CARDIAC TAMPONADE AS THE INITIAL SYMPTOM OF LUNG CARCINOMA

Editor;

Presentation of a pericardial effusion may be secondary to a wide variety of clinical situations. Overall, 22.6% of these effusions are secondary to uraemia, 14.5% to neoplasias, 11.3% to bacterial pericarditis, 11.3% to acute idiopathic pericarditis and the remaining 40.3% are secondary to other causes with 56.7% of the cases present with pericardial tamponade[1]. From the perspective of cardiac tamponade, according to a study published in 2002 by Oliver Navarrete et al.[2], when this occurs the aetiology is neoplastic in 52.1%, idiopathic in 14.6%, uraemic in 12.5%, iatrogenic in 7.3%, mechanical in 4.2%, tuberculous in 2.1% and due to other lesser causes in the remaining 7.3%.

On the other hand, from 5% to 15% of the patients with neoplasms may have tumoural invasion of the pericardium. The most usual clinical manifestation consists in haemorrhagic pericardial effusion and pericardial tamponade. The most frequent causes of this are lung and breast neoplasms, followed by haemotologic neoplasms[3]. Involvement is produced by continuity (haemotogenic or lymphatic) and the fundamental mechanism is blockage of the lymph nodes of the heart and pericardium.

A 52-year-old active smoker (20 cigarettes per day) male receiving treatment with alprazolam for a depressive syndrome arrived to the Emergency Department for persistent cough and pharyngeal irritation since five days previously. Piroxicam was prescribed and the patient was discharged. The patient did not improve and developed intense asthenia, anorexia, general malaise, dyspnoea and haemoptoic sputum for which he returned to the Emergency Department. He did not report fever or chest pain at any time. However, the day of admission he presented haemoptoic sputum and a significant increase in dyspnoea. Only jugular ingurgitation, tachycardia of 119 bpm and hyperventilation in the pulmonary base were of note on examination. Arterial pressure was 120/100 mmHg and arterial oxygen saturation by pulsioxymetry was 88%. Chest x-ray showed cardiomegaly and mediastinic widening to the detriment of the left side, with fibrotic tracts and a possible loss of volume (Figure 1, upper image). The electrocardiogram showed diminished voltages with no relevant findings. Blood and biochemical analyses were normal. Computerized tomography of the pulmonary arteries ruled out the presence of pulmonary embolisms, although a large pericardial effusion was observed (Figure 1, middle image) as was a pulmonary nodule located in the left vertex. Emergency echocardiography confirmed massive pericardial effusion with signs of tamponade (Figure 1, lower image) as well as spontaneous echogenicity in the pericardial fluid (suggesting the presence of blood) and dilatation of the inferior vena cava which increased inspiration volume. Three hours after admission to the Intensive Care Unit the patient presented haemodynamic deterioration with a reduction in systolic arterial pressure under 80 mmHg and evacuation pericardiocentesis was performed. During the following 24 hours pericardiocentesis evacuated 2,290 ml of fluid of serohaemorrhagic appearance. In the apical segment of the upper left lobe, fibrobronchoscopy showed an image for which biopsy could not be performed. However, cytology of the bronchial aspirate and the pericardial fluid allowed the diagnosis of non-small cell lung cancer. Analytical study of tumour markers showed: CEA of 11.) ng/mL (normal range: 0-4.7), CA125 of 172.5 U/mL (normal range: 0-31) and 21 value of 10.65 n/ML (normal range: 0-3). Treatment with carboplatin and paclitaxel was initiated and the patient was stable at two months with no recurrence of pericardial effusion.

