

LETTERS TO THE EDITOR

Idiopathic retroperitoneal fibrosis: a case report**Editor:**

Idiopathic retroperitoneal fibrosis (RPF) is a rare disease of unknown etiology. It is characterised by a retroperitoneal inflammatory process often involving the ureters and producing unilateral 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 maintenance 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 deposits³, generally in the retroperitoneum, 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.
- Bromocriptin.
- Betablockers.
- Methyldope.
- Hydralazine.
- LSD.

Retroperitoneal lesion:

- Radiation.
- Haemorrhage.
- Surgery.
- Extravasation urine.

Inflammatory processes:

- Periaortitis (abdominal aorta aneurysm).
- Sarcoidosis.
- Pancreatitis.
- Endometriosis.
- Autoimmune collagen vascular disease.

Infection:

- Tuberculosis.
- Histoplasmosis.
- Actinomycosis.

tion has been used, such as cyclophosphamide⁸, azathioprine^{8,10} or tamoxifen^{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.

References

- 1 Kottra JJ, Dunnick NR. Retroperitoneal Fibrosis. *Radiol Clin North Am* 1996;34:1259-75.
- 2 Mitchinson MJ. Retroperitoneal fibrosis, revisited. *Arch Pathol Lab Med* 1986;110:784-6.
- 3 Lepor H, Walsh PC. Idiopathic retroperitoneal fibrosis. *J Urol* 1979;122:1-6.
- 4 de Sá J, Pimentel J, Carvalho M, Evangelista P, Martins P. Spinal cord compression secondary to idiopathic retroperitoneal fibrosis. *Neurosurgery* 1990;26:678-81.
- 5 Scully RE, Mark JE, McNeely WF, Ebeling SH. A 31 year-old woman with lumbar and abdominal pain, hypertension and a retroperitoneal mass. *N Engl J Med* 1996;335:650-5.
- 6 Baker LRI, Mallinson WJW, Grogory MC, Menzies EAD, Cattell WR, Whitfield HN. Idiopathic retroperitoneal fibrosis. A retrospective analysis of 60 cases. *Br J Urol* 1988;60:497-503.
- 7 Mitchinson MJ. Some clinical aspects of idiopathic retroperitoneal fibrosis. *Br J Surg* 1972;54:58-60.
- 8 Monev S. Idiopathic retroperitoneal fibrosis: Prompt diagnosis preserves organ function. *Cleve Clin J Med* 2002;69:160-6.
- 9 Cogan E, Fastrez R. Azathioprine. An alternative treatment for recurrent idiopathic retroperitoneal fibrosis. *Arch Intern Med* 1985;145:753-5.
- 10 McDougal WS, MacDonell RC. Treatment of idiopathic retroperitoneal fibrosis by immunosuppression. *J Urol* 1991;145:112-4.
- 11 Spillane RM, Whitman GJ. Treatment of retroperitoneal fibrosis with tamoxifen. *Am J Roentgenol* 1995;164:515-6.

María Isabel LLULL FERRETJANS¹,
Miquel CALDENTEY TOUS²,
Antonia ROCA CASAS³

¹Centro de Salud Tramontana. Mallorca. Ib-salut, España.

²Centro de Salud Coll d'en Rebassa. Mallorca.

Ib-salut, España. ³Centro de Salud Es Trencadors. Mallorca. Ib-salut, España.

Subacute cardiac rupture as a complication of acute myocardial infarction

Editor:

Acute cardiac tamponade due to cardiac 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 duration. After 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 presents in 1-4% of hospitalised AMI patients, responsible for 15-20% of AMI-related deaths¹. 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 reperfusion⁴.

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 age⁷⁻⁹.

