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Severe chronic aortic regurgitation after percutaneous coronary intervention: a case report and literature review

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Background Severe aortic regurgitation (AR) is an extremely rare complication after coronary catheterization and percutaneous coronary intervention (PCI), where most reported cases have required relatively urgent surgical intervention due to acute-onset AR and cardiac decompensation.

Case summary We report a case of a 60-year-old woman that previously presented with a non-ST-elevation myocardial infarction (NSTEMI) due to an ostial right coronary artery stenosis. During the course of 2 years, she developed five recurrent NSTEMI due to in-stent thrombosis, necessitating either a new coronary stent or balloon. She developed a chronic severe AR due to a drug-eluting coronary stent protruding from the right coronary artery and underwent successful aortic valve replacement and coronary artery by-pass grafting.

Discussion We performed a literature review and identified 16 reported cases of iatrogenic severe aortic regurgitation related to coronary catheterization or percutaneous coronary intervention. All patients developed an acute aortic regurgitation and, thus, we report the first case of a delayed complication caused by a protruding coronary stent. The surgical strategy is related to the extent of the damage, where smaller perforations or lacerations seems to be feasible for aortic valve repair and larger defects more often lead to aortic valve replacement. Our patient developed a fibrotic right coronary cusp which could not be used to perform a successful aortic valve repair.

Keywords Aortic regurgitation • Aortic valve replacement • Case report • Coronary angiography • Iatrogenic • Percutaneous coronary intervention

Learning points

- Iatrogenic severe aortic regurgitation is an extremely rare complication after coronary catheterization or percutaneous coronary intervention.
- All but one reported cases to date describe the development of acute severe aortic regurgitation.
- We show that chronic severe aortic regurgitation can be caused by a protruding ostial coronary stent due to repeated mechanical trauma and/or hypersensitivity reaction to the stent.

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Introduction

Aortic regurgitation (AR) is the diastolic backflow of blood from the aorta to the left ventricle (LV) through an incompetent aortic valve. In the Framingham Heart Study, the estimated prevalence of chronic AR was about 5% in the general population, with moderate to severe AR occurring in approximately 0.5%.[1] Chronic AR may be caused by a primary valve disease, such as congenital malformation [bicuspid aortic valve (BAV)] and degeneration of a tricuspid aortic valve (TAV), or result from dilatation of the thoracic aorta.[2] Acute AR is uncommon and can be caused by type A aortic dissection, endocarditis or trauma, and less common iatrogenic causes.[3]

We report the first case of a delayed percutaneous coronary intervention (PCI) complication, where the patient developed a chronic severe AR due to a protruding right coronary artery stent. We also provide a review of the literature to summarize the present knowledge of coronary angiography/PCI-induced AR.

Timeline

28 months prior to admission	Received a 12 mm drug-eluting stent in the ostial part of the right coronary artery due to a non-ST-elevation myocardial infarction (NSTEMI)
24 months prior to admission	In-stent thrombosis causing NSTEMI, dilatation using a 4.0/20 mm drug-eluting balloon (DEB)
17 months prior to admission	In-stent thrombosis causing NSTEMI, balloon dilatation and subsequent stenting
14 months prior to admission	Cardiac magnetic resonance imaging revealed a moderate aortic regurgitation (AR), a mildly dilated left ventricle (LV) and normal left ventricular ejection fraction (LVEF)
11 months prior to admission	In-stent thrombosis causing NSTEMI, placing of a 4.0/24 mm drug-eluting stent
5 months prior to admission	In-stent thrombosis causing NSTEMI, dilatation using a 4.0/20 mm DEB
3 months prior to admission	In-stent thrombosis causing NSTEMI, dilatation using a 4.0/30 mm DEB
2 months prior to admission	Transthoracic echocardiography showed a severe AR, severely dilated LV with mildly reduced LVEF (45%)
At admission	Underwent aortic valve replacement and concomitant coronary artery by-pass grafting

