LETTERS TO THE EDITOR

Myocarditis associated with the use of energy drinks: a case report

Miocarditis relacionada con el uso de bebidas energéticas: a propósito de un caso

To the editor:

The consumption of energy drinks has increased over the last two decades, and with it the number of emergency consultations due to their adverse effects[1]. Energy drinks generate multiple side effects on the cardiovascular system due to the high caffeine content. Most consumers are young men, and there is also a tendency to associate this consumption with alcoholic beverages[2].

Cans of energy drinks contain approximately 152-160 mg of caffeine, and both acute consumption (> 480 mg of caffeine in a day) and chronic consumption (> 200 mg of caffeine daily, for more than one week) have been associated with adverse cardiovascular events[1]. In addition, plasma catecholamines have been reported to increase at doses of caffeine > 250 mg and may even increase further at rapid and excessive consumption of energy drinks[1]. The excess of catecholamines stimulated by caffeine leads to increased heart rate and blood pressure secondary to increased systemic vascular resistance and positive inotropism generated by increased intracellular cyclic MPA[2,3]. Taurine, present in these energy drinks, is known to have a positive inotropic and arterial hypotensive effect by attenuation of angiotensin II. However, this does not appear to be sufficient to counteract the sympathomimetic activity of caffeine[2].

This is a case of a 45-year-old male who came to the ED for dyspnea. As a background he had a smoking habit, he was not a regular user of alcoholic beverages or other drugs. He referred during the previous week to minimal effort dyspnea, orthopnea and bendopnea. He denied chest pain, syncopes and did not present fever or infectious semiology in the previous days. In the directed anamnesis, a daily consumption of 1-2 cans of energy drinks (355- 710 ml) was identified for 6 months due to its working conditions. Physical examination showed: heart rate at 110 beats per minute (bpm), blood pressure 100/70 mmHg, respiratory rate at 24 brpm, temperature 36.5ºC, basal oxygen saturation of 92%, arrhythmic heart sounds, without murmurs or pericardial rubbing, with third noise, and lung crackles. Chest radiology showed a cardiothoracic index greater than 0.5 and interstitial edema. The electrocardiogram showed atrial fibrillation with mean ventricular rate at 110 bpm, QRS axis at -30º, with diffuse disorders of ventricular repolarization. The study of coagulation, hemogram, thyroid hormones, liver function and glomerular filtration were normal. The presence of ethanol in blood and illegal drugs (cannabis, cocaine and opiates) in urine were ruled out. The immunological study (antinuclear antibodies, rheumatoid factor), as well as the serological study for viruses (enterovirus, adenovirus, parvovirus B19, human herpes virus type 6, Epstein-Barr virus Influenza A/B, HIV) and bacteria (*Mycobacteria, Chlamydia, Mycoplasma, Streptococci*), were negative. He presented elevation of acute phase reactants (PCR = 100 mg/dl, normal < 3 mg/dl; fibrinogen = 6.5 g/L, normal = 1.9-4.3 g/L), conventional cardiac troponin I (4,050 ng/mL, normal = 0.03-0.08 ng/mL) and natriuretic peptide (2,650 pg/mL). Transthoracic echocardiography revealed an enormously dilated left ventricle with global hypokinesia and a 25% left ventricular ejection fraction. After deplective treatment with furosemide, vasodilator with nitroglycerin, inotropic with levosimendan and anticoagulation with enoxyparin, the patient gradually improved. A cardiac catheterization was performed, which ruled out coronary artery disease. On the fourth day of admission, magnetic resonance imaging with gadolinium was performed, which showed edema and late intramyocardial gadolinium uptake at the inferolateral level, with a 30% ejection fraction from the left ventricle (Figure 1). In the absence of an autoimmune, infectious or inflammatory trigger, and applying Naranjo's algorithm4 to analyze the causal relationship between energy drinks intake and the appearance of the adverse reaction, a "probable" relationship was obtained, with the diagnosis of acute myocarditis in relation to energy drinks intake. In its subsequent evolution, after 6 months of treatment for heart failure with severe left ventricular systolic dysfunction and absence of energy drinks intake, left ventricular systolic function recovered its ejection fraction (60%).

