LETTERS TO THE EDITOR

Particularities of epidural hematoma in the infant

Sir,

Slight closed head injuries are extremely frequent in childhood. They usually are of little clinical relevance and patients may be discharged after a few hours, with standard home observation. There is however an age group, below two years and especially below one year of age, requiring special consideration. These patients may present an epidural hematoma with hardly any clinical symptoms. This often raises doubts about what approach to take with children visiting ED for seemingly banal trauma. We present a case in point of this problem.

An 8-month-old infant girl attended the ED of a local hospital after an accidental fall from her cot 14 hours earlier. She had right temporal trauma without loss of consciousness, with irritability in the following hours and phases of drowsiness accompanied by vomiting three times. Physical examination showed right temporal contusion with a Glasgow Coma Score (GCS) of 15, normotensive fontanel, without signs of neurologic focality, and hemodynamic stability. Blood pressure (BP) was 96/54 mm Hg, heart rate 151 bpm, arterial oxygen saturation measured by pulse oximetry 100% and axillary temperature of 37ºC. The rest of the physical examination was normal except for pale skin and irritability. Blood test on admission showed hemoglobin 5.9 g/dL. Computed tomography (CT) cranial scan showed a right temporoparietal epidural hematoma, with maximum dimensions of 25 x 85 x 70 mm and a craneocaudal length of 7 mm, resulting in a mass effect with pressure on the right lateral ventricle and a midline shift leftwards of 7.5 mm. She presented bone fracture of the right temporal scale (Figure 1). She was transferred to a neurosurgical center, given the short duration of the transfer (30 minutes) and the GCS of 15, without intubation. The ambulance care team observed during transport a decrease in the GCS to 10, so hypertonic saline perfusion was administered. On arrival at the receiving centre, she presented hypotension (BP 64/41 mm Hg); she received orotracheal intubation, transfusion of red blood cells and volume expansion with crystalloids and noradrenalin infusion pump. She presented generalized hypertonia with bilateral mydriasis which responded to a bolus of midazolam and hyperventilation. Right parieto-temporal craniotomy and hematoma drainage were performed, with extubation 24 hours later. Thereafter, she showed good clinical evolution.

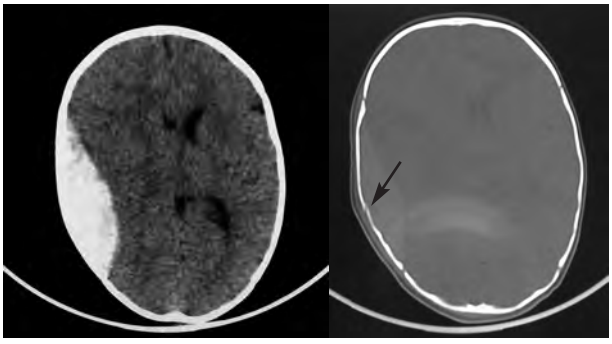

Figure 1. Right temporoparietal epidural hematoma (left) and fracture of the right temporal bone (right, arrow).

The incidence of epidural hematoma in children is lower than that in adults. Suspected diagnosis is made, as in the case of the adult patient, by the presence of clinical symptoms such as vomiting, decreased consciousness or neurologic focality after suffering head trauma. The diagnosis is confirmed by CT scan findings[1].

Infants present a peculiarity: the existence of an open fontanel and lack of closure of the sutures, which confers high cranial vault distensibility in these children, and allows the accumulation of a large amount of blood without signs of brain structure compression. Therefore, on appearance of a collection of blood in the epidural pace, especially if the bleeding is slow, intracranial pressure is accommodated, extending the window period between trauma and the emergence of clinical symptoms[2]. This is especially relevant in this age group where epidural hematomas occur after slight trauma caused by minor impacts.

In our case the patient suffered a fall from the cradle and the symptoms did not appear until 12 hours later. Even so, at the time of ED admission in the first hospital, they were scarce and consisted solely of irritability, paleness, and vomiting. Despite the presence of a large epidural hematoma, the infant had a GCS of 15, a normotensive fontanel and there were no signs of neurologic focality.

The second noteworthy fact is the large decrease in hemoglobin, solely attributable to the epidural hematoma. Infants are the only group that may present hypovolemic shock due to intracranial hemorrhage. This fact is due to their head volume which, in relation to body size, is proportio-

nally much larger than in adults[3]. On first admission, the patient maintained normal blood pressure despite the hemoglobin level of 5.9 g/l. On arrival at the reference hospital, she showed clinical signs of hypovolemic shock. This is the third important point: children, especially infants, maintain normal BP values because of vasoconstriction, despite having lost up to 35% of blood volume. After this point, blood pressure falls sharply.

