LETTERS TO THE EDITOR

Noninvasive ventilation as a therapeutic option in carbon monoxide poisoning

Sir,

I have recently read the article by Oliu et al., published in Emergencies, dealing with carbon monoxide (CO) poisoning and treatment[1]. This is without doubt an excellent review that emphasizes the importance of oxygen administration in both normobaric and hyperbaric conditions, as appropriate. They point out that oxygen should be administered by masks with a reservoir, to ensure the highest possible concentration of oxygen. They also highlight that when subsequent intubation and mechanical ventilation is required, a high concentration of oxygen is necessary, as is known.

However, they do not comment on the possible use of noninvasive ventilation (NIV), although there may be controversy over its use or perhaps lack of sufficient scientific evidence to recommend it unconditionally. The fact is that many of our emergency departments have good NIV equipment, and those who do not can always get access to Boussignac type CPAP devices. I believe this resource may be most useful in selected cases of CO poisoning. Rodríguez Fernández et al.[2] in a well-known article on NIV, indicated that it may be a therapeutic option with 100% oxygen, maintained until the carboxyhemoglobin value falls to less than 5%. They noted that since CO poisoning is often associated with smoke and irritant inhalation, bronchospasm is frequently observed, so NIV may be useful in this context[2]. Moreover, Poignon García et al.[3] noted that in the pathophysiology of CO poisoning, besides finding patients with symptoms of acute hypoxic respiratory failure due to alterations in oxygen transport and tissue absorption, the picture is also hypercapnic, depending on the degree of neurological dysfunction. Thus, from this point of view, we consider the use of NIV to be useful, with the aim of trying to improve gas exchange, not only to ensure good oxygenation, but also adequate ventilation. This could prevent intubation and its complications, and would be more appropriate to use than oxygen administration via mask with reservoir, since the latter method could be detrimental to the regulation of breathing in the clinical context of hypercapnia. They suggest that CPAP (or BiPAP pressure support), in the absence of contraindications, are the most beneficial forms of NIV. Their use increases functional residual capacity, reduces intrapulmonary shunt and work of breathing and opens collapsed alveoli. One may start with a low IPAP and EPAP of 6 to 8 and from 3 to 5 cm H_2O, respectively, and continue adapting these parameters to the patient's clinical condition and laboratory test results. One should ensure a tidal volume about 7 ml/kg, and try to maintain a respiratory rate less than 25 bpm.

Oliu et al.[1] also include in their review the importance of rapid displacement of CO from hemoglobin, to accelerate its elimination and prevent it reaching the cells from bloodstream, among other benefits. In this regard, Chapa Iglesias[4] compared the washout rate of carboxyhemoglobin in two of his patients with CO poisoning. One received oxygen (O_2) by a mask with reservoir and the other by Boussignac CPAP with O_2 flowmeter set to obtain a pressure of 10 cm H_2O. He noted that the second patient treated with CPAP reached the target level of 5% carboxyhemoglobin in 120-150 minutes, while the other, despite having lower initial levels of carboxyhemoglobin, required an extra 2 hours of oxygen therapy. Folgado also Pérez et al.[5] noted that clinical observation supports the hypothesis that oxygen therapy and CPAP improve arterial oxygen content, dissolved in plasma, and the elimination of carboxyhemoglobin, compared with the oxygen mask and a reservoir. And in this line they reported a clinical case of CO poisoning where they decided to optimize arterial oxygenation with Bousignac CPAP and a pressure of 7 cm H_2O with helmet interface and Fi $O_2$100%, with O_2 flow of 35 l/m to maintain adequate pressurization of the helmet and a stable CPAP, which was very well tolerated and achieved a decrease of carboxyhemoglobin levels from 29.3% on admission to 16.9% after one hour of treatment, and 9% and 4.9% after 2 and 3 hours of treatment, respectively.

They also highlighted the versatility of the Boussginac CPAP device which can easily be applied using a helmet interface, without CO_2 re-inhalation and maintaining a stable CPAP.

Given all this, we would argue that different modes of NIV may be most useful in the treatment of selected patients with CO poisoning and furthermore allow optimal use of the material resources we have.