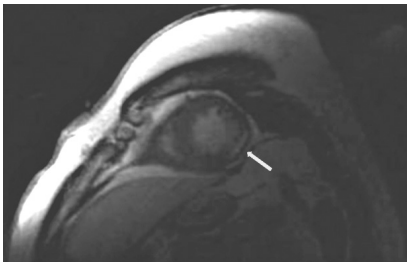

Figure 1. Magnetic Resonance Imaging, short axis apical projection. Inferolateral, late enhancement with gadolinium (white arrow).

Infections, drugs, autoimmune and toxic diseases can trigger acute myocarditis, and viral infections are the most common aetiology[5]. After a systematic review of the literature in the database of the National Library of Medicine of the USA, no case of myocarditis associated with energy drink consumption has been described. The cardiovascular events that have been described as associated with energy drinks are: supraventricular and ventricular arrhythmias, coronary vasospasm, aortic dissection and acute intracoronary thrombosis, among others[2]. Grasser et al. demonstrated that polymorphic alterations in genes related to cytochrome CYP1A2 involved in caffeine metabolism could intervene in molecular and cellular changes and lead to anomalies in cardiac chamber geometry and ventricular modeling.

Therefore, from our point of view, the combination of compounds contained in these energy drinks can produce a much higher cardiovascular risk, by generating uncertain interactions, than any component alone. Furthermore, the potential for dependence and abstinence after regular consumption of energy drinks has been demonstrated, in addition to anxiety, aggression and sleep disorders[6]. This case shows the importance of asking in the ED anamnesis about energy drinks consumption, given the key role that these services have in detecting clinical events related to the intake of substances of abuse[7-10].

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Thoracic tamponade: as a diagnostic challenge

El taponamiento torácico como desafío diagnóstico

To the editor:

Cardiac tamponade is a clinical syndrome generally secondary to pericardial effusion. However, a massive pleural effusion is capable of causing cardiac tamponade in the absence of pericardial effusion[1,2].

This study presents the case of a 42-year-old man with no relevant medical history, who was admitted to the emergency department with pleural effusion. While under observation, the patient suddenly reported poor general condition and dyspnea (7 points, Borg scale) and tachycardia (140 bpm), tachypnea (> 30 rpm) and hypotension (80/50 mmHg) with significant jugular vein inflammation and right lung hypophonesis. Due to clinical deterioration, orotracheal intubation and mechanical ventilation were implemented. The echocardiogram showed the collapse of the right atrium, with low filling velocities and respiratory variation greater than 25%. Pericardial effusion was ruled out, and the presence of massive pleural effusion was confirmed. With the change in diagnostic orientation, an urgent thoracentesis was performed, with an initial drainage of 700 ml (total 3,500 ml) which resulted in a decrease in right atrial collapse, and normalization of hemodynamic status. Finally, a thoracic tamponade caused by pleural effusion was confirmed as a complication of mesothelioma. The patient was able to continue his oncological treatment.

Thoracic tamponade is a similar medical condition to cardiac tamponade, but it is derived from pleural effusion[3]. Physiopathologically, the transmission of intrapleural pressure to the pericardial space is theorized, even without effusion in the latter, resulting in collapse and decreased filling of the right chambers, a highly sensitive (100%) and specific (82%) finding in cardiac tamponade (Figure 1)[2,4].

Although transthoracic echocardiography was the choice for this patient, a portable transthoracic bedside device was used which did not allow the recording of all images. Although the described pattern of tamponade is most often seen in the context of pericardial effusion, pleural effusion should be considered in the differential diagnosis, as the therapeutic strategy is clearly different. In addition, analysis of the vena cava, which is usually dilated without respiratory variation due to increased pressure in the right atrium, and pleural examination should not be overlooked in the emergency examination.

