Chicken pox with multiorgan involvement

Sir,

Chickenpox is a worldwide contagious infectious disease caused by primary infection with varicella zoster virus (VZV) or human herpesvirus 3 and is characteristic of childhood[1]. It is usually a mild disease, benign and self-limiting, characterized by the appearance of a prodrome followed by a generalized vesicular rash about 15 days after exposure to the virus. Complications can involve the skin (bacterial superinfection of the lesions), the nervous system (meningitis, encephalitis), the respiratory system (pneumonitis) and, rarely, multiorgan involvement[2,3].

A 55 year-old man without relevant medical history consulted the emergency department (ED) for fever up to 39°C during 10 days, general malaise, central chest pain which increased on coughing and deep breathing, generalized cutaneous purpura and mucous bleeding when touched (Figure 1). He was diagnosed with chickenpox. Physical examination showed a blood pressure of 125/89 mmHg, temperature 36.5°C and oxygen saturation 94%. He presented isolated hissing and crackling and selective pain on palpation of the epigastrium and the skin lesions. Laboratory tests showed arterial oxygen pressure of 69 mmHg, creatine kinase (CK) 419 IU/L, CK-MB 50 IU/L, troponin I level 0.139 ng/ml due to probable myocarditis, a pattern of hepatitis (ALT 109 IU/L, GGT 315 IU/L, GPT 90 IU/L, FA 218 IU/L, LDH 2161 IU/L); leukocytes 9.700/mm^3 (neutrophils 84.6%); and platelet count was 53.000/mm^3. Chest radiography showed multiple alveolar nodular confluent opacities with a diffuse, bilateral distribution, consistent with pneumonitis (Figure 2). The electrocardiogram (ECG) showed sinus rhythm of 93 bpm, and left bundle-branch block. The diagnosis was confirmed by positive serology for VZV and other microbiological tests were negative. Echocardiogram and abdominal ultrasound abdomen were normal. He received intravenous acyclovir and ceftriaxone, the latter being added for extra antibiotic coverage and to prevent possible secondary superinfection, and the patient evolved favorably. Final diagnosis was chickenpox with visceral involvement (pneumonia, myocarditis, hepatitis) with hypoxemia and thrombocytopenia.

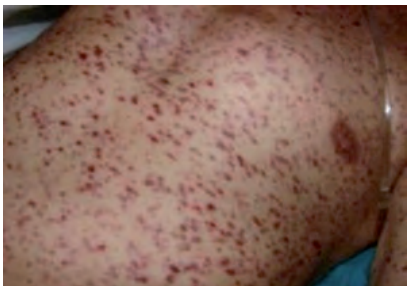

Figure 1. Image showing purpuric, polymorphic and widespread skin lesions.

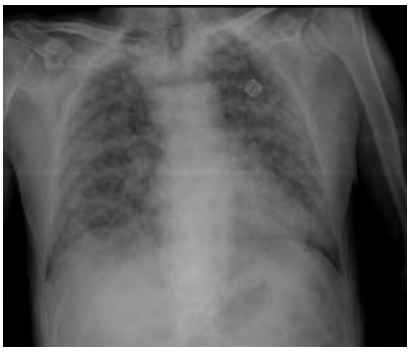

Figure 2. Chest X-ray showing multiple alveolar nodular confluent opacities with a diffuse, bilateral distribution

This case illustrates highly unusual multiorgan involvement. The most important complication was varicella pneumonia (with an estimated mortality of 10-30% in immunocompetent patients, and up to 50% in immunocompromised patients)[4] which was successfully resolved by the early administration intravenous acyclovir and ceftriaxone. Altered myocardial enzymes and the chest pain were attributed to myocarditis, a rare complication which may present with arrhythmia and/or heart block associated with sudden death[5,6]. Unusually, the patient also had hepatitis, which in immunocompetent patients is usually mild and evolves satisfactorily[7]. However, severe cases of autoimmune hepatitis triggered by VZV have been described in the medical literature[8]. In conclusion, the prevalence of chickenpox in adults is low but extremely serious multiorgan complications can develop if antibiotic treatment is not started early. For this reason chickenpox should be considered in the differential diagnosis of any exanthematous illness attended in the ED. We would highlight the importance of thorough anamnesis, since this is the main tool allowing us to establish the initial suspected diagnosis.

