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.

1. Rasquin-Weber A, Hyman PE, Cucchiara S, Fleisher DR, Hyams JS, Milla PJ, et al. Childhoood functional gastrointestinal disorders. Gut 1999;45(Suppl II):1160-68.
2. Duckett A, Pride PJ. Cyclic vomiting syndrome in an adult patient. Hosp Med 2010;5(4):251-2.
3. Kenny P. Síndrome de vómitos cíclicos: un enigma pediátrico vigente. Arch Argent Pediatr 2000;98(1):34.
4. Barrio A. Síndrome de vómitos cíclicos. An Esp Pediatr 2002;56:151-64.
5. Yang HR. Recent concepts on cyclic vomiting syndrome in children. Neurogastroenterol Motil 2010;16(2):139-47.
6. Erturk O, Uluduz D, Karaali-Savrun F. Efficacy of nebivolol and amitriptyline in the prophylaxis of cyclic vomiting syndrome: a case report. Neurologist 2010;16(5):313-4.

M.J. Izquierdo Ortiz, V. Mercado Valdivia, P. Abaigar Luquin
Nephrology Department. Healthcare University Complex of Burgos, Spain
Correspondence: M.J. Izquierdo Ortiz
Sección de Nefrología.
Complejo Asistencial Universitario de Burgos, Avda. del Cid. 09006 Burgos. Spain.
maridetrespa@hotmail.com
veronicamercado2@hotmail.com.

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

Nefrologia 2011;31(6)761-3
doi:10.3265/Nefrologia.pre2011.Oct.11123

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.

Table 1. Most common aetiologies in renal tubular acidosis

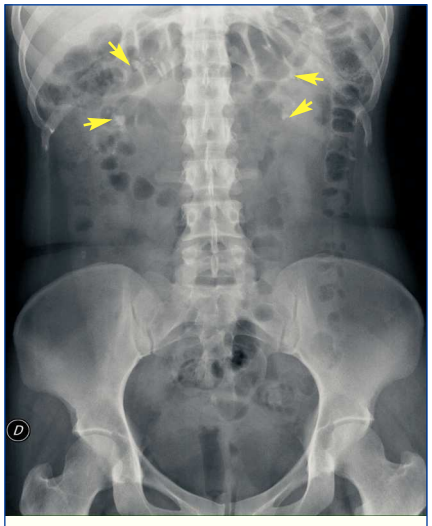
Primary aetiology
1. Idiopathic, sporadic
2. Hereditary
- Autosomal recessive (some forms are associated with nerve deafness, others with osteopetrosis)
- Autosomal dominant
Secondary aetiologies
1. Autoimmune disease
- Sjögren's syndrome
- Systemic lupus erythematosus
- Primary biliary cirrhosis
- Thyroiditis
2. Drugs
- Aspiration of toluene
- Lithium
- Amphotericin B
- Ibuprofen
- Ifosfamide
3. Other aetiologies
- Hypergammaglobulinaemias
- Sickle-cell anaemia
- Obstructive uropathy and chronic pyelonephritis.

RTA in children tends to be primary or idiopathic. A genetic analysis allows for distinguishing the responsible mutation (over 50 have been described), the most relevant of which are alterations to the genes ATP6V0A4 and ATP6V1B1, this last one related to deafness.[1] Early diagnosis can allow for correction of the metabolic acidosis, thus avoiding stunted development and rickets.[2]

Adult patients require a differential diagnosis in order to rule out associated autoimmune pathologies.[3] We asked the patient to undergo a complete examination on several occasions without being able to establish an autoimmune aetiology. Hypercalciuria, hypocitraturia, and abnormally alkaline urine (and less frequently, hyperuricosuria) are situations that predispose the patient to the development of nephrolithiasis and nephrocalcinosis,[4] making the patient prone to recurrent urinary infections. Renal function is conserved initially, but without appropriate treatment, patients develop chronic renal failure, AHT, and other complications. There is also a risk of acute renal deterioration with various aetiologies due to urinary infections or acute pyelonephritis, obstructive phenomena, or even rhabdomyolysis secondary to severe hypokalaemia.

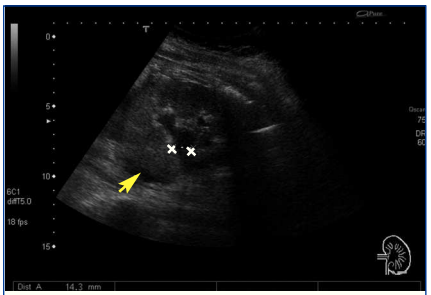
Treatment in the initial phase consists of alkalising agents such as bicarbonate or citrate and correction of the hypokalaemia. In cases of RTA related to secondary aetiologies, controlling the responsible pathology aids in correcting the hydroelectrolytic disorders of RTA.[5]

We conclude that RTA is an uncommon tubulopathy whose early diagnosis avoids severe renal repercussions, although the evolution and prognosis of this condition are both favourable with proper treatment.



Bilateral renal lithiasis (arrows)

Figure 1. Simple abdominal x-ray.



Grade II-III/IV pyelocaliceal dilation of left kidney and perirenal fluid

Figure 2. Left renal ultrasound.

Conflicts of interest

The authors have no conflicts of interest to declare.

1. Karet FE, Finberg KE, Nelson RD et al. Mutations in the gene encoding B1 subunit of H+-ATPase cause renal tubular acidosis with sensorineural deafness. Nat Genet 1999; 21(1):84-90.
2. Morris RC, Sebastian A. Alkali therapy in renal tubular acidosis: who needs J Am Soc Nephrol 2002;13: 2186.
3. Rodríguez Soriano J. Renal tubular acidosis: the clinical entity. J Am Soc Nephrol 2002;13:2160.
4. Buckalew VM Jr. Nephrolithiasis in renal tubular acidosis. J Urol 1989;141:731.
5. Emmett M. Treatment of distal (type 1) and proximal (type 2) renal tubulur acidosis. 2011 Up ToDate. www.uptodate.com.

