LETTERS TO THE EDITOR

Venous-arterial extracorporeal membrane oxygenation in combination with the Impella CP® heart pump for the early treatment of refractory cardiogenic shock

Oxigenación con membrana extracorpórea venoarterial y soporte ventricular (Impella CP®) en el tratamiento precoz del shock cardiogénico refractario

To the editor:

In cardiac arrest and refractory cardiogenic shock, circulatory aids such as extracorporeal venoarterial membrane oxygenation (ECMO-VA) provide adequate hemodynamic support, oxygenation and tissue perfusion[1]. Its use has become widespread in Spain with increasing experience[2], allowing an early onset of assistance through the peripheral approach, essential to prevent the progression of shock and reverse this situation[2].

A 68-year-old man with no relevant background consulting for angina effort. Physical examination was normal and ergometry under medical treatment was early positive. Coronary angiography showed chronic occlusion of the proximal anterior descending artery (ADA), with distal vessel filled by collateral circulation from the right coronary artery (RCA), and injury to the proximal circumflex artery. Cardiac magnetic resonance showed viability in the ADA territory and percutaneous retrograde coronary revascularization was decided. During the procedure, the patient showed elevation of the ST segment in the lower face and there was spasm of the RCA and loss of collateral circulation to the ADA, which was complicated by cardiac arrest (CA) due to ventricular fibrillation. Advanced cardiopulmonary resuscitation (CPR) manoeuvres were initiated, with several electric shocks, intubation and perfusion of vasoactive amines. In refractory CA, an ECMO-VA (CARDIOHELP™, MAQUET Cardiopulmonary and AG, Hirrlingen, Germany) was implanted by right femorofemoral access in the haemodynamic laboratory. Angiography showed an acute occlusion of the circumflex artery, which was treated with a pharmacoactive stent and flow was also restored in the RCA. Despite circulatory support, great electrical instability persisted (alternating episodes of ventricular fibrillation and extreme bradycardia), severe ventricular dysfunction, and pulmonary edema developed, so an Impella CP® ventricular support device (ABIOMED, Danvers, Massachusetts, USA) was implanted through the left femoral artery and a transient pacemaker (Figure 1). After achieving hemodynamic stability, the patient was admitted to the intensive care unit. He progressively improved electrical instability and pulmonary congestion and ventricular function, and mechanical support was removed four days later. Subsequent evolution was satisfactory, and he was discharged with good functional statusl.

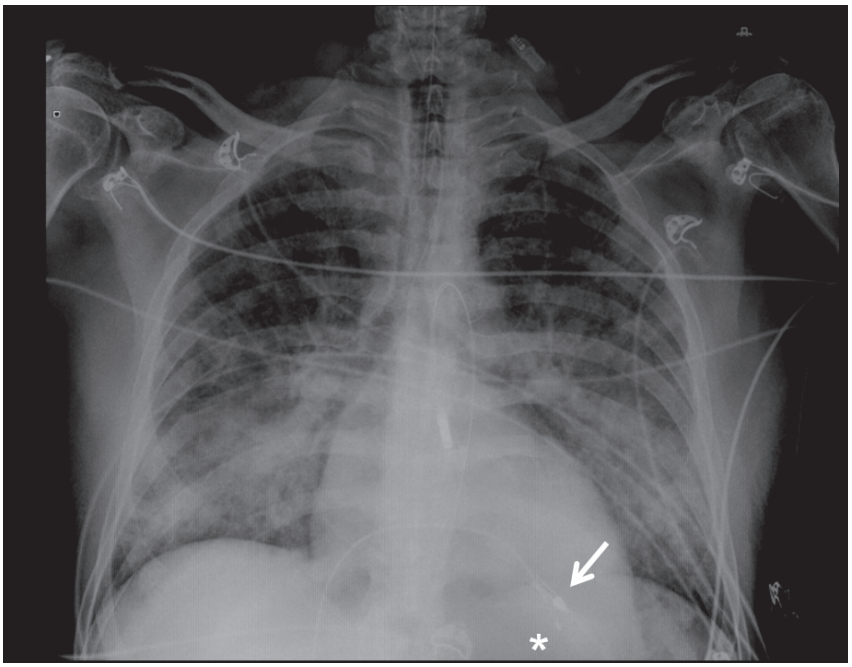

Figure 1. CP® Impeller device with left ventricle terminal (arrow). Transient pacemaker in the apex of the right ventricle (asterisk).

An ECMO-VA was implanted in our patient to stabilize his hemodynamic situation, but the ventricular discharge was compromised by an increase in post-loading, with the development of pulmonary edema. The use of Impella CP® in this context[3] can reduce filling pressures and favour ventricular discharge, while improving pulmonary congestion and the hemodynamic situation. Our work is novel in that it represents the first successful case in which ECMO-VA and Impella CP® are used in combination in Spain in the treatment of refractory cardiogenic shock.