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

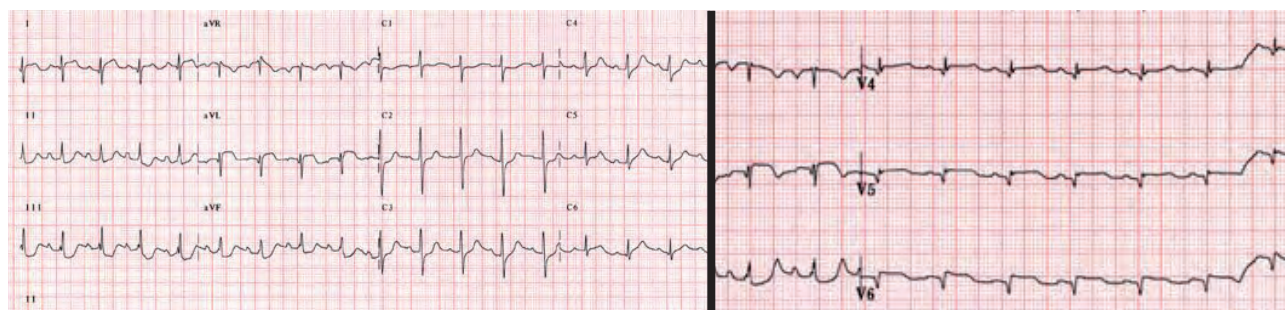


Figure 1. ECG on admission: Sinus tachycardia 110 bpm. ST segment elevation in lead DI y aVL with anterior ST and V2-V3 descent, with ST elevation in posterior leads (V4-V6 ECG of the right).

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 cardiogenic shock, may include measures such as pericardiocentesis and intra-aortic balloon counterpulsation, useful in cases of haemodynamic instability that fail to respond to inotropic drugs¹². Although the literature contains cases of survival with conservative treatment¹¹, 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 age¹³.

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.

References

- Antmann E, Braunwald E. Acute Myocardial infarction. En: Braunwald E, Zipes D, Libby P editores. Heart Disease: A Textbook of cardiovascular medicine (6th edition). Philadelphia: Saunders 2001;1114-1232.
- Raitt MH, Kraft CD, Gardner CJ, Pearlman AS, Otto CM. Subacute ventricular free wall rupture complicating myocardial infarction. *Am Heart J* 1993;126:946-55.
- López-Sendón J, González A, López de Sa E, Coma-Canella I, Roldán I, Domínguez F. Diagnosis subacute ventricular wall rupture after acute myocardial infarction: sensibility and specificity of clinical, hemodynamic and echocardiographic criteria. *J Am Coll Cardiol* 1992;19:1145-53.
- Raposo L, Andrade MJ, Ferreira J, Aguiar C, Couto R, Abecasis M, et al. Subacute left ventricle free wall rupture after acute myocardial infarction: awareness of the clinical signs and early use of echocardiography may be life-saving. *Cardiovasc Ultrasound* 2006;4:46.
- Pollak H, Nobis H, Mlczech J. Frequency of left ventricular free wall rupture complicating acute myocardial infarction since advent of thrombolysis. *Am J Cardiol* 1994;74:184-186.
- Honan MB, Harrell FE Jr, Reimer KA, Califf RM, Mark DB, Pryor DB, et al. Cardiac rupture, mortality and the timing of thrombolytic therapy: a meta-analysis. *J Am Coll Cardiol* 1990;16:359-67.
- Moreno R, López-Sendón J, García E, Pérez de Isla L, López de Sá E, Ortega A, et al. Primary angioplasty reduces the risk of left ventricular free wall rupture compared with thrombolysis in patients with acute myocardial infarction. *J Am Coll Cardiol* 2002;39:598-603.
- Bueno H, Martínez-Sellés M, Pérez-David E, López-Palop R. Effect of thrombolytic therapy on the risk of cardiac rupture and mortality in older patients with first acute myocardial infarction. *Eur Heart J* 2005;26:1705-11.
- Keeley FC, de Lemos JA. Free wall rupture in the elderly: deleterious effect of fibrinolytic therapy on the ageing heart. *Eur Heart J* 2005;26:1693-4.
- Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. *J Am Coll Cardiol* 1993;22:720-6.
- Figueras J, Cortadellas J, Evangelista A, Soler-Soler J. Medical managements of selected patients with left ventricular free wall rupture during myocardial infarction. *J Am Coll Cardiol* 1997;29:512-8.
- Amir O, Smith R, Nishikawa A, Gregoty ID, Smart FW. Left ventricular free wall rupture in acute myocardial infarction: a case report and literature review. *Tex Heart Inst J* 2005;32:424-6.
- Daya SK, Tan D, Tolerico PH, Gowda RM, Khan IA. Survival of an octogenarian after rupture of the left ventricular free wall caused by myocardial infarction. *Tex Heart Inst J* 2004;31:178-80.
- Purcaro A, Constantini C, Ciampani N, Mazzanti M, Silenzi C, Gili A, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. *Am J Cardiol* 1997;80:397-405.
- Joseph MX, Disney PJ, Da Costa R, Hutchinson SJ. Transthoracic echocardiography to identify or exclude cardiac cause of shock. *Chest* 2004;126:1592-7.

