

LETTERS TO THE EDITOR

Non-cardiogenic acute pulmonary edema secondary to tamoxifen

Sir,

The diagnosis of drug-induced non-cardiogenic acute pulmonary edema (NPE) is rare and generally made by exclusion of other causes. We present a case of NPE probably induced by tamoxifen.

The patient was a 25 year-old man without known drug allergies or toxic habits. He had engaged in bodybuilding during 4 years and self-medicated anabolic androgenic steroids during 1 year, and for the last 3 weeks had taken tamoxifen to prevent secondary gynecomastia. He consulted the emergency department for self-limiting pre-syncope with progressive dyspnea, tachypnea and cough, but no chest pain or other symptoms. On arrival he was normotensive, without fever, but presented tachycardia and tachypnea, with basal oxygen saturation of 83%. He also presented dry bibasilar rales on auscultation. Laboratory tests showed negative troponin curve, CRP 23.59 mg / L, leukocytes 17.330/ μ m and D-dimer 100 ng / ml. Other parameters, including urinary toxicological analysis, were normal. Basal arterial blood gas showed a pH of 7.41, pCO₂ of 41.3 mmHg and pO₂ of 48.9 mmHg. Electrocardiogram was normal. Chest X-ray showed bilateral patchy infiltrates with a cotton wool reticular appearance without cardiomegaly, confirmed by chest CT which ruled out pulmonary thromboembolism (Figure 1). With the diagnosis of acute respiratory failure with pulmonary infiltrates, the patient was admitted to intensive care, without requiring mechanical ventilation. Treatment began with empirical antibiotics, high dose corticosteroids and diuretics. Infectious serology, including mycobacteria, fungi, viruses and pneumocystis, and laboratory tests for vasculitis, were all negative. Bronchoscopy showed acute inflammation on biopsy. Echocardiogram showed an ejection fraction of 65% and pulmonary artery pressure of 41 mm Hg with all pulmonary cavities being normal. Within 24 hours the patient showed marked improvement with corticosteroids, since the antibiotics and diuretics were withdrawn on receiving negative laboratory results. The patient was diagnosed with NPE, despite the absence of a hemodynamic study, attributable to drugs and especially tamoxifen, as this was the last drug used before the onset of symptoms, supported by clinical evidence of rapid improvement with corticosteroid treatment.

NPE is a process with acute-onset lung damage, PaO₂/FIO₂ $\leq$ 300, bilateral alveolar infiltrates on chest X-ray and wedge pressure of 18 mmHg

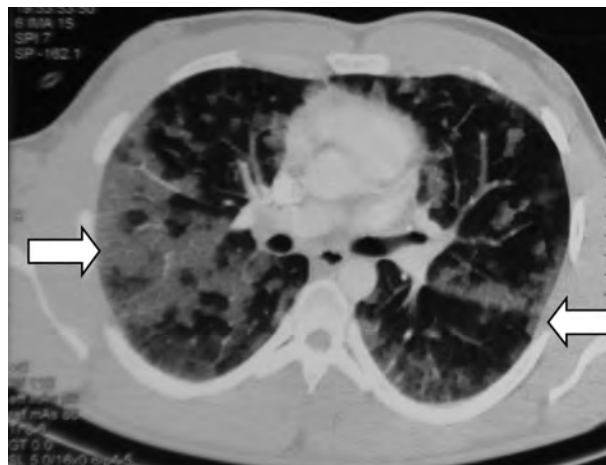


Figure 1. Chest computed tomography showing increased density in ground glass opacities, bilateral, predominantly sub-pleural and in the lower lobes, associated with septal thickening (arrows).

or higher (or no evidence of hypertension in the left atrium in the absence of this measurement)^{1,2}. Causes include drugs, especially opiates, salicylates, hydrochlorothiazide, protamine, contrast agents, tocolytics, tricyclic antidepressants and calcium antagonists³⁻⁵. The pathophysiology is probably due to an idiosyncratic non-immunological reaction^{1,6-8}. Symptoms appear within minutes to several days after administration of the drug⁶. Chest X-ray shows uni- or bilateral alveolar edema indistinguishable from other diseases but without cardiomegaly². The diagnosis must be made by exclusion, and it is necessary to rule out a cardiac, infectious, metabolic and inflammatory origin⁹. Tamoxifen (anti-estrogen) has a very low incidence of adverse side effects (<0.01%), although it is associated with an increased risk of pulmonary thromboembolism⁸ and to a lesser extent interstitial pneumonia¹⁰. In conclusion, in the history of patients with NPE it is important to consider any exposure to drugs, and include this possibility in the differential diagnosis, given the good prognosis associated with it.