Pablo Díez Villanueva[1],
Fernando Rivero[1],
Nieves García Vázquez[2],
Fernando Alfonso[1]

[1]*Cardiology Department, Hospital Universitario La Princesa, Madrid, Spain.*
[2]*Intensive Care Unit, Hospital Universitario La Princesa, Madrid, Spain.*

pablo_diez_villanueva@hotmail.com

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Acute nicotine poisoning: a rare cause of paroxysmal atrial fibrillation

Intoxicación aguda por nicotina: una causa infrecuente de fibrilación auricular paroxística

To the editor:

Nicotine is a cholinergic agent whose cardiovascular effects are based primarily on stimulation of the sympathetic nervous system[1]. The increasing use of electronic cigarettes (EC) is a potentially new toxic source of nicotine[2]. References in the literature to episodes of atrial fibrillation (AF) in the context of acute nicotine poisoning are anecdotal.

A 51-year-old woman who had been a passive smoker, with no other cardiovascular risk factors, came to our emergency department. She referred dizziness, nausea and vomiting during the two hours after accidentally rinsing her mouth with a small amount of liquid to fill the EC, without ingesting it. This product was composed of 1.2% nicotine (12 milligrams per milliliter) and an unknown concentration of propylene glycol and glycerin. The patient had a blood pressure of 126/86 mmHg, a heart rate of 150 bpm, 96% oxygen saturation, and was eupneic and afebrile. Rapid, arrhythmic heart sounds were prominent, and the rest of the physical examination was normal. The electrocardiogram showed AF at 150 bpm, maintained until three hours after contact with the toxicant, when it spontaneously reverted to sinus rhythm with normal ventricular frequency. No anticholinergic antidotes or antiarrhythmic drugs were used. One month later, a 24-hour ambulatory electrocardiographic monitoring study and a transthoracic echocardiogram were performed which were normal. No episodes of AF occurred within six months of the episode described.

The temporary association between the development of the AF episode and contact with nicotine, as well as the absence of known heart disease, suggest that acute nicotine poisoning was the trigger of the arrhythmia. Most of the described cases of AF attributable to nicotine exposure occurred after relatively prolonged consumption over time or after contact with high doses of nicotine[1,3]. It also contrasts with our case that many of the patients with complications related to tobacco substitution treatments were still active smokers or had cardiovascular comorbidities[1].

Smoking tobacco increases the risk of AF. Nicotine increases heart rate as a consequence of increased plasma catecholamine concentrations due to its stimulating effect on the sympathetic neurotransmission. Nicotine can also alter the conduction through the ionic channels of the auricular myocytes, due to the release of neurotransmitters or to the direct interaction on the ionic channels, which could increase the vulnerability to fibrillar[4]. Inhalation, ingestion or physical contact with solutions containing nicotine can be toxic, given its good absorption through the respiratory tract, skin and mucous surfaces[5].

The recent appearance of ECs, which are recharged with highly concentrated nicotine compound liquids, is a new source of potentially toxic nicotine doses. Different health authorities have reported that nicotine poisonings from these products are increasing alarmingly, especially among children. It is essential for the emergency physician to know the clinical and management of acute nicotine poisoning in the face of this novel source of poisoning[2].

Alberto Cózar-Llistó[1],
Manuel Pérez-Torres[2],
Fernando de Andrés-Moro[3],
Emilio Mencías-Rodríguez[4]

[1]Internal Medicine Department, Hospital Universitario Puerta de Hierro-Majadahonda, Madrid, Spain.
[2]Intensive Medicine Department, Hospital Universitario Puerta de Hierro-Majadahonda, Madrid, Spain.
[3]Emergency Department, Hospital Universitario Puerta de Hierro-Majadahonda, Madrid, Spain.
[4]Toxicological Information Unit, National Institute of Toxicology and Forensic Sciences, Madrid, Spain.

acozarll@gmail.com

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Hyperdense middle cerebral artery sign: traumatic dissection of the internal carotid artery

Signo de la arteria cerebral media hiperdensa en la disección traumática de la arteria carótida interna

To the editor:

The sign of the hyperdense middle cerebral artery[1] is found in 30-40% of patients with angiographically demonstrated middle cerebral artery occlusion[2]. It has a high specificity and is an early sign of ischemic stroke. It is a marker of poor prognosis[3], as it indicates an extensive infarction in the area of the middle cerebral artery[4].

A 25-year-old woman with no personal history of interest except being a smoker and without chronic home treatment. She came to the emergency department after suffering trauma with a sharp object in the Oropharyngeal region while riding on horseback with a pencil in her mouth. She was transported in an ambulance with stable basic life support, haemodynamically and asymptomatically, except for the pharyngeal wound. In the physical examination, the following stood out: score on the Glasgow Coma Scale of