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Blood transfusion in the emergency

Sir,

We have read with great interest a recent insightful letter by Madrazo

et al.[1] on the allegedly poor effectiveness of allogeneic blood transfusion (ABT). We share with the authors their concern about the habitual lack of attention given to anemia, little studied and scarcely treated with corrective measures. We also share their view on the lax use of ABT, without universal application of restrictive criteria established by national and international guidelines[2,3], and the non-application of alternative measures to ABT despite current evidence and national consensus documents[4].

However, we disagree when Madrazo *et al.* claim that the "ultimate objective of ABT is to rapidly supply the tissues with oxygen and prevent and/or correct the consequences of hypoxia"[1]. The objective should be the correction of tissue hypoxia and its symptoms or signs that appear when compensatory mechanisms fail in any type of anemia[5]. ABT, by increasing red cell mass, attempts to increase transport. But this does not ensure increased transfer of oxygen to the tissues, because the relationship between oxygenation and transport is not linear; under normal conditions only one quarter to one fifth of the oxygen transported is supplied (and consumed). Only when a "critical point" is reached, which is about 12-15% hematocrit in healthy volunteers[6], does consumption and transport show a linear relationship, and then the benefits of ABT become evident[5-7].

Madrazo *et al.*[1] commented that in the absence of more reliable physiological indicators regarding oxygen supply and consumption by cells and tissue, the concentration of hemoglobin and the percentage of hematocrit are two key biological parameters to estimate transfusion needs. However, certain groups have shown that oxygen tissue pressure can be monitored and used as the most reliable parameter of ABT need and effectiveness[8]. This is the objective of currently developing transdermal devices for non-invasive monitoring of tissue hypoxia in critical patients[5].

The authors also express concern about the variable quality of packed red blood cells (RBC) and progressive reduction of viability[1]. An accepted classical criterion of viability is that three quarters of the RBCs transfused continue circulating at 24 hours. In donated RBCs after 42 days of refrigerated storage in non-physiological conditions, 33% are senescent and the remainder malfunctioning. This could explain the increased morbidity and mortality of patients with heart or onco-hematological disease or critical processes when transfused with "old" RBCs. This ethical problem of "old" blood underlies proposals to reduce the expiry period to 28 days, and to 15 days for pediatric patients or those with cancer and heart disease. The logistical problems of probable shortages have not allowed the health authorities to take this logical sPTE to date.

We agree with the authors about anemia being highly prevalent in emergency services, and that management should involve a multidisciplinary approach supported by an effective therapeutic arsenal, optimizing available resources[2,4] and treating each patient according to their particular characteristics; ABT should only be administered when shown to be necessary[3,7]. It is worth recalling that the effect of ABT treatment is transient, that deficiencies will re-occur unless the cause is properly identified and corrected whenever possible, and treatment must be personalized, with ABT administered only after individualized evaluation[3].

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Authors' reply

Sir,

We appreciate the interest shown by Dr. García-Erce *et al.* in our letter and fully agree with their definition of the ultimate goals of allogeneic blood transfusion (ABT). Our letter was basically to express our concern about the indiscriminate use of ABT, applying strictly "numerical" criteria often based on little (or at least questionable) scientific evidence, and about the generally low level of interest shown in a disease or comorbidity as prevalent and relevant as anemia[1,2]. The scientific community recommends applying restrictive transfusion criteria with selected hemoderivatives selected for each patient when ABT is necessary, and consideration of effective alternative therapies[3-7]. Hemoglobin and hematocrit values are classically used as indicators for ABT, ignoring (as Garcia-Erce *et al.* point out) the Individual physiological response and compensatory mechanisms[8-10]. We look forward to the validation of new "physiological" indicators for ABT - tissue consumption and oxygenation parameters , not only transport - for application in routine practice, trusting they will prove more reliable than the simple determination of hemoglobin[8-11]. As indicated by García-Erce *et al.,* promising results are being obtained with biological parameters in this field (tissue O_2 pressure, O_2 extraction rate, P300 cerebral latency, gastric intramucosal pH and others), and we may soon be witnessing a more judicious use

of a resource as valuable (and scarce) as ABT[11].