Case presentation

A previously healthy 60-year-old Caucasian woman presented to a secondary referral centre emergency department (ED) with central chest pain 28 months prior to admission to our clinic. There was a slightly elevated troponin and a non-ST-elevation myocardial infarction (NSTEMI) was suspected. Coronary angiography revealed an ostial stenosis in the right coronary artery (RCA) which was successfully treated with a drug-eluting stent (DES) (*Figure 1*). Afterwards, the patient had a total of five recurrent in-stent thrombosis necessitating either a new DES or dilatation using a drug-eluting balloon (DEB), see *Timeline*. About 14 months prior to admission, she underwent a cardiac magnetic resonance imaging (cMRI) to assess the myocardial viability due to recurrent NSTEMI. She had normal biventricular function and normal chamber dimensions, and there were no signs of late gadolinium enhancement. Instead, a mild to moderate AR was found with a calculated regurgitation volume of 34 mL (corresponding to a 30% regurgitation fraction) (*Figure 2*).

Three months prior to admission, she once again developed a NSTEMI due to in-stent thrombosis in the proximal segment of the RCA and a new DEB was placed. She had complaint of progressive dyspnoea in the past 6–12 months, in New York Heart Association (NYHA) functional Class III, and underwent a TTE 2 months prior to admission. This revealed a severe AR with an eccentric regurgitant jet covering almost the whole LVOT, a short pressure half-time (147 ms), significant diastolic reversing in the aortic arch, a dilated LV and a mildly reduced left ventricular ejection fraction of 45% (normal value 54–74%) (*Figure 3*). She had a tricuspid aortic valve with suspected prolapse of the right coronary cusp (RCC), but the mechanism of AR remained uncertain. The patient was accepted for surgery and aortic valve repair was planned, as well as coronary artery bypass grafting (CABG) to the RCA. At the admission, she was in a compensated state but experienced exertional dyspnoea corresponding NYHA functional Class IIIA. The physical examination revealed nothing remarkable except for a holodiastolic murmur at the site of the aortic valve.

After induction of general anaesthesia, the routine perioperative transoesophageal echocardiography (TOE) confirmed the diagnosis of a severe AR (*Video 1*). In an X-plane view including the mid-oesophageal aortic valve short- and long-axis views, a structure with the appearance of a stent protruding from the right coronary ostium, was found above the RCC (*Figure 4*, *Video 2*). In systole, the RCC seemingly struck the protruding stent (*Video 3*). A 3D TOE was carried out to better characterize the nature of the AR (Supplementary material online, *Video S1*) but the mechanism remained uncertain. A full median sternotomy was performed, and the heart was accessed after complete opening of the pericardial sac. Retrograde cardioplegia was delivered through the coronary sinus and asystole was induced. First, the distal anastomosis to the RCA was completed using the great saphenous vein as a conduit. The distal anastomosis was placed as proximal as possible to ensure adequate retrograde flow to the proximal parts of the RCA. An aortotomy was subsequently performed above the level of the aortic valve. In line with what was seen on the TOE, a stent was protruding about 1 cm into the Sinuses of Valsalva from the right coronary ostium (*Figure 5*). The stent was trimmed, and an autologous pericardial patch was used to permanently close the right coronary ostium. The aortic valve was then inspected and the RCC was fibrotic and retracted with poor coaptation to the other cusps, but there was no prolapse. Both the LCC and NCC had normal morphology and function. It was

