

Letters to the Editor

Prognostic markers in infected patients

Dear Sir,

Nowadays there is a great deal of interest in sepsis. Its increasing incidence and significant mortality rate¹ make it a very important issue. From the moment we suspect this kind of condition, we need to distinguish whether the patient is has sepsis, severe sepsis or septic shock^{2,3}. Each of these conditions has a different prognosis and requires a different kind of treatment. Early, objective-based treatment improves the chances of survival for patients⁴. In most cases an accurate diagnosis cannot be made based solely on the classic clinical symptoms of low blood pressure and tachycardia. Other markers such as lactic acid should also be taken into consideration^{5,6}. Lactic acid is a weak acid which is a product of glycolysis. In situations where limited oxygen is available for cells, the Krebs cycle is altered and levels of lactic acid increase. This becomes a biomarker for tissue hypoxia⁶. Tissue hypoxia occurs in different circumstances for example: a change in oxygen supply (hypoxemia, hypoperfusion caused by low cardiac output or low blood pressure), an increase in oxygen demand (convulsions, hyperthermia) and/or changes in oxygen extraction capacity. The presence of high levels of lactic acid in patients, irrespective of whether they have low blood pressure or not, marks the difference between sepsis, severe sepsis and septic shock in septic patients^{4,7}. Until now, studies such as that carried out by Shapiro⁸ link levels of lactic acid in patients when they arrive at the emergency department with a higher mortality rate. However, variables such as the presence or absence of comorbidity or the haemodynamic state are not taken into consideration.

The objective of our study was to analyse the possible prognostic markers (levels of lactic acid, haemodynamic state and comorbidity) in patients admitted to hospital via the emergency department with syndromic diagnosis of a known infection, and the relationship between these markers and the chances of survival.

This was a prospective study carried out in the Emergency Department of Hospital Río Hortega in Valladolid, Spain. The inclusion criteria were as follows: all patients over the age of 18 who came to the emergency department with syndromic diagnosis of any kind of infection. Different variables were recorded from each patient including the presence or absence of comorbidity, temperature, average blood pressure, heart rate, oxygen saturation, mental state, oedemas, etc. When the patient was admitted to the emergency department different additional tests were carried out (full blood test, coagulation test, gasometry and arterial lactic acid levels) and fi-

nally information was recorded about the patients progress, amino acid requirements, etc.

Statistical analysis was carried out using the software program SPSS 11.0. Qualitative variables were compared using the chi-square test and statistical significance was taken into consideration at $p < 0.05$.

The study included a series of 99 patients with a mean age of 71.5 years. Of these, 20 died (20.2%). There were significant statistical differences ($p < 0.05$) between the final outcome (death or survival) and the levels of lactic acid when the patient was admitted to the emergency department. There were also significant statistical differences between the patient's final outcome and haemodynamic instability (blood pressure below 70 mm Hg, systolic blood pressure below 90 mm Hg or a drop of more than 40 mm Hg). There was a higher death rate among patients who were haemodynamically unstable and those with lactic acid levels over 2 mmol/l.

At the same time, we also observed how lactic acid values can be used as markers for the final prognosis, irrespective of the patient's haemodynamic situation or the presence or absence of comorbidity.

Although the results obtained were limited because of the number of patients included in the study, they support the fact that lactic acid levels and the haemodynamic state of the patient can be used as prognostic markers in septic patients, irrespective of comorbidity. We have also seen how the link between the levels of lactic acid and the patient's final outcome is not affected by the presence or absence of low blood pressure or other comorbidity factors. This makes arterial lactic acid a very significant prognostic marker for hospital emergency departments. Early evaluation of lactic acid levels in patients with a suspected secondary infection who have been admitted to the emergency department could help to establish the seriousness of the case and allow medical staff to administer the correct treatment as early as possible³.

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Spontaneous bilateral pneumothorax and asthma attacks

Dear Sir,

Spontaneous pneumothorax is defined as a condition that is not a result of trauma or medical treatment. It is a primary condition when it cannot be substantiated by clinical injuries or x-rays and is secondary when accompanied by pulmonary diseases, in particular COPD¹. It is not commonly associated with asthma, especially when the pneumothorax is bilateral (BP) and in patients with spontaneous breathing. In these cases, which are clinically similar to an asthma attack, a diagnosis of pneumothorax may be delayed or even overlooked there by putting the patient at great risk.