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Noninvasive mechanical ventilation of an immunocompromised patient with community-acquired pneumonia and multiorgan failure

Sir,

Noninvasive mechanical ventilation (NIV) has been used for years in the emergency department (ED) for patients with acute respiratory illness (ARI)[1] who do not respond to oxygen therapy. Scientific evidence supports its use in chronic obstructive lung disease (COPD), acute cardiogenic pulmonary edema and immunocompromised patients, but in patients with community acquired pneumonia (CAP) its indication is controversial[2].

A 33 year old man consulted the ED with fever (39.5 °C), cough and hemoptysis, accompanied by progressive dyspnea during one week. Medical history included heavy smoking, occasional consumption of cocaine and 3 episodes of herpes zoster. His partner was seropositive for HIV. On ED arrival he was febrile, tachycardic, tachypneic and hypotensive; pulse oximetry showed an SaO_2 of 85% without oxygen therapy. Laboratory tests showed neutrophilic leukocytosis, C-reactive protein (CRP) 315.7 mg/L, PaO_2/FiO_2 < 200 without hypercapnia, plasma creatinine 1.53 mg/dl, and coagulopathy. Chest X-ray showed an alveolar infiltrate in the right hemithorax and left lower lobe (Figure 1). Diagnosis was CAP with severe multiorgan dysfunction. Hemodynamic support was initiated with fluid and noradrenaline as well as antibiotic therapy with cefepime and levofloxacin. He presented hypoxemia refractory to standard oxygen therapy, so it was decided to evaluate his response to NIV given the suspected immunodeficiency, and postpone invasive mechanical ventilation with tracheal intubation (IMV). Ventilatory support was performed with NIV (see figure), using inspiratory pressure 14 cmH_2O, end tidal positive pressure of 6 cmH_2O and FiO_2 60%. Blood and urine cultures were positive for *Streptococcus pneumoniae* and HIV serology showed a viral load of 70,200 copies/ml and a CD4 lymphocyte count of 49. The patient showed rapidly progressive clinical improvement; the inotropics were suspended on day 3 after admission and NIV was withdrawn on day 5. Coagulopathy was gradually corrected without requiring transfusion and renal function normalized: the patient was discharged 10 days after hospital admission.

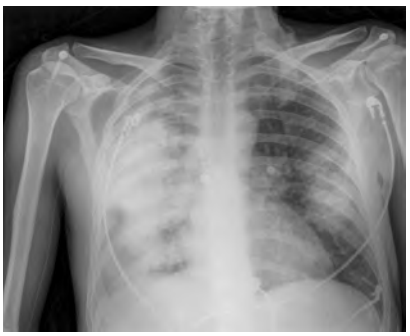

Figure 1. Chest-x ray showing alveolar infiltrates in the right hemi-thorax and left lower lobe.

Our case illustrates the positive response to NIV in an immunosuppressed patient with CAP, pneumococcal bacteremia, and multiorgan dysfunction. Since the trachea is not invaded and sedation is not required, NIV reduces the risk of nosocomial infection and hospital stay times, which is especially important in severely immunocompromised patients[3,4]. From the first hour of therapy it can be seen if there is response to NIV, by assessing improvement in three areas: gas exchange, work of breathing and subjective dyspnea[5,6]. Hence the importance of intensive monitoring of evolution. If no improvement is evident, IVM without delay is recommended[7]. Experience of the care team in the management of NIV, close monitoring of response to therapy and technical improvements in the new NIV ventilators are factors that support more widespread use of this technique in selected patients with severe CAP and immunosuppression in the context of a life-threatening emergency, given the high mortality of patients requiring VMI in these cases[2,8,9].

