

Comment on "Applying a risk scale in interhospital transfer: need for adaptations and component redesign"

Sir:

Cardenete et al. retrospectively applied our interhospital transport scale (SVPTS) and describe its validity to optimize resources and increase efficiency¹. We appreciate their positive opinion of the scale and are pleased that it has again demonstrated its usefulness. However, we must highlight the fact that the SVPTS was created in 1981 in a very different healthcare scenario, before the existence of pre-hospital emergency teams, the development of intensive care units, regional emergency services and regional hospitals. Sometimes the distances to hospitals exceeded 200 km, and helicopter transport was an exceptional alternative². We sought to regulate secondary transfers in a setting without rules, without professionals or appropriate vehicles, and without legislation to regulate the transport of patients². Once the scale's validity was proven and results of its use were published³, its use progressively spread to other areas, as the authors describe¹.

After 30 years since its creation, during which time there has been a satisfactory growth of healthcare resources and technologies, we must reconsider the risk scale and adapt it to current reality. It seems appropriate to take into account some of the new resources (ultrasound, impedance hemodynamic monitoring, non-invasive ventilation, semi-automatic defibrillation, multi-parameter control), and technological advances (induced hypothermia, telecommunications), as well as the new drugs available, which affect the scale and make some of the items obsolete.

As stated by the authors - in relation to the development of small hospitals lacking the ability to offer certain techniques - percutaneous coronary intervention (PCI) in low-

risk patients is required in more than half of the indications for patient transfer, at least in their geographic area^{1,4}. With the growth of the hospital network, current ambulance transfers do not normally exceed 30 minutes, and helicopters are generally used for longer transfers. However, we believe there should be tighter control of returning patients after transfer to undergo PCI, given the high overall incidence of post-PCI complications: ventricular fibrillation or tachycardia in 5.7% of these patients (and increases their mortality from 3.6% to 23.2%); major bleeding in 3.5% and cardiogenic shock or stent thrombosis to a lesser extent⁵⁻⁷. Therefore, we would emphasize the need for a modern scale that determines the risks and facilitates safe, good quality transfers.

Consequently, we invite our SUMMA 112 colleagues, and all professionals (doctors, nurses and health emergency technicians) involved in this activity to reflect on possible changes to adapt the SVPTS to current needs, thus maintaining its validity and allowing quality, safe and efficient transfer. For this purpose, we intend to create a multicenter research team, open to all suggestions that may be useful.

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Emilio MORENO MILLÁN¹,
Jerusalén VILLEGAS DEL OJO¹,
Juan Manuel GARCÍA TORRECILLAS²,
María del Carmen LEA PEREIRA³,
Francisca PRIETO VALDERREY⁴

¹Servicio de Medicina Intensiva, Hospital "Santa Bárbara", Puertollano, Spain. ²Servicio de Cuidados Críticos y Urgencias, Complejo Hospitalario "Torrecárdenas". Almería, Spain. ³Servicio de Medicina Interna, Empresa Pública Hospital de Poniente. El Ejido, Spain.

Authors' reply

Sir:

Moreno et al¹ suggest the need to adapt their risk rating scale for inter-hospital transfer (IHT) to the current situation and redesign the component items. We read with interest their comments on our brief original article. We must say that during the process of data collection, which included the reports on each transfer made by the professionals involved in IHT, we observed that some of the SVPTS³ variables, rather than obsolete, did not fully correspond to current therapeutic possibilities or clinical status parameters, and therefore could be updated. Indeed, the SVPTS must be adapted to the current reality of our public health system, not only to reflect the technological and pharmacological advances now employed in medical emergency services, but also to take into account the high level of training and capability of modern health transport professionals (doctors, nurses and technicians), the sustainability of the system and the real needs of our patients in IHT.