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Figure 1. Facial computed tomography (detail) showing left orbit floor fracture (arrow) and hemorrhage in the left maxillary sinus.

Traumatic optic nerve avulsion

Sir,

Optic nerve avulsion (ONA) is uncommon but serious; it is usually the consequence of eye or facial concussion¹. Clinical manifestations consist of reduced visual acuity, visual field defects, afferent pupillary defect and funduscopy optic nerve abnormalities².

We present the case of a 52-year-old epileptic woman who had suffered facial trauma on falling to the ground during an epileptic seizure. After recovery, she consulted for loss of vision in the left eye. Physical examination showed blood pressure of 129/82 mm Hg, temperature 37 ° C, heart rate 76 beats per minute, left periorbital hematoma without crepitus or deformity, with no other remarkable findings. Left eye examination showed a visual acuity of 1/10, subconjunctival hemorrhage, normal intraocular pressure, pupil mydriasis and unresponsive to direct and consensual pupillary reflex stimulus, and orbital floor hemorrhage. Facial computed tomography (CT) centered on the left orbit showed a fracture of the orbital floor and of the left maxillary sinus wall with intrasinus hemorrhage (Figure 1). CT scan provided no information on the optic nerve. Suspecting ONA, high-dose corticosteroid was administered. The diagnosis was subsequently confirmed by electrophysiological study.

The mechanism whereby ONA is produced is usually anterior displacement and rotation of the eyeball due to orbital trauma (penetrating or not), ocular contusion or traumatic head injury¹. The diagnosis is mainly based on clinical findings: visual loss or afferent pupillary defect after

trauma. Visual impairment is often severe and permanent. The diagnosis must be actively sought in patients with loss of consciousness who cannot report visual alterations³. Orbital CT or MRI may locate the site of injury, but in most cases there is no evidence of alteration (especially in indirect trauma)⁴. In these cases, electrophysiology study of the optic nerve by means of an electro-retinogram or evoked potentials is essential⁵. ONA constitutes a true emergency that requires prompt intervention. Despite the absence of validation studies for any treatment, the therapeutic use of high-dose corticosteroids has been widely accepted^{6,7}.

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Unusual finding of a mediastinal mass: pericardial cyst

Sir,

Pericardial cysts are caused by a defect in the embryogenesis of the coelomic cavity. They are uncommon (accounting for 7% of all mediastinal masses, and incidence in the general population is 1: 100,000) and unrelated to age or gender.

The patient was a 56 year-old woman, smoker, with hypertension and chronic bronchitis, who for some months had suffered oppressive chest pain, radiating to the back, lasting several minutes on moderate exertion but subsiding with rest. She was referred to the emergency department for evaluation of probable effort angina. Among the tests performed, chest X-ray showed an oval image in the right cardiophrenic region. Thoracic computed tomography (CT) and subsequent magnetic resonance imaging (MRI) confirmed the presence of a pericardial cyst (Figure 1). This was considered an incidental finding unrelated to the chief complaint. In the absence of clinical symptoms, a conservative approach has been adopted to date.

The most common site of pericardial cysts is the right cardiophrenic angle (70%). They may also appear in the left cardiophrenic angle (20%) and other more unusual locations related to pericardial recesses (10%). Most are silent (60%) but when symptoms are present, they are related with compression (dyspnea, chest pain, cough, arrhythmia), or with a complication (infection, hemoptysis, hemorrhage). The diagnosis is suggested by conventional X-ray findings and confirmed by CT or MRI. There are several treatment options, but surgical excision is recommended for cysts that are symptomatic, very large or in unusual locations.