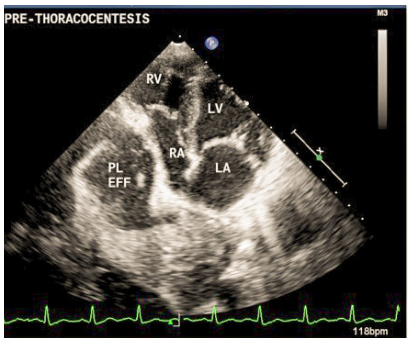

Figure 1. Image of intrapleural spill resulting in collapse and decreased filling of right chambers, taken from Mahajan K[2].

Although thoracic tamponade secondary to pleural effusion is rare, it should be considered as a differential diagnosis in emergency departments with cardiac tamponade. This will allow the avoidance of harsh therapeutic strategies, such as emergency pericardiocentesis, and the alternative use of thoracocentesis, which in this case was the appropriate option[5].

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Erythema multiforme induced by topical imiquimod

Eritema multiforme inducido por imiquimod tópico

Sr. Editor:

Imiquimod is a topical immunomodulatory agent approved as an alternative to surgery for the treatment of low-risk basal cell carcinomas in immunocompetent patients. It is a well-tolerated drug and when adverse effects occur they are strictly to the skin.

This is the case of a multiform erythema associated with the administration of topical imiquimod, which occurred in an 84-year-old male with a history of high blood pressure and atrial fibrillation who was treated with losartan 100 mg per day and rivaroxaban 20 mg per day. He was diagnosed with a basal cell carcinoma in the left temporal region, and it was decided to administer 5% topical imiquimod three times every seven days. Four weeks after starting treatment, he visited the emergency department for skin lesions distributed in the trunk and upper extremities. In addition, he presented a general condition of fever, dry cough, dysphagia and conjunctival pruritus. On examination, vital signs were normal. On auscultation, cardiac tones were arrhythmic and vesicular murmur was preserved. Erythematous-violet macules with vesicular center and pustules (Figure 1) were observed, predominantly in the face, neckline and upper limbs, accompanied by meliceric scabs and pustules on the oral mucosa and lips, as well as erythematous papules on the glans. In the left temporal region, area of application (Figures 1 and 2), there was a large erythematous area with scabs, which when detached exposed erosions. The hemoglobin value was 13.5 g/dl, the leukocyte count was 6.8 x 10^3/µl and the platelet count was 162 x 10^3/µl. The coagulation study showed an INR (international normalised ratio) of 2.23 and the biochemical profile showed a C-reactive protein of 25 mg/l. Serologies for *Mycoplasma pneumoniae*

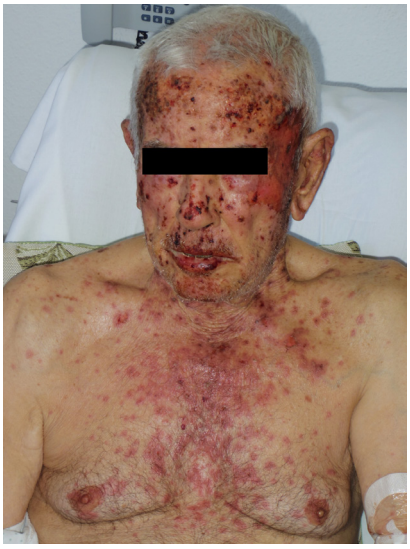

Figure 1. Erythema-violate macules with vesicular and pustular centre. Extensive erythema with scabs in left temporal region.

and herpes group viruses did not show data of acute infection. Erosive epidermal necrosis with necrotizing folliculitis was reported in the skin biopsy. In addition to topical treatment and removal of imiquimod, the patient received intravenous boluses of 125 mg of methylprednisolone every 8 hours for 3 days and then prednisone 60 mg per day for 10 days. The evolution was favorable, with resolution of skin lesions in about 3 weeks.

