Staphylococcus aureus endocarditis complicated by bilateral pneumothorax

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ABSTRACT

Staphylococcus aureus is the most common cause of endocarditis in intravenous drug users. The organism gains access by intravenous injection or from the direct invasion of skin at injection sites. Known for its aggressiveness, the right sided endocarditis that ensues can lead to complications such as pulmonary abscesses and even death. We report the unusual case of an intravenous drug abuser, who following the occurrence of extensive pulmonary abscesses, developed bilateral pneumothoraces within a few days.

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Intravenous drugs abuse is a common social and medical problem. It is associated with serious consequences since the often unsterile injection techniques pose a high risk of infective endocarditis.[1] Associated bacteremia arises from the direct intravenous injection, skin flora or local infections surrounding the injection sites such as cellulites, abscesses and suppurative thrombophlebitis.[2] *Staphylococci* are the cause of more than 60% of cases of endocarditis in intravenous drug abusers, more than all other species combined.[3] The course of the ensuing illness is often acute and potentially lethal.[1] We report a case of right sided staphylococcal infective endocarditis in an intravenous drug user (IDU), who's hospital course, following the development of extensive pulmonary abscesses, was further complicated by the unusual occurrence of bilateral pneumothoraces and discuss the pulmonary complications in IDU.

Case Report. A 37-year-old Saudi male patient was referred to the Accident and Emergency Department of Riyadh Medical Complex, Riyadh, Kingdom of Saudi Arabia, from a mental asylum with a 5 days history of fever, productive cough, and dyspnea class III (NYHA). He had a long history of drug abuse and high serum levels of both opiates and cannabis were reported by the referring institution.

Physical examination revealed a toxic, pale patient with first degree clubbing and an elevated jugular venous pressure of 10 cm from the sternal angle with a prominent V wave. Vital signs showed a temperature of 38.6^{O}C, blood pressure 120/60 mm Hg, pulse rate 100 beats/minute, and a respiratory rate of 34/minute. Chest examination revealed bilateral coarse crackles more on the right side mainly in the upper and middle zones. A loud p2 was evident on auscultation of the heart. Abdominal and central nervous system examinations were normal. The electrocardiogram showed sinus tachycardia and a chest radiograph revealed cardiomegaly and bilateral multiple heterogenous opacities with cavitations within both lungs in the upper and mid-zones. A complete blood count revealed a white cell count of

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20×10^9, Hemoglobin 8.6 g/dl and platelet count of 352×10^3. Coagulation profile showed prothrombin time 16.9 seconds and partial thromboplastin time 41.5 seconds, with an international normalized ratio of 1.37. Cardiac enzymes revealed a creatinine phosphokinase of 57 u/dl and a lactate dehydrogenase 758 u/l. Apart from a raised aspartate serum transferase 112 u/l his liver enzymes were normal. Glucose and renal function tests were normal. While breathing 5 l/minute oxygen via face mask his arterial blood gases showed: pH 7.5, PCO_2 25.4 mm Hg, PaO_2 127 mm Hg, HCO_3 22.5, and an oxygen saturation of 99.3%. Immediately, he was subjected to a transthoracic echocardiogram (TTE), which showed dilatation of the right atrium and ventricle with paradoxical septal motion. The tricuspid valve had 2 large and freely mobile vegetations measuring 14 x 9 mm and 13 x 11 mm, with failure of coaptation of the valve leaflets resulting in severe tricuspid regurgitation with an estimated pulmonary artery pressure of 65-70 mm Hg (**Figure 1**). The inferior vena cava was dilated and non-collapsing, indicating that the right atrial pressure was elevated and in the range of 20-25 mm Hg. He was then shifted to the intensive care unit, and a septic screen was performed. He was started on a triple antibiotic regimen consisting of vancomycin, gentamicin and ceftazidime. On the third day of hospitalization, while he continued having fever, the sputum culture grew methicillin resistant *Staphylococcus aureus* (MRSA), sensitive to vancomycin, clindamycin, and erythromycin, resistant to oxacillin and penicillin G. On the fifth day of hospitalization blood cultures were positive for the same MRSA organisms. That day, he developed a sudden onset of severe shortness of breath and chest pain and a chest radiograph revealed left sided pneumothorax (**Figure 2**). He was sedated, intubated and an intercostal tube was inserted. A TTE was repeated at the bedside and this time showed a new mobile vegetation at the right ventricular outflow tract just above the aortic valve in the parasternal short axis view, while the other vegetations had remained the same size. A complete blood count showed a raised leukocyte count of 23 x 10^3 and hemoglobin of 10.2 g/dl.