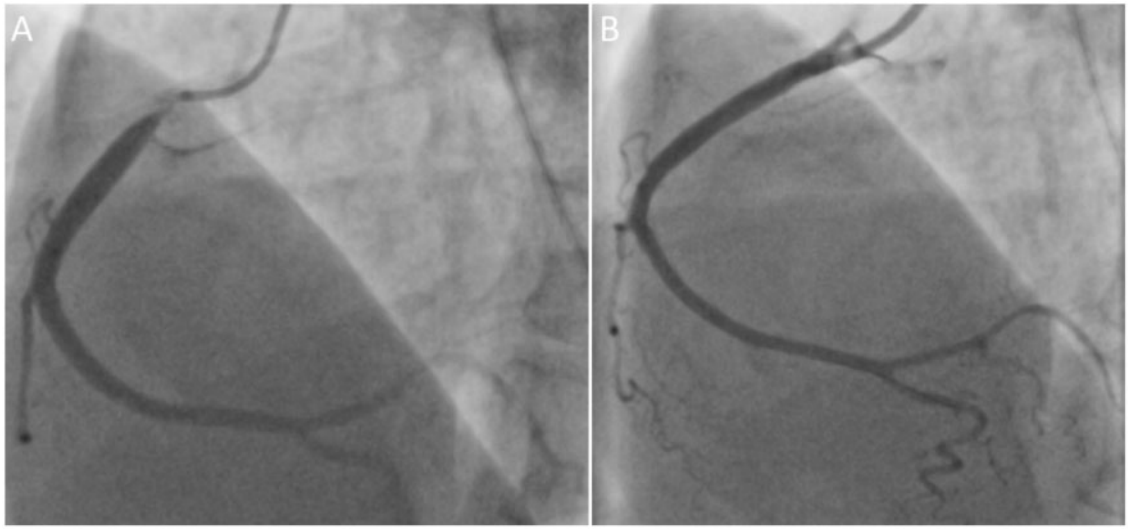
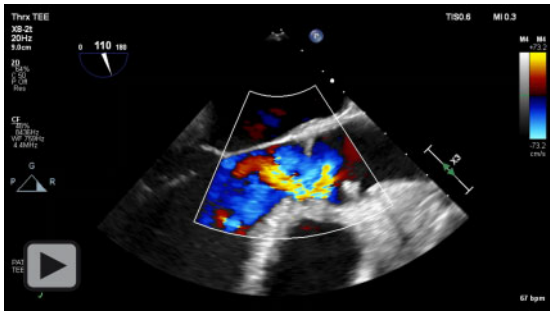
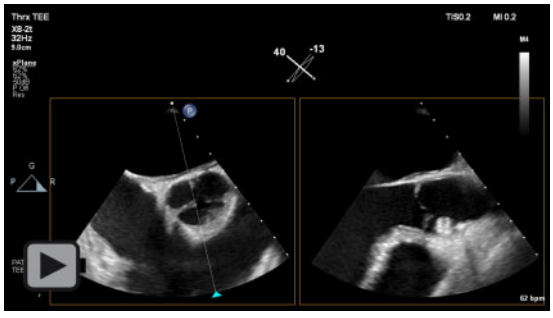


Figure 1 The patient underwent her first coronary angiogram when she presented with central chest pain 28 months prior to admission to our institution. (*A*) The LAO 30° view showed an ostial total occlusion of the right coronary artery. (*B*) The culprit lesion was successfully treated with a drug-eluting stent (*B*).



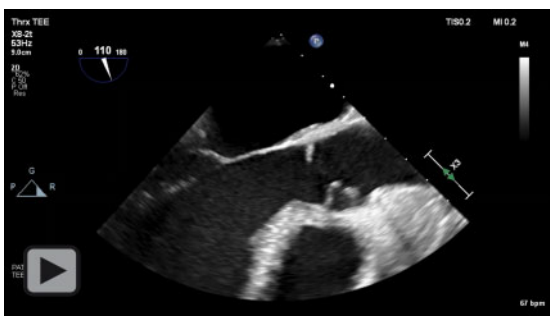
Video 1 Peroperative midesophageal aortic valve long-axis view (ME AV LAX) showing a significant aortic regurgitation.



Video 2 Peroperative X-plane imaging over the aortic valve, including the midesophageal aortic valve short- and long-axis views, showing a structure with the appearance of a stent protruding from the right coronary ostium, above the RCC.

determined that aortic valve repair could not be performed and a mechanical aortic valve prosthesis (Carbomedics 23 mm) was placed instead. Weaning from extra-corporeal circulation was uncomplicated and the patient was extubated a couple of hours after the procedure in the thoracic intensive care unit (ICU).

