ACUTE AORTIC SYNDROME WITH DISSECTION: A PENETRATING ATHEROSCLEROTIC ULCER

Mr. Director;

The acute aortic syndrome (AAS) is an acute process which weakens the middle layer of the aorta, producing a potential risk of severe complications. Dissection of the aorta occurs in 80% of the cases, intramural haematoma in 15% and penetrating atherosclerotic ulcer (PAU) in 5%[1].

The patient was a 49-year-old hypertensive, dyslipemic, not pharmacologically controlled male smoker who attended the emergency department for central chest pain at rest of sudden onset which was intense and not irradiated of 3 hours of evolution. Blood pressure (BP) was 170/90 mm Hg. He did not present vegetative symptoms or data of haemodynamic compromise. Physical examination was normal including cardiac auscultation and distal pulses. The pain was not alleviated with nitroglycerin or morphine. Electrocardiogram showed a sinusal rhythm without alterations. Chest x-ray was normal including the mediastinum and the cardiothoracic index. Seried cardiac necrosis markers were normal.

On clinical suspicion of AAS, contrast enhanced computerised tomography (CT) was performed (Figure 1) and showed aortic bands, half a centimetre after the exit of the left subclavian artery, an indented sack-like formation, over an area of parietal calcifications compatible with PAU. The study was completed with transoesophageal echocardiogram which demonstrated a large atherosclerotic ulcer protruding in the aortic lumen at the final portion of the band and catheterism showed non significant coronary lesions and an image in the shape of a crater in the final portion of the band.

The patient continued with episodes of chest pain and 10 days after admission percutaneous treatment was performed after weighing the risks of a conventional surgical approach. A Relay 30 x 100 mm endoluminal prosthesis was implanted via the femoral vein under general anaesthesia requiring intentional occlusion of the left subclavian artery to place the device correctly. Sequelae of this occlusion included acral insensitivity, discomfort and a slight reduction in the strength of the upper left extremity. At three months this symptomatology was minimum demonstrating partial recanalization of the subclavian artery in the control CT. Treatment with enalapril, simvastatin and AAS was implemented.

Penetrating atherosclerotic ulcer is the result of progressive erosion of an atherosclerotic plaque in the wall of the aorta which weakens the aortic wall mainly in the distal third of the descending thoracic aorta[2]. The localisation in the band as in this case is infrequent. It most often affects elderly patients with cardiovascular risk factors and other levels of atherosclerosis. The potentially progressive nature of this syndrome

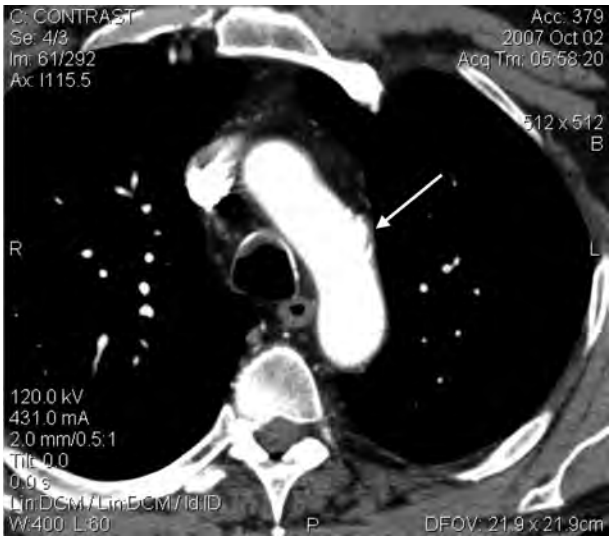

Figure 1. Computerised tomography was performed.

may lead to severe complications such as aneurism, pseudoaneurism, embolisms of thrombotic material and dissection[3].

The most commonly used diagnostic methods for the diagnosis of this syndrome are CT and magnetic resonance (MR) since these present a high sensitivity[4]. Transoesophageal echocardiogram is also useful, although its sensitivity and 24-availability is usually lesser. The risk of acute, symptomatic PAU is greater than or equal to that of classical dissection, particularly those localised in the ascending aorta which evolve to dissection or rupture in 57% thereby requiring the need to carry out invasive treatment[5,6].

The mortality of PAU in its acute phase is greater than or equal to that of classical dissection making clinical suspicion, the implementation of early imaging tests to confirm the diagnosis as well as medical treatment in the acute phase aimed at controlling the pain and BP the fundamental factors to reduce early mortality.