Finally, CT scan showed the presence of skull fracture. Several authors have described that, in the infant, there is a direct relationship between a skull fracture and the presence of epidural hematoma, so the question arises: should skull X-ray be routinely performed in infants presenting minor head injury, as a screening method? The answer depends on the clinical evaluation. In patients with no clinical expression and no external signs of injury, standard home observation or short-stay admission for a few hours of observation may be sufficient[4]. Patients with significant skull hematoma and thus a high possibility of skull fracture, despite the absence of symptoms, should receive plain X-ray of the skull, followed by CT scan if there is fracture[5-7]. Finally, infants showing signs of alarm such as vomiting and irritability, despite a high GCS and no neurological focality, should undergo CT scan[8,9].

References

1 Tasker RC, Morris KP, Forsyth RJ, Hawley CA. Severe head injury in children: emergency access to neurosurgery in the United Kingdom. Emerg Med J 2006;23:519-22.
2 Benii-Adani M, Flores Jspektor S, Urnansky F, Constantini S. Epidural hematoma in infants. J Trauma 1999;46:306-11.
3 Shade SJ, Lie ST, Draaisma JM. Three infants with epidural hematoma. Ned Tijdschr Geneeskd 2001;45:182-8.
4 Holsti M, Kadish HA, Sill BL, Firth SD, Nelson DS. Pediatric closed injuries treated in an observation unit. Pediatr Emerg Care 2006;22:392.
5 Gruskin KD, Schtzman SA. Head trauma in children younger than 2 years: are there predictors for complications? Arch Pediatr Adolesc Med 1999;153:15-20.
6 Jiménez García R, García Teresa MA. Traumatismo craneoencefálico leve. En: Casado Flores J, Castellanos A, Serrano A, y Teja JL, editores. El niño politraumatizado. Madrid. Ergon 2004. p. 153-160.
7 Cambra FJ, Palomeque A. Traumatismos caraneoencefálicos. An Pediatr Contin 2005;3:327-34.
8 Simon B, Letourneau P, Vitorino E, McCall J. Pediatric minor head trauma: indications for computed tomographic sacanning revisited. J Taruma 2001;51:237-8.
9 Bor-Seng-Shu E, Aguiar PH, Matushita H, Manreza LA, Ferreira AA. Actual asyntomatic epidural hematomas in chilhooh. Report of three cases. Childs Nerv Syst 1997;13:605-7.

Eduardo CARRERAS GONZÁLEZ[1],
Beatriz BALSERA BAÑOS[2],
María Teresa COLL SIBIÑA[2],
Patricia FEBLES GONZÁLEZ[1]

[1]UCI Pediátrica Hospital Sant Pau. Universidad Autónoma de Barcelona, Spain. [2]Servicio de Pediatría. Hospital General de Granollers. Barcelona, Spain.

Influence of gender in the management of patients with non-traumatic chest pain in the Emergency Department of a tertiary hospital

Sir,

Recently, Riesgo et al. have published an article in your journal on the differences between men and women in the management of non-traumatic chest pain. Our hospital has a chest pain unit (CPU) attached to our ED and coordinated together with a heart unit. The patients included in this CPU are those presenting non-traumatic chest pain with non-diagnostic ECG and positive myocardial markers at the time of arrival at ED but negative 6 hours later. This type of patient corresponds to the protocol P3 of the Riesgo publication[2]. Of 1,091 patients included between 01 June 2006 and 31 June 2008, 441 were women (40.4%), with a mean age significantly higher than that of the men [63.9 (SD 11.7) vs. 59.4 (SD 13.9); $p < ,001$], similar to the population studied in the above-mentioned publication[1]. We found no significant gender differences in door-ECG time; median (interquartile rangel) for women being 14 (18) minutes vs. 15 (18) minutes for men. One of the reasons for this was the more atypical presentation of these episodes in the women[3]. Our data base included a series of variables related with the symptoms of these episodes (Table 1) and no significant gender differences were found in these or in the percentage of normal ECG findings.

Non-significant differences were found in positive ergometry (11.6% in women vs. 15.4% in men; $p = 0,123$). According to these results, despite the age difference between the women and the men, we can conclude that the presentation of chest pain in women is similar to that in men, with very similar percentages of positive ergometry. In the initial management expressed as door-ECG time, no gender differences were observed, although this is basically due to application of a consensus protocol for managing these patients that marks a maximum period for ECG performance after triage. Such protocols tend to guarantee equity in terms of gender management in the ED. On interpreting these results, it should be noted that they were obtained from a particular population with low-moderate risk of heart disease, with only a slight gender difference in prevalence, despite the clear difference in age.

