CASE REPORT

A chest wall pulsating mass

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SUMMARY

A 34-year-old HIV-positive man with intravenous drug addiction presented with a 2-week history of fever and a pulsatile presternal mass; 2 weeks prior he had suffered a traumatic sternal fracture. The CT scan showed a bulky abscess with presternal and retrosternal extension that contacted the heart (which explained its pulsatile effect) and pulmonary necrotising lesions. Drainage of the thoracic wall abscess was performed, and antibiotics (flucloxacillin plus gentamicin) were started on the presumption of staphylococcal bacteraemia given the patient's intravenous drug habit. Blood cultures and culture of the aspirated pus confirmed growth of methicillin-sensitive *Staphylococcus aureus*; transoesophageal echocardiography showed endocarditis of the tricuspid valve. We therefore assumed pulmonary septic embolisation from right-sided endocarditis and acute sternal osteomyelitis, and the patient was treated for 6 weeks with clinical resolution.

BACKGROUND

Infective endocarditis (IE) was recognised to be a complication of injection drug use in the 1950s. *Staphylococcus* is the most common cause of IE among injection drug users (IDUs), accounting for more than half the cases. Most cases of right-sided endocarditis occur among this population, and pneumonia and septic pulmonary emboli are common, occurring in up to 75% of patients with tricuspid valve involvement. However, pulsatile masses in the anterior chest wall usually occur in the setting of bone destruction and cardiac contact. We were impressed to find that in this case the pulsatile presternal mass represented the clue to a right-sided IE.

CASE PRESENTATION

A 34-year-old man with known HIV infection, without previous treatment or medical follow-up, attended our consultation due to progressive swelling, warmth, erythema and pain in the anterior chest wall, accompanied by high fever (40°C) and chills. He denied other symptoms such as cough, sputum production, anorexia, weight loss, gastrointestinal or urinary symptoms; he referred to having resumed drug addiction during the last month (intravenous heroin). Two weeks before, he had suffered a traumatic sternal fracture without bone exposure or cutaneous integrity breakdown. Medical advice was to rest and take analgaesic medication, with no indication for surgical intervention. At clinical examination, there was a painful, inflamed, small orange pulsatile mass over the sternum, and the patient was febrile (39°C). The cardiac auscultation was irrelevant, namely for

the presence of heart murmurs; there were no lymphadenopathies or hepatosplenomegaly, and no cutaneous or nail lesions. He was admitted to our ward to make the necessary investigations.

INVESTIGATIONS

As the sternal lesion was pulsatile, we performed a CT of the thoracic before attempting to drain it. The CT scan revealed a complete misaligned fracture of the sternal body and a voluminous fluid accumulation measuring 7.8×5.7 cm in the presternal area, with extension to the retrosternal space (5.2×1.8 cm). The accumulation intercommunicated through the intercostal spaces, and the retrosternal component intimately contacted the anterior wall of the heart, applying pressure on this organ (figure 1). The internal mammary arteries were located adjacent to the fluid collection. There were also two pulmonary rounded lesions with central cavitation (figure 2). We performed surgical drainage of the mass, revealing pus, a sample of which was sent for microbiological studies (tuberculosis, fungal and aerobic cultures); three sets of blood cultures were also collected. Antibiotics were empirically started (flucloxacillin 2000 mg four times a day plus gentamicin 5 mg/kg once daily) on the suspicion of staphylococcal infection. The blood analysis showed the following: haemoglobin 13.2×10⁹ g/L, leucocytes 13.70×10⁹/L with neutrophilia, platelets 382×10⁹/L, creatinine 0.98 mg/dL, hepatic aminotransferases and alkaline phosphatase

Figure 1 Initial thoracic CT scan (sagittal view) showing complete fracture of sternal body, with a voluminous collection of presternal and retrosternal components; the presternal area measures 7.8×5.7 cm and the retrosternal area 5.2×1.8 cm, both communicating through the intercostal spaces. The retrosternal component contacts the anterior face of the heart, maintaining an adipose cleavage plan.

