cles for *Treponema pallidum*, TPPA (agglutination assay) and EIA (enzyme immunoassay).

These tests are still reactive for life in virtually all individuals regardless of previous treatment.

The treatment recommended by the Centres for Disease Control and Prevention (CDC), including ocular syphilis are:[10] aqueous crystalline penicillin G (3 to 4 million units intravenously every four hours, or 24 million units per day as a continuous infusion) for 10 to 14 days, or procaine penicillin G plus probenecid. Doxycycline or ceftriaxone at high doses could be used as an alternative to penicilin.[11]

Follow-up should be carried out three and six months after treatment and then every six months until the CSF is normal and VDRL is unresponsive. Treatment must be repeated if any CSF follow-up sample increases four times in the VDRL titre or if there is an increase in leukocytes.[8]

CONCLUSION

Syphilis is an infectious disease which is not very prevalent in our setting and as such, is not often suspected.

Neurosyphilis has been considered a late manifestation of syphilis. In the case of our patient, the presence of a clinical profile, in which the symptoms of secondary syphilis (skin involvement) and neurological symptoms occur almost simultaneously is striking. We suspect that this is a consequence of the fact that it involves a patient on immunosuppression due to renal transplant anti-rejection medication.

We believe that, in addition to the lack of prevention by the patient, it is very important to consider immunodeficiency acquired by the renal transplant itself and we believe that it is an excellent opportunity to highlight the susceptibility of these patients to exposure to these type of microbiological agents; in cases of immune competitiveness, the presentation of this type of disease is probably not very aggressive.

Conflicts of interest

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

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Hydroelectrolytic disorders secondary to refeeding syndrome

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To the Editor:

The refeeding syndrome (RFS) is a severe hydroelectrolytic diorder, which is generated following dietary supplementation in patients with major basic nutritional deprivation. In this syndrome, multiple electrolyte disorders, vitamin deficiencies and severe cardiovascular effects appear.[1]

We report the case of a 70-year-old female with high blood pressure and endometrial adenocarcinoma diagnosed in 2010, who was treated with hysterectomy, chemotherapy and pelvic radiation

therapy. About a month after completing chemotherapy, she was admitted with febrile neutropenia, ileal enteritis and *E. coli* bacteraemia. Tests highlighted severe leukopenia ($0.22x10^9$/l, 0 neutrophils), creatinine 70.2mmol/l sodium 139mmol/l, potassium 3.74mmol/l, serum calcium 2.45mmol/l, serum phosphate 1.2mmol/l, metabolic acidosis (pH 7.27, pCO_2 45mmHg, bicarbonates 20.7mmol/l) and hypoalbuminaemia (32mg/l).

Bowel rest, intravenous fluid therapy, empirical antibiotic therapy and granulocyte colony-stimulating factor are introduced. After slow abdominal evolution, she started on parenteral nutrition (PN) on the third day with intake of 1500kcal/day. After 24 hours, she presented neurological symptoms with generalised tremors, hyperreflexia, Chvostek and Trousseau signs, peripheral oedema and QTc prolongation on electrocardiogram. Tests highlighted hypocalcaemia (1.6mmol/l-1.76 albumin-corrected), hypomagnesaemia (0.45mmol/l), hypophosphataemia (0.68mmol/l), hypokalaemia (2.59mmol/l), normal renal function and acid-base balance with pH 7.41, pCO_2 31mmHg, pO_2 48mmHg and bicarbonate 19.6mmol/l. Given the temporal relationship with the start of PN, RFS was suggested as the clinical profile.

DISCUSSION

RFS is a series of hydroelectrolytic disorders (hypophosphataemia, hypomagnesaemia, hypokalaemia) that may occur after initiation of enteral or parenteral nutrition with high caloric intake, and that may have life-threatening consequences. The condition of chronic malnutrition (marasmus, malabsorption syndrome, chronic alcoholism, multiple comorbidities in elderly patients and morbidly obese patients after bariatric surgery), anorexia nervosa, poorly controlled diabetes mellitus, chronic use of antacids, high metabolic stress lasting more than 7 days, recent surgery and cancer are predisposing factors for developing RFS.[2] In these circumstances, the decrease of baseline metabolism and insulin levels produce a number of adaptive mechanisms that lead to increased protein catabolism and fat metabolism with ketone body generation and enhancement of gluconeogenesis as an energy generation source (Figure 1).[3]

After refeeding, nutrient availability generated increased levels of insulin with the subsequent introduction of phosphorus, potassium, magnesium and thiamine intracellularly, used to reactivate the glycolysis process. These components decreased rapidly in plasma, which added to metabolic and water overload in a depressed baseline myocardium, produces serious clinical consequences (Table 1).[2]

RFS treatment focuses on prevention. The most important steps are to identify the patients at risk, start nutrition with a low energy intake (20kcal/kg/day or 1000kcal/day) and gradually introduce the requirements for a period of one week. If ionic disturbances are present, they should be corrected before starting refeeding. NICE[4] also recommends thiamine intake. When signs and symptoms of RFS appear, nutrition should be discontinued, and early correction of electrolyte abnormalities as well as additional supportive measures must be carried out according to the profile (vasopressors, oxygen, diuretics, etc...).[4]

Our case dealt with a patient with multiple comorbidities, cancer, with a high risk of suffering RFS, who started PN with standard caloric intake, which resulted in hypophosphataemia, hypokalaemia, hypomagnesaemia (secondary hypocalcaemia) and hypervolaemia, with neurological consequences and electrocardiography repercussions. Caloric intake decreased to 1000kcal per day (but did not stop) as well as the volume of intravenous fluid therapy. Corrective treatment of the electrolyte imbalances was started and thiamine supplements were administered, with improvement of neurological symptoms, peripheral oedema and correction of electrolyte imbalances.

