

CASE REPORT

GUIDER INDUCED CORONARY DISSECTION WITH RETROGRADE EXTENSION TO AORTA

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43 years old male, current smoker, with a history of dyslipidemia was admitted with unstable angina. His physical examination was unremarkable. Electrocardiography demonstrated minor ST-T changes with Q waves in inferior leads. Medically refractory angina developed after hospital admission and he underwent cardiac catheterization three days later.

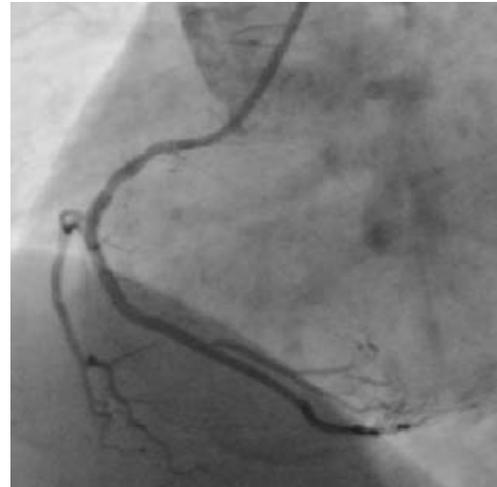
Cardiac catheterization revealed dominant RCA with critical tubular lesion in mid RCA. (Fig 1) There were minor luminal irregularities in proximal LCX and proximal RCA. Left ventricular function was normal. PCI was the preferred treatment strategy.

Fig. 1 - Severe tubular lesion in mid RCA.



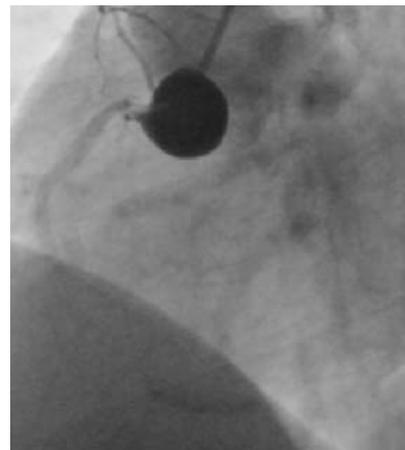
RCA was engaged with 6Fr Judkins right (JR4) side holes guiding catheter because there was damp in pressure. The lesion was crossed with BMW wire, than we pre-dilated the lesion with 2.5x10 mm compliant (sapphire) balloon at 12 atmospheres for 20 seconds. The lesion was covered with 2.75x23 mm sirolimus eluting stent. (Fig 2)

Fig. 2 - Successful PCI – Mid RCA



After stent deployment in mid RCA, there were no symptoms, ECG changes and had stable haemodynamics. Final angiographic views showed dissection of proximal RCA with dye staining in right coronary sinus (Fig. 3)

Fig. 3 - Dissection with retrograde extension to right coronary sinus



At this point patient was asymptomatic and hemodynamically stable and no significant ECG change was noted. Again the lesion was crossed with

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BMW wire and another bare metal stent of 2.75x29 was placed at 12 atmosphere for 20 seconds in proximal RCA covering the ostium of RCA. After stenting, the area of contrast staining in aortic sinuses was minimal. (Fig-4) After procedure patient remains pain free and hemodynamically stable

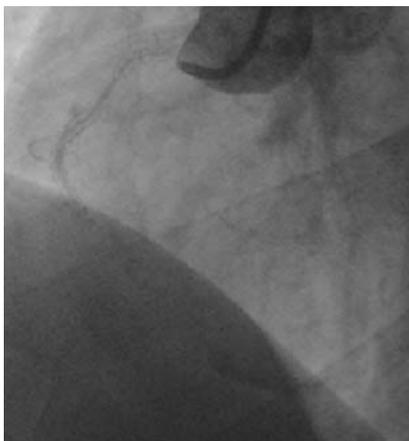
Fig. 4 - PCI-Proximal RCA with minimal dye staining of Coronary Sinus



Further hospital course was uneventful and patient was discharged in FC 1.

On routine follow up patient was enquired regarding symptoms and ensured about the compliance of dual antiplatelet therapy. After three months of initial procedure patient complained of chest pain of FC-3 with SOB. Repeat angiogram showed total in-stent restenosis of prior proximal RCA stent. (Fig 5)

Fig. 5 - Diffuse ISR



PCI was advised again. RCA was engaged with 6 Fr Judkins right guiding catheter, lesion was crossed with

fielder FC wire, and pre-dilated with 2.5x15 balloon at 12 atmosphere for 20 seconds, finally 3x33 sirolimus eluting stent (SES) was deployed at 18 atmosphere for 30 seconds. TIMI-3 flow established at the end of procedure (Fig.6). Patient remained stable during and after the procedure.

Fig. 6 - TIMI 3 flow after DES



At present, patient is asymptomatic and in FC 1.

