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Atrial Myxoma Complicating The Course Of ARDS: A Case Report

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Abstract

We report a case of a 42 year-old with community acquired pneumonia and hypoxic respiratory failure. Thus, intubated and shifted to intensive care unit (ICU) for ventilator support on (acute respiratory distress syndrome) ARDS protocol. Then, all of sudden his condition deteriorated. Thus, bed side echocardiography was done by the intensivist, which showed a big myxoma causing obstruction to the mitral flow with good systolic function. That can explain the reason for pulmonary edema and the deterioration which happened. He was extubated successfully and then transferred to Mater hospital for surgical removal of the tumour. He was discharged home few days later.

Key words: acute respiratory distress syndrome, atrial myxoma, pulmonary edema

INTRODUCTION

Myxomas are the most common primary cardiac neoplasm. The prevalence of cardiac tumors at autopsy ranges from 0.001% to 0.3%, and more than 50% of benign cardiac tumors are myxomas. In 7%, it has genetic origin and arises as a component of a heritable disorder with some clinical manifestations. Over 72% of primary cardiac tumors are benign. In adults, the majority of benign lesions are myxomas[1,2].

Its clinical manifestations are variable and non specific. Commonly observed symptoms and signs are dyspnea, orthopnea, paroxysmal nocturnal dyspnea, pulmonary edema, cough, hemoptysis, edema, and fatigue[6].

Case report:

We report a case of 42- year old male patient with a history of gout, hypercholesterolemia and ex-smoker. He was admitted to the hospital ward with cough, fever, vomiting and rigors. He was hypoxic on presentation with Spo2 80% on room air which improved on oxygen mask 5L/m to 94%. Initial diagnosis was community acquired pneumonia.

2 days later, he became more hypoxic with respiratory distress and worsening bilateral infiltrates on the CXR (Figure 1A and B). His laboratory findings showed leukopenia (WBC 2.6), Hb 14.9, neutrophils 3.5, lymphocytes 0.66, platelets 188, CRP 103, urea 7.7, creatinine 88, sodium 138, influenza A +ve, ECG was normal, sinus tachycardia, thus he was treated with ceftriaxone, clarithromycin and oseltamavir. ICU on call was consulted at that stage for worsening respiratory distress(respiratory rate 40/minute, heart rate 125 beat/minute, desaturating on 5 liters/minute oxygen), temperature 38.6 C^{o}. He was therefore intubated and moved to the ICU for ventilatory support and further management. ABG: PH 7.36, PaO_2 7.8, $PaCO_2$ 4.8, lactate 0.8, (Figure 2a)

He was treated as severe ARDS P/F was 58 on fio_2 1.0 and PEEP 14, lung protective strategy was applied, sedation and paralysis for 24 hours. He was hemodynamically stable throughout.

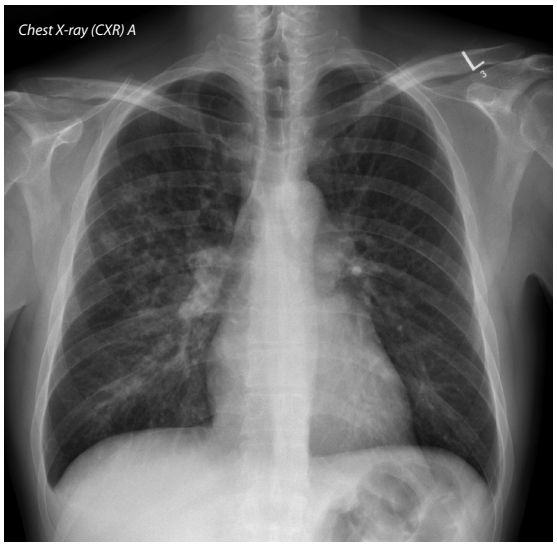

Figure 1A: Showing the CXR on admission and 2 days later, with worsening bilateral lung infiltrates