RFS is a severe disorder and is avoidable, and as such, it should be consid-

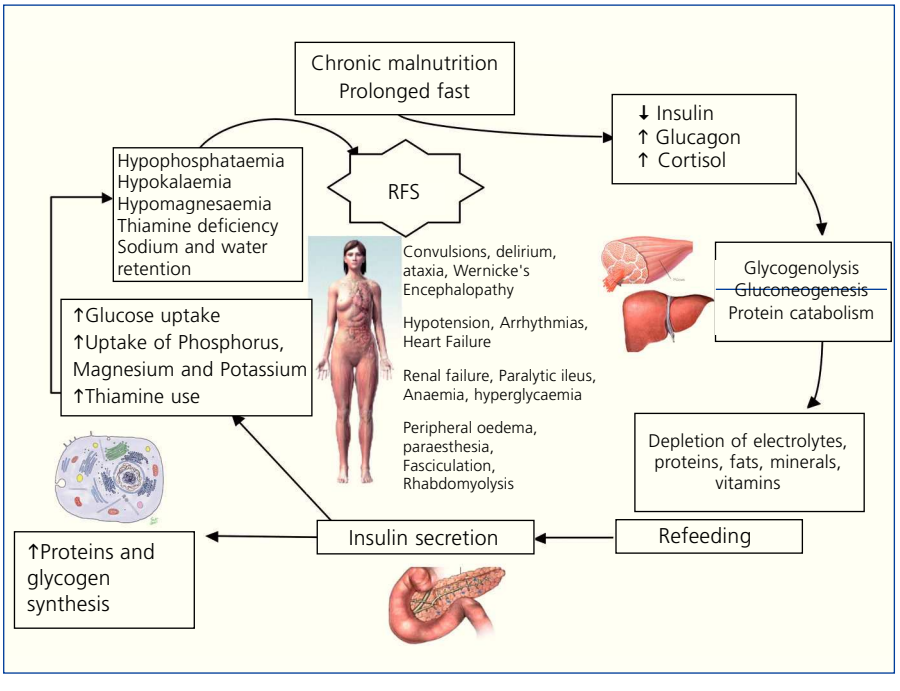

Figure 1. Refeeding syndrome physiopathology.
RFS: refeeding syndrome.

Table 1. Disorders and symptoms associated with refeeding syndrome

Hypophosphataemia	Nausea, vomiting, heart failure, arrythmias, haemolytic anaemia, pancytopenia, rhabdomyolysis, acute tubular necrosis, cranial nerve palsy, muscular paralysis, confusion, coma.
Hypomagnesaemia	Hypocalcaemia, arrhythmias, tachycardia, tremor, ataxia, confusion, irritability, paraesthesia, abdominal pain, convulsions, tetany.
Hypokalaemia	Arrhythmia, hyporeflexia, low blood pressure, paralytic ileus, paraesthesia, cramp, muscular paralysis, respiratory depression, myoglobinuria, polyuria, metabolic alkalosis.
Thiamine deficiency	Wernicke's encephalopathy Korsakoff's syndrome
Intolerance to carbohydrates, water intolerance	Hyperosmolar condition, fatty liver. Dehydration, water overload, peripheral oedema, heart failure, low blood pressure, prerenal renal failure, sudden death.

ered in all patients at high risk of early nutritional support.

Conflicts of interest

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

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Middle aortic syndrome as the cause of renovascular hypertension in a 3-year-old girl: difficulties in the differential diagnosis

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To the Editor:

High blood pressure (HBP) occurs in 1% of children,[1] and 10% of cases are renovascular in origin; fibromuscular dysplasia (FD), Takayasu arteritis (TA) and the middle aortic syndrome (MAS) are the most commonly associated aetiologies. These diseases pose difficulties in differential diagnosis due to their clinical similarities. This article describes the case of a girl with MAS as aetiology of HBP. Reports on this disease in paediatric age are scarce, as was its form of presentation and evolution.

CASE STUDY

Patient who was hospitalised when she was two years due to HTA associated with loss of strength and sensation in her lower limbs. The arteriography showed decrease in aortic diameter, 20% stenosis at the right renal *ostium*, critical stenosis of the left renal artery and no flow in the left lower renal pole. The decision was made to perform left renal autotransplantation with renal anastomosis of the iliac artery and biopsy of the renal artery, which reported findings consistent with FD (Figure 1). She was discharged with minoxidil and propranolol.

A year later, the patient is hospitalised due to hypertensive crisis. The renal scintigraphy showed exclusion of the left autotransplanted kidney and impaired perfusion of the right kidney. Due to suspected large vessel vasculitis type TA, paediatric rheumatology assessment was requested. Although the patient met the criteria for TA classification (abdominal aorta and renal arteries stenosis associated with HBP), prior biopsy showed no findings of vasculitis.

Further differential diagnoses pointed to FD but this was ruled out because FD renal lesions have a characteristic image of pearls necklace[2] and rarely affect the *ostium* or the proximal segments. In this patient, there was no image of pearls necklace and the involvement of the renal artery was at the proximal portion of the artery. Following these imaging findings, MAS was eventually diagnosed.

Another arteriography was performed, which reported irregular abdominal aorta with progressive distal thinning, arterial anastomosis autotransplant occlusion and progression of right renal artery stenosis (Figure 2); it was decided that a right renal