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.

1. Mehanna HM, Moledina J, Travis J. Refeeding syndrome: what it is, and how to prevent and treat it. BMJ 2008;336:1495-8.
2. Khan LUR, Ahmed J, Khan S, MacFie J. Refeeding Syndrome: A Literature Review. Gastroenterology Research and Practice. Volume 2011.
3. Stanga Z, Brunner A, Leuenberger M, Grimble RF, Shenkin A, Allison SP, et al. Nutrition in clinical practice. The refeeding syndrome: illustrative cases and guidelines for prevention and treatment. Eur J Clin Nutr 2008;62:687-94.
4. National Institute for Health and Clinical Excellence. Nutrition support in adults. Clinical guideline 32. 2006. Available at: http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

Javier D. Macías-Toro, Anna Saurina-Solé, Mònica Pou-Potau, Vicent Esteve-Simó, Verónica Duarte-Gallego, Miguel Fulquet-Nicolás, Fátima Moreno-Guzmán, Manel Ramírez-de Arellano Serna
Servicio de Nefrología. Consorci Sanitari de Terrassa. Terrassa, Barcelona. (Spain).

Correspondence: Javier D. Macías Toro
Servicio de Nefrología.
Consorci Sanitari de Terrassa,
Carretera Torrebonica, s/n.
08277 Terrassa, Barcelona. (Spain).
deivith1978@gmail.com

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

Nefrologia 2013;33(2):281-3
doi:10.3265/Nefrologia.pre2012.Oct.11769

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

primary angioplasty was to be carried out, which was unsuccessful due to the persistence of stenosis. Because of the difficult monitoring of blood pressure figures, selective venous sampling of the renal veins was performed to measure renin, with a difference of 10:1 being found in the concentrations of autotransplanted kidney against the right kidney. This confirmed the suspicion of renovascular HBP originating in the autotransplanted kidney. It was not possible to perform autotransplanted renal artery embolisation due to the risk of extensive necrosis, as parasitic branches were found that provided flow to the autotransplanted kidney and the intrinsic muscles of the pelvis. It was decided to carry out nephrectomy of the left autotransplant. Evolution was satisfactory with better control of blood pressure figures, and as such, the patient was discharged.

DISCUSSION

Renovascular hypertension is defined as the presence of HBP secondary to an obstructive lesion of the renal arteries[3]; it is the third leading cause of HTA[4] amongst children and major diseases that are associated with it are FD, TA and type 1 neurofibromatosis. MAS is a rare cause, but must be considered as a differential diagnosis.[5]

MAS is characterised by stenosis of the proximal abdominal aorta and ostial stenosis of its major branches (renal arteries 90%)[2]; the mean age of onset is 20 years,[4] although recent papers report earlier diagnosis.[6,7] Its aetiology is diverse; most cases are idiopathic,[4] although it has been associated with diseases such as neurocutaneous syndrome and Williams syndrome.[6] Among the hypotheses proposed is inadequate fusion of the two dorsal aortas during embryonic development and it has also been linked to congenital rubella syndrome.[8] The most important clinical marker is severe HBP caused by ostial stenosis of the renal artery, which leads to increased production of renin,[4] as happened in this patient. Commonly observed histological findings are intimal fibrodysplasia, distortion of internal elastic lamina with absence of inflammatory changes, with lesions similar to those observed in some forms of FD.[9]

For its diagnosis, arteriography remains the gold standard; it is also useful to define the extention of disease and conduct surgical therapy.[10] Additional tests are measuring renin levels and renal function tests. In differential diagnosis, it is important to note that the acute phase reactants are normal, unlike with TA, and there are no systemic symptoms such as fever, weight loss, abdominal pain or skin lesions.

Treatments vary depending on the severity of each case, and range from conservative to surgical management, such as autotransplant[6] and nephrectomy when revascularization is not possible.[4,7] Percutaneous transluminal balloon angioplasty and stenting are used in older children,[10] however, successful results have not been displayed, as is the case with this patient.[10] Medical therapy is important for aggressive control of blood pressure, which is often difficult to manage.[8]

To conclude, this article reports the case of a girl diagnosed with middle aortic syndrome caused by renovascular hypertension, with a clinical presentation that proved to be a diagnostic challenge, as it was difficult to distinguish from other more common causes, such as TA and FD. Associated HBP was difficult to treat and required multiple therapeutic interventions such as autotransplant and balloon angioplasty, which were unsuccessful and required nephrectomy for adequate control of blood pressure.

