

Review

Minimal Change Disease in Young Adults: A Case Report

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Abstract

Minimal Change Disease (MCD) represents the most common type of nephrotic syndrome in children (~80%), whereas in adults it accounts for 10% to 20% of cases. It seems to be associated with pollen allergy, asthma, NSAIDS use, and Hypoxia. A 25 years old girl with edema in both legs and facial puffiness and no other complain came to the clinic, her lab test was normal except for proteinuria 3.34g/24h and blood Albumin 1.9mg/dl, her ultrasonography for the kidneys was normal. Kidney Biopsy was normal and IF study was negative for immune related diseases. She was treated with corticosteroid; cyclosporine then tacrolimus so she could have good response.

Keywords: Kidney Biopsy; Minimal Change Disease; Nephrotic Syndrome; NSAIDS

Introduction

Minimal Change Disease (MCD), previously known as lipoid nephrosis or minimal lesion represents the most common type of nephrotic syndrome in children (~80%), whereas in adults it accounts for 10% to 20% of cases [1]. MCD received its name because of the minimal, if any, glomerular abnormalities present by light microscopy and by immunofluorescence, immunoglobulins or complement deposits are rarely found. If present, deposits are limited to the mesangium. MCD seems to be associated with pollen asthma, NSAIDS use, and Hypoxia [2].

Renal function is generally preserved but creatinine could slightly be elevated and hematuria is rare. MCD displays a higher rate of remission after corticosteroid treatment, has better long-term renal outcomes, and has an earlier onset than other patterns of nephrotic syndrome so corticosteroid treatment is usually effective in inducing remission, but relapse is common and repeated therapy often

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required. Among children with MCD, 25% never relapse, 25% relapse infrequently, and 50% have numerous relapses.

Case Report

A 25 years old woman (64 Kg in weight, 160 in Height) came to the clinic suffering from fatigue and swelling in her face and legs started 15 days ago with no other complain. She mentioned an onset of collapse after standing long time for once only, but no other previous medical history, and no medication use except for paracetamol with doctor prescription if needed. Her BP was 80/50mmhg at the time of collapse.

At the clinic: She looked well, her physical exam was perfect except for edema in both legs and around her eyes, her blood pressure was 110/70mmhg, heart beats 70 b/min, respiratory rate 17/min and body temperature 37°C.

An ultrasound for the abdomen was done which showed normal findings and her kidneys were as following:

- RK normal in shape and size and appearance 10.5cm long.
- LK normal in shape and size and appearance 11cm long.
- Chest X-ray was normal

She had lab tests which come as the following (Table 1):

Lab Tests	Results
White blood count	7890/mm ³ (normal 3500-13000/ mm ³)
Hemoglobin	12g/dl (normal 12-16g/dl)
Platelet count	278,000 mm ³ (normal 150,000-400,000 mm ³)
Serum Creatinine	0.9(normal 0.6-1.2 mg/dl)
ESR	105/110 mm/h (normal 0-15 mm/h)
ALT	32 IU/L (normal 7-40 IU/L)
AST	40 IU/L (normal 5-35 IU/L)
Albumin	1.9 g/dl (normal 3.5-5 g/dl)
TSH	4 mIU/L (normal 0.4-4.8 mIU/L)
C3	109mg/dl (normal 63-192 mg/dl)
C4	42 mg/dl (normal 15-52 mg/dl)
ANA	Neg
Anti DNAds	Neg
Anti GBM	Neg
ANCAc	Neg
ANCAp	Neg
HBsAg	Neg
Anti HCV	Neg
Urinalysis:	normal except for protein +++
24 hour protein	3.34g/dl, volume 1670 ml

Table 1: Lab tests.

She had a Kidney Biopsy

No glomerular disease is present. Acute tubular injury, IF study ruled out immune mediated study. There were two differential diagnoses: ATN and Acute Tubular Injury and MCD (Minimal Change Disease) [3]. She was treated with prednisolone 1mg/kg/day for 6 months with good response to treatment then corticosteroid trapped slowly until it was stopped and she became free of disease for only one month (no symptoms or complains, serum creatinine 0.6 and no proteinuria in 24 hour urine) so she had a relapse with increasing in proteinuria 5g/24h urine. She took cyclosporine 5mg/kg bid (therapeutic blood range CO=100) for 3 months with no response to treatment so the medication was stopped and replaced with tacrolimus 1mg bid (therapeutic blood range = 5.6) with fast response to treatment in only one month (her protein in 24 h urine was 500mg,no symptoms). Her proteinuria now after 3 month of treatment is 80 mg in 24 hour urine [4,5].

Discussion

MCD is a disease of childhood we should never miss it in young adults with overt proteinuria and normal biopsy especially when the patient has a story or a diagnosis of ATN (Hypotension, Hypovolemia...etc.) ATN alone is not a usual cause of overt proteinuria and the decision for having biopsy should be taken ASAP in such case. But even if the biopsy was normal (no obvious reason for proteinuria) we should keep in mind the chance of having MCD. As known ATN should be recovered in a few days by itself with supportive treatment and with resolving its cause, but in this case we added corticosteroid because the proteinuria continued to increase even after resolving the cause and the patient had normal blood pressure and euvolemic blood statuses for at least 2 months which support the diagnosis of MCD. MCD is treating first with corticosteroid and if it relapsed after trapping it we can treat again with corticosteroid for longer time (another year) if another relapse happened we move to cyclosporine and if there is no response we can replace it with another immunosuppressive drug.

But as mentioned before she had a relapse with greater proteinuria than the first soon after trapping corticosteroid that we had to put the patient on immunosuppressive therapy in order to stop the proteinuria. The questions that is still in mind whether it is preferable to treat with immunosuppressive therapy exactly at the time of diagnosis rather than corticosteroid ,since the rate of relapse is high among young adults especially after trapping it, or starting with corticosteroid and follow the protocols, and was it better choice if we started with tacrolimus first or not?!!...Studies should be done until we have the answers....

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