

Malaria in a Patient with Undiagnosed Severe Mitral Stenosis: A Case Report

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Abstract

Background: Mitral stenosis can complicate into pulmonary edema and hemoptysis as progress of the disease or from acute hemodynamic changes. An Indian young male previously healthy presented with malaria and pulmonary edema and finally found to have severe mitral stenosis, the association which has not been described previously in the literature.

Case Presentation: A previously self-reported healthy 32 years old Indian male was admitted at emergency then ICU due to hypoxia and vomiting. Prior, malaria was diagnosed and under treatment but his clinical condition was worsening by unstable cardiorespiratory hemodynamics. Ultrasound confirmed severe mitral stenosis and pulmonary edema. After diagnosis, vasopressors and fluids were tapered down and patient discharged in stable condition for surgical operation in his home country.

Conclusion: This is the first reported case of malaria in a patient with unknown severe mitral stenosis. While bedside echocardiography differentiated mitral stenosis as cardiogenic to non-cardiogenic cause of pulmonary edema, the management was challenged by lack of literature. The management was based on physiologic principles. In settings with limited resources, if a patient presents with malaria and cardiorespiratory complications, simplified bedside echocardiography should be done to exclude any cardiac cause. In case of isolated severe mitral stenosis with normal ventricular function, the management should aim normal low heart rate with limited fluid intake.

Keywords: Mitral Stenosis; Hemodynamic; Malaria; Mitral Stenosis and complication

Abbreviations

BP: Blood Pressure; HR: Heart Rate; ICU: Intensive Care Unit; IVC: Inferior Vena Cava; MVA: Mitral Valve Area; NYHA: New York Heart Association

Background

Clinical diagnosing of mitral stenosis in a patient with low threshold of suspicion may be difficult yet missing the diagnosis has grave outcome [1]. While mitral stenosis is a risk factor for patients to develop pulmonary edema, hemoptysis and hypoxia [2] any illness may precipitate these complications [2]. A multidiscipline team approach is important to investigate hemoptysis and hypoxia in context of febrile illness and to affect the treatment outcome. Malaria may complicate into pulmonary edema, acute respiratory distress syndrome, hemoptysis hence hypoxia [3]; failure to differentiate these complications against severe mitral stenosis as possible underlying cause or pneumonia as associated diagnosis in patient having severe mitral stenosis has a deleterious effect. It has implication to management and the outcome of patient. We present a young man presenting with hemoptysis, hypoxia and diffuse lung crackles in the context of malaria and finally found with severe mitral stenosis.

Case Presentation

A previously self-reported healthy 32 years old Indian male was transferred into our hospital from a local clinic due to hypoxia and vomiting. Our hospital is a tertiary University Teaching Hospital that manage advanced cases. Prior to admission in our settings, the patient had a febrile illness associated with headache confirmed to be malaria by blood smear and came on second day of artesunate from the clinic. His symptoms rather than improving, worsened with vomiting, short of breath, chest pain and hemoptoic cough, the reason of referral.

At presentation in our setting, he presented in mild respiratory distress with SPO₂ of 84% on room air and respiratory rate: 22cpm; BP: 106/70 mmHg; HR 129 bpm; T°: 39.2°C; the reported abnormalities on physical exam were diffuse bilateral crackles with reduced breath sound and regular tachycardia, the rest of other systems were normal. Chest X Ray was reported with diffuse infiltrate more in right side of the lung with trace pleural effusions in the right pleural space, though could not differentiate from pulmonary edema, atypical pneumonia or acute respiratory distress syndrome, the radiologist suggested to be more likely pulmonary edema in consideration of clinical scenario and mild pleural effusion. Creatinine of 0.8 mg/dl and urea 22 mg/dl, Na 134 and Cl 108; Full blood count with hemoglobin of 12, platelets of 149, white blood cells of 9.8, neutrophils of 98%; Repeated malaria blood smear came negative; electrocardiogram reported only sinus tachycardia.