Imiquimod is a topical agent used in the treatment of basal cell carcinomas, actinic keratoses and genital warts[1,2]. It acts as an anti-tumor and antiviral agent by different mechanisms that exaggerate the immune response, including agonist activity on TLR7 and TLR8 (Toll Like Receptor) receptors, cytokine release and lymphocyte stimulation[2]. Local reactions, such as erythema, itching, burning sensation, erosions and excoriations, represent the most frequent adverse effects. Systemic reactions are rare, as imiquimod absorption through the entire skin is minimal. Muscle weakness, myalgia, headache and flu-like symptoms, among others, have been described[3]. Erythema multiforme in relation to topical administration of imiquimod has been reported exceptionally. The appearance of extensive local reactions has been suggested to favour, as in the case described, greater systemic absorption of the drug and an immune-mediated inflammatory response[4]. It is important to point out that, despite its topical use, dermatological emergencies such as the erythema multiforme described can be observe[5].

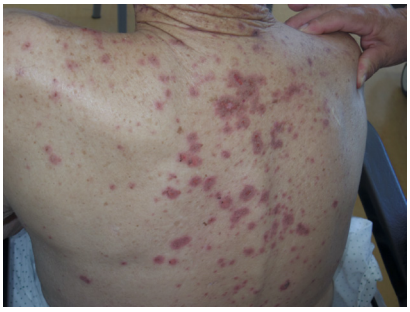

Figure 2. Erythemate-violate macules on back, some of them with vesicular center and others with a pustule inside

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Capillary leak syndrome: often forgotten in differential diagnosis

El síndrome de fuga capilar: el gran olvidado en el diagnóstico diferencial

To the editor:

Idiopathic systemic capillary leak syndrome, also known as vascular hypermeability syndrome or Clarkson's syndrome, is a rare entity with less than 500 cases reported since 1960, characterized by the extreme hypotension, hemoconcentration and hypoalbuminemia triad[1-3]. The severity of these conditions varies and can be fatal. Outbreaks may be preceded by infections, especially respiratory, suggesting the role of inflammation in their origin.

A 56-year-old woman with no history of interest who attended the emergency department for asthenia, nausea and vomiting of acute onset. Physical examination showed hemodynamic instability with undetectable blood pressure and poor peripheral perfusion. The analytical study highlighted the presence of acute renal failure with a glomerular filtrate of 3.2 ml/min, hypoproteinemia (4.5 g/dl) with hypoalbuminemia (2.1 g/dl), hemoconcentration (Hb 21.4 g/dL, Hto 65.8%) and metabolic acidosis with pH 7.16 and HCO_{3-} 11.5 mmol/l. She was admitted to the intensive care unit (ICU) and underwent fluid therapy, extraction of 900 ml of blood (bleeding) and empirical antimicrobial treatment, with both clinical and analytical improvement without the need for vasoactive drugs. During admission, only the identification of a monoclonal IgG gammopathy of uncertain origin stood out. Four months after discharge, the patient presented a similar clinical picture to the previous one, with the presence of generalized edema. Intensive therapy with vasoactive drugs, fluid therapy and albumin replacement was required. The infection and autoimmune markers were negative, except for the persistence of monoclonal gammopathy, a differential element with the cases described in children, where its presence has not been observed in published series[4]. After ruling out other entities, it was classified as idiopathic systemic capillary leak syndrome, vascular hypermeability syndrome or Clarkson's syndrom[1].