Table 1. Characteristics of chest pain by gender

Clinical characteristics of Chest pain	Men (%)	Women (%)	p
Type of pain			
Oppressive	84.1	82.1	0.804
Sharp	5.6	6.1	
Malaise	5.0	7.2	
Other	5.2	4.6	
Location of pain			
Retrosternal	48.5	49.4	0.614
Precordial	41.7	38.4	
Epigastrium	5.7	5.2	
Rib ends	2.2	4.4	
Upper limbs	1.9	2.6	
Associated symptoms			
Vegetative cortège	45.9	51.9	0.118
Dizziness	17.5	15.4	
Dyspnea	17.1	14.2	
Syncope	1.2	2.5	
Other	18.3	16.0	
Pain trigger			
None	39.4	35.5	0.787
Sleep	31.9	29.0	
Effort	26.4	30.5	
Stress	2.3	5.0	
Modifiers of pain			
Breathing	2.7	2.6	0.596
Palpation	0.7	0.5	
Movement	2.7	3.8	
None	93.9	93.2	
Duration			
< 5 minutes	9.6	15.2	0.060
5-20 minutes	52.2	48.9	
> 20 minutes	38.2	35.9	
Number of crises in the last 24 hours			
0-2	78.8	81.1	0.408
> 2	21.2	18.9	
Relief from pain:			
Sleep	46.6	51.5	0.183
Sublingual nitrates	49.2	45.0	
Anxiety	1.9	1.7	
Analgesics	2.3	1.7	

References

1 Riesgo A, Bragulat E, López-Barbeito B, Sánchez M, Miró O. Aproximación diagnóstica al dolor torácico en urgencias: ¿existen diferencias entre mujeres y hombres? Emergencias 2008;20:399-404.

2 Bragulat E, López B, Miró O, Coll-Vinent B, Jiménez S, Aparicio MJ, et al. Análisis de la actividad de una unidad estructural de dolor torácico en un servicio de urgencias hospitalaria. Rev Esp Cardiol 2007;60:276-84.

3 Kaul P, Chang W-Ch, Westerhout CM, Graham MM, Armstrong PW. Differences in admission rates and outcomes between men and women presenting to emergency departments with coronary syndromes. CMAJ 2007;177:1193-7.

Pablo HERRERO PUENTE,
Luis ANTUÑA MONTES,
José Juan GIL ROMÁN,
Carlos GARCÍA CERECEDO

Área de Urgencias. Hospital Universitario Central de Asturias, Spain.

Authors' response:

Sir,

We are extremely pleased to answer the letter by Herrero et al. regarding our article[1], because we believe it leads to an interesting debate and possibly future joint and/or multicentre studies. In essence, the authors highlight two facts in the letter. The first is that in their series of patients initially diagnosed with chest pain of possible coronary origin in the chest pain unit (CPU) of their hospital emergency department (ED), there were no significant differences in Door-ECG time between men and women. This does not substantially differ from the results of our study, which concluded that chest pain diagnosed in the ED may appear less intense in women, but many of the initial differences observed disappear when time was stratified by age.

It is obvious that the more advanced age of the women (compared to the men) consulting ED for chest pain found in both studies is an essential factor to consider. The second aspect that Herrero et al. stress is the absence of differences in symptom presentation of chest pain between the sexes. The different presentation of chest pain according to sex has been described in studies from other countries, especially the United States and specifically for black people, which has been largely attributed to differences (or inequalities, depending on point of view) in the management of patients with acute coronary syndrome[2-4]. Although not one of our original objectives, we reanalysed our series taking into account some aspects of the type of chest pain for which data were available. In addition, we limited our analysis to the same group of patients which Herrero et al have described. Without considering the different prevalence found in each of the series (Table 1), we want to emphasize that, in our experience, chest pain of possible coronary origin shows oppressive features more frequently in women than in men, and also that pain is reproduced more frequently with chest pressure.

The only thing these discrepancies illustrate is the need for a more general approximation to the problem, since both our series and that of Herrero et al. will surely have their own biases due to idiosyncrasies of each reference area, hospital, ED or even each CPU. Although there are Spanish multi-centre studies that have magnificently illustrated the reality of care of ischemic heart disease in the ED[5,6], the characteristics of chest pain and equity of attention in those services are still unknown. Thus we believe that a future multi-centre study is highly necessary to elucidate unanswered questions. We would therefore encourage all those authors with data to offer them for the common good.

