Bacterial endocarditis

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Abstract

Infective endocarditis is clinically known for three hundred years.

Forms of presentation are diverse and nowadays more subtle, however still is a frequent clinical situation. The clinical expression, sometimes fussy, should not prevent us of timely suspecting and diagnosing it.

Although in this clinical case the patient background made a very likely diagnosis this was hampered by the peculiarity of some circumstances.

Keywords: congenital heart disease, systemic lupus erythematous, bacterial endocarditis.

Introduction

Infective endocarditis defined by Osler in 1855[1] as "a microbial infection of the heart endocardial lining" is known whilst a clinical entity for around three hundred years. [2]

Although it can present itself in several forms which are more subtle from a clinical point of view, it arises in later stages of life[3] attaining evermore the right heart. It is more frequent in the male gender although there are not sensitive changes in its incidence, throughout the last few decades for which the United Kingdom, for instance, is quoted in seven new cases per 100,000 inhabitants per year. [3]

Endocarditis is therefore a frequent condition and often with a difficult clinical expression. The search of rare cases should not keep us away of the suspicion and the diagnosis of the common cases which make everyday medicine.

The clinical care presented here is also to remind us of exactly that.

Case Report

In October 1988, it was seen as a Cardiology Outpatient in Coimbra University Hospital, to study a congenital heart blow, a patient MMS, 41 years old, Caucasian, professional engineer, born in residing in a town in the north of the country.

The complementary exams carried out at the time led to the diagnosis of subaortic intraventricular communication, and all the controls he was subject to throughout the last few years have shown a moderate but progressive dilation of the left cavities keeping though the patient asymptomatic.

In April 1993, suddenly high-temperature, arthralgia and petechiae emerge having been admitted in hospital near his home to clarify the clinical condition.

He was discharged sometime after with a diagnosis of systemic lupus erythematous, a diagnosis based on the clinical criteria and on the positivity of ANA and anti-dsD-NA. The family history including his mother suffering of SLE, and a sister with ITP (Idiopathic thrombocytopenic purpura) episodes reinforced such diagnostic conclusion. It was then started on cortical therapy and it was verified the disappearance of fever and "petechial lesions", keeping however an important arthralgic component.

He was allowed to retake his professional activity, which happened only two weeks after that, time at which the high temperature came back with arthralgia worsening, onset of thoracic pain, first on the left and then on the right, and such deterioration led to be admitted in the same hospital.

From the tests performed then it should be highlighted the gasometry, showing hypoxemia and hypocapnia, and the thorax X-ray showing changes construed as pneumonic evolving processes, waiting then the result from the hemocultures requested in the meanwhile.

With SLE diagnosis, complicated by pneumonia, he was transferred for family reasons to Coimbra

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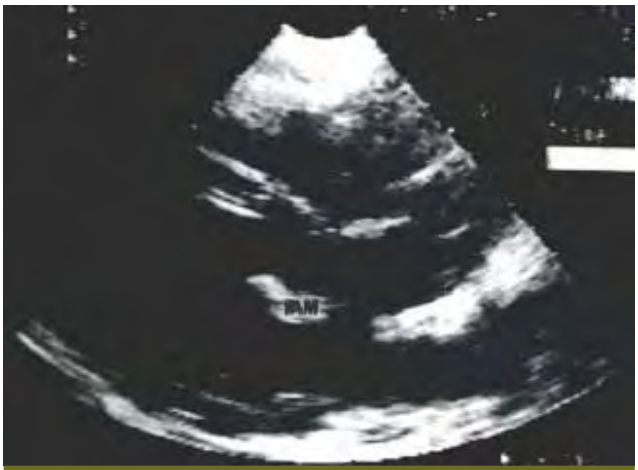
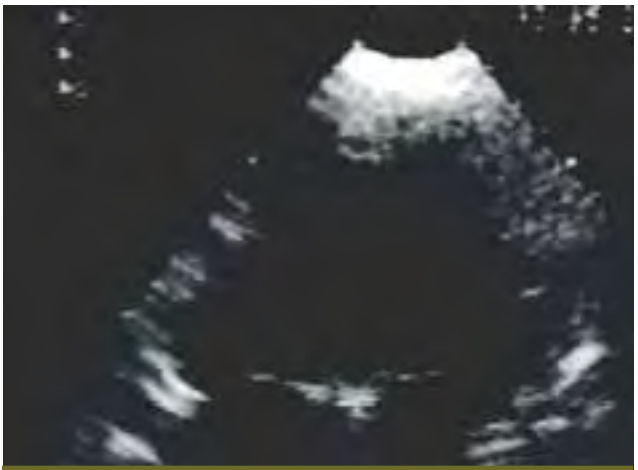


