LETTERS TO THE EDITOR

Malignant tumor of the autonomic plexus, a rare cause of pain in the right iliac fossa

Sir,

Abdominal pain is one of the most frequent symptoms in emergency department (ED) patients[1]. We describe a patient with abdominal pain and gastrointestinal tumor of the autonomic plexuses. These are rare neoplasms whose incidence in Spain[2] is 457 new cases per year[3]. They appears as from the age of 50 years, with no gender predominance. The most common location is in the stomach (55%)[3-6]. The most reliable criterion of malignancy is the presence of metastasis at diagnosis. The most common symptom is abdominal pain that appears in advanced stages[3,5]. The diagnosis[2,3,5] is complicated. Treatment[2-5] depends on the extent at the time of diagnosis: with early detection, surgery may be sufficient, and with metastatic disease, imatibit mesylate is the first line of treatment.

A 46 year-old male with a history of rectal bleeding for minute polyps of the ileocecal valve with nonspecific inflammatory changes visited the ED for abdominal pain during 15 days. He reported good general health and right abdominal pain. Additional tests were normal. He was discharged then readmitted for persistent pain and fever, with very painful abdomen and signs of peritoneal irritation. Laboratory tests were normal. Abdominal CT showed inflammatory plastron of the cecum and ascending colon up to the hepatic flexure, with probable involvement of the loops of ileum, involvement of right lateroconal fat and inflammatory lymph nodes. He was hospitalized with a diagnosis of inflammatory plastron of probable appendicular origin versus inflammatory bowel disease. With conservative treatment the patient responded favorably. Colonoscopy showed an obstruction as from 30 cm of the sigma. He began steroid treatment for suspected inflammatory bowel disease. Deterioration continued, treatment was discontinued and a new CT scan with punch biopsy of the mass showed torpid evolution with increased mass and the lymph nodes, new liver lesions, ascites and peritoneal implants. Histopathology of tissue corresponded to a malignant tumor of the autonomic plexus. Imatibit chemotherapy was started but the patient's general condition continued to deteriorate and he died soon after.

The patient's main symptom was abdominal pain which required multiple differential diagnosis with malignant tumors, benign tumors, Crohn's disease, foreign bodies, and infectious ileitis diverticulitis[1,6,7]. In our case, antibiotic treatment was begun considering the infectious origin, and given the patient's evolution, steroids were added for suspected Crohn's disease. No surgical treatment was given because of the size of the mass, and chemotherapy proved ineffective due to the advanced stage of disease and the patient's declining general condition.

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Disasters and carbon monoxide poisoning

Sir,

Recent cases of poisoning by smoke and carbon monoxide (CO) reported in Catalonia after a snowfall that affected part of our territory (1 person died, 6 hospital admissions and more than 100 were poisoned)[1] should make us reflect on the importance of adopting certain measures to prevent such accidents (information to the public by the authorities about precautions in the use of portable electric generators when their use is expected to increase), as well as the role of disaster-related epidemiology[2]. The United Nations International Strategy for Disaster Reduction (ISDR) has defined a disaster as a severe disruption of the functioning of a community or society causing extensive loss of life, property damage, economic or environmental losses which exceed the capacity of the affected community or society to manage using their own resources[3]. Although the magnitude of the disaster reported means it did not strictly fall within the scope of this definition, and CO poisoning in our setting only accounts for approximately 4-5% of all serious poisoning[4], the nature of the episode reminds us that the epidemiology of disasters has previously described the relationship between disasters and CO poisoning[5,6]. The impact of disasters is not limited to effects on the health of the affected population, but extends to disruption of basic services (energy, communications, sanitation, etc.), which often have greater social impact. The relationship between a disaster and CO poisoning is determined by the fact that, quite often, the power supply is interrupted and people seek alternative sources of energy. The main alternative are portable electric generators, which require a basic knowledge of safety to avoid poisoning[7]. After Hurricane Katrina in the United States in 2005, health facilities with hyperbaric chambers treated 51 cases of CO poisoning[6]. The study authors acknowledge that the real numbers are likely to be well above this figure of 51 cases because only 6% of patients presenting to emergency departments with CO poisoning require hyperbaric treatment[8]. All cases except one were due to the use of portable generators, specifically misuse or use in poorly ventilated locations. As the

epidemiology of disasters had already shown the relationship between the disaster and CO poisoning, the Centers for Disease Control and Prevention (CDC) in Atlanta proposed a preventive strategy for these cases by producing a guide for users and precautions to be taken with portable electric generators in cases of emergency or disasters[9]. This initiative of the CDC provides the necessary bridge between epidemiology and disaster medicine in emergency care. And this step has not yet been taken in our country, probably because there are no accessible and reliable data on the magnitude of the problem (in the UK, for example, CO poisoning is the most common cause of fatal poisoning each year and kills more than 50 people 10). It would therefore be particularly useful, for research purposes, to make available and publish the data and analysis of CO poisoning episodes in normal situations and after an emergency or disaster.