Regarding patients being returned to their local hospital after undergoing PCI, the risk of adverse events cannot really be avoided, but we would like to point out that the

majority of ventricular fibrillation or tachycardia events (64%) occurred before the end of the PCI procedure⁴ and cardiogenic shock mainly occurred before or during the procedure⁵. In our sample², as in other studies on IHT with properly selected patients, we found a very low incidence of complications and no mortality during transfer⁶⁻⁸.

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César CARDENETE REYES,
Gemma TÉLLEZ GALÁN,
Carlos POLO PORTES

*Servicio de Urgencias Médicas de Madrid
(SUMMA 112), Spain.*

Paraplegia and anticoagulation therapy: a case report

Sir:

Of all the complications associated with anticoagulation, bleeding is

the most frequent¹. When it affects the central nervous system (CNS), the location is usually the brain. Although brain hemorrhage represents less than 1%, it is a life-threatening condition and generally fatal¹⁻³. Spinal cord involvement is even rarer, and symptoms are often nonspecific or silent, so suspicion is of great importance for diagnosis which should be made early, as spinal compression may produce irreversible neurological impairment.

An 81 year-old woman was admitted for inability to walk and remain standing of a few hours duration. She had a history of hypertension, abdominal aorta aneurysm and atrial fibrillation for which she was being treated with acenocoumarol. The patient reported that 36 hours before she had experienced sudden intense pain at the lumbar level, irradiating to the waist and both legs, but it was self-limited and she had not considered it important. She denied any history of trauma, fever or other clinical symptoms. Physical examination revealed nothing except the known cardiac arrhythmia and a hypogastric palpable non-pulsatile abdominal mass, related with a distended bladder which resolved after urinary catheterization. Arterial pulse in both legs were preserved. In contrast, neurological examination revealed a 3/5 paraparesis and areflexia in both legs, was preserved. Laboratory tests showed impaired renal function (creatinine 2.1 g / dL) and excessive hypocoagulability (prothrombin activity: 7%; INR: 5.4), which was treated with vitamin K and withdrawal of anticoagulation. Cranial computed tomography (CT) scan was normal, and abdominal CT ruled out aneurysm rupture, but showed a wedge fracture of the vertebra T12, already present in previous studies, without spinal involvement. Two days after admission, her back pain was controlled and renal function normalized, but the patient deteriorated neurologically to develop paraplegia and L1 nerve root sensitivity. Lumbar CT scan with contrast showed the previously described fracture and a hypodense collection in the anterior epidural location (Figure 1) extending from T10 to L1, compatible with a spinal hematoma

with compressive effect. The case was discussed with the department of neurosurgery, which ruled out sur-

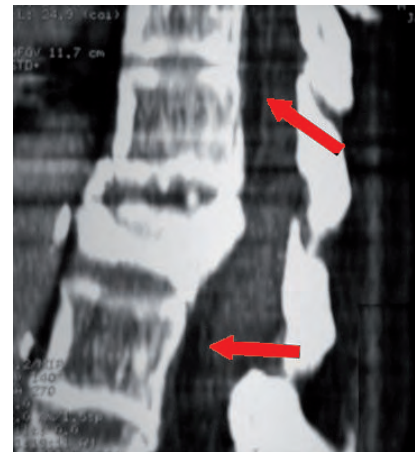


Figure 1. Detail of thoracolumbar computed tomography scan showing crushed T12 vertebral body and anterior epidural hematoma (arrows).

gical decompression given the high risk status of the patient. At 4 weeks after admission, CT scan confirmed resorption of the hematoma, but the 3/5 paraparesis of both legs persisted and neurogenic bladder.

