Acute adrenal failure, insufficiency a rare cause of pediatric cardiorespiratory arrest

Sir:

Adrenal crisis is rare in childhood[1] (0.014% in USA[2]). A significant proportion of these patients have chronic adrenal insufficiency[3] (primary or secondary), while adrenal crises are extremely rare in patients not previously diagnosed with adrenal insufficiency.

A 7 year-old boy with no history of interest except hospitalization four weeks before for gastroenteritis and possible severe hyponatremia was brought to our Critical and Emergency Care (CEC) unit in his father's arms due to seizures. The father was alerted by strange noises and found his child in bed with abnormal face and right body movements. The patient was unconscious, with pallor, gasping, a respiratory rate of 8 bpm, heart rate of 135 bpm, blood pressure 60/30 mmHg, cold skin, without other signs of hypovolemia (normal skin turgor and good capillary refilling), basal oxygen saturation 91%, capillary blood glucose 25 mg/dl and a skin temperature of 36°C. No hyper-pigmented areas were evident. Intramuscular glucagon treatment was started and the child presented partial facial and right hemisphere seizures. Five mg of diazepam rectal and oxygen by mask with reservoir were administered, while attempting to establish venous access. The situation of venous vascular collapse forced us to resort to right leg intraosseous access, and 500 cc of saline was infused. No pathological cardiac rhythm was observed. We again measured blood glucose which showed a value of 15 mg/dl, for which we administered 30 ml of hypertonic glucose at 50%. Since the seizures persisted, it was decided to sedate the patient with intravenous diazepam and isolate the airway by self-inflating endotracheal balloon intubation. Within two minutes, glucose was 10 mg/dl, his breathing had decreased to 4-5 bpm and heart rate was 10 bpm in sinus rhythm. A further 30 cc of hypertonic glucose was administered and advanced cardiopulmonary resuscitation (CPR) was continued, with left leg intraosseous access and rapid administration of serum glucosaline, and adrenalin and atropine by intraosseous route. A nasogastric tube was used to administer another 30 ml of hypertonic glucose (capillary blood glucose at 1 minute was 10 mg/dl). After continuous advanced CPR and a cardiac rhythm of asystole and no response during approximately 45 minutes, the child was declared dead. Autopsy showed atrophy of the adrenal glands and a significant brain edema.

Endocrine processes that are life-threatening are relatively uncommon, although hypoglycemia and adrenal crises are more frequent than others[4]. Acute adrenal insufficiency can prove fatal[5]. It may appear suddenly in previously healthy subjects, without any apparent precipitating cause. Our pediatric patient was not diagnosed with adrenal insufficiency, showed no fatigue, nausea, vomiting or abdominal pain, was not asthmatic[7] or receiving corticosteroids and there was no stress trigger[5,6]. The seizures, hypotension, hypoglycemia and suddenly impaired level of consciousness should have aroused suspicion of an Addisonian crisis. However, the medical team considered hypoglycemia as the cause of the seizures and the patient was initially and correctly treated with rectal diazepam[8]. He was then treated with intramuscular glucagon and subsequently successive boluses of hypertonic glucose, but hydrocortisone or other intravenous steroids were not considered[9]. The gasping was confused with normal breathing, and CPR was not considered initially. It was started with 5 Ambu rescue ventilations and chest compressions without waiting for a situation of asystole to appear. The team was unable to resolve the hypotension, hypoglycemia and shock. It is clear the approach to such a serious refractory condition is complicated by its relative rarity, particularly in children of primary school age.

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Hypoglycemia after venlafaxine and paroxetine overdose

Sir:

Venlafaxine and paroxetine are anti-depressant drugs and overdoses can produce serotonin syndrome, gastrointestinal, cardiac or neurological alterations, and even death[1-5]. Hypoglycemia, however, is an exceptional complication[6].

We report the case of a 41 year-old woman with a history of congenital intestinal malrotation, episodes of depression and drug overdose (40 mg lorazepam and 400 mg paroxetine). She had no toxic habits. She was admitted to the ED 5 hours after taking, at breakfast time the following drugs: lorazepam 36 mg, 400 mg paroxetine and 4500 mg venlafaxine. She and her family denied the use of any other psychoactive substances, insulin and oral anti-diabetic drugs. Blood alcohol content was 0.04 g/L. She had good general condition,

was afebrile, hemodynamically stable and good oxygen saturation. She had no edema or increased CK. Capillary blood glucose was 52 mg/dl. Neurologically, we observed somnolence, a Glasgow coma score of 13, poor muscle tone, mydriatic reactive pupils, but no other abnormalities. She was treated with hypertonic glucose at 50%, KCl and dextrose for 52 hours, with progressive normalization of blood glucose (capillary blood glucose 70-80 and 90-100 mg/dl at 20 and 30 hours respectively). Fifty two hours after the overdose, fluid therapy was suspended and no new episodes of hypoglycemia were recorded.