Table 1. Characteristics of chest pain by gender

	Women N (%)	Men N (%)	p
Type of pain			**0.02**
Oppressive	714 (78.2%)	883 (73.7%)	
Other	199 (21.8%)	315 (26.3%)	
Location of pain			**0.20**
Retrosternal or precordial	881 (88.8%)	1,041 (86.9%)	
Other	102 (11.2%)	157 (13.1%)	
Pain trigger			**0.49**
Stress	303 (33.2%)	416 (34.7%)	
Other or none	610 (66.8%)	782 (65.3%)	
Modification of pain with breathing			**0.34**
Yes	154 (18.2%)	183 (16.4%)	
No	693 (81.8%)	931 (83.6%)	
Modification of pain with palpation			**< 0.001**
Yes	139 (16.6%)	94 (8.4%)	
No	700 (83.4%)	1,024 (91.6%)	
Number of episodes in the last 24 h			**0.93**
0 2	494 (61.6%)	658 (61.9%)	
More than 2	308 (38.4%)	405 (38.1%)	

References

1 Riesgo A, Bragulat E, López-Barbeito B, Sánchez M, Miró O. Aproximación diagnóstica al dolor torácico en urgencias: ¿existen diferencias entre hombres y mujeres? Emergencias 2008;20:399-404.

2 Shaw LJ, Shaw RE, Merz CN, Brindis RG, Klein LW, Nallamothu B, et al. Impact of ethnicity and gender differences on angiographic coronary artery disease prevalence and in-hospital mortality in the American College of Cardiology-National Cardiovascular Data Registry. Circulation 2008;117:1787-801.

3 Hravnak M, Whittle J, Kelley ME, Sereika S, Good CB, Ibrahim SA, et al. Symptom expression in coronary heart disease and revascularization recommendations for black and white patients. Am J Public Health 2007;97:1701-8.

4 Pezzin LE, Keyl PM, Green GB. Disparities in the emergency department evaluation of chest pain patients. Acad Emerg Med 2007;14:149-56.

5 García-Castrillo Riesgo L, Loma-Osorio AL, Recuerda martínez E, Muñoz Cacho P. La cardiopatía isquémica en los servicios hospitalarios. Proyecto EVICURE. Emergencias 2000;12:183-90.

6 García-Castrillo Riesgo L, Recuerda Martínez E, Loma-Osorio A, García-Camarero T, García-Cases C, Epelde Gonzalo F, et al. Características y manejo de los pacientes con dolor torácico no traumático en los servicios de urgencias hospitalarios. Resultados del estudio EVICURE II. Emergencias 2008;20:391-8.

Alba RIESGO, Ernest BRAGULAT,
Miquel SÁNCHEZ, Òscar MIRÓ

Sección de Urgencias Medicina, Área de Urgencias, Hospital Clínic, Barcelona, Spain.

Painful left bundle branch block

Sir,

Consensus guidelines recommend reperfusion as the treatment of choice for patients with acute left bundle branch block (LBBB) and clinical features suggestive of acute myocardial infarction (AMI)[1]. However, cases of chest pain and acute LBR in patients with healthy hearts have been reported, whose appearance is dependent on heart frequency. It is called "painful LBBB", the treatment of which does not include therapeutic reperfusion[2]. We describe the case of a patient who presented painful LBBB, whose evolution after conservative treatment was satisfactory.

This was a 52-year-old woman, with a history of high blood pressure (BP) and depression. Admitted in 2003 and 2008 for episodes of chest pain, in both cases with normal coronariography. In April 2008 she underwent stress ergometry performed by her cardiologist, due to persistence of chest discomfort, when heart rate-dependent LBBB was observed. She began treatment with oral verapamil (80 mg every 12 hours). She consulted our ED for a new episode of chest pain which was non-oppressive and without irradiation, continuous, of several hours' evolution, accompanied by a feeling of general weakness, headache and palpitations. These episodes had occurred for several days before the consultation and coincided with stress associated with moving house and poor BP control. Physical examination only showed tachycardia on auscultation, blood pressure of 202/98 mm Hg and a heart rate of 104 beats per minute. Electrocardiogram (ECG) showed sinus tachycardia with acute LBR, and she was transferred to a resuscitation bay. Despite the ECG findings and the presenting symptoms, the case was not treated as AMI, since the patient related her cardiologic history perfectly. Suspecting painful LBBB, treatment started with intravenous nitrite and 40 mg of oral propranolol in order to monitor heart rate. Relief from the chest discomfort was achieved within minutes, BP was controlled and the ECG changed to a sinus rhythm without bundle branch block. At no time were ischemic ST segment changes observed. Serial enzyme determination was negative after 12 hours of observation and the patient was discharged. Discharge recommendations included initiating treatment with bisoprolol, to control hypertension and heart rate.