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Figure 2 Thoracic CT scan (axial view) showing one of the cavitated pulmonary nodules.

within normal range, C reactive protein 183.9 mg/L (<5.0 normal value); urine analysis without pathological findings. CD4 count $0.620 \times 10^9/L$, viral load 314 copies/mL, without antiretroviral therapy resistance in the tests; negative syphilis, hepatitis B and C serology. Blood and pus cultures grew methicillin-sensitive *S. aureus*. The direct microscopy, nucleic acid amplification test and mycobacterial culture of the drained material were negative for *Mycobacterium tuberculosis*. The transoesophageal echocardiography showed mobile vegetations (14 mm large) in the tricuspid valve, prolapsed to the right atrium.

TREATMENT

Given the evidence of IE and the antibiogram of the isolated pathogen, we decided to maintain the antibiotic therapy previously instituted: gentamicin for 5 days and flucloxacillin for 6 weeks.

OUTCOME AND FOLLOW-UP

The patient became afebrile 5 days after starting the antibiotics; 2 weeks after starting treatment, there was substantial reduction of the abscedated-purulent accumulation (figure 3). He was discharged on the fourth week of therapy, asymptomatic and without evidence of cardiac or extracardiac endocarditis complications during this period. He completed antibiotic therapy with oral flucloxacillin 2000 mg four times per day for two more weeks at ambulatory. The patient has been regularly evaluated by us over the past 6 months and remains well. He abandoned drug addiction, returned home to his wife and daughter, and is now happily working and starting a new life. We started him on antiretroviral therapy with efavirenz+tenofovir+emtricitabine.

DISCUSSION

Injection of particulate matter (such as talc) along with illicit drugs and its repetitive use produces cumulative subclinical damage to the tricuspid valve.^{1 2} In the process of drug injection, patients may also inject bacteria or fungi present on the surface of the skin, in the drug itself, or in diluents, fillers, or filters used to prepare drugs for injection.¹ In addition, IDUs have higher rates of nasal and cutaneous colonisation of *S.*

Figure 3 Thoracic CT scan (sagittal view) 2 weeks after starting treatment, showing substantial volume reduction of the abscess.

aureus than patients who use illicit drugs by the oral route exclusively³; staphylococcal infection is the most common cause of IE among IDUs, accounting for more than half the cases.⁴

As the sternal fracture in our patient was not associated with cutaneous breakdown or bone exposure, infection by contiguity was considered improbable. Therefore, the bone infection with presternal abscess and the IE are explained by haematogenous spread of *S. aureus*, probably due to the intravenous drug injection. We wonder if these two *loci* of infection occurred more or less simultaneously, or if the sternal fracture was the originally infected site, with posterior spread to the heart, and then to the lung by septic embolisation. The inflammation caused by the unstable sternal fracture represented a site of fragility, ideal for the seeding of haematogenous bacterial dissemination. From this site and the eventual drainage of infectious phlebitis from the veins, a secondary right endocarditis could have evolved.

With respect to the differential diagnoses, tuberculosis or other mycobacterial infection would be a possible explanation for the osseous infection, with ossifluent abscess and simultaneous cavitory pulmonary lesions, especially in patients with HIV-infected.^{5 6} These mycobacterial ossifluent lesions are

Learning points

- ▶ Always be suspicious of infective endocarditis in injection drug users (IDUs) with fever or with pulmonary or systemic abscedated lesions; do not begin antibiotics before blood culture collection.
- ▶ Thoracic wall abscesses and cavitated pulmonary lesions in an HIV patient should bring to mind the possibility of tuberculosis.
- ▶ Pulsatile masses of the anterior chest wall should be approached cautiously with a previous radiological examination before drainage.
- ▶ IDUs are still a discriminated group, with difficulties in accessing the health system, but they deserve the same treatment as other patients. Hospital admittance is an opportunity to advise and create new habits.

classically described as ‘cold’ abscesses, with insidious progression, distinguishable from the bacterial ‘hot’ abscesses, which are rapid in progression with local inflammation signs and systemic toxicity. Other infectious agents can provoke osseous infections and abscess formation (fungi *Histoplasma*, *Coccidioides*, *Blastomyces*, bacteria, anaerobes such as *Actinomyces israelis*,⁷ leprosy⁸ and melioidosis⁹).

Pulsating masses in the anterior chest wall can occur due to bone destruction and cardiac/aortic contact (local or metastatic neoplasms, multiple myeloma^{10 11}), or in the setting of highly vascularised lesions such as angiomas. In ancient times, syphilitic thoracic aorta aneurysms could cause sternal erosion and present as a classical pulsatile mass capable of expansion.

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