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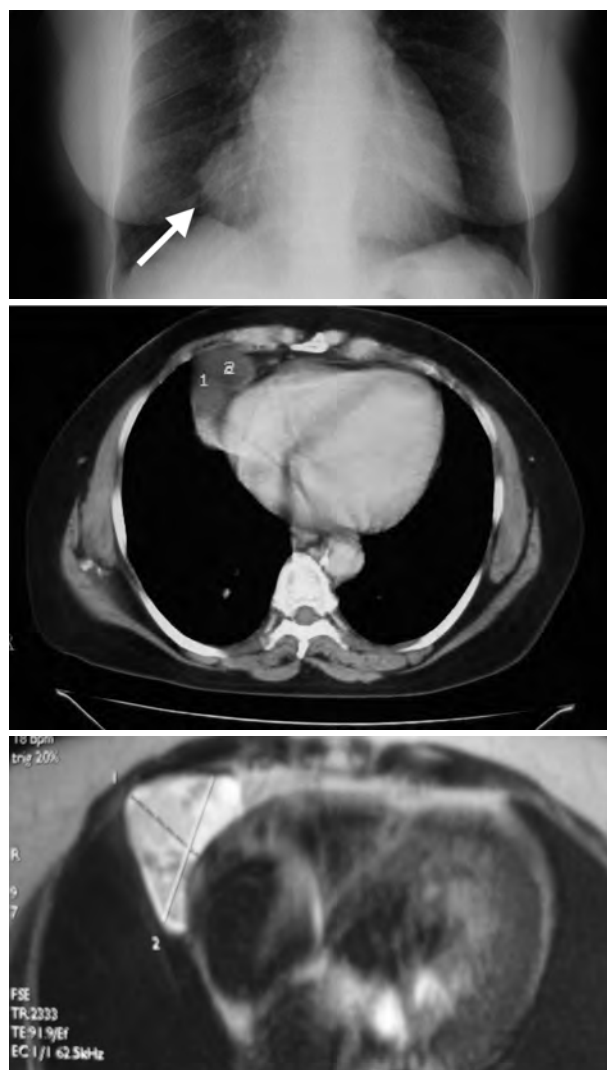


Figure 1. Anteroposterior chest X-ray (top), showing a homogeneous oval image of water density at the right cardiophrenic angle (arrow). Chest CT (middle) showing an elliptical mass of water density. Cardiac MRI (bellow) showing a discrete lobulated cystic mass measuring 33 x 52 x 30 mm, hypointense in weighted sequences at T1 and hyperintense at T2, without contrast capture, with a broad base of contact with the right atrioventricular parietal pericardium which does not produce mechanical compression of the heart.

Acute generalized exanthematous pustulosis induced by clindamycin

Sir,

Dermatological clinical manifestations are a common form of adverse drug reactions. We present a case of acute generalized exanthematous pustulosis (AGEP) due to clindamycin.

A 58 year-old woman, with no history of allergic reaction to drugs or other agents, consulted for ery-

thematous lesions on the trunk, limbs and face, accompanied by itching and burning sensations. A few days before she had an odontogenic cyst removed and had been prescribed metamizol, ibuprofen and clindamycin. On the sixth day of treatment an erythematous rash appeared on the face, neck and upper chest, which did not improve with antihistamine treatment. On arrival at the emergency department (ED), the patient had fever (38.5 ° C) and physical examination showed multiple pustules 1-4mm in diameter on an erythematous base, with facial and eyelid edema (Figure 1). Distribution was symmetrical on the nose, chin, preauricular regions, neck, back and thighs. She presented positive Nikolsky sign and also some laterocervical adenopathies. There was no palmoplantar or mucous involvement, and the rest of the examination was normal. Additional ED tests showed 19,600 leukocytes/ul (86% neutrophils), and normal blood count and biochemical parameters. The patient was admitted for treatment with intravenous prednisone 60 mg/day and topical antiseptics of denuded areas. Antistreptolysin titers were normal. Blood, urine and skin cultures for common germs were negative, as were direct immunofluorescence for herpes zoster virus and serology for cytomegalovirus, Epstein-Barr virus and hepatitis. Skin biopsy showed intraepidermal pustules with polymorphonuclear infiltrate and erythrocytes, together with monocytes and leucocytoclastic vasculitis. Given the clinical picture, history of drug intake and histopathology, AGEF was diagnosed. Outcome was favorable; at 15 days the lesions had almost completely disappeared, and the skin was intact.