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Authors' response

Sir,

First, we appreciate the interest shown by Dr. Álvarez Rodríguez in his critical reading of our article[1]. In the literature search for writing that article, we found no references to the routine use of noninvasive ventilation (NIV) in patients poisoned by carbon monoxide (CO). In conscious patients with normal airway, it seems unnecessary to apply the kind of pressure delivered by NIV for good ventilation and oxygenation. In cases of isolated CO poisoning, alveolar collapse and intrapulmonary shunt are uncommon. In mixed poisoning from smoke inhalation, the possibility exists of bronchospasm, which depends on the composition and characteristics of smoke. If bronchospasm is present, it is reactive and usually shows good response to conventional pharmacological therapy. Its pathophysiology is different from that of bronchospasm in chronic obstructive lung disease (COPD), which may require NIV in cases of non-response to drug treatment, fatigue and risk of respiratory hypercapnia on administering oxygen at high concentrations. In this regard, a recent review on the clinical management of poisoning due to fire smoke[2] refers to the administration of oxygen with reservoir mask and tracheal intubation (OTI) as necessary, without citing NIV systems.

On the other hand, as pointed out by Dr. Rodríguez, in cases of severe neurological dysfunction with associated hypoventilation, acute hypoxic respiratory failure may occur and even hypercapnia. But in these cases, if clinical conditions are severe, it is difficult to avoid the OTI, as altered level of consciousness is a contraindication for the application of NIV.

Finally, we should also consider the balance between the theoretical benefit obtained with NIV and the discomfort caused to the patient and the workload involved in the application of NIV, in the context of a busy emergency department.

The Boussignac system is more convenient and less complex to manage, but the only clinical situation in which it has been shown to increase survival and decrease the need for intubation is in cardiogenic pulmonary edema. Indications for NIV are severe exacerbation of COPD and severe cardiogenic pulmonary edema[3]. In severe hypoxemic respiratory failure from other causes, the indication for NIV is controversial.

Therefore, we believe that NIV is a good method for ventilatory support in selected patients, but there is no evidence to recommend its routine use in cases of CO poisoning. The only exception would be patients with decompensation of other pathologies because of CO poisoning or smoke, and this is where the indication of NIV is supported by scientific evidence.

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On optic nerve avulsion

Sir,

We are writing to thank Drs. Purriños and Balado for publishing a case of ophthalmic pathology in a general journal on emergency medicine[1]. In their case of optic nerve avulsion they correctly describe an uncommon lesion with poor visual prognosis. However, we would offer some remarks about it. Optic nerve avulsion presents with pupil alterations. Indeed, the direct pupillary reflex may be altered, even abolished, as occurs in relative afferent pupillary defect[2]. However, the consensual pupillary reflex will be normal if the contralateral eye is intact. For this same reason anisocoria is not always detected, and if found, many physicians suspect other causes, such as iris contusion or damage to the pupillary parasympathetic fibers that accompany the third cranial nerve. Since it is a disease that affects the optic nerve, the diagnosis can be made by ophthalmoscopy, and electro-retinography or evoked visual potentials are not necessary tests. In cases of suspicion one should not hesitate to consult an ophthalmologist or use direct ophthalmoscopy. Even in the absence of previous experience in exploration of the optic nerve, the pathological image should be evident.

Finally, imaging tests are accurate in optic nerve trauma[3], not so much for the diagnosis of avulsion as to assess other orbital structures, especially bone fractures or hematomas that compress the optic nerve, for which there are specific treatments that avoid loss of vision. It is true that sometimes computed tomography (CT) provides no additional information in relation to the eyeball and the optic nerve, but its sensitivity can be increased by the radiologist on being requested to perform 1mm coronal slices of the orbit with hyperextension of the neck (assuming there is no cervical disease associated). Cranial CT scan is not useful for orbital study[4].

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Nerve injury possibly caused by drinking methanol

Sir,

Methanol poisoning is associated with high mortality and morbidity,

with serious neurological sequelae, affecting the optic nerve and basal ganglia, especially the putamen[1-3].

A 42 year-old woman with a psychiatric history of anxiety-depressive disorder, treated with citalopram, etumina, clonazepam and lormetazepam, was taken to the emergency department after her family found her with drowsiness, blurred vision, abdominal pain and difficulty speaking from the previous night. At 12 hours after ED arrival she presented hypotension, metabolic acidosis (pH 7.04, bicarbonate 3.8 mmol/l and base excess of –24.8 mmol/l) with increased anion gap of 25 mEq/L (normal 7 ± 4 mEq/L). Gradual hemodynamic and neurologic deterioration led to transfer to the intensive care unit of a referral hospital. Four days later she showed clinical improvement, but with dystonic movement disorders in the upper extremities. In a second interview with the family, they reported finding an empty bottle of methylated spirits (96°) in her room. The patient has continued outpatient consultation in the departments of psychiatry and neurology, and dystonic movements have persisted.