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THE OSBORN, OR J, WAVE IN HYPOTHERMIA

Mr. Director;

A 60-year-old male patient with a previous history of diabetes mellitus was brought to the emergency department in a mobile ICU after having been found on the floor of his home with a low level of consciousness, after neighbours had alerted the healthcare services. On physical examination a Glasgow score of 12/15, blood pressure of 100/60 mm Hg, a cardiac frequency of 43 beats per minute, rectal temperature of 34ºC and signs of dehydration were of note. Blood analysis in the emergency department showed glycaemia of 610 mg/dL and metabolic acidosis. The electrocardiogram (Figure 1) demonstrated sinusal bradycardia and a positive deflexion at the end of the QRS complex (Osborn J wave), although with many artefacts due to the trembling of the patient. The patient was diagnosed with diabetic ketacidosis as the cause of the reduction in the level of consciousness which led the patient to remain on the floor causing hypothermia and oxygen and intravenous insulin therapy were administered. To correct the hypothermia progressive warming was performed with a thermic air blanket and the patient evolved favourably.

The persistence of the Osborn wave, also known as the J wave, is highly sensitive and specific, although not pathognomonic, for hypothermia[1]. Cases in patients with subarachnoid haemorrhage without hypothermia have been described in normothermic patients with acute cardiac ischaemia and even in healthy persons.

This feature is a positive deflexion between the end of the QRS and the beginning of the ST segment (J point), which is more evident in middle left precordial leads which show depolarisation of the left ventricle[2]. The Osborn wave has been associated with hypothermia under 34ºC. Some studies support the suggestion that the presence and the size of this wave are based on body temperature[3], with the size of the wave being inversely proportional.

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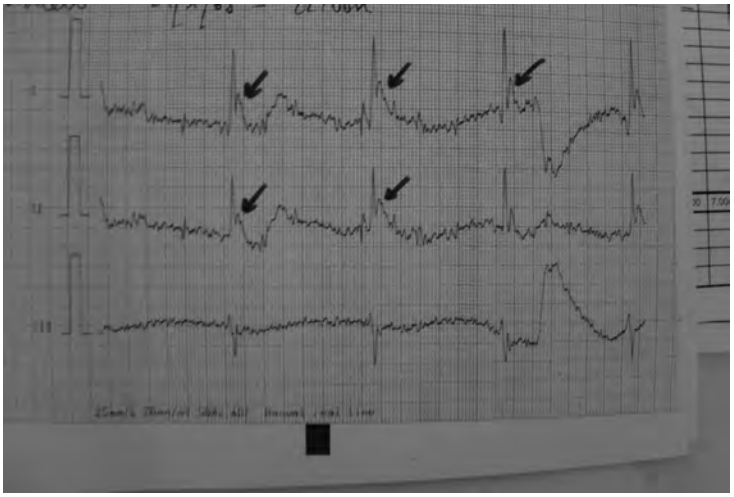

Figure 1. Sinusal bradycardia with positive deflexion at the end of the QRS (Osborn J wave).

VOMITING AND DYSPNOEA IN A 50-YEAR-OLD WOMAN

Mr. Director;

A 50-year-old female patient with a history of laparoscopic Nissen fundoplication one year after the present clinical manifestations consulted for unconstrainable vomiting, chest pain and dyspnoea of 24 hours of evolution. On clinical examination tachycardia (120 bpm), hypotension (SBP 90 mmHG), tachypnoea (28 r/m) basal oxygen saturation of 91%, and a reduction in movement of the left hemiothorax with abolition of the vesicular murmur were of note. Blood analysis demonstrated leucocytosis with neutrophilia (17.80 xv103/µL), renal dysfunction (urea/creatinine 74/2.4 mg/dL), hypopotassaemia (K 2.8 mmol/L), hepatic cytolysis (GOT-GPT 171/174 U/L) and an elevation of inflammatory reactants (CRP 24.4 mg/dL, fibrinogen 885 mmol/dL). Chest x-ray (figure 1) showed a veiling of the left hemithorax with a small, well ventilated pulmonary area (arrow 1), mediastinic displacement and an image of a semicircular wall superimposed to the cardiac apex (arrow 2). The lateral projection (Figure 2) showed hydropneumothorax with the superior pole of the collapsed lung (Arrow 3), with the second level corresponding to a paraoesophageal hernia (arrow 4). These findings were confirmed on computerised tomography (CT) in both the mediastinic and the parenchyma windows (Figure 3). Thoracic drainage was placed obtaining purulent fluid. On surgery a large paraoesophageal hernia with a small strangulated ring trapped in the stomach at the intrathoracic level with perforation and massive necrosis was observed. Total gastrectomy with Y in Roux oesophageal-jejunostomy for alimentation was performed. The postoperative evolution was difficult but favourable.