O. Fikri Benbrahim[1], F. Cazalla Cadenas[1], A. Valentín Martín[2], E. D. Valladares Molleda[2], R. García Agudo[1], J. Mancha Ramos[1]

[1] Servicio de Nefrología. Hospital La Mancha Centro. Alcázar de San Juan. Ciudad Real.

[2] Servicio de Radiología. Hospital La Mancha Centro. Alcázar de San Juan. Ciudad Real.

Correspondence: Oussamah Fikri Benbrahim
Servicio de Nefrología.
Hospital La Mancha Centro.
Avda. la Constitución, s/n.
13600 Alcázar de San Juan. Ciudad Real. Spain.
fikrioussamah@yahoo.fr
lourdeshreynals@yahoo.com.ar.

Systemic lupus erythematosus and hypothyroidism

Nefrologia 2011;31(6):763-4
doi:10.3265/Nefrologia.pre2011.Jun.11007

To the Editor,

The combination of systemic lupus erythematosus (SLE) and altered thyroid function has been described in several different studies. The most common alteration has been described as primary hypothyroidism. However, the presence of central hypothyroidism in patients with SLE is very uncommon. Here we present the case of a patient with SLE that, in the course of an outbreak of lupus nephritis, developed severe suprathyroid hypothyroidism

Our patient was a 33-year old male diagnosed with SLE in 2000 based on an analysis of polyarthralgia and cutaneous lesions. In 2001, he developed pure nephrotic syndrome. A renal biopsy indicated membranous glomerulonephritis (stage V) that went into complete remission following immunosuppressant treatment. In 2005, he had another bout of nephritis in the form of impure nephrotic syndrome; we performed another biopsy and detected membranous glomerulonephritis (GN), accompanied by necrosis and proliferation in half of the glomeruli and centres of fibrosis, as well as interstitial atrophy (stage 4-5). He responded partially to several immunosuppressants, and was stabilised at a plasma creatinine level of 1.5-1.8mg% and proteinuria in the non-nephrotic range. He then was treated with losartan, prednisone (5mg/day), and simvastatin.

In March 2011, the patient sought treatment for the appearance of tibiomalleolar oedema. The physical examination revealed blood pressure of 210/120mm Hg, pallor of the mucosa, and pitting oedema in both legs. A laboratory analysis revealed: haemoglobin (Hb): 10g/dl; creatinine: 4.3mg/dl (glomerular filtration rate [GFR]: 15ml/min/1.73m^2); albumin: 25g/l; antinuclear antibodies (ANA): 23 (positive >1); anti-DNA: 405U/ml (positive >15); C3: 28mg/dl (76-181); C4: 4.4mg/dl (12-49); proteinuria/24 hours: 11g, and sediments with haematuria. We started the patient on prednisone and mycophenolate, with anti-hypertensives to control the arterial hypertension. We observed the progressive disappearance of the oedema, as well as improved renal function (creatinine: 3mg/dl and reduction of proteinuria to 3g/24h). Seven days after starting treatment, the patient complained of severe asthenia that impeded mobility, constipation, and a constant feeling of cold. The thyroid analysis revealed: thyrotropin (TSH): 0.09μU/ml (0.34-4.9); free T4 thyroxin: 0.60mg/dl (0.69-1.48), free T3 triiodothyronine: 1.4pg/ml (1.71-3.71); reverse triiodothyronine: 0.19ng/ml (0.10-0.34), and thyroid peroxidase antibodies (TPO): 6.92U/ml (0-5.6). The measurements of gonadotropins (FSH, LH), prolactin, human growth hormone, testosterone, and somatomedin C (IGF-1) were all normal. A thyroid ultrasound and magnetic resonance of the hypophysis resulted normal. We started treatment with levothyroxine, observing a progressive disappearance of the hypothyroid symptoms, and normalised plasma levels of free thyroxin.

Several different studies have shown that altered thyroid function is more common in SLE patients than in the general population.[1-4] Primary hypothyroidism, both clinical and subclinical, is the most commonly observed alteration. Two recent studies compared patients with SLE with a control group and observed a prevalence of primary clinical hypothyroidism of 6% and 14%, and subclinical hypothyroidism of 12% and 17%, respectively.[5,6] The prevalence of clinical hypothyroidism in the general Western population is less than 1%. Based on the presence of antithyroid antibodies in patients with SLE and hypothyroidism, half of all cases have an autoimmune origin, and the percentage of positivity for antithyroid antibodies in patients with SLE and euthyroidism ranges between 6% and 47%.

On the other hand, the majority of these studies suggest that there is no difference in the prevalence of hyperthyroidism between patients with SLE and the general population. We must point out that the association between SLE and central hypothyroidism is rare. The cases described have been of patients with SLE that develop lymphocytic neurohypophysis, which also produces altered secretion of other hormones in addition to the thyroid hormones.[7]

Taking into account this prevalent association and the fact that clinical and laboratory manifestations of hypothyroidism can simulate a lupus outbreak,[8] we suggest performing an analysis of thyroid function in patients with SLE.

Conflicts of interest

The authors have no conflicts of interest to declare.

1. Goh KL, Wang F. Thyroid disorders in systemic lupus erythematosus. Ann Rheum Dis 1986;45:579-83.
2. Tsai RT, Chang TC, Wang CR, Chuang CY,