

Case Report

Acute Mitral Regurgitation: an important cause of respiratory distress

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Abstract

Acute mitral regurgitation (acute MR) is a rare cause of acute respiratory distress, which can present diagnostic challenges. We present the case of a 57 year old man who developed acute shortness of breath subsequently associated with fever, raised white cells and elevated CRP. Chest x-ray revealed unilateral shadowing and he was treated for pneumonia, despite the finding of severe mitral regurgitation on echo. Failure to respond to antibiotic treatment following 3 weeks on ITU led to the consideration of acute MR as the cause of his symptoms and he responded well to diuretics. He subsequently underwent mitral valve repair. The causes and clinical presentations of this condition are discussed.

Learning points:

- Consider acute MR in the differential diagnosis for patients presenting with acute respiratory distress or cardiogenic shock.
- An acute inflammatory response is common in patients with acute MR, which may lead to misdiagnosis as sepsis or pneumonia.
- Unilateral (predominantly right sided) pulmonary oedema maybe seen in acute MR.
- Severe mitral regurgitation in the context of normal LV size and function is highly suggestive of acute regurgitation.
- Medical treatment involves supportive care, diuretics, nitrates and intubation if necessary but the aim should be to stabilise the patient for surgery.
- The definitive treatment for acute MR is urgent valve repair or replacement.

Case

A 57 year old gentleman presented to the emergency department (ED) with a two day history of worsening shortness of breath and some mild central chest pain. He described the shortness of breath as acute in onset. The chest pain was not pleuritic and there was no radiation. He denied any fever or cough. He was previously fit and well, worked as a lorry driver and was not on any regular medications. He did not have any risk factors for coronary artery disease.

On admission, his observations were as follows:

Temperature 35.5°C (spiked to 38 degree C within 12 hours of admission), BP 130/80, Heart rate 110 bpm, Oxygen Saturation 88% on 4L oxygen.

Clinical examination revealed he had bilateral crackles with a soft pan systolic murmur. He was noted to be euvolaemic.

A Chest radiograph (CXR), undertaken at the time of admission is shown (figure 1).

Initial bloods showed a high white cell count, troponin of 0.09 and a 6 hour repeat of 0.07. Urea, electrolytes and liver function tests were unremarkable. C-reactive protein (CRP) also peaked on day 3 of admission and remained persistently elevated (table 1)

ECG on admission showed sinus tachycardia with no ischaemic changes.

The initial working diagnosis was of severe pneumonia. Over the next 24 hours he developed haemoptysis and required increasing amounts of oxygen to maintain saturations of just over 90%.

He was moved to the intensive therapy unit (ITU) where he was intubated and ventilated.

The repeat CXR prior to ITU transfer is shown (figure 2).

Following transfer to ITU, a Trans Thoracic Echo (TTE) was arranged in light of the murmur that had been identified. This showed he had normal Left Ventricle (LV) size and function in the presence of severe posteriorly directed Mitral Regurgitation (MR). A moderately dilated Left Atrium (LA) and normal Right Ventricular (RV) size and function was noted. Abnormal thickening of the mitral valve with Barlow appearance (severe prolapse) was noted which raised the possibility of a vegetation.

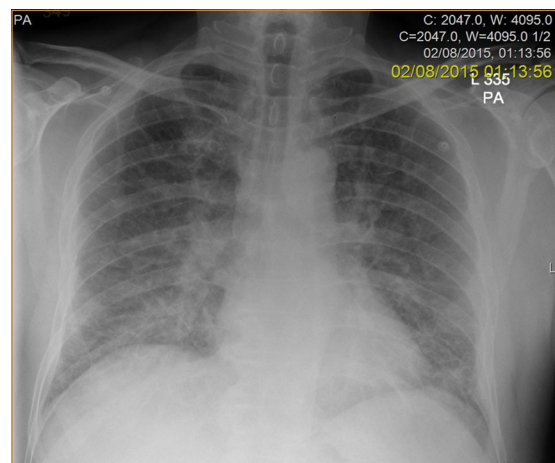


Figure 1. Admission chest radiograph

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Table 1. Sequential blood results following admission