Carlos BEAUMONT CAMINOS¹,
Isabel IDOATE SANTESTEBAN²,
Clint JEAN-LOUIS LAWRENCE¹,
Ana Carmen CABODEVILLA GÓRRIZ¹

¹Servicio de Urgencias. ²Servicio de Cardiología. Hospital de Navarra, España.

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

Editor:

In the light of the EMERGENCIAS editorial¹ 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 Ressuscitació" (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

	Age	Material	Spiral Training	EXTRAMURAL FAMILIAR Y DISCUSION
Pre-School	P3 P4 P5	Danger Awareness Dial 112	Knowledge	
Primary Education				
Initial Cycle	6 7	+ Dial 112 PLS	Refresh & Add	
Intermediate Cycle	8 9	+ Open the airway Check for Breathing	Refresh & Add	
Higher Cycle	10 11	+ Chest Compressions Conocerlas	Refresh & Add	
ESO* Secondary Education	12 14	Basic Life Support, Complete,	Refresh & Add	
	15 16	Refresher course	MONITORS Refresher course	

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

currently engaged in analysis of the results, not only of what was learnt by students but also the organizational and teaching aspects.

References

- García Vega FJ, Montero Pérez FJ, Encinas Puente R. La comunidad escolar como objetivo de la formación en resucitación: la RCP en las escuelas. *Emergencias* 2008;20:223-5.
- Miró O, Escalada X, Jiménez-Fábrega X, Díaz N, Sanclemente G, Gómez X, et al. Programa de Reanimación Cardiopulmonar orientado a Centros de Enseñanza Secundaria (PROCES). *Emergencias* 2008;20:229-36.
- Miró O, Sánchez M, Jiménez-Fábrega X, Escalada-Roig X. Teaching basic life support in schools: still waiting for public funding. *Resuscitation* 2008;77:420-1.

Manel Cerdà VILA¹,
Manel R. CHANOVAS BORRÀS^{1,2},
Cinta ESPUNY VIDAL³,
Xavier ESCALADA ROIG¹ y
Grupo de Trabajo de SVB a Educació
de les Terres de l'Ebre¹⁻³

¹Consell Català de Ressuscitació.

²Serveis Territorials de Salut a les Terres de l'Ebre, España.

³Centre de Recursos, Serveis Territorials d'Educació a les Terres de l'Ebre, España.

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.

Francisco Javier GARCÍA VEGA^{1,2},
Francisco Javier MONERO PÉREZ¹,
Rosa María ENCINAS PUENTE¹

¹Secretaría de Formación de la SEMES, España.

²Secretario del Comité Ejecutivo del Consejo Español de RCP (CERCP), España.

Brugada Syndrome patient with an automatic implantable defibrillator that simultaneously delivered appropriate and inappropriate 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 tachyarrhythmic 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 tachyarrhythmia, 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 car-

diac 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 tonsillitis. His AID generated effective antiarrhythmic therapy. Antipyretic treatment was initiated with paracetamol, benzodiazepine 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 be 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.