This case is an infrequent cause of acute chest pain and dyspnoea. In paraoesophageal hernias, the fundus and part of the gastric body are trapped in a herniated peritoneal sack in the mediastinum. The clinical presentation varies greatly, although it generally represents an incidental finding on chest x-ray. In other cases, digestive (dysphagia, postprandrial fullness) or respiratory manifestations (dyspnoea and chest pain) due to compression of the hernia sack have been reported[1,2]. Complete herniation of the stomach is a rare complication, being even more exceptional with necrosis and intrathoracic performation[3]. Since simple radiographic findings may be equivocal contrast CT should be performed. This presentation has a high mortality (> 60%)[4], with early diagnosis and surgical treatment being required. A high index of suspicion in patients with the symptoms and history described in necessary and this complication should be included in the differential diagnosis of chest pain and acute dyspnoea.

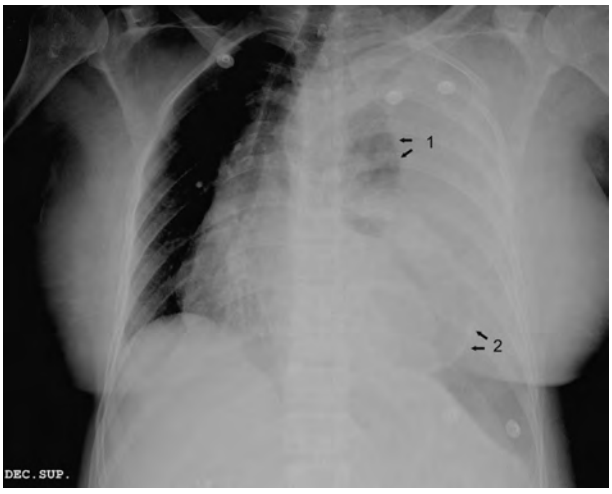

Figure 1. Veiling of the left hemithorax with displacement of the mediastinum and a small well ventilated pulmonary area (arrow 1) and an image of a semicircular wall superimposed to the cardiac apex (arrow 2).

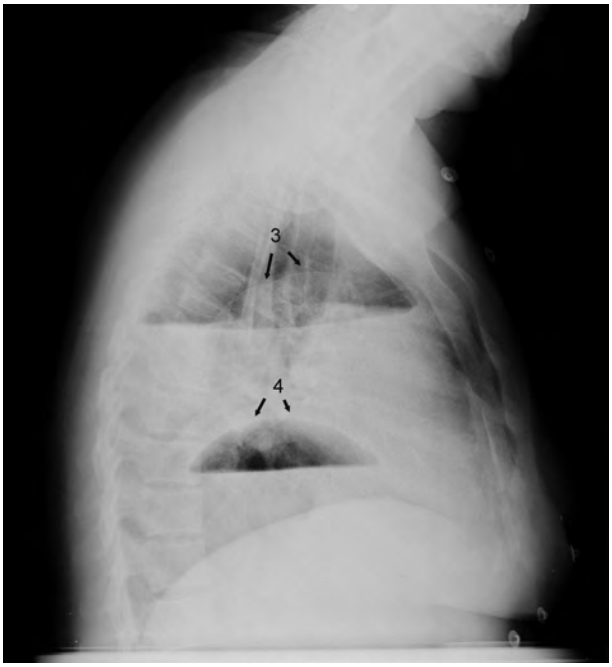

Figure 2. Hydropneumothorax with the superior pole of the lung collapsed (arrow 3) and a second level corresponding to a paraoesophageal hernia (arrow 4).

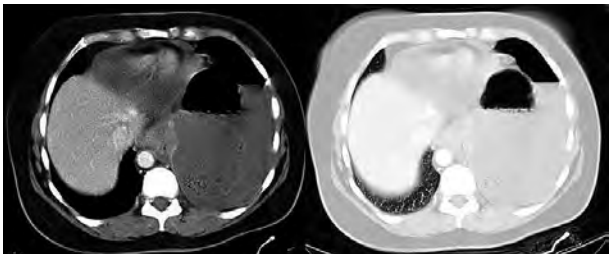

Figure 3. Computerised tomography in mediastinic window (left) and in a parenchyma window (right).

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OIL, GUNPOWDER AND MEMORIES OF CHILDHOOD: A CASE REPORT

Mr. Director;

The "déjà vu" phenomenon is based on an erroneous impression of familiarity to a present experience which is shocking for having been previously known. The most frequent cause is temporal lobe epilepsy (TL), although other pathologies may also be the cause[1,2].