It was decided to shift the patient to a tertiary care facility for possible surgical intervention. Before transfer on the ninth hospital day, he sustained a drop in his O_2 saturation to 77% and this time a chest radiograph showed a right sided pneumothorax. (**Figure 3**) Another inter-costal tube, with negative slow-motion suction was inserted and the patient's oxygen saturation improved. He was then shifted to King Faisal Specialist Hospital and Research Center

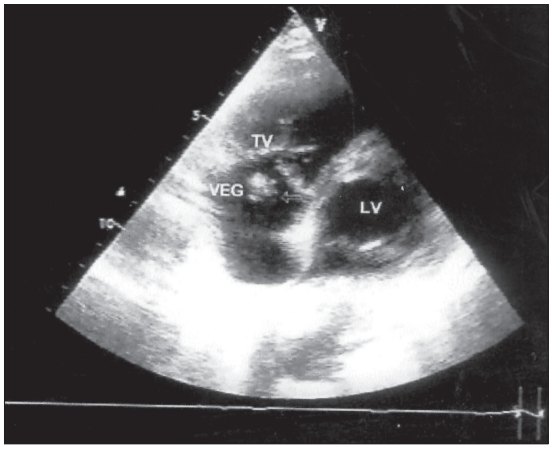

Figure 1 - Transthoracic echocardiogram 4 chamber view showing vegetation (VEG) on tricuspid valve (TV). LV - Left valve.

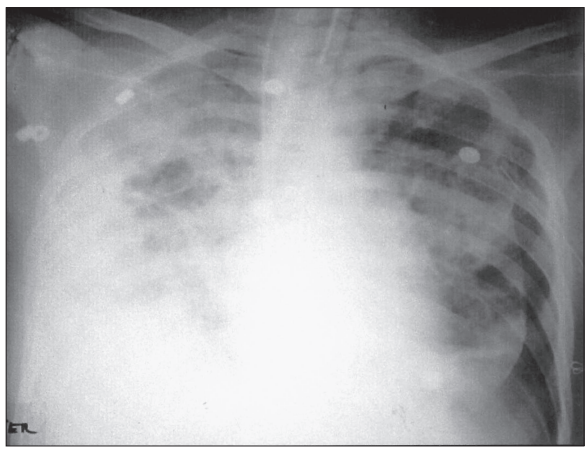

Figure 2 - Chest radiograph showing left sided pneumothorax.

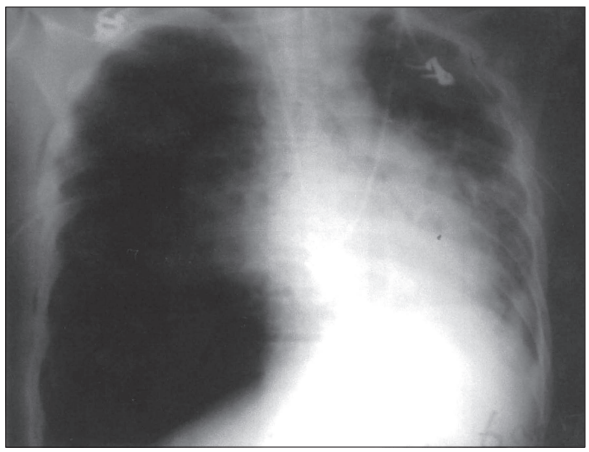

Figure 3 - Chest radiograph showing right sided pneumothorax.

where his case was discussed, and it was decided to continue antibiotics and observe the patient closely by repeated echocardiograms. Three days later the patient had improved, weaned from the ventilator, and was afebrile. However, he administered himself heroin through his central line and eventually, absconded and his family were informed upon, which he was briefly readmitted, but discharged himself against medical advice. No further information regarding the patient after discharge is available.