References

- 1 Antzelevitch C, Brugada R. Fever and Brugada syndrome: Pacing Clin Electrophysiol 2002;25:1537-9.
- 2 Peinado Peinado R, Martín Martínez A, González Torrecilla E, Laguna Del Estal P, Ormaetxe Merodio J, Suero Méndez C, et al. Manejo de los pacientes portadores de un desfibrilador automático implantable en los servicios de urgencias hospitalarias. Emergencias 2005;17:180-96.
- 3 Martín Martínez A, Peinado Peinado R, González Torrecilla E, Ormaetxe Merodio J, Álvarez López M, Del Arco Galán C, et al. El desfibrilador automático implantable. Actualización para médicos de urgencias. Emergencias 2007;19:77-87.
- 4 Mok NS, Chan NY. Brugada syndrome presenting with sustained monomorphic ventricular tachycardia. Int J Cardiol 2004;97:307-9.
- 5 Pinar E, García-Alberola A, Martínez J, Sánchez JJ, Valdés M. Spontaneous sustained ventricular tachycardia after administration of ajmaline in a patient with Brugada syndrome. Pacing Clin Electrophysiol 2000;23:291-2.
- 6 Boersma LV, Jaarsma W, Jessurun ER, Van Hemel NH, Wever EF. Brugada syndrome. A case report of monomorphic ventricular tachycardia. Pacing Clin Electrophysiol 2001;24:112-5.
- 7 Dinckal MH, Davutoglu V, Akdemir I, Soyuncu S, Kirilmaz A, Aksoy M. Incessant monomorphic ventricular tachycardia during febrile illness in a patient with Brugada syndrome: fatal electrical storm. Europace 2003;5:257-61.

Jesús Ángel CRUZADO QUEVEDO¹
Juan José SÁNCHEZ MUÑOZ²

¹Unidad de Urgencias. Hospital Universitario Santa María del Rosell Cartagena. Murcia, España.

²Unidad de Arritmias. Hospital Universitario Virgen Arrixaca. Murcia, España.

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 prostration, dyspnea and pleural chest pain in the previous few days. A sister working in the same place had been diagnosed with pneumonia without requiring hospital admission. The patient presented a temperature of 40°C, respiratory frequency of 24 per minute, cardiac frequency of 110 bpm; lung auscultation revealed right base lung rales. Analytic findings included respiratory insufficiency (pO₂ 56 mmHg; pCO₂ 31 mmHg) and increased acute phase reactants (GSR 69 mm, CRP 30 mg/dl). Chest radiography (Figure 1a) 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 levofloxacin 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 levofloxacin. 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 1996¹, Vigo in 2000², Alcoy in 1999³, 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, histiocytosis X, sarcoidosis and lymphangioleiomyomatosis.

When spontaneous pneumothorax appears associated to an infectious lung process, the most frequent cause is tuberculosis followed by *Pneumocystis jirovecii* 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 pneumothorax¹⁻⁵. Tan et al⁸ 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 before⁹⁻¹¹, 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.

References

- 1 Informe preliminar de brote de neumonía por *Legionella* en Alcalá de Henares. Bol Epidemiol Sem 1996;16:129-36.
- 2 Dirección Xeral de Saúde Pública. La gestión de un brote de legionelosis vivido como una crisis de salud pública. Gac Sanit 2001;15:77-9.
- 3 Fernández JA, López P, Orozco D, Merino J. Clinical study of an outbreak of Legionnaire's disease in Alcoy, Southeastern Spain. Eur J Clin Microbiol Infect Dis 2002;21:729-35.
- 4 Jerico C, Nogués X, Santos MJ, Féliz M, Garcés JM, Mariñosa M, et al. Brote epidémico de neumonía comunitaria por *Legionella pneumophila* en Barcelona: "el brote de la baceloneta". Efectos del diagnóstico y tratamiento precoz. Rev Clin Esp 2004;204:70-4.
- 5 Garción-Fulgueiras A, Navarro C, Fenoll D, García G, González-Giego