Cardiac tamponade as the initial manifestation of pulmonary neoplasm is a rare clinical manifestation[4-7] and this case is particularly significant because the initial clinical manifestations leading to the study and diagnosis of this case consisted only in the presentation of a persistent cough and pharyngeal irritation, increasing dyspnoea accompanied by mild haemoptoic sputum and slight tachycardia. These scarcely discriminatory data seem to be in agreement with those provided by the last review by the National Cancer Institute[5] which states that up to 21% of the patients with cancer present malignant pericardial effusions which are often not suspicious because of the scarce presentation of clinical signs or symptoms of pericardial tamponade. Half of these cases with pericardial effusion evolve to cardiac tamponade and this effusion is the first sign of malignant disease. The symptomatology presents with dyspnoea in 93% of the cases and specific symptomatology associated with tamponade includes tachycardia, pulsus paradoxus, high jugular vein venous pressure and hypotension. In the present case dyspnoea, tachycardia, jugular ingurgitation and cough were the fundamental axes of the clinical presentation. The electrocardiography corresponded to an evident pattern of generalised diminishment of the wavelength which characterises this type of picture, although the present case did not present other electrical variants cited in the literature such as alternation[9].

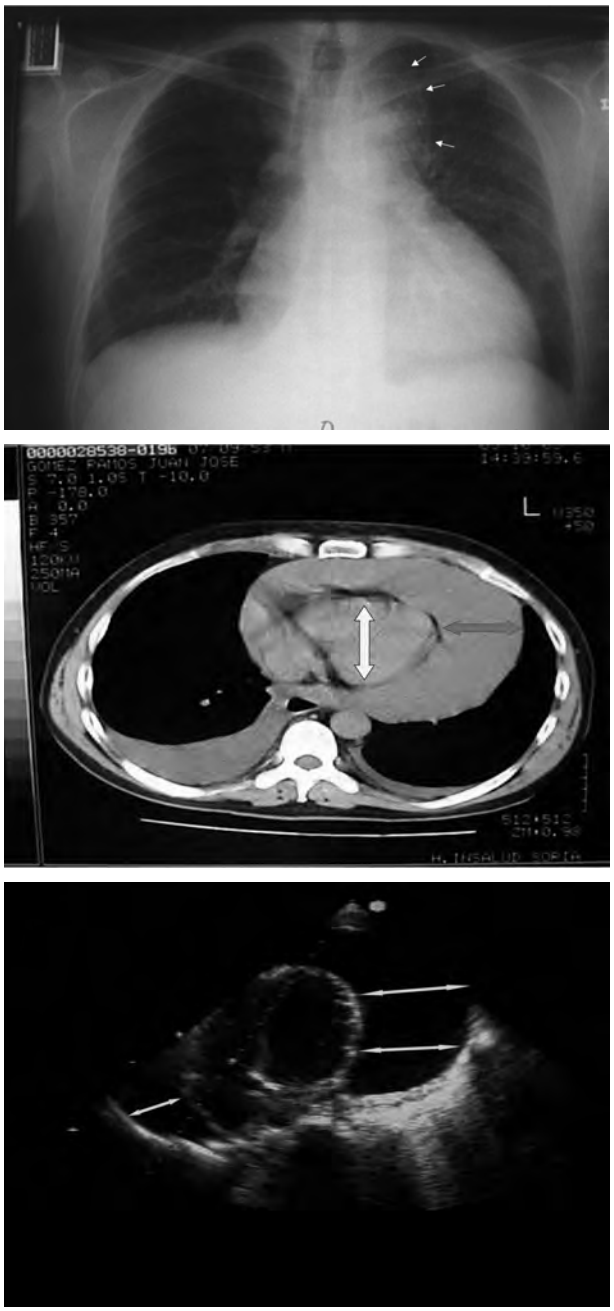

Figure 1. Pericardial effusion observed on chest x-ray (top, arrows indicate the loss of left lung volume), in the computerised tomography (centre, arrows) and in the echocardiography (bottom, arrows).

