Can the differential diagnosis of acute vestibular syndrome be improved? The eyes hold the key

Sir,

Since Gutierrez Solis et al.[1] first published their work in this journal in 1996, diagnostic methods of vertigo in emergency department (ED) patients have changed considerably.

Vertigo remains a common cause of ED visits, with a prevalence of around 5%[2]; of which 4-6% are associated with a cerebrovascular accident[3]. Moreover, an incorrect approach in the ED can lead to misuse of imaging tests that do not help the diagnostic process[4].

For this reason, the application of diagnostic protocols based on ocular examination and vestibular-ocular reflex (VOR) with high sensitivity and specificity should be promoted to distinguish between peripheral or central causes of vertigo[5]. These protocols are based on the study of three clinical signs: the head impulse test or head-thrust test, direction of nystagmus and eye alignment during the occlusion test[3,6].

In the head impulse test[7] the patient is asked to fix their gaze on a target point (usually the examiner's nose) and the examiner, holding the patient's head with both hands, then makes a forceful but controlled head movement to the left or to the right. If the patient's horizontal duct VOR is conserved, he/she will be able to keep their eyes on that target, but if the VOR fails, the patient's eyes will be diverted to the left or to the right and he/she will need one or more corrective saccades to regain the initial position.

The direction of nystagmus is easily examined. Peripheral nystagmus is characterized by the fact that the eye always twitches in the same direction regardless of where the patient is looking. By contrast, in central nystagmus the nystagmus usually twitches towards the side to which the gaze is directed (directional nystagmus)[8].

Finally, the alternating occlusion of the eyes can produce a movement of vertical realignment if there is a problem in the brainstem, while this is very unusual if the problem is vestibular[9].

The overall sensitivity of these three tests is greater than 95% and specificity close to 85% (higher than radiological tests applied indiscriminately) and they must be considered especially in patients with vascular risk factors[3]. For this reason we consider it vital to know and perform these tests in patients presenting to the ED with any form of dizziness

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Angel BATUECAS CALETRIO[1],
Juan Manuel ESPINOSA-SÁNCHEZ[2],
José Antonio LÓPEZ-ESCAMEZ[3]

[1]Servicio de ORL y PCF, Unidad de Otoneurología, Hospital Universitario de Salamanca, Spain. [2]Grupo de Otología y Otoneurología CTS495, Hospital San Agustín, Linares, Jaén, Spain. [3]Grupo de Otología y Otoneurología CTS495, Hospital de Poniente, El Ejido, Almería, Spain.

Wunderlich syndrome, a rare cause of acute abdomen

Sir,

Renal, perirenal or spontaneous retroperitoneal bleeding or Wünderlich syndrome is a vital emergency to be included in the differential diagnosis of acute abdomen. CT scan is essential for diagnosis and the appropriate therapeutic approach. We present a case of Wünderlich syndrome associated with anticoagulation and antiplatelet therapy, an exceptional cause of this condition.

An 81 year-old man suffered sudden abdominal pain and hemodynamic deterioration three hours after receiving a loading dose of antiplatelet and anticoagulation treatment with low molecular weight heparin for acute coronary syndrome (ACS). Physical examination showed malaise, pallor, tachycardia (120 beats / min) and hypotension (70/30 mmHg) which recovered after administration of plasma expanders. Abdominal palpation was very painful, with defense and sense of mass in the upper right quadrant. Laboratory tests showed: hemoglobin 8.1 g / dl and hematocrit 24.6% (he presented normal values on admission) with an INR of 2.4 and cephalin time of 74 seconds. Abdominal CT scan showed a right renal subcapsular collection with hyperdense foci of active bleeding with occupation of the retroperitoneal space (Figure 1). The anterior surface of the kidney in contact with the collection showed cortical cystic lesions. The patient presented major hemodynamic instability and required emergency nephrectomy. In the resected specimen, three cysts were identified with signs of bleeding