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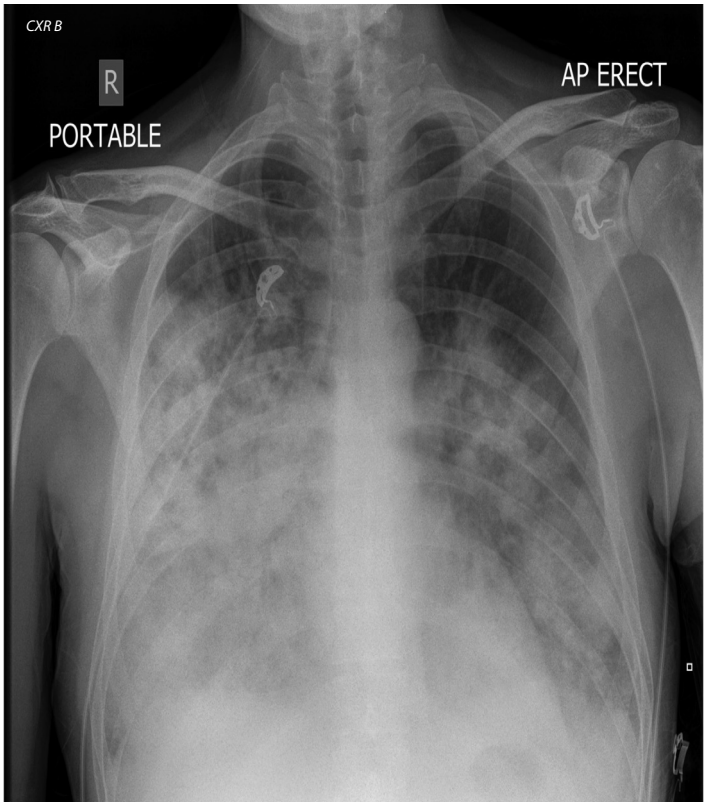

Figure 1B: Showing the CXR on admission and 2 days later, with worsening bilateral lung infiltrates

All septic screening, bronchoalveolar lavage, respiratory panel were –ve except for influenza A positive.

Four days later the ventilatory parameters started to improve, fio2 45%, PEEP 10 cmh_2o on pressure support ventilation, therefore, weaning from the ventilator was planned.