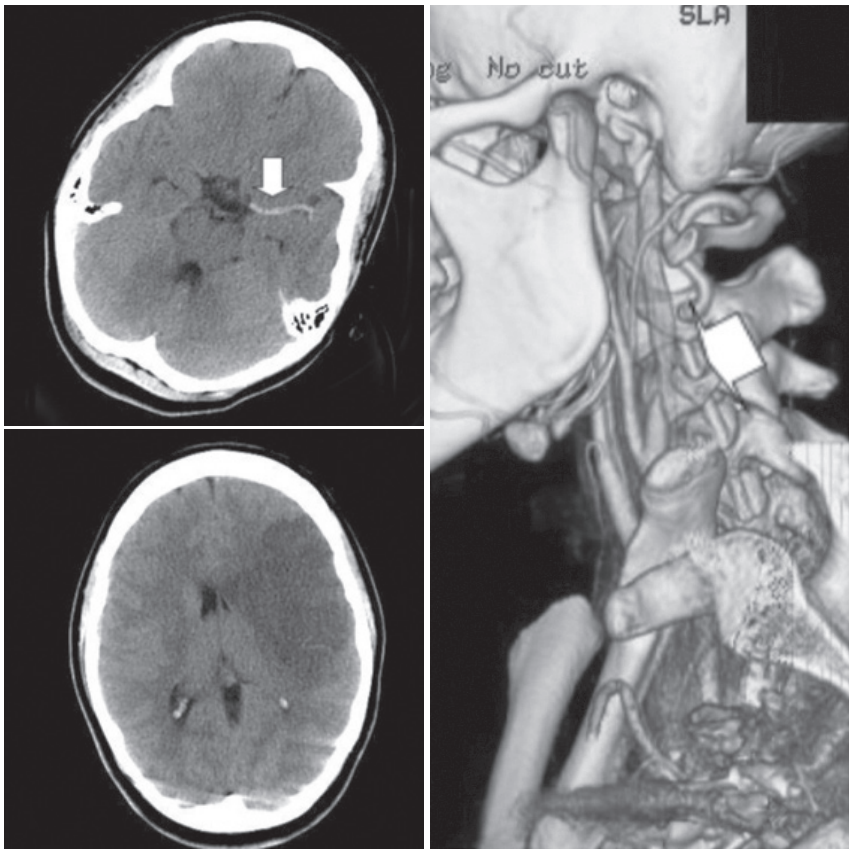

Figure 1. Top left: sign of hyperdense left middle cerebral artery from M1 to M3 (arrow). Bottom left: softening and image of ischemic edema in the left middle cerebral artery area. Right: dissection of the internal carotid artery (arrow).

15 points, without neurological focus and incised/contuse wound in the left sub-tonsillar region. During her stay in the department, she began right hemiparesis clinic with global aphasia, psychomotor agitation and decreased consciousness with a Glasgow Coma Scale score of 10 (O:3 M:5 V:2). In view of this symptomatology, a cranial computed tomography (CT) scan was performed and an image of hyperdensity was observed in the territory of the left middle cerebral artery indicating its entire route (M1 to M3) (Figure 1 left above) without other neuroradiological alterations. On suspicion of ischemic stroke, she was admitted to the stroke unit. The 24-hour control CT scan showed ischemic edema and softening (Figure 1 right below). Subsequently, CT angiography was performed where dissection of the left internal carotid artery with extension to the left middle cerebral artery was observed (Figure 1 right) with a discrete increase in the fat attenuation values of the left parapharyngeal space and retropharyngeal space. In this space only air is observed, up to vertebra C6. The etiological suspicion of stroke was related to a penetrating oropharyngeal trauma. Interventional vascular radiology was contacted and after angiographic study the existence of an arterial dissection of the internal carotid was confirmed with a significant reduction in caliber until almost its disappearance to approximately 1.5 centimeters from the bifurcation of the common carotid artery, without evidence of any permeability to the middle cerebral artery in its entirety, but since many of the collateral were competent, as well as the vertebro-basilar system that partially irrigated its territory, a conservative attitude was decided.

Dissection of the internal carotid artery is a common cause of stroke in the young population. Extracranial dissection is much more common than intracranial dissection and accounts for 90%. The pathogenic mechanism involved in most cases is craniocervical trauma. In terms of clinical manifestations, they are variable, and only 10-15% manifest as ischemic stroke. Other symptoms are headache, which appears in 70% of cases[5], Horner's sign and pulsating tinnitus, which may precede the establishment of the stroke between 5 and 10 days. The prognosis is variable and will depend on the severity of the stroke and the viability of the collateral circulation of the Willis polygon.

Germán Pérez Fajardo,
Elvira García Castro,
Aurora Sánchez González

Emergency Department, Hospital Universitario Nuestra Señora de Candelaria, Tenerife, Spain.

gperfaj@gmail.com

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Valproicacid–induced hyperammonemic encephalopathy in the emergency department

Encefalopatía hiperamoniémica inducida por ácido valproico en el servicio de urgencias

To the editor:

Hyperammonemia is an occasional adverse effect in patients undergoing treatment with valproic acid (VA). It is typically asymptomatic, although it may lead to hyperammonemic encephalopathy[1], a rare but potentially fatal complication, characterized by lethargy, vo-

miting, bradipsychia, focal neurological deficits, and decreased consciousness or coma. We now present a case.