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Multiorgan dysfunction in a patient with enteritis due to *Strongyloides stercoralis*

Sir,

Strongyloides stercoralis (SS) is a cosmopolitan nematode, endemic to tropical and sub-tropical rural regions. In Spain its prevalence is probably underestimated; the area of highest incidence is the Mediterranean coast[2]. SS has an autoinfection cycle with the capacity of chronic parasitism during decades, usually being asymptomatic or producing mild gastrointestinal symptoms. In immunocompromised patients it may produce hyperinfection manifesting in fever, gastrointestinal symptoms, pneumonia, petechial purpura, meningitis and septic shock. Attributable mortality is 30-80% depending on early or late diagnosis and treatment[3-5].

We report the case of a 40 year-old Ecuadorian man, resident in Spain for eight years, with a history of glioblastoma multiforme treated by surgery, radiotherapy and chemotherapy with temozolomide and dexamethasone. He consulted for green-colored diarrhea with epigastric pain and vomiting. He denied recent travel or bad food intake. Physical examination showed blood pressure of 80/55 mmHg, heart rate 130 bpm, respiratory rate 33 rpm, dehydration and diffuse abdominal pain without signs of peritoneal irritation. Laboratory tests showed: leukocytes 14.880/µL (11.400/µL neutrophils, 800/µL eosinophils), hemoglobin 19.6 g/dL, pH 7.25, bicarbonate 12.7 mEq/L, lactate 4.9 mmol/L and creatinine 4 mg/dl. Abdominal CT scan showed distended loops of the jejunum with thickened circumferential mucosa. In the ED, resuscitation was started with intravenous fluids and antibiotic therapy with piperacillin tazobactam, and blood and stool cultures were ordered. His condition showed no improvement and he was admitted to the intensive care unit, where hemodynamic monitoring showed a pattern of distributive shock. Intensive fluid therapy was continued and noradrenaline (0.3 ug/kg/min) and hydrocortisone (100 mg/8 h) were administered. The outcome was favorable with clinical stabilization and lab test improvement, but diarrhea persisted. In the study of feces, SS larvae were observed and treated with ivermectin, which resolved the diarrhea.

As in our case the main risk factor for severe SS infection is cellular immunosuppression (due to glucocorticoids and other immunosuppressants)[5]. In chronic forms, detection of the parasite in feces is low, unlike in other forms or in severe hyperinfection6. Serology may be falsely negative in immunocompromised patients. Diagnosis in Spain is often delayed and the consequences can be fatal. In addition to early diagnosis and proper management of severe sepsis[7], the treatment of choice is ivermectin in two single consecutive doses (200 mg/kg/day orally)[8]. In cases of hyperinfection the necessary dosage is unknown but some authors suggest treatment during 7 days. For primary prevention SS should be studied and treated in all patients with epidemiological risk factors and those receiving immunosuppressive therapy.

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Angioedema of the tongue related to enalaparil

Sir,

We read with interest the article by Cordoba *et al.* describing the case of a hypertensive patient who developed a lingual and oral cavity edema within 24 h of initiating treatment with enalapril[1]. Although the relationship between the occurrence of angioedema and angiotensin-converting enzyme inhibitors (ACEI) is well established, it seems to be not well known among physicians[3]. While the scenario described by the authors is the most common, it is not unusual for angioedema to appear years after starting ACEI therapy[4,5]. This means that when patients present with lingual edema, treatment with ACEI may be maintained, showing ignorance of its pathogenic role in angioedema[3,5]. In a review by Tocornal *et al.* of 5 cases of enalapril-induced angioedema, most of the patients had had 1 to 7 episodes before enalapril was suspended, and time to onset of symptoms ranged from 1 to 96 months[6].

Recently, an 80 year-old woman with a history of hypertension consulted the ED of our center; she reported that three years before she had had an episode of lingual edema 5 months after starting treatment with enalapril 20 mg/day, which resolved with conventional treatment, although enalapril was not discontinued. This time the patient had a large lingual edema and mild stridor without signs of bronchospasm. She reported no new medication. The symptoms resolved after receiving hydrocortisone 100 mg/6 h iv with dexchlorpheniramine 5 mg/8 h iv and withdrawal of enalapril; finally, after more than 48 h in the observation area, the patient was discharged. Regarding her hypertension, treatment was initiated with oral amlodipine 5 mg/12 h, one of the alternative therapies.

