LETTERS TO THE EDITOR

Idiopathic retroperitoneal fibrosis: a case report

Editor:

Idiopathic retroperitoneal fibrosis (RPF) is a rare disease of unknown etiolgy. It is characterised by a retroperitoneal inflammatory process often involving the ureters and producing unibilateral hydronephrosis and sometimes renal failure[1,2]. Early diagnosis of this entity may avoid important complications.

A 50-year-old man with a history of arterial hypertension and type 2 diabetes mellitus presented at Emergency Department with lumbar pain radiating diffusely to the abdomen of two weeks evolution. Physical examination showed diffuse abdominal pain, and laboratory test showed slight anemia (Hb 102 g/l, MCV 75 fl) and ESR 63 mm. Ultrasound showed calcified aortic wall, without aneurysm but with periaortic tissue. CT scan showed a mass of tissue involving the abdominal aorta from immediately below the origin of the renal veins to the aortoiliac bifurcation and the proximal portion of the iliac vessels. Slight left hydronephrosis was observed. Given the patient´s symptoms, he was admitted. Rheumatoid factor, antinuclear antibodies and anticitoplasmas de neutrófilio (ANCA) were negative. Fine needle aspiration ruled out malignancy and confirmed the diagnosis of RPF. Corticoid treatment was initiated with doses of 60 mg and then tapered by 5 mg every 15 days. Patient evolution was good, with rapid pain relief from the initiation of treatment. The patient is currently on maintenace therapy with 5 mg prednisone and remains asymptomatic. Follow up CT scan every two months has shown near disappearance of the mass.

RPF It is characterised by a retroperitoneal inflammatory infiltration, fibroblastic proliferation and collagen deposits[3], generally in the retroperitonium, occasionally trapping and compressing the ureters, arteries, veins and lymphatic vessels which gives rise to symptoms; rarely, this may extend to the mediastinum, mesenterium, biliary pathways, duodenum, bladder and the epidural space[1,4]. The etiology of most cases of RPF is considered idiopathic (up to 50%) or forms of Ormond´s disease[1,5]. There is also another form of RPF secondary to drug intake or retroperitoneal lesion (Table 1). The incidence of RPF is estimated at 1/200.000 people, generally affecting adults, especially in the fifth and sixth decade of life, with a male:female ratio of 2/1.

The most common symptom is lumbar and/or diffuse abdominal pain. Other symptoms include anorexia, nausea, vomiting and weight loss. Physical examination can show fever, arterial hypertension and edema of the lower limbs or scrotum. occasionally with oliguria or anuria. The most frequent analytic alterations are elevated ESR, anemia or impaired renal function. The absence of diagnosis of this disease is associated with important mortality in patients with ureter obstruction and complications such as loss of renal function or edema secondary to obstruction of the inferior vena cava or lymphatic vessels.

CT scan is currently the first choice of test for the diagnosis of RPF and follow up of these patients, although histology of biopsy samples is mandatory to rule out malignancy[1,6]. Other useful tests include nephro-urologic ultrasound, endovenous pyelography and magnetic resonance imaging.

Management of RPF patients may be medical or surgical. Surgery is indicated in cases of ureter compression and severe hydronephrosis. In the initial phases of fibrosis with slight ureter compression, as in our case, or when surgery is not possible due to the poor condition of the patient, medical treatment alone is recommended. The best results reported in different series[6,7] have been obtained with both treatments: firstly with surgical removal of the mass and liberation of the ureter, followed by the administration of corticoids. Recently, other immunosuppressive medica-

Table 1. Causes of secondary retroperitoneal fibrosis

Malignant Processes:
– Lymphoma.
– Sarcoma.
– Carcinoma.
– Metastasis.
Drugs:
– Methysergide.
– Bromocryptin.
– Betablockers.
– Methyldope.
– Hydralazine.
– LSD.
Retroperitoneal lesion:
– Radiation.
– Haemorhage.
– Surgery.
– Extravasation urine.
Inflammatory processes:
– Periaortitis (abdominal aorta aneurysm).
– Sarcoidosis.
– Pancreatitis.
– Endometriosis.
– Autoimmune collagen vascular disease.
Infection:
– Tuberculosis.
– Histoplasmosis.
– Actinomicosis.

tion has been used, such as cyclophosphamide[8], azathyoprine[8-10] or tamo-xifen[8,11].