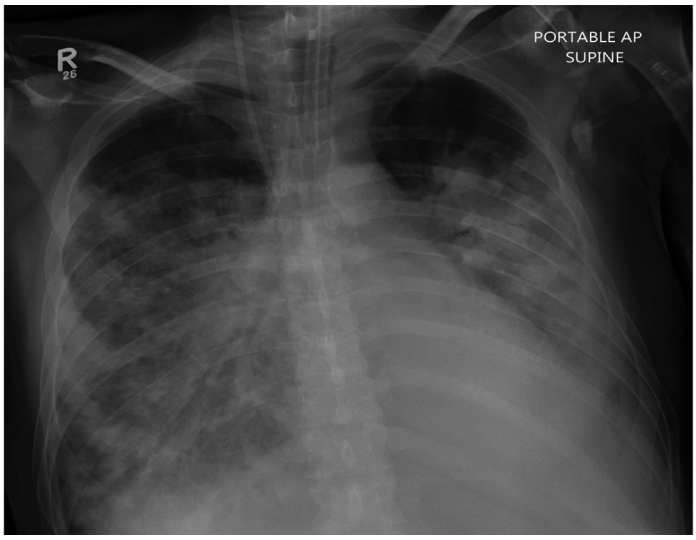

Figure 2A: CXR post-intubation

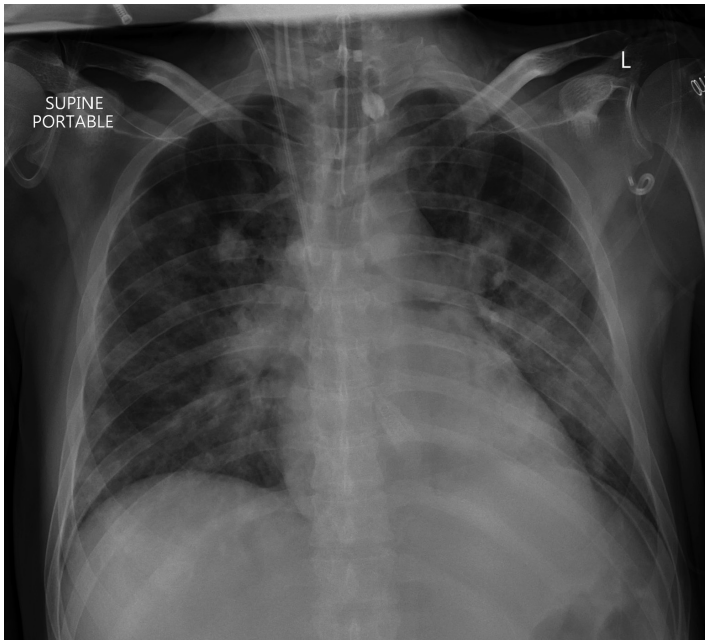

Figure 2B: : CXR on the day of planned extubation

In spite of keeping him on negative fluid balance, all of sudden, two days later, his oxygen requirements increased again to fio_2 90%, PEEP 12 and P/F ratio 90 with radiological deterioration (worsening bilateral infiltrares) (Figure 3).

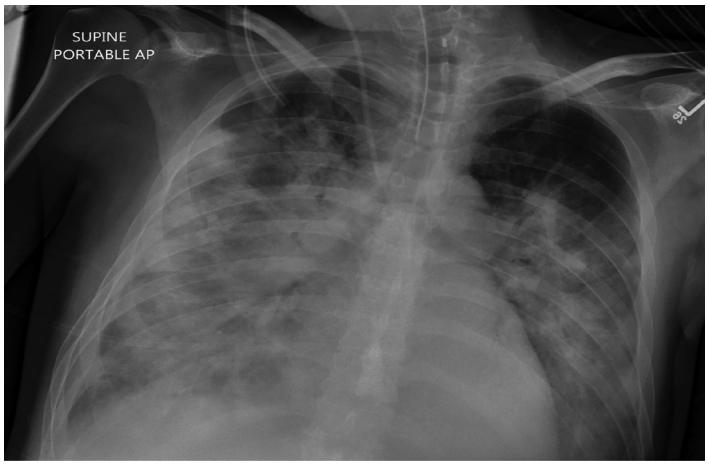

Figure 3: CXR shows worsening bilateral lung infiltrates

Thus, bed side transthoracic echocardiography was performed by the intensivist, which showed a big myxoma (6x4 cm) which is attached to inter-atrial septum and protruding through the mitral valve leaflets to the left ventricle causing obstruction to the mitral flow with good systolic function. That can explain the reason for pulmonary edema and the deterioration which happened (Figures 4 a &b).