A 55-year-old woman who came to the emergency department due to disconnection from her surroundings. She had a history of depression, bipolar disorder, and conversational episodes, and was institutionalized in a psychiatric hospital. She underwent treatment with valproic acid (1,000 mg daily), lorazepam, clotiapine, quetiapine and bupropion. On arrival at the emergency department, she had a blood pressure of 143/93 mmHg, a heart rate of 90 bpm, was afebrile and had an oxygen saturation of 97%. Cardiopulmonary and abdominal exploration was normal. The patient was conscious but disconnected from the environment with 11 points on the Glasgow Coma Scale, she only responded verbally with guttural sounds and did not respond to commands, but she did locate painful stimuli and sometimes fixed her gaze on the explorer. The threat reflex in both eyes was abolished and showed a loss of strength with claudication in Barré and Mingazzini of the four limbs in less than 10 seconds, and sometimes spontaneously performed uncontrolled movements of any limb against gravity. There were no alterations in the osteotendinous reflexes; and showed myoclonias in lower limbs. An analytical study was carried out with ions, hepatic profile and normal urine sediment, a chest x-ray that did not show any findings and a urine toxicity test was requested including opioids, cocaine, cannabis, amphetamines and benzodiazepines, which was positive for the latter. A CT scan of the skull without contrast was performed, which was normal, and plasma concentrations of VA were measured at 50 µg/ml (therapeutic range 50-120 µg/ml). Despite the initial diagnostic doubts with a conversive picture, without ignoring the suggestive findings of encephalopathy, blood ammonium concentrations were requested, which were 248 µmol/l (normal values: 9-30). In the observation room, the VA was suspended and hererotherapy started, with progressive and complete recovery of consciousness, language and mobility. After confirming normalization of ammonia concentration, the patient was discharged from the emergency department after 48 hours.

Hyperammonemic VA encephalopathy is a rare condition that requires a high degree of clinical suspicion for diagnosis. Its incidence and severity are not related to blood concentrations of the drug[2] and there is no linear correlation between ammonium concentration and encephalopathy grade[3]. It is potentially reversible with drug withdrawal, supportive treatment and antiencephalopathy measures. Treatment with L-carnitine[4], of which a starting dose of 100 mg/kg followed by an infusion of 50 mg/kg every 8 hours[5] is recommended, may be useful. The present case demonstrates the importance of the inclusion of this entity in the differential diagnosis of neurological symptoms in the emergency department in those patients treated with VA, as this can avoid invasive interventions and hospital admissions.

Raúl Pliego Cosano,
Juan Manuel Moreno Rosauro,
Marta Clavero Olmos

Emergency Department, Hospital Universitario Hospital Infanta Elena, Madrid, Spain.

jmmrosauro@gmail.com

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Lemierre syndrome due to Klebsiella pneumoniae

Síndrome de Lemierre por Klebsiella pneumoniae

To the editor:

Lemierre syndrome (LS) or necrobacillosis was originally described as the association of an oropharyngeal infection, bacteremia, septic thrombophlebitis of the internal jugular vein (STIYV) and septic metastases, mainly pulmonary and less frequently articular or of soft tissues[1,2]. It has also been described occasionally associated with sinusitis, mastoiditis, pyomyositis or dental infections. LS is an infection classically produced by Fusobacterium necrophorum[1,3-5]; however, cases caused by Streptococcus spp., Bacteroides spp., Eikenella corrodens, Enterococcus spp. among others have also been reported. The following is a case of LS produced by Klebsiella pneumoniae.

A 31-year-old woman, no previous known history, who had been treated with amoxicillin-clavulanate for otalgia, bilateral otorrhea and pharyngeal discomfort for two weeks, without being able to complete treatment for incoercible vomiting, oral intolerance and abdominal discomfort. She came to the emergency department with a low level of consciousness and fever. Diabetic ketoacidosis (pH 7, 02, blood glucose 930 mg/dl, HbA1 9.5%) was objected to, and treatment with intravenous insulin was initiated. Chest X-ray showed no alterations and in the analysis the leukocyte count was 43,180 leukocytes/µl (93% PMN), a procalcitonin of 7.2 mg/l (0-0.5 mg/l) and a renal function alteration with creatinine of 2 mg/dl. Despite normalization, the patient continued to be bradipsychic and disoriented, which is why a cranial computed tomography (CT) scan was performed, in which bilateral otomastoiditis was observed, associated with abscess in the right retroauricular region with mastoid and epidural extension to the right posterior fossa and right septic thrombophlebitis suggestive of LS (Figure 1). Drainage and treatment with meropenem 1 g/8 h iv was performed. Klebsiella pneumoniae (with decreased sensitivity to quinolones) was isolated from blood cultures and abscess culture. Ceftriaxone 2 g/24 h treatment was de-escalated until a total period of 42 days was completed. Bemiparine treatment (5,000 IU/day) and, subsequently, acenocoumarol was used until the resolution of the picture.

LS has an incidence of 0.6-2.3 million cases/year and a mortality rate of 4-18%[3]. A high suspicion and