Prognosis for treated RPF is generally good. Evolution is worse in elderly patients or those with important renal affection. Long-term follow up of these patients is required since disease activity may persist for many years and relapse is not infrequent, especially after corticoid withdrawal.

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Subacute cardiac rupture as a complication of acute myocardial infarction

Editor:

Acute cardiac tamponade due to car-diac rupture is a severe mechanical complication of acute myocardial infarction (AMI), with a mortality rate that remains very high despite aggressive treatment. Pericardiocentesis is a method that may be applied in emergency situations, since it temporarily improves the clinical and haemodynamic condition of the patient, until such time as surgical repair of the ventricular wall is possible.

We present the case of an 83-year-old woman with a history of hyperlipemia, two episodes of transitory ischemia accidents 10 years before, hypothyroidism and depression under treatment. The patient lost consciousness while walking in public, preceded by chest pain of 3-5 minutes de duration. Alter being attended by an emergency ambulance, she was transferred to hospital. Physical examination showed generally poor state, poor peripheral perfusion, hypotension (75/50 mmHg), tachycardia (120 bpm) and tachypnea (35 breaths/minute), important jugular ingurgitation, and auscultation showed symmetrical cardiopulmonary ventilation in both lungs, weak and rhythmic, without murmur. Her daughter reported that the patient had complained of central chest pain which resolved after some minutes. Electrocardiogram (ECG) showed slight ST segment elevation in D1 and aVL with anterior and inferior descent, and a subsequent ECG obtained with posterior leads showed ST segment elevation (Figure 1). Transthoracic ECG showed lateral and inferolateral akynesia with severe pericardial haemorrhage-like effusion and signs of acute tamponade, compatible with sub-acute cardiac rupture but the exact location was not observed. Based on all these data, the patient was diagnosed with evolved posterolateral AMI complicated by cardiac tamponade due to cardiac rupture. The treatment option adopted was conservative and subxiphoid pericardiocentesis extracted 320 cc of haematic liquid; the patient improved and was extubated within the first 24 hours. Two days later she suddenly worsened. Echocardiography showed a new event of severe pericardial effusion, and the patient died a few minutes later.

Rupture of the left ventricle free wall pesents in 1-4% of hospitalised AMI patients, responsible for 15-20% of AMI-related deaths[1]. In the great majority of cases (80-90%) rupture occurs in the first week, and in half of these (30-50%) within the first 24 hours[2,3]. Certain risk factors have been described: advanced age, female sex, hypertension without left ventricular hypertrophy, anterior localization of the infarction, first episode of infarction, and especially prolonged time to reperfusion of the infarcted area or absence of reperfusion[4].

Revascularization techniques have markedly reduced the incidence of this complication[5,6].

Recently, primary angioplasty (PA) has been shown to offer the greater benefit than fibrinolytic therapy, reducing the risk of cardiac rupture, especially in elderly patients, so PA is now recommended for patients over 75 years of age[7-9].

This complication should be suspected in patients complaining of persistent chest pain (occasionally pericardial), which is often erro-