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Hypothermia during transport of an infant with hypoxic-ischemic encephalopathy

Sir,

Perinatal hypoxic-ischemic encephalopathy in Spain affects between 500 and 1,500 newborns a year. Supportive care has long been the only option, in the absence of any therapy that improves neurological outcome. The advent of moderate hypothermia in clinical practice as a mechanism of neuroprotection is now a reality[1]. Moderate hypothermia is the reduction of brain temperature by 3-4 ºC by means of whole-body cooling or selective brain cooling started within 6 hours of life and maintained for 72 hours. Four meta-analyses[2-5] and follow-up results after two years of major multicenter studies have shown significant improvement in the neurological prognosis of these children[6,7]. Moderate hypothermia used in the safe and controlled setting of a tertiary hospital performed by a trained multidisciplinary team means that for the first time in many years we now have a tool to reduce neurological injury in hypoxic-ischemic encephalopathy. The importance of starting treatment before 6 hours of life and the need to centralize the care of these patients makes cooling during transportation crucial.

In Catalonia, we have formed a working group to establish a program of hypothermia which takes into account the particularities of our region, including the distance between hospitals, the existence of an exclusively pediatric transport system and the fact that there are hospitals prepared to administer centralized hypothermia in Barcelona. A detailed review of the literature, years of experience of neonatal transport and 24 months of applying hypothermia during transportation has led us to certain perceptions on the basic management of these cases. A stable non-oscillating temperature is essential, as increased temperature has been associated with seizures and hemodynamic instability[8,9]. Infants who suffer hypoxic-ischemic insult tend to reduce their temperature. Thus, the first few hours are the most difficult to maintain temperature within the therapeutic range. The risk of over-cooling and its possible influence on the neuroprotective effect of hypothermia requires very careful temperature control[9]. In most multicenter studies, acceptable management of temperature during transport was achieved with the use of bags of cold water and the incubator's own heating system[10]. We observed variability in temperature according to the different vehicles used (ambulances and helicopters) and season. There are many examples of over-cooling in the literature when attempting to maintain a temperature of 33-34ºC without the use of automatic mechanisms of hypothermia[8-11]. The use of automated equipment to manage hypothermia during transport is currently under development; when this is not available, in our opinion, the range of safe and effective temperature during transport is 35°C ± 0.5. With a temperature of 35.5°C, cold water bags can be placed around the body, avoiding direct skin contact, to create a cold environment. Ice in direct contact with the skin should be avoided. If the temperature falls below 34.5ºC, incubator heat can be applied. Temperature control of infants with hypoxic ischemic encephalopathy in the first hours of life is very important to ensure effective neuroprotective effect. Many of these children come from other centers and must be transferred to a tertiary center capable of managing hypothermia with appropriate multidisciplinary care. The management of hypothermia presents most difficulty and complications during transport. Continuous rectal temperature probes and the use of a conservative target temperature help prevent overcooling.

Annex

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Exercise-induced rhabdomyolysis without associated acute kidney injury

Sir,

Rhabdomyolysis is a rare syndrome that affects skeletal muscle and can lead to systemic manifestations. It appears in a variety of situations such as enzyme deficiencies, muscular dystrophy, immune disorders, excessive muscle activity, direct muscle injury, acute ischemia, infection, impaired thermoregulation, drugs, toxic and metabolic or idiopathic alterations. It is characterized by muscle cell necrosis and release of contents into the bloodstream. Myoglobin is nephrotoxic, damaging the tubular epithelium and causing acute renal failure (ARF) in 30% of patients with rhabdomyolysis. Among the infections that cause ARF by rhabdomyolysis is Legionnaire's disease[1]. Correct diagnosis is important for appropriate therapy to prevent associated complications such as ARF, electrolyte alterations, disseminated intravascular coagulation and hepatic and lung dysfunction[2].

