

All Breathlessness in Mitral Stenosis is Not Pulmonary Edema- A Case of Mitral Stenosis with Cardio Respiratory Arrest

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ABSTRACT

Anaphylactic shock in periprocedural period poses diagnostic and therapeutic challenge. Here we report a case of severe mitral stenosis developed sudden cardio respiratory arrest after parenteral antibiotic injection while being prepared for elective balloon mitral valvotomy. After initial resuscitation patient was taken up for emergency balloon mitral valvotomy in view of refractory shock. Patient developed air embolism during the procedure in view of reduced right atrial pressure which was successfully managed. She underwent balloon mitral valvotomy. Patient developed renal failure and deranged haematological parameters which were managed accordingly, finally the patient made a complete recovery.

Key words: Anaphylactic shock, Air embolism, Balloon mitral valvotomy.

INTRODUCTION

Anaphylactic shock in periprocedural period poses diagnostic and therapeutic challenge. We report a case of severe mitral stenosis developed sudden cardio respiratory arrest after parenteral antibiotic injection before the planned balloon mitral valvotomy (BMV). After initial resuscitation patient was taken up for emergency BMV in view of refractory shock. Patient developed air embolism during the procedure in view of reduced right atrial pressure which was successfully managed. To the best of our knowledge this is the first case report of air embolism into the right sided cardiac chambers during BMV. Patient made a complete recovery subsequently.

CASE HISTORY

A 16 year old girl presented with the complaints of worsening breathlessness for few weeks. Clinical examination, chest X-ray, electrocardiogram (ECG) and

echocardiography revealed features suggestive of severe mitral stenosis with pulmonary hypertension. The valve was suitable for balloon dilatation and she was planned for BMV.

On the day of procedure, routine pre procedure protocol (including injection cefazolin 1 gm intravenous administration after test dose) was followed. She developed sudden cardio respiratory arrest when she was about to be shifted to cardiac catheterisation laboratory. Code blue was announced. ECG showed bradycardia at a rate of 30 /min. Blood pressure was not recordable, no respiratory efforts were noted and was started on cardiopulmonary resuscitation. She was intubated and was connected to the mechanical ventilator. Being a case of tight mitral stenosis possibility of flash pulmonary edema with subsequent cardio respiratory arrest was considered as the first diagnosis. But the preceding events were not typical of the same lungs were clear on auscultation. A second possibility of anaphylactic shock secondary to the antibiotic administration was also considered. She was administered parenteral adrenaline and hydrocortisone. She was started on intravenous inotropes and intravenous crystalloids. Central venous pressure was low (zero). Echocardiography revealed normal left and right ventricular systolic function with severe mitral stenosis. Chest X Ray showed a decrease in the size of cardiac silhouette (Figure 1) when compared

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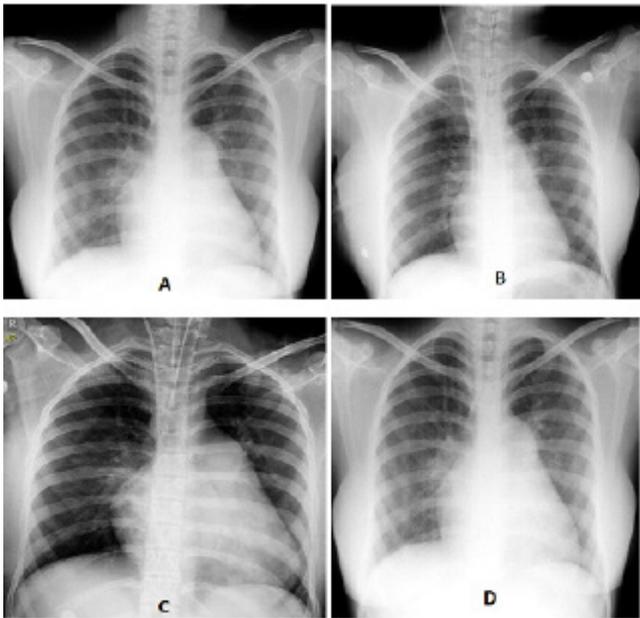


