1Ra), which in turn would stimulate tumour necrosis factor alpha (TNFα) and interleukin 6 (IL-6) and the activation of NFκ-β which stimulates the production of SAA.[8]

In the case of our patient, given the long evolution of the HIV infection and long history of parenteral drug consumption, it would be impossible to discern the cause of the amyloidosis, which could be due to drug consumption and recurrent infections, the HIV infection, or perhaps the sum of all of these factors.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Acute renal failure secondary to cyclic vomiting syndrome

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To the Editor,

Cyclic vomiting syndrome (CVS) is a functional gastrointestinal disorder characterised by episodes of severe, unpredictable, and explosive vomiting, separated by intervals of perfect health.[1] The start of these symptoms can occur in infancy, and it normally appears between the ages of three and seven years, although cases have been described when symptoms commence in adulthood.[2]

The aetiology and pathogenesis of this condition are still unknown, although the hypothesis has been put forward of a disorder of the cerebro-intestinal communication, which is activated by certain stimuli (stress, infection, some foods).[3] The most common duration of an episode can be one to four days, and may last as long as 14 days. During each episode, vomiting occurs as frequently as every 10 to 15 minutes, and can occur anywhere from several times a year to several times per month, with a regular recurrence rate.

The symptoms include vomiting preceded by forceful gagging and abdominal muscular contractions, accompanied by uncontrollable nausea and extreme fatigue. Patients suffer a sort of "conscious coma" during each episode, and describe themselves as being in a state of stupor until the episode passes.[4] Among the most common complications are dehydration, electrolyte disorders, improper secretion of anti-diuretic hormone (ADH), and oesophagitis.[4]

The optimal treatment for this condition consists of establishing prophylaxis with anti-migraine medications such as amitriptyline along with propranolol. In the prodromal phase, we must attempt to abort the episode using ketorolac or sumatriptan. In the acute phase, ondansetron or lorazepam are used, with chlorpromazine, promethazine, or intravenous morphine as the possible alternatives.[4] The patient occasionally must be sedated in order to assuage the unstoppable vomiting.

Here we present the case report of a 31-year old male who has suffered from crises of nausea, uncontrollable vomiting, and abdominal distress associated with prodromal nervousness since infancy (three-five years old), frequently related to triggering factors such as emotional stress and infections. The patient later had symptom-free periods with variable frequency. He was diagnosed with periodical functional syndrome with uncontrollable vomiting and erosive oesophagitis at the age of 14 years, which persisted in spite of treatment with chlorpromazine. After being examined by several different specialists, the patient was diagnosed three years ago with CVS. The patient takes a prophylactic dose of 20mg propranolol (half at breakfast, half at dinner) and 75mg amitriptyline (half tablet at dinner), abortive therapy consists of microenemas with diazepam, and during acute crises he takes ondansetron at 4mg every 8 hours, one vial of lorazepam every 8 hours, and one vial of chlorpromazine intravenously every six-eight hours or promethazine at 50mg every six-eight hours, in the hospital.

Since one year ago the patient has required three hospitalisations due

to complicated crises with hydroelectric imbalance and acute renal failure. The last episode caused intense dehydration with prerenal acute renal failure, with creatinine of 2.2mg/dl, K at 2.9mEq/l, metabolic alkalosis, and a urinary infection that may have triggered the episode. We started the patient on aggressive hydration therapy and antibiotics, and had to sedate him with chlorpromazine at half a vial every eight hours and ondansetron at 4mg every eight hours for two days in order to prevent the uncontrollable vomiting and worsening of the dehydrated state. During his stay in the hospital, the patient's hydroelectric imbalance was corrected, along with creatinine levels that reached 1.1mg/dl upon discharge.

Here we have discussed the case of prerenal acute renal failure secondary to dehydration, a very common pathology in our daily practice, but that was caused by CVS, a very uncommon and rarely seen phenomenon amongst adult nephrologists. This review, illustrated by our case report, serves to show how to effectively approach the treatment of a patient with this syndrome. We must highlight that the treatment of these patients does not only consist of rehydration, but also abortive therapy for vomiting crises with sedation in order to avoid the perpetuation of acute renal failure.[5,6]

Conflicts of interest

The authors have no conflicts of interest to declare.

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Distal renal tubular acidosis in a seven-week pregnant woman: Diagnosis, complications and treatments

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To the Editor,

Distal renal tubular acidosis (RTA) is a relatively uncommon tubulopathy that is characterised by hyperchloremic metabolic acidosis, hypokalaemia, elevated urine pH (>5.5), and a negative anion gap. Early diagnosis can facilitate providing adequate treatment, which avoids potentially severe complications. Here we present the case report of a gestating mother (7 weeks) diagnosed with RTA.

We treated a 28-year old pregnant woman (7 weeks gestation) that sought emergency treatment for intense weakness with vomiting and abdominal pain. She had a history of rhabdomyolysis secondary to severe hypokalaemia of an unknown cause, bilateral nephrocalcinosis, and nephrolithiasis (Figure 1). We reviewed the patient's previous laboratory results and observed that she had hyperchloremic metabolic acidosis and hypokalaemia with persistently alkaline urine pH with several years' evolution. Upon arrival in the emergency room, she had: AHT: 103/71mm Hg, HR: 78 systoles, deep abdominal palpation produced pain in the left hypochondria and fossa, with positive left renal percussion.

Blood analysis highlighted a pH of 7.18, bicarbonate at 12.4mmol/L with normal plasma anion gap, PCO_2 at 35mm Hg, K^+ at 3.3meqL, chlorine at 121 meq/l, creatinine at 0.62mg/dl, calcium at 8.3mg/dl, albumin at 3.3g/dl, and phosphorous at 3.6mg/dl. The urine sample resulted in: urine pH: 8; negative anion gap [Cl (66.2mEq/l) < Na^+ (86mEq/l) + K^+ (14.17mEq/l)]; diuresis: 3200ml/24h; calciuria: 137.7mg/24h; hypocitraturia (citraturia <102mg/24h), and normal oxaluria. The immunological analysis did not reveal any significant abnormalities.

A renal ultrasound revealed grade II-III/IV pelvicalyceal dilatation of the left kidney and fluid collection in the left perirenal space (Figure 2). The patient was gestating with a live foetus. The urology department was advised of the situation, and they placed a left double J ureteral catheter, making passage through a left ureterolithiasis, resulting in the flow of urine with a purulent aspect. We later started treatment with antibiotics and intravenous potassium and bicarbonate, achieving clinical improvement. Based on the clinical symptoms, the previous laboratory analyses, and the current values, the patient was diagnosed with RTA.

RTA is a renal tubulopathy with hereditary aetiology that is idiopathic or secondary to any one of a variety of causes (Table 1). It is diagnosed based on the presence of electrolytic disorders that appear in blood and urine samples through venous gas measurements.