Systemic capillary leak syndrome usually appears in middle-aged patients, with no sex preference. The etiology remains unknown. Different theories about the etiopathogenesis of the entity are postulated, although it is recommended to rule out an acute infectious process (sepsis) in the acute phase. Its diagnosis is a challenge for the clinician because it is infrequent and unknown within the differential diagnosis of shock. Clinically, it is characterized by three phases: a prodromal phase with nonspecific symptoms such as fatigue, abdominal pain, nausea and myalgia; a second with fluid extravasation where the characteristic triad of severe hypotension, hypoalbuminemia (< 3 g/dl) and hemoconcentration is observed; and finally the phase of recovery or fluid recruitment, with massive absorption of the edema with polyuria and weight loss[4]. It is often accompanied by the finding of a monoclonal gammopathy that supports the diagnosis. Survival can be 70% at 5 years with prophylactic treatment[5].

Treatment can be divided into two phases. The acute phase requires treatment of hemodynamic instability and would be equivalent to the management of distributive shock, with monitoring of the evolution of peripheral and different organ edema (myocardium, kidney) and treatment of possible complications (venous thrombosis, myocardial contractionary dysfunction, metabolic acidosis, among others). The preventive phase corresponds to monthly infusions of intravenous immunoglobulins at a dose of 2 g/kg. In patients who do not tolerate it, a combination of theophylline (10-20 mcg/ml) and terbutaline (5 mg/6 hours) is suggested[5]. We believe that this syndrome should be included in the differential diagnosis of shock, as it is a little-known entity that is difficult to manage in the acute phase, with possible relapses without prophylactic treatment.

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Emergency extracorporeal membrane oxygenation in a case of fatal acute poisoning by solvent intake

Intoxicación aguda mortal por ingesta de disolvente con indicación emergente de oxigenación por membrana extracorpórea

To the editor:

The use of extracorporeal membrane oxygenation (ECMO) in intoxicated patients has been documented for more than a decade and, in recent years, has been considered a therapeutic option in severe drug and other toxic poisoning with particular toxic potential and damage to the respiratory and cardiovascular systems[1]. The early indication of ECMO in cases of cardiogenic shock or toxin-induced adult respiratory distress syndrome, as in the case reported, significantly improves survival rates[2,3].

A 38-year-old woman with a psychiatric history of schizophrenia treated with

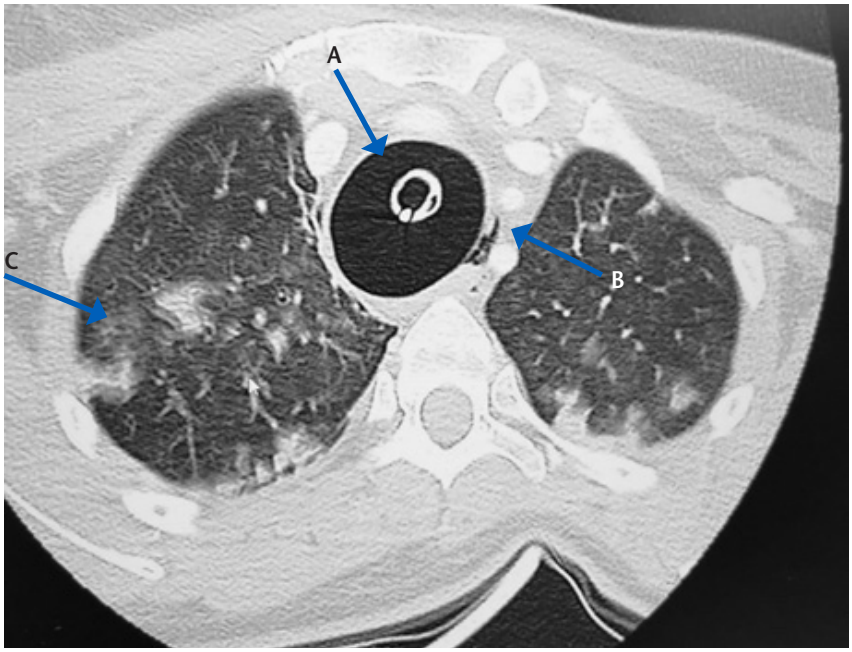

Figure 1. CT scan showing tracheal laceration (A), pneumomedistine (B) and parenchymal lung images secondary to bronchial aspiration (C).