The most frequent routes of dissemination are direct tumour invasion, retrograde lymph emigration and haemotogenesis[4]. In the present case, the initial route of dissemination seems to have been direct tumour invasion since retrograde lymph emigration could not be demonstrated in the absence of lymph adenopathies. Neither does a haematogenic route seem plausible due to the absence of differentiated metastases in other organs. Direct tumour invasion seems to have been confirmed by the bronchoscopic finding of infiltration of apical segments. Computerised tomography ruled out mediastinic involvement and the presence of extracardial metastases. The lack of clinical data which may aid in relating cardiac tamponade and a neoplastic aetiology to the tamponade, cytology and biopsy continue to be fundamental diagnostic tools.

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RENAL INFARCTION. APROPOS OF TWO CASES

Editor;

Renoureteral pain is a frequent cause of consultation in emergency departments. Most of these patients present nephritic colic or acute pyelo-

nephritis but a small percentage have renal infarction (RI), the diagnosis of which may allow the detection and treatment of predisposing factors for thromboembolic phenomenon[1-4].

Two clinical cases attended in our hospital are described.

The first case was a 30-year-old male who consulted for very intense, non irradiating right lumbar pain accompanied by vomiting which initiated during sleep. He presented pain of right hypochondrial palpation and very positive right renal fist percussion. Urine sedimentation, chest and abdominal radiographies and basic biochemical analysis did not show any alterations. In the haemogram, leucocyte values of 12,300/mL with 93% neutrophils were of note. The diagnosis of nephritic colic (NC) was considered and analgesic treatment was initiated with major opioides also being used. Abdominal echography was requested and was also normal. Since the initial diagnosis of NC did not respond to treatment the patient was hospitalised.

A more extensive routine analysis performed 48 hours after admission showed lactate dehydrogenase (LDH) of 2,759 U/L (240-480) together with the appearance of microhaematuria. In view of this elevation in LDH an abdominal computerised tomography (CT) with contrast was requested which demonstrated massive right RI. The patient received anticoagulants and a thrombophilia study was made and found positive for antiphospholipid antibodies. Other studies showed neither arteriopathy nor thromboemboligenic cardiopathy.

The second case was a 47-year-old male with a history of 2 superficial thrombophlebites more than 5 years previously as well as deep vein thrombosis in the lower left extremity 3 years before, for which he was receiving oral anticoagulation. The patient attended the Emergency Department with pain in the renal renal fossa irradiating to the right hemiabdomen of 8 hours of evolution. He presented positive renal fist percussion and diaphoresis. Urinary sedimentation, abdominal radiography, blood and basic biochemical analyses were not altered. The INR was 2.3. Symptomatology improved with intramuscular diclofenac and the patient was discharged with a diagnosis of NC.

Four hours later the patient returned due to the reappearance of pain. Urine sedimentation and the blood analysis remained without alterations. On this occasion, the patient was administered tramadol intravenously and abdominal echography was performed due to the persistence of pain with no alterations being observed.

In view of the history of hypercoagulability, despite an INR within the therapeutic range and considering the doubts with respect to the diagnosis of NC (persistent pain, without haematuria or evidence of lithiasis or ectasia) LDH determination was made and was 705 U/L (12 h of evolution), with the previous value being 420 U/L (8 hours of evolution). The new determination of LDH at 24 h of evolution was of 1,050 U/L. After this progressive increase in LDH contrast CT was requested showing segmentary infarction of the right kidney.

Microhaematuria appeared after 48 hours of evolution. During admission, arteriography demonstrated segmentary occlusion with alterations of the main renal artery, echocardiography was normal and a thrombophilia study (incomplete) showed no alterations. The patient was discharged one week later with a diagnosis of left RI by non filiated thrombophilia and maintenance of the previous anticoagulant.

Renal infarction is infrequent. Although the exact incidence is unknown, some studies have reported an incidence of around 0.007%[1]. Little literature is available on RI with reports of isolated cases 7-9 and some retrospective studies[1-6].

The most frequent causes are emboligenic cardiopathies[2] while other causes include thrombophilias, connective tissue disease and aneurysm or dissections of the aorta or the renal artery[7,8]. Renal infarction may appear in anticoagulated patients, particularly if the therapeutic range is not achieved[1,3].

