Case Report

Management of a case of dynamic (intermittent) severe ischaemic mitral regurgitation

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Abstract

We report a patient with Canadian Cardiovascular Society (CCS) class III-IV symptoms and intermittent pulmonary oedema, with 2D echocardiogram evidence of non dilated left ventricle (LV) with grade 3-4 mitral regurgitation (MR) during episodes of pulmonary oedema and almost complete recovery in between episodes. The patient's coronary angiogram revealed a proximal left circumflex (LCx) artery critical stenosis which was treated with percutaneous coronary intervention (PCI) with implantation of a drug eluting stent (DES) and resulting in improved clinical status. Post procedure 2D Echocardiogram revealed minimal MR with normal LV function. Approach to the patient with ischaemic mitral regurgitation (IMR) is a controversial topic as IMR can present in 3 different settings. They are, IMR in acute myocardial infarction (AMI), IMR in chronic phase with remodeled LV, and intermittent IMR in non remodeled LV. This case illustrates the value of PCI as a viable option for properly selected cases with intermittent IMR with non remodeled LV.

Introduction

Mitral regurgitation (MR) can be classified as primary and secondary. Primary MR is usually due to an organic disease of the mitral valve (MV) itself especially involving the mitral valve leaflets. Secondary MR is usually due to dilatation of the mitral annular ring following LV remodeling in cases of cardiomyopathy or myocardial ischaemia affecting the functionality of the MV. The mechanism of ischaemic MR(IMR) is a complex phenomenon. In secondary MR the MV leaflets can have different functionality depending on localized remodeling, chronic LV remodeling, papillary muscle or chordal rupture and transient functional abnormality with intermittent ischemia. Basically IMR is classified as 3 different clinical entities. Firstly, acute IMR which is seen in AMI usually due to damage or ischaemia to the posterior papillary muscle. The condition is serious, and warrants urgent surgical revascularization with MV repair or replacement.[1] Secondly, chronic IMR is seen in chronic myocardial ischaemia with LV remodeling and this entity is more commonly encountered in clinical practice.[2]

Chronic IMR of moderate to severe severity with remodeled LV has a good response to surgical revascularization with MV surgical intervention. Thirdly, (dynamic) intermittent IMR occurring in patients with non remodeled LV with myocardial ischaemia due to either right coronary artery (RCA) or LCx territory causing intermittent ischaemia of the myocardium affecting the functionality of the MV.[3] These patients typically present with flash pulmonary oedema with episodes of worsening angina.[4]

The echocardiogram findings may not reveal moderate or severe MR once pulmonary oedema and symptoms are resolved. Therefore this condition is underestimated and frequently missed. MR results from an imbalance between increased tethering forces and reduced closing forces of the MV leaflet(s).[4]

Case presentation

A 48 year old male with no significant past medical history presented with chest tightness on exertion, with episodes of shortness of breath which progressed to class 111 angina over the past one month. He had been treated for one episode of pulmonary oedema at a peripheral hospital. His cardiovascular examination was normal. He had a normal resting ECG, normal left ventricular ejection fraction of 60% with non dilated LV and mild MR with a morphologically normal appearing mitral valve on 2 D Echocardiogram.

He developed flash pulmonary oedema with worsening angina while in hospital. He was resuscitated and treated as acute coronary syndrome (ACS). During the episode of acute pulmonary oedema with ACS, patient demonstrated grade 3-4 MR with mild impairment of LV systolic function and new regional wall motion abnormalities(RWMA) of infero-lateral LV segments from base to apex. However there was no evidence of chordal or papillary muscle rupture. Once the pulmonary oedema was resolved study of the coronary anatomy was contemplated.

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Diagnostic coronary angiography via right radial artery access demonstrated dominant left circumflex artery with normal left main coronary artery and left anterior descending artery ,anatomically dominant LCx showing proximal 99% tight stenosis [Figure 1], and the non dominant, small RCA showing proximal minor stenosis. The diagnosis of single vessel coronary artery disease with proximal LCx critical stenosis was made. It was understood that his pulmonary oedema was due to papillary muscle dysfunction due to myocardial ischaemia of the LCx territory and the diagnosis of (dynamic) intermittent IMR was confirmed. This was a clear demonstration of IMR and PCI was the choice of revascularization mode. Therefore patient was offered the benefit of revascularization by PCI [Figures 1, 2, 3] with DES implantation.

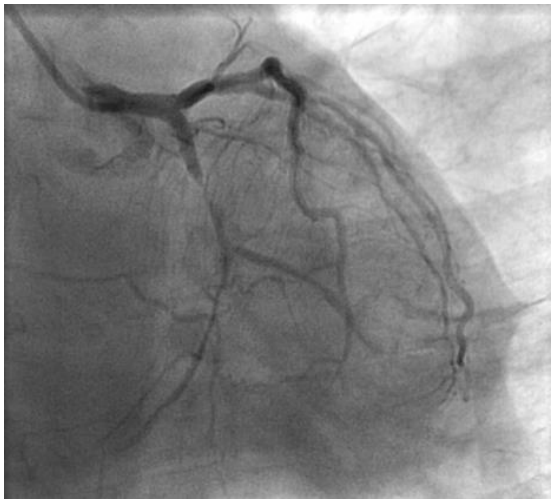

Figure 1- RAO Caudal view showing critical stenosis in LCx.