Bidimensional echocardiography – longitudinal parasternal plan – AE Left auricle VE- left ventricle: VD right ventricle: RAo Aorta root, VAM Mitral anterior valve with vegetation and anterior valve prolapse.

FIG. 1



Bidimensional echocardiography – two cameras apical plan: AE – left auricle: VE left ventricle: VM Mitral valve with vegetation and anterior valve prolapse

FIG. 2

University Hospital, Medicine Service II, where he was admitted on 16th May.

On the day of admission the patient showed discolored skin and mucosa, prostration, dyspnoea, productive coughing leading to thorax pain, generalized arthralgia, bilateral tibial-tarsal arthritis, high fever and meso-cardiac blow grade IV/VI.

From the research performed immediately it was evident the existence of hypoxaemia and hypocapnia, a marked increase on the fibrin degradation products, as well as scintigraphy of ventilation – perfusion compatible with pulmonary embolism diagnosis.

Knowing the patient's clinical history and with these data we pondered as diagnosis hypothesis, septicemia with pulmonary embolism by septic and/or non-septic material. As the patient was also a CIV carrier, the existence of bacterial endocarditis was strongly likely in spite of the normal ultrasound recently carried out at the hospital he came from.

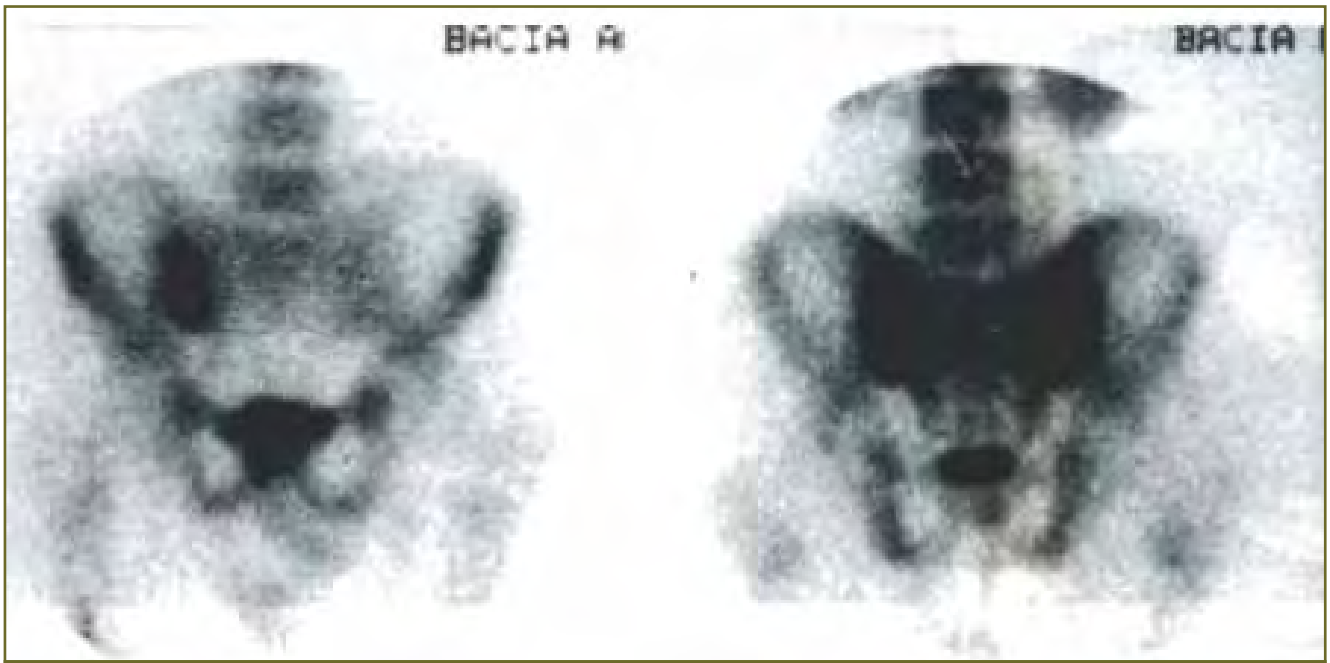
On the following day after being admitted we were informed that the hemocultures collected in the hospital he came from were positive for Staphylococcus aureus. He underwent then an echocardiography that apart of the previous changes (subaortic CIV and aortic valvular fibrosis with a light insufficiency) suggested the presence of vegetation at the level of the anterior mitral valve (*Fig. 1*). The pulmonary CT, carried out in the meanwhile, showed signs of cavitation on the right pulmonary medium lobe.

