance imaging performed during admission (*A*) and follow-up (*B*) showing resolving intramural haematoma (white arrows).

significantly reduced in size. The patient was normotensive and free from exertional angina.

Discussion

Intramural haematoma is a rare and potentially fatal complication of percutaneous procedures (mainly CTO PCI⁴ or catheter ablations⁵) as it may cause compression of cardiac chambers with similar haemodynamic effects as a proper pericardial tamponade,⁶ but with no evidence of significant pericardial effusion. This condition is thus referred to as pseudotamponade or dry tamponade. There are no international guidelines for its management and only few reports are available from literature, each suggesting a different approach with

various outcomes. For example, a recent case report⁷ showed a large $(4 \times 7 \text{ cm})$ IMH of the antero-inferior portion of the right ventricle caused by a perforation of a septal collateral during a CTO PCI of the RCA, with patient complaining of chest pain and ST elevation on ECG. Despite its dimensions, it was managed conservatively in view of patient's haemodynamic stability, with good clinical outcome. Such an approach⁸ may not be advisable in all cases,⁹ as another report¹⁰ showed right ventricular haematoma developing after wire perforation in a CTO PCI of the RCA. The patient was transferred to Coronary Care Unit nearly asymptomatic and normotensive but after 1 h rapidly deteriorated and collapsed in asystole. In previously by-pass grafted patients, IMH poses a particular challenge 11-13 because the bleeding might extend from the wall into the residual pericardial space contained by post-surgical adhesions. Management of such cases is usually surgical although recently successful CT-guided percutaneous procedures have been described. 14,15

Our case is unique for several reasons. First of all, hypotension was the main and almost only clinical sign, with no chest pain or ST changes on ECG. The raised venous pressures led to suspicion of a possible pericardial tamponade, but neither coronary perforation nor pericardial effusion were evident. In fact, a thorough off-line reassessment of angiographic images (Supplementary material online, Video 6) after the CT allowed to appreciate that some contrast extravasation (likely causing the IMH) was already evident during the procedure, even if the actual mechanism leading to it remains unclear. It might have been due either to wire perforation towards the right ventricular wall during antegrade wire escalation or following balloon angioplasty when an extensive dissection occurred. It was probably reduced after deployment of DES in proximal and mid segments of RCA. It is interesting that the final diagnosis was made as an incidental finding on a CT scan performed for a different reason. In a setting like this, transthoracic echocardiogram performed urgently may often miss the diagnosis. We suggest that an emergency thoracoabdominal-pelvic CT scan be performed in cases of unexplained hypotension refractory to standard treatments occurring during PCI. To the best of our knowledge, this is the first published case of IMH located at the atrioventricular groove. Even a small haematoma here will lead to significant haemodynamic compromise as it will compress the tricuspid valve and thus reduce inflow and outflow of the right atrium. An IMH in the right ventricular free wall would have to be significantly larger to give similar haemodynamic effects. Eventually, IABP helped managing the pseudotamponade conservatively. Serial imaging assessments with cardiac MRI were helpful to monitor the resolving IMH. A limitation in the management of this case is that a stress test was not performed prior to the PCI. It would ideally have been beneficial to have demonstrated ischaemia in the RCA territory prior to embarking on PCI. Despite this the operators were confident that, given the typical symptoms, no evidence of Q waves in the inferior leads on the ECG and no evidence of akinesia in inferior wall on transthoracic echocardiogram with no further angiographic targets, the RCA lesion was indeed the correct culprit vessel. Interestingly a cardiac MRI performed during the admission for evaluation of IMH confirmed viability with <50% subendocardial enhancement in the basal and mid inferior segments.

Lead author biography



Dr Vincenzo Vetrugno qualified in Medicine and Surgery from the University of Perugia in 2013. He started his Cardiology training at the Catholic University of the Sacred (Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome) in 2014. He is currently a clinfellow in Interventional Cardiology at the Queen Elizabeth Hospital (University Hospitals Birmingham NHS Foundation Trust, United Kingdom) under the supervi-

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Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