The appearance of rate-dependent LBBB does not necessarily imply the existence of underlying heart disease. In 1976 Wieweg et al[3] first described some patients who, during the course of a stress test, had chest pain which coincided with the appearance of LBBB. Coronary catheterization in these patients showed no heart disease, and this picture was called painful LBBB[4]. The appearance of rate-dependent LBBB is not preceded or followed by alterations of the ST segment associated with ischemic block[4-5]. Although its pathophysiology is unknown, two theories have been postulated to explain the pain in patients with normal coronary angiography. The first posits an ischemic cause and is supported by the increase of lactate in the coronary sinus, the appearance of minor alterations in the ECG and the improvement brought about by nitroglycerin. The second, more widely accepted, proposes contraction disynergy with sti-

mulation of the chest mechanoreceptors as the cause of pain[5].

Other authors have described good prognosis in patients with medium and long-term rate-dependent LBBB and normal hearts, regarding infarction and mortality[6-9]. In these patients, the development of permanent LBBB is not uncommon with disappearance of symptoms during follow-up.

Exceptionally, LBBB has evolved to advanced AV block, with the consequent need for implantation of a pacemaker. This progression to complete heart block may explain some cases of sudden death reported in patients with healthy coronary status[7-9].

Even when no underlying heart disease exists, regular monitoring of these patients is recommended because of the possibility of evolution to permanent LBBB or, exceptionally, to more advanced forms of AV block.

The interest of this case lies in our being able to observe the LBBB during an acute episode of chest pain and its disappearance after treatment was administered. We believe that the increased heart rate during an episode of pain does not allow exclusively attributing the conduction disorder to a heart rate-dependent mechanism, as the previous episodes had occurred during stress ergometry, and in this case the tachycardia did not exceed 104 beats per minute.

Reperfusion therapy should be initiated early in all patients who meet the criteria for chest pain and acute LBBB of coronary origin[1]; possible contraindications should be explored and it must be determined that the patient has less than 12 hours of evolution. The earlier the therapy is applied, the greater the benefit and the decrease in mortalidad1. Although painful LBBB is an entity that has been known for over 20 years, we should not consider the diagnosis except when the history of the patient guide us to it. Diagnostic ECG criteria of acute ischemia in LBBB patients are not easily applicable[5], because the abnormal ventricular depolarization produces a secondary alteration in the recovery process, a phenomenon that is reflected in ECG changes in repolarization in the opposite direction to the main QRS deflection or “appropriate discordance” between the QRS complex and the ST segment. ST-segment elevation in association with positive QRS complexes or depression of the ST segment in V1, V2 and V3 are not present in uncomplicated bundle branch block, called 'inappropriate discordance " and are indicative of acute ischemia[5]. Given the high mortality in myocardial infarction electrically expressed by LBBB, the rule should be to administer immediate reperfusion therapy.

References

1 Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. A report of the American College of Cardiology / American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infaction. J Am Coll Cardiol 2004;44:671-719.

2 Virtanen KS, Heikkilä J, Kala R, Siltanen P. Chest pain and rate-dependent left bundle branch block in patients with normal coronary arteriograms. Chest 1982;81:326-31.

3 Wieweg WVR, Stanton KC, Alpert JS, Hagan AD. Rate-dependent left bundle branch block with angina pectoris and normal coronary arteriograms. Chest 1976;69:123-4.

4 Vasey C, O’Donnell J, Morris S, McHenry P. Exercise-induced left bundle branch block and its relation to coronary artery disease. Am J Cardiol 1985;56:892-5.

5 Kafka H, Burggraf GW. Exercise-induced left bundle branch block and chest discomfort without myocardial ischemia. Am J Cardiol 1984;54:676-7.

6 Puppo A, Fernández E, Fournier J. Bloqueo de rama izquierda doloroso. Rev Esp Cardiol 2005;58:1130-3.

7 La Canna G, Giubbini R, Metra M, Arosio G, Curnis A, Cicogna R, et al. Assessment of myocardial perfusion with thallium-201 scintigraphy in exercise-induced left bundle branch block: diagnostic value and clinical significance. Eur Heart J 1992;13:942-6.

