Acute Aortic Insufficiency: a case report

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Abstract

A 46 years old patient, male, black, building worker, with a clinical condition of progressive dyspnea of recent onset and a previous history suggesting a breathing infection.

The clinical exam was pointing, from the onset, to a global cardiac insufficiency with a left dominance.

There was a considerable improvement on the symptoms with the medication started, having the patient shown occasionally for the three weeks he was admitted, short periods of dyspnea. He was febrile only in the last 5 days of admission, and the hemoculture performed in that period, only got the results after the obituary.

The supplementary exams performed, namely the three first echocardiograms, were not conclusive regarding an eventual valvular pathology. Only the last echocardiography, performed with Doppler study, has given us the diagnosis of acute aortic insufficiency. The patient died on the following day due to a complication of the underlying disease – bacterial endocarditis.

Key words: Acute aortic insufficiency, endocarditis, echocardiography, Propionibacterium acnes.

Introduction

Acute aortic insufficiency is a rare condition, with a difficult diagnosis in its earlier stages but crucial as it is possible actually to start an effective surgical therapy, sometimes life-saving.[1,2]

Bacterial endocarditis, whether diphteroids or Propionibacterium are rare: <1% in native valves, <5% in drug addicts and 5% in valvular prothesis.[3]

Recently we have seen a clinical case of acute aortic insufficiency by bacterial endocarditis where the clinic and the supplementary exams performed did not allow for an early diagnosis to be made on time to perform the medical-surgical treatment appropriate and on time, where the responsible bacterial agent was a saprophyte infecting the apparently healthy valves.

Clinical case

E. M. T, 46 years old, male, black, born in Cape Verde, residing in Portugal for 14 years, a builder.

He was admitted in the emergency service, due to a recent onset dyspnea.

The patient complained since the 20th July 1993, of a pain in the neck posterior area irradiating for the shoulder and interscapular region starting after a sudden movement; this pain was constant, grinding, getting worst with movement.

On the 28th July 93 it started with dyspnea complains and tiredness for medium efforts, with afternoon cold chills, not known if he had a fever.

On the 4th August 93 he went to the HSAC emergency service with a condition of asthenia, anorexia, thoracic pain (featured anteriorly) and a light non productive cough. He had an axillary temperature of 37.3ºC. The clinical condition was construed as a respiratory infection, reason why he received amoxycillin and ambroxol and was referred to the Internal Medicine outpatient service to clarify the cardiomegaly and slight anemia seen in the supplementary tests.

On the 9th August 1993, the patient was back to the emergency service with a worst clinical condition, in spite of the therapy started, showing dyspnea to small efforts, reason why he was re-admitted.

From his personal history it should be mentioned the patient was healthy until the beginning of the current story, mentioning a high blood pressure, in some assessments and twice venereal diseases treated with injectable medication. He had marked ethylic habits with a 200 g/day alcohol intake but denied smoking or drug addiction. There is to mention sexual

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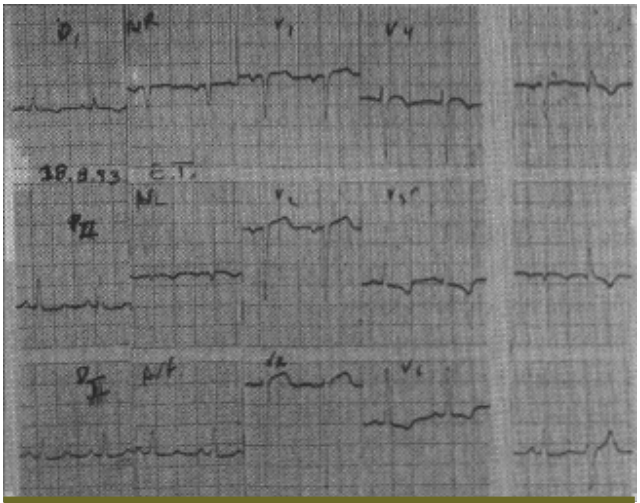
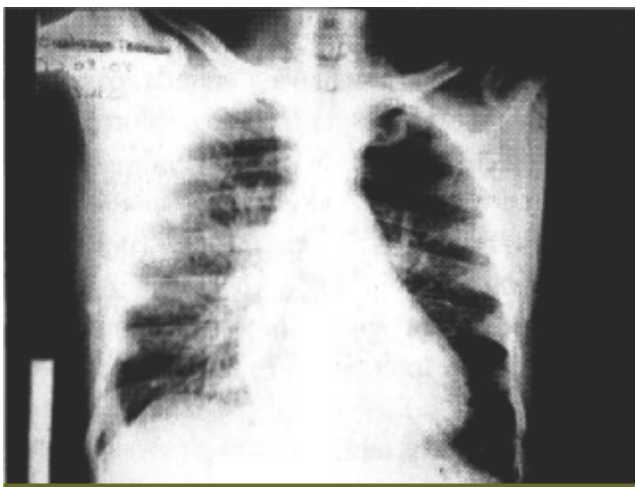


ECG (18 August 93).

