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Case Report

Secondary Membranous Nephropathy: "Full-House" Pattern in a Syphilis and Parvovirus B19 Coinfection

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Abstract

Introduction: The Syphilis can share many similarities with multiple autoinmune diseases, mostly when it evolves to secondary syphilis with its visceral involvement (kidney, digestive system, central nervous system). The syphilitic nephropathy usually results in a classic membranous glomerulopathy.

Description: We present a clinical case which shows biological and histological features compatible to a lupus nephritis. A 53-year-old caucasian male presenting a patent nephrotic syndrome meanwhile he suffers from an inflammatory bowel disease and a sudden onset of a Meniere's-like Syndrome.

Diagnoses: The serology tests provide a positive result to Syphilis and Parvovirus B19 co-infection, being consistent with the histological injury and the motley semiology that presented the patient.

Results: The nephrotic syndrome was totally solved along with the digestive and otological symptomatology after the antibiotic administration as CNS dosing infection.

Conclusion: The Syphilis and Parvovirus B19 co-infection should be recognised as a lupus-like nephritis. Eventually, we would like to highlight the importance of a differential diagnosis of the multiple secondary causes of Membranous Nephropathy supported by a complete clinical history.

Introduction

The Membranous Nephropathy (MN) is the most frequent cause of Nephrotic Syndrome (NS) in adults. It is a disease caused by antibodies targeted to specific antigens, that they deposit in the subepithelial verge of the Glomerular Basement Membrane (GBM), forming Immune Complexes (IC) [Immnunoglobulin (Ig) and Complement (C)]. The podocyte foot process is injured by this ICs increasing its permeability, causing proteinuria and the potential NS [1]. Its principal etiology is primary or idiopathic, that is to say that the responsible antigen is unknown. The Phospholipase A2 Receptor (PLA2R) has been described as the antigen in the 70%-80% of idiopathic cases [2]. The secondary etiology is those triggered by autoimmune diseases, neoplasm, infections or drugs. A correct diagnosis of the several secondary causes is achieved with a complete clinical history, that it should comprehend new drugs or toxic abuse, serological tests and tumoral pathology screening [3]. Besides, it is required a renal biopsy study because some histological findings could guide us towards secondary causes [4].

Case Presentation

A 53-year-old caucasian male without behavior of toxic abuse. His medical background is asthma and hypotiroidism. In the previous months was diagnosed of an Inflamatory Bowel Disease (IBD). The patient was admitted with bad general condition, low digestive bleeding and onset of edema in lower limbs. Endoscopies had been performed, showing chronic pangastritis and mucosal ulcers in colon and rectum; without evidence of infectious etiology. At the same time, our patient complained about instability and hypoacusia of 15 days of evolution, being diagnosed of Meniere's-like Syndrome. Finally, the patient was referred to Nephrology because he had a pathological urinary sediment with proteinuria and generalized edema. In the clinical interview, the patient denied any general symptoms nor starting any new medication except of descendent corticotherapy. At the physical examination the only noticeable thing was pitting edema in the lower limbs. In the laboratory tests, it was demonstrated 13 grams of daily proteinuria (Protein/Creatinine Ratio 9.1), a pronounced hypoalbuminemia (1.4 g/dL) associated with hyperlipemia (LDL-cholesterol 240 mg/dL). In this regard, we extended the study of NS by performing imaging test; proteinogram, autoimmunity and serology test, and a kidney biopsy. The symptomatic treatment was initiated: a diuretic, the antiproteinuric double blockade, a statin and anticoagulation. Achieving a drop of weight of 7 Kg and the reduction of P/C Ratio from 9.1 to 0.8. The abdominal echography was unremarkable. The proteinogram didn't show any relevant disturbance. Concerning to the autoimmune test, the Antinuclear Antibodies (ANA), Antineutrophil Citoplasm Antibodies (ANCA) and Anti-PLA2R antibodies were negative. The serology test was negative to HBV, HCV and HIV. But a positive result was obtained for the syphilitic tests and for the presence of IgM and IgG antibodies of Treponema pallidum and Parvovirus B19. The kidney biopsy described in the Figure 1 give us the histopathological diagnosis of MN stage I with a "Full-House" pattern in the Immuneflourescence (IF). After reaching this diagnosis, the instability and hearing loss was attributed to a possible neurosyphilis or an otosyphilis. The analysis of cerebrospinal fluid was normal. But as the otosyphilis is diagnosis of exclusion, and without another cause to justify the Meniere's-like syndrome, we decided to initiate antibiotherapy with sodium G

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penicillin with neurosyphilis dosage (4 MUI/4h) [5]. In the follow-up, the patient showed complete resolution of the NS with normal kidney function and disappearance of proteinuria. As regards the otological symptomatology, the patient refers improvement in deafness and instability; and he didn't have any gastrointestinal disturbance as the previous episodes again. In summary, the complete semiology that our patient presented could be explained by the syphilitic infection. As it is, our case could receive its famous appellation: "The great imitator".