A 45-year-old male attended the emergency department reporting 10-minute episodes of dizziness accompanied by an intense olfactory sensation, being, on one occasion, the "smell of gunpowder" and on another of "motor oil" of sudden appearance and which disappeared within a few minutes since one month previously. These "strange smells" reminded him of something "already lived", from episodes of his infancy which he described as ·"walking hand in hand with my father…", who was already deceased. Coinciding with the clinical picture the patient presented trembling of the left hand of a few seconds in length. Headache, instability or symptomatology at other levels was not reported. The temperature was 36ºC, blood pressure 140/80 mmHg. Physical and neurological examinations were normal. Cranial computerised tomography (CT) detected a rounded hypodense lesion in the right TL with intense peripheral enhancement in an "irregular ring" with contrast (Figure 1). Cerebral magnetic resonance (MR) confirmed a well defined, homogeneous tumour with ring-shaped enhancement and moderate vasogenic oedema (Figure 2). Electroencephalogram revealed desynchronised fundus activity and the presence of an intense focality of delta waves in the right TL. Chest x-ray was normal. Following surgical removal the morphologic diagnosis was glioblastoma multiforme (GM).

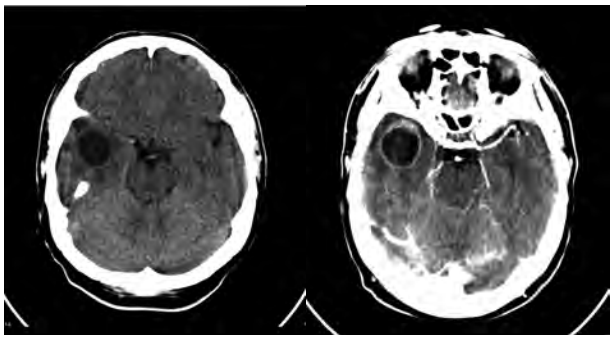

Figure 1. Cranial computerised tomography (CT) with round-shaped hypodense lesion in the right temporal lobe, perilesoinal oedema and mass effect with ring enhancement after contrast.

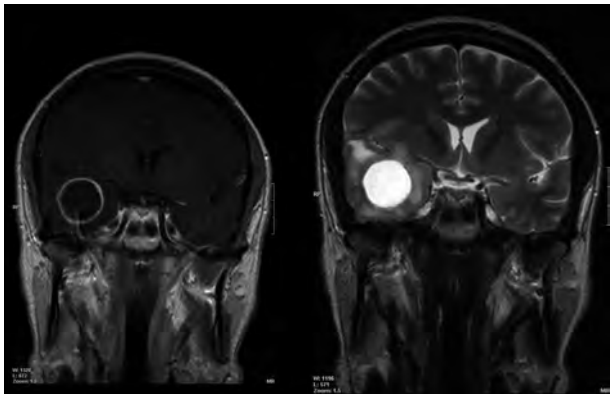

Figure 2. Cerebral magnetic resonance (MR) with homogeneous right temporal lesion with ring-shaped enhancement in the T1 and T2 sequences.

Glioblastoma multiforme is the most frequent primary brain tumour[3], representing 25% of all brain tumours and more frequently affecting males from 50 to 60 years of age. It is a malignant infiltrating tumour which may attain an enormous size before presenting clinical manifestations because of its rapid growth. Ten percent begin with visual, olfactory, taste or other hallucinations together with dysmnesic symptoms of the "déjà vu" type. CT and cerebral MR are the complementary tests of choice, demonstrating an irregular shaped lesion with ring-shaped contrast reuptake. The differential diagnosis should be with metastasis, lymphoma or abscess. Treatment included surgical resection followed by radiotherapy and occasionally chemotherapy with alkylating agents[4,5]. Postoperative survival at 18 months is 15%. This case demonstrates that, sometimes, anamnesis is not

sufficiently taken into account and emphasises that the appearance of complex psychic symptoms may reveal a severe organic disease.

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TRANSIET APICAL DYSKINESIA ASSOCIATED WITH UPPER DIGESTIVE TRACT BLEEDING

Mr. Director;

The transitory apical dyskinesis syndrome (TADS) is a recently described clinical entity which was initially confounded with acute myocardial infarction (AMI)[1,2].

A patient who developed severe ventricular dysfunction related to upper digestive haemorrhage (UDH) is presented. Practically normal recovery of contractility was achieved. A 79-year-old woman attended the emergency department for retrosternal chest pain of 14 hours of evolution with the sensation of loss of life, dyspnoea and vegetative symptoms.