This picture of drug intoxication coursed with mydriasis, confusional state, hypotonia and hypoglycemia. The credible denial of having consumed other substances, the absence of previous episodes of hypoglycemia, and the fact that the patient had no access to hypoglycemic drugs and none of her immediate family had diabetes, make it likely that one of the three drugs ingested was responsible for her episode of hypoglycemia. In the literature we found no cases of hypoglycemia after benzodiazepine or paroxetine overdose, but some references to other selective serotonin reuptake inhibitors (SSRI) (sertraline and fluoxetine)[7,8]. We also found a case of hypoglycemia after sertraline and venlafaxine poisoning[6]. One cannot rule out the possible role of benzodiazepines in hypoglycemia after venlafaxine overdose, since the two cases cited had consumed both substances. Moreover, since SSRIs have been associated with hypoglycemia, we cannot rule out that paroxetine had a supporting role, although the patient had no hypoglycemia in the previous intoxication with similar doses of paroxetine and lorazepam. The duration of our patient's hypoglycemia was consistent with the half-life of venlafaxine[9]. Therefore, although this hypoglycemic effect is little known, we believe it should be considered in cases of poisoning by both SSRIs and venlafaxine.

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Complicated mycotic aneurysm abdominal aorta with severe sepsis and rupture of the aortic-vena cava fistula

Sir:

An infected aneurysm is known as mycotic aneurysm (MA). MA of the abdominal aorta is a rare entity.

We report the case of a 68 year-old man with an indwelling urinary catheter for neurogenic bladder and a history of multiple renal stones, urinary infection urinary and sacral ulcers. He presented with malaise, decreased appetite and oliguria of 15 days duration. The patient was hypotensive, tachycardic, afebrile, tachypneic, hypoperfused, dazed, dehydrated, pale, and had a globulous abdomen with voluntary resistance and purulent sacral ulcers. Laboratory tests showed hemoglobin 8.5 g/dL, hematocrit (Hct) 26.5% (anemia present), leukocytes 32.1 x 10^3/µL, neutrophils 82.5%, platelets 503 x 10^3/µL, prothrombin time 18 secs, INR 1.54, kaolin cephalin clotting time 28.4 secs, lactate 6.89 mmol and pH 10.4. Urine analysis showed over 100 leukocytes/c and presence of bacteria. With suspected severe sepsis, the patient was stabilized with early therapy guided by objectives[1]. Shortly after he presented clinical and hemodynamic worsening, with hematocrit 13.6%. Computed tomography (CT) scan showed saccular aneurysm of the infrarenal aorta with filling of the inferior vena cava, compatible with aortocaval fistula, and contrast extravasation compatible with active bleeding to the renal fossa and right space; there was abundant blood in the flank, right perihepatic, perisplenic abdominal wall, and at the bottom of pouch of Douglas. Aneurysmorrhaphy was performed, revealing purulence. The patient died 10 hours later after multiple organ failure.

The incidence of infectious aneurysms secondary to bacteremia is low (from 0.06 to 0.65%[2]). Since the findings of Baker[3], its pathogenesis is related to intimal disease secondary to intimal septic embolism, congenital defect, atherosclerosis, occlusion of the vasa vasorum or trauma. Microorganisms frequently involved are gram negative[4] *(Salmonella sp. Escherichia coli)* and *Staphylococcus sp.* The clinical picture is nonspecific: sepsis that is difficult to control or manifestations related to the early rupture of the aneurysm and its characteristic rapid expansion, which acts as a local mass compressing adjacent structures[5]. The evolution to rupture or sepsis determines the high rate of mortality, even with early surgical treatment and continuous antibiotic therapy. The diagnosis of MA requires high clinical suspicion. In this case, the context of multifocal infection, the evolution of the picture during several weeks, the clinical data obtained in the ED and the response to fluid therapy supported the syndromic diagnosis of severe sepsis. Subsequent developments suggested a bleeding complication, confirmed by complementary tests. This was a rare occurrence of successive presentation in the same patient of the three possible complications of MA of the abdominal aorta: severe sepsis, a fistula to the cava and its rupture.

