although the timing of the clinical presentation makes this less likely.

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Juan Antonio Martín-Navarro[a,*], Vladimir Petkov-Stoyanov[a], María José Gutiérrez-Sánchez[a], Luis Pedraza-Cezón[b]

[a] *Servicio de Nefrología, Hospital del Tajo, Aranjuez, Madrid, Spain*
[b] *Servicio de Farmacia, Hospital del Tajo, Aranjuez, Madrid, Spain*

[*] *Corresponding author.*
E-mail address: juanmartinnav@hotmail.com (J.A. Martín-Navarro).

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Case reports and misdiagnosis of renal tubular acidosis☆

Informe de casos de acidosis tubular renal y errores de diagnóstico

Dear Editor,

Renal tubular acidosis (RTA) is a pathophysiological alteration of acid–base metabolism, characterised by the presence of hyperchloraemic metabolic acidosis, which is caused by renal loss of bicarbonate or a reduction in hydrogen ion excretion by the renal tubules.[1] A suspicion of RTA is based on the clinical presentation of various signs and symptoms such as anorexia, vomiting, polyuria, polydipsia, delayed growth, muscle weakness, rickets, nephrocalcinosis and sensorineural deafness.[2] The diagnosis is validated with laboratory examinations that should include the demonstration of hyperchloraemic metabolic acidosis, with a normal blood anion gap and a blood pH lower than 7.35, in patients with decompensated metabolic acidosis. In the case of secondary RTA, it is important to diagnose the systemic disease that is causing it.[1] It should be noted that intestinal losses of bicarbonate, whether they are due to diarrhoea or a fistula, are a common cause of the same acid–base alterations, and so they should not be present when a diagnosis of RTA is made.

RTA has been reported to be overdiagnosed in Mexico,[3,4] and it has been associated with allergy.[4–6] For this reason, we conducted a study with the aim of documenting the diagnosis of RTA in children from different hospitals. A total of 170 children with a prior diagnosis of RTA were enrolled; the majority were receiving alkaline treatment. Treatment was suspended 5 to 7 days prior to the initial assessment, which consisted of a medical history, laboratory examinations and an assessment by the allergy department. A diagnosis of RTA was only confirmed in 3 patients (1.8%), one with distal RTA and 2 with RTA secondary to cystinosis, which were accompanied by Fanconi syndrome. None of them had an allergy. The rest of the patients' prior diagnosis of RTA was erroneous; failure to thrive was caused by other conditions such as nutritional deficiency, Turner syndrome, giardiasis, coeliac disease, familial short stature, hypophosphataemic rickets, coenzyme Q10 deficiency or cardiomyopathy. The cases with RTA are described below.

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Case 1: A 12-year-old eutrophic female patient with normal anthropometric measurements for her age and gender. She was diagnosed with RTA at one month of age, and since then she has been receiving a potassium citrate solution. On the third day of suspending alkaline treatment she had vomiting, metabolic acidosis, blood pH 7.22; [HCO_3^-] 10 mmol/l; blood anion gap 11; K^+ 2.2 mEq/l; Cl^- 114 mEq/l; urine calcium/creatinine 1.38; urinary pH: 7.5. Alkaline treatment — bicarbonate and potassium — was restarted. Kidney ultrasound: grade III medullary nephrocalcinosis. Molecular study: mutation of the *ATPV60A4* gene, not previously reported in the literature, which shall be the subject of a subsequent publication.

Case 2: A 22-month-old male patient with delayed growth since sixth months of age. At 11 months, he was diagnosed with distal ATR, with vomiting, polydipsia and polyuria, and he received treatment with a potassium citrate solution. Entry into the study: weight 7.88 kg ($p < 3$); height 71 cm ($p < 3$); weight/age 71%; weight/height 86.3%; height/age 91.8%; BMI Z-score: −1.47. Blood gases pH 7.39; [HCO_3^-] 16.2 mmol/l; K^+ 3.0 mEq/l; HPO_4^- 2.4 mg/dl; urine pH: 7.5; trace albumin, +glucosuria; microscopic haematuria, 45% tubular reabsorption of phosphate. Administration of furosemide: did not have urinary acidification. Ophthalmology: birefringent crystals in the cornea with a slit lamp. A diagnosis of infantile nephropathic cystinosis was considered. Molecular study of the *CTNS* gene (17p13, NG_012489.1 RefSeqGene): compound heterozygous genotype, predictor of severe form of infantile nephropathic cystinosis with deletion of 57 kb, deletion of the first 10 exons, mutation more common in Caucasian, Mexican and Latin American patients[7,8] with cystinosis and a minor deletion previously reported in European populations.[9] Treatment with cysteamine bitartrate, phosphates, bicarbonate and potassium, with satisfactory evolution.