The term Wunderlich syndrome refers to renal, peri-renal or spontaneous retroperitoneal bleeding of non-traumatic etiology[1,2], first described by Wunderlich in 1856[3]. It is a rare entity, with only 20 cases reported in Spain before 2009[4]. A literature review by Kendall in 1974 reported that the most frequent cause was renal tumors (50%), among which adenocarcinoma (33%) was the most frequent. Other less common causes are: vascular (20%), infectious (10%), coagulopathy (5.1%) and miscellaneous (3%)[5,6]. The use of anticoagulants and antiplatelets may fa-

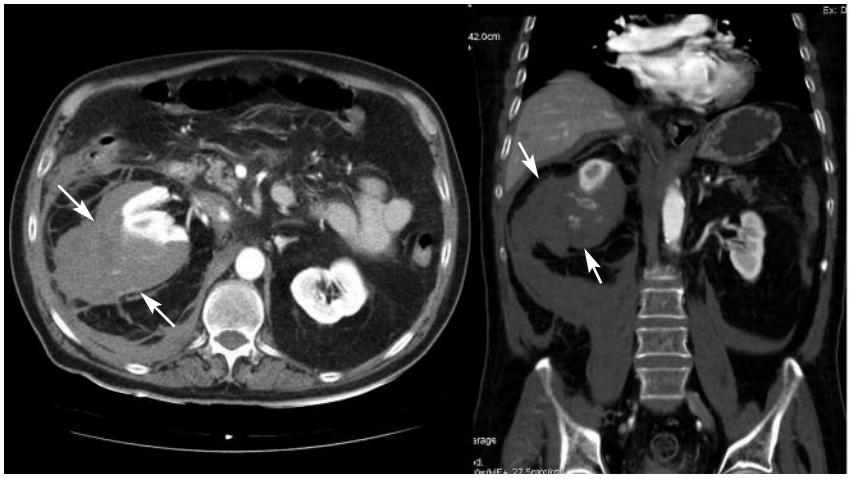

Figure 1. CT image of the abdomen with contrast (left: axial section; right: cross-section) showing a right subcapsular collection (arrows) with foci of active bleeding occupying the right pelvic retroperitoneal space.

vor their appearance. A typical clinical picture is known as Lenk's triad: sharp pain in the flank, palpable abdominal mass and rapid onset hypovolemic shock[3]. But this presentation only occurs in 20% of cases. There are more insidious presentations as a result of slow or scarce bleeding[7]. Imaging with abdominal ultrasound and CT are fundamental for diagnosis and aid decisions on therapeutic measures[1]. CT detects the extent of bleeding and can identify the causes, and guides the treatment plan for reconstruction of the kidney and vascular structures[3,7,8]. Conservative management is usually adopted in clinically stable cases of benign etiology, with CT follow up[9]. Nephrectomy is reserved for malignant cases and emergency nephrectomy for urgent cases in which, despite the benign etiology, there is destabilization that cannot be treated with other methods[10].

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José GÓMEZ RUBIO[1],
Ana Belén BÁRCENA ATALAYA[2],
Francisco RIVERA MUÑOZ[3]

[1]Servicio de Medicina Interna, [2]Servicio de Cuidados Críticos y Urgencias, [3]Servicio de Urología, Hospital Universitario de Valme, Sevilla, Spain.

Infant acute myocardial infarction due to congenital coronary anomaly

Sir,

The diagnosis of acute myocardial infarction is extremely rare in Pediatric medicine because of its low prevalence and the lack of clinical suspicion in children, where cardiovascular risk factors are usually absent. The most common etiology is congenital coronary anomalies[1] especially anomalous left coronary artery from the pulmonary artery (ALCAPA).

A previously healthy 2-month-old boy was brought to the emergency department for intermittent episodes of intense crying followed by syncope. Physical examination showed signs of cardiogenic shock. Chest radiography showed cardiomegaly with pulmonary edema. The ECG showed ST elevation and pronounced Q waves in leads I, aVL and left precordial leads suggestive of left anterolateral infarction. Biochemistry revealed elevated troponins. Echocardiography showed dilated cardiomyopathy and severe mitral regurgitation, but could not confirm the origin of the left coronary artery of the aortic root. Upper parasternal short axis echocardiography showed the absence of a connection between the left coronary artery and the aortic root, as well as reverse flow from the pulmonary artery (Figure 1). With these data, ALCAPA was suspected and confirmed by catheterization (Figure 1). After hemodynamic stabilization with iv infusion of milrinone and nitroglycerin, iv, the infant underwent surgery with re-implantation of the anomalous coronary in the aortic root without complications. At 6 months the patient was asymptomatic, without treatment, with normal imaging test results.