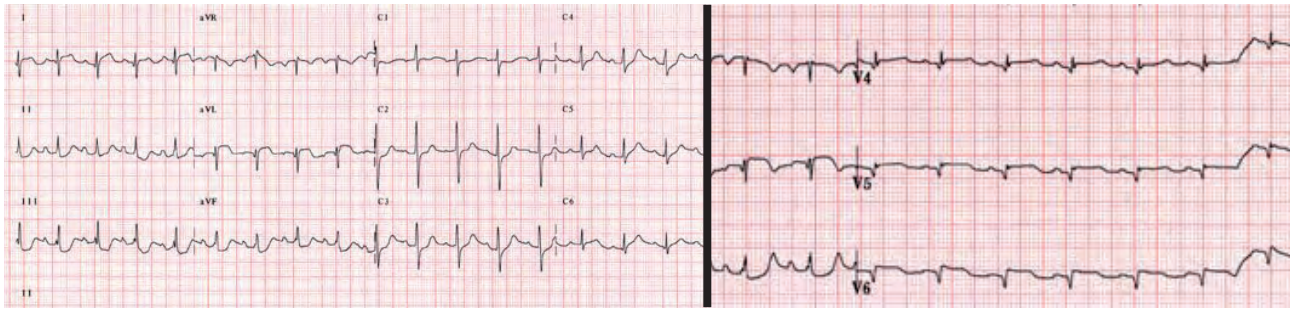

Figure 1. ECG on admission: Sinus tachycardia 110 bpm. ST segment elevation in lead DI y aVL with anterior ST and V2-V3 descent, with ST elevation in posterior leads (V4-V6 ECG of the right).

neously attributed to ischemia, newly appearing changes in the ST segment or persistent ST elevation, vomiting, sudden deterioration of haemodynamic state, transitory or persistent hypotension, syncope or cardiac tamponade[2,4,10-12].

Transthoracic echocardiography is the first, sometimes the only, complementary test necessary to confirm the diagnosis, in the presence of suggestive symptoms, especially in the Emergency Department (ED). Pericardial effusion is the most frequent finding, but compromised right cavity filling may be observed, and even wall defects[4,13,14]. Haemodynamic monitoring with right cardiac Swan-Ganz catheterization is indicated in all cases suspected of mechanical complications, to improve diagnosis and management of these patients.

Medical treatment, in addition to liquids and vasopressors to treat cardigenic shock, may include measures such as pericardiocentesis and intra-aortic balloon counterpulsation, useful in cases of haemodynamic instability that fail to respond to inotropic drugs[12]. Although the literature contains cases of survival with conservative treatment[11], the treatment of choice is surgical repair of the ventricular defect.

Despite high perioperative mortality, ranging from 33% to 52%[3,14], some cases of survival have been reported in patients of very advanced age[13].

Echocardiography should constitute part of the arsenal of diagnostic techniques for ED physicians in the resuscitation room, at the head of the patient, since in cases of shock suspected to be of cardiogenic origin this device allows identifying most causes of cardiac complications (tamponade, left or right ventricle systolic dysfunction, post-infarction mechanical complications etc.)[15].

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Pilot plan for teaching basic life support in schools: a spiral approach

Editor:

In the light of the EMERGENCIAS editorial[1] referring to teaching CPR in schools, so well reported by García Vega et al, we would like to make known our experience with a "Pilot project" for teaching basic life support (BLS) in schools, which is being carried out in educational centres of Tortosa. From the PROCES[2,3] experience, mentioned in the editorial, our evaluation and follow up has shown that students learn better when instructed by their teachers, and that most students have forgotten most of what they learnt after one year.

For these reasons we have implemented a project, involving physical education teachers in both the design of the teaching plan (methodology and teaching material) and its practice, in order for students to acquire long-lasting BLS knowledge and skills which are now included in the teaching curriculum of these schools. A pilot project was elaborated, directed not only at secondary school students but also at younger children, starting from initial schooling at the age of 4 years and continuing to the end of compulsory schooling.

BLS knowledge and skills are introduced step by step, geared to the age of the students, which means that learning takes place in a spiral manner to achieve consolidation and permanence. Figure 1 shows the specific learning objectives for each cycle. As the authors comment in their editorial, to maintain and extend this training to all educational centres in Cataluña, the pilot project is backed by official collaborating entities: the "Consejería de Salud" (Health Authority) and the "Consejería de Educación" (Education Board) of the autonomous government "Generalitat de Catalunya" and the "Consell Català de Ressuscitacio" (CCR), member of the national "Consejo Español de RCP" (CERCP).

After two years preparing the pilot project, it was first implemented in May 2008, and we are currently engaged in analysis of the results, not only of what was learnt by students but also the organizational and teaching aspects.