Methanol (CH3-OH), also known as methyl alcohol, is a highly toxic substance that causes liver degradation by formaldehyde and formic acid, responsible for the toxicity, so that the clinical manifestations appear late (12 to 24 hours)[1,2].

The diagnosis is based on suspicion of intake, the presence of visual disturbances, metabolic acidosis with elevated osmolar and anion gap, and laboratory tests[4,5]. In addition, computed tomography or magnetic resonance imaging shows bilateral putaminal necrosis (a characteristic but rare finding) with or without bleeding (Figure 1), diffuse necrosis of white matter, subarachnoid hemorrhage, necrosis of the corpus callosum and bleeding in the brain parenchyma[6-8].

Treatment is based on life support measures, gastric lavage in the first two hours (activated carbon is not effective), correction of acidosis with bicarbonate hemodialysis and the use of antidotes such as ethanol (100 times more affinity) and 4-methylpirazol[10,19], although the latter is not marketed in Spain. In our case, methanol poisoning was diagnosed late, and treatment with these antidotes was not administered by either our ED or the referral hospital. Given the difficulty of obtaining a reliable history in psychiatric patients, especially in cases of suicide attempts, high initial clinical suspicion is necessary, with a blood gas

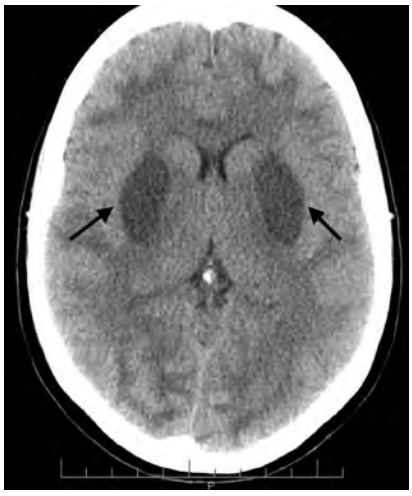

Figure 1. Cranial CT scan showing increased bilateral and symmetric density (black arrows) in putaminal areas consistent with hemorrhage and signs of cerebral edema.

diagnostic approach to rule out, inter alia, alcoholic ketoacidosis, lactic acidosis, hyperproteinemia, hyperlipidemia, mannitol or salicylate poisoning, paraldehyde and ethylene glycol. Early diagnosis and rapid initiation of treatment may be critical in the evolution of these patients.

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Pulseless ventricular tachycardia, atrial flutter and respiratory failure in a young woman: a rare association of events

Sir,

In young people, the coexistence of important arrhythmias - ventricular tachycardia originating from the right ventricular outflow tract and right atrial flutter - is a rarity and should lead to ruling out any structural cardiac abnormality. In the case reported, congenital lung failure was associated with these two types of arrhythmia.

A 39 year-old woman attended the emergency department for self-limited palpitations experienced during one month before admission, accompanied by dizziness. Thirteen years earlier she had consulted a cardiology department for palpitations; the finding then was isolated ventricular extrasystole. Medical history also included euthyroid goiter and congenital hypofibrinogenemia. Physical examination showed: blood pressure 115/80 mmHg, heart rate 115 beats per minute, afebrile, with pulmonary insufficiency murmur 2/6, ventricular extrasystole, and other tests were normal. In the ED she presented atrial flutter. She was hemodynamically stable and received oral atenolol 25 mg. The patient developed sinus rhythm with ventricular extrasystole and soon after two episodes of ventricular tachycardia without clearly palpable pulse, so electrical cardioversion was applied at 360 J, and she reverted to sinus rhythm. She was admitted to the intensive care unit and the arrhythmias were controlled with intravenous procainamide. Pulmonary insufficiency was confirmed by echocardiogram (pulmonary valve with prolapse of one of the leaflets and two moderate pulmonary regurgitation jets) and the morphology of ventricular tachycardia was consistent with ventricular tachycardia of the right ventricular outflow tract. Cardiac magnetic resonance examination was normal. In the electrophysiological study ablation of

atrial flutter circuit was performed, but ventricular tachycardia could not be induced. After this, the patient was treated with atenolol and discharged asymptomatic.

Congenital pulmonary insufficiency is generally benign[1] but may be associated with ventricular tachycardia and atrial flutter. Pulmonary valve disease has little hemodynamic significance, and does not produce heart failure unless there is severe lung hypertension[2]. The most common acquired pulmonary insufficiency is secondary to dilated valve annulus due to severe pulmonary hypertension, reflected in the physical examination finding of a high-frequency diastolic murmur, decreasing and hissing at the left sternal border (Graham Steell murmur)[3]. To our knowledge, the literature contains no case reports on the combination of these two arrhythmias and congenital pulmonary insufficiency.