ALCAPA is the most frequent congenital coronary anomaly in infants (incidence 1 / 300,00 live newborns, 0.25-0.5% of all congenital heart disease)[2]. It causes severe heart failure due to severe dilated cardiomyopathy secondary to progressive left ventricular ischemia, territory irrigated by blood which is oxygen desaturated and of low pressure from the pulmonary artery[3].

It should be suspected in infants over 1 month of age with sudden episodes of irritability triggered by normal effort, such as crying or feeding, with signs of poor peripheral perfusion and syncope, with electrocardiographic signs of left anterolateral myocardial ischemia[4]. The diagnosis is confirmed by echocardiography, CT angiography or catheterization[5,6].

Preoperative management should include sedoanalgesia and oxygen, inodilator drugs such as milrinone and nitroglycerin to achieve an appropriate balance between systemic and pulmonary pressures and prevent diastolic theft of the coronary artery from the pulmonary artery[7], unless there is severe hypotension

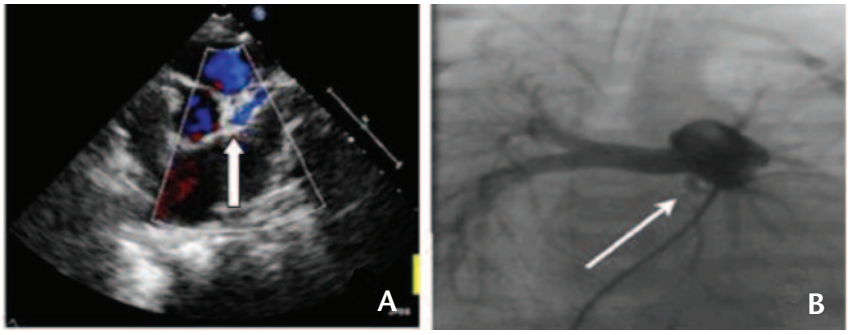

Figure 1. (A) Echocardiography in the upper parasternal short axis showed the absence of a connection between the left coronary artery and the aortic root (arrow), and reverse flow from the pulmonary artery. (B) Pulmonary aortography showed the origin of the left coronary artery from the main pulmonary artery (arrow).

when adrenaline is recommended. Furthermore, given the absence of coronary obstruction, the following are not indicated: acetylsalicylic acid, fibrinolysis with rTPA or angioplasty. Surgical treatment is effective and the prognosis is excellent, with high survival rates without sequelae but this requires early diagnosis, before the onset of ventricular myocardial necrosis, in which case mortality rises dramatically (90%)[8-10].

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Moisés RODRÍGUEZ-GONZÁLEZ[1],
Miguel Ángel MATAMALA-MORILLO[1],
Antonio SEGADO-ARENAS[2]

[1]Sección de Cardiología Pediátrica, [2]Sección de Neonatología, Hospital Universitario Puerta del Mar, Cádiz, Spain.

Coagulation disorder after a bite from *Vipera latastei*

Sir,

In Spain, snakebites are relatively rare but constitute an emergency when they do occur[1]. In the Iberian Peninsula there are five species of poisonous snakes, three belonging to the *viperidae* family and two to the *colubridae* family[2]. Although the venom of these snakes acts on coagulation, the signs and symptoms are often restricted to the affected extremity and coagulopathy is highly unusual.

A 44 year-old man, out riding a mountain bike in a coastal area of Barcelona (Montseny) in the month of September, was bitten by a snake (probably *Vipera latastei*) on the index finger of the left hand. On arrival at the first emergency department room, he presented a hemorrhagic phlyctena at the site of the bite as well as edema of the hand and forearm to the armpit, a palpable axillary lymphadenopathy, abdominal pain, nausea and vomiting. The thrombocytopenia (30 x 10^9 / L) and the absence of an antidote in the first medical center prompted his referral to our hospital. On arrival, abdominal pain persisted. Laboratory tests confirmed the thrombocytopenia (21 x 10^9 / L), a decrease of prothrombin time (37%), normal fibrinogen (2 g / L), with negative PDF and activated partial thromboplastin time was 29.7 seconds. The arm edema continued to progress so a vial of anti-venom was administered. This was associated with rapid recovery of platelet count and prothrombin time (Table 1), and the edema ceased to progress, although a large local ecchymosis appeared (Figure 1). The patient improved gradually and was discharged after 5 days.