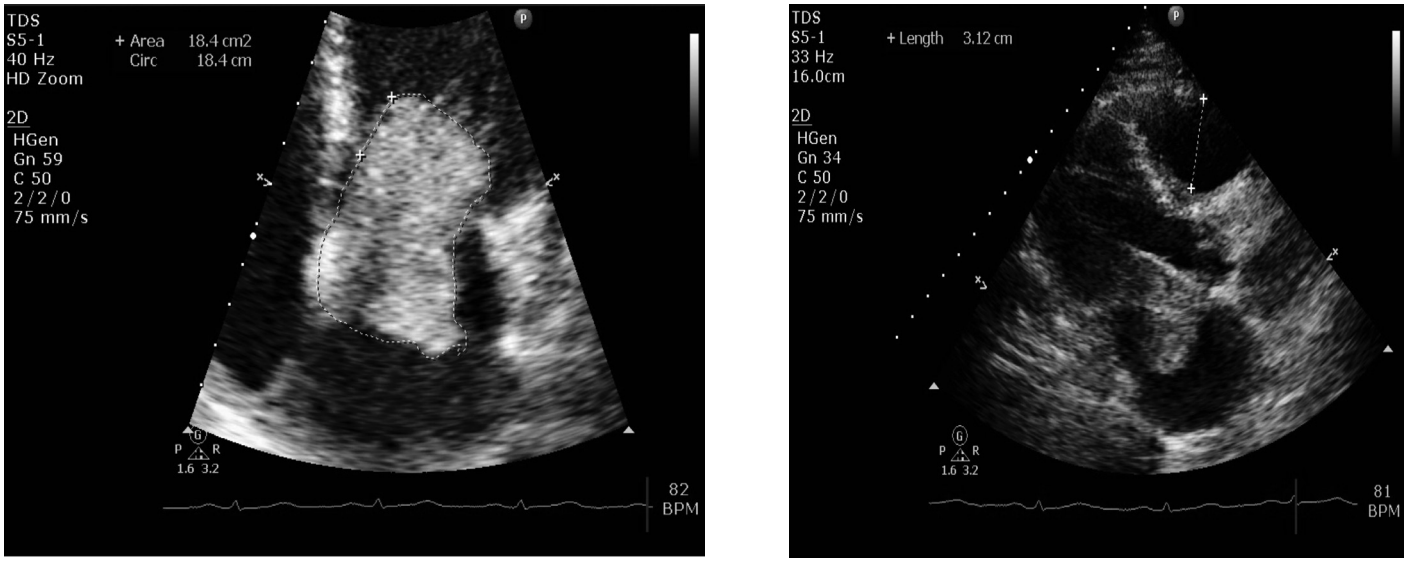

Figure 4A: transthoracic echocardiography shows left atrial myxoma protruding through the mitral valve

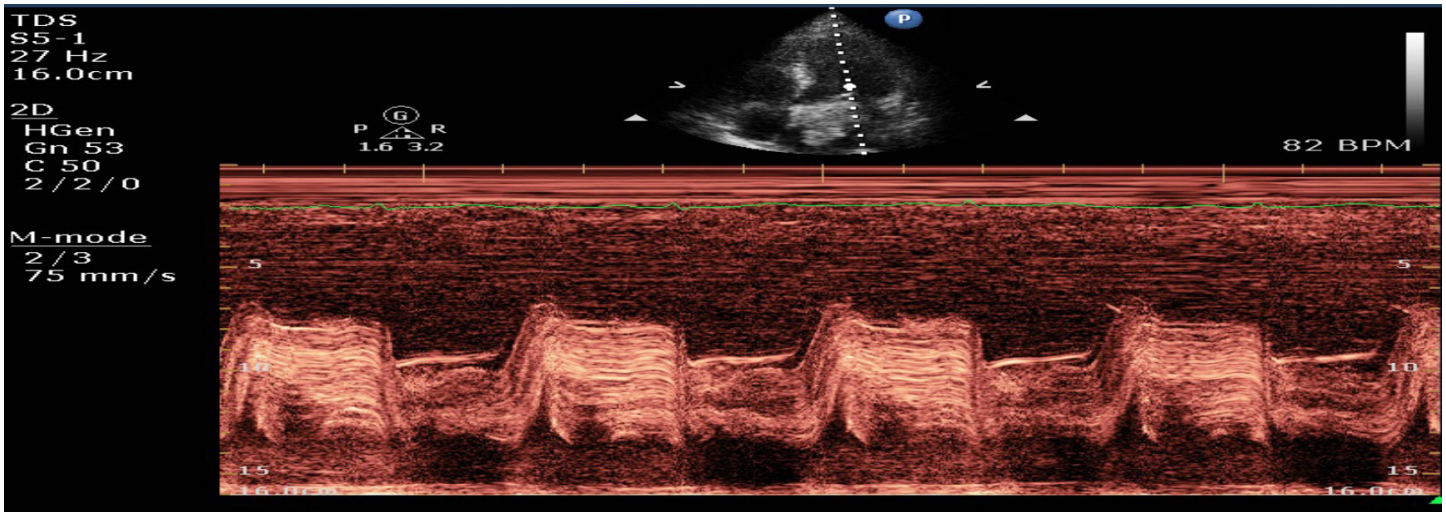

Figure 4B: transthoracic echocardiography shows left atrial myxoma

Cardiologist was contacted to confirm the findings and she did the TTE which confirmed our findings. Microbiologist escalated the antibiotics coverage and amphotericin B was added to cover the possibility of fungal endocarditis, however all were de-escalated when all cultures came back negative. CT thorax was done and showed bilateral consolidations, effusion and cardiac filling defects (Figure 5). Transesophageal echocardiography was done and confirmed the findings.

Five days later a weaning trial was done on pressure support ventilation, Fio2 30% and PEEP 5, ABG: PH 7.47, PaCO2 6, PaO2 11.6, SPO_2 98%, while keeping him on negative fluid balance over the previous days and he was extubated successfully and kept on HFNC 25% with good gas exchange (Figure 6). Mater hospital was contacted and the patient was transferred for urgent cardiac surgery.

The surgery was performed 2 days after transfer and the patient was discharged home few days after that.