A 32 year-old woman who regularly performed moderate exercise (45 minutes spinning) visited the ED. Twenty-four hours after a spinning session she began to feel generalized muscle pain with greater intensity in both quadriceps on urinating dark urine. Physical examination showed tenderness to palpation of both thighs. Temperature was 36ºC, heart rate 80 beats/minute and blood pressure 130/70 mmHg. The rest of the examination was normal. Laboratory tests showed creatine kinase (C K) 100,928 U/l, myoglobin 3,000 ng/l, creatinine 0.7 mg/dl and the remaining parameters were within normal limits. Urine sediment showed more than 300 erythrocytes per field and proteinuria was > 100 mg/dl. The patient was admitted to the observation unit with suspected rhabdomyolysis-induced by exercise. Treatment was initiated with intense fluid therapy, urinary alkalinization with bicarbonate and osmotic diuresis with intravenous mannitol. The patient remained under observation during 30 hours and showed hemodynamic stability and excellent diuretic response. Laboratory tests revealed no renal failure at any time.At discharge she had urea 17 mg/dl, creatinine 0.6 mg/dl, sodium 141 mEq/l, potassium 3.6 mEq/l, total bilirubin 0.2 mg/dl, AST 1426 U/L, GPT 543 U/L, GGT 10 U/L, alkaline phosphatase 51 U/l, CK 30,334 U/L and myoglobin 776 U/l. One month later CK was 92 U/L.

Rhabdomyolysis is a potentially serious syndrome that must be recognized and treated immediately. It was first described in patients wounded during the second World War. Incidence varies widely, but it seems greater in men than in women. Its causes are classified into four categories: trauma or direct injury, excessive muscle activity (as in our case), hereditary enzyme deficiencies and other medical causes (drugs such as statins, cyclosporin A, risperidone, antithyroid agents and terfenadine, toxins, viral and bacterial infections, temperature changes and myopathy)[3-6]. The most important complication is acute renal failure, with an incidence of 10-30%[7,8]. Rhabdomyolysis is produced by an increase in the production of adenosine triphosphate, resulting in an increase of intracellular calcium, causing cell destruction with release of creatine and myoglobin into the bloodstream[2]. In exercise-induced rhabdomyolysis the patient presents myalgia, weakness, sweating, and decreased pain threshold. Spinning is one of the most popular exercises practiced in gyms. It consists of a static bicycle program in a motivational class, of forty minutes duration, in which variable resistance is applied to simulate real cycling[2]. The patient reported a history of intense physical exercise with a slow debut of symptoms. Only two cases have been reported in the literature of rhabdomyolysis induced by spinning. Another important fact is the appearance of dark colored urine, brown to red in 50% of cases. The most useful laboratory test is serum CK elevated two or three times above normal levels, with a peak at 24 hours, and a 40% decrease per day thereafter[2]. Some authors have argued that the higher the level of serum CK, the greater the risk of developing acute renal failure[7,10]. Coinciding with the events described, our patient presented myalgia and dark urine 24 hours after exercise. CK in our case rose disproportionately but she did not develop kidney failure.

Diagnosis is based on clinical history and laboratory tests, based primarily on blood CK values. Our patient did not undergo the orthotoluidine test for myoglobinuria, but there was clear evidence of rhabdomyolysis due to the high elevation of blood CK and myoglobin values. In patients who have performed intense physical activity, we must suspect this disease once other processes have been ruled out[2].

The main goal of treatment is to prevent acute renal failure and this includes rapid and aggressive fluid replacement, urine alkalinization with bicarbonate, a urine output of 200-300 ml/hr, management of hyperkalemia and a decline in blood CK levels shown by repeated measurements. The possible appearance of hypocalcemia and metabolic acidosis should be monitored.

If there is evidence of renal failure and no response to initial measures,

hemodialysis should be considered. Lastly, the inflammatory process should be monitored and physical activity must be limited to avoid further destruction of muscle tissue[2,7,9].

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Blood transfusion and the increase in hemoglobin concetration: a marriage of convenience?