From the point of inoculation, venom diffuses through the interstitial space and enters the lymphatic system. Lymph nodes are a significant barrier against further diffusion patients generally have no systemic manifestations[3]. The coagulopathy observed may be due to the fibrinogenolytic activity of the venom i.e. the direct destruction of fibrinogen with prolongation of prothrombin time, and also to direct rupture of platelet walls by A2 antiphosfolipase platelet activity, which would explain the thrombocytopenia[4-8]. Therefore, we do not consider it likely that disseminated intravascular coagulation occurred. In the case presented here, the abdominal pain, vomiting and especially the coagulopathy all indicate diffusion of the venom and constitute an indisputable indication for antidote administration[9]. In Spain, the Ministry of Health distributes

a polyvalent anti-venom for European vipers, with fragmented antibodies which reduce the risk of an anaphylactic reaction[10].

Table 1. Evolution in time of coagulation factors

Variables	AVA								
Time from bite (hours)	+2	+7	+9	+11	+16	+20	+44	+92	+140
Platelets (10^9 / L)	30	21		174	226	237	249	178	281
Prothrombin time (%)	37	37,1		49,7	51,6	58,2	69,9	92	98
Fibrinogen (g / L)				2					3,5
Products of fibrinogen degradation (PDF)		Negative							

AVA: Administration of anti-venom (4 ml).

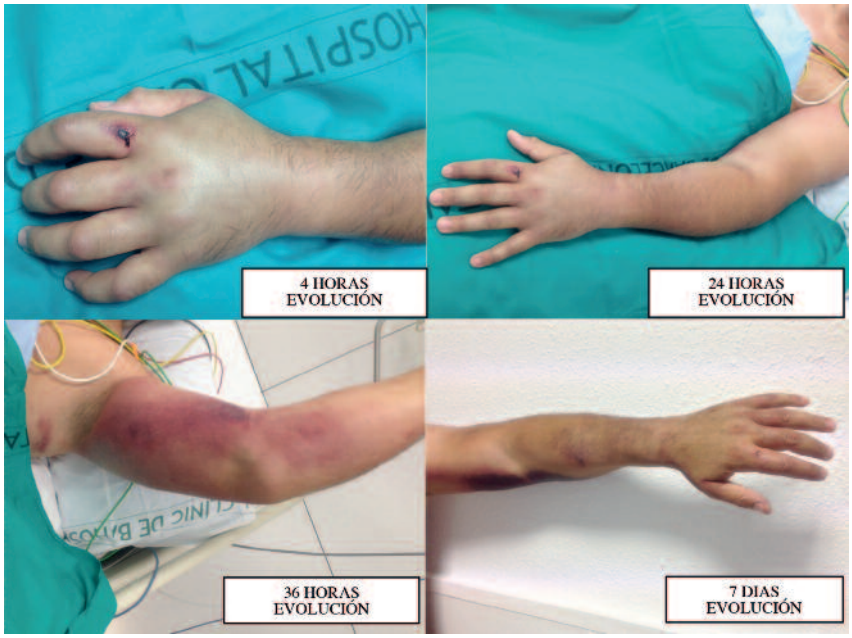

Figure 1. Clinical course of the lesion in the left arm.

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Ona ESCODA-TURON[1],
Cristina MARTÍN-SIERRA[2],
Joan Carles REVERTER[3],
Santiago NOGUÉ-XARAU[4]

[1]Servicio de Medicina Interna, Hospital Clínic, Barcelona, Spain. [2]Mutua Asepeyo, Teruel, Spain. [3]Servicio de Hemoterapia-Hemostasia, CDB, Hospital Clínic, Barcelona, Spain. [4]Área de Urgencias, Hospital Clínic, Barcelona, Spain. Grupo de Investigación "Urgencias y Patologías", IDIBAPS, Barcelona, Spain.