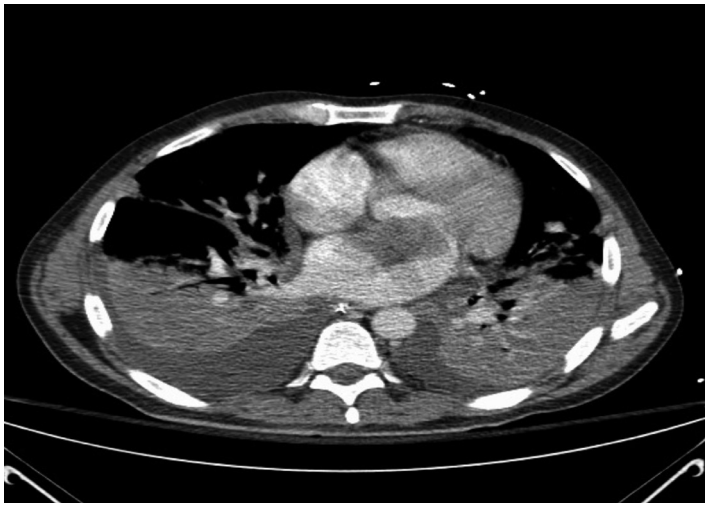

Figure 5: CT thorax

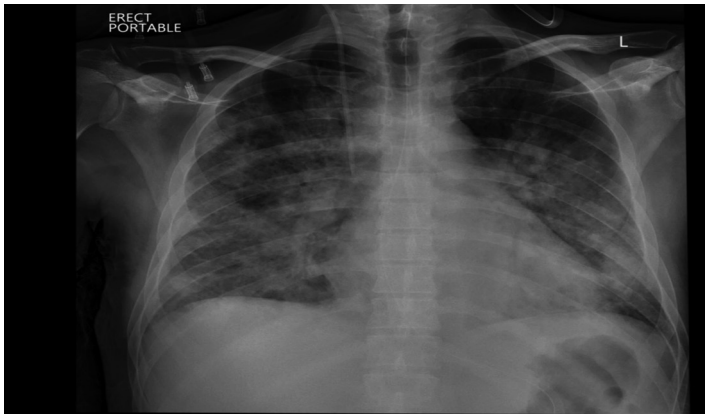

Figure 6: CXR post extubation

DISCUSSION

The ARDS is a well known complication of pneumonia and its management is well established. Cardiac myxomas can present with symptoms secondary to obstruction of blood flow, nonspecific constitutional symptoms such as fever, malaise, arthralgias, rash, or thromboembolic phenomena such as stroke or transient ischemic attack[3,4]. Sudden cardiac death has been reported in the literature, the incidence being only 0.01%–0.005%. Syncope is reported as the most common symptoms in patients with sudden cardiac death, and the cause is obstruction of blood flow either due to ball valve mechanism or due to embolization of tumor to the coronary circulation[5].

In our scenario, when the patient improved and he was ready for extubation, he developed an acute deterioration that mandates finding out the reason. Bedside Echocardiography is widely available and provides a simple, noninvasive technique for rapid evaluation of the cardiac function in addition to detection of any underlying pathology which might contribute to the patient's sudden deterioration. Transthoracic echocardiography is sufficient to confirm the diagnosis of atrial myxoma[6]. Obstructing left atrial myxoma is reported in many cases as a cause of cardiogenic pulmonary edema[7-10]. Moreover, Echocardiography helps in distinguishing cardiac myxomas from atrial thrombus. A thrombus usually arises from the posterior wall of the atrium and is generally immobile and does not have the characteristic stalk[4].

In our patient, the cardiac mass was pedunculated with a stalk arising from the interatrial septum which is the common site of origin of an atrial myxoma. Its definitive treatment is surgical excision.

CONCLUSION

Cardiac masses are rare and they could present with obstructive shock or pulmonary edema which might complicate the course of a disease like ARDS. They need to be diagnosed at the earliest, so as to initiate timely interventions. Echocardiography remains the only modality which makes a point of care diagnosis in cases of cardiac masses and makes an invaluable tool in the armament of an intensivist. This case, however highlights the importance to equip intensivists with point of care ultrasound skills to help in the diagnosis of such cases with confidence

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