Figure 1: Chest X Ray of the patient at admission (A) showing a prominent main pulmonary artery with pulmonary venous hypertension. Chest X Ray after cardiac arrest showing a decrease in the size of cardiac silhouette with clear lung fields (B). Repeat X Ray after stabilisation showing an increase in the size of cardiac shadow (C) and at discharge the cardiac size is similar to that at admission (D)

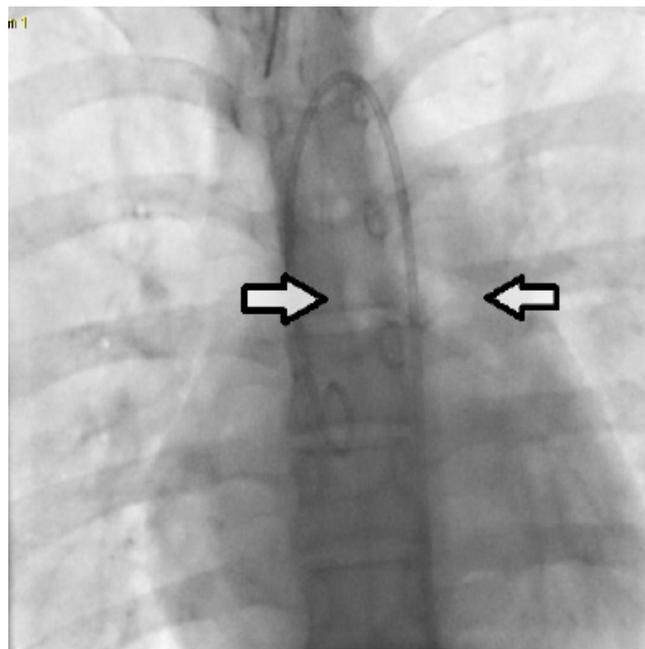


Figure 2 : Fluoroscopic image during the procedure shows the presence of air shadow inside the pulmonary arteries (Arrow mark) during the right heart study.

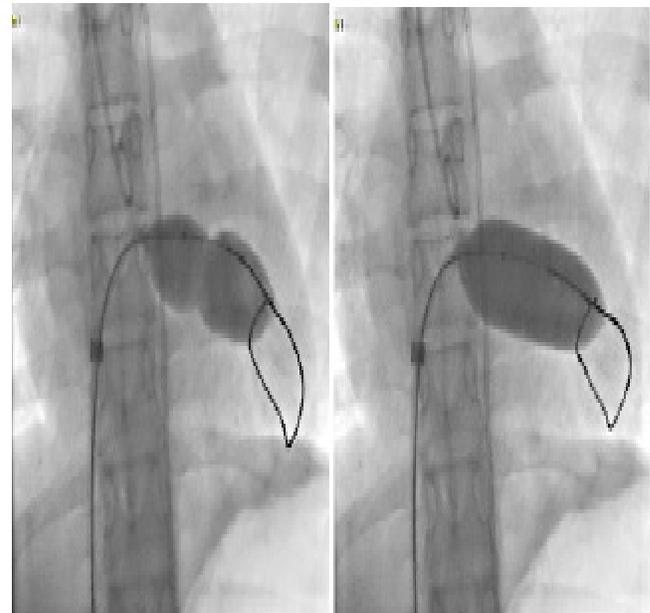


Figure 3: Image showing the Joseph balloon across the mitral valve with a waist (Left) which yielded on subsequent inflation of the balloon (Right)

to the previous X ray. No evidence of pulmonary venous hypertension. In spite of inotropes, steroids and intravenous crystalloids there was no improvement in hemodynamics. Hence a decision to do an emergency BMV was made.