paliperidone and suicidal drug intoxication 3 years earlier with benzodiazepines. She was treated at home by an advanced life support (ALS) unit of the Emergency Medical System (EMS) in a comatose state after the ingestion with suicidal ideation of an unknown quantity of Nitro Dipistol® solvent (composition: toluene: 50-75%; methyl acetate: 10-25%; methanol: 10-25%; 2-methylpropan-1-ol: 1-2.5%; 1-methyl-2-methoxyethyl acetate: < 1%). The patient underwent initial treatment of shock and severe respiratory failure with orotracheal intubation (difficult due to severe glottic edema), vasoactive support and fluid therapy, and was admitted to the intensive care unit. Analysis showed metabolic acidosis with lactate at 24.3 mg/dl. Thoracoabdominal CT scan showed an extensive pneumomediastinum with emphysema in the supra- and infra-glottic cervical region, extensive consolidation in both lower lobes, middle lobe, lingula and upper left lobe suggestive of possible iatrogenic tracheal laceration (Figure 1). Fibrogastroscopy showed esophageal causation (Zargar 2A) and acute duodenitis. During admission pronation, deep sedation and recruitment maneuvers were required. After 8 hours of admission, following a joint telephone assessment with the third level hospital, the emergent initiation of venous-vein (VV) ECMO and stabilization was agreed upon. Together with the EMS, coordination and inter-hospital transfer were made with this therapy and protective mechanical ventilation.

Fibrobronchoscopy showed a longitudinal tracheal laceration in its left middle quarter that required surgical repair and tracheotomy. Methanolemia on admission was 0.76 g/l, so an ethanol perfusion was scheduled. Acute renal failure was treated with hemodiafiltration. The initial evolution was favorable from the respiratory point of view, and was attributed to treatment with ECMO, although 13 days after admission she presented massive bleeding through the jugular cannula that was complicated by spontaneous decanulation and a picture of secondary hemorrhagic shock that could not be reversed, and the patient died.

It is considered a relevant toxicological case, as it presents concomitant oropharyngeal, esophageal and tracheal injuries secondary to the irritant effect of hydrocarbons (which contributed to the iatrogenic tracheal laceration during the out-of-hospital intubation maneuver) and pulmonary injuries due to the aspiration of the product. Finally, the serious effect of the metabolic toxicity associated with the intake of methanol and objectified in the analyses performed should be noted. In mass solvent poisoning with secondary aspiration, extreme care should be taken in handling the airway, due to the possible involvement of the tracheobronchial respiratory mucosa[4]. The application of ECMO could be useful in the treatment of cases with pulmonary involvement secondary to hydrocarbon poisoning and should be considered in the initial stages of treatment[4,5].

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Acute pericarditis secondary to COVID-19

Pericarditis aguda secundaria a COVID-19

To the editor:

COVID-19 is a global pandemic, caused by the SARS-CoV-2 coronavirus, which has so far affected more than 600,000 people in over 150 countries[1]. It is estimated that 7% of patients suffer myocardial damage from this infection[2]. However, the involvement of the pericardium is