The clinical manifestations of RI are similar to NC or acute pyelonephritis thereby delaying diagnosis or even not being considered[1,5,6].

Urinary sedimentation shows microhaematuria in practically all the cases throughout the evolution but may be normal in the early phases[1,2]. Lactate dehydrogenase is elevated in all patients with RI in the first 24 hours[2]. Its determination is inexpensive and accessible in the emergency department and should be requested in patients with renoureteral pain and thromboembolic risk factors1 and in cases of initial diagnostic doubt. The elevation of LDH in these patients indicates the urgent need for contrast CT.

With respect to the treatment, revascularisation (intravascular intervention, surgery or fibrinolysis) is controversial[10] but may be considered in patients with previous renal disease to avoid the need for dialysis. Perhaps what is most important is to take the diagnosis into account and prevent new thromboembolitic events with anticoagulation[6].

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IMPACTION OF A MEAT FRAGMENT IN THE CARINA

Editor;

Death by choking is defined as the accidental ingestion or inhalation of a foreign object which results in the obstruction of respiration[1]. Food, mainly meat, is the object most frequently involved.

An 80-year-old male with a medical history of arterial hypertension and ischaemic heart disease who developed complete airway obstruction with posterior cardiorespiratory arrest is presented. Advanced CPR manoeuvres were initiated and airway permeability was attempted. None of the manoeuvres employed, including surgical access, were effective.

The posterior autopsy study discovered a piece of meat of 5 cm in length by 2 cm in width impacted in the tracheal carina completely obstructing the airway (Figure 1). Large food fragments, mainly meat, of up to 7 x 4 cm were also found in the stomach. Other autopsy diagnoses were ischaemic heart disease, cerebral lacunar infarction, hepatic steatosis, aortic valve calcification and severe generalised arteriosclerosis.

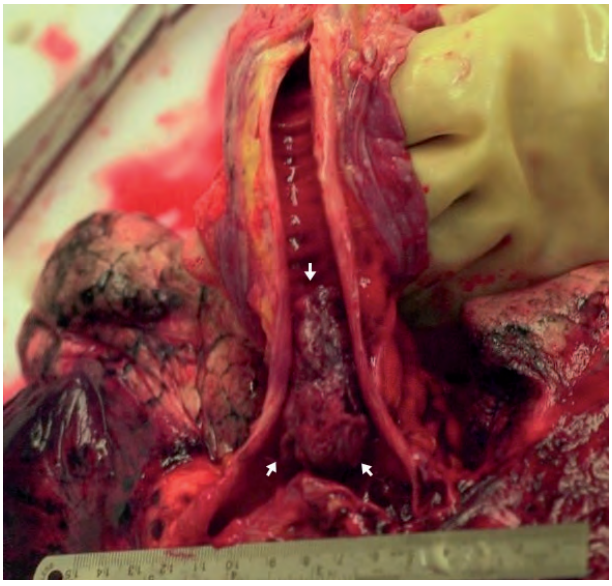

Figure 1. Fragment of meat with an approximate size of 5 cm in length by 2 cm in width.

Chemical-toxicological study demonstrated the presence of nordiazepam and an ethylic alcohol concentration of 1.56 g/L in the blood.

During 2003 4,300 deaths by choking were reported in the United States representing the fourth cause of accidental death. The groups of risk are children under the age of 4 years and the elderly[1]. Given the form of presentation, this entity has also been denominated "cafeteria infarction"[2-4].

One autopsy study[5] described that up to 1.3% of deaths which had initially been erroneously attributed to an acute coronary syndrome corresponded to pictures of choking. Many studies have shown that advanced age, swallowing alterations, dental problems, sedative drugs and neurological disease, especially Parkinson disease, are predisposing factors for choking.