After spending 1 day in the thoracic ICU, she was transferred to the surgical ward for further post-operative care. The post-operative course was uneventful. Routine post-operative echocardiogram showed a normally functioning mechanical aortic valve prosthesis with adequate max and mean gradients (29/17 mmHg) and no paravalvular leakage. The patient was feeling well and was discharged to her home after a total hospital stay of 7 days. At a 5-month follow-up, she had returned to normal physical activity and was in NYHA Class I. She underwent an elective myocardial scintigraphy at the referral hospital as she had experienced brief episodes of angina-like chest pain during the past 2 weeks. The myocardial scintigraphy showed no



Video 3 Peroperative ME AV LAX view illustrating how the RCC repeatedly struck the protruding RCA stent in systole.

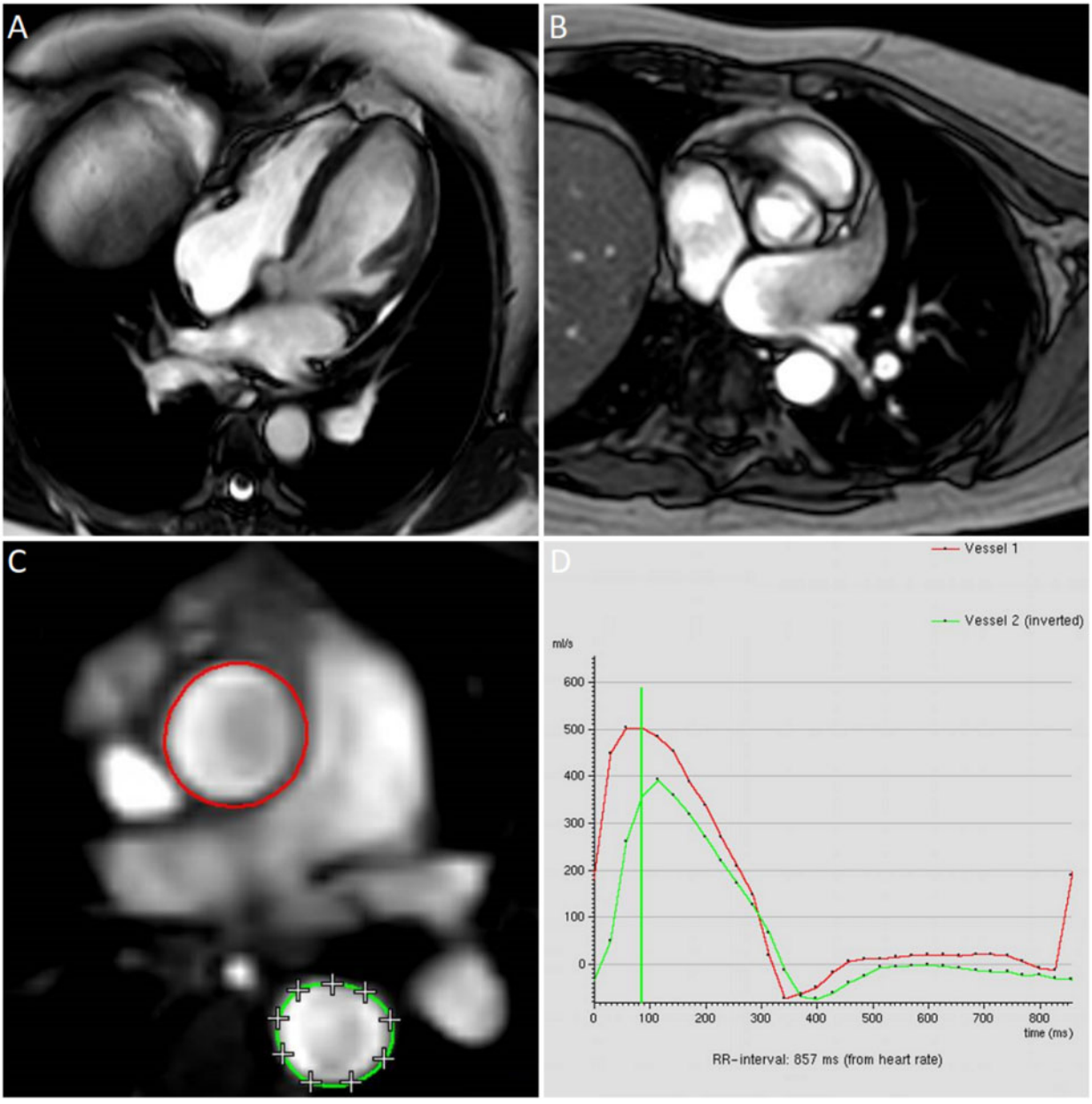


Figure 2 Cardiac magnetic resonance imaging revealed a mild to moderate aortic regurgitation in a tricuspid aortic valve, and a mildly dilated left ventricle with a normal systolic function (left ventricular ejection fraction 66%). (*A*) The aortic regurgitant jet visualized in the five-chamber view. (*B*) Short-axis view showed a tricuspid aortic valve. (*C*) Region of interests for antegrade flow in the ascending aorta (red) and retrograde flow in the descending aorta (green). (*D*) Flow-vs.-time plot in the ascending aorta with a calculated antegrade flow of 113 mL/beat and a retrograde flow of 34 mL/beat, and aortic regurgitant fraction 30%.

signs of ischemia and the symptoms disappeared shortly afterwards and has not recurred ever since.

Discussion

Coronary angiography/PCI-induced severe AR is extremely rare, with a reported frequency of approximately 0.0001%,[4] and often associated with an acute presentation. We report an unusual presentation of PCI-induced severe AR, where the patient developed a chronic AR with associated progressive symptoms. This has never been reported, to the best of our knowledge.

To review the present knowledge of coronary angiography/PCI-induced AR, we performed a literature search of Medline and Embase databases using the terms 'Aortic regurgitation OR Aortic insufficiency' AND 'Coronary Angiography OR Percutaneous Coronary Intervention OR Coronary Catheterization' AND 'Iatrogenic OR Traumatic' AND 'Aortic Valve Injury OR Aortic Valve