FIG. 1



Thorax X Ray(4th August 93).

FIG. 2

promiscuity – multiple unprotected sexual contacts with prostitutes in Portugal, He denied odontology treatment and surgical interventions and he had no usual medication. line

On the objective exam when arriving at the service, he had dyspnea while resting, with light infracostal chest indrawing < acyanotic and pale mucosa; jugular regurgitation at 45º; TA-150/90, FC-100 ppm rr, FR-36 cycles per minute, without fever; under cardiac auscultation it was heard tachychardia, with a galloping rhythm and it seemed to be listened a mitral rumble; the pulmonary auscultation had an expiratory time slightly increased and there were coarse crepitations in the lower third in both hemithoraxes; from the abdominal observation it was highlighted a painful hepatomegaly 4 cm below the costal boarder in the median clavicular ; lower limbs without edema.

From the tests made there is to refer: normocytic normochromic anemia with Hgb 10.6 g/dL, stable during admission; Fe 30 mcg/dL and transferrin 1.72 g/L; leukocytosis with neutrophilia (GB 16400 N – 80%) down to 10000 on the second week of admission; ESR 40-20-30; AF 198/U/L and GGT 199 U/L persistently high; urea 136 mg/dL and creatinine 1,5 mg/dL, urine II with GV (r) and proteins (++) within normal range within a week after admission; arterial gasometry with PO2 of 75 mm Hg; low C3 (18.7 mg/dL) and normal C4 (14 mg/dL); and negative TASO, PCR, HIV, ANAs, W. Rose, serum for Legionella Pneumophila, Mycoplasma, rickettsiosis, Ac Hbc +; IgG + for EBV and CMV; VDRL + (2Dill) and FTA ABS +.

ECGs changes on overload/ischemia deteriorating during admission: in the initial ECG there was a probable hypertrophy of the left auricle and a slight lower lever in ST in V5 and V6; in the ECG of 18 August 1993 (Fig. 1) there was an inverted T wave in D1 and AVL, a negative T wave from V4 to V6 and an isolated ventricular extrasystole.

In the thorax X ray (*Fig.* 2) it is highlighted the costal arch flattening, cardiomegaly, hilum ingurgitation, pulmonary vessels "cephalization" and diffuse reinforcement of the pulmonary reticulum.

Echocardiography – several were performed in two hospital facilities: 10 August 1993 normal ventricular function, degenerative/fibrosis(?) change in the mitral valvular system leading to a stenosis pattern in the mitral valve; 13 August 1993 without valvular pathology (the Doppler was not made), left ventricle slightly dilated with a systolic function kept, small pericardic effusion. This echocardiography was performed in a cardiology appointment, and was not helped as the patient had a sudden and intense dyspnea episode, without an identifiable precipitating cause, requiring the patient compensation in the intensive care unit, and was construed as dyspnea of a main respiratory cause; 19 August 1993 – "changes compatible with incipient stenosis of the mitral valve and/or change of the left ventricle compliance/distensibility".

Other supplementary exams of interest:

Abdominal echography (14 August 1993): hepatic

congestion, being the other organs normal.

Holter (25 August 1993) where it is to be highlighted a sinusal basis rhythm with a minimum heart rate and average abnormally high; some isolated supraventricular extra-systoles, multiform and 3 couplets; lower leveling ST/T in V5, where point J lowers more than 1 mm.

Thorax CAT scan (18 August 1993): moderated cardiomegaly, hilum ingurgitation and the vascular structures reach the parenchyma periphery, changes which favor the pathologic process of vascular predominance. The therapy implemented was guided to the treatment of heart failure with the following drugs: furosemide, digoxin, captopril, DNI, hydroxizin and fraxiparin; it was given amoxycillin and clavulamic acid in the first 8 days of admission, therapy already started as outpatient.