Intentional ingestion of 20% hydrochloric acid solution

Sir,

Poisoning by caustic agents is rare[1]; 80% of cases involve accidental ingestion by children. In adults it is more common in attempted suicide[2] with higher volumes and more corrosive agents resulting in more serious injuries. We present a case of voluntary intake of hydrochloric acid (HCl).

A 35 year-old man was transferred to the ED after ingesting 20 ml of HCl with suicidal intent. He complained of a burning sensation in the oropharynx. Physical examination was unremarkable. Electrocardiogram, blood test and X-rays of the chest and abdomen were normal. Omeprazole 80 mg was administered intravenously and perfusion was started with 8 mg / hr. Emergency gastroscopy showed Zargar IIa injury of the esophagus, and IIIa of the stomach (Figure 1). He was admitted, with nil per os (NPO) and perfusion of omeprazole, methylprednisolone 1mg / kg and iv amoxicillin / clavulanate 1 g / 8 hours. After 72 hours NPO, gastroscopy was repeated without changes being observed. Food intake was started and the patient was discharged after 4 days, on omeprazole 40 mg / 24 hours and oral sucralfate 1000 mg / 8 hours.

Hydrochloric acid is a potent solution used for cleaning and disinfection of floors and toilets. The severity of injury depends on the concentration and amount ingested. The usual symptoms are oropharyngeal, retrosternal or epigastric pain, dysphagia, hyper-salivation, nausea and vomiting[3]. If the pain is persistent or there are there signs of shock, perforation should be suspected. However, clinical manifestations do not correlate with mucosal damage[4]. Gastroscopy should be performed within 24h of ingestion[5]. Treatment depends on endoscopic findings[6] according to Zargar's classification system of lesions[7]: grade 0 or I (edema and hyperemia) should be treated with proton pump inhibitors (PPI) and soft diet; grade IIa (superficial ulcers) requires NPO and iv PPI; grade IIb / IIIa (circumscribed ulcer or limited necrosis) requires NPO, iv PPI, and possibly steroids and antibiotics; and grade IIIb / IV (extensive necrosis or perforation) requires ICU admission and treatment with NPO, iv PPI, and possibly corticoids, antibiotics and surgery. Although steroids are widely used, there is no evidence for their systematic use to prevent the onset of stenosis, and they may even have adverse effects[8]. Antibiotic use should be limited to treating established infection, perforation, bronchial aspiration or corticoid use[9]. Other authors support their use in conjunction with corticosteroids to prevent stenosis[10]. Our patient presented serious injury despite the mild clinical symptoms, and the limited amount of HCl ingested favored his recovery.

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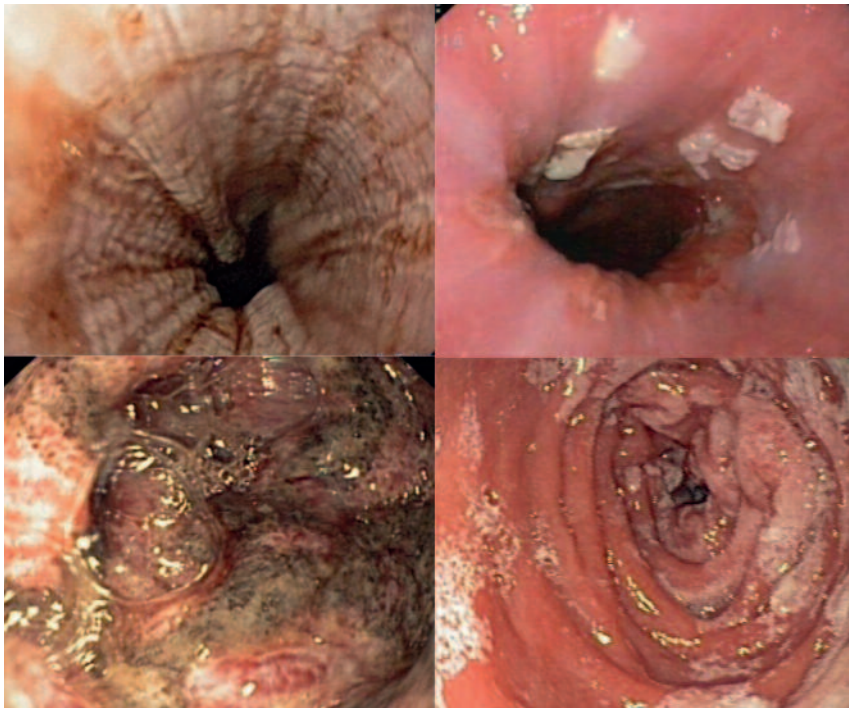