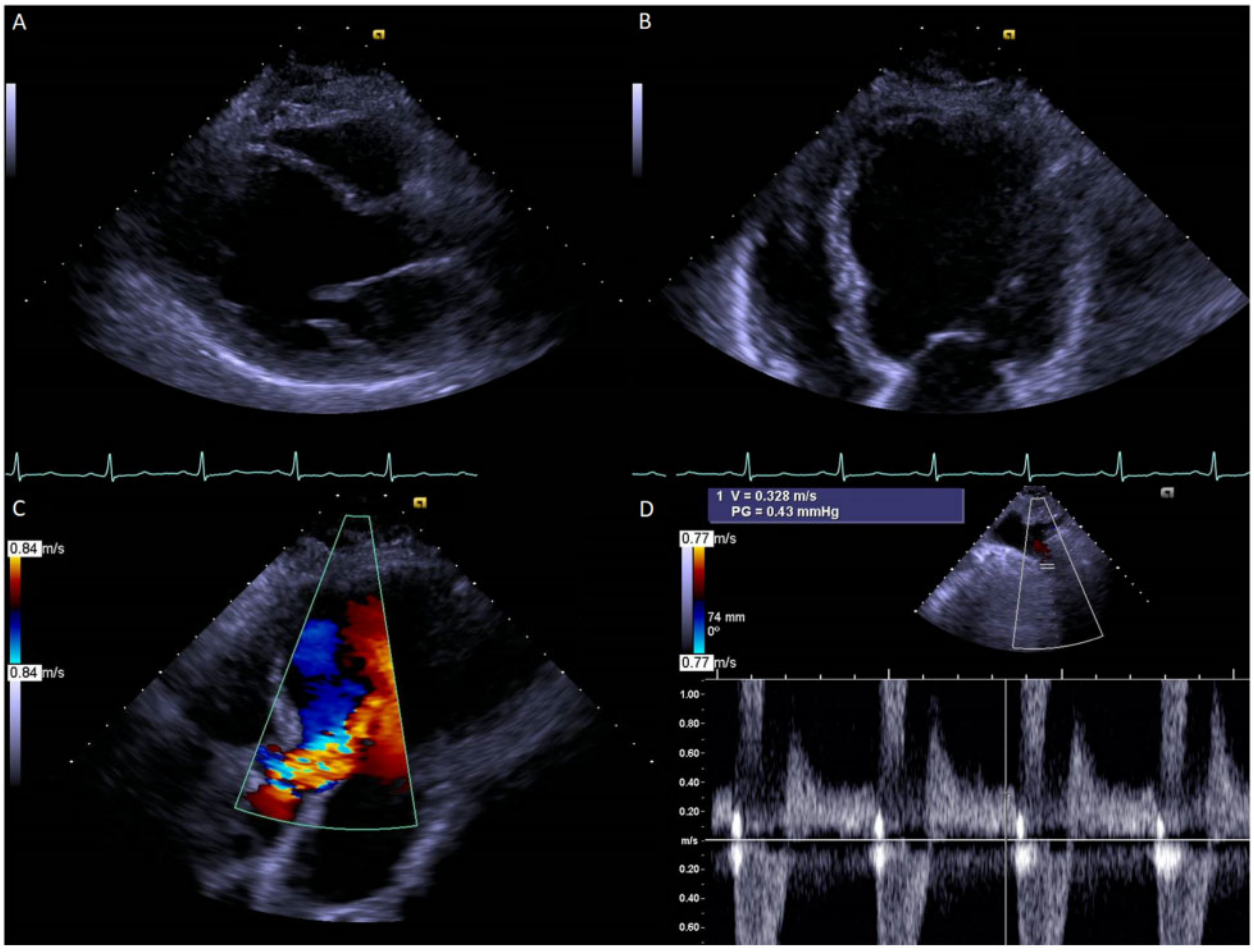


Figure 3 Transthoracic echocardiography showed a severe aortic regurgitation in a severely dilated left ventricle (indexed left ventricular end-diastolic volume 94 mL/m^2, normal value 29–61 mL/m^2**)** and a mildly depressed systolic function (left ventricular ejection fraction 45%, normal value 54–74%). (*A*) Parasternal long-axis view showing the severely dilated left ventricle. (*B*) In the apical four-chamber view, the spherical form of the adversely remodelled left ventricle was appreciated. (*C*) The apical five-chamber colour-Doppler view displaying a severe aortic regurgitant jet. (*D*) Suprasternal view showing a holodiastolic backflow in the descending aorta with an end-diastolic velocity of 33 cm/s.

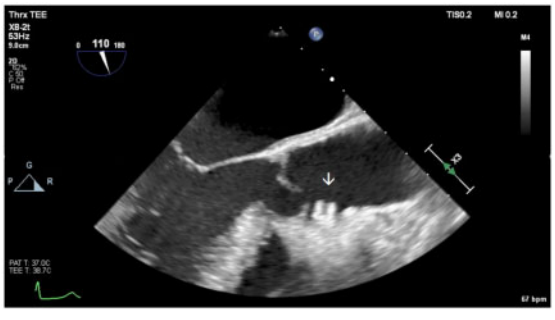


Figure 4 Perioperative transoesophageal echocardiography mid-oesophageal aortic valve long-axis view showing a stent protruding from the right coronary artery almost 1 cm into the Sinus of Valsalva (arrow).