Conflicts of interest

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

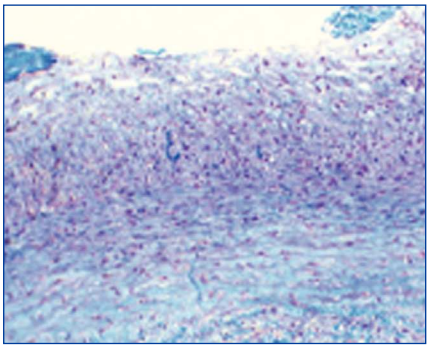

Figure 1. Renal artery biopsy. Trichrome stain: a rough organisation of the outer (bottom), middle (centre) and inner (top of image) layers is observed without evidence of a true muscle layer or internal or external elastic lamina. Findings consistent with renal artery dysplasia.

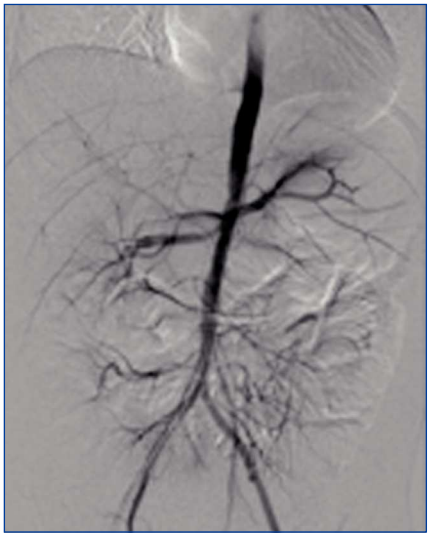

Figure 2. Abdominal arteriography. Abdominal arteriogram showing irregularity of the middle-aortic segment and progressive distal thinning of the aorta in addition to ostial stenosis of the right renal artery with poststenotic truncal and/or dysplastic dilation and an absence of the left renal artery due to previous autotransplantation.

1. Denis G, Schaefer F. Comprehensive Pediatric Nephrology. 1 ed. Philadelphia: Mosby Elsevier; 2008.
2. Van den Driessche A, Van Hul E, Ichiche M, Verpooten G, Bosmans JL. Fibromuscular dysplasia presenting as a renal infarction: a case report. J Med Case Rep 2010;4:199.
3. Mehta AN, Fenves A. Current opinions in renovascular hypertension. Proc (Bayl Univ Med Cent) 2010;23(3):246-9.
4. Lin Y-J, Hwang B, Lee P-C, Yang L-Y, Meng CCL. Mid-aortic syndrome: a case report and review of the literature. Int J Cardiol 2008;123(3):348-52.
5. Brunner J, Feldman BM, Tyrrell PN, Kuemmerle-Deschner JB, Zimmerhackl LB, Gassner I, et al. Takayasu arteritis in children and adolescents. Rheumatology (Oxford) 2010;49:1806-14.
6. Tummolo A, Marks SD, Stadermann M, Roebuck DJ, McLaren C, Hamilton G, et al. Mid aortic syndrome: long-term outcome of 36 children. Pediatr nephrol 2009;24(11):2225-32.
7. Ozen S, Pistorio A, Iusan SM, Bakkaloglu A, Herlin T, Brik R, et al. EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. Ann Rheum Dis 2010;69(5):798-806.
8. Sethna CB, Kaplan BS, Cahill AM, Velazquez OC, Meyers KEC. Idiopathic mid-aortic syndrome in children. Pediatr Nephrol 2008;23(7):1135-42.
9. Bonvini RF, Rastan A, Sixt S, Righini M, Hofstetter R, Zeller T. Diffuse fibromuscular dysplasia successfully treated with scoring balloon angioplasty in a 3-year-old boy. Heart Vessels 2009;24(6):460-2.
10. Brountzos EN, Ptohis N, Triantafyllidi H, Panagiotou I, Spyridopoulos TN, Misiakos EP, et al. Renal artery rupture following cutting balloon angioplasty for fibromuscular dysplasia: a case report. Cases J 2009;2:8881.