Figure 1. Gastroscopic images showing the lesion produced by HCL ingestion. Above: grade IIb circumscribed ulcers in mucosa at one third of the esophagus (left) and cardias (right). Below: Grade IIIa ulcers with necrotic areas on the wall of the proximal gastric body (left) and grade IIa superficial ulcers in the distal gastric body (right).

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David NAVARRO GONZÁLEZ,
Ignacio PÉREZ LITAGO,
Iñigo ALONSO SEGURADO,
Laura SÁNCHEZ IÑIGO

Servicio de Urgencias, Hospital García Orcoyen, Estella, Spain.

Unilateral mydriasis after contact with flowers of the genus *Brugmansia* (angel's trumpet)

Sir,

Spontaneous unilateral mydriasis is an uncommon condition in the emergency department (ED). The absence of other ocular or systemic pathology data often disconcerts the examiner[1].

A young man attended the ED for left unilateral atraumatic mydriasis of 4 hours duration, without loss of visual acuity in either eye, oculomotor paresis or other local or systemic symptoms. He showed non-reactive left mydriasis to artificial light, and convergence without response to pilocarpine 2%. Basic lab tests and cranial computed tomography were normal. The patient reported having rubbed his eye while doing some gardening work. On being shown internet photographs, the patient reported having touched a large bell-shaped flower called angel's trumpet, known in Spain as floripondio (*Brugmansia* or *Datura*). The mydriasis resolved spontaneously after 48 hours

The differential diagnosis of pupillary mydriasis includes multiple entities such as Adie syndrome or Adie's tonic pupil, partial third cranial nerve palsy and some poisonings.

Pilocarpine, initially at low doses (0.2%) is useful. Adie's pupil (being denervated it is hypersensitive) shows a miotic reaction. If there is no reaction, pilocarpine at 2% is administered; if it reacts (meiosis), partial third cranial nerve palsy is probable. If there is no reaction, one can infer a toxic ocular cause affecting the ciliary receptors[2]. The main toxins are atropine, hyoscyamine and scopolamine contained in species such as belladonna, henbane, nightshade, tomato, potatoe, eggplant, pepper and other solanaceae. A dozen cases of unilateral mydriasis due to contact with Brugmansia and Datura have been published[3-10]. Systemic toxicity is common in recreational use by boiling the flower[4], causing anticholinergic syndrome consisting of mydriasis, tachycardia, dry skin and mucous membranes, disinhibition, agitation and confusion; high doses may induce convulsions, impaired consciousness and coma. Treatment is support, anti-adherents and physostigmine. The interest of this case lies in the use of internet images to identify the plant most probably involved which helped make the diagnosis. Knowing the species responsible is useful for identifying them in the interview.

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Figure 1. Flower known as angel's trumpet, or floripondio in Spain.

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Alejandro GALLARDO-TUR,
Francisco J. GARZÓN-MALDONADO

Unidad de Gestión Clínica Intercentros de Neurociencias de Málaga, Instituto de Investigación Biomédica de Málaga (IBIMA), Hospital Universitario Virgen de la Victoria/Universidad de Málaga, Málaga, Spain.

Pregnant body packer

Sir,

We have read with great interest the recent article by Dueñas et al on body packers. We would like to report our recent experience with a pregnant women who required unconventional diagnostic methods; the whole process meant a disturbing delay in initiating treatment which greatly increased risks for the mother and child.