This rash is rare in our setting, with an incidence of 1-5 cases per million inhabitants per year¹. Presentation is acute, characterized by the appearance of sterile pustules on erythematous edematous skin, often accompanied by fever, peripheral leukocytosis and sometimes facial edema². The confluence of pustules may result in a positive Nikolsky sign that can lead us to erroneous diagnosis of toxic epidermal necrolysis³. Some cases have been attributed to infection by Mycoplasma or viruses, but in most patients AGEF is an uncommon type of drug-induced hypersensitivity⁴⁻⁹. Evolution after exposure is acute (1-5 days), usually self-limiting and benign⁷. The underlying causative mechanisms are unclear, although recent research suggests an immunological process^{9,10}.

The patient had previously taken metamizol and ibuprofen for other problems of an inflammatory nature without any complications. We therefore believe that the cause of the AGEF was clindamycin. As mentioned, the diagnosis of this entity, besides the temporal relationship with the presumed agent involved,



Figure 1. Photograph showing facial, neck and upper chest involvement.

is based on clinical evolution, histopathology, and exclusion of other causes of dermatitis. And although reproducing the rash would be feasible by re-exposing the patient to the drug, ethical factors disallow this possibility, and we are limited to inconclusive in vitro techniques¹⁰.

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Fluctuating decreased level of consciousness in a patient with atrial fibrillation

Sir,

An 80-year-old woman with a history of chronic atrial fibrillation and cardioembolic stroke fifteen years before consulted for slight residual right hemiparesis. She was being treated with digoxin, amiodarone and acenocoumarol. During the previous two weeks she had experienced unstable gait, and three days before she also had slurred speech and facial asymmetry. On physical examination she was tachycardic (140 bpm), while other vital constants were normal. Cardiac auscultation showed arrhythmia, and lung auscultation showed vesicular murmur. Neurological examination showed decreased level of consciousness, intermittent dysarthria and left mouth line deviation, all three signs of changing intensity during the examination. Chest X-ray showed cardiomegalia. Laboratory tests showed urea 89 mg/dl and creatinine 1.52 mg/dL. Coagulation test showed INR of 1.84. Computed tomography (CT) scan was normal. Electrocardiography showed atrial fibrillation with frequent bursts of wide QRS complex tachycardia and the morphology of left bundle branch block (LBBB) (Figure 1). The patient was monitored by ECG, which showed that decreased level of consciousness coincided with the more prolonged episodes of tachycardia and aberrant conduction of basal atrial fibrillation.

Frequency-dependent branch block or Ashman's phenomenon (AP) is a transient ventricular conduction disorder dependent on heart rate change. As the time required for repolarization of the conduction system depends on the length of the cardiac cycle that precedes it, short cycles with fast heart rate require short repolarization time, and long cycles with slow heart rate require longer repolarization time. Generally, the right branch of the His bundle is the last part of the infranodal system to be repolarized. Thus, after a long cycle, any subsequent atrial extrasystole of a shorter cycle can be conducted aberrantly, generally with right bundle branch block (RBBB). This is what is

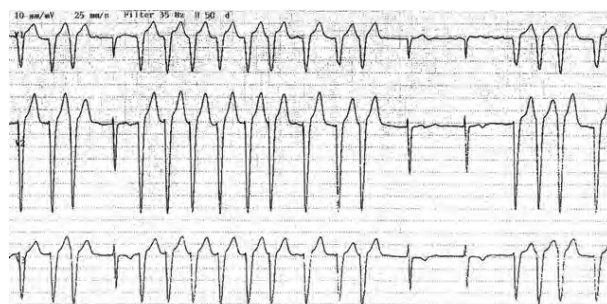


Figure 1. Symptomatic Ashman phenomenon over atrial fibrillation. Series of supraventricular extrasystoles with left bundle branch block morphology.

called long cycle-short beat, which is the most common, although the opposite short-cycle-long beat has also been documented¹. In addition to heart rate, the refractory periods are also affected by factors influencing the ionic environment, drugs and pathological states such as ischemia or hypoxia.