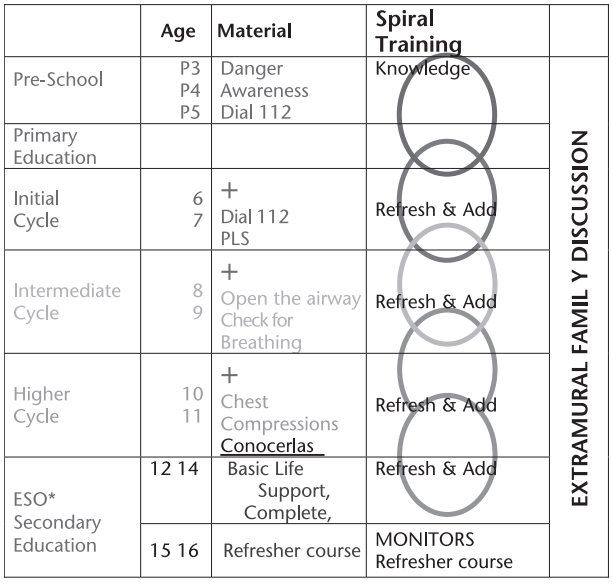

Figure 1. Learning using a spiral approach. Curricular content for teaching basic life support in schools.

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Reply

Editor:

It is most gratifying to learn that initiatives, with or without institutional support, are sprouting throughout the country to consolidate training in cardiopulmonary resuscitation (CPR), so

that this becomes considered a normal activity in our education and culture. Undoubtedly, without demeaning any other Autonomous Community, Cataluña is becoming a pioneer in this field. The pilot project of teaching Life Support in the schools of Tortosa, one of the fruits of PROCES, is good evidence of this.

Various links in the chain of CPR training are missing, and we have to resort to efforts on all fronts: public, private, voluntary, professional etc. From schools to universities, professional training and the health administration together may achieve a strong and consistent chain. Much remains to be done, with a lot of work and effort, but all investment in CPR training is worth it in the short, medium and long term.

Congratulations to all those responsible for the Tortosa schools BLS training project, and every encouragement to publicize their results so that all of us involved in emergency medicine training are kept informed and to serve as an example for other similar initiatives.

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Brugada Syndrome patient with an automatic implantable defibrillator that simultaneously delivered approriate and inapproriate electrical discharges

Editor:

Polymorphic ventricular tachycardia is the most frequent arrhythmia found in patients with Brugada syndrome, while monomorphic tachycardia is exceptional. Among the causes triggering tachyrhythmic events in these patients is hyperthermia. Treatment consists in placement of an automated implantable defibrillator (AID) that acts with antiarrhythmic therapy or electrical discharge synchronised with ventricular tachyrhythmia, treatment of triggering factors and patient sedation. One of the undesirable effects of the AID device is that it may deliver electrical discharge inappropriately due to erroneous diagnosis of ventricular arrhythmia, producing pain and anxiety in the patient.

We present the case of a 21-year-old man with Brugada syndrome diagnosed after an episode of sudden cardiac arrest in 1999 causing postanoxic encephalopathy, with good resultant quality of life limited by slight-moderate cognitive deterioration, in whom a prophylactic AID device was implanted. In August 2007 he presented at our emergency department with monomorphic ventricular tachycardia, triggered as on a previous occasion by hyperthermia, secondary to purulent tonsilitis. His AID generated effective anti-arrhythmic therapy. Antipyretic treatment was initiated with proparacetamol, benzodiacepine and isoproterenol iv. During observation, the AID began delivering inappropriate discharges related by the patient despite our monitor screen observation of normal sinus rhythm. The patient had to transported 50 Km away to a reference hospital with an arrhythmia unit. On the way in the emergency ambulance, the AID did not deliver any sort of therapy. When the device was tested, it was shown to deliver inappropriate therapy due to oversensitivity to T wave signals.

This case illustrates the dilemma produced when an AID begins simultaneous delivery of appropriate and inappropriate therapy, and the delay in its re-programming due to the absence of an arrhythmia unit in the same hospital. In this case we believe it recommendable to begin by treating known causes, sedate the patient, then, while monitoring the patient with placement of external electrical cardioversion electrode pads, disconnect the AID until specialised attention can be given by a reference hospital.