A 33 year-old woman was arrested at the airport on suspicion of being a body packer. Her medical history was unremarkable but she was 24 weeks pregnant. She initially denied the charges and refused to undergo the usual imaging test (abdominal radiography) for which her consent was required since radiography is not indicated in a pregnant woman. This led to a delay in diagnosis and treatment, increasing the risk of wrapper rupture. She consented to examination by a gynecologist and abdominal ultrasound, but the results were inconclusive. Finally, the diagnosis was made using magnetic imaging (MRI) and the woman admitted having ingested 58 packs of cocaine the day before (Figure 1). From this point, controlled evacuation began, lasting two days without complications.

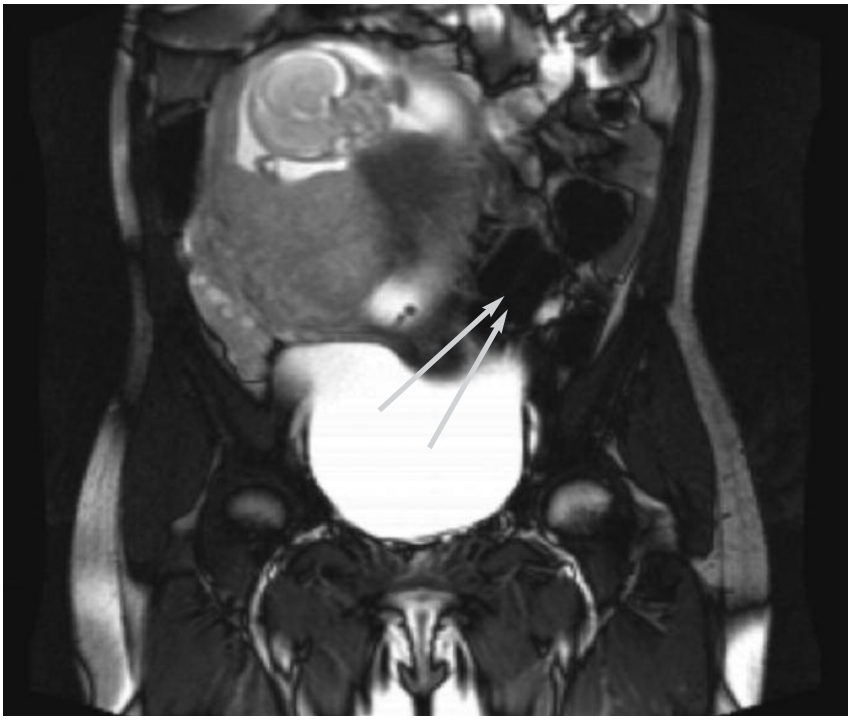

Figure 1. Magnetic resonance images showing cylindrical shapes with sharp and defined contours, with a non-anterior position, compatible with foreign bodies (arrow). There was also a distended bladder and pregnant uterus, and no free fluid at the level of the pelvic floor.

We do not know the reasons behind the use of this type of body packer but it seems they exist and that this situation will be repeated in the future. Therefore we believe specific protocols for the management of pregnant patients should be developed. Such patients require means not always available in all centers. For these cases we propose MRI as the diagnostic method of choice and that the attending hospital should have a neonatal intensive care unit available.

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Ricardo RUBINI PUIG[1],
Eva SAN VALERO CARCELÉN[1],
Benjamín CLIMENT[2],
María RUBINI GIMÉNEZ[3]

[1]Servicio de Urgencias, [2]Departamento de Toxicología - Medicina Interna, Consorcio Hospital General Universitario Valencia, Spain.
[3]Departamento de Cardiología, Hospital Universitario de Basilea, Suiza.

Acute muscle weakness in the emergency

Sir,

We present a case of asthenia with a rare etiology: hypokalemic

thyrotoxic periodic paralysis (TPP), associated with poor prognosis unless diagnosed and treated early, and this makes it especially relevant for emergency physicians.