Collected date		02-Aug-15 00:43	02-Aug-15 06:05	04-Aug-15 00:00	04-Aug-15 08:24	05-Aug-15 06:00	05-Aug-15 15:00	06-Aug-15 05:15	07-Aug-15 07:00	08-Aug-15 00:00
Hb	g/L	158	161	149	151	127	134	126	117	115
WBC	10 ⁹ /L	14.8	14.8	26.3	15.8	13.6	15.2	12.0	9.8	10.8
NEUT	10 ⁹ /L	12.5	12.8	24.0	13.9	10.5	11.4	8.7	7.1	9.3
PLT	10 ⁹ /L	145	136	131	108	145	133	121	107	106
Troponin	ug/L	0.09	0.07							
CRP	mg/L	3	9	218	200	233	235	198	278	243

(Normal Values : Hb 120 - 160g/L, WBC 4 - 11 10⁹/L, NEUT 2 - 7.5 10⁹/L, PLT 150 - 400 10⁹/L, Troponin < 0.04ug/L, CRP < 7 mg/L)

A Trans-oesophageal Echocardiogram (TOE) was undertaken within 24 hours to further investigate this. This confirmed severe MR with flow reversal in the pulmonary veins but no evidence of endocarditis was seen. The suggestion of a possible vegetation on TTE reflected the severe prolapse of the Anterior Mitral Valve leaflet (AMVL). There was also no clinical suspicion of endocarditis.

The severe MR was not pursued further at this stage as it was felt it was coincidental and the clinical suspicion of severe pneumonia remained, supported by the high white cell count, elevated CRP, fever, mainly unilateral CXR findings, acute onset and associated Acute Respiratory Distress Syndrome (ARDS). Despite ongoing treatment on ITU his respiratory failure did not improve. Furthermore, initial blood cultures were negative.

After he had been ventilated for 3 weeks, it was felt his MR and fluid overload were significantly contributing to his difficult respiratory wean; he was commenced on intra-venous (IV) diuretics and he slowly started to improve. His tracheostomy was decannulated and he was transferred to the ward after a total of four and a half weeks' stay in ITU.

At this point, the history and echo images were reviewed again by the cardiology team. The dramatic improvement in his condition following treatment with diuretics raised the suspicion of acute MR being the cause of his presentation. The abrupt onset of his symptoms supported the suspicion that his initial presentation was likely following a primary mitral valve leaflet prolapse. He was transferred to the cardiology ward and was worked up for mitral valve surgery. There were some initial delays in going ahead with the surgery due to the deterioration in his lung function tests; a result of his severe MR and prolonged ITU stay. He stayed on the ward for another 10 days during which his lung function was optimised with continued diuresis. He then went on to have a successful mitral valve repair. His post op recovery was uneventful and he was discharged home a few days later. He continues to make good

progress with exercise tolerance and now walks at least a mile every day.

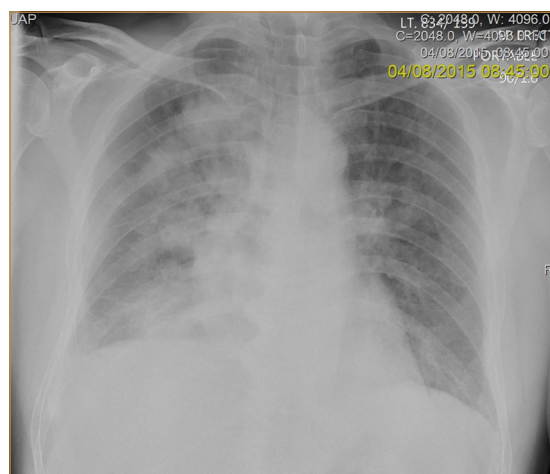
Discussion

Acute MR is often misdiagnosed as a primary pulmonary process or as heart failure due to LV dysfunction. Echocardiography is diagnostic and a high level of clinical suspicion is needed.

Acute MR could be due to defects in any of the structures involved in mitral integrity; chordae tendineae rupture (secondary to myxomatous disease, trauma, spontaneous rupture, infective endocarditis, and acute rheumatic fever), papillary muscle rupture (secondary to acute myocardial infarction, trauma) or secondary to a dysfunctional prosthetic valve.¹

Males are affected more than females in those under 20. However in those over 20 there is no difference in prevalence between males and females. Older males (>50) have been noted to be more severely affected.²

Acute MR often presents with signs and symptoms of acute pulmonary oedema. This is often determined by left atrial compliance. In acute MR, the increase in volume of blood in the left atrium results in a sudden increase in pressure in the left

**Figure 2.** Repeat chest radiograph

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atrium. This increased pressure is transmitted back to the pulmonary circulation leading to pulmonary oedema.¹ The sudden left atrial stretch and associated myocardial dysfunction has also been identified as one of the factors that contributes to the acute inflammatory response often seen in acute MR. The elevation in white cell count and CRP often leads to initial misdiagnosis as sepsis due to pneumonia, as in this patient.³ Of note, the CXR in our patient showed these changes were slightly more prominent on the right side, which appeared to support the clinical suspicion of pneumonia. Unilateral pulmonary oedema is well recognised in acute MR, affecting up to 9% of cases; this is frequently right sided, as the regurgitant jet is commonly directed towards the right pulmonary veins in a flail posterior mitral valve leaflet.⁴ In our patient however it was the anterior leaflet that was affected.