References

- Neumann F-J, Sousa-Uva M, Ahlsson A, Alfonso F, Banning AP, Benedetto U, Byrne RA, Collet J-P, Falk V, Head SJ, Jüni P, Kastrati A, Koller A, Kristensen SD, Niebauer J, Richter DJ, Seferović PM, Sibbing D, Stefanini GG, Windecker S, Yadav R, Zembala MO; ESC Scientific Document Group. 2018 ESC/EACTS Guidelines on myocardial revascularization. Eur Heart J 2019;40:87–165.
- 2. El Sabbagh A, Patel VG, Jeroudi OM, Michael TT, Alomar ME, Mogabgab O, Fuh E, Roesle M, Rangan BV, Abdullah S, Hastings JL, Grodin J, Kumbhani DJ, Alexopoulos D, Fasseas P, Banerjee S, Brilakis ES. Angiographic success and procedural complications in patients undergoing retrograde percutaneous coronary

- chronic total occlusion interventions: a weighted meta-analysis of 3,482 patients from 26 studies. *Int | Cardiol* 2014;**174**:243–248.
- Sapontis J, Salisbury AC, Yeh RW, Cohen DJ, Hirai T, Lombardi W, McCabe JM, Karmpaliotis D, Moses J, Nicholson WJ, Pershad A, Wyman RM, Spaedy A, Cook S, Doshi P, Federici R, Thompson CR, Marso SP, Nugent K, Gosch K, Spertus JA, Grantham JA. Early procedural and health status outcomes after chronic total occlusion angioplasty: a report from the OPEN-CTO registry (Outcomes, Patient Health Status, and Efficiency in Chronic Total Occlusion Hybrid Procedures). IACC Cardiovasc Interv 2017;10:1523–1534.
- Khan MF, Brilakis ES, Wendel CS, Thai H. Comparison of procedural complications and in-hospital clinical outcomes between patients with successful and failed percutaneous intervention of coronary chronic total occlusions: a metaanalysis of observational studies. Catheter Cardiovasc Interv 2015;85:781–794.
- Looi J-L, Lee AP-W, Chan C-P, Chan JY-S, Chan AK-Y, Tong M, Wong K-T, Yu C-M. Intramural left atrial haematoma complicating catheter ablation for atrial fibrillation. J Am Coll Cardiol 2013;62:252.
- 6. Adler Y, Charron P, Imazio M, Badano L, Baron-Esquivias G, Bogaert J, Brucato A, Gueret P, Klingel K, Lionis C, Maisch B, Mayosi B, Pavie A, Ristic AD, Sabaté Tenas M, Seferovic P, Swedberg K, Tomkowski W; ESC Scientific Document Group. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 2015;36: 2921–2964.
- Ghobrial MSA, Egred M. Right ventricular wall haematoma following angioplasty to right coronary artery occlusion. *J Invasive Cardiol* 2019;31:E66.
- Talanas G, Garbo R, Nusdeo G, Sanna GD, Bilotta F, Parodi G. Conservative management and 1-year follow-up of a huge left atrial intramural hematoma related to an epicardial collateral perforation during a chronic total occlusion percutaneous coronary intervention. *IACC Cardiovasc Interv* 2018;11:e207—e208.
- Kawana M, Lee AM, Liang DH, Yeung AC. Acute right ventricular failure after successful opening of chronic total occlusion in right coronary artery caused by a large intramural haematoma. Circ Cardiovasc Interv 2017;10. pii: e004674.
- Steinwender C, Hofmann R, Leisch F. Pseudo-pericardial tamponade after perforation of the right coronary artery. Heart 2004;90:e36.
- Almeida J, Garcia R, Monteiro V, Pinho P. Pseudo-pericardial tamponade after cardiac surgery. J Am Soc Echocardiogr 2009;22:211.e5–6.
- Akowuah EF, Rajnish R, Tomkins S, Hutter J. Pseudo cardiac tamponade: a rare complication of temporary epicardial pacing wires. Eur J Cardiothorac Surg 2008; 33:738.
- 13. Allencherril J, Allencherril R, Joseph L. Left atrial intramural haematoma after surgical aortic valve replacement. *Methodist Debakey Cardiovasc J* 2017; **13**:81.
- Wilson WM, Spratt JC, Lombardi WL. Cardiovascular collapse post chronic total occlusion percutaneous coronary intervention due to a compressive left atrial haematoma managed with percutaneous drainage. Catheter Cardiovasc Interv 2015;86:407–411.
- 15. Adusumalli S, Morris M, Pershad A. Pseudo-pericardial tamponade from right ventricular haematoma after chronic total occlusion percutaneous coronary intervention of the right coronary artery: successfully managed percutaneously with computerized tomographic guided drainage. Catheter Cardiovasc Interv 2016; 88:86–88.