

Minimal change disease with concurrent thin basement membrane disease: A case report

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ABSTRACT

Minimal change disease (MCD) is a main cause of the nephrotic syndrome. Thin basement membrane disease (TBMD) is another disease characterized by microscopic hematuria. The present case is a young adult female who presented with classic nephrotic syndrome, but she had microscopic hematuria as well. Hematuria can be part of MCD in 21% of patients, but in this case, histopathological diagnosis confirmed MCD with concurrent TBMD. This was reported only in two cases, up to our literature review. Using steroids resulted in nephrotic syndrome improvement, but microscopic hematuria has persisted, which is mostly related to TBMD rather than a primary part of MCD. Up to our knowledge, this is the first case report of MCD with concurrent TBMD in Arab countries.

Keywords: Case report, female, minimal change disease, thin basement membrane disease

Introduction

Minimal change disease (MCD) is characterized by heavy proteinuria, leading to intravascular volume depletion and edema. Moreover, it is a major cause of nephrotic syndrome and it occurs in 15–40% of adults with nephrotic syndrome, but its incidence is more dominant in children.^[1] Thin basement membrane disease (TBMD) is characterized by microscopic hematuria without additional symptoms or progression to renal impairment.^[2,3] While hematuria could be part of MCD in 21% of patients,^[4] searching the literature, only two reported cases have linked hematuria in MCD to the concurrence with TBMD rather than a part of MCD.^[5,6] Here, we present the first case in Arab world.

Case Report

A 18-year-old female patient, previously healthy, presented with lower limb edema and puffy face that started 1 week before her presentation, associated with frothy urine. She reported another two previous similar attacks in the past 3 months, but with shorter duration and spontaneous recovery. This time, her symptoms did not improve spontaneously, for which she sought medical advice. She denied any history of recent upper respiratory infections, shortness of breath, chest pain, abdominal distention, or change in her urine color. There was no history for fever, skin rash, joint pain, hearing impairment, nonsteroidal anti-inflammatory drugs, or any new medications use. A family history of renal or hearing diseases, particularly Alport syndrome, was negative. Her surgical history was negative. Her vaccinations were up to date. She had no known allergy. She is a single, non-smoker, studying in the 12th grade with good

scholastic performance. On examination, her blood pressure and other vital signs were normal. Her face was puffy and she has bilateral pitting lower limb edema. There was no skin rash or active synovitis. Her cardiovascular, respiratory, and abdomen examinations were unremarkable. Laboratory tests showed normal complete blood count, blood urea nitrogen, creatinine, and electrolytes. Her albumin was 19 g/L (normal 40–50 g/L). Her urinalysis showed 3+ protein and 3+ blood and red blood cell (RBC) > 50/HPF, but it was negative for white blood cell. Her urinalysis results were confirmed twice. Her 24 h urine protein was 5.1 g/day (normal <150 mg). Her protein/creatinine random urine was 341 mg/mmol (normal < 15 mg/mmol). She had normal complements. Her low-density lipoprotein was 8.67 mmol/L. Her antinuclear antibody (ANA), anti-neutrophil cytoplasmic autoantibody (ANCA), cryoglobulins, hepatitis B virus, hepatitis C virus all were negative. Renal ultrasound showed mildly echogenic normal-sized kidneys. Considering significant microscopic hematuria, which is not classical in most cases of MCD, a renal biopsy was done. It showed features of MCD with normal light microscopy and kidney background [Figure 1a and b] and negative immunofluorescence. Electron microscopy (EM) revealed diffuse foot processes effacement and glomerular basement membrane with areas of thinning with an average thickness of 218 nm with no immune deposits [Figure 2a and b]. Hence, her biopsy EM findings explained her microscopic hematuria. She was started on prednisone 1 mg/kg. As a follow-up, 10 days after steroid, her symptoms resolved completely, her urinary protein became negative, and her protein/creatinine random urine was 21.2 mg/mmol (baseline 341 mg/mmol). She achieved clinical and biochemical complete remission of her MCD, but she continued to have persistent microscopic hematuria.