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Differential diagnosis of lactic acidosis in a patient with multiple comorbidities

Sir,

The most important aspect of this case was the difficulty involved in reaching an etiologic diagnosis of lactic acidosis in the context of renal failure in a patient with multiple diseases and medications. It is important to consider adverse pharmacological effects in the differential diagnosis of any clinical picture, and the problem of a global understanding is accentuated in patients with multiple systemic diseases.

A 59 year-old man consulted the ED for vomiting, abdominal pain and hyporexia since three days before. A few minutes later, he presented epigastric pain, poor peripheral perfusion and decreased level of consciousness, and was transferred to the intensive care unit (ICU). Medical history included hypertension, diabetes mellitus II, atrial fibrillation and ischemic cerebrovascular accident (CVA) with residual hemiparesis, receiving treatment with aspirin, candesartan, warfarin, atorvastatin and metformin.

Lboratory tests showed pH below 6.8, lactacte >15 mmol/L, potassium 6.1 mlq/L, creatinine 8.6 mg/dl and glucose 29 mg/dl. In the ICU he was given continuous venovenous hemofiltration (CVVHF) during the first 24 hours. Recovery of the acute renal failure began and he entered a phase of polyurea. In this situation he was transferred to the department of internal medicine with good clinical evolution, although with fluctuating neurological status (dysarthria and hemiparesis). Cmputed tomography (CT) scan showed no recent injuries. Finally, after sustained stability, he was discharged to a rehabilitation center.

Metformin is an antidiabetic drug considered in most guideline as the first choice of therapy in diabetes mellitus II[1]. Its main effect is to decrease resistance to insulin and hepatic gluconeogenesis[4]. Clearance occurs entirely via the kidneys without being metabolized[5]. Typical manifestations of poisoning by this drug are associated with lactic acidosis (nausea, vomiting and diarrhea). In more severe cases it can cause hypotension, acute renal failure and cardiac arrest. There are a number of circumstances that favor the accumulation of lactate, and poisoning is rarely observed in the absence of renal, hepatic, cardiac or hemodynamic instability. As for treatment, it is essential to stabilize the airway and cardiovascular system, and to correct metabolic disorders. Hemodialysis and its variants, such as CVVHF, are preferred when the basic measures fail[2]. In our case, the most plausible hypothesis is that in the presence of mild renal insufficiency initially, probably pre-renal, a certain amount of metformin accumulated which led to frank renal failure, which in turn increased the circulating levels of metformin in a feedback system.

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Fibro-osseous pseudotumor of the finger simulating osteosarcoma

Sir,

Fibro-osseous pseudotumor of the digits is a benign tumor that affects young adults and presents as a fusiform mass located in the soft tissue of the fingers, usually in the proximal phalanx. The importance of this entity is the possible clinical, radiological and histopathological confusion with malignant neoplasms (osteosarcoma).

A 29 year-old man, with no relevant medical history, presented with tumor of 2 cm in diameter on the first phalanx of the right hand index finger. The lesion appeared 6 months earlier, with no history of trauma, phlogosis or pain. It was firm, hazel-nut sized, painless and not attached to bone. Plain radiography showed an image of bone density, calcified from the periphery to the center with normal adjacent bone structure. Surgical excision was performed. Pathologically, there were abundant fibroblasts and bone trabeculae with osteoblast and osteoclast border and abundant mitoses. MRI ruled out the hand bone involvement. With the clinical, radiological and pathological data, the case was diagnosed as fibro-osseous pseudotumor of the finger. At 12 months the patient remained asymptomatic.

Fibro-osseous pseudotumor of the digits is a rare disease of benign etiology, affecting adults with a mean age of 38 years[1,2]. It presents as a fusiform tumor 1-3 cm in diameter,

of hard consistency, well circumscribed, with inflammatory signs in 57% of cases. It is located in the soft tissue of the fingers, most often in the proximal phalanx, without muscular involvement. Radiologically it appears as a dense calcified mass in the periphery in 58% of cases and rarely with periosteal reaction. Histopathologically the excised lesions are ovoid or lobulated, with hardened tissue in the periphery and soft tissue or jelly in the center. The growth pattern is multinodular, disorganized, with poorly defined margins; there is fibroblastic proliferation, with varying degrees of cellular atypia, osteoid and bone trabeculae. Unlike fibroblasts, osteoblasts show no evidence of cellular atypia[3]. Its rapid growth and the radiological and pathological lesion findings mean that it may easily be confused with extraskeletal osteosarcoma. This, usually located in the lower extremities and rarely affectis the fingers, is only exceptionally found in patients younger than 35 years and there is pleomorphism and atypical mitosis. The differential diagnosis is important, because the error could lead to mutilating surgical procedures[4,5].