Catalina Vélez-Echeverri[1,7], Margarita Suárez[2], Lina Serna-Higuita[1,7], Ana K. Serrano-Gayubo[3], Juan J. Vanegas-Ruiz[1,7], José M. Hidalgo[4], Luis F. Arias[5], Ruth M. Eraso[6,7]

[1] Unidad de Nefrología. Hospital Pablo Tobón Uribe. Medellín, Antioquia (Colombia).
[2] Departamento de Pediatría. Hospital Pablo Tobón Uribe. Medellín, Antioquia (Colombia).
[3] Unidad de Nefrología.
Facultad de Medicina, Universidad de Antioquia y Hospital Universitario San Vicente de Paúl. Medellín, Antioquia (Colombia).
[4] Unidad de Radiología. Hospital Pablo Tobón Uribe. Medellín, Antioquia (Colombia).
[5] Departamento de Patología. Facultad de Medicina, Universidad de Antioquia y Hospital Universitario San Vicente de Paúl. Medellín, Antioquia (Colombia).
[6] Sección de Reumatología. Hospital Pablo Tobón Uribe. Medellín, Antioquia (Colombia).
[7] Departamento de Pediatría. Facultad de Medicina, Universidad de Antioquia, Medellín, Colombia.

Correspondence: Lina Serna Higuita
Unidad de Nefrología.
Hospital Pablo Tobón Uribe.
Transversal 39 a número 71-57, Medellín, Antioquia (Colombia).
lm.serna@hotmail.com

Treatment for renovascular hypertension due to fibromuscular dysplasia of renal arteries with renal autotransplantation

Nefrologia 2013;33(2):283-4
doi:10.3265/Nefrologia.pre2012.Oct.11766

To the Editor:

Renovascular hypertension (RVH) is the most common form of secondary hypertension, with renal arteriography being the gold standard study for confirming diagnosis.[1] It presents clinically with arterial hypertension (AHT) that does not respond to treatment (three antihypertensive drugs including a diuretic) and progressive deterioration of renal function due to ischaemic renal atrophy.[2] Angioplasty is the technique of choice in cases of renal artery fibromuscular dysplasia; but there is the possibility of performing nephrectomy, back table procedure and autotransplantation in cases that cannot be treated with angioplasty.[3]

We report the case of a 59-year-old female, without harmful habits, a history of dyslipidaemia, obstructive sleep apnoea syndrome and chronic iron deficiency anaemia, followed up in the Nephrology outpatient service for about 20 years due to AHT unresponsive to treatment (5 drugs).

Physical examination was normal except for sustained AHT (170/90mmHg under antihypertensive medical treatment), including funduscopic examination, where no retinopathy was observed. Ambulatory monitoring of arterial pressure was carried out on various occasions and confirmed resistant AHT. One of these measurements showed the following readings:

Mean systolic blood pressure (SBP): 143mmHg (110-168), mean diastolic blood pressure (DBP): 80mmHg (62-90), mean heart rate: 61 (64-67). SBP load: 70.7%, DBP load: 25.9%. NON-DIPPER night-time pattern (lower night-time SBP: -1.15%, lower night-time DBP: 0%).

The blood test showed haemoglobin: 11.7g/dl, haematocrit 37.1%, creatinine: 1.22mg/dl, urea: 40mg/dl, glucose: 88mg/dl, total cholesterol: 249mg/dl and normal urinary sediment without microalbuminuria. Thyroid hormones, catecholamines and metanephrines were normal.

The electrocardiogram showed signs of left ventricular hypertrophy which was not confirmed by Doppler echocardiography.

Imaging studies showed normal chest X-ray and renal ultrasound with right kidney with no structural abnormalities, except for the presence of two simple cysts in the upper pole and it was not possible to visualize the left kidney. Subsequently, renal artery CT