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Atypical complication of atypical pneumonia

Editor:

We present the case of a 56-year-old woman with a history of tuberculous meningitis at the age of 8 years, smoking 40 packets/year, and being a worker at a paper factory in an industrial area. She consulted our Emergency Department (ED) for fever of one week duration with slight unproductive cough, intense postration, dyspnea and pleural chest pain in the prvious few days. A sister working in the same place had been diagnosed with pneumonia without requiring hospital admission. The patient presented a temperature of 40ºC, respiratory frequency of 24 per minute, cardiac frequency of 110 bpm; lung auscultation revealed right base lung rales. Analytic findings included respiratory insufficiency (pO_2 56 mmHg; pCO_2 31 mmHg) and increased acute phase reactants (GSR 69 mm, CRP 30 mg/dl). Chest radiography (Figure1a) showed the presence of alveolar interstitial infiltrate in the right lung. Blood and sputum cultures were performed, as well as pneumococcus antigen test and serotype 1 *Legionella pneumophila* in urine.

She was diagnosed with acute respiratory failure and community-acquired pneumonia (CAP) FINE IV. Empiric antibiotic treatment was initiated with levofloxacine and she was admitted to the ED short-stay unit (SSU). Urine test was positive for Legionella and it was decided to continue treatment with levofloxacine. At 48 hours after admission the patient became afebrile. Chest radiography at that time showed the formation of a giant bleb in the right upper lobe (Figure 1b). Computerised tomography (CT) scan confirmed the presence of this bleb (Figure 1c). On the third day the patient developed complete pneumothorax of the right lung (Figure 1d) requiring the placement of a chest drainage tube.

In recent years there have been Spanish community outbreaks of legionellosis, in Alcalá de Henares in 1996[1], Vigo in 2000[2], Alcoy in 1999[3] , Barcelona and Murcia in 2001[4,5]. Furthermore, increased incidence has been detected, currently 3.5 per 100.000 persons/year. This increase is due to greater diagnostic capacity fundamentally as from the introduction of the urinary antigen assay in 1995.

In our case the availability in ED of this test not only allowed early aetiological diagnosis; collaboration with the department of preventive medicine resulted in the detection of Legionella in the patient´s sister as the causal agent of her pneumonia, and an investigation into the possible epidemic outbreak was launched.

Another noteworthy aspect of the case in terms of evolution was the complication of a large lung bleb and the subsequent development of secondary pneumothorax. Spontaneous pneumothorax usually presents in patients

with underlying lung pathology such as pulmonary emphysema or other less frequent diseases: connective tissue diseases (Marfan syndrome, Ehlers-Danlos), lung neoplasia, histocytosis X, sarcoidosis and lymphangioleiomyomatosis.

When spontaneous pneumothorax appears associated to an infectious lung process, the most frequent cause is tuberculosis followed by Pneumocistis jorivicii pneumonia in HIV patients and *Staphylococcus aureus* pneumonia with pneumatocele formation. The presence of pulmonary embolism in right endocarditis has also been described as an exceptional cause of spontaneous pneumothorax[6,7].

The presence of spontaneous pneumothorax is a rare complication of pneumonia due to *Legionella*. Radiological findings in CAP due to *Legionella* are not specific and vary from interstitial infiltrate to alveolar condensation; the presence of cavitation or pleural effusion is not at all frequent. In different series of pneumonia due to Legionella published in our country, none have described the presence of associated spontaneous pneumothorax[1-5]. Tan et al[8] published a retrospective study on radiological patterns of pneumonia due to *Legionella* and make no mention of bleb formation or pneumothorax. However, this rare complication has been reported before[9-11], with one recent publication of bilateral spontaneous pneumothorax associated with pneumonia due to *Legionella*[12].

To our knowledge, this is the first reported case with radiological evidence showing the evolution from alveolar infiltrate to bleb formation and subsequent pneumothorax associated with pneumonia due to Legionella.

In conclusion, we believe that rapid diagnostic tests for Legionella in EDs situated in high prevalence areas is amply justified, and that *Legionella* should be considered as a causal agent in patients with pneumothorax associated to pneumonia.