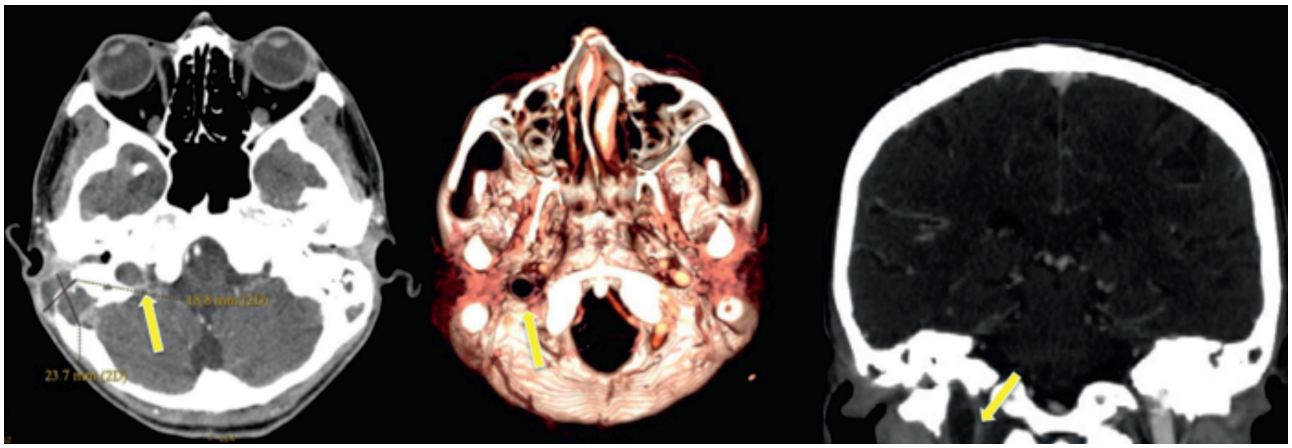

Figure 1. Dimensions of the right mastoid abscess (left image, arrow). Septic thrombophlebitis of the internal jugular vein (central and right images, arrows).

an imaging test such as ultrasonography, magnetic resonance or CT are essential for its diagnosis. Among the cases of STIYV by Klebsiella pneumoniae described, the incidence is similar in both sexes, and mainly affects adolescents and young adults[5]. In many published cases, the patients were diabetics with poor glycaemic control, positive blood cultures in 54% and positive purulent exudate cultures in 90.9%. There is controversy regarding the need for anticoagulation for these patients, which should perhaps be limited to those cases where thrombotic progression exists. However, most of the cases described were anticoagulated. In all but one case[5], the final outcome was a cure after an average period of 42 days of treatment. We conclude that in diabetic patients with septic picture and infection in one of the mentioned outbreaks, the possibility of an LS should be considered and, therefore, a diagnostic imaging test should be performed. Likewise, in addition to the infections classically associated with LS, in diabetic patients the empirical antibiotic treatment should take into account the possibility of infection by Klebsiella pneumoniae, with strains producing extended spectrum betalactamases, in 10-30% of cases[6].

Miguel Angel López Zúñiga[1],
Concepción López-Robles[2],
María Concepción Fernández-Roldán[2],
Antonio Tapia-Gómez[2]

[1]*Internal Medicine Department. Complejo Hospitalario de Jaén, Spain.*
[2]*Infectious Diseases Department. Hospital Virgen de las Nieves, Granada, Spain.*

miguelangellopezzuniga@gmail.com

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On the usefulness of N-terminal prohormone of brain natriuretic peptide for the diagnosis of incomplete Kawasaki disease

Reflexiones sobre la utilidad de NT-proBNP en el diagnóstico de la enfermedad de Kawasaki incompleta

To the editor:

We have read very carefully the paper written by Rodríguez-González et al.[1], published in the last issue of this journal. The conclusion reached by the authors when observing that NT-proBNP concentration in plasma could be a valid diagnostic biomarker in incomplete Kawasaki disease (KD) in a pediatric population is very interesting.

NT-proBNP increases in the acute phase of the KD, but the cause of its elevation is not clear. It also elevates in other inflammatory substrate diseases, such as community-acquired pneumonia[2]. Although the etiology of KD is not fully identified, epidemiological evidence points to infection and genetic predisposition[3]. It seems congruent to think that a systemic disease that generates vasculitis in medium calibre vessels, with secretion of pro-inflammatory cytokines possibly in the context of an altered immune response to infection, may develop cardiovascular stress, increased tension in the wall of the myocardiocytes with synthesis and release of natriuretic peptides by modification of peripheral vascular resistances. NT-proBNP could also be elevated by KD complications such as myocarditis, pericarditis, valvular

insufficiency, ventricular dysfunction and the most devastating complication, coronary artery disease with aneurysmal dilatation and coronary thrombosis.

We would have liked to know the initial NT-proBNP concentrations in the five patients who developed coronary aneurysm compared to the rest of the population diagnosed with incomplete KD. This data seems relevant to us, since its values could guide on the potential severity.

We understand that Rodríguez-González et al.[1] assume the cut-off points of McNeal-Davidson et al.[4] which, in turn, are based on a study of the Israeli and German populations[5]. McNeal-Davidson et al. explore three different criteria for choosing the cut-off point that will define the positivity of the NT-proBNP test: based on the analysis of ROC curves, 95 percentile (p95) according to age and values greater than 2 for the Z value for age. The latter were used by Rodriguez-González et al. Due to the greater sensitivity of the criterion based on p95 compared to Z > 2.0 (80.2 vs 70.4), we suggest using in clinical practice the cut-off points based on p95 as proposed by Nir et al.[5], since given the potential severity of the non-diagnosis, we think that sensitivity should be prioritized over the specificity of the test. Since it is not possible to rule out that KD is associated with a genetic component, perhaps a study with a local population that reconsiders or confirms these cut-off points would be appropriate.