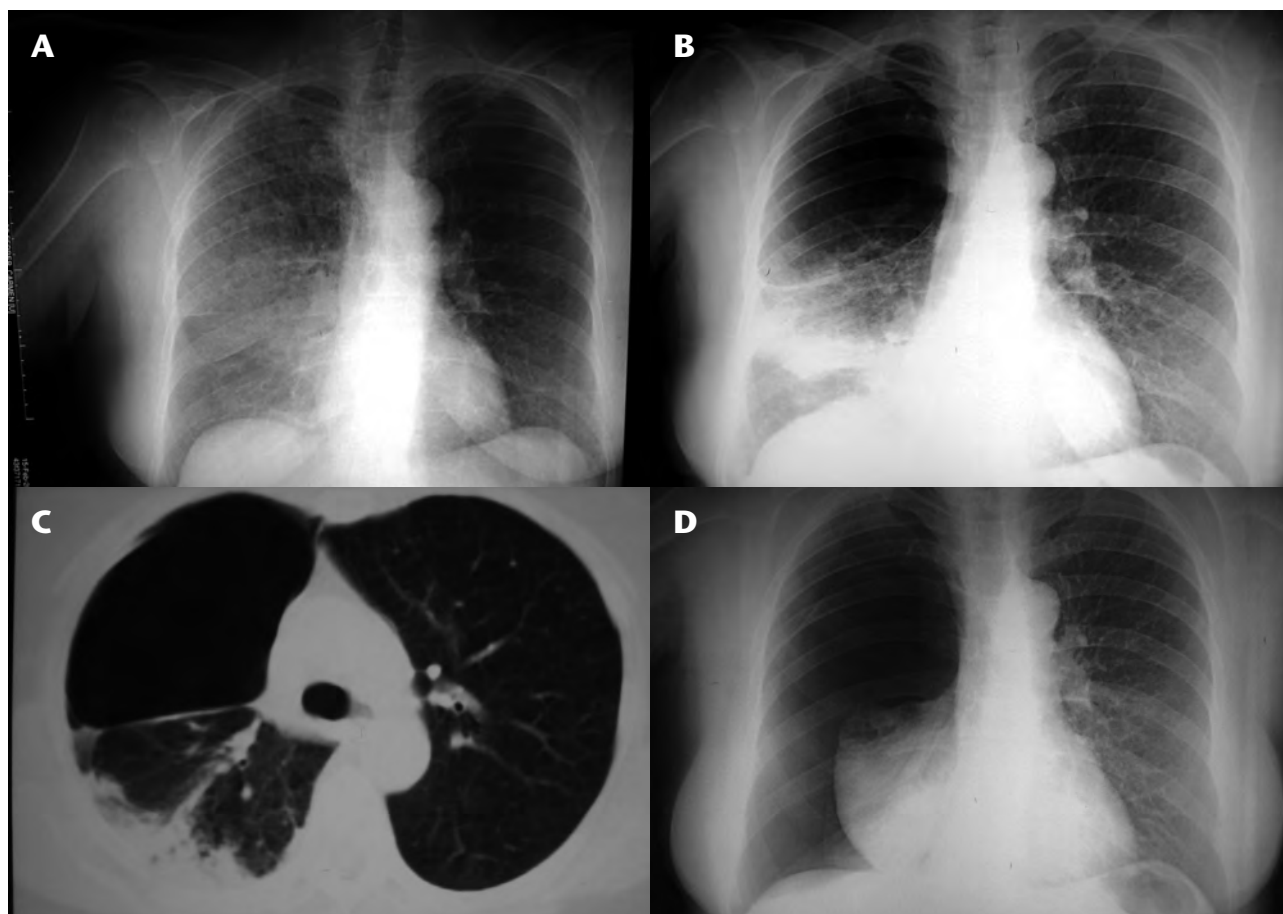


Figure 1. A) Interstitial-alveolar infiltrate in the whole right lung, predominantly in the left lower and middle lobe. B) Giant bleb in the upper half of the right lung. Infiltrate-condensation in lower and middle lobe. C) Bleb in upper right lobe. D) Complete pneumothorax of the right lung with mediastinal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

- P, Jiménez-Buñuales T, et al. Legionnaire's disease outbreak in Murcia, Spain. *Emerg Infect Dis* 2003;9:915-21.
- 6 Tumbarello M, Tacconelli E, Pirroni T, Cauda R, Ortola L. Pneumothorax in HIV-infected patients: role of *Pneumocystis carinii* pneumonia and pulmonary tuberculosis. *Eur Respir J* 1997;10:1332-5.
- 7 Sahn SA, Heffner JE. Spontaneous pneumothorax. *N Engl J Med* 2000;342:868-74.
- 8 Tan MJ, Tan JS, Hamor RH, File TM Jr, Breiman RF. The radiologic manifestations of Legionnaire's disease. The Ohio Community-Based Pneumonia Incidence Study Group. *Chest* 2000;117:398-403.
- 9 Gervais A, Beghetti M, Rimensberger P, Posfay-Barbe K, Barazzone C. Bullous emphysema after *Legionella* pneumonia in a two-year-old child. *Pediatr Infect Dis J* 2000;19:86-7.
- 10 Sundkvist T, Carlsson MG. Legionnaires Disease: unusual presentation with pneumothorax. *Scand J Infect Dis* 1983;15:127-8.
- 11 Fraux B, Macher MA, Loirat C, Tournier G. Pneumothorax au cours d'une légionellose chez un transplanté rénal. *Press Med* 1991;1394-5.
- 12 Gunha B, Pherez FM, Nouri Y. *Legionella* community-acquired pneumonia (CAP) presenting with spontaneous bilateral pneumothoraces. *Heart Lung* 2008 ;37:238-41.