A 28-year-old patient with a history of a possible allergy to paracetamol, who smoked 10 cigarettes a day, had chronic rhinitis and had been suffering from bronchial asthma since he was 9 years old, was admitted to hospital because of an asthma attack. He had not reacted well to beta₂ antagonists and was given an oxygen reservoir bag at 15 litres per minute. Arterial gasometry showed pH 7.34, PaCO₂ 40 mmHg, PaO₂ 64 mmHg, EB -4.2, O₂ Sat 91% and he was promptly admitted to intensive care.

When he was admitted we observed tachypnea at 32 breaths per minute, 89% oxygen saturation with the oxygen reservoir bag and mask, general hypoventilation and bilateral ronchus and wheezing.

The full blood test showed 22,800 leukocytes/mm³ with 98% segmented and 3.7% lymphocytes. The chest x-ray showed significant pulmonary hyper-insufflation, which was more marked in the right lung with laminar atelectasis. We began treatment with intravenous levofloxacin, high flow oxygen, salbutamol, nebulised ipratropium and intravenous methyl prednisolone and observed a general improvement at first.

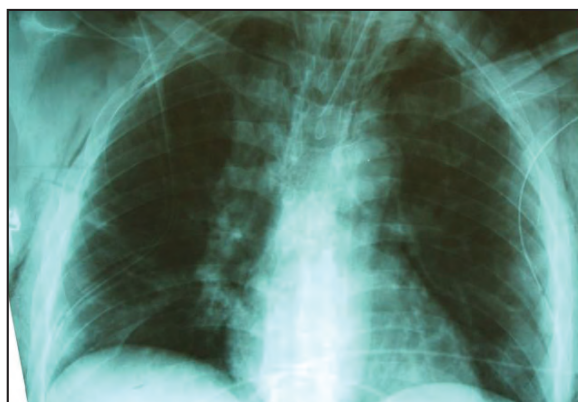


Figure 1. Chest x-ray which shows extensive bilateral subcutaneous emphysema and bilateral pleural effusion.

Eighteen hours after admission the patient deteriorated and presented significant anxiety, tachypnea, respiratory difficulties and severe bronchial spasms with 72%O₂ Sat which did not improve following medical treatment. Suddenly subcutaneous emphysema appeared on the neck and right chest area without tracheal deviation. Blood pressure was 120/65 mm Hg; heart rate was 125 beats per minute with arterial-venous pressure at 18-20 cmH₂O with oliguria. Faced with a suspected tension pneumothorax, a diagnostic chest wall puncture was carried out and the result was positive. An anterior chest tube was inserted and the patient improved, oxygen saturation was 90%. After 15 minutes the patient began to show signs of oxygen desaturation of up to 80% with subcutaneous emphysema on the left side of the chest area and tympanic percussion sounds which prompted insertion of a chest tube in the left side. Finally, the patient was intubated because of persistent bronchospasms.

In the x-ray afterwards we observed significant subcutaneous bilateral emphysema (Figure 1). The initial peak pressure (PP) was 52 cm H₂O with a tidal volume (TV) of 480 ml and breathing rate of 12 with auto-PEEP of 15 cmH₂O. The gasometry with FiO₂ 100% showed pH 7.04 with PaCO₂ 117 mmHg and PaO₂ 149 with bicarbonate at 22 and O₂ Sat 98%. The patient was put on a Servo 300 mechanical ventilator. He had permissive hypercapnia and we also observed the following values: PaCO₂ 145 mmHg with pH 6.93 and VT 5 ml/kg. The patient was given 200 mEq of Sodium Bicarbonate 1M, intravenous and inhaled salbutamol, two 0.2 mg doses of intravenous adrenalin, 1g of magnesium sulphate, methyl prednisolone and euphylline. The patient was sedated with propofol and was relaxed using cisatracurium. After 8 hours his PP was 32 cmH₂O with pH 7.07, Pa-CO₂ 98 mmHg, bicarbonate of 21 and O₂ Sat 92%. The patient was intubated for 12 days and the chest tubes were taken out on the 15th day. He was discharged after 17 days in the ICU and 8 days as an in-patient. Spontaneous BP is a rare condition which makes up

1%-4% of pneumothorax cases². Trauma, central venous catheterisation, intubation, tuberculosis, pulmonary tumours³, menstruation, pregnancy, sarcoidosis, radiation therapy, Marfan syndrome⁴, subpleural bullae^{5,6} and pulmonary emphysema⁴ are common causes of BP.

Two cases of spontaneous unilateral pneumothorax secondary to a severe asthma attack in patients with spontaneous breathing^{7,8} have been published. Another two cases of tension bilateral pneumothorax have been described in patients with from severe asthma attacks during cardiac arrest in whom the final cause of pneumothorax was not clear⁹.