The heading "Other febrile diseases" refers to etiological agents, but does not specify the specific nosological entities. If one of them potentially increases NT-proBNP concentrations, it could act as a confounding factor.

The article by Rodríguez-González et al. seems to us to be an important approach through NT-proBNP to a substrate disease that is still unclear. Perhaps in this entity, which is the most common cause of acquired cardiopathy in children from developed countries, this and other biomarkers may shed light in the future to clarify its aetiology, diagnosis and prognosis.

Manuel Antonio Tazón-Varela[1,2],
Héctor Alonso-Valle[1,3],
Pedro Muñoz-Cacho[1,4]

[1]*Community Health Group, Valdecilla Health Research Institute (IDIVAL), Santander, Cantabria, Spain.*
[2]*Emergency Department, Laredo Hospital, Laredo, Cantabria, Spain.*
[3]*Emergency Department, Marqués de Valdecilla University Hospital, Santander, Cantabria, Spain.*
[4]*Teaching Unit of the Primary Care Management, Cantabrian Health Service, Santander, Cantabria, Spain.*

hectoravt@telefonica.net

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Authors' reply

Respuesta de los autores

To the editor:

We appreciate the interest shown by Tazón-Varela et al. and their critical reading of our article[1].

We would like to clarify that the exclusion criteria described in the methodology prevented the inclusion of diseases that could raise NT-proBNP. In addition, the patients who developed coronary aneurysm were not 5, but 3 in acute phase, persisting in 2 of them at 12 months. Both cases had NT-proBNP concentrations above 2 for Z for their age (2,714 and 5,063 pg/ml for 4 years and 6 months, respectively), and required a second dose of gamma globulins. Although NT-proBNP elevation in Kawasaki disease (KD) appears to be multifactorial, inflammation would play a major role[2]. Recently NT-proBNP has been suggested as a marker of TNF-D activity in KD, the most important pro-inflammatory cytokine in its pathophysiology[2]. In addition, the development of coronary aneurysms and refractoriness to gamma globulins has been associated with higher concentrations of NT-proBNP[3], which would reflect a high degree of inflammatory activity in KD and could have a role as a prognostic biomarker yet to be defined.

Table 1. VNT-proBNP cut-off values for Kawasaki disease

Age of patients	Value of plasma NT-proBNP (pg/ml)
1-11 months	1,000
1-2 years	900
2-3 years	800
3-4 years	700
4-6 years	600
6-8 years	500
8-10 years	400
10-15 years	300

These NT-proBNP values correspond to a Z = 2 value for age. They are easy to remember and have a sensitivity of 43.6% and a specificity of 98% for the diagnosis of Kawasaki disease[5].

The choice of a suitable cut-off point for the diagnosis of KD must be adjusted to the age of the patient[4]. For the study, we used McNeal's complex formula to determine the Z-value > 2 for age. Shiraishi et al.[5] propose other cutting points that are used in our center because of their easy applicability to clinical practice (Table 1). Both methods stand out for their high specificity (98 and 91,8). We understand the appreciation of the authors regarding the search for a high sensitivity that minimizes non-diagnosis. However, we believe that the main limitation of the current KD criteria is precisely that they are nonspecific4. In our experience, due to the potential seriousness of non-diagnosis, the threshold for treating patients with suspected KD is usually low, which sometimes leads to unnecessary administration of gamma globulins to patients without KD. Therefore, the greatest value of NT-proBNP in this

context would be its ability to confirm the picture in preselected patients using current criteria, having to use very specific cut-off points. In centres with little experience in the recognition of KD we would find it advisable to use values with high sensitivity.

Finally, due to the low prevalence of KD, prospective and multicentre studies would be necessary to confirm our results and establish reference values for NT-proBNP and its prognostic capacity in the local population.

Moisés Rodríguez-González[1],
Ana Castellano-Martínez[2],
Álvaro Antonio Pérez-Reviriego[1],
Almudena Alonso-Ojembarrena[3]

[1]UPediatrics Clinical Management Unit, Department of Paediatric Cardiology, Puerta del Mar University Hospital, Cadiz, Spain.
[2]Paediatric Clinical Management Unit, Department of Paediatric Nephrology, Puerta del Mar University Hospital, Cadiz, Spain.
[3]Pediatrics Clinical Management Unit, Department of Neonatology, Puerta del Mar University Hospital, Cadiz, Spain.

doctormoisesrodriguez@gmail.com

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Severe hyponatremia after a purification ritual using an Amazonian frog poison (Kambô)

Hiponatremia grave por ritual de purificación con veneno de rana amazónica (Kambô)

To the editor:

In Western Europe, natural medicine has become popular and purification rituals are becoming more popular. Traditionally, the ceremony of the large monkey frog, Phyllomedusa bicolor or Kambô, is performed by Amazonian indigenous people, mainly in northwestern Brazil and the border with Peru. Its purpose is to propitiate hunting, purify the body or treat diseases[1]. The shaman, after exposing the amphibian to heat, scrapes its skin with a spatula. Thus he obtains a secretion called Kambô, which he dries and stores. This secretion can be commercialized in this format. This juice is rich in various bioactive peptides. As a previous preparation, the participant drinks water. The shaman then burns one limb superficially and applies Kambô[1,2] (a quantity adjusted according to weight). Absorption is immediate and the effects are noticeable within a few minutes, some of them are lethal[2-4]. We describe a case of severe hyponatremia by Kambô in an urban environment.