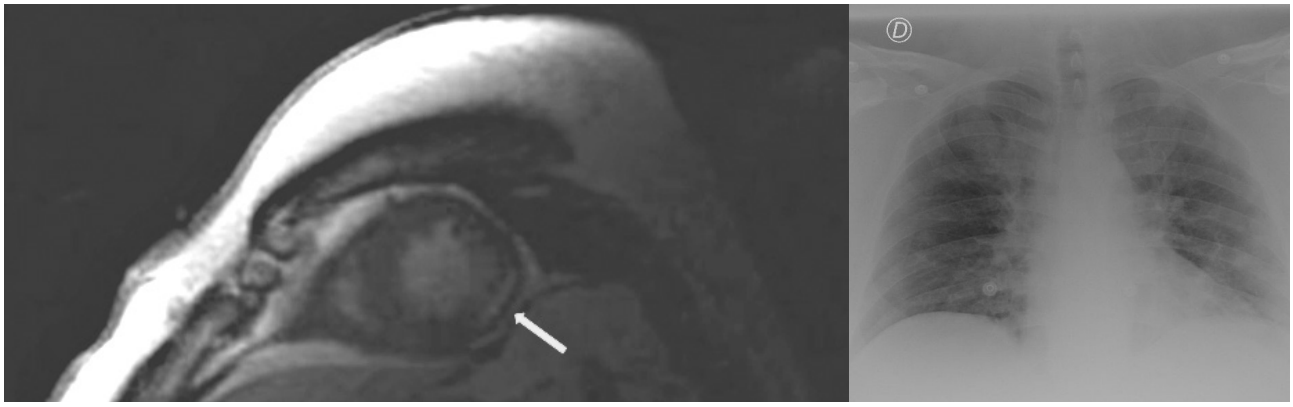

Figure 1. Electrocardiogram (left) and chest X-ray (right) of the case.

anecdotal as reflected in the literature[3,4].

A 35-year-old man with no personal history of interest who attended the emergency department for constant, stabbing central thoracic pain that increased with deep inspiration and decreased with forward inclination. The patient also reported a 5-day history of fever (39°C), dry cough and dyspnea of minimal effort. On arrival, the patient had a blood pressure of 122/75 mmHg, a heart rate of 98 bpm, oxygen saturation of 89% in room air and a body temperature of 38°C. During cardiopulmonary auscultation, bibasal crackles were observed, as well as pericardial rubbing. The following values stood out in the analysis: troponin T-ultra-sensitive: 6 ng/ml (normal value -NV-: < 15), PCR: 18.9 mg/dl (VN: < 0.5), LDH: 539 IU/l (VN: 110-225), ferritin: 391 ng/ml (VN: 150-300), D-dimer: 865ng/ml (VN: < 500) and lymphocytes: 580 cells/ul. Basal arterial gasometry showed partial respiratory failure (pH: 7.43, pCO_2: 36.4 mmHg, pO_2: 57.2 mmHg, HCO_3-: 24.3 mEq/l). The electrocardiogram (Figure 1A) showed a concave elevation of the diffuse ST segment with predominance of the lower and lateral faces, in addition to a subtle decrease in the PR interval. Chest X-ray showed bilateral interstitial-alveolar infiltration (Figure 1B). No data on ventricular dysfunction were observed in the echocardiography performed at the bedside.

Upon clinical and radiological suspicion of bilateral COVID-19 pneumonia, which was confirmed by PCR positivity for SARS-CoV-2, the patient was admitted to hospital and treatment with hydroxychloroquine, lopinavir/ritonavir, interferon-B and azithromycin was initiated. Acute pericarditis was also diagnosed and treatment was prescribed with acetylsalicylic acid (ASA) and colchicine. The patient showed a favourable evolution and was discharged with good general condition after 9 days of admission.

Acute pericarditis is an inflammatory syndrome with an infectious cause, mainly of viral etiology[5]. However, only two cases are reflected in the literature describing pericardial involvement by SARS-CoV-2[3,4]. While the first case refers to pericarditis secondary to acute myocarditis with concomitant deterioration of ventricular function, the second case describes cardiac tamponade in association with COVID-19. Therefore, the case presented here is the first description of an isolated acute pericarditis caused by SARS-CoV-2 with typical clinical and electrocardiographic manifestations.

Given the debate over the safety of using non-steroidal anti-inflammatory drugs (NSAIDs) in patients with COVID-19, the European Medicines Agency states that at present there is no evidence linking the use of NSAIDs to worsening of COVID-19 infection[6]. Therefore, we consider that the therapeutic recommendations of the European Society of Cardiology for acute pericarditis are applicable in patients with COVID-19, always taking into account the possible drug interactions that could occur, especially between antiretroviral agents, azithromycin and colchicine[5].

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