Once choking has occurred rapid intervention to open the airway is essential. Taking into account the type of patients involved and the form of presentation, two specific groups may benefit from training in basic vital support manoeuvres and particularly airway de-obstruction techniques: restaurant personnel and care givers in elderly residences. This suggestion has been reinforced by the findings of a recent study[6] which showed that the victims of choking in a restaurant are attended later than those at home, since the latter are rapidly assisted by the family or friends with telephone aid from the emergency systems.

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ATRIAL MYXOMA. APROPOS OF A CASE

Editor;

Auricular myxoma is not a frequent diagnosis in the emergency department, mainly because of its scarce incidence which ranges from 0.002% to 0.3% in several autopsy series[1]. This is a benign primary tumour of the heart which is most frequently found in women, usually with symptoms of oppression or pain in the chest, respiratory difficulty on effort, dizziness, syncope or the sensation of palpitations after position changes. A frequent complication is embolisms which may affect the brain, eyes and extremities[2].

We herewith present a case which is of interest because of the diagnostic difficulty in the emergency department.

A 38-year-old male with a history of arterial hypertension (AHT) and smoker of 15 cigarettes per day presented to the Emergency Department for intense pain, insensitivity and paleness of the lower extremities during coitus.

On physical examination arterial blood pressure was 177/110 mmHg, cardiac frequency was 74 bpm, respiratory frequency 20 rpm, and arterial oxygen saturation (O2Sat) 98%. The patient was not febrile, was conscious, oriented and with great anxiety. No findings of interest were found on cardiopulmonary auscultation. The lower extremities demonstrated lack of sensitivity and bilateral cerea paleness under the knees with an absence of pedial and posterior tibial pulse. The bilateral femoral pulse was preserved and motor deficiency was not observed. Laboratory data (blood and biochemical and coagulation analyses), chest radiography and ECG were normal. Abdominal CT showed the abdominal aorta and the primitive internal and external iliac arteries to be normal. Magnetic nuclear resonance (MNR) was also normal while echo Doppler of the lower extremities detected a flow deficiency from the popliteal arteries in both lower limbs.

After admission to the cardiovascular surgery ward for bilateral arterial ischaemia of probable emboligenic origin, an angio CT of the thoracic-abdominal aorta was performed and detected a left auricular repletion defect of 9 mm x 2 cm (Figure 1) compatible with thrombus within or an auricular tumour (auricular myxoma).

Auricular myxomas may manifest clinically as: 1) asymptomatic or oligosymptomatic[3] pictures, the diagnosis of which is casual, 2) with systemic or constitutional symptoms due to the release of inflammatory cytokines (interleukin-6), 3) with cardiovascular symptoms such as myocardial infarction[4], cardiac insufficiency, syncope[5,6], and arrhythmias, and may simulate mitral stenosis, 4) with neurological symptoms and cardioembolic pictures[7,8], and 5) cutaneous manifestations[9], such as skin spots, erythematous maculae, nevus associated with the Raynaud phenomenon and distal ischaemias as a consequence of embolic events. The severity of these tumours lies in their emboligenic potential.

Echocardiography is the diagnostic test of greatest relevance since it provides a good definition of the localisation and extension of the myxoma. Transoesophageal echocardiogram (TEE) is fundamental in the search for the emboligenic source since it may detect masses and tumours of 1 to 3 mm in diameter[10,11]. On the presence of a cardiac tumour, CT and MNR make up part of the screening process because they are able to differentiate among the different types of tissues as well as solids, fluids and the space occupied by the tumour. Resection is usually curative. It may therefore be concluded that although auricular myxoma is an infrequent disease in the emergency department and may be difficult to diagnose, it must be taken into account as a possible cause of embolism.