The antibiotic therapy he had been undergoing was then adjusted to the strongly likely diagnosis of bacterial endocarditis, guided by the antibiogram result which had followed those of the hemoculture. The identified germ was only sensitive to vancomycin, rifampicin, fusidic acid and not very sensitive to co--trimoxazole. At this point in time, steroid reduction was started until being withdrawn altogether.

The patient went on with high temperature in the first days of this new admission period, and both the clinical and laboratory data were pointing out for a difficult antibiotic therapy response being detected a septic focus at the level of the external face of the left leg inferior third, which was drained because it was presenting fluctuation being identified the Staphylococcus aureus in the purulent material collected for analysis.

As the clinical condition had an onset of heart failure he was subject, in a context of clinical laboratory monitoring to a new ultrasound exam which has revealed, apart of vegetations, a prolapse in the mitral anterior valve body due to a probable chordae tendineae rupture, leading to regurgitation with important haemodynamic significance (*Fig. 2*). He was then transferred to the Coronary Intensive Care Unit and introduced to heart surgery.

As the patient haemodynamic stability was achie-



Skeleton scintigraphy – hip; hyper-fixation on the sacroiliac joints level

FIG. 3

ved, the Heart Surgery decided to postpone the procedure, in order to intervene with a better infectious condition, and the patient underwent surgery a few days later.

The procedure was to close the CIV, a surgical cleaning of all existing vegetations both in the septal as the anterior wall of the right ventricle, as well as the drainage of the abscess located also in the right ventricle. The mitral valve was replaced by mechanic processes of the type Bjork Shiley no. 29, and it was also carried out the aortic valvuloplasty with sub commissure annuloplasty.

After surgery the patient developed a right hemiparesis of brachial predominance followed by extrapyramidal signs, followed by spontaneous contractions of the left lower limb and algic hypoesthesia of the foot dorsum, and this complication was due to spine emboli confirmed by scintigraphy.

The after surgery period evolved slowly but positively and the patient left hospital by the end of July with oral anticoagulant.

Comments

The clinical condition described is a paradigmatic case of acute bacterial endocarditis due to a highly invasive germ with a highly destructive capacity, Staphylococcus aureus, being verified in the evolution almost all the possible complications in such conditions.