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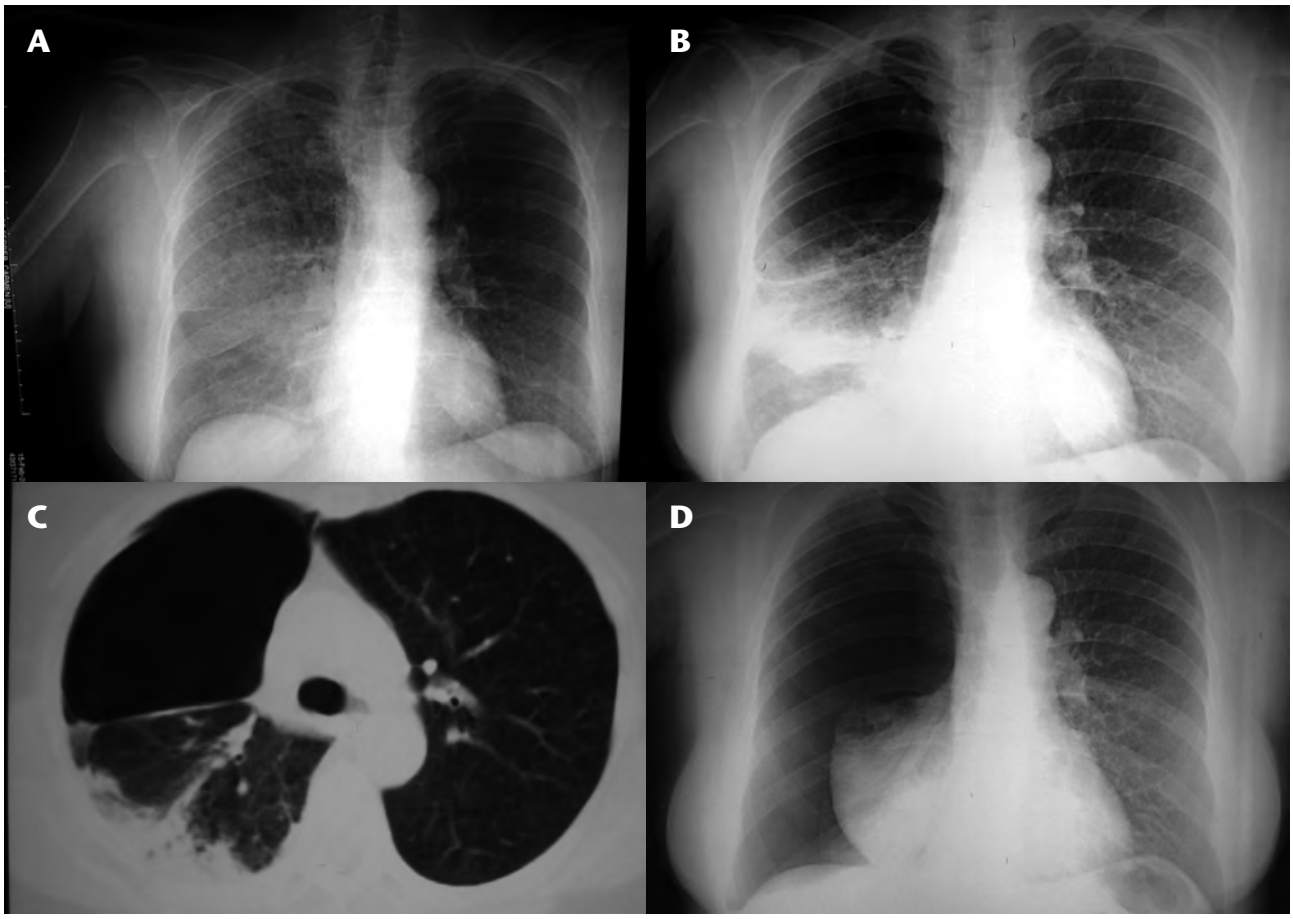

Figure 1. A) Interstitial-alveolar infiltrate in the whole right lung, predominantly in the left lower and middle lobe. B) Giant bleb in the upper half of the right lung. Infiltrate-condensation in lower and middle lobe. C) Bleb in upper right lobe. D) Complete peumothorax of the right lung with mediastinal displacement.