Spontaneous epidural hematoma is rare; it represents 0.3-0.6% of spinal space-occupying lesions³. Its origin is mainly anticoagulation therapy, but neoplasia, coagulopathy and vascular malformation may also be the cause^{4,5}. Epidural hematomas can be either subdural or intraparenchymal, with few cases described^{6,7}. In anticoagulated patients, the origin is more frequently post-traumatic than spontaneous, and the most common location is the thoracic spine⁷. Although the secondary development of neurological symptoms is not frequent, appearing in only 30% of cases³, this type of hematoma can compress the spinal cord and nerve roots, giving rise to clinical symptoms that allow us to reach an early diagnosis, such as acute pain (single most common symptom), focal or radicular pain, followed by signs of spinal involvement such as urinary retention or sensory and motor compromise of the legs, which may progress to paraplegia as in our case. Symptomatic extrinsic compression of the spinal cord by any cause is a medical emergency, and best treatment involves early diagnosis and correction of any underlying coagulopathy and surgical decompression when possible. The approach to treatment is either conservative, which involves withdrawal of anticoagulation

and the administration of drugs to reverse its effects, or surgical measures involving drainage and decompression. Regarding the decision to perform surgery or not, the results of surgical treatment depend on initial neurological status, the severity of other comorbid conditions, location of the bleeding and the association with subdural hematoma⁸. Less preoperative symptom severity and faster surgical decompression are the important variables most likely to allow complete clinical recovery⁸. The present case is most instructive. Despite the low incidence of spinal hematoma, in all anticoagulated patients with non-traumatic acute vertebral bone pain associated with neurological symptoms, we should consider this condition and perform early imaging studies to rule it out, since effective treatment involves urgent surgical decompression, when possible, or else neurological deficit will be difficult to resolve.

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Miguel Ángel NÚÑEZ VIEJO,
Ana FERNÁNDEZ MONTES

Servicio de Medicina Interna. Hospital Universitario Marqués de Valdecilla. Santander, Spain.

Cardiopulmonary resuscitation in the classroom: the "RCP na aula" program for lower-secondary school students

Sir:

The teaching of cardiopulmonary resuscitation at a basic level (CPR-B) to children at compulsory school levels is a great opportunity to reach and instruct a large number of citizens¹. In Europe, CPR-B is included in the school curriculum in Norway, Denmark, France, Britain and Spain. Although the Spanish Education Act² includes basic protocols of first aid in the subject physical education (PE) for 4th year secondary education students, in practice, CPR-B training is not standardized.

Classroom CPR (called RCP na aula in Galicia) is a system in which health professionals of the emergency service 061 Galicia train the heads of PE departments as instructors in CPR-B and semi-automatic external defibrillator (SAED) use, and these instructors teach other teachers and students. It is aimed at all secondary school students where the subject of physical education is mandatory.

The initial course has one part online (<http://rcpnaula.iesollosgrandes.org/>) and another face-to-face part lasting 5 hours. Heads of PE departments who complete the course then train their colleagues and these teachers teach their pupils and other school staff. Each department designs its own teaching unit. The course consists 9-10 sessions of 45 minutes in one school term and a final exam. Finally, with back-up 15-minute sessions for 4 weeks during the school year, and then once a term in successive years. The system is essentially based on teachers, which allows reaching all the students, and ensures they know the matter from their first year of secondary school to the end, and receive refresher sessions during five years. The main difference between classroom PCR and other similar national programs³⁻⁵ is that it is teacher-centered; he or she chooses the method, which is intended to ensure continuity.

The emergency service 061 has ceded 15 Laerdal Little Anne manikins and 5 defibrillator simulators (Phillips FR2 AED®). In Lugo, twenty-six school centers of the 56 that exist (46.4%) have started their participa-

tion, as well as 3 centers in other provinces. Initially, 061 of Galicia trained 37 teachers as CPR-B instructors, and 29 of them continued with the program, teaching 28 other teachers from their centers. In 2 years (2007-2009), these 57 teachers have taught CPR-B to 1,900 students and 210 employees. In the academic year 2010-2011, 16 more centers joined the program, so expectations are high regarding the training of the general population of Lugo through this system.

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María del Carmen LÓPEZ UNANUA,
Miguel FREIRE TELLADO,
Rosario RASINES SISNIEGA,
Antonio IGLESIAS GONZÁLEZ

*Fundación Pública Urgencias Sanitarias de Galicia
061. Base Medicalizada de Lugo, Spain.*

Lipschütz ulcer: an infrequently seen emergency

Sir:

Genital ulcers are a frequent reason for visits to the emergency department. Idiopathic vulvar or Lipschütz ulcer is a rare entity, which presents in adolescents. It manifests as a painful genital ulcer accompanied by malaise, fever and flu-like symptoms which often cause intense concern and uncertainty in the patient, family members and health staff. This ulcer may suggest sexually transmitted disease and even sexual abuse. There are few cases described

in the literature, and it is probably under-diagnosed in primary care, regional and even tertiary hospital emergency departments.

We present two cases of Lipschutz ulcer attended at our hospital emergency department. Two female patients aged 12 and 13 years old consulted for pain and swelling in the genital area with foul-smelling discharge about 48 hours earlier. Both had initiated antibiotic treatment some days before for sore throat and fever up to 40°C. Both denied any sexual intercourse. Physical examination showed an elevated ulcerated lesion on the external genitals approximately 3 cm in diameter, with necrotic base, purulent edges and serohematic secretion, painful on palpation and with urination, unrelated to previous trauma (Figure 1). There was no inguinal lymphadenopathy, nor axillary or oral ulcers. Two ulcer exudate samples per patient were collected for culture and histological study. Also, laboratory blood tests included blood count, acute phase reactants, serology (HBV, HCV, HIV, HSV type 1 and 2, EBV, CMV, syphilis) and immunologic profile (ANA, ANCA and rheumatoid factor).

Among the results we found elevated PCR (> 90 mg / dL), and IgG antibodies positive for Epstein Barr virus in one patient and for varicella zoster in both. The culture was negative. The pathology report described vulvar fragments with epithelial hyperplasia and mixed inflammatory infiltrate (acute and chronic). Scintigraphy with labeled leukocytes was normal. Both patients were diagnosed with idiopathic vulvar ulcer and treated with antibiotics cefuroxime and amoxicillin-clavulanate. Outcome was favorable and ten days later the vulvar lesion had disappeared in both patients.

Lipschutz's ulcer, aka acute or idiopathic vulvar ulcer, is characterized by the appearance of a genital ulcer in children and adolescents without previous sexual relationships. It was first described by Lipschutz in 1913 who attributed the ulcer to inoculation by *Bacillus crassus* (*Lactobacillus Döderlein*)^{1,2}. This theory has not been confirmed in studies published to date. Lipschutz's ulcer is little known and probably under-diagnosed, which reduces the possibility of clinical suspicion. Onset is abrupt, very painful, accompanied by febrile syndrome, sore throat, headache, as-



Figure 1. Vulvar ulcer in the two patients described.

themia, myalgia, and inguinal adenopathy. Genital lesions are characteristically necrotic, deep and edematous.

Two forms of presentation have been described. The gangrenous form, associated with systemic symptoms, is more frequent: it presents very acute, deep ulcers with a grayish-white background which leave a scar after healing. Then there is a miliary form that causes superficial, small, purulent fibrinous ulcers with an erythematous halo. Not associated with systemic symptoms, these ulcers heal in 1-2 weeks without leaving a scar or recurrence.

The etiology and pathogenesis remain unknown, although it has been linked in multiple studies to primary infection with Epstein-Barr virus and infection by ureaplasma, typhoid, paratyphoid and HIV^{3,4}. To date, criteria for hospital admission have not been described. The presence of painful genital ulcers in young women without sexual activity and generalized symptoms helps guide the diagnosis, although this is by exclusion. Therefore, correct differential diagnosis is important, including all other causes of acute genital ulcers⁵. They can be divided into those of infectious origin, both venereal (syphilis, lymphogranuloma venereum, herpes sim-

plex, chancroid) and non-venereal (Epstein-Barr virus, cytomegalovirus, brucella)⁶, and those of non-infectious origin (Crohn's disease, Behcet's disease, vulvar pemphigus, sclerosis lichen, idiopathic aphthosis, fixed drug or multiform erythema)⁷. The literature contains descriptions of diagnostic criteria as lack of clinical evidence of recurrent oral aphthosis, to rule out Behcet's disease and negative results for diseases of sexual transmission⁸.

Additional tests include CBC, erythrocyte sedimentation rate, blood chemistry, serology (Epstein-Barr virus, cytomegalovirus, brucella, HIV and syphilis), exudate, blood and stool culture, HSV and the detection of *Chlamydia trachomatis*⁹.

Treatment is primarily symptomatic, although administration of broad spectrum antibiotics has proven effective in gangrenous forms¹⁰.

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Raysé RODRÍGUEZ REYES,
Mercedes HERNÁNDEZ SUÁREZ,
Ana Isabel PADILLA PÉREZ,
Francisco Javier DE LA TORRE
FERNÁNDEZ DE VEGA

Servicio de Ginecología y Obstetricia. Hospital Universitario de Canarias. Tenerife, Spain.

Long-QT interval secondary to antimicrobial treatment

Sir:

Antimicrobials represent a tenth of the pharmaceutical market in our country, and the third most frequently taken medicinal drugs after analgesics and antihypertensives.

In recent years, the prescription of antibiotics in Spain has changed and they are increasingly associated with prolongation of the QT interval¹. Given the frequency of use, aging of the population and increased prevalence of structural heart disease, potentially arrhythmogenic drugs should be prescribed with caution.

A 75 year-old woman was admitted to our emergency department for three syncopal episodes in the last 12 hours. The last episode was accompanied by tonic-clonic seizures without presenting a postcritical period. The patient had a history of long-standing hypertension treated with enalapril 20 mg daily. In the last 5 days the patient had had symptoms of upper respiratory tract catarrh, for which reason she attended a primary care center and was prescribed azithromycin 500 mg daily. She took a total of three doses and on the third day of treatment her symptoms had not improved. She consulted another physician who prescribed moxifloxacin 400 mg every 24 hours. After two days of moxifloxacin treatment the patient experienced three episodes of abrupt loss of consciousness. Admission echocardiogram (ECG) showed atrial fibrillation of 76 bpm and a long QT (520 ms) with frequent ventricular extrasystole and a few episodes of polymorphic ventricular tachycardia. Transthoracic ECG showed no significant alterations. The clinical picture was interpreted as QT interval prolongation with secondary ventricular arrhythmia. Because of concomitant drugs that could be involved in the genesis of these findings and the reversibility of the picture, it was considered that both azithromycin and moxifloxacin could be responsible. Maintenance therapy was applied and ECG monitoring revealed episodes of self-limiting ventricular tachycardia, which disappeared in the first 12 hours of admission, and the QT interval normalized at 72 hours.

The antimicrobial groups most frequently involved in the genesis of QT prolongation are the macrolides,

fluoroquinolones, antimalarials, anti-protozoals and antifungals^{2,3}. The predisposing factors reported as encouraging QT prolongation in patients treated with antimicrobials include female gender, structural heart disease, taking another drug that enhances QT elongation, reduced elimination of the drug due to liver or renal failure, hypokalemia, hypomagnesemia, basal bradycardia that alters corrected QT interval (over 450 ms) and genetic predisposition⁴. Congenital long QT interval⁵ may become apparent after exposure to certain drugs. Precisely the fluoroquinolones and macrolides have shown a significant increase in prescription, as they are used in high-incidence conditions such as respiratory infection⁶. For this reason we believe that clinicians should be aware of the potential arrhythmogenic effect of antimicrobials.

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Francisco EPELDE,
María Luisa IGLESIAS-LEPINE,
Leopoldo ANARTE

*Servicio de Urgencias. Hospital de Sabadell.
Corporació Sanitària i Universitaria Parc Tauli.
Sabadell, Spain.*

Resuscitation using intravenous lipid emulsion or epinephrine

Sr. Editor:

Nogué S et al.¹ have produced

an excellent review of the role of intravenous lipid emulsion (ILE) in cardiac arrest (CA) and refractory shock secondary to highly lipid soluble drugs. As they point out, the scientific evidence supporting their use is very low, relying on expert opinion, animal experimentation and clinical observations. However, they also that the recommendations of the American Heart Association (AHA) 2010 include their use in refractory cases of severe poisoning with beta-blockers and local anesthetics².

The authors recommend reducing or suspending the use of adrenaline in these cases of CA or refractory shock, since it worsens prognosis when used in conjunction with ILE. We disagree with this recommendation, since the use of adrenaline in CA is recommended with a level of evidence A in the same AHA guidelines of 2010³. But the recommendation to suspend adrenaline is based solely on two experimental animal studies 4,5 with important limitations, as recognized by the authors. We would also point out that the use of ILE in cases of beta-blocker intoxication in the AHA recommendations is based on the description of two cases².

In the situation of CA it seems more appropriate to continue using the doses of adrenaline recommended in the current guidelines on cardiopulmonary resuscitation and, in situations of shock, vasoactive amines.

If there is no response to these measures, and assuming the situation is secondary to highly soluble drug toxicity, ILE should be administered at recommended doses. The effectiveness of ILE will be reflected in the recovery of spontaneous circulation in cases of CA, and hemodynamic improvement which will allow amine dose reduction. This latter situation is described by Engels et al⁶ in a case of amitriptyline intoxication and by Young et al.⁷ in a case of verapamil intoxication, both of which are covered in the review.

We agree with the authors that ILE should be included in our therapeutic arsenal for highly soluble drug toxicity refractory to conventional measures, but without forgetting that the level of evidence in support of ILE use is very low and ILE is not exempt from adverse effects. We do not agree that adrenaline or other amines should be suspended or re-

duced in situations of cardiac arrest or refractory shock.

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Francisco Ramón PAMPÍN HUERTA,
Fernando MOSTEIRO PEREIRA
*Servicio de Medicina Intensiva. Complejo
Hospitalario Universitario A Coruña.
A Coruña. Spain.*

Authors' reply

Sr. Editor:

In the review published in EMERGENCIAS we stated that the current level of scientific evidence supporting the use of intravenous lipid emulsion (ILE) is low, that dosage is not definitely established and that some negative interactions described, with adrenaline for example, are based on animal experimentation¹. Nevertheless, we proposed that in some cases ILE can be used as rescue therapy, as do the American Heart Association recommendations of 2010. But readers of this Journal must be clear that we never suggested that ILE should replace cardiopulmonary resuscitation or any of the conventional drugs administered during the procedure; only that in some cases of intoxication where the cardiovascular system is refractory to conven-

tional measures, it was time to evaluate the use of ILE.

With respect to interference between ILE and adrenaline, we believe the work of Hiller et al.², albeit experimental (rats), is of great interest in asystole induced by bupivacaine, where the effectiveness of lipid infusion was reduced by concomitant use of adrenaline at high doses (> 10 µg/kg). Harvey et al.³, in another experimental model (rabbits), showed that in the same situation as that described above, although adrenaline was essential to achieve return of spontaneous circulation, the animals simultaneously treated with adrenaline and ILE presented life-threatening hemodynamic deterioration, a situation not observed with lipid perfusion alone. Likewise, in another study with similar characteristics carried out by Hicks et al.⁴ in pigs, the infusion of high-dose adrenaline (100 µg/kg) did not allow lipid perfusion to offer better prognosis regarding cardiac arrest, while Weinberg et al.⁵ in bupivacaine-induced asystole showed that the resuscitation capacity of ILE was significantly higher than that of adrenaline. But it is also true that in human clinical practice this association has been made without apparent drawbacks. In a very recent review by Wolfe et al⁶, after weighing the pros and cons of the association between ILE and adrenaline and confirming the absence of consensus, the authors concluded that in cardiac arrest induced by local anesthetics, ILE should be initiated with relatively small doses of adrenaline (1mg / kg), a drug still lacking strong evidence for use in cardiac arrest to improve prognosis.

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Santiago NOGUÉ,
Juan CINO,
Nuria COROMINAS,
Dolors SOY

*Unidad de Toxicología Clínica. Área de Urgencias.
Hospital Clínic. Barcelona, Spain.*

Necrotizing fasciitis: a case report

Sir:

Soft tissue infections (STI) are frequently attended in the emergency department¹.

They may be necrotizing or not². Necrotizing STI (NSTI), including necrotizing fasciitis, are rare and should be diagnosed rapidly because they are associated with high mortality which decreases with early treatment²⁻⁷.

A 40 year-old woman with chronic alcoholism was attended for general state deterioration and back pain after a fall 2 days before. She was drowsy, but had no focal neurological deficits. On admission she had blood pressure of 120/60 mmHg, heart rate 90 bpm, arterial oxygen saturation 91% (digital pulse oximetry), fever (38°C) and presented erosions and crepitation of the right paralumbar area. Laboratory tests showed leukocytosis and neutrophilia, hyponatremia, hypokalemia (2.7 mmol / L), increased creatinine (1.5 mg / dl), CRP of 32 mg / dL and non-compensated metabolic alkalosis. Lumbar x-ray showed gas in the soft tissue gas (Figure 1) and computed tomography (CT) with iv contrast showed that the deepest tissue was most affected, characteristic of necrotizing fasciitis. Soon after, she presented hemodynamic instability (blood pressure 69/44 mmHg with no response to fluid therapy, with tachycardia (heart rate 115 bpm) and worsening state of consciousness. The patient was transferred to the intensive care unit of a reference hospital after receiving a first dose of antibiotic. She underwent debridement of necrotic subcutaneous tissue and received broad spectrum antibiotic combination the-

rapy; tissue samples collected for culture were positive for *E. Coli* and *Proteus penneri*. The patient eventually died.

Certain groups of patients are predisposed to NSTI: alcoholics, diabetics, immunosuppressed patients and parenteral drug users^{2,3}. The precipitating factor may be slight trauma (as in this case), surgical wound infection or a pressure ulcer, but the trigger is unknown in 20% of cases. High fever, hypotension, prostration and multiorgan failure with physical examination findings suggestive of STI lead to the suspected diagnosis of NSTI, although only 10-40% of cases present this picture³. The importance of early diagnosis of NSTI and the different therapeutic approach to STI have prompted investigators to seek different laboratory parameters⁴, physical examination findings⁵ and imaging test results^{1,8} to differentiate between the two processes.

The analytical parameters, such as leukocyte count, sodium, serum BUN⁴, or others such as the LRINEC scale⁹ comprising glucose, creatinine, sodium, hemoglobin and white blood cell count, need prospective validation studies and lack sensitivity⁵. Physical examination findings alone allow diagnosis only in advanced stages of the disease where cutaneous hemorrhagic blisters, necrosis and



Figure 1. Lateral x-ray of the lower lumbar spine showing abundant soft tissue gas.

crepitus appear⁵. Plain x-ray may demonstrate the presence of soft tissue gas, which is characteristic of NSTI but only occurs in less than 50% of cases⁸. CT scan has greater sensitivity than x-ray for detecting gas in the deep soft tissues³. As many as 80% of NSTI cases show polymicrobial infection, by aerobic and anaerobic bacteria, and therefore broad spectrum antibiotic treatment is necessary^{1,2}, although early surgical debridement of necrotic tissue improves the prognosis^{1,2,4,7}.

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Elena GUERRA DEL BARRIO¹,
Ignacio GONZÁLEZ IGLESIAS²,
Juan Alberto GARCÍA CACHERO²,
Lorenzo PASTOR HERNÁNDEZ¹

¹Servicio de Urgencias. ²Servicio de Radiodiagnóstico. Hospital V. Alvarez-Buylla. Asturias, Spain.