The initial physical examination showed involvement of the general status, paleness and sweating; blood pressure 86/45 mm Hg, cardiac frequency of 67 bpm and O_2Sat 98%. The electrocardiogram (ECG) showed anterolateral subepicardic lesion with elevation of the V2-V6 of the ST segment. Blood analysis: haemoglobin 9 g/dL, hematocrit 0.27%, normal troponine T and creatinine. The initial diagnosis was acute coronary syndrome (ACS) and anaemia with no aetiological diagnosis. Treatment was initiated with aspirin, enoxaparin, omeprazol and concentrated red blood cell transfusion. The patient was transferred to the haemodynamic department of reference for urgent catheterism in which apical dyskinesis of the left ventricle with angiographically healthy coronary arteries were observed. An echocardiogram was performed showing the left ventricle to be of normal size; apical dyskinesis with compensating response from the remaining segments and a slightly depressed ejection fraction. Gastroscopy revealed an active bleeding ulcer lesion in the gastric antrum. The outcome of the patient was favourable.

The TADS is infrequent (0.5-1% of patients with suspicion of ACS)[3] and is normally presented with chest pain and enzymatic and electrocardiogram changes similar to those of an AMI. In contrast to this, coronariography shows arteries without significant lesions and left ventricular dysfunction with transitory apical akinesis-dyskinesis[3]. None of the hypotheses on the aetiology of this syndrome has clearly been related[2,4-6]. It is usually preceded by

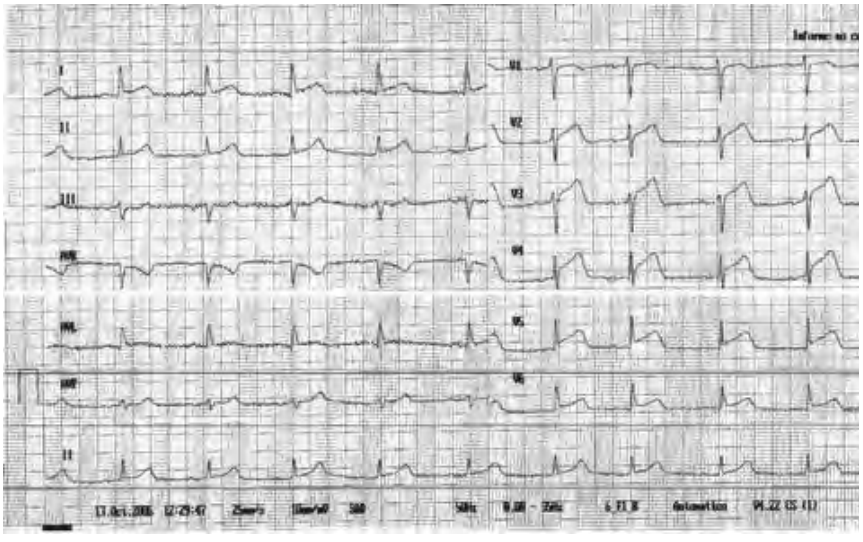

Figure 1. Electrocardiogram: anterolateral subepicardic lesion (elevation of V2-V6 in ST segment).

physical or emotional stress or an exacerbation of an underlying disease[1,3,7,8] (in the present case the triggering factor may have been stress produced by acute haemorrhage on originating the release of catecholamines[9,10]).

The ECG showed an elevation of the ST segment in the anterior side (90%)[3,8], Q waves (25%) and in almost all the cases, negative T waves and prolongation of the QT interval in the previous days[3,8,11]. Coronariography is fundamental for the diagnosis, showing coronary arteries with significant lesions accompanied by left ventricular dysfunction[3]. The segmentary alterations of contractility recover within weeks and although the prognosis is good[3,5], acute complications frequently occur such as acute pulmonary oedema, cardiogenic shock, arrythmias[12]. The differential diagnosis with AMI is important since the treatments used in ACS (fibrinolysis, heparinisation, platelet IIb-IIIa receptor antagonists) may be detrimental in patients with TADS[3,5]. Purely clinical criteria which allow the difference to be reliably established have not been described. However, there are data of suspicion[3,8] including an elevation of the ST segment in the acute phase which is more marked in V4-V6 than in V1-V3 with an absence of reciprocal changes in the inferior leads, Q waves which disappear after the acute phase, very prominent T waves in V1-V6 after the second day, prolongation of the QTc, predominance in females over the age of 50 years, and physical or emotional stress which may trigger the picture. In the emergency department, the ACS should be managed based on the current recommendations, although the suspicion of TADS and performing a catheterisation in the acute phase may avoid unnecessary and potentially dangerous therapeutic approaches[12].

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