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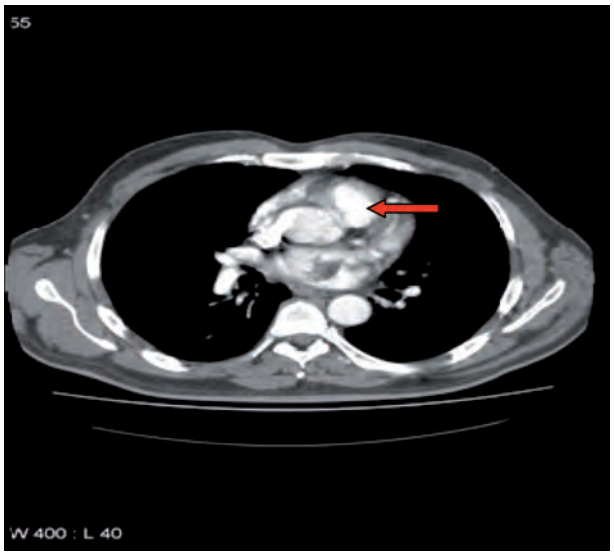

Figure 1. Image of angio-CT of the myxoma in the left auriculum.

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SPONTANEOUS CAROTID CAVERNOUS FISTULA, A DIFFERENTIAL DIAGNOSIS OF RED EYE

Editor;

Cavernous-carotid fistulas (CCF) are anomalous communications of traumatic or spontaneous origin between the cavernous sinus and the system of the carotid artery which lead to a set of signs and symptoms of venous insufficiency by high pressure blood flow in the veins affected[1,2]. Seventy-five percent of CCF are of traumatic origin (craniofacial or iatrogenic traumatisms by invasive techniques) and the remainder is of spontaneous origin (rupture of a carotideal aneurysm, arteriovenous malformation, atheromatous lesions, arterial hypertension or connective tissue diseases)[1,2].

An 87-year-old woman attended our Emergency Department for non painful ocular reddening of the left eye of 5 days of evolution with no accompanying symptomatology or any recent related traumatism. The patient had essential arterial hypertension and chronic venous insufficiency and was receiving treatment with captopril, pentoxfylline and torasemide. On examination, moderate conjunctival chemosis was observed as was reddening and vasodilatation of the episcleral vessels in the shape of a "head of medusa" in the anterior pole of the left eye (Figure 1). Intraocular pressure and fundus of the eye were normal. The remainder of the general and neurological examination was normal. On suspicion of CCF, cranial computerised tomography (CT) was performed with signs suggestive of a low flow CCF in the left eye. Given the good clinical status of the patient, her clinical history and advanced age, conservative treatment was initiated with intermittent manual compression of the left carotid artery, three times a day until the next check up. After 4 months of treatment the patient was asymptomatic and the picture had completely remitted (Figure 1).

The most frequent symptoms of CCF are: orbital pain, palpebral oedema, variable degrees of exophthalmos with or without murmur, tinnitus, diplopia, conjunctival injection, proptosis, ophthamoparesis (most frequently of par cranial VI) and an increase in intraocular pressure[1,2]. Different vascular retinal alterations, conjunctival and episcleral chemosis in the "head of medusa", twisting, haemorrhages and obstruction of the central vein of the retina may also be observed[1-3]. There is the risk of intraparenchymatous venous and haemorrhagic infarctions.

Diagnosis must be fundamentally based on the clinical history and physical examination accompanied by imaging tests: transcranial colour echo-

Doppler, with angiography providing confirmatory diagnosis[4]. The initial differential diagnoses include unspecific conjunctivitis, acute phase thyroidal ophthalmopathy, tumoural orbitary venous compression, orbital varices, the Sturge-Weber syndrome and the superior vena cava syndrome. Traumatic CCF normally requires endovascular therapy with selective embolisation techniques for resolution[5,6]. In low flow fistulas intermittent manual compression of the common carotid may be used.

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COMPLETE AURICULAR-VENTRICULAR BLOCKADE BY CHAGAS DISEASE

Editor;

Apart from the habitual diseases seen in the emergency department every day, there are others of tropical origin which require the widening of the scope of aetiologies of certain, up to now, infrequent diseases. This is the case of Chagas disease, an endemic disease exclusive of the Central and South American continents, from Mexico to the south of Argentina and Chile[1].

