scribed as isolated and infrequent (1/100-1000 of patients);[4] it seems to be present upon the first administrations and subsequently dissapears.[5] The symptoms described by the patient as a burning sensation of the tongue and peribuccal hyperaesthesia, although subject to subjective assessments, did not correspond to the neurological disorders commonly described as paraesthesia or taste disorders[4]. The clinical profile that our patient presented seemed to signal an anaphylactoid reaction which was not confirmed. Generally speaking, reactions secondary to intravenous iron have been attributed to rapid infusion with an oversaturation of transferrin and the release of free iron, which is responsible for toxicity and vasomotor reactions.[6] This limited the total dose of iron administered and the rate of infusion in older formulations. Although ferric carboxymaltose is in this sense better tolerated, there are few studies comparing it to the rest of the formulacions.[7] Furthermore, the potential development of severe, even fatal adverse effects remains a source of concern. Many of these reactions have been associated with high molecular weight iron dextran preparations and seem to have an immunological base[1,7]. However, controlled clinical trials for different intravenous iron preparations are limited by design to detect rare adverse effects as they are conducted in a small number of patients over short follow-up periods.[1,2]

Given the manifestation of infrequent side effects, it is vitally important that the intensity and seriousness of the reaction be established since this may require the permanent suspension of the drug, with the resulting limitation of the therapeutic arsenal available.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Emphysematous cystitis

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To the Editor:

We present the case of an 86-year-old female with a history of poorly controlled type 2 diabetes mellitus, who sought care for an episode of arterial hypotension, fever, dysuria, and acute renal failure in the context of sepsis of a urinary aetiology. Laboratory tests revealed the following values: leukocytes: 27 200x10e3/μl; glucose: 170mg/dl; creatinine: 5.1mg/dl; urea: 217mg/dl; and C-reactive protein: 232mg/l. An abdominal x-ray revealed gas surrounding the urine bladder, indicative of emphysematous cystitis (Figure 1 A).

Cultures of tissue samples did not reveal microbiological growth. We started treatment with intravenous hydration, insulin, and antibiotics with meropenem, which, along with catheterisation of the urine bladder, produced an adequate response. The patient recovered after three weeks of antibiotic treatment, with no complications and with normalisation of renal function (Figure 1 B).

Emphysematous cystitis is a rare, progressive, and fatal disease if it is not detected early. More than 90% of cases of this disease occur in diabetic and immunodepressed patients. The most commonly involved microorganisms are *Escherichia coli* and *Klebsiella pneumoniae*. The mechanism through which gas is produced in emphysematous infections is not well understood. In diabetic patients, one reason could be the production of CO_2 by the microorganism through the glucose fermentation pathway, which occurs when glucose concentrations are high. The best diagnostic method is radiological imaging (simple x-ray or computed axial tomography). This potentially fatal and rare complication must be kept in mind during patient diagnosis, especially in elderly diabetic patients with urinary tract infections.

Conservative treatment with antibiotics and catheterisation of the bladder is generally successful, with a rate of complications <20%. This strategy reduces patient mortality without requiring surgical interventions, and aids in preserving renal function.

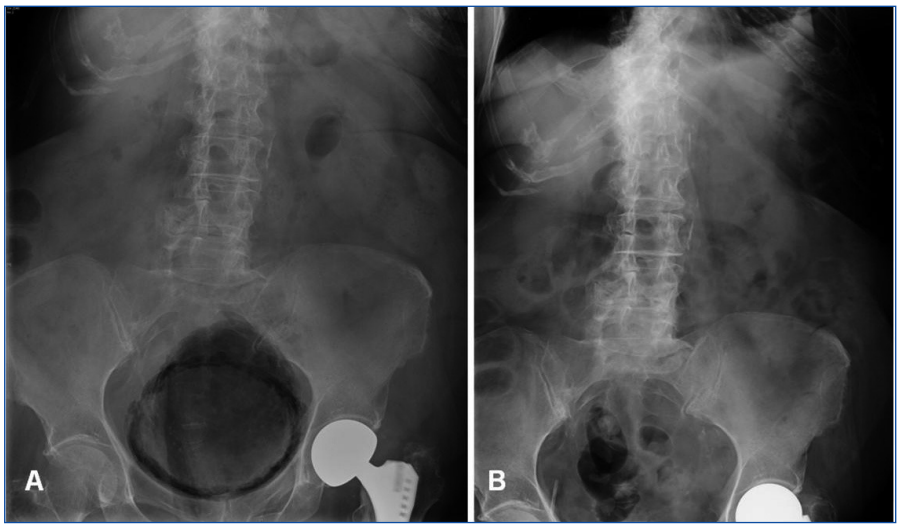

Figure 1. Emphysematous cystitis.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Recurrent cutaneous necrosis of a multifactorial origin in a patient on haemodialysis

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To the Editor:

We present the case of a 40-year-old female, who was an ex-smoker until 2010, when she retook the habit. The patient suffered from lupus with joint, haematological, and pericardial manifestations; the patient also suffered from anti-phospholipid syndrome with arterial manifestations (cerebrovascular accident, renal artery stenosis, and stenosis of the arteriovenous fistula [AVF]), poorly controlled arterial hypertension, advanced chronic kidney disease (CKD) secondary to focal and segmental hyalinosis (insufficient evidence to rule out the coexistence of type V lupus nephritis), and on haemodialysis treatment since 1990. The patient received a kidney transplant in 1991, but restarted dialysis in 1998 after a recurrence of the underlying disease, which coincided with pregnancy. Severe tertiary hyperparathyroidism with regular/poorly controlled phosphorous levels for several years (variable compliance with treatment) and osteoporosis of trabecular bone (t-score: -2.5; z-score: -2.3). The patient also exhibited hyperhomocysteinemia, hyperuricemia, and a hidden hepatitis C infection with haemosiderosis. The right humerobasilic AVF was functional. Normal treatment included cinacalcet, pepsamar, calcium acetate, risedronate, folic acid, polyvitamin B1/6/12, omeprazole, allopurinol, carvedilol, hydroxychloroquine, and acenocoumarol.

CURRENT DISEASE

In September 2009, acenocoumarol was replaced with tinzaparin (anti-activated factor X: 0.8-1.2IU/ml) due to upper gastrointestinal bleeding, and the patient remained on this treatment regimen for one year. In November 2010, she developed purple lesions on the pad of the third finger of the left hand, contralateral to the AVF (Figure 1 A), which were cold and painful to the touch. Radial pulse was normal. An ipsilateral Doppler ultrasound revealed **mild calcified atheromatosis of the axillary and brachial arteries, with no haemodynamically significant stenosis**. During the first 24 hours, acenocoumarol was reinitiated, and topical nitroglycerin was added to the treatment regimen. An INR of 4 was reached in 72 hours, which allowed for the suspension of tinzaparin. Five days after the start of symptoms, the patient complained of intense pain in the finger and a slow progression towards cutaneous necrosis and a new lesion with similar ischaemic characteristics to the second ipsilateral finger. Treatment was started with antibiotic therapy and alprostadil (500ug i.v. over 10 doses). Dicoumarin was suspended, and tinzaparin was recommenced. We took a