Discussion. Strains of *Staphylococcus aureus* species cause more than 60% of cases of endocarditis among IDU, more than all other species frequently combined with an acute course.[1,3] The IDU are 300 times more likely to die suddenly with infective endocarditis than are nonusers.[4] Infections are so widespread among IDU due to the presence of the following reported risk factors: a high rate of staphylococcal and other bacteria carriage in skin, mucus membranes and nasopharynx, unsterile injection technique, contamination of injection equipment used or substance of abuse taken, poor dental hygiene, drug induced impairment of cough and gag reflexes, and a low socioeconomic status with exposure to certain pathogens such as tuberculosis.[5,6] Parenteral drug abuse is an important risk factor for endocarditis,[5] even in Saudi Arabia where syringes are freely available. It is important; however, to note that although endocarditis is the infection most commonly attributed to the injection of an illicit substance, skin and soft tissue infection are the most common reasons for admission.[5] Over a 6 month study period of a cohort of addicts only 1% were treated for endocarditis compared with 10% with skin abscesses.[5]

The clinical manifestations of endocarditis associated with IDU differ from non-abusers; in IDU the right side of the heart is more often affected and presents with fever associated with pulmonary emboli rather than involving the left side of the heart with its accompanying systemic embolization.[5] In various series, the tricuspid valve was involved in 60-70% of cases, in contrast to aortic and mitral valve involvement of 30-40%.[3,7] Tricuspid valve vegetation commonly embolize to the lungs, causing septic pulmonary infarcts, which results in multiple focal opacities on the chest radiograph sometimes associated with cavitation. These were the findings in our patient. This radiological finding is reported to be a highly characteristic sign of acute right sided endocarditis in a febrile IDU.[8]

Pulmonary manifestations are extremely common in IDU.[5] The lung is the target of numerous infectious and non-infectious insults.[5] The latter includes drug induced bronchospasm, acute pulmonary edema, and talc granulomatosis.[5] Heroin overdose may be associated with unilateral or bilateral pulmonary edema and may be accompanied by fever and leukocytosis.[5] In a series of pulmonary complications of IDU, septic emboli were the most common complication followed by community acquired pneumonia and mycobacterium tuberculosis infection.[5]

Our patient developed bilateral septic pulmonary emboli complicated by bilateral pneumothorax, one of which occurred prior to initiation of ventilation. This was due to the most common organism in IDU MRSA. A diagnosis of pulmonary tuberculosis could not be substantiated. This is important since pneumonia was reported in one series to be the most common cause of fever in IDU.[5] Lung abscesses may arise from aspiration pneumonia, necrotizing pneumonitis or septic emboli from right sided endocarditis as in our patient.[5] The dominant causative organism to cause endocarditis in IDU is *Staphylococcus aureus* followed by *Streptococcus* groups A, B and G.[5] Polymicrobial endocarditis with up to 8 organisms were seen in IDU.[5] Unusual organisms reported include *Corynebacterium xerosis*, neisseria subvalvular and neisseria flavescens. There is an overall poor response to antibiotic therapy with persistent fever and embolic events require prolonged antibiotic therapy, and in our opinion an input from a clinical pharmacologist, to determine minimal inhibitory concentrations and appropriate dosing schedules to fine tune therapy where available.[8] Valve replacement is usually required in one third of patients with left sided endocarditis and in less than 10% of right sided cases.[9] Therefore, detection of valvular vegetations does not necessarily indicate the need for valvular replacement. Surgery is indicated when patients become hemodynamically unstable, mobile vegetations persist after embolic events such as in the case of our patient or vegetations become larger while the patient is on treatment.[9,10]

Chambers et al[11] advised that young IDU with acute *Staphylococcal endocarditis* have a good prognosis and should therefore, only have surgery if they develop intractable heart failure, definite signs of treatment failure such as persistent fever and enlargement of vegetations during therapy. Our patient therefore was a candidate for surgery.

In conclusion, our patient had a typical case of endocarditis and its complications in an IDU apart from occurrence of pneumothorax, which in our opinion, should now be kept in mind of those dealing with IDU patients. The management of such patients is problematic, not only from the point of the medical

problem and threat to life posed by the endocarditis, but also as a result of the difficulties in dealing with patients addicted to a self-destructing course in life.

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