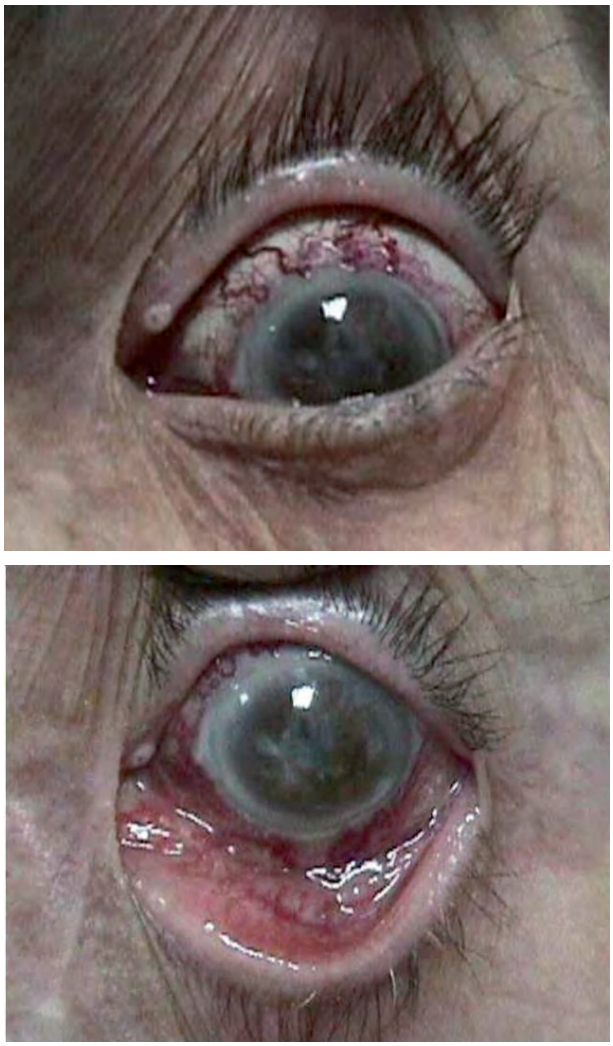

Figure 1. Vasodilation of the episcleral vessels in the form of "head of medusa" (upper) which disappeared after 4 months (lower).

A 23-year-old female Bolivian carrier of Chagas disease presented a medical report from her country with a cardiology study dated in 1998 for cardiac anomalies of unspecified aetiology. No treatment was given. She reported two births with normal deliveries. She neither smoke nor drank. She had been in Spain for three days and one sister had been diagnosed with Chagas disease.

The patient arrived to the Emergency Department for abundant metrorrhagia dizziness, occasional instability, asthenia and dyspnoea of 15 days of evolution. She also referred lumbar traumatism by accidental fall a few days before with an episode of dizziness. The date was situated as between 8 to 10 weeks previously. A pregnancy test was positive. Blood pressure was 80/60 mmHg, haemoglobin values were 10.6 g/dL, haematocrit 29.7 and beta-HCG was 135.5 mU/ML (non pregnant women up to 3 mU/mL).

On suspicion of the threat of miscarriage, obstetric echography was performed demonstrating endometrial

thickening of the uterus with a gestational sac. On confirmation of an ongoing miscarriage, curettage was programmed. On performing a preoperative ECG, important auricular-ventricular (O-V) conduction and Hiss system[2] disorders were observed and the patient was admitted to the ICU for cardiac monitoring. At the time of admission, the patient presented complete A-V blockade and complete blockade of the right branch of the bundle of Hiss (BCRDHH) with a ventricular frequency of 40 bpm (Figure 1). A transitory pacemaker (PM) was implanted via the left subclavian artery and the patient was informed of the need to implant a definitive PM, a treatment which she rejected. During admission parasite serology was performed which was positive for *Tripanosoma cruzi* with antibodies for Chagas disease (ELISA Ig positive for both ELISA 2.15 and indirect immunofluorescence 1/160). Cardiomegaly was not observed on chest x-ray. An echocardiogram did not demonstrate structural or cardiac dilatation.

