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Case Report

Membranous glomerulonephritis: a case report

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ABSTRACT

A clinical case is presented with the aim of performing an analysis that allows to provide timely and comprehensive care to patients diagnosed with kidney diseases such as membranous glomerulonephritis, characterized by edema of the lower limbs, fatigue, and loss of protein in the urine. The case is a 55-year-old male patient who came to the NAPOLEÓN DÁVILA CÓRDOVA hospital because of anasarca and dyspnea with medium to minimal effort. Paraclinical tests are performed where leukocytosis with neutrophilia, slight elevation of creatinine and CRP, proteinuria, with hypoalbuminemia are evident; abdominal ultrasound reports significant ascites. In this context of a patient with generalized edema plus proteinuria and reduced albuminemia, nephrotic syndrome is suspected, for which reason measures are initiated to achieve a negative balance and with the diagnostic suspicion, a renal biopsy is performed in which a membranous lesion pattern + PLA 2R positive in basal membranes is reported. Analyzing the presentation of the patient's symptoms, it can be classified as idiopathic. The importance of diagnosing membranous glomerulonephritis is demonstrated.

Keywords: Kidney disease, Membranous glomerulonephritis, Arterial hypertension, Urine protein, Renal biopsy

INTRODUCTION

Membranous glomerulonephritis (MG) is one of the most common causes of nephrotic syndrome in adults and the elderly, while it is rare in children and adolescents. The incidence ranges from 5 to 10 cases per million inhabitants. GM can occur at any age, being more frequent in 80 to 90% of adult patients who are over 30 years of age at the time of diagnosis. In 80% of cases, MG begins with complete nephrotic syndrome (proteinuria >3.5g/24 h, hypoalbuminemia, hyperlipidemia).[2] There are two broad subtypes of MG that have been differentiated, such as primary or idiopathic membranous glomerulonephritis (PMG) and secondary membranous glomerulonephritis.

In the first case, an unknown responsible antigen was present and there was no disease or drug related to the onset of the condition; in the second case, the process is caused by systemic diseases, tumors, infections or drugs.[3] Patients with GM require prompt initial intervention, with checks every 1 to 2 months, to observe the evolution of proteinuria and renal function. Changes in these parameters will announce the spontaneous resolution of nephritic syndrome or an aggressive course, which are usually defined in the first months of diagnosis of the disease.[4] Due to the evolutionary differences of GM that have been reflected so far, it is important to propose a global treatment strategy that can be adapted to the particular characteristics of each case. Careful medical and family history, a complete physical examination and laboratory tests are essential for diagnosis. Due to the low incidence of this disease and the novelty of its diagnosis, it was decided to present this case.

CASE REPORT

A 55-year-old male patient with a history of poorly controlled arterial hypertension was admitted to a secondary hospital due to anasarca and dyspnea with

moderate to minimal exertion. Upon questioning, the patient reported productive cough, dyspnea and orthopnea. He noted the use of homeopathic medication, which he did not mention the dose or name of. Physical examination revealed: decreased vesicular murmur and significant generalized edema. Laboratory tests revealed the presence of leukocytosis with neutrophilia, altered renal function, elevated acute phase reactants, and urine test with proteinuria.

Imaging tests reported the presence of ascites on ultrasound and pleural effusion on chest CT. In this context of the patient with generalized edema plus proteinuria, nephrotic syndrome is suspected, and tests are performed to confirm this suspicion. Proteinuria in 24 hours, giving results of 5736.00 mg/24 hours and a urinary volume of 1720.00 ml/24 hours. Total protein 4.10, albumin 1.30, total bilirubin 0.16, sodium 141.90, potassium 3.46, chloride 103.40. Immunological tests, anti-neutrophils anca negative: c-anca, p-anca, a-anca Ab-antinuclear, ANA negative, anti-DSDNA negative. Complement c3 137.90 mg/dl, complement c4 33.20 mg/dl.

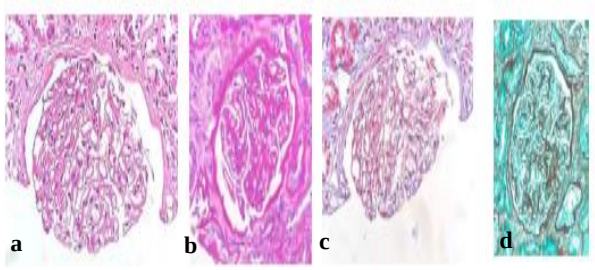

Figure 1 (a-d): Renal biopsy: histopathological result.

Renal parenchyma consisting of cortex, medulla and corticomedullary junction, which included 32 glomeruli, 4 of which showed global sclerosis, not segmental sclerosis; the rest showed global and diffuse thickening of the glomerular basement membranes, with filling defects and spicules evident with Jones methenamine silver stain; no endo- or extra capillary hypercellularity. The tubules showed atrophy affecting 15-20% of the tissue surface evaluated. The interstitium showed fibrosis foci in 15-20% of the cylinders studied. The vessels had the usual histology.

A histopathological study of the kidney was performed, where at a microscopic level renal parenchyma was observed, consisting of cortex, medulla and cortico-medullary junction, which had 32 glomeruli, 4 showed global sclerosis, not segmental sclerosis the rest showed global and diffuse thickening of the glomerular basement membranes, with filling defect and evident spicules with methenamine silver Jones stain no endo or extra capillary hypercellularity.