8 Heinsimer JA, Irwin JM, Basnight L. Influence of underlying coronary artery disease on the natural history and prognosis of exercise-induced left bundle branch block. Am J Cardiol 1987;60:1065-7.

9 Candell J, Oller G, Vega J, Ferreira I, Peña C, Castell J, et al. El bloqueo de rama izquierda inducido por el ejercicio en pacientes con y sin enfermedad coronaria. Rev Esp Cardiol 2002;55:474-80.

José Antonio FRANCO HERNÁNDEZ[1],
María del Carmen AURED GUALLAR[2],
Pedro SORRIBAS RUBIO[1],
José Gabriel GALACHE OSUNA[2]

[1]Servicio de Urgencias. [2]Servicio de Cardiología. Hospital Universitario Miguel Servet, Zaragoza, Spain.

About the medical language of Emergency Medicine specialists

Sir,

I have read with interest the work by Hernandez and Bustabad recently published in EMERGENCIAS[1]. It is certainly a formidable article: not only the authors are to be congratulated but also the Editorial Board for publishing a work that apparently has nothing to do with "our specialty." But nothing could be further from reality; our language and our use of Spanish concerns us all and in all areas of our lives, particularly the very special language that is used in medical jargon.

I do not publish regularly, nor am I a linguistic purist, but I do read; and I appreciate the lesson delivered by the authors, even if it does not flatter us.

It is true that we sometimes make mistakes in writing, concordance, punctuation and style, or even excessive use of neologisms from other languages or from other sectors similar or related to healthcare[1], for different reasons as noted by Miró in the Editorial[2]. But it is also true that there is in-

creasing effort to correct such mistakes, or at least that is how I see it. Evidence of this are the manuals on use of language, blogs on the internet, and the publications on the topic listed in the bibliography of the work by Miró or Hernández without looking further. I believe that the transmission of knowledge or experience should include the purpose of reader edification, and the language used should be respectful and elegant.

Someone once said that literary texts should be reviewed in medical faculties from time to time (I suppose in reference to the fact that, academically, we come from the sciences). To which I would add that some classic work in emergency medicine written in our tongue would not be amiss.

I would like to congratulate Miró for the self-criticism expressed in his editorial[2], a rare but laudable act nowadays. The new image of EMERGENCIAS is a marked improvement and with gestures and lessons like these, embedded in classical works of the speciality, the journal is consolidating its role as a reference text.

What I cannot come to terms with as yet is the idea of abandoning the Spanish term "urgenciólogo", especially now that the specialty of emergency medicine is closer to becoming a reality.

References

1 Hernández H y Bustabad S. Características lingüísticas de los trabajos científicos de la medicina de urgencias. Emergencias 2009;21:133-140.
2 Miró O. Escribir bien para entendernos mejor. Emergencias 2009;21:81-82.

Juan María FERNÁNDEZ NÚÑEZ
Servicio de Urgencias. Complejo Hospitalario Universitario. Badajoz, Spain.

Fecal pneumothorax: an exceptional diagnosis in the Emergency Department

Sir,

Abdominal viscera hernias due to traumatic diaphragmatic rupture are well-known clinical entities. Frequency varies according to the published series, but it is accepted that it may occur in 5% of blunt trauma[1] and in 10-15% of penetrating trauma of the thorax[2]. A unique and rare complication is tension fecal pneumothorax (TFPT) due to gas from the digestive system being released into the pleural cavity. Its identification in the emergency department (ED) is exceptional; there have only been 15 cases documented in the literature. A history of trauma, the presence of varying degrees of dyspnea, radiological findings and feculent drainage from the chest tube constitute the basic elements of diagnostic suspicion.