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Bilateral Chilaiditi's sign

Sir:

As published in a recent issue of EMERGENCIAS[1], Chilaiditi sign is a rare radiological finding consisting of colon or small intestine interposition between the liver and diaphragm, with an incidence of 0.02 to 0.28%[2]; exceptionally it can be bilateral. When associated with abdominal symptoms (pain, nausea, constipation) it is called Chilaiditi syndrome , which is sometimes complicated by colonic volvulus and bowel perforation or obstruction[3-5]. Differential diagnosis must be made with pneumoperitoneum and subphrenic abscess[3]. In the ED, knowledge of this entity may help avoid unnecessary examinations.

We report the case of an 80 year-old man with a history of chronic arterial ischemia of the lower limbs and hypertension who was admitted to the emergency department with diffuse abdominal discomfort and long-standing constipation. Physical examination showed a distended and tympanic abdomen. Posteroanterior chest x-ray showed interposition of the colon between both hemi-diaphragms and the abdominal viscera (bilateral Chilaiditi bilateral). The patient responded favorably to conservative treatment.

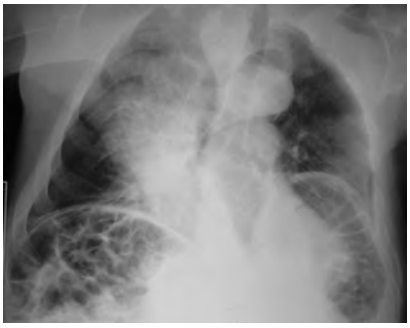

Figure 1. Chest X-ray showing interposition of the colon and small intestine in both sub-phrenic spaces and severe spinal scoliosis.

Chilaiditi sign is a radiological finding consisting of intermittent or chronic interposition of part of the colon, usually the right, and occasionally of the small intestine, between the liver and diaphragm, very rarely on the left side[6] and exceptionally bilateral[7,8]. The etiology is multi-factorial and the following predisposing factors have been identified: chronic constipation, colonic elongation, congenital anomalous position, paralysis of the phrenic nerve, absence of liver right lobe, diaphragmatic hernia, disease chronic obstructive pulmonary disease, adhesions, previous abdominal surgery, cirrhosis and scoliosis, although it is clearly related with age and is more common in older men over 65 years with a male-female ratio of 4:1. It is usually an incidental finding in asymptomatic patients, but in some cases it is associated with gastrointestinal symptoms (nausea, vomiting, anorexia, flatulence, constipation) and then it is called Chilaiditi syndrome. It was first described by Cantini in 1865[2], but named after the Viennese radiologist Demetrius Chilaiditi on the basis of his findings in three asymptomatic patients. This finding has also been linked to chest pain, dyspnea, cough, volvulus and intestinal obstruction[2,9,10]. Diagnosis of Chilaiditi syndrome is always radiological and treatment is conservative in most cases, except when acute abdomen appears in which case emergency surgery is required.

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Usefulness of laboratory findings in the early diagnosis of necrotizing soft tissue infections

Sir:

We have read with interest the article by Guerra et al[1] in which they highlight the importance of early diagnsis of soft tissue necrotizing infection in order to reduce the very high rates of morbidity and mortality associated with this condition.

Indeed we have few reliable tools for the diagnosis of this disease in its early stages where initial clinical findings are often scarce, and we agree that it must be based on medical history (compatible signs and symptoms in a patient with comorbidity that predisposes to this type of infection) and radiological evidence. However, we also consider laboratory parameters very useful for diagnosis[2]. Those included in the Laboratory Risk Indicator for Necrotizing Fasciitis scale, although nonspecific, are routinely obtained; together they provide useful information on the probability of necrotizing fasciitis. A score above 6 on the scale has a positive predictive value of 92% and a negative predictive value of 96%[2]. We agree with other authors who have recently expressed their support for this scale as a useful tool for the clinician when treatment decisions must be taken in the shortest possible period of time[3,4].

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Whole-body computed tomography for the early assessment of severe traumatic injury

Sir:

In cases of severe trauma or multiple injuries, avoidable mortality is estimated to be 21-33% in general hospitals and 6% in specialized trauma centers[1]. Whole-body CT (WBCT) is a relatively recent diagnostic procedure which has not yet been introduced as a routine technique in severe trauma, nor is it mentioned in the relevant guidelines[2,3]. However, studies on the diagnostic safety of WBCT and its efficacy and effectiveness show its is gaining ground as the gold standard diagnostic tool for severe trauma.