The tubules showed atrophy that compromised 15-20% of the tissue evaluated. The interstitium showed fibrosis foci in 15 to 20% of the cylinders studied. The vessels had usual histology. It had five glomeruli, which are observed in Table 1, and Figure 1 estimates the microscopic view of the biopsy taken. Determining a diagnosis of membranous lesion pattern global sclerosis in 10-12% tubular atrophy 15-20%, interstitial fibrosis 15-20 %.

Table 1: Direct immunofluorescence.

IGG	fine granular deposits +++/+++ in glomerular basement membrane
IGA	Negative
IGM	fine granular deposits +/+++ in glomerular basement membrane
C3	fine granular deposits ++/+++ in glomerular basement membrane
CLQ	fine granular deposits +/+++ in glomerular basement membrane
KAPPA	fine granular deposits ++/+++ in glomerular basement membrane
LAMBDA	fine granular deposits ++/+++ in glomerular basement membrane

Own elaboration.

DISCUSSION

The term glomerulonephritis or glomerulopathies is widely used to designate diseases that affect the glomerular structure and function, as they are heterogeneous clinical entities, both in their etiology, clinical manifestations, course and prognosis.[1] The histological finding of inflammation of the glomerular tuft (with four specific exceptions: diabetic nephropathy, hypertensive nephrosclerosis, amyloidosis and hereditary nephropathies) and which involve an immune mechanism in their pathophysiology. The rapid and effective recognition of the cause of the glomerular disease will result in a rational, safe and effective therapeutic approach, with special impact on the preservation of renal function. It should be mentioned that, worldwide, primary glomerular disease is the second cause of chronic terminal renal disease, preceded only by diabetic nephropathy, which is a secondary glomerulopathy.[1,2] Currently, two entities have been described as special forms of membranous glomerulonephritis.

Most cases of membranous glomerulonephritis present with complete nephrotic syndrome and are diagnosed relatively quickly, because the patient perceives the edema typical of this pathology.[4] Some cases present with non-nephrotic proteinuria and the diagnosis can be masked by the absence of symptoms. The presence of microhematuria is relatively frequent and macroscopic hematuria is very rare and requires ruling out the presence of renal vein thrombosis or urological tumors.[5]

The clinical manifestations and complications present as those of a nephrotic syndrome (edema, hyperlipidemia,

hypercoagulability). Venous thrombosis and, occasionally, pulmonary thromboembolism resulting from hypercoagulability are usually the first clinical manifestation and edema usually presents less abruptly than in clinical lesions. At diagnosis, most cases present normal renal function and blood pressure, and the appearance of arterial hypertension is usually related to the development of chronic renal failure. Patients with massive proteinuria and severe hypoalbuminemia may present a progressive deterioration of renal function in the first months of the clinical course.

Membranous nephropathy may develop gradually. Most people have edema early in the disease, but others may not develop any serious symptoms until kidney disease is advanced. Signs and symptoms of membranous glomerulonephritis are summarized in Table 2.

Health seeking behaviour of people is dependent on the perception of people regarding the quality of health care services in health centers. The perception of the people has to be changed to attract them more to government hospitals and health centers. It can be done through improving the quality of care, proper maintenance of facilities and also by inculcating a caring and sympathetic attitude in health professionals while dealing the patients.

Table 2: Main signs and symptoms of GM.

Sudden shortness of breath, which may be related to a complication of pulmonary thrombosis
Hematuria
Edema in the legs and ankles
Weight gain
Decreased protein in the blood, particularly albumin
High cholesterol level
Lack of appetite
Urine that appears foamy
Increased protein in the urine (proteinuria)
Decreased protein in the blood, particularly albumin
Hematuria
Fatigue
Increased blood pressure
Sudden pain between the upper abdomen and the middle back

The KDIGO guidelines recommend the initiation of immunosuppressive treatment only in those patients who maintain nephrotic proteinuria after an observation period of at least 6 months, and provided that the proteinuria does not have a clear tendency to decrease during this period. During this observation phase, a series of conservative measures should be established aimed at reducing the risks of nephrotic syndrome, reducing edema or facilitating the appearance of spontaneous remissions such as ACEI or ARB.[6]

During the observation period, patients should be excluded in whom a progressive deterioration of renal function is observed (increase in creatinine >30% of the baseline value during the first 6-12 months of evolution), those who present complications caused by nephrotic syndrome (for example, pulmonary thromboembolism) or cases with extreme hypoalbuminemia.

During the observation period, patients should follow a low-salt diet and receive diuretics (thiazides, furosemide, mineralocorticoid antagonists) in the doses and combinations required to reduce edema and allow a normal life. Treatment with ACEI or ARB, in addition to its indication in hypertensive patients, decreases the amount of proteinuria and facilitates the appearance of spontaneous remissions, as discussed above.

However, its use should be cautious, particularly in patients without hypertension or with an effective circulating volume compromised by severe hypoalbuminemia. Lipid-lowering therapy should be instituted in patients with dyslipidemia. A controversial issue is the indication of anticoagulant treatment. Although the evidence is based only on observational studies, the KDIGO guidelines recommend its initiation in patients with significant hypoalbuminemia, although an algorithm has been developed that generates a risk-benefit ratio for prophylactic anticoagulation in patients with GM.[7]

CONCLUSIONS

Membranous glomerulonephritis is a rare disease in the fifth decade of life. Identifying its cause is very important because when it occurs, it is necessary to establish its cause, which, if secondary, could be due to a neoplasia. Determination of PLA 2R in the renal biopsy sample clearly establishes the difference between primary or secondary MG.

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