José Manuel MURCIA ZARAGOZA,
Fadoua LAGHZAOUJ,
Francisco ROMÁN CERDÁ,
Pere LLORENS SORIANO

Unidad de Corta Estancia. Servicio de Urgencias.
Hospital General Universitario de Alicante, España.

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, paroxysmic auricular fibrillation and cholecystectomy, and she was under treatment with sertraline, omeprazol, amlodipine, nitrates, diltiazem, lorazepam and acenocumarol. Physical examination showed mucocutaneous paleness, general malaise, hypotension (80/60 mmHg) and intense epigastralgia without signs of peritoneal irritation. Intravenous metemizol and omeprazol were administered. The pain intensified and abdominal defence began. Salient laboratory findings included: leucocytes 16.000/ μ l (12.700/ μ l neutrofiles), RBC 3.44×10^6 / μ l, haemoglobin 9.3 g/dl, haematocrit 27%, platelets 317,000/ μ l 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 adhesences after recurrent pancreatitis, occlusion of the splenic vein due to pancreatic inflammation, or acute inflammation of ectopic pancreatic tissue within the spleen itself²⁻⁵.

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 pancreatitis⁴.

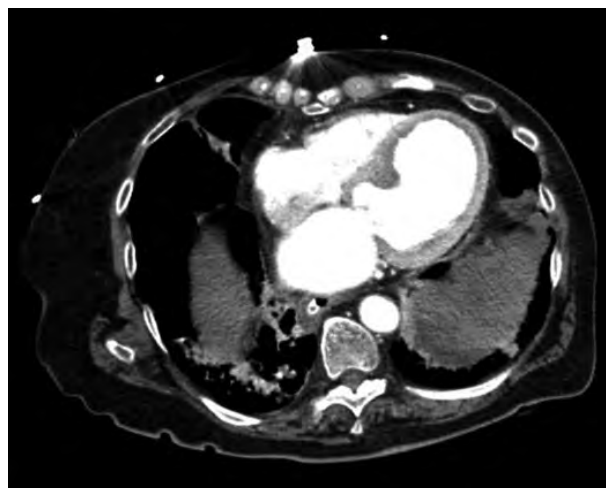


Figure 1. Perisplenic haematoma.

References

- 1 Hernández Siberio González N, Pérez Palma J, Márquez MA, Díaz Flores L. Rotura espontánea esplénica relacionada con enfermedad pancreática. *Cir Esp* 2002;72:359-61.
- 2 Ramos Ramos JC, García Díaz JD. Rotura esplénica espontánea como complicación de una pancreatitis aguda. *Med Clin (Barc)* 2002;119:478-9.
- 3 Gázquez I, Vicente de Vera P, García Valencia MJ, López A. Infartos esplénicos y hematoma subcapsular en el curso de una pancreatitis crónica. *Med Clin (Barc)* 2003;120:559.
- 4 Ortega Carnicer J. Rotura esplénica espontánea como complicación de una pancreatitis aguda. *Med Intensiva* 2006;30:474-5.
- 5 Nicolás de Prado I, Corral de la Calle MÁ, Nicolás de Prado JM, Gallardo Sánchez F, Medranda MÁ. Complicaciones vasculares de la pancreatitis. *Rev Clín Esp* 2005;205:326-32.

Isabel GIL ROSA,
Emilio SÁNCHEZ-PARRA MARÍN,
M^a Cruz DE JUAN SÁNCHEZ,
Antonio MARTÍNEZ GARCÍA,
Juan José CERVANTES CONESA

*Servicio de Urgencias. Hospital General Universitario
"Reina Sofía". Murcia, España.*