Another distinguishing feature to differentiate from infective pathologies may be the often dramatic and acute onset of symptoms. Cardiovascular collapse is a common presentation due to the systolic flow reversal from left ventricle to left atrium. The degree of hemodynamic compromise in acute MR depends upon the cause and severity of MR.¹

In addition to signs of acute pulmonary oedema examination often reveals an audible systolic thrill. The murmur is often harsh and can radiate to multiple sites including the axilla, back and left sternal border. In some cases, however findings can be quite subtle.²

Echocardiography is the investigation of choice in diagnosing acute MR and can often help demonstrate the mechanism and therefore the aetiology of MR. Left Ventricular (LV) size and function can help determine the chronicity of the MR; the presence of normal or hyperdynamic LV in the context of severe MR is suggestive of acute regurgitation.⁵ In patients with chronic regurgitation, acute worsening may be difficult to assess as the ventricular size is enlarged; comparison with prior studies may help in this situation.⁶ If the TTE is inconclusive the TOE can often still help greatly in identifying the aetiology. TOE is also important in planning surgical repair options.

The definitive treatment of severe acute MR remains surgery. The mainstay of medical treatment includes oxygen, diuretics, nitrates, intra-aortic balloon pump, vasoactive drugs and early intubation if respiratory failure results. The crucial point however is that medical management should not delay surgery. The aim of medical management should be to stabilise the patient in preparation for surgery.¹

Mitral valve repair is the optimal choice, given the lower risk of infective endocarditis and better postoperative LV function. However mitral valve replacement is often necessary, particularly if diagnosis is delayed.

The prognosis of patients with acute MR depends on the aetiology of the MR and the effect it has had on the LV function. Complications of acute MR such as acute pulmonary oedema and cardiogenic shock can affect the prognosis and the success of interventions such as surgery. Early recognition of even minimal symptoms is crucial; the earlier the intervention, the more the likelihood of success and preservation of the LV function.² Surgical mortality in acute MR remains high, although the mortality rate is lower than with medical management alone.¹

This case highlights that acute MR can mimic severe pneumonia, as well as acute left ventricular failure, acute respiratory distress syndrome and other causes of pneumonitis. In this case although the severe MR was picked up on his initial echo, it wasn't considered to have been the cause of his presentation and diuretic treatment was therefore delayed. Mitral regurgitation is a common finding amongst medical patients, and the significance of this can therefore easily be overlooked, without a high level of clinical suspicion. Severe MR noted in the context of an abrupt onset of acute respiratory distress or unexplained shock should trigger referral for an urgent cardiology opinion. This patient would have been a good candidate for urgent valve surgery; although he improved with conservative management, the delay in diagnosis contributed to his prolonged hospital stay.

Conflict of interest

Nothing to declare.

References

1. Otto C M (2015) Acute Mitral Regurgitation in Adults. UpToDate. [Online] Available at http://www.uptodate.com/contents/acute-mitral-regurgitation-in-adults?source=search_result&search=acute+mitral+regurgitation&selectedTitle=1%7E30 [Accessed: 9/11/15]
2. DiSandro D, Brenner BE. (2013) Acute Mitral Regurgitation, Medscape, [Online] Available at <http://emedicine.medscape.com/article/758816-overview#a6> [Accessed: 9/11/15]
3. Yusoff MR, Keevil B, Jenkins NP, Ray SC. Activated vascular inflammatory response in chronic severe mitral regurgitation with preserved left ventricular function. *European Journal of Echocardiography*. 2005; **6**; p 68.
4. Morris PD, Warriner DR, Channer KS. Focal pulmonary oedema: an unusual presentation of acute mitral regurgitation. *Thorax*. 2013; **68**(5); p498
5. Gaasch WH, Meyer TE. Left ventricular response to Mitral regurgitation implications for management. *Circulation*. 2008; **118**:2298–2303.
6. Stout KK, Verrier ED. Valvular heart disease: Changing concepts in disease management. Acute Valvular regurgitation. *Circulation* 2009; **119**:3232–3241