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Chronic lumbar back pain in the emergency department: treating the patient with vascular disease

Sir,

Back pain lasting more than three months is chronic; etiology is most often benign, of musculoskeletal causes. We report the case of a patient whose chronic lower back pain was due to a more serious process[1].

A 73 year-old man with hypertension, dyslipidemia and coronary heart disease consulted the emergency department with back pain of 4 months duration, which worsened with movement and interfered with sleep on occasions, without fever. He had no gastrointestinal or urological symptoms but reported weight loss. Physical examination revealed only a painful lower abdomen, no mass or visceromegaly, palpable and symmetrical pulses, and decreased sensation in the right quadriceps. Laboratory tests showed discreet normocytic anemia. Lateral X-ray of the lumbar spine (Figure 1a) showed erosion of the vertebrae L4-L52-4. Tumor markers were negative. Computed tomography (CT) showed a giant saccular aneurysm of the infrarenal abdominal aorta, measuring 9 x 7 x 6 cm, which conditioned the vertebral lysis of L3, L4 and L5, with an area of increased uptake on the right, which suggested intrathrombus bleeding[5]. Magnetic resonance imaging revealed dramatic bone disease caused by the aneurysm (Figure 1b).

Abdominal aortic aneurysms are usually asymptomatic, and rupture is the first manifestation in 25% of cases. In the ED we are accustomed to think about them in a patient with symptoms of suspected ruptured abdominal aneurysm. The differential diagnosis of back pain involves multiple entities, including discopathy[6], spondylolisthesis or erosion of lumbar vertebrae as in our case, which is rare but cases have been reported in the literature since 1961[7]. The diagnosis of diseases causing vertebral lysis is usually delayed while differential diagnosis is performed with processes such as bone metastases, primary tumors, spondylodiscitis or infection[8]. Abdominal X-ray is mandatory, sometimes showing calcifications of the aneurysmal wall, but abdominal ultrasound, CT angiography, MRI and angiography are useful to confirm the diagnosis, although CT angiography remains the technique of choice in the ED. The choice of MRI avoids ionizing radiation and the administration of contrast, which can be beneficial for certain patients.

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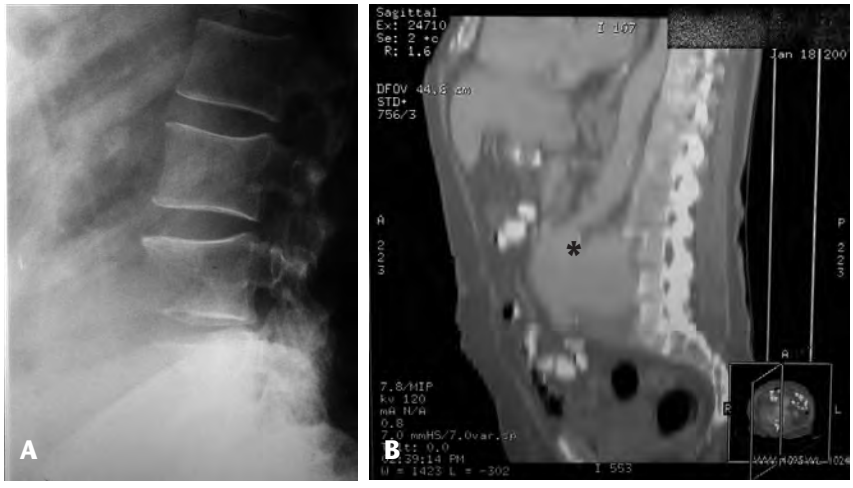

Figure 1. A) lateral X-ray showing lysis of the vertebral bodies from the aneurysm. B) MRI showing the aneurysm (*) and the full extent of spinal injuries.

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Bilateral testicular microlithiasis in a man with acute testicular pain

Sir,

Acute scrotum is often seen in emergency departments, usually of traumatic, vascular or inflammatory origin. Ultrasound plays a key role in their differential diagnosis. Testicular microlithiasis (TM) is an uncommon finding, usually incidental, and can manifest as testicular pain[1].