A 36-year-old man consulted our ED (Central Middlesex Hospital, London) for weakness, lethargy and dizziness. He reported loss of appetite and reduced food intake for several weeks, as well as mental problems and grieving due to the recent death of a relative. He had a history of depression and attempted suicide (one week before he had been discharged from the department of psychiatry with lorazepam treatment for depression). Vital constants were normal except for a breathing rate of 26 breaths / minute and heart rate of 109 bpm. Oxygen saturation was 96% in ambient air. Also, his history included asthma treated with fluticasone-salmeterol and salbutamol. No family history of interest was recorded. Physical examination showed a decrease in breathing movement on the left side of chest, moderate wheezing in the upper half of the right lung and pain on palpation of the lower left rib area at the posterior axillary line. He also exhibited visible scars on the anterior chest and left wrist. Spray salbutamol and ipratropium and analgesia with paracetamol and codeine were administered. Laboratory tests showed an infection and a thrombosis. Chest radiograph (Figure 1) showed a tension pneumothorax. Drainage by aspiration yielded 2.2 liters of fetid air. The control X-ray image is shown in Figure 1. Further drainage by aspiration of the pleural cavity yielded 1 liter of air followed by white-yellowish liquid, also foul-smelling. CT scan showed the following injuries: left lung collapse with pleural effusion, diaphragmatic defect with hernia of the stomach fundus in the left hemithorax. After chest tube insertion and administration of appropriate antibiotic coverage, the patient was referred for surgery. The patient was questioned and admitted that 10 weeks before he had stabbed himself several times in the chest, which required hospitalization for 2 weeks and thoracic drainage. After that discharge, he had flown from Ireland to London.

TFPT secondary to traumatic diaphragmatic hernia is a rare clinical entity with only 15 cases described in literature at the time of writing[1,3,4]. Delay in the diagnosis of this entity causes significantly increased morbidity and mortality in these patients. Failure to diagnose TFPT at the time of the trauma has been attributed to the nonspecific, diverse and confusing signs and results of radiological studies[5]. Non specific symptoms and signs of this clinical picture at the time of evaluation was a common feature in all these cases. The cause of the trauma (14 knife wound cases) and greater or lesser degrees of dyspnea (all 15 cases) were the most frequent features[1,3,4]. Only one case began with a picture of shock.

The slow evolution of this picture, with highly variable intervals between penetrating trauma and the appearance of TFPT (from 2 days to 10 years)[1,3] and good symptom tolerance by the patient could explain the absence of typically sudden appearance of tension pneumothorax. Furthermore, the relationship between the trauma and the subsequent pneumothorax has not always been clearly demonstrated[1,4,6,7]. Chest X-ray has been described as conclusive for the diagnosis of pneumothorax, but not for hernia of the digestive system in the chest, which requires the use of spiral CT scan or intestinal transit test[4,8]. In itself, the presence of foul-smelling air from chest drainage is highly suspicious of TFPT. Similarly, feculent serous fluid drained after the air is also highly suspicious.

After stabilization, patient management includes initial evacuation of the pneumothorax, which may be accomplished by aspiration of the pleural air, and once TFTP is diagnosed, the insertion of a pleural drainage tube, administration of appropriate antibiotics and transfer of the patient for thoracic surgery for definitive surgical treatment[1,3,4].

References

1 Vermillion JM, Wilson EB, Smith RW. Traumatic diaphragmatic hernia presenting as tension fecopneumothorax. Hernia 2001;5:158-60.
2 Reber PU, Schmied B, Seiler CA, Baer HU, Patel AG, Büchler MW. Missed diaphragmatic injuries and their long-term sequelae. J Trauma 1998;44:183-8.
3 Jarry J, Razafindratsira T, Lepront D, Pallas G, Eggenspieler P, Dastes FD. Tension faecopneumothorax as the rare presenting feature of a traumatic diaphragmatic hernia. Ann Chir 2006;131:48-50.
4 Ramdass MJ, Kamal S, Paice A, Andrews B. Traumatic diaphragmatic herniation presenting as a delayed tension faecopneumothorax. Emerg Med J 2006;23:e54.
5 Kelly J, Condon ET, Kirwan WO, Redmond HP. Post-traumatic tension faecopneumothorax in a young male: case report. World J Emerg Surg. 2008;3:20. Disponible en http://www.wjes.org/content/3/1/20.
6 Orr KB. Faeco-pneumothorax as the presenting feature of a traumatic diaphragmatic hernia. J R Soc Med 1989;82:445-6.
7 Phipps RF, Jackson BT. Faeco-pneumothorax as the presenting feature of a traumatic diaphragmatic hernia. J R Soc Med 1988;81:45-50.
8 Iochum S, Ludig T, Walter F, Sebbag H, Grosdidier G, Blum AG. Imaging of diaphragmatic injury: A diagnostic challenge? Radiographics 2002;22:S103-S118.

Juan Carlos MEDINA ÁLVAREZ,
Jesús DÍAZ-GUIJARRO HAYES
Servicio de Urgencias. Central Middlesex Hospital. Londres. Reino Unido

Prothrombin complex concentrate for the rapid reversal of oral anticoagulation in patients with severe bleeding

Sir,

Hemorrhage is the most serious side effect of oral anticoagulants, and constitutes a major cause of mortality. Rapid reversal of anticoagulation is essential for survival and for minimizing complications in these patients with severe bleeding[1]. Years ago, the treatment was based on the administration of fresh frozen plasma, a slow method with the risk of fluid overload which is not recommended in the elderly or in patients with heart disease[2].