We report the case of a 46 year-old woman transferred to our emergency department with multiple injuries after falling from a tree. She suffered head injuries with transient loss of consciousness, as well as neck, chest and abdominal trauma. She was initially attended on the scene by the mobile emergency team (061) who referred the patient after primary evaluation using the standardized protocol of the American College of Surgeons Advanced Trauma Life Support2, and this team had applied a cervical collar, administered high-flow oxygen, fluid therapy and sedoanalgesia with fentanyl and midazolam. Primary re-evaluation in the ED, following the same protocol of care, showed: A) unobstructed airway; B) resting tachypnea (18-25 bpm) with high flow oxygen therapy; C) no evidence of external bleeding, normal color, well perfused, blood pressure 100/60 mmHg, heart rate 105 bpm, oxygen saturation 93%, D) Glasgow coma score 15/15 and normal isochoric pupils. Secondary evaluation with routine craniocaudal examination showed: scalp lesion in the left parietal region reaching the left eyebrow area, about 12 cm long, hematoma in soft tissue of the left lateral cervical area and diffuse superficial erosion on the anterior surface of the upper left chest. She presented decreased vesicular murmur and rhonchi at the upper left level; abdomen was soft and depressible, without defense, with no sign of peritoneal irritation and painful right upper quadrant. High-flow oxygen at 50% was administered. Fluid therapy was administered via a left femoral central line, and the patient received analgesia with metamizol, diclofenac and fentanyl. Laboratory tests showed: leukocytes 17.800/µl, hematocrit 31.5%, urea 48 mg/dl, AST 797 IU/L, ALT 880 IU/l, GGT 41 IU/l, CK 1,259 IU/l and arterial blood gas with pH 7.31, pCO_2 32 mmHg, pO_2 61mmHg and HCO_3 19 mmol /L. Given the clinical and laboratory findings, it was decided to perform a WBCT, the results of which are described in Figures 1-5. During the process of WBCT, a picture of worsening psychomotor agitation was observed, motivated by hypotension (70/30 mmHg), which resolved after the administration of 10 ml/kg of saline solution. Subsequently, the patient was admitted to the intensive care unit for follow up and definitive but conservative treatment of her lesions.

The advent of helical CT scanners in clinical practice in the early 1990s revolutionized radiological diagnosis[4]. Its use in the initial care of serious trauma was first described by Löw et al.[4] in 1997. Subsequent studies have shown the safety of single-pass WBCT scanning in this type of patient[5]. Multislice WBCT saves time when compared with other diagnostic radiology techniques such as plain X-ray, ultrasound or non-multislice CT[6]. However, until recently the benefit of this technique in reducing mortality had not been demonstrated[7]. The authors of the cited study concluded that WBCT, compared to segmental CT, was associated with a statistically and clinically significant reduction of 30% in mortality rate[7-9] and recommended that it be the standard diagnostic tool for the initial evaluation of patients with severe trauma. The cited study has been criticized on the grounds of unnecessary exposure to radiation and iodinated contrast[10], and owing to cumulative doses, WBCT increases individual risk of cancer[11]. However, single-pass WBCT has been shown to expose the patient to lower levels of radiation than a series of segmental CT scans[12].

Salim et al.[13], in a prospective study of 592 trauma patients without obvious signs of injury, reported relevant clinical findings in 20% of patients undergoing WBCT and as a result patient management changed in 19% of cases, so their conclusion was that this technique should be more widely employed in this type of patient.

Some authors[4] believe that WBCT is contraindicated or can be dangerous in patients with hemodynamic

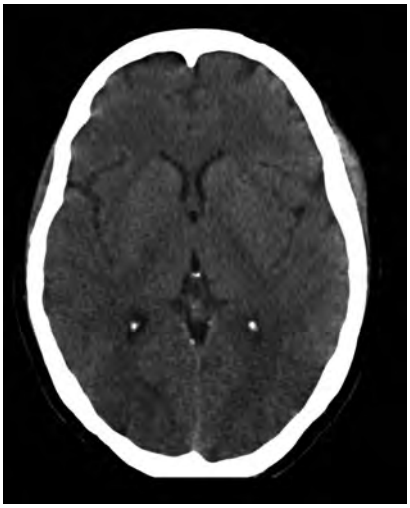

Figure 1. Cranial CT, axial plane. Left cephalohematoma (arrow).

