

Recurrent IgM nephropathy associated with seasonal allergic rhinitis

M. Al-Ahmad, S. Betschel*, P. Vadas

Key words: allergens; nephrotic syndrome; recurrent; seasonal rhinitis.

IgM nephropathy is an ill-defined type of glomerulonephritis characterized by predominant, if not exclusive, deposition of IgM in the glomerulus and is considered a variant of the nephrotic syndrome. We report a case of recurrent IgM nephropathy associated with seasonal allergic rhinoconjunctivitis in an adult.

A case of recurrent IgM nephropathy associated with seasonal allergic rhinoconjunctivitis in an adult.

A 36-year-old woman, with a history of seasonal allergic rhinoconjunctivitis due to type-1 allergic hypersensitivity to tree and grass pollen, presented with recurrent attacks of ankle swelling and frothy urine during the grass pollen season. She was found to have nephrotic range proteinuria and elevated cholesterol levels. She had a total of three attacks of nephrotic syndrome during three consecutive grass pollen seasons. Her recurrent nephrotic syndrome was associated with symptoms of rhinoconjunctivitis. Two years prior to her first presentation of nephrotic syndrome, she received desensitization therapy with Pollinex preseasonal tree, grass, and ragweed pollen vaccine. One year later, she only received Pollinex-T tree pollen vaccine. Immunotherapy reduced but did not eliminate her rhinoconjunctivitis symptoms. She had no immunotherapy during the years she presented with the nephrotic syndrome.

Skin prick testing was positive to tree (8 mm wheal diameter), grass (8 mm wheal diameter), and ragweed (6 mm wheal diameter), and histamine (6 mm wheal diameter). A 24-hour urine

collection showed 7.7 g of protein. Her serum immunoglobulin levels, including IgE, were measured during the first two presentations of the nephrotic syndrome and were normal. Pollen specific IgE was not quantitated. A kidney biopsy performed during the first episode of nephrotic syndrome revealed 'minimal changes' on light microscopy. Immunofluorescence identified IgM deposits and electron microscopy detected dense mesangial deposits.

The first two episodes of nephrotic syndrome were treated successfully with prednisone 50 mg/day for 1 month with a tapering course and her proteinuria resolved within 4 days of treatment. During her third relapse, addition of azathioprine 75 mg/day was required as she relapsed when treated only with prednisone. She responded well to this therapy with complete resolution of her proteinuria.

The etiology and pathogenesis of the steroid-sensitive nephrotic syndrome remains obscure. It is associated with 'minimal' histological changes in the glomeruli and although relapses often follow infections, the nature of the triggering process is unknown. Only a few publications have reported a role for allergy in the development and maintenance of this disease, particularly in childhood (1, 2). Reports of an association between recurrent nephrotic syndrome and inhaled allergens are very uncommon and are absent from the recent literature (3, 4). IgM nephropathy has been considered a variant of the nephrotic syndrome. However, patients with this disorder are less responsive to steroid therapy than those with minimal change nephrotic syndrome (5), which may have accounted for this patient's need for azathioprine during her third relapse.

The beneficial effects of desensitization therapy on the course of nephrotic syndrome have been uncertain. Some have reported a positive outcome with specific allergy therapy (6). It is not clear in our case if the previous specific allergy therapy had delayed presentation of nephrotic syndrome or precipitated it. The recurrent IgM nephropathy as described in this patient suggests that the potential

absorption and renal deposition of pollen may result in glomerular inflammation manifesting as nephrotic syndrome. Recurrent nephrotic syndrome with minimal change disease has been reported in association with seasonal allergic rhinitis (4), however, IgM nephropathy associated with seasonal allergic rhinitis has not previously been described in an adult patient. Allergists should be aware of this association in patients with a history of recurrent nephrotic syndrome as it may impact on their decision to institute immunotherapy. Furthermore, prophylactic immunosuppression may be warranted during the relevant pollen season in patients with the nephrotic syndrome.

*Division of Allergy and Clinical Immunology
St. Michael's Hospital
30 Bond St.
Toronto
Ontario
Canada M5B 1W8
Tel: +1-416-8851566
E-mail: wonbet@sympatico.ca

Accepted for publication 4 April 2005

Allergy 2005; 60:1332

Copyright © Blackwell Munksgaard 2005

DOI: 10.1111/j.1398-9995.2005.00889.x

References

1. Lin CY, Lee BH, Lin CC, Chen WP. A study of the relationship between childhood nephrotic syndrome and allergic diseases. *Chest* 1990;**97**:1408-1411.
2. Meadow SR, Sarsfield JK. Steroid-responsive and nephrotic syndrome and allergy: clinical studies. *Arch Dis Child* 1981; **56**:509-516.
3. Hardwicke J, Soothill JF, Squire JR, Holti G. Nephrotic syndrome with pollen hypersensitivity. *Lancet* 1959;**1**:500-502.
4. Reeves WG, Cameron JS, Johansson SG, Ogg CS, Peters DK, Weller RO. Seasonal nephrotic syndrome. Description and immunological findings. *Clin Allergy* 1975;**5**:121-137.
5. Border WA. Distinguishing minimal-change disease from mesangial disorders. *Kidney Int* 1988;**34**:419-434.
6. Richards W, Olson D, Church JA. Improvement of idiopathic nephrotic syndrome following allergy therapy. *Ann Allergy* 1977;**39**:332-333.