Although AP may present in any atrial arrhythmia, it is typically seen in atrial fibrillation, characterized by sequences of long and short cycles. As isolated QRS complexes and RBBB morphology, AP is a relatively common asymptomatic electrocardiographic finding in clinical practice². More rarely, QRS complexes adopt an LBBB morphology, as in the case presented here. Also, unlike the more frequent isolated QRS complex, our patient had a variable number of QRS complex series - between 2 and 9 (Figure 1). Finally, compared to the usual asymptomatic presentation, AP in our patient was accompanied by symptoms.

Fisch criteria for the diagnosis of AP are as follows³: a relatively long aberrant cycle preceding the QRS complex (a short-long-short cycle is even more likely to initiate aberrant conduction, which may be LBBB or RBBB, and even in the same patient), aberrant conduction with RBBB morphology and normal orientation of the initial QRS vector (hidden perpetuating aberrant conduction is possible, so there may be a series of supraventricular beats of the wide QRS complex); irregular coupling of aberrant QRS complexes; and absence of a complete compensatory pause.

Differential diagnosis should consider premature ventricular complexes, intermittent ventricular pre-excitation as in Wolff-Parkinson-White syndrome, and ventricular tachycardia, which have different prognoses and treatment. In this regard, several studies have shown AP to have

low sensitivity and specificity in the differential diagnosis between aberrant conduction and ventricular rate.

Per se, AP does not produce symptoms. When symptoms are present, they are due to the tachycardia and the possible underlying heart disease. In our patient, transthoracic echocardiography performed five months earlier had shown moderate depression of systolic function influenced by the heart rate, which was 45% for frequencies around 130 bpm and above 50% after longer diastoles. In the emergency department, her decreased level of consciousness coincided with higher frequencies. Similarly, we interpreted the changing dysarthria and mouth line deviation as a result of brain hypoperfusion secondary to low intermittent cardiac output associated with tachycardia, which coincided with aberrant conduction. AP does not require treatment when expressed as isolated complexes; when symptomatic, beta-blockers can be used.

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Smart-drugs: a new clinical challenge in Emergency Medicine

Sir,

The recently published Annual Report of the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) 2010¹ warns of the increased use of smart drugs or legal highs, especially in young people, confirming trends already observed in previous reports, in monographs by the same body² and by research groups such as the Psychonaut Web Mapping Project³.

Smart drugs are a group of substances of different nature and origin, which include products such as ancestral peyote or psilocybin fungi, plants such as *catha edulis* (khat) or *salvia divinorum*, drugs for veterinary use such as piperazines (anthelmintic) and clandestinely produced cannabis analogues (HU-210, JWH-018, CP 47,497) or synthetic cathinone (mephedrone, methylone, methedrone and MDPV)^{1,3}.

As these products have a similar chemical structure to classical synthetic drugs (ecstasy, GHB, LSD, etc.), they are used for recreational purposes (hallucinogenic, euphoric, relaxing, aphrodisiac, increased psychomotor performance, decreased fatigue and sleep, etc), invigorating (weight loss or gain, fitness, etc.) or to combat the unwanted effects of other drugs. They can be purchased through internet from smart shops (the EMCDDA registered 170 online stores in 2010, up from 39 in 2006) for prices that are quite low and, above all, legally, since their distribution and sale is not legally penalized in most cases¹. Some of the products on sale, such as piperazines (BZP), have been banned in Spain as from July 2009 after several attributable deaths, following a European Union directive adopted in 2008⁴. In others, such as synthetic cannabis or "spice"⁵, ease of component change means that, as certain synthetic cannabinoids are being banned, others are incorporated before being subject to regulation. Special mention should be made of mephedrone, known as cat or meow meow. This synthetic analogue of cathinone has rapidly become the most popular legal alternative to ecstasy and cocaine, consumed heavily in England and probably in Spain⁶. In any case, the line between legal and illegal drugs has become very tenuous. Naturally, most of these synthetic products have been introduced on the market without any support from clinical studies on their pharmacology and toxicity, not even in animal models, and the only sources of pseudo-clinical information are user forums on the internet.