Sir,

Anemia is highly prevalent in all emergency departments, and management involves a multidisciplinary approach supported by an armamentarium of therapeutic measures, optimizing available resources [allogeneic blood transfusion (ABT), parenteral iron, oral iron therapy, erythropoiesis-stimulating agents] depending on the particularities of each patient[1-3]. Anemia is an extremely common comorbid condition in elderly patients (17-63% prevalence in people aged over 65 years), cancer patients (14-77%) and heart failure patients (10-50%), related with iron-deficiency, chronic disease, hemolytic disease, of myelosuppressive, multifactorial or unknown etiology, inter alia[4,5]. ABT is a quick and effective means of correcting anemia, despite potential complications and limited availability[1,3]. The most common complications of ABT are the transmission of infectious diseases, allergic or anaphylactic reactions, cardiopulmonary or thromboembolic complications, prolonged hospital stay, increased hospital mortality and neoplastic recurrence[1,2,6]. Among non-infectious mechanisms are acute lung injury (TRALI effect, incidence 1:2000-1:8000 transfusions), immunosuppressive effect (TRIM effect, associated with various infectious and the recurrence of neoplasm) and circulatory overload (TACO effect)[6]. The ultimate goal of ABT is to rapidly increase oxygen delivery to tissues and prevent and/or correct the effects of hypoxia[1,3]. In the absence of more reliable physiological indicators of oxygen supply and consumption at the cell and tissue level, hemoglobin (Hb) and percentage of hematocrit are two of the main biological parameters for estimating transfusion requirements[1,3]. Based on indirect parameters, we estimate the transfusion trigger threshold in normovolemic patients without cardiovascular disease at around 7 g/dl Hb[1,3,7]. Classically, each unit of packed red blood cells (PRBC) is considered to increase Hb levels by 1 g/dl and hematocrit by three points (3%)[3,8]. Paradoxically, few studies in humans have been performed to support or validate this claim, especially considering the considerable variability in volume (200-350 ml) and hematocrit (55-80%) of each PRBC and other factors, such as the progressive reduction of the viability of stored red cells and then transfused, the possibility of loss or destruction of red blood cells, the simultaneous administration of fluids, and so on[1,3,9]. Several recent studies suggest there is systematic underestimation of transfusion requirements in applying the classical premise of "an increase of 1 g/dl in Hb for each concentrate transfused" (with hematocrit increments that vary widely from 1.5 to 9% after transfusion of a PRBC), with the consequent risk of insufficient blood transfusion and thus "sub-optimal" Hb levels. Some authors recommend a more individualized estimate of transfusion requirements (depending on the value of hemoglobin, weight, sex and even the hematocrit value of packed red cells) in order to maximize the clinical benefits of ABT and more accurately approximate the target Hb value established by the physician[1,3,9,10]. Among other aspects, the review by Liumbruno et al. of clinical recommendations for ABT analyzed the increase in Hb after transfusion of a single pack of red blood cells according to patient weight and sex, and found a range of increases in Hb that ranged from 0.5 to 2.5 g/dl for each pack transfused[1]. Davies et al. proposed a mathematical formula to calculate the need for transfusion depending on the weight of the patient, the desired increase in Hb and the hematocrit level of the red blood cells[10]. Elzik et al. found an increase in hematocrit of 1.9 ± 1.2% per 300 ml (considered the standard volume) of packed red blood cells transfused, and estimated that 500 ml of RBC (almost double that of the standard blood volume) were necessary to achieve the classic '3% increase in hematocrit"[9]. Clinical evidence continues to make inroads into the field of transfusion medicine. The strict 10/30 rule to automatically start ABT, the post-transfusion interval of 4-48 hours for analytical testing of its effectiveness ... all these myths have fallen by the wayside[8,10].

Is it now time to rethink the old assumption that a single pack of RBC increases Hb levels by 1 g/dl and hematocrit by three per cent?

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Genital necrotizing fasciitis

Sir,

Genital necrotizing fasciitis, also known as Fournier's gangrene[1] or streptococcal gangrene of the scrotum, is characterized by abrupt onset of necrotizing infection of the soft perirectal tissues and genital region with rapid evolution, involving the surrounding tissues and lower abdominal floor.

We report the case of an 82 year-old man with diabetes mellitus type 2, multiinfarct dementia and bearer of a urinary catheter for urethral stenosis. He presented with high fever of seven days evolution, abdominal pain, deterioration of the condition and swelling of the right scrotal area with an ulcerated wound on the right scrotum of septic appearance. Blood pressure was 80/40 mmHg and temperature 38.3 °C. He was poorly perfused and showed a low level of consciousness. There was an erythematosus plaque, indurated at the level of the lower abdomen, painful on palpation, and an enlarged right testicle, fluctuating, with an ulcerated-necrotic lesion that spontaneously drained a foulf-smelling purulent fluid (Figure 1, top). Laboratory tests showed: 17,400 leukocytes/uL (92.8 N, 3.3 L), urea 127 mg/dl, sodium 150 mEq/L, potassium 3.6 mEq/L, and creatinine 1.4 mg/dL. Abdominal computed tomography (CT) scan showed a collection of gas bubbles in front of the left anterior rectal muscle at the level of the prostate gland, which extended from the opening of the planes on the right to level of the FID (Figure 1, lower image). Treatment was initiated with imipenem 2 g/8 h i.v. Urgent right inguinal orchiectomy was performed with extensive debridement of necrotic tissue. The patient died in the immediate postoperative period. A posteriori, Morganella morganii and Providencia stuartii were isolated in microbiological samples of exudate culture and Enterococcus faecalis in blood cultures.