A right femoral arterial and venous access was taken. Right heart study was done with multipurpose catheter showed a very low right atrial pressure (Zero). During the process of right heart study patient developed air embolism into the right ventricle and pulmonary arteries (Figure 2) which was identified by fluoroscopy and was aspirated with the same catheter. Subsequently septal puncture was done with standard technique. Left atrial pressure was measured to 3 mmHg and gradient across the mitral valve was nil. Over the wire BMV was done with 24 mm x 4 cm Joseph balloon (Figure 3). The left atrial pressure post BMV was 2 mm hg. There was no gradient between left atrium and left ventricle during diastole. The aortic pressure was 76/35 mm hg at the end of the procedure.

She was continued on inotropic and ventilatory support. Post procedure she developed bleeding from vascular access sites. She had elevated activated partial thromboplastin time (>150 s) prothrombin time (International normalised ratio was 8.50), decreased fibrinogen (<43 mg/dl) that required multiple units of fresh frozen plasma and cryoprecipitate to normalize. She also developed metabolic acidosis and renal impairment secondary to hypotension which were managed with multiple cycles haemodialysis. Sepsis workup was negative. With all supportive measures she

made a gradual recovery. Her haematological and renal parameters improved gradually and normalised after a period of one week. She was weaned off inotropes and ventilatory support slowly. Her echocardiography at discharge revealed a mitral valve area of 2.5 square centimetres with no significant mitral regurgitation or PAH. A detailed history retrospectively revealed probable evidence of drug allergy in the past.

DISCUSSION

Our patient is a case of severe rheumatic mitral stenosis sustained cardio respiratory arrest while being prepared for balloon mitral valvotomy. Respiratory distress in a patient with mitral stenosis is most commonly due to pulmonary congestion.¹ But in our case patient did not have clinical features typical of pulmonary edema as evidenced by the clear lung fields and she sustained cardiac arrest immediately after the onset of breathlessness which is also not typical of pulmonary edema. Since the patient received an antibiotic injection minutes before the event, anaphylactic shock secondary to the same was also considered as a differential diagnosis. Cephalosporins are among the most commonly-used antibiotics in the treatment of routine infections and their use is increasing over time.² Anaphylactic reactions to cephalosporins are rare (frequency 0.0001 to 0.1 percent).³ Any cephalosporin can cause anaphylaxis.^{4,5} Cephalosporin-induced anaphylaxis may be fatal.^{6,7}

In spite of aggressive fluid management and inotropic support patient's hemodynamics did not improve. Anaphylactic shock refractory to conventional management is rare.⁸ She was taken up for emergency BMV in an attempt to improve hemodynamics. Our patient had a very low right sided chamber pressures because of shift of fluids from intravascular to extravascular space. Massive shift of fluid out of intravascular space in anaphylactic shock is well known.⁹ This explains the decrease in size of cardiac silhouette in chest X Ray.

Per procedural air embolism during BMV is rare.¹⁰ Air embolism in to the left sided chambers or coronary arteries can happen when the balloon ruptures inside the left ventricle.¹⁰ Air embolism into the right sided chambers is uncommon during BMV in view of elevated right sided chamber pressures. Our patient had air embolism during

catheter manipulation inside the right sided chambers in view of low right atrial pressures. To the best of our knowledge this is the first case report of air embolism into the right sided chambers during BMV which was managed successfully. Periprocedural air embolism in to the systemic circulation would have caused major consequences. Post procedure she was noted to have deranged haematological parameters. Deranged bleeding parameters had been reported in anaphylactic shock¹¹ She made an uneventful recovery subsequently.

CONCLUSION

This case was reported to highlight the possibility of anaphylactic shock in periprocedural period which poses diagnostic and therapeutic challenge. This needs to be considered when a patient develops sudden hemodynamic compromise when it is not explained by primary disease or procedure related complications. A detailed drug history and history of drug allergy might have prevented this complication. Air embolism during cardiac interventional procedure is an important complication which needs to be identified promptly for better management.

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