With this comment on the case published in emergencies we wish to illustrate that the causal link between enalapril and angioedema is often not as clear as in the case presented by Cordoba *et al.* The potential involvement of ACEI therapy should be considered in all cases of angioedema regardless of the period of treatment time.

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Acute bilateral pulmonary emboli and complete atrioventricular block: an atypical association

Sir,

Pulmonary thromboembolism (PTE) includes deep vein thrombosis (DVT) and pulmonary embolism; 90% of PET originate with DVT. PTE is a severe entity with non-specific clinical presentation which may range from breathlessness, chest pain or hypotension to an absence of symptoms. Diagnosis therefore requires a high index of suspicion[1]. PTE may be massive or sub-massive, according to whether vascular obstruction is greater or less than 50% (arteriographic criteria) or whether there is shock or not (clinical criteria). Predisposing factors include advanced age, previous PTE, cancer, pregnancy, surgery, multiple trauma, immobilization, thrombophilia and oral contraception[2]. One fifth of cases are idiopathic[2]. Electrocardiographic manifestations are non-specific. The most frequent is sinus tachycardia and changes in the T wave and the ST segment. The S1Q3T3 pattern, right branch block and right shift of the QRS complex is observed in 26% of PTE[3]. For the therapeutic approach, initial risk stratification is clinical: high risk (shock or hypotension, systolic blood pressure (SBP) < 90 mmHg); intermediate risk (normotensive, with right ventricular (RV) dysfunction or overload; and low risk (normotensive without RV dysfunction)[2].

A 46 year-old man with no relevant history consulted the ED for left popliteal pain during several days. Physical examination showed a swollen left leg and bradycardia of 35 bpm. Doppler ultrasound of the lower limbs showed DVT in the popliteal and superficial femoral veins; D-dimer was 2113 mg/l. The ECG showed 2nd degree atrioventricular (AV) block Mobitz type II (2:1) and incomplete right bundle branch block. Given these findings, we performed chest CT scan with contrast, which showed bilateral massive PTE. He was admitted to the intensive care unit where transthoracic echocardiography (TTE) showed a dilated RV with preserved ejection fraction (EF). The patient was treated with low molecular weight heparin (LMWH) and an external pacemaker (PM). Fibrinolysis was not used since he was classified as intermediate-risk 2. Study of thrombophilia, polycythemia, myocardial ischemia and tumor markers showed no alterations. During his stay, the patient was hypertensive (blood pressure 180/90 mmHg) and alternated between complete AV block and Mobitz II (2:1) 2nd degree AV block. His past medical history included left bundle branch block (LBBB). After six days of anticoagulation, complete AVB persisted and a type DDD pacemaker was implanted. On control TTE, RV was not dilated.

The finding of this PTE was fortuitous; although massive, it was asymptomatic, and was only suspected because of the high D-dimer value and the AV block. The association between PTE and complete AV block is uncommon, with few cases reported in the literature[3-5]. Several theories may explain complete AV block in PTE: by vagal reflex causing hypotension and AV conduction delay, which is transient[3]; previous LBBB together with RBBB due to PTE produces complete AV block[4,5] or ischemia secondary to shock[3]. In our case, there is doubt as to whether the association between PTE and complete AV block was causal or not. An ischemic origin was ruled out (normal enzymes), as was a vagal reflex (no hypotension and the AV block was not transient). The previous LBBB could be causally related. There are ECG alterations in 70-80% of PTE patients. Several ECG scoring scales exist to stratify the risk of PTE and to guide treatment[6]. The higher the score, the greater the RV hypokinesia and the worse the prognosis[7]. So, with DVT confirmed, despite the absence of PTE symptoms, we should always perform an ECG; if there are signs of RV overload and D-dimer is high (> 500 mg/l), the diagnosis of PTE should be considered.

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