Case 3: A 20-month-old female patient with polyuria, polydipsia, anorexia and delayed growth. RTA was suspected, and she was referred to our institution without being treated. Weight 6.9 kg ($p < 3$); height 75.5 cm ($p < 3$); weight/age 60.9%; weight/height 73%; height/age 91.3%. Blood gases: pH 7.47; [HCO_3^-] 13.8 mmol/l; K^+ 3.6 mEq/l; HPO_4^- 2.2 mg/dl; Cl^- 114 mEq/l; urine pH 7.0; glucosuria 100 mg/dl; +albumin. Ophthalmology: birefringent corneal cystine crystals. Molecular study of the *CTNS* gene: homozygous genotype, microdeletion of exon 12, which deleted amino acids 346–349 of the 7th transmembrane domain of cystinosin, which confirmed the diagnosis of infantile nephropathic cystinosis, reported in Europeans[10]. Treatment with cysteamine bitartrate, phosphates, bicarbonate and potassium.

We concluded that RTA is an uncommon tubulopathy, not associated with allergy, and confirmed that it is overdiagnosed in Mexico. We recommend a comprehensive paediatric approach in children with delayed growth, considering other diseases in addition to RTA, with special caution in the studies that are requested and their quality. When RTA is diagnosed, the presence of primary diseases with secondary RTA should be ruled out, and suitable guidance and nutritional support should be provided.

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Conflict of interest

The authors have no conflict of interest to declare.

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Mara Medeiros[a,b,*], Sandra Enciso[a], Ana María Hernández[a], Hector Rodrigo García Hernández[a], Georgina Toussaint[a], Claudia Pinto[c], Elsy Maureen Navarrete Rodríguez[d], Blanca E. del-Rio-Navarro[d], Omar Josué Saucedo-Ramírez[d], Patricia Medina Bravo[e], Sergio Miranda[f], Liliana Worona[f], Germán Sosa[c], Leticia Belmont Martinez[g], Miguel Ángel Alcántara Ortigoza[h], Laura Escobar[i], Ricardo Muñoz Arizpe[a]

[a] *Laboratorio de Investigación en Nefrología y Metabolismo Mineral Óseo, Hospital Infantil de México Federico Gómez, México, D.F., Mexico*
[b] *Departamento de Farmacología, Facultad de Medicina, Universidad Nacional Autónoma de México, México, D.F., Mexico*
[c] *Departamento de Nefrología «Dr. Gustavo Gordillo Paniagua», Hospital. Infantil de México Federico Gómez, México, D.F., Mexico*
[d] *Servicio de Alergia e Inmunología Clínica Pediátrica, Hospital Infantil de México Federico Gómez, México, D.F., Mexico*
[e] *Departamento de Endocrinología, Hospital Infantil de México Federico Gómez, México, D.F., Mexico*
[f] *Departamento de Gastroenterología, Hospital Infantil de México Federico Gómez, México, D.F., Mexico*
[g] *Laboratorio de Errores Innatos del Metabolismo y Tamiz, Instituto Nacional de Pediatría, México, D.F., Mexico*
[h] *Departamento de Biología Molecular, Instituto Nacional de Pediatría, México, D.F., Mexico*
[i] *Departamento de Fisiología, Facultad de Medicina, Universidad Nacional Autónoma de México, México, D.F., Mexico*

* *Corresponding author.*
E-mail addresses: medeiro.mara@gmail.com, maramedeiros@hotmail.com (M. Medeiros).

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Creation of the Working Group on Diagnostic and Interventional Nephrology of the Spanish Society of Nephrology☆

Creación del Grupo de Trabajo en Nefrología Diagnóstica e Intervencionista de la Sociedad Española de Nefrología

Dear Editor,

At its meeting of 9 July 2014, the Board of Directors of the Spanish Society of Nephrology (SEN) approved the creation of the SEN Working Group on Diagnostic and Interventional Nephrology (DIN). This letter, based on the group's founding document, which is available on the SEN website, aims to set forth the reasons why it is necessary to create this group, its objectives and its plan for action.

Ultrasound is an essential tool in the practice of medicine, with multiple applications in kidney patients. In addition to being a very informative and non-invasive diagnostic method, it is the vehicle through which interventions such as biopsy on the kidneys may be performed.[1–5] Furthermore, it is crucial for performing various interventions that are not strictly related to the kidneys, but are the responsibility of the nephrologist, such as placing central lines and managing arteriovenous fistulas for haemodialysis.[6] Finally, it allows the arteries to be visualised for early diagnosis of subclinical artery disease, or evolution of atheromatous disease, which is known to be the basis of the majority of cardiovascular events and mortality in the general population and to an even greater extent in the kidney population.[7]

For this reason, it is important that the nephrologist learn appropriate ultrasound techniques so as to perform and interpret ultrasound examinations in order to achieve more comprehensive and efficient patient management.

Suitable and timely placement of the appropriate catheter for peritoneal dialysis determines the prognosis for the patient and the technique. The nephrologist is trained to place and remove peritoneal catheters, and this is in fact what happens in some Spanish departments of Nephrology.

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