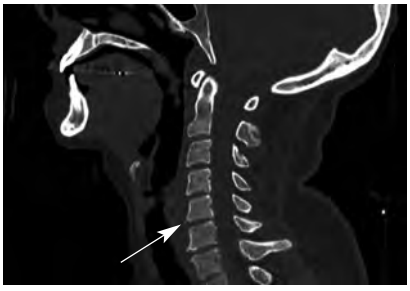

Figure 2. Cervical CT, medial plane. Vertebral fracture was observed with bone fragment anterior to vertebra C5 (arrow). Medullary canal intact.

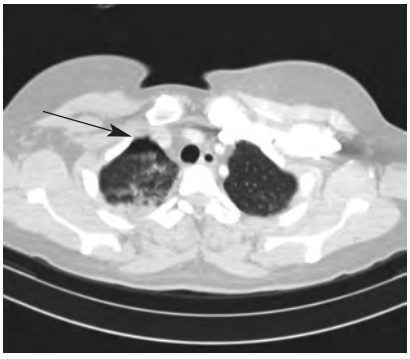

Figure 3. Contrast-enhanced CT scan of the chest, axial plane. Lung contusion with right hydropneumothorax (arrow).

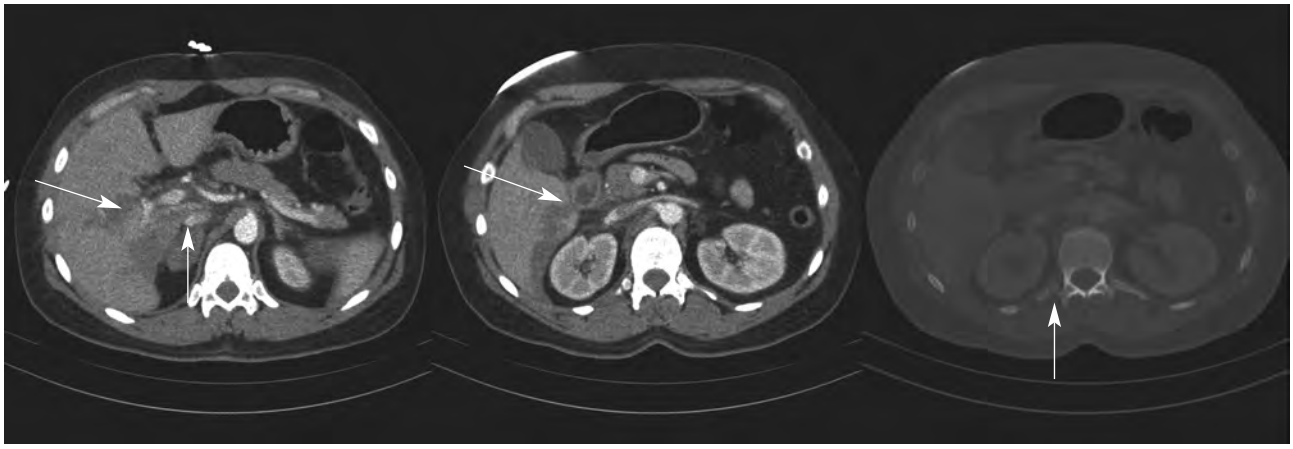

Figure 4. Contrast-enhanced abdominal CT scan, axial planes. Left: liver laceration with a small amount of free fluid (arrow). Center image: right adrenal hematoma (arrow). Right: transverse process fracture of lumbar vertebra (arrow).

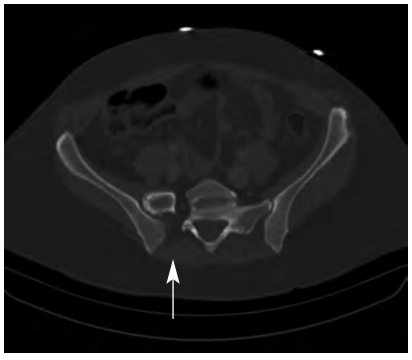

Figure 5. Contrast-enhanced pelvic CT scan, axial plane: right sacroiliac joint diastasis.

instability due to hypovolemic shock. It is true that the probability of survival decreases by 1% every 3 minutes[15] in patients with hemorrhagic shock, but WBCT can be performed in 3-6 minutes[5]. In view of this controversy, prospective randomized studies are needed. For hemodynamically unstable patients, the CT scanner must be rapidly available, adjacent to or in the same resuscitation area, and that the scanning process be carried out in the shortest possible time.

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