The clinical challenge for those working in emergency services and departments is that these drugs are not detectable by qualitative or semi-quantitative tests used in such departments and laboratories of most hospitals⁷, nor even by most toxicology laboratories using chromatographic and spectrometric techniques⁸ due to the absence of reference patterns for identification⁹. Thus, diagnosis currently depends only on clinical suspicion based on knowledge and up-

dated information about this group of drugs and toxidromes¹⁰; and incidentally avoid costly and unnecessary complementary tests or clinical assessments¹¹. At discharge, preventive measures at the family and individual level must be adopted.

Another similar toxicological phenomenon faced by clinicians is that known as pharming (recreational pharmacological drug use), with doses that differ from therapeutic use, generally seeking adverse effects. In USA, this group of drugs accounts for 29% of recreational substance abuse by adolescents¹², mainly involving dextromethorphan. Similar results have been obtained in Spain through the Observatory of Medication Abuse (OMA), sponsored by the College of Pharmacists of Barcelona. In addition to the classical drugs (benzodiazepines, opiates, ketamine, etc), products such as propofol, benzylamine, pregabalin, methylphenidate or modafinil are emerging as substances of abuse^{1,3}.

In conclusion, clinical toxicology should form part of the training of future specialists in accident and emergency medicine¹³. Until this happens, continuous updates, awareness of the limitations of laboratory tests and clinical suspicion are our only weapons to monitor the changing world of substance abuse which has burst on the market, making these drugs highly accessible, globalized, sophisticated and very attractive to young people.

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Pain and Guillain-Barré syndrome

Sir,

Currently, Guillain-Barré syndrome (GBS) comprises multiple subtypes of inflammatory demyelinating polyneuropathy: acute inflammatory demyelinating neuropathy, acute motor axonal neuropathy, acute sensory-motor axonal neuropathy and Miller-Fisher syndrome. The diagnosis is based on criteria that with the passage of time have proved their utility (Table 1)¹. However, there are other non-cardinal symptoms, which may be the first to manifest, whose recognition is important to avoid delayed diagnosis and therapeutic measures that are erroneous or counterproductive. One of these symptoms is pain. We would like to share our experience in handling a case of GBS, with an initial manifestation of intense back pain.

A 40 year-old woman attended the emergency department for severe back pain and tingling sensations in the hands and feet some 24 hours earlier. On physical examination she complained of selective pain between D6 and D12, with no other early clinical signs. Hemogram, biochemistry, ECG, chest X-ray and chest computed tomography (CT) were normal, while dorsal CT showed a dorsal medial hernia at D8-D9. She received analgesics according to WHO scale, and required infusion pump morphine for the pain. At 12 hours after the first evaluation in the emergency department, symmetrical achilles and patellar hyporeflexia was detected. She underwent a lumbar puncture and albumin-cytological dissociation was found. A diagnosis of GBS was established. Two days later the patient suffered respira-

Table 1. Diagnostic criteria for Guillain-Barre syndrome**Necessary for the diagnosis**

- Progressive weakness of the limbs
- Areflexia

Support the diagnosis

- Progression up to 4 weeks
- Relative symmetry of signs
- Mild sensory symptoms or signs
- Involvement of cranial nerves
- Gradual recovery after the end of the progression
- Autonomic dysfunction
- Absence of fever at onset

Laboratory studies that support the diagnosis

- Increased proteins in CSF with less than 10 cells
- Neurophysiological studies

tory arrest, requiring admission to the hospital intensive care unit (ICU) for quadriplegia during 56 days, 40 of which with endotracheal intubation and mechanical ventilation. She was treated with intravenous immunoglobulin. Recovery was progressive from day 38 of admission to the ICU.