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Hypovolemic shock after spontaneous splenic rupture during an episode of acute pancreatitis

Editor:

A 79-year-old woman was attended at Emergency Department for nausea, vomiting and abdominal pain of 12 hours evolution. Medical history included arterial hypertension, primary hypothyroidism, gastro-duodenal ulcer, hiatus hernia, ischemic cardiopathy, paroxistic auricular fibrillation and cholecystectomy, and she was under treatment with sertraline, omeprazol, amlodipine, nitrates, diltiazem, lorazepan and acenocumarol. Physical examination showed mucocutaneous paleness, general malaise, hypotension (80/60 mmHg) and intense epigastralgia without signs of peritoneal irritation. Intravenous metamizol and omeprazol were administered. The pain intensified and abdominal defence began. Salient laboratory findings included: leucocytes 16.000/μl (12.700/μl neutrofils), RBC 3.44 x 10^6/μl, haemoglobin 9.3 g/dl, haematocrit 27%, platelets 317,000/μl and glucose 373 mg/dl, urea 48 mg/dl, creatinine 1.9

mg/dl, creatininkinase 60 U/L, troponine I: 0,62 ng/ml, α-amilase: 726 U/L, prothrombin activity: 12% (INR: 4.69). With a provisional diagnosis of acute pancreatitis (AP), treatment was initiated with insulin, saline therapy and subcutaneous petidine, but evolution was poor and she required orotracheal intubation and mechanical ventilation. Cerebral and abdominal computerised tomography (CT) scans (Figure 1) showed a perisplenic haematoma and a reduction of normal splenic volume, with suspected active bleeding. Emergency laparotomy was performed during which haemoperitoneum (1.000 cc) was found due to splenic rupture with a large perisplenic haematoma. Splenectomy was then performed and haemostasia achieved. Despite this, the patient died during the post-operative period.

Splenic rupture without prior trauma is a rare entity; it may be spontaneous (rupture of a normal spleen) or pathologic (rupture of a diseased spleen)1. Although the distal portion of the tail of the pancreas maintains a close relation with splenic vessels and the splenorenal ligament, spontaneous splenic rupture is described as a rare complication of pancreatitis in relation with: extension of necrotizing pancreatitis to the spleen, splenic hilum erosion due to pseudocyst in the tail of the pancreas, perisplenic adherences after recurrent pancreatitis, occlusion of the splenic vein due to pancreatic inflammation, or acute inflammation of ectopic pancreatic tissue within the spleen itself[2-5].

The abdominal CT scan did not reveal structural alterations of either the spleen or the pancreas. On reviewing clinical case reports, we found enzyme liberation in acute pancreatitis as a possible cause of the inflammatory lesion of the splenic capsule, perisplenic haemorrhage and abdominal haematoma[2,4]. Therefore, rupture of the spleen should be included in the differential diagnosis of shock secondary to acute pancreatitis[4].

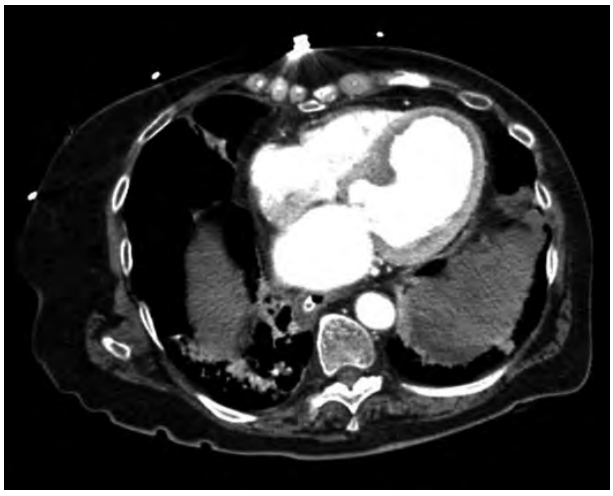

Figure 1. Perisplenic haematoma.

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