A woman with no previous history of interest who was brought to the emergency department after participating in the Kambô ritual. Previously, she had consumed 2 liters of water and fasted for 12 hours. The shaman had made superficial burns on her ankle (Figure 1) to apply the secretion of Phyllomedusa bicolor (Figure 2). A few minutes later she vomited and had liquid stools that self-limited. Subsequently, she ingested 1.5 liters of water and rehydration serum. Six hours later she began a picture of disorientation with incoherent language. At 10 a.m. she suffered a generalized tonic crisis, so the emergency services were alerted.

The first assessment documented a Glasgow Coma Scale (GCS) score of 9 attributed to the post-critical state. During transfer, she received 2 mg of midazolam intravenously by psychomotor agitation. On arrival at the emergency department, 11 hours after the rite, her vital signs were: blood pressure 110/74 mmHg, heart rate 110 bpm, respiratory rate 14 brpm and oxygen saturation of 100% (FiO2 21%). It persisted with a GCS score of 9 (O1 + V3 + M5), alternating with episodes of psychomotor agitation. On physical examination, the pupils were mildly midriatic, but reactive, and showed acute urine retention. In the first analysis the following stood out: pH 7.38, bicarbonate 22 mmol/l, lactate 57.9 mg/dl, CK 296 UI/l, osmolarity 240 mOsm/kg with natremia of 118 mEq/l, kaliemia 2.4 mEq/l, calcemia 7.3 mg/dl and magnesemia 1.2 mg/dl. Urinary osmolarity was 277 mOsm/kg and recent urine natriuresis was 100 mEq/l. An emergency screening for drugs of abuse was carried out, which was negative. The electrocardiogram showed a sinus rhythm with a QTc interval of 480 ms.

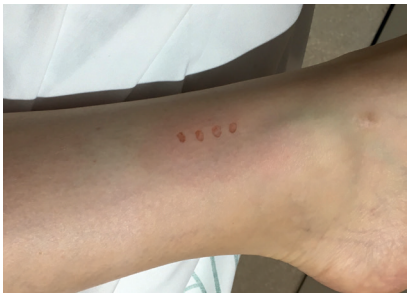

Figure 1. Puntiform and aligned wounds found in the external supramaleolar area of the left lower extremity, consistent with superficial burns where Kambô would have been applied.

Figure 2. Phyllomedusa bicolor or large monkey frog.

Based on the findings and the clinical context, it was treated as a comitology crisis for severe hyponatremia secondary to syndrome of inadequate antidiuretic hormone secretion, excessive losses and dilution. Sodium chloride was initiated at 2% for 4 hours (1.5 ml/kg/h). Also, intravenous administration of 10% calcium gluconate (one 4.6 mEq ampoule), magnesium sulphate (1.5 g) and potassium chloride (20 mEq in 3 hours). She was transferred to the intensive care unit. To control the agitation, dexmedetomidine was used (up to 1.17 µg/kg/h). Four hours after starting the treatment, the natremia was 131 mEq/l and the rest of the ions were corrected. The degree of consciousness was optimal at 22 hours and she only had amnesia of previous events. The study was completed with an electroencephalogram, which showed a diffuse slowdown of brain activity, and a

cranial CT scan that was normal. The only aspect to highlight during recovery was a transient elevation of CK (peak of 1,064 IU/l). On the fifth day she was discharged from hospital.

The effects of Kambô are due to the bioactive peptides contained in the secretion of the frog. At present, numerous have been identified: phyllomedusin and phylloquinin, vasodilators[1,5]; phyllocaerulein and sauvagin, hypotensors[1,2]; dermorphins and deltorphins, potent opioids[1,6]; adenoregulin and phylloseptin B-2, anti-inflammatories and antimicrobials[1,5]; and finally, dermaseptin B2 and antitumor[7]. The rapid absorption of these molecules is responsible for their effects.

Adverse reactions reported to date are scarce; however, their variability is very notable: hyponatremia[3], acute hepatitis4, emetic syndrome[8], psychomotor agitation9 and death[10]. In addition, a broad spectrum of severity has been documented in terms of its appearance. In our case, we expose a severe hyponatremia with important neurological repercussions and the need for pharmacological containment due to psychomotor agitation.

In countries where the tradition of healing rituals is more entrenched, legal measures have been initiated to prevent the sale and advertising of substances with "purifying powers". However, the lack of knowledge about these preparations in Europe could lead to harmful and even lethal effects on the health of the individuals who are exposed to them. In order to be able to develop joint lines of action to address their dangerous and growing popularisation, the declaration and collection of all cases is paramount.