A 34 year-old Peruvian man with a history of hyperthyroidism (Graves Basedow disease diagnosed 3 months before [TSH <0.01 mU / l (0.34 to 5.6), free T4 3.06 mU / l (normal: 0.61 to 1.24), antithyroid peroxidase Ab 2468.4 IU / ml (normal <60.0), Ac antireceptor TSH 7.93 U / l (normal: 0-0.7)] consulted in the emergency department for muscle weakness to the point of being unable to walk - predominantly distal and lower limbs, of three hours duration, which progressed symmetrically reaching the upper limbs, but no other accompanying symptoms. The patient had been taking propranolol 20 mg / day and tiamizol 5 mg / day from the diagnosis of hyperthyroidism, and he reported similar episodes which had been self-limiting. Physical examination showed nervousness, warm and clammy skin, heart rate 75 bpm and symmetrical limb tetraparesis mainly of the legs, with hypotony and hyporeflexia. Lab tests showed K 1.2 mmol / L (normal: 3.5-5.5) with K in urine 7.7 mmol / day, total CK 212 mU / mL (normal: 15-190) and phosphate 2.8 mg / mL (normal: 2.2 to 4.4). ECG showed sinus rhythm at 60 bpm, PR 0.12 ", serum diluted lengthened QTc (0.42 "). Treatment was initiated in the ED with iv KCL interval and continuous infusion.

The clinical symptoms reversed at 10 h, with K value higher than 4.7 mmol / L (at 36 h of admission). The thyroid profile showed: TSH 0.00 mU / L and free T4 3.56 mU / L. These data indicated a probable diagnosis of TPP. He was prescribed tapering tiamizol from 30 mg / day and propranolol from 30 mg / day with oral potassium.

TPP is an acute, possibly life-threatening complication of hyperthyroidism, particularly Graves Basedow disease[6,7,9]. Acute muscle weakness is more common in 20-40 year-old men[1,2], with a clear geographical distribution (predominantly Asian)[1,4,6]. Cases of familial hypokalemic thyrotoxic periodic paralysis involving HLA-DRw8 have been reported[1,3,7]. The pathophysiology is the result of a rapid and massive movement of extracellular K to intracellular space by the Na / K ATPase pump, due to stimulation of the ß2-adrenergic system by thyroid hormones. This happens in different situations: hyperinsulinemia (exaggerated response of insulin secretion to glucose overload in these patients), drugs (steroids, adrenaline, amiodarone)[7,10], exercise, carbohydrate or alcohol intake[1,3,4]. Testosterone also facilitates this, which explains the higher incidence in men[1,2]. The classical clinical triad consists of generalized muscle weakness (with previous episodes of lower intensity and nocturnal episodes triggered by precipitating factors), tachycardia and hypertension, in the context of hypokalemia and hyperthyroidism[1]. Urinary potassium levels below 15 mEq / l rule out renal loss as in our case. The degree of hypokalemia correlates with the intensity of muscle weakness, but not with thyroid hormone values. Differential diagnosis should be performed with Guillain-Barré syndrome, urinary losses of K, familial and sporadic paralysis, myasthenia gravis, multiple sclerosis and thyrotoxic myopathy. The distinction is easily made by the determination of thyroid function, potassium levels and is urinary potassium levels[1,7]. The goal of treatment is to prevent the onset of life-threatening cardiac arrhythmia8 as well as respiratory failure due to hypophosphatemia[1,8], and reverse the muscle paralysis by regulating K to a safe rather than normal level, to prevent rebound hyperkalemia[3]. Potassium replacement with iv KCl 10 mEq / h if K <2.5 mEq / L is recommended and orally when higher than this value (we found no protocol in the literature specifying that the replacement of K should be adjusted according to patient weight). Avoid additives with intravenous dextrose due to the exaggerated response to carbohydrates in these patients. Propranolol at a dose of 3-4 mg / kg quickly reverses the symptoms and prevents refractory hyperkalemia[3]. Prevention against future attacks involves avoiding precipitating factors and the use of propranolol 20-80 mg every 8 h until definitive treatment of hyperthyroidism is initiated[4].

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Jessica RUIZ IZQUIERDO[1],
Javier RAMOS LÁZARO[1],
Abraham CHAVARIN[2],
Álex SMITHSON[2],
Margarida FERRER CAMPS[3]

[1]Servicio de Medicina Interna, [2]Servicio de Urgencias, [3]Servicio de Endocrinología, Fundació Hospital de l'Esperit Sant, Santa Coloma de Gramenet, Barcelona, Spain.