The evolution was favorable with an improvement on the complaints the patient showed on admission, passing through asymptomatic periods to medium efforts (for instances, going up two sets of staircases steps without complaints) and paradoxically having sudden dyspnea periods while resting or to the small efforts (for example, turning in bed).

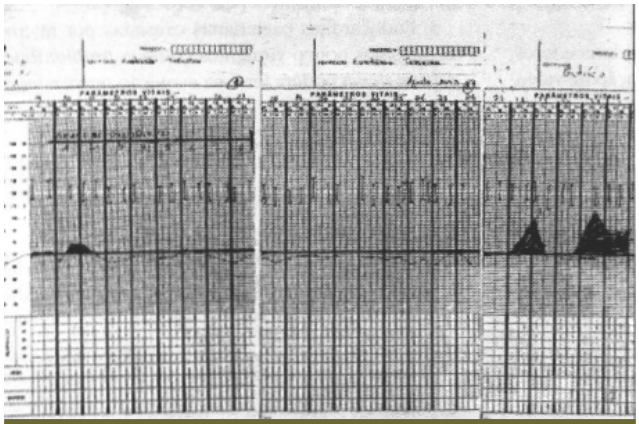
The fever graph (*Fig. 3*) shown displays on the 2nd day of admission, a febrile peak of 37.3ºC and from the 20 to the 24th day (five last days of admission) with febrile peaks reaching 38.8ºC, and at that time several hemocultures were made.

On a 3rd cardiology appointment, on the 1st September 1993, it was found on the cardiac auscultation a S3 or S4, an aortic systolic blow and a short diastolic blow. He made an echocardiography showing that the mitral valve did not have a stenosis pattern, no valvular vegetation, the left ventricle was not dilated and it was hyperdynamic due to volume overload, the left auricle was slightly delayed.

The echo-Doppler made confirmed the presence of aortic insufficiency grade IV/IV, with an aortic reflux to its apex.

The patient transfer was proposed on the following day to the cardiology service with a diagnosis of acute aortic insufficiency by a probable bacterial endocarditis.

The patient died in our service on the 2 September 1993, after a short episode (around 10 min) of an intense precordial pain, sweating and bradycardia, having the ECG taken at the time, a total 3rd degree AV block. This episode reached its peak with a cardiorespiratory arrest, and resuscitation was attempted unsuccessfully.



Fever graph.

FIG. 3

The autopsy has confirmed the clinical diagnosis of aortic endocarditis with this valve border erosion, with vegetation around 1,5 mm, aortic root normal, 3 cuspids aortic valve ; other valves normal; left ventricle hypertrophy without macroscopic myocardial lesions, serosa pericarditis with a small pericardial effusion; pulmonary congestion, small bilateral pleural effusion; chronic stasis liver; without other organs lesions.

Histologically it was verified the destruction of the aortic valve by abundant colonies of isolated positive gram bacilli and grouped identified as *Propionibacterium acnes* and pulmonary congestion with alveoli destruction (emphysema).

The three hemoculture results received two days after the patient's death revealed colonies of *Propionibacterium acnes*.

Discussion

In the last few years the evolution of diagnosis techniques and therapy has enabled to isolate the Acute Aortic Insufficiency as a clinical entity with particular features.[1]

It seldom appears and it is expressed as cardiac failure with left predominance, of sudden onset, usually progressive, non-treatable and lethal unless there is a surgical intervention.[1] In the case we present, the AAI was formulated based in clinical data and the echo-Doppler result made evident the existing

aortic reflux.[1,2]

The question raised is whether it would be possible to reach an early diagnosis enabling the patient to get an adequate and timely medical-surgical treatment.

The dyspnea differential diagnosis seemed evident the existing a heart dysfunction as there were signs and symptoms suggesting a heart insufficiency of recent onset and the thorax X rays, thorax ECGs and CAT scans, although non specified, supporting this hypothesis.

We did not think about AAI as likely, as it is a rare condition and different from usual[1,2] dyspnea at rest, main symptom on admission, has improved considerably with the implemented therapy: the patient would walk around the service apparently well, being asymptomatic for average efforts, in spite of mentioning short dyspnea episodes sometimes triggered by a minimum effort.

Different from what is the usual[1,2] the patient did not have signs of low debit, peripheral vasoconstriction, severe pulmonary congestion or edema, neither had he alternating pulse.

Blood pressure characteristically normal or low,[1,2] was always in the upper normal limits.