The description of pain as a symptom of GBS cases dates back to initial case reports by Landry² and Guillain-Barré-Strohl³. Patients can discriminate several types of sensory impairment and describe numbness, paresthesia and dysesthesia. The pain can be sufficiently intense as to elicit spontaneous description. It may precede the onset of motor symptoms or accompany them from the start. The location is usually precise and commonly involves the flexors of the thighs, buttocks and dorsal muscles; less commonly, the lumbar or dorsal regions. Pain is more often experienced in the evening and worsens with both active and passive mobilization. It is often described as intense stiffness⁴. The frequency of pain in GBS is variable because, in most series pain data mainly refer to back pain only, which tends to underestimate the actual incidence. The highest rates range from 72%⁴ to 85%⁵. Treatment is based on the use of non-steroid anti-inflammatory drugs and, in resistant cases, opioids. The differential diagnosis when a motor process is involved without sensory impairment includes poliomyelitis, porphyria, polymyositis, hypokalemia, botulism and myasthenia gravis⁶. In our case, as the pain presented as the initial symptom, and due to its intensity and location, we initially considered myelitis or spinal cord compression by a tumor or bleeding and ruled out aortic dissection. Misdiagnosis of poliomyelitis would have led to the use of corticosteroids that are contraindicated in GBS. In our case, the main guiding symptom was areflexia detected on re-examination at 12 hours in the emergency department. From our experience, we would emphasize the need for evolutionary moni-

toring control for such a common symptom as back pain in the emergency department, since discrepancy between the intensity of pain and examination findings might suggest a functional or a banal process.

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Upper limb positional paresthesia in a young woman

Sir,

Thoracic outlet syndrome (TOS) includes different clinical conditions due to compression of neurovascular elements that pass from the chest to the arm. TOS produced by a cervical rib is a congenital anomaly present in 1% of the population, predominantly in young women. It is originated by a supernumerary rib, which reduces the space of the interscalene triangle, compressing the subclavian artery and brachial plexus.

A 37 year-old woman with a history of depressive anxiety disorder complained of difficulty mobilizing the right arm after getting out of bed and numbness of the right chest and arm to the hand. She reported no history of trauma or overexertion. She had no headache or other sensory alterations. Physical examination showed a painful bilateral paravertebral contracture aggravated by cervical mobility, and especially hyperextension. Cardiovascular and pulmonary examination was strictly normal. Neurological examination showed a loss of sensation in the ulnar nerve region of the right arm. Chest and neck X-ray showed an appendix at the transverse process of the seventh cervical vertebra and a marked correction of physiological cervical lordosis (Figure 1). With a tentative diagnosis of cervicobrachialgia secondary to cervical rib, physical measures were



Figure 1. Anteroposterior (left) and lateral (right) X-rays of the cervical spine, showing images compatible with bilateral supernumerary cervical rib (arrows) and loss of physiologic lordosis.

recommended (local heat, avoidance of effort), analgesics and muscle relaxant; she was referred to outpatient traumatology. She currently receives medical and rehabilitation treatment, and shows good symptomatic control.

The clinical manifestation of cervical rib is due to neurological disturbances secondary to compression of the medial cord of the brachial plexus, affecting the cubital, ulnar, pectoral and medial brachial cutaneous nerves. There is pain and dysesthesia of the supraclavicular region radiating to the inner arm and hand, with anterior chest involvement. It may also cause vascular manifestations such as distal coldness, paleness, Raynaud's phenomenon and weak radial and ulnar pulse. The diagnosis is clinical, by provoking symptoms with maneuvers and hyperextension of the neck, and radiological, with X-ray evidence of a super-

numerary rib. It is advisable to perform differential diagnosis with neurological diseases (amyotrophic lateral sclerosis, multiple sclerosis, spinal tumor, syringomyelia, etc.), trauma (cervical osteoarthritis, cervical spondylosis, myofasciitis trapezius, shoulder bursitis), rheumatic disease (Raynaud's, arteritis), and oncologic disease (Pancoast tumor).

The treatment of choice is medical, with B12, B6, B2, vasodilators, analgesics and muscle relaxants, and physical therapy to strengthen scapular waist muscles and improve improper postures. Surgical treatment is recommended when complications appear (severe vascular compromise, muscle atrophy) or when medical treatment fails.

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