The patient was started on Oxygen 5 l/min per facial mask, artesunate continued for malaria, ceftriaxone for possible pneumonia and doxycycline in addition to cover possible atypical pneumonia and metoclopramide to reduce vomiting. Figure 1 shows timeframe of blood pressure and heart rate trends from admission to emergency till admission in Intensive Care Unity.

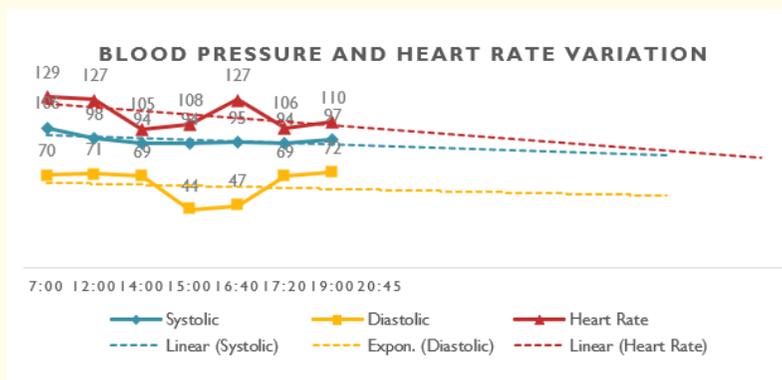


Figure 1: Normal saline 500 ml given slowly at 2 pm and a small dose of Lasix given at 3 pm worsened the hemodynamics.

At emergency, from 7 am till 2 pm urine output was below 200 ml and Coca-Cola in color with dropping BP to 94/44 mmHg, intravenous 500 ml of normal saline was given slowly in considering possible pulmonary malaria complications pulmonary edema or acute respiratory distress syndrome. Rather than improving, the hemodynamic instability worsened with tachycardia and dyspnea with hypoxia requiring oxygen increment; a dose of 20 mg of Lasix was given intravenously at 15:00 with slight improvement of dyspnea. Vital signs kept unstable in association to respiratory distress with hypoxia and increased frequency of vomiting; hence at 8:45 pm, the patient was transferred in ICU for hemodynamic and respiratory support in case of need. Adrenaline 0.01 mg/kg/min in addition to increased high flow oxygen by facial mask was started for hemodynamic support with presumptive impression of either ARDS or non-cardiogenic pulmonary edema due to malaria.

Two days later still in ICU, though fever and vomiting had resolved, the vital signs kept unstable with still diffuse lung crackles. Due to lack of familiarity of bedside chest echography use in diagnosis with patients presenting with respiratory complaints, the mitral stenosis with MVA of 0.8 cm^2 by pressure half time and planimetry (Figure 3 and 6) was diagnosed on 3rd day of admission. The Sonosite M Turbo echography was used to make the diagnosis and establish treatment plan. Parasternal long and short views, apical and subxyphoid imaged were taken (Figure 2-4). The echocardiography was performed on bedside and the patient was of lean body with small intercostal space to capture good images to complete all measurements in the machine's capacity. In comparison to admission, a careful auscultation found a grade 1/4 diastolic rumbling murmur at mitral area, the patient recalled childhood approximately biannual febrile illness treated at his hospital in India and reported that he used to carry all daily activities but could sense dyspnea of physical exercise that he felt it is normal (NYHA class I). Otherwise no other relevant family or past medical history reported by the patient. With use of BLUE protocol in critically ill patient, bilateral B Profile with normal sliding pleural line was noted; 2 to 3 B lines on left lung and 4 to 5 B lines on right lung were noted, in favor of pulmonary edema.



Figure 2: Diffuse lung infiltrates with mild right pleural effusion.

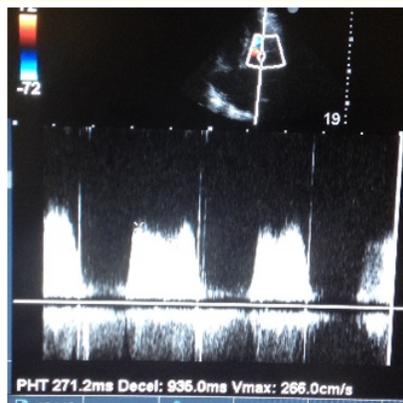


Figure 3: Pressure half time of 271.2 ms in favor of severe mitral stenosis.