The auscultation signs can easily go unnoticed and tachychardia makes difficult its characterisation[1,2]: S1 and S2 were well audible and what is not usual, an S3 was heard and only at a later stage it was heard a short dyastolic blow.

Difficult was also the diagnosis of bacterial endocarditis, more common cause of AAI.[1,2] In this clinical situation, fever is the most frequent sign of appearance.[4]

The patient had an axillary temperature of 37,3ºC on the 2nd day of admission and ≤ 38.5 ºC on the four days preceding death, being kept pyretic on the 10 days following amoxycillin and clavulamic acid suspension.

Bacterial endocarditis evolve in 3 to 15% of cases with normal or sub-normal[3] axillary temperature among other causes due to antibiotic administration – fever recurring after therapy suspension is characteristic,[3] what did not happen with the patient we followed.

The septic condition was clinical and laboratorial discreet, has given in to the antibiotic therapy not very aggressive and the hemoculture results were only known after the patient's death, what did not allow for an earlier diagnosis, although this seems the presentation frame of sub-acute bacterial endocarditis.

Bacterial endocarditis caused by diphtheroid or Propionibacterium in native valves are rare (<1%);[3] it is to be highlighted that the patient was not a drug addicted nor was it detected any previous heart condition, predisposing factors to valve infection.

The isolated agent, *Propionibacterium acnes,*[5-10] is a gram positive anaerobic bacilli, saprophyte which is part mainly of the skin bacterial flora, intestinal and urinary and oropharynx tract.[5] It is a frequent hemoculture contaminating agent, however when isolated in two or more hemocultures collected at different times and within the clinical context, must be valued as infecting agents and not only as simple contaminating agents.[5] These microorganisms are seldom accountable for bacterial endocarditis.[5,7,8,9,10] Infect more often valvular prothesis than native valves but in spite of not being very virulent there are cases described of perivalvular abscesses.[5,7] Such microorganisms are sensitive to, among other antibiotics, penicillin and clavulanic acid.[5]

We thing the uncharacteristic evolution of AAI in our patient is due to the fact it was caused by a not very virulent agent, sensitive to the antibiotic therapy initially started as there was a suggestive history of respiratory infection.

The unexpected terminal accident leading to death in a short time after a total AVB raises the question regarding the etiology.

It is known that one of the most serious intracardiac complications of bacterial endocarditis is the development of myocardial abscesses,[4] that one thinks being present in 20% of patients dying by valvular infections.[4] Infections spreading to the adjacent structures are more common in aortic valves.[4,5] The formation of perivalvular abscesses may lead to the appearance of serious changes in the auricle-ventricle conduction: 1st, 2nd and 3rd AVB degrees.[1,2,4]

In spite of not being isolated cardiac abscesses in the autopsy or histology one can not set aside its existence any longer, as they can not have escaped the histological cuts made. Besides, the patient has almost all criteria[4] suggesting the existence of valve ring abscesses: aortic valve infection, regurgitation of recent onset, serious AVB, death preceded of short duration symptoms.

The echocardiography was not clarifying, in spite of being important while evaluating valvulopathies and bacterial endocarditis.[1,2,4]

The AAI echocardiography findings are not specific [1,2]. However no suggestive change was detected.[1,2] of this clinical situation: mitral valve early closure and late opening with fluttering. No suggestive changes of valvular involvement were detected or previous cardiac disease.[1]

M-mode and transthoracic bidimensional echocardiography detect around 75% of valvular vegetation;[1] the biggest difficulties emerge in the echocardiography diagnosis in patients with sub-acute bacterial endocarditis due to the small dimension of vegetation associated to low virulence microorganisms.[4,5] Only in the 4th echocardiogram performed to the patient it was made an echo-Doppler decisive to confirm the diagnosis: an aortic regurgitation flow IV/IV up to the apex was detected. An early performance of the Echo-Doppler would be more useful to clarify this clinical situation. The Echo-Doppler is the most sensitive and accurate test for valvular regurgitation detection[1,2], detecting moderate aortic insufficiency not suspected on auscultation.[2]

Conclusion

Acute aortic insufficiency is a rare clinical condition, of a difficult diagnosis and requires an urgent therapy. One should think on this hypothesis in heart failure of recent onset conditions.

Echo-Doppler is decisive to confirm a diagnosis of acute aortic insufficiency.

Bacterial endocarditis caused by low virulence microorganisms as *Propionibacterium acnes* may have a serious and fatal evolution.

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