Daiana L. Agüero-González[1,2],
Adriana Pané-Vila[1],
Víctor Gil[1],
Pedro Castro[2,3]

[1]SEmergency Department, Hospital Clínic, Barcelona, Spain.
[2]Intensive Surveillance Area, Hospital Clínic, Barcelona, Spain.
[3]Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Universitat de Barcelona, Barcelona, Spain.

dlaguero@clinic.cat

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Poisoning by Chinese rocks: fatal outcome of a suicide attempt

Intoxicación letal por piedras chinas con ideación suicida

To the editor:

Toads have been used for ritual and medicinal purposes since ancient times, particularly in Asian and Mesoamerican countries, but the skin, parotid glands and eggs of certain species contain alkaloids and cardioactive steroids[1] that can produce intoxication similar to digitalis. In addition, there is a preparation in the form of a dry extract obtained from toad secretions that is used for sexual purposes. This extract, called love stone, or Jamaican stone, is applied to the ventral side of the glans, producing an anaesthetic effect that delays ejaculation. It is a resinous substance of approximately 1 cm^2. After application, hands should be washed and oral sex avoided. This is the first case reported in Spain of lethal poisoning with suicidal intent by this substance.

A 23-year-old woman who came to a primary care center for nausea, vomiting, anxiety and agitation following the ingestion of a substance called Chinese rocks with suicidal ideation three hours earlier. She had PA 153/70 mmHg, FC 55 bpm, basal oxygen saturation 99%, normal body temperature and capillary glycaemia 122 mg/dl. An electrocardiogram showed sinus bradycardia at 50 bpm. It was decided to transfer by conventional ambulance to our center, during which severe deterioration occurred. She arrived with signs of poor perfusion and preagonal breathing. The first rhythm recorded was asystole, and advanced cardiopulmonary resuscitation manoeuvres were initiated, which lasted 60 minutes, without achieving a proper rhythm. K 7.76 mmol/l (VN: 3.5-5.1 mmol/l) stood out in the analysis. No digoxin determination was requested. Judicial necropsy was performed, which revealed generalized congestion, edema and diffuse pulmonary hemorrhage. Microscopic findings confirmed these findings. The presence of digoxin was detected by enzymeimmunoassay (Indiko Plus, Thermo Scientific, DRI®) in concentrations of 0.004 mg/l. Caffeine and paracetamol were also detected in blood and tetracaine in bladder washing through chromatographic techniques. No bufotenin or other substances were detected.

Cardioactive steroids found in animals are called bufadienolides while those found in plants are called cardenolides[2]. Buffadienolides are extracted from the cutaneous secretions of some species of toads of the genus Bufo. As they are structurally similar to digital molecules, concentrations of digoxin can be detected in the blood of patients who have consumed them. Some cases of fatal poisoning by these substances have been described[1,3,4]: one case after intravenous administration, in a patient who believed that it was ecstasy[4], the other cases after oral ingestion[1,3]; two of these poisonings presented bradycardia and hyperpotasemia[1,3]. Necropsy was performed in only one, which showed nonspecific findings[4], similar

to the necropsy performed on our patient. This makes it difficult to diagnose by necropsy, so it is necessary to identify digoxin and, preferably, bufadienolides in the samples analyzed.

Our hypothesis on the evolution of the patient is that after the initial bradycardia, a ventricular tachycardia appeared during the transfer to our center, followed by ventricular fibrillation and asystole. The administration of antidigital antibodies could be effective in the treatment of intoxications by bufadienolides, but due to the rapid outcome, they could not be administered.

August Supervía[1],
Ignasi Galtés[2],
Julián Errasti[3],
María Barbal[4],
Patricia Gallardo[1],
Isabel Cirera[1]

[1]*Unitat Funcional de Toxicologia, Emergency Department, Hospital Universitario del Mar, Barcelona, Spain.*
[2]*Department of Forensic Pathology, Institut de Medicina Legal i Ciències Forenses de Catalunya, Barcelona, Spain.*
[3]*Emergency Department, Hospital Universitario del Mar, Barcelona, Spain.*
[4]*Department of Forensic Laboratory, Institut de Medicina Legal i Ciències Forenses de Catalunya, Barcelona, Spain.*

asupervia@parcdesalutmar.cat

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Still with neither a specialization nor a spokesperson

Seguimos sin especialidad y sin interlocutor

To the editor:

Please allow us a few lines, written shortly after learning that Parliament is dissolved again and new general elections are called, to denounce the paralysis in which the development of our specialty, Emergency Medicine (EM), has entered in recent years. Following the annulment of Royal Decree 639/2014 of 25 July, which relegated training in our speciality to a specific training area, several ministers have taken over without having achieved the much-needed regulation of primary speciality training in EM via MIR. We will not repeat here the benefits and reasons that assist not only our group of emergency physicians, but also the health system and the general population, so that this legislation is produced without further delay. Ministerial changes do not contribute to the solution of the problem, and the delay in adopting such a solution deepens it. The Spanish Society of Emergency Medicine (SEMES) will once again offer its experience and help to its next tenant. In the meantime, we will continue promoting and disseminating the knowledge, training and research required by our professionals in Spain[1-5], which in most European countries[6] is carried out during a period of regulated specialisation.

Manuel J. Vázquez Lima[1],
Juan J. González Armengol[2]

[1]*Vice-president of the Spanish Society of Emergency Medicine.*
[2]*President Spanish Society of Emergency Medicine.*

mvazlim@gmail.com

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