Perforation OR Aortic Valve Laceration'. All articles in English published until 31 July 2020 were eligible for inclusion. References cited by these articles were also reviewed for eligibility. Reports of secondary AR, e.g., due to aortic dissection, were excluded. Only 14 case reports involving 16 patients have been documented in the literature,[4–17] of which all but one describe the development of acute severe AR in contrast to our report (*Table 1*). Most cases report catheter or guidewire as probable cause of laceration/perforation, while AR related to a protruding coronary stent was reported in only two cases. The patient in the case reported by Denyer *et al.*[17] also developed a chronic AR, but the mechanism was leaflet perforation and it is likely that the AR emerged instantly. We attributed the AR to previous PCI with the placement of a DES which, due to the ostial RCA stenosis, was placed proximal and obviously protruding from the RCA. We propose that the mechanism of chronic severe AR in this patient was due to repeated mechanical trauma to the RCC

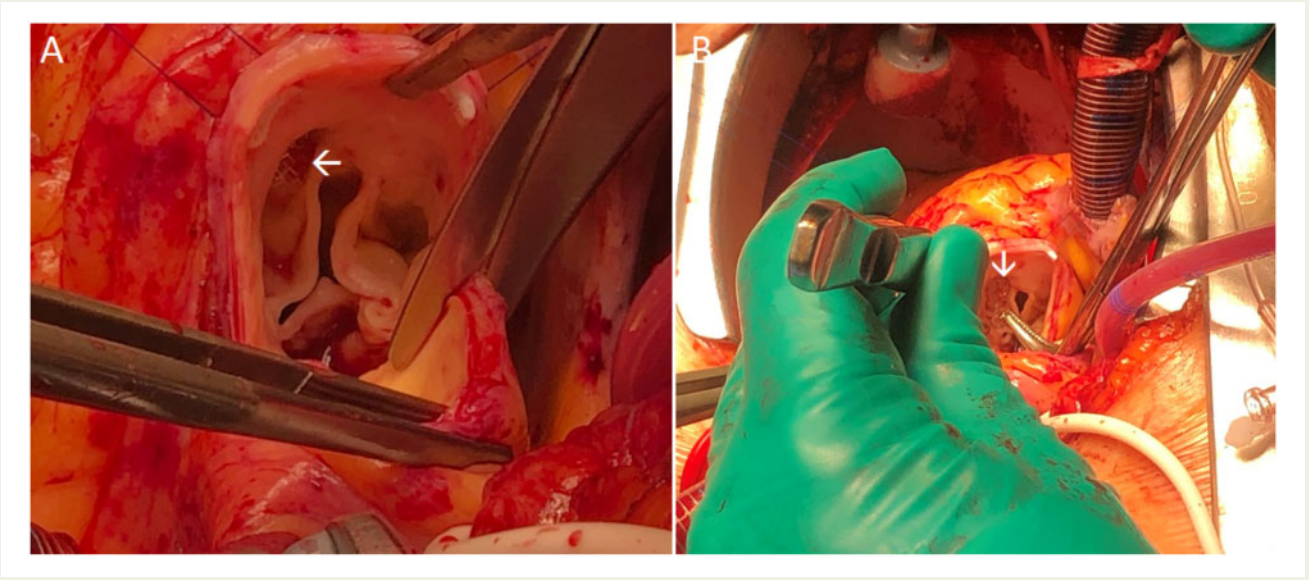


Figure 5 Surgical aortic valve replacement to a mechanical aortic valve prosthesis was performed. (*A*) The aortotomy was performed and the protruding right coronary artery stent was visualized (arrow). (*B*) The protruding stent was grasped and gently pulled with forceps and the trimmed at the level of the right coronary artery ostium using a Mayo scissor (arrow).