Twenty-four hour Holter registry reported a mean cardiac rhythm of 58 bpm and rhythms of less than 40 bpm were reported during 30% of the registry. The most significant pauses were of 260 msec.

The patient was again informed of the need to implant a definitive PM which was again refused by the patient who requested a voluntary discharge.

Chagas disease or tripanosomiasis americana is an often lethal chronic infection produced by *Tripanosoma cruzi* which penetrates the human body through a bite by the insect, triatomino (Figure 2). This insect may be found in rural areas and is closely associated with poverty. It is endemic in Latin America and reportedly causes around 50,000 deaths per year, with up to 100 million inhabitants in conditions of risk. Of these, between 16 and 18 million are infected by *Tripanosoma cruzi* and 10% to 14% present manifestations of the disease.

The main forms of contamination are entomological, transfusional and transplacental.

Chagas disease is generally a chronic parasitosis which leads to death in the long term but may occasionally have a fulminant evolution. The course of this disease is chronic and may, therefore, remain unperceived until long after the infection, up to the point that a high percentage of infected patients are healthy carriers[1]. Indeed, in 95% of the cases, the parasitosis does not produce symptoms or these are so mild that they remain unnoticed. In benign cases, symptoms such as fever, vomiting, diarrhoea, and deterioration of the general status may hardly be evident. Three systems present the greatest involvement by the parasite: the circulatory, the digestive and the neurological systems. Chagasic cardiomyopathy is one of the most frequent manifestations of these patients. The size of the heart is augmented with signs of

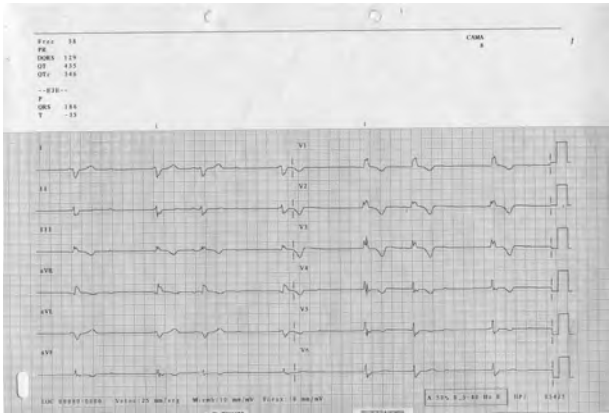

Figure 1. Image of biphasicular blockage. Third degree A-V blockage plus complete blockade of the right branch.

Figure 2. *Triatoma infestans.*

cardiac insufficiency[3]. Sudden death may occur in severe cases due to rupture of aneurysms located in the apex and may even coexist with thromboembolic events[4]. The presence of ECG alterations such as arrhythmias and conduction disorders is frequent and BRDHH and/or left supero-anterior hemiblockade (HAS)[5] is of note and is the most frequent ECG finding in 62.5% of the patients. Negative T waves and ventricular extrasystoles or polyfocal origin have also been described.

On suspicion by the symptoms described or the origin of the patient from endemic areas, diagnosis requires serologic confirmation[6,7] and may be performed with different measures. Fresh smear examination shows tripomastigoids but is more effective if the culture undergoes previous centrifugation. Thus, thick smear sample study involves a greater quantity of blood, xenodiagnosis and the blood cultures may provide definitive diagnosis.

There is no specific treatment for this parasitosis. Only palliative treatments may be administered to reduce the discomfort produced by the disease and these include megavisceral surgery, cardiac surgery, etc. However, two drugs have shown tripanomicide action: Nifurtimox with 10-12 mg/Kg/day during 60 days and Beznidazol with 5 to 7 mg/Kg/day for 60 days[8-10]. Secondary effects include leucopenia, irritability, cutaneous manifestations and digestive disorders. Criteria of cure are considered to be negativity of the serologic controls.

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