Patient was observed in the coronary care unit for one day and was haemodynamically stable throughout. He was transferred to the ward on 2nd day and discharged from hospital on day 3 with per oral medications (asprin 75 mg nocte, clopidrogrel 75 mg nocte, atorvastatin 40 mg nocte, enalapril 5 mg mane, bisoprolol 5 mg mane). 2D echocardiogram demonstrated no significant mitral regurgitation and improvement of LV function following revascularization to the LCx. He was reviewed in 2 weeks and 6 weeks follow up and found to be free of angina and shortness of breath with persistently near normal mitral valve function on echocardiogram. He has reported that he has improved exercise tolerance at 6 weeks follow up while on same medication.

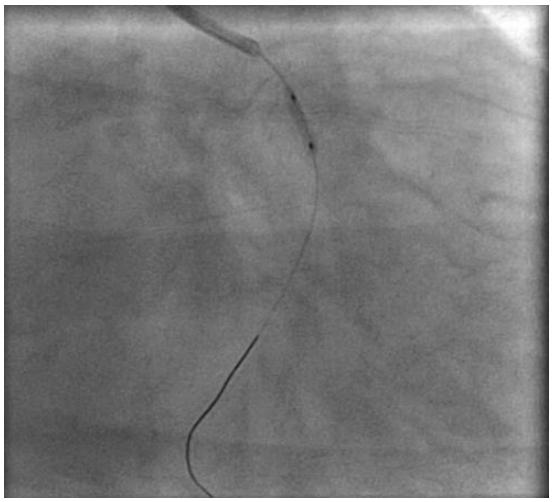

Figure 2- LCx is wired and lesion is predilated with balloon.

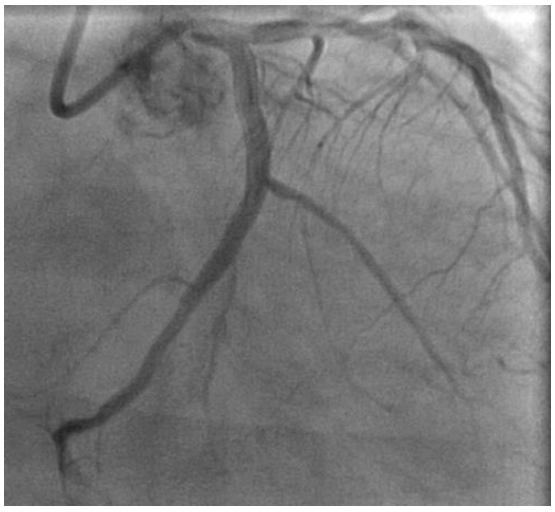

Figure 3- Following stent deployment in LCx

Discussion

This case illustrates a patient with proximal LCx critical stenosis with symptoms of stable angina and episodic shortness of breath presenting with acute coronary syndrome. His initial 2D echocardiogram demonstrated normal appearing MV with mild mitral regurgitation without papillary muscle damage or chordal rupture. LV ejection fraction (LVEF) was normal with no significant regional wall motion abnormalities. He developed acute pulmonary oedema with worsening angina while in hospital and 2D echocardiogram revealed severe mitral regurgitation with LVEF of 50%, confirming papillary muscle dysfunction with severe MR which almost completely resolved following recovery of pulmonary oedema.

The MR was therefore diagnosed as IMR with dynamic variation due to intermittent myocardial ischaemia in the RCA or LCx territory affecting the posterior papillary muscle of MV.[5]

IMR with restricted motion of leaflet(s) is usually seen following an AMI.[6] IMR can occur with normal leaflet motion and isolated annular dilation in certain patients with isolated basal MI. In others IMR can occur with excess leaflet motion resulting from either an acute (ruptured papillary muscle) or chronic (fibrotic and elongated papillary muscle) following a myocardial ischaemic event. Chronic IMR is seen in patients following LV remodeling subsequent to chronic myocardial ischaemia. This clinical entity is more commonly encountered in practice.

Acute ischemia with papillary muscle dysfunction that would reverse with revascularization alone not requiring MV surgical intervention is now recognized to be valid in only a small percentage of patients with IMR. [7] Our patient belongs to this group.

Trivial IMR is inconsequential and can be left alone. Management of mild to moderate IMR is controversial. In patients with mild-moderate IMR, the ischaemic symptoms usually will help to decide the treatment strategy other than medical management. Severe IMR generally needs MV surgery if and when the patient is recommended for CABG as the method of revascularization. However dynamic acute severe (intermittent) IMR with papillary muscle dysfunction due to LCx critical lesions could be successfully treated with revascularization by PCI.

Conclusion

Diagnosing acute intermittent IMR accurately can be a challenging task, as it could be dynamic and hence easily missed. Our case illustrates that acute intermittent IMR can be successfully treated with timely PCI.

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References

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