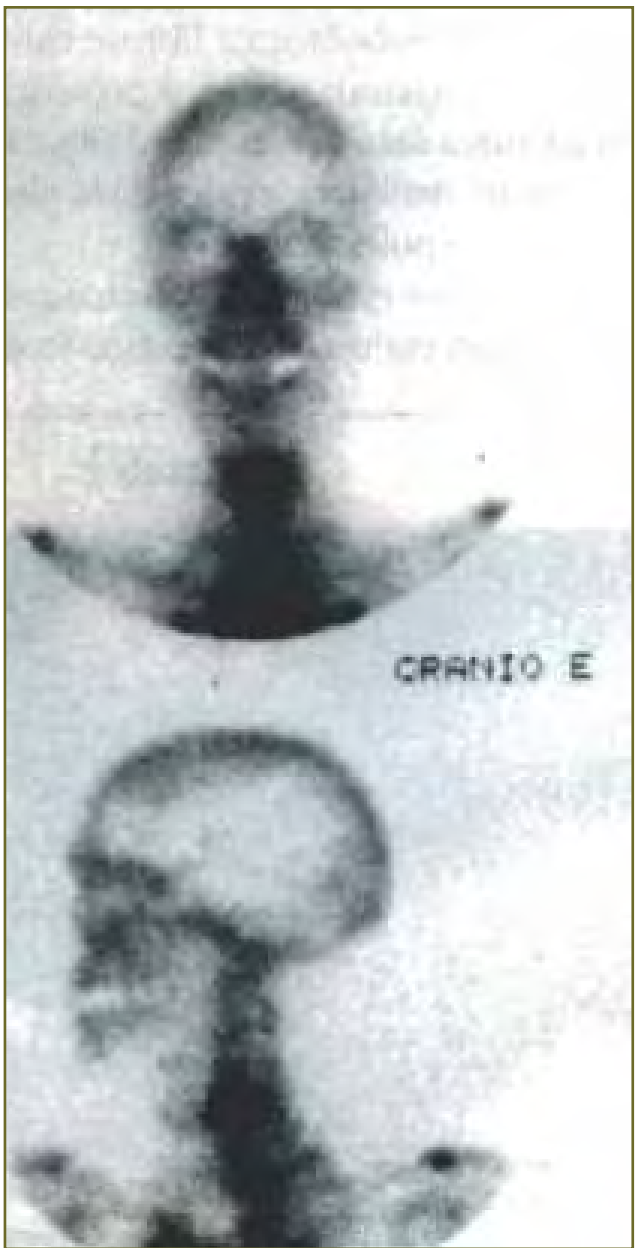
Bacterial endocarditis is before anything an intracardiac and intravascular infection, but it is also a systemic infection which leads to marked immune allergic reactions. We cannot therefore find odd if certain markers were positive, they are the translators of the immune system stimulation such as the rheumatoid factor, autoantibodies, immune complexes and so on and so forth as happened in the evaluated case.[5]

The persistent fever exists in 85 to 95% of cases, and can show itself whether as a subfebrile condition, or as a permanent hypothermia or in peaks.[6]

The new occurrence of heart blows happens in 30% of cases. However if there were previous heart blows to those setup by infectious condition, only at a later stage of the of the evolution process they will suffer any change.[6]

Some signs described classically, as Roth retinitis, Osler nodes and Janeway spots today are less frequent due mainly to early diagnoses.

Secondary septic locations are frequent and should be feared mainly when it is in cause the Staphylococcus.[6]



Skeleton scintigraphy – cranium and cervical column; hyper – fixation on the 7th cervical vertebrae and the 1st dorsal vertebrae

FIG. 4

In the described case, besides the pulmonary embolism with a posterior cavitation, secondary locations in the right sacral iliac joint (*Fig. 3*) and at the level of the seven cervical vertebra and the first dorsal one (*Fig. 4*) were detected. Particularly dangerous are embolism to the central nervous system.

On the complementary exams more often used in such circumstances just some considerations.

The ECG apart of an increase on the heart rate secondary to hyperthermia does not show again significant changes. However it is recommended that it is performed every two days considering the detection of serious complications as for instances the myocardial infarction due to septic emboli or the appearance of conduction due to the presence of peri-valvular abscesses.[6]

The thorax X-ray, besides signs of heart failure can show the existence of infiltrates or abscesses secondary to embolism.[6] In this particular CTs are a precious assistant.