Among the factors that favor the appearance of this entity are immunosuppression (diabetes mellitus, HIV infection), alcoholism, cancer, urethral strictures or bearing a catheter[2]. The origin of the infection is usually[3] colorectal or urological[4]. Bacteriological studies usually show mixed aerobic and anaerobic flora: The main microorganisms involved are Gram-positive aerobes (enterococci), Gram negative *(E. coli, P. aeruginosa, P. mirabilis, K. pneumoniae, P. Stuarti)* and anaerobic bacteria *(Bacteroides fragilis, Bacteroides melaninogenicus, streptococci and clostridia)*[5]. But the diagnosis of Fournier's gangrene is usually based on clinical signs and symptoms, with imaging tests being an important complement.

These include conventional radiology, ultrasound and CT, where not only can one visually identify the presence of subcutaneous emphysema[6], a characteristic finding with this entity, but also asymmetries in the fascia or purulent collections[7]. Treatment is based on broad-spectrum antibiotics together with early extensive surgical debridement[8].

Despite this, mortality is high, ranging from 20-40% in most series, 9 although there are variables such as age, coexisting diseases, the level of healthcare offered in the country or timing of treatment, which may increase or decrease this range[5,8,10].

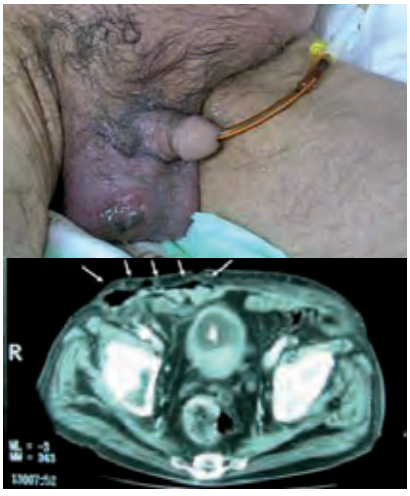

Figure 1. Image showing increased size right testicle with ulcerated lesion and drainage (above). Abdominal CT scan showing a collection of gas bubbles in front of the anterior rectus (below, arrows).

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A "rebellious" urinary bladder catheter

Sir,

Benign prostatic hypertrophy (BPH) is the cause of 53% of acute urine retention in men[1] and treatment most commonly consists of bladder catheterisation. We report the case of a patient who required catheter replacement according to the stipulated protocol of the referring primary care center.

An 84 year-old male patient was referred by the primary care center due to

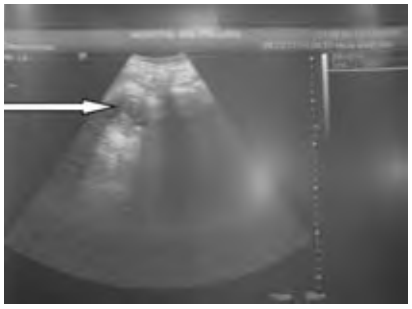

Figure 1. Bladder ultrasound image showing the walls of the catheter balloon (arrow).

Figure 2. Foley catheter with Drum® catheter guidewire used in this case.

the impossibility of replacing a Foley nº 15 catheter. He had used the bladder catheter during several months for various episodes of urine retention, and the only relevant medical history was possible prostate gland cancer (specific PSA 25 ng/ml) for which he was awaiting evaluation for transurethral resection. We found it impossible to deflate the balloon with a syringe using the traditional method of saline extraction. We decided to cut the distal end of the catheter just above the fork, in order to eliminate the possible effect of the retaining valve of the drainage port. Even so, the catheter continued to resist attempts at extraction. Suspecting that the balloon could have burst and the catheter was attached to the walls of the urethra, bladder ultrasound was performed by the radiologist who observed that the balloon was inflated (Figure 1).

We then attempted to deflate the balloon by inserting an intravenous needle through the cut made in the catheter and using pressure aspiration, to no avail. After considering several possibilities, we then decided to use the guidewire for a Drum® catheter (central venous access catheter for peripheral access, 14G) to unblock the probable obstruction, passing it through the inflation channel (Figure 2). This was easily done to the end of the inflation channel, where slight resistance was encountered, but then saline flowed and we were finally able to remove the catheter.

In reports of similar incidents, different types of actions have been described[1-4], including a suprapubic catheterization with puncture under endoscopic guidance, puncture of the balloon with a flexible biopsy needle via a rectal or vaginal tract, hyperinflation of the balloon with saline or air, or chemically-induced rupture using injected paraffin oil or ether into the balloon to cause it to dissolve and burst. Given the success of the guidewire method, a non-aggressive and easy technique, we believe it should be considered a very good option for such situations.

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