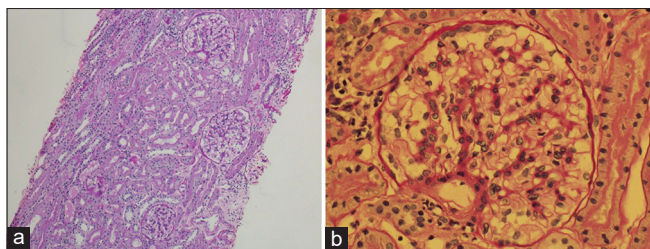


Figure 1: (a) Low power light microscopy showing normal kidney background (periodic acid-Schiff stain). (b) High power light microscopy showing normal-looking glomerulus (periodic acid-Schiff stain)

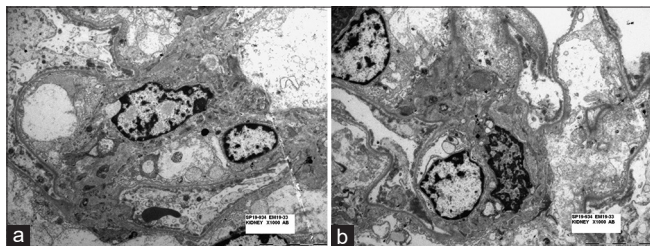


Figure 2: (a and b) Electron microscopy showing diffuse foot processes effacement and thinning of the basement membrane

Discussion

It is known that all MCD patients present with nephrotic-range proteinuria, while microscopic hematuria appears only in 21% of patients.^[4] In addition, MCD generally is characterized by normal-appearing glomeruli on light microscopy, absence of immune deposits, yet with diffuse foot processes effacement on electron microscope and negative staining on immunofluorescence.^[7] On the other hand, TBMD is characterized by microscopic hematuria without additional symptoms or progression to renal impairment.^[2,3] In addition, TBMD is diagnosed by EM by the presence of thinning in the basement membrane.^[3] Our patient had a puffy face with lower limb edema. Laboratory tests showed nephrotic range proteinuria and negative secondary workup. Her renal biopsy showed normal light microscopy, negative immunofluorescence, and diffuse foot processes effacement on EM, which are compatible with MCD. The present case had no family history of renal diseases, particularly Alport syndrome, but she had microscopic hematuria with urine RBCs of >50/(HPF). In addition, EM revealed glomerular basement membrane thinning to 218 nm in some areas instead of the normal range which was reported to be 330–460 nm in adults.^[8] These features reflect the presence of TBMD in addition to her MCD. Based on the histopathological diagnosis, she has MCD with concurrent TBMD. As mentioned above, hematuria can be part of MCD in 21%^[4] but also it can be related to concurrent TBMD. Based on our literature review, there were only two case reports reported the coincidence of MCD with TBMD,^[5,6] and our patient was the first case in Arab world. The first case report^[5] revealed elderly patients with combined MCD and TBMD in whom proteinuria responded to steroid. The second case report was for a 15-year-old boy who was

having TBMD associated with MCD and had hematuria in urinalysis. He was treated with corticosteroid and complete remission was achieved, but hematuria was persistent during the follow-up period.^[6] The primary drug used for MCD is steroid treatment with prednisone.^[7] In our patient, we used prednisone. In 10 days follow-up, there was an improvement in both her proteinuria and her symptoms which resolved completely, but microscopic hematuria has persisted. This was in agreement with the findings of the previous case report.^[6] This indicates that steroids could treat MCD-related proteinuria; however, hematuria was not affected by this management as it was caused by TBMD not MCD. Having a patient with a picture of MCD, but with microscopic hematuria, the differential diagnosis will include MCD itself in addition to concurrent TBMD.

Conclusion

In addition to the classic presentation with nephrotic syndrome, MCD can present also with microscopic hematuria as a part of the disease. Furthermore, hematuria can be related to a concurrence with TBMD. Treating condition will result in improvement in proteinuria but no hematuria. The persistence of hematuria after treatment in our case is related to the concurrence of TBMD rather than being a primary part of MCD.

Patient Consent

Written informed consent was taken from the involved patient.

Competing Interest

None.

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