Hemocultures are positive in 90% of cases if carried out in good quality laboratories and soon after the first collections.[7] However 5 – 15% of endocarditis evolve with negative hemoculture.[8] It is recommended to carry out 2 to 3 collections from different sites of percutaneous venepuncture, and to perform 2 aerobic cultures for each one of them. Anaerobic cultures as well as those made in an enriched culture should be carried out if the clinical situation justifies it.

It is important that samples are collected before any antibiotic therapy. However due to the frequency as this happens nowadays, such attitude might not be possible, therefore the collections should be made at times of lower serum levels of the antibiotic using more samples and more blood volume (20 – 30 mL), and passing such information on to the laboratory. The dialogue between the clinician and the pathologist is absolutely crucial here.[9]

The echocardiography enables the confirmation of the diagnosis by intensifying the cardiac lesions and complications intensifying the pathology previously existent.[10,11,12] It is also the ideal way to control the efficacy of treatment and the clinical evolution[13]. However a normal echocardiographic exam does not exclude an endocarditis diagnosis. In spite of such statement a negative transoesophageal ultrasound for endocarditis, in an unclear clinical situation, makes less likely its existence.[14] Regarding the sensitivity and specificity of such diagnosis techniques they have been frankly improved with transoesophageal echocardiography which became particularly important to evaluate the prosthetic endocarditis, namely the mitral one.[15]

The endocarditis diagnosis is therefore based in three main pillars: clinic, hemocultures and the echocardiography. First to suspect and then to confirm

the diagnosis are crucial steps to the beginning of the therapy.[6]

In terms of endocarditis treatment, our considerations in this space are abbreviated. The treatment is clinical or clinical and surgical.

From a medical point of view, antibiotic therapy guided usually by the antibiogram should be extended for 4 to 6 weeks intravenously using bacterial antibiotics and their efficacy and tolerance monitored. Medical treatment outside antibiotic therapy is usually directed to the therapy of heart failure and probably thromboembolic complications.[6]

The surgical treatment happens during the acute process or after the medical treatment already in the stage of quiescence in case there was a valvular deterioration justifying it. In the acute stage the indication for surgery is of haemodynamic nature (uncontrolled heart failure) or infectious nature (persistence of the infection condition and/or appearance of uncontrolled septic foci).[6]

Regarding the evolution, bacterial endocarditis can show a good response to antibiotic therapy curing without valvular lesion or minimal ones, but it can also evolve in a complicated manner.

Complications due to this onset of heart failure happen in 40 to 50% of cases and are usually due to acute valvular failure by chordae rupture. Heart abscesses, pulmonary embolism and mycotic aneurysms might be present or not.[16]

The bacterial endocarditis prophylaxis is recommended in all individuals with valvular prothesis, in all those who had endocarditis, in those carrying systemic-pulmonary "shunts", as well as all acquired valvulopathies, whether they are rheumatic or degenerative. Also indicated for prophylaxis are the obstructive hypertrophic myocardiopathy and the mitral valve prolapse with significant insufficiency. [6] The circumstances requesting it, the antibiotics and doses to be used vary and are perfectly defined in the rules set up for such purpose by the NYHA.[17]

Conclusions

In 10 to 20% of individuals with less than 60 years of age developing bacterial endocarditis have previous heart pathology with congenital anomalies present, such as the VSD, Marfan syndrome, PCA, aortic coarctation, etc...

The existence of valvular anomalies with significant insufficiency and in those we include the mitral prolapse as well as the valvular prothesis, place the individual in a high risk for endocarditis where the prophylaxis is compulsory.[6,17]

Staphylococcus aureus, responsible for 20 to 40% of all endocarditis native valves,[1] has usually a skin entrance. In around 50% of cases the evolution is acute, with possible quick valvular degradation, haemodynamic worsening and frequent appearance of secondary septic foci.

Antibiotic sensitivity has suffered modifications and to date is unthinkable to get an effective therapy which is not guided by the antibiogram.[18]

The early diagnosis, the intensive treatment, the correct monitoring of the clinical condition and its complications as well as a close medical and surgical interlink are the most effective weapons to control such clinical entity whose prognosis even today is shady. ■

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