Letter to the Editor

Renal involvement in Sneddon's syndrome☆

Afectación renal en el síndrome de Sneddon

ARTICLE INFO

Article history:

Dear Editor,

Sneddon's syndrome (SS) is a rare and slowly progressive vascular disease that affects small and medium-sized arteries, characterised by the association of *livedo reticularis* and recurrent cerebrovascular accidents (CVA).[1]

Approximately 80% of SS patients are women with a median age of 40 years.[1] The aetiopathogenesis is unknown, with two proposed primary mechanisms (autoimmune/inflammatory versus thrombophilia), and it is classified mainly as positive or negative antiphospholipid.[1] The main symptoms are cutaneous, which usually precede neurological symptoms by more than 10 years. The vast majority of patients present with labile systolic hypertension and it can also affect the heart valves or the kidneys, inter alia.[1,2] After reviewing the literature, histopathological renal involvement in this syndrome is extremely rare.[2,3] Below we describe a case of SS with biopsy-proven chronic kidney disease associated with features of membranous nephropathy:

A 53-year-old woman diagnosed with SS, arterial hypertension, arterial toe ischaemia and an active smoker, receiving treatment with acetylsalicylic acid (ASA), atorvastatin and valsartan. She is referred to nephrology due to the onset of albuminuria. Asymptomatic, with good blood pressure control. Normal physical examination except for *livedo reticularis* in the facial area, back of the hands, upper and lower limbs. An investigation was started, which revealed plasma levels of creatinine of 1.3 mg/dl, urea 48 mg/dl, sodium 141 mEq/l, potassium 3.8 mEq/l, total cholesterol 155 mg/dl, triglycerides 153 mg/dl, total protein 6.5 mg/dl, albumin 3.9 mg/dl, normal immunoglobulins (Igs), C3 77.8 mg/dl, C4 18 mg/dl, positive antinuclear antibodies 1/1280 (1/320 centrometric), anti-Jo-1 1/18 and anticardiolipin antibodies >160. The remaining autoimmunity, including anti-phospholipase A2 antibodies, protein electrophoresis and serologies were negative. Normal blood count and coagulation. Urinalysis revealed an albumin/creatinine ratio (ACR) of 1478.8 mg/g. Sonography revealed kidneys of decreased size and a diffuse increase in bilateral cortical echogenicity.

Due to renal involvement, a renal biopsy was performed that revealed chronic arteriolar/arterial vascular lesions with extensive interstitial and glomerular kidney damage associated with incipient membranous glomerulopathy. Glomerular damage was associated with ischaemia and deposition of immune complexes in capillary membranes without complement deposition. Immunofluorescence showed negativity for C1q, C3, C4, fibrinogen and albumin.

To complete the study of this systemic entity, a cranial CT scan was performed, which found vascular-degenerative leukoencephalopathy.

Regarding the treatment of SS, antiplatelets and anticoagulants are indicated as secondary CVA prophylaxis, in addition to improving the prognosis.[4] In patients with antiphospholipid syndrome, anticoagulants give rise to a better outcome than antiplatelets.[4] The use of anti-inflammatory or immunosuppressive therapies is controversial. In some cases, treatment has been started with intravenous immunoglobulins in patients with *livedoid* vasculopathy, with adequate outcomes.[5] In our case, anticoagulant and antiproteinuric treatment was administered, with stability of renal function and a decrease in the ACR to 175 mg/g.

In conclusion, it is a rare clinical syndrome that is probably underdiagnosed and in which kidney involvement is rare, so its detection adds complexity to the case.

The case described is interesting since, in addition to presenting with vascular lesions associated with SS in the kidneys, there was associated membranous nephropathy, which has not been previously reported in conjunction with SS in the literature, and may be related to the existence of an associated antiphospholipid syndrome.

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Elena Hernández García[a,*], María José Sánchez Torres[b]

[a] *Servicio de Nefrología, Hospital Universitario San Cecilio, Granada, Spain*
[b] *Servicio de Nefrología, Hospital Universitario Virgen de las Nieves, Granada, Spain*

[*] *Corresponding author.*
E-mail address: elena_46hg@hotmail.com (E. Hernández García).

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Letter to the Editor

Evolution of renal replacement therapy in Mexico in the last 10 years☆

Evolución del tratamiento sustitutivo de la función renal en México en los últimos 10 años

ARTICLE INFO

Article history:

Dear Editor,

In Mexico, 11% of the general population suffers from some degree of chronic kidney disease. The main causes are diabetes mellitus and arterial hypertension. There is no kidney health programme and 6% of patients are admitted for dialysis each year.[1] Over the last 10 years, dialysis treatments have increased at the expense of haemodialysis, which goes against current evidence.[2] The Instituto Mexicano del Seguro Social [Mexican Institute of Social Security] (IMSS) covers 65 million users in its different administrative regimes that offer its social security, and includes 50% of the patients that are on dialysis.[3]

A 10-year retrospective study (June 2008–June 2018) was conducted based on the institutional registry of adult and paediatric patients in chronic dialysis. It did not include continuous renal replacement therapy. It included 73,730 patients, of which 70,158 (95%) were being treated in secondary healthcare. A 57.5% were male, the average age was 63 years (range:

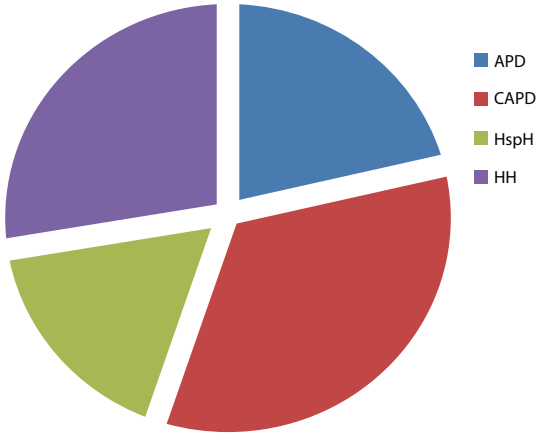

Fig. 1 – Distribution of treatment by type of dialysis.

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