Figure 4: MVA of 0.8 cm² by planimetry.



Figure 5: Hockey stick shape of anterior mitral valve leaflet and calcification of valves in favor of mitral stenosis.



Figure 6: Small IVC diameter of 1.39 cm.

With this new diagnosis, the management changed to stepping down adrenaline, no diuretics, judicious fluids in case of need in addition to continuation of antimalarials. He went out of the ICU after one day with clear lungs and discharged off the hospital on infective endocarditis prophylaxis with atenolol and penicillin V tablets and recommended to surgical treatment in home country.

Discussion and Conclusion

This case highlights the importance of a bedside echocardiography in investigating a patient presenting with febrile illness with respiratory signs and hemodynamic instability. It also shows how treating the pulmonary edema or acute respiratory distress syndrome assumptions due to malaria may have a poor outcome in patient with unrecognized mitral stenosis. For this case at admission, respiratory symptoms and signs were not differentiate clinically either as due to malaria complication (i.e. pulmonary edema or acute respiratory distress syndrome) or to primarily pneumonia in association to malaria. With low suspicion of mitral stenosis, malaria confirmation and respiratory symptoms and signs, it was reasonable to think of pulmonary edema or pneumonia associate septic shock or possible ARDS on top of malaria hence the initiated treatment focusing on these entities of diseases. Either fluids, diuretics or vasopressor worsen hemodynamic physiology of mitral stenosis [4,5].

Patients with mitral stenosis may remain asymptomatic or adapt to sedentary [5,6]. It is not uncommon to miss signs of mitral stenosis on auscultation especially in case of low suspicion. Most patients don't recall remote symptoms related to Strep Group A infections, probably the reason he recalled the childhood twice annually febrile illness treated at local hospital after mitral stenosis diagnosis focused history [6]. His tiredness considered normal to his daily activities could be classified as NYHA class I [7].

Pulmonary edema which may be due either mitral stenosis decompensation or malaria was highly suspected based on the BLUE Protocol B-profile found with the patient and it has 97%, 95%, 87%, 99%, sensitivity, specificity, positive predictive value and negative predictive value respectively [8]. The chest x-ray with bilateral diffuse infiltrate with right mild pleural effusion support the diagnosis despite not truly classical with cephalization, acute respiratory distress syndrome could be excluded by the presence of severe mitral stenosis on cardiac ultrasound [9]. The high-grade fever at second day under artesunate would still be from malaria [10] or other infection like pneumonia as the patient presented with diffuse infiltrate not typical to pulmonary edema. Pulmonary edema, in patient with unrecognized mitral stenosis, could have been precipitated by hemodynamic change related to malaria, received fluid, SIRS from malaria or other infection. Though blood culture did not grow and full blood count normal, others infections could not be excluded.

After concluding on pulmonary edema by the BLUE Protocol probably from severe mitral stenosis confirmed by echocardiography in a patient with malaria, the management plan changed. There is no such case in literature reporting the association and how to take care of associated physiologic and pathophysiologic changes. The treatment recommendations were based on physiology concepts to taper down adrenaline, to no or less diuretic and fluids to keep the mean arterial pressure above 65 and HR below 80 and to keep antimalaria, initiated antibiotics and oxygenotherapy. From this case, every effort to assess hemodynamic instability and respiratory compromise by bedside echography to exclude concomitant associated cardiovascular disease should be made, especially in setting with limited resources. Clear guidance is needed in case of managing acute respiratory complication of mitral stenosis in case of conditions raising end diastolic pressure and peripheral vasoconstriction with instable vital signs.

Author's Contributions

JPS drafted the manuscript and got patient consent for publication; HS, internists who took care of the patient; AM, internal medicine Resident who admitted the patient, JCG is anesthetist who followed the patient in ICU. All authors read and approved the final manuscript.

JPS is a consultant internist with interest in echocardiography and pulmonary ultrasound.

Competing Interests

None declared.

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