Table 1 Reports of coronary angiography/PCI-induced severe aortic regurgitation

Author (year)	Gender/age	Mechanism of AR	Cause	Acute/chronic	Surgical strategy
Madigan (2020)[5]	M/44	Laceration of LCC	Catheter/wire	Acute	Valve replacement
Kolla (2018)[6]	M/67	Laceration of RCC	Catheter	Acute	Valve repair
Gamaza Chulián (2017)[7]	M/63	Laceration of NCC	Catheter	Acute	Valve replacement
Roy (2016)[8]	M/55	Laceration of NCC	Catheter/wire	Acute	Valve replacement
Grisoli (2012)[9]	M/69	Laceration of NCC	Catheter	Acute	Valve replacement
Shim (2012)[10]	1. M/70	1. Laceration of NCC	1. Catheter	1. Subacute	1. Valve replacement
	2. F/72	2. Laceration of NCC	2. Catheter	2. Acute	2. Valve replacement
Rolf (2011)[11]	M/56	Laceration of NCC	Catheter	Acute	Valve replacement
Ong (2011)[4]	1. M/60	1. Perforation of LCC	1. Catheter	1. Acute	1. Valve replacement
	2. F/84	2. Perforation of LCC	2. Catheter	2. Acute	2. Valve replacement
Bouabdallaoui (2011)[12]	M/41	Laceration of NCC	Catheter	Acute	Valve repair
Safar (2010)[13]	M/66	Prolapse of NCC	Catheter/wire	Acute	Valve replacement
Quintana (2009)[14]	M/49	Perforation of NCC	Stent	Acute	Valve replacement
Kotoulas (2007)[15]	M/66	Perforation of RCC and LCC	Catheter/stent	Acute	Valve replacement
Fundaró (1996)[16]	M/54	Laceration of RCC	Catheter	Acute	Valve repair
Denyer (1988)[17]	M/59	Perforation of RCC	Catheter	Chronic	Valve repair

AR, aortic regurgitation; M, male; F, female; LCC, left coronary cusp; RCC, right coronary cusp; NCC, non-coronary cusp.

during systole (seen in the mid-oesophageal aortic valve long-axis view, *Video 3*), making it progressively fibrotic and retracted. Our theory is supported by the fact that the patient underwent a cMRI which revealed a mild to moderate AR and only mildly abnormal LV dimensions about 2 years after the first PCI and subsequently developed progressive symptoms and LV dilatation during the following 6–12 months. Another potential mechanism behind the fibrotic RCC might be the impact of the DES on the aortic valve. It has been shown that some patients are predisposed to recurrent in-stent restenosis due to a hypersensitivity reaction causing inflammation.[18,19] Indeed, our patient had no less than five recurrent in-stent restenoses, raising the possibility that the aortic valve was affected by recurrent exposure to the eluted drug or the metal from the protruding stent. Histological examination of the aortic valve could have helped to reveal the mechanism, but this was not performed.

Among the 16 reported cases, 12 patients underwent aortic valve replacement while aortic valve repair was possible in 4 patients (*Table 1*). Small perforations and lacerations seemingly had a higher probability of being repaired. Although initially planned, it was not possible to perform aortic valve repair in this patient due to the poor quality of the RCC.

Conclusions

Acute severe AR is uncommon after coronary catheterization or PCI but may have serious consequences for the patient. We report a rare case of iatrogenic chronic severe AR, where the protruding RCA stent probably caused a repeated mechanical trauma to the aortic valve, making the RCC fibrotic and retracted. This illustrates that severe AR can appear as a delayed complication after PCI, which should be acknowledged as a potential risk when dealing with an ostial coronary artery stenosis.

Lead author biography

Dr Johan Wedin is currently working as an intern at Uppsala University Hospital and as a PhD student at the Department of Cardiothoracic Surgery, Uppsala University Hospital, Sweden. He also has a background as a cardiac sonographer. His areas of interest include valvular heart disease, speckle-tracking echocardiography and diastolic dysfunction. Dr Wedins' research focuses on echocardiographic and bioinformatic characterization of bicuspid aortic valve disease.

Supplementary material

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

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

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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