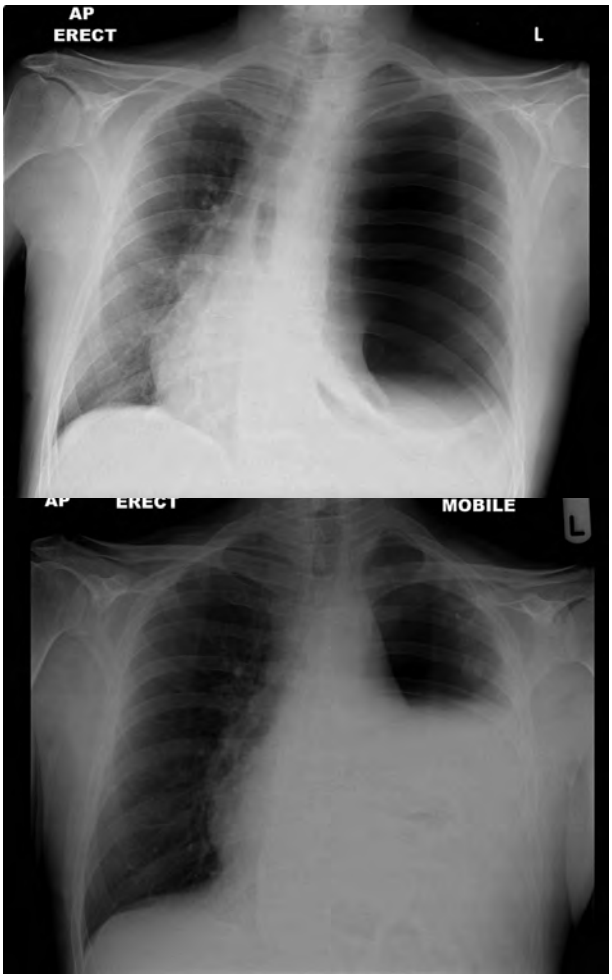

Figure 1. Chest X-ray at the time of admission (above) showing tension pneumothorax in the left lung, and (below) after resolution of the pneumothorax showing space occupation by pleural effusion.

Preparations with blood clotting factors II, VII, IX and X (Prothrombin Complex) have represented a breakthrough in reversibility and treatment of severely bleeding patients taking oral anticoagulants[3]. We believe that this type of treatment should be employed at an early stage, even prehospital, in anticoagulated patients with severe bleeding (especially upper digestive bleeding) and hemodynamic instability, before coagulation levels are available. Apart from prothrombin complex, intravenous vitamin K should be administered[4]. The recommended dose of prothrombin complex

concentrate is 500 units (one vial) initially, until laboratory test results become available. This dose is sufficient for patients with INR less than 55. The main drawback of this treatment is cost, but that is more than justified in life-threatening hemorrhage.

References

1 Lubetsky A, Hoffman R, Zimlichman R, Eldor A, Zvi J, Kostenko V, et al. Efficacy and safety of a prothrombin complex concentrate (Octaplex) for rapid reversal of oral anticoagulation. Thromb Res 2004;113:371-8.
2 Riessa HB, Meier-Hellmannb A, Motschc J, Eliasd M, Kurstene FW, Dempfle CE. Prothrombin complex concentrate (Octaplex) in patients requiring immediate reversal of oral anticoagulation. Thromb Res 2007;121:9-16.
3 Pindur G, Mörsdorf S. The use of prothrombin complex concentrates in the treatment of hemorrhages induced by oral anticoagulation. Thromb Res 1999;95:S57-61.
4 Makris M, Greaves M, Phillips WS, Kitchen S, Rosendaal FR, Preston EF. Emergency oral anticoagulant reversal: the relative efficacy of infusions of fresh frozen plasma and clotting factor concentrate on correction of the coagulopathy. Thromb Haemost 1997;77:477-80.
5 Yasakaa M, Sakatab T, Naritomia H, Minematsua K. Optimal dose of prothrombin complex concentrate for acute reversal of oral anticoagulation. Thromb Res 2005;115:455-9.

Enrique ALONSO FORMENTO,
Blanca Mar ENVID LÁZARO
Servicio de Urgencias. Hospital Obispo Polanco de Teruel, Spain.