Discussion

The syphilis as the "great imitator" could affect to multiple organs and could present a diverse semiology. In the stages where the spirochaete is disseminated in the organism, the disease would be easily mistaken with other systemic diseases as the Systemic Lupus Erythematosus (SLE) [6]. The syphilitic nephropathy is rare but it could be seen in the secondary/terciary stage or in latent infections [7]. It is presented as a flourish nephrotic syndrome, needing a kidney biopsy to its diagnosis. The membranous nephropathy is the most frequent found injury, probably caused by an immunological cross-reactivity between the syphilis and a kidney antigen. There has been described other more infrequent histopathological patterns [8]. The finding of "Full-House" pattern is highly indicative of autoimmunity and widely observed in Lupus Nephropathy (LN). These found elements could make us dismiss from the typical syphilitic affection [9]. However, the C1q deposits on the kidney tissue had been described in some viral infections as the Parvovirus B19, a well-known pathogen that could mimic LN [10,11]. In the co- infection of those two pathogens, the Treponema pallidum and Parvovirus B19, we could find overlapped histopathological features simulating a Lupus-like Nephritis, as it shows the IF of our case. By doing a review in the published literature we found two similar cases [7,12]. For this reason, the clinical-pathological diagnosis is Membranous Nephropathy due to Syphilis and Parvovirus B19 co-infection. On the otosyphilis, an early initiation of the treatment should allow a full recovery of deafness and the disoperation of instability, although is also frequent to leave some permanent damages [13]. At the same time, it has been described in the literature some cases of gastrointestinal symptoms caused by the Treponema pallidum as syphilitic gastritis or proctitis in the secondary syphilis, simulating an IBD symptomatology [14].

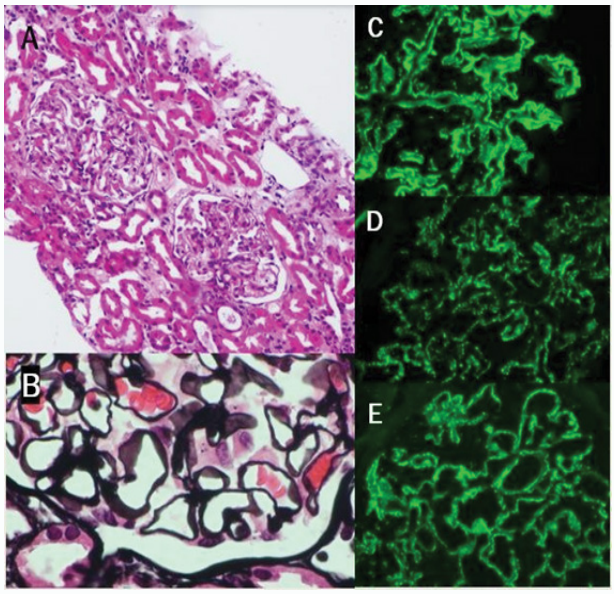

Figure 1: A) Renal biopsy with preserved parenchyma and normal featured glomeruli without clear deposit on the GBM nor mesangium, without important tubulointerstitial inflammation nor tubular athrophy (PAS stain *200). B) Abscense of spikes nor moth-eaten images all along the GBM (Methenamine silver stain *400). C, D and E) If of glomerular immune deposit (IgG, C3 and C1q, respectively).

Conclusion

Being in front this apparently motley case we could think of the syphilis as a sufficient reason that could explain the whole symptomatology of our patient, although we could be mistaken by using this "Ockham's razor". To be sure in our diagnosis, we must value the importance of a complete medical history with a wide study of the nephrotic syndrome's etiology due to a its vast variety of possible causes. And without forgetting that some infections could causes similar features as autoimmune diseases and could lend us to wrong diagnosis.

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