



Jameela A. Kari · Hussain Bamashmous  
John D. Mahan

## Steroid-sensitive nephrotic syndrome and juvenile idiopathic arthritis

Published online: 15 August 2002  
© IPNA 2002

**Keywords** Steroid-sensitive nephrotic syndrome ·  
Juvenile idiopathic arthritis

Sirs,

Secondary nephrotic syndrome (NS) and kidney involvement are common in adults with rheumatoid arthritis (RA). Nephropathy in patients with RA varies and could be due to RA itself and/or to anti-rheumatic therapy [1]. Renal involvement usually occurs several years after the diagnosis of RA [2] and can present as glomerulonephritis (mesangial, membranous, focal proliferative, and minimal change lesions), amyloidosis, or interstitial nephritis [1, 2]. Similarly, NS may complicate juvenile idiopathic arthritis (JIA) and has been described in adults [3], children [4], and infants [5] with JIA. In these patients, it has been characterized by amyloidosis [3, 4, 5] and glomerulonephritis [6].

We report a case of primary steroid-sensitive NS (SSNS) and JIA in which nephrosis preceded the development of JIA by years. A 2.5-year-old Serlankian girl presented to King Abdul Aziz University Hospital with SSNS. She responded to steroid therapy, but demonstrated a frequently relapsing NS. She was maintained on low doses of alternate-day prednisolone to prevent relapses of NS. Four years later, at the age of 6.5 years, she presented with monoarticular arthritis in the knee joint. She had left knee effusion that lasted for more than 6 months. Slit-lamp examination of her eyes revealed no abnormalities. At presentation of her arthritis, her erythrocyte sedimentation rate was elevated at 96 mm/h, her complement levels were normal, and anti-nuclear antibody and

anti-DNA antibody titers were negative. Radiographs demonstrated left knee effusion with slight enlargement of the left femoral epiphysis, thought to be due to chronic inflammation. Magnetic resonant imaging revealed marked effusion in the left knee with features suggestive of synovitis, but no cartilaginous or osseous lesions. The patient was diagnosed with monoarticular JIA after excluding infection, as synovial fluid showed a normal cell count and negative culture. A skin test for tuberculosis was negative. She required intra-articular steroid injections and oral naproxen to control her joint symptoms. Her NS continued in