A 26 year-old patient presented with acute left testicular pain after physical exertion. Urgent scrotal ultrasound was performed to rule out testicular torsion; it revealed multiple hyperechogenic calcified foci without acoustic shadowing, diffusely distributed and abundant in both testes, with vascularization as assessed by normal echo-Doppler (Figure 1).

TM is an ultrasound finding of undetermined clinical significance and etiology, usually affecting both testes. It may be associated with a number of gonadal conditions including hydatid torsion, testicular torsion, varicocele, epididymitis, hydrocele and testicular pain[2]. It has also been described in association with cryptorchidism, infertility, Klinefelter syndrome, Down syndrome, Carney syndrome, cystic fibrosis, pulmonary alveolar microlithiasis, calcification of the sympathetic nervous system and brain[3]. But its association with malignant testicular tumors is what has generated controversy over whether or not it is a pre-malignant entity and, therefore, what diagnostic approach to adopt. The association of TM with carcinoma in situ and its relationship with testicular tumors, especially non-seminomatous germ cell tumors, is well documented. TM is thought to be a predisposing factor, a possible indirect indicator of pre-malignant disease or a tumor marker[5], although not a pre-malignant condition itself. In general, it is now accepted that patients with TM should be followed periodically with blood tests (alpha-fetoprotein and beta-HCG) and ultrasound[6], although the duration of follow up remains undetermined. The use of testicular biopsy is not justified except in very specific cases[7]. Knowledge of this rare condition of the testes and increasingly generalized use of scrotal ultrasound with high frequency probes will undoubtedly result in increased detection of MT, which may enable us to study large series and establish more precisely its origin and relation twith testicular cancer.

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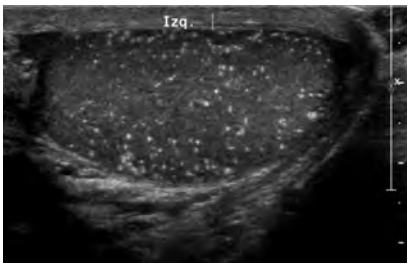

Figure 1. Hyperechogenic multiple millimeter-sized points and diffuse distribution in the left testicle.

Couple with asymptomatic fever: tularemia in emergency department

Sir,

Tularemia is an anthropozoonosis caused by the Gram negative coccobacillus *Francisella tularensis,* which is aerobic, intracellular and affects mainly animals and occasionally human beings[1-3]. The human disease is caused by contact with body fluids and tissues of contaminated animals (rabbits, hares, crabs, arthropods, etc.). Since 1997 there have been more than a thousand cases in Castilla and León, with outbreaks related to contact with animal hosts[4-7]. Infection in humans produces varied clinical manifestations but it can also be asymptomatic. Incubation period is three to seven days. The most common symptoms are fever, regional lymphadenopathy and general symptoms. Classically, six clinical forms have been described: ulceroglandular (common), nodal, typhoid, oculoganglionar, oropharyngeal and lung symptoms[8-10]. We report a case of tularemia with the typhoid form.

A 69 year-old man living in a rural area, previously healthy, was referred by his physician for symptoms of fever with no focus of origin. The patient had fever of 39ºC, predominantly in the evening, well controlled by antipyretics for 4 days. The previous week he had caught and handled hares, and he reported that his wife also had fever. Physical examination was normal, chest X-ray normal, blood tests showed LDH 557 U/L and CRP > 9 mg/dL, with the remaining parameters normal. Microhematuria was detected in urine. Blood serum tests were requested and the patient was empirically treated with doxycycline 100 mg every 12 hours for 14 days, and asked to return three weeks later. At 3 weeks the patient was asymptomatic. The local physician had also treated the man's wife with doxycycline and she was cured. The test results were negative for *Brucella, Chlamydophila,*

Mycoplasma, Legionella and tularemia. We repeated blood extraction for a new blood serum test and after two weeks the results were negative for all the above microorganisms except tularemia, with a titer of 1/5120, and seroconversion with respect to the previous test.

We present this case report because emergency departments are often asked to act at the request of a medical colleague, working in either primary care or a hospital. The results are striking, but the main beneficiary is the patient. This diagnosis of tularemia could have been made elsewhere, but the patient would probably have had a longer delay in diagnosis. The lesson in all this is that "high-resolution" consultation is increasingly necessary and this could be led by emergency physicians (revision of cases after empiric treatment, eg. pneumonia). In addition, this case confirms once again that medical history is the doctor's best diagnostic tool.

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