DISCUSSION

Catheter induced dissection with retrograde extension to aortic root, a feared complication of cardiac catheterization is rare and has been estimated to occur in approximately 0.008-0.2% of diagnostic catheterization and 0.06-0.07% of percutaneous interventions (PCI)^{1,2}, but the overall incidence of catheter induced dissection remains unknown. The majority of cases involve the right coronary artery (RCA) and rarely the left main coronary artery (LMCA). This may be because periostial and sino tubular junction of LMCA are formed by more smooth muscle cells and by a dense matrix of collagen type 1 fibre. This is in contrast to similar areas of RCA.^{3,4} Histologically, the ostium and the first 2 to 4 mm of LMCA lack adventitia, with aortic smooth muscle arranged perpendicular to and surrounding the ostium. The tunica media of LMCA is largely made up of more circulatory and spiral smooth muscle cell than RCA. These cells are arranged in concentric layers with abundant elastic fibers, which could explain the LMCA being more resistant to retrograde dissection⁵. Risk factors for aorto coronary dissection include hypertension, older age, extensive atherosclerosis and underlying structural weakness of media (eg, cystic

medial necrosis)⁶. Pande et al⁷ reported a case of iatrogenic aortic dissection during PCI of RCA in a patient with cystic medial necrosis. Our patient was not hypertensive and without aortic root dilatation, and had neither clinical evidence nor family history of Marfan's syndrome or other causes of medial necrosis, although we don't have any histopathological specimen. A history of myocardial infarction (MI) has been suggested as a cause of aorto-coronary dissection. Dunning et al⁸ described two patients with an extensive aorto-coronary dissection. Both of these patients underwent coronary angiogram due to acute MI. Our patient also had a recent acute coronary syndrome but not ST-elevation MI. Other reported risk factors include variant anatomy of the coronary ostia (e.g., downward sloping origin of the left main coronary artery), vigorous hand injection of contrast material, and even vigorous inspiration during contrast injection. The size of the diagnostic catheters may also be important. We routinely use 6 Fr catheters for this purpose, in this case a side hole guider was used as there was damp in pressure. When there is ventricularization or damping of aortic pressure, corrective measure by using guide with side holes can give false security because the tip of the guide could be located under a plaque and manipulation or injection of contrast could cause severe dissection.¹⁰ Retrograde dissection of the CSV is easily diagnosed by coronary angiography, which usually reveals dye staining persistently localized or extending to the entire aorta when this angiographic finding was observed, the forceful injection of contrast medium should be avoided in order to prevent the propagation of the dissection. If there is retrograde propagation of the dissection to involve the aortic wall, the extent to which the aorta and its branches are involved will dictate the immediate management. Aorto-coronary dissections can remain localized to the sinus of valvula, or may extend into ascending aorta.¹⁻³ They have even been described to extend to the aortic bifurcation⁹. It has been recommended that cases of localized aorto-coronary dissections not complicated by ischemia or hemodynamic instability can be managed conservatively. However if ischemia of any aortic branches occurs, if there is extensive dissection or if there is hemodynamic instability, urgent surgery is the treatment of choice. It is reasonable to attempt to seal the entry site of dissection with PCI and stenting first, and then the extent of dissection can be assessed. A cardiac surgical consult should be sought early. Any

ischemia, hemodynamic compromise or extensive dissection should prompt immediate treatment with surgical repair of aortic dissection. However in the absence of these high risk features, class I or II dissections may be managed conservatively¹¹. A proposal has been made by Dunning and colleagues⁸ for classification of system based on extent of aortic involvement:

Class I: the contrast staining involves only coronary cusp.

Class II: Contrast extends up to aortic wall < 40 mm.

Class III: Contrast extends > 40 mm up the aortic wall.

In their series patient with class III had poor outcomes. This classification may be useful for risk stratification. According to this classification our patient had class 1 dissection with favorable long term outcomes. Moles et al⁹ reported the first cases of aortic dissection as a complication of PCI. Their 2 cases had different evolutions. In their first case, the dissection of aorta was limited to left coronary sinus of valvula (CSV) and surgical intervention was not needed. In their second case, surgical intervention was necessary because the entry was in the aortic intima adjacent to conal artery, leading to dissection of ascending aorta. Dunning and colleagues⁸ described two patients with class 3 dissection who were submitted to surgery and who expired before discharged. Sutton et al¹² described a case in which retrograde dissection of aorta necessitating urgent surgical repair occurred during an attempt to open a chronically occluded RCA. A patient reported by Varma et al⁴ with RCA dissection during PCI extending into the aortic root died within 48 hours with conservative treatment.

In the present case iatrogenic guider induced aortic dissection was possibly due deep throated placement of the guider (with the side hole. In the presence of side hole any damp in pressure which gets masked which might have predisposes to dissection with forceful injection hitting some plaque. Dissection was limited to coronary cusp (class 1) which was successfully managed with stenting of ostium and proximal segment of RCA.

It is always useful to assess carefully before initiating a procedure regarding appropriate size and shape catheter to avoid the contrast injection being directed

at a plaque and contrast should not be injected if pressure is damped as this may be due to the catheter resting against plaque in the artery and extra careful approach if side hole diagnostic guiders are being used which masked the pressure damp. There should be careful removal of a deep throated guider in the presence of plaque in the proximal segment of RCA. Initial contrast injections for final injection should not be forceful until correct coaxial alignment of the catheter and injection should not be directed at the plaque. Finally, the choice of guide catheter is a risk-benefit tradeoff between extra backup and the possibility of deep intubation and subsequent coronary artery dissection. This decision must be at the operator's discretion and must be made on a case-by-case basis; however, the operator should be aware of the possibility of dissection when using more aggressive guide catheters .

CONCLUSION

The present case had an initial aorto-coronary dissection due to deep engagement of right coronary side hole guider that was treated with bare metal stenting, which was followed by total or diffuse ISR most likely due to long BMS stents and at ostial location. Finally managed with placement of in-stent drug eluting stent

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