The differential diagnosis between tension pneumothorax and an asthma attack is difficult because of the clinical similarities and symptoms. Chest pain, tracheal deviation, venous distension and low blood pressure are extremely rare in patients with spontaneous breathing¹⁰.

In an asthmatic patient, hypoventilation may be a sign of a bronchospasm, pulmonary hyper-inflation or pneumothorax. Asymmetric signs suggest the latter which can be ruled out after a chest x-ray.

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The treatment of community-acquired pneumonia

Dear Sir,

Having spent some time reading the letter sent in by Pacios et al¹, I was quite shocked about the references made to

the treatment of community-acquired pneumonia. I read this letter quite carefully because all topics related to pneumonia are always of interest to me, and I was shocked because attributes certain statements to me that I have never written. In the article that this letter refers to, which is titled "How and when should we use third generation cephalosporins for respiratory infections in the emergency department?", my suggestions, which were summarised in CAN, were as follows: a 400 g dose of oral cefditoren pivoxil every 12 hours for two weeks for treating pneumonia caused by pneumococcus (with positive antigenuria) in patients that do not need to be admitted to hospital, and as a substitute for ceftriaxone or cefotaxime in the case of follow up treatment. I advised that in cases involving acute exacerbations of COPD, cefditoren may work in the same way as amoxicillin/clavulanic and can be used as an alternative².

I am not of the opinion that pneumonia that requires hospital admission should only be treated with betalactamic drugs. Recent IDSA/ATS³ recommendations suggest using betalactamic drugs with azithromycin because this can reduce the patient's stay in hospital (evidence level I)⁴.

Unfortunately, I do not agree with the bacteriological argument for the reasons I am about to explain. Traditionally, penicillin has been used as a susceptibility marker of *Streptococcus pneumoniae*, which does not mean that strains with a certain MIC to penicillin have the same MIC to other antibiotics. It seems to me that Pacios believes that *Streptococcus pneumoniae* with a MIC ≥ 2 µg/ml to penicillin is also resistant to Cefditoren, which is not correct.

The most recent studies published show that Cefditoren has a MIC₉₀ of 0.5 µg/ml against *Streptococcus pneumoniae*, including strains with reduced sensitivity to penicillin (intermediate and resistant) which means that 96% of isolated pneumococcus, including those which have a high resistance (MIC ≥ 2 µg/ml), would respond to Cefditoren^{5,6}.

We also must not overlook *Haemophilus influenzae* as the main pathogen responsible for community respiratory infections given that the increase in BLNAR⁷ strains recorded should be considered when dealing with these infections. A recent study⁸ show that only Cefditoren is effective against *Haemophilus influenzae* regardless of the resistance phenotype, which is something that cannot be said of the other antibiotics studied like cefuroxime or amoxicillin-clavulanic acid.

One of the factors for predicting the effectiveness of betalactamic drugs is the mean time above breakpoint MIC₉₀, which is 40% above the dose interval. In the case of Cefditoren for *Streptococcus pneumoniae*, the mean time that the plasma concentrations are above MIC₉₀ after a dose of 400 mg is over 40% even for MIC values of 1 µg/ml⁹. The latter coincides with the values that appear in the information sheet for Cefditoren approved by the Spanish Drug Agency which are < 0.5 µg/ml for sensitive strains and >2 µg/ml for resistant strains.

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Hypothermia and Osborn waves

Dear Sir,

A 75-year-old male patient with advanced vascular dementia was brought to the emergency department because his level of consciousness had deteriorated. When the patient underwent a physical examination he was lethargic, with blood pressure at 80/55 mm Hg and a body temperature of $< 34^{\circ}\text{C}$. The electrocardiogram showed the typical signs of hypother-



Figure 1. An ECG of twelve derivations where we can see the Osborn waves (arrow).

mia: sinus bradycardia, prolonged PR interval, prolonged QTc and Osborn waves (arrow) in all derivations.

The Osborn wave, also known as the J-wave, is the most typical indication of hypothermia in electrocardiograms¹.

The Osborn wave is a positive deflection occurring between the QRS complex and ST segment (point J), which is clearer in middle and left precordial derivations which includes left ventricular depolarisation. This is almost always present when body temperature is below 32°C and its size and duration are linked to the level of seriousness of the hypothermia. When the J-wave is very high, the T-wave is usually inverted in the same derivation. Although it is a typical indicator of hypothermia, Osborn waves are not exclusively associated with hypothermia and can also be found when dealing with normothermic patients suffering from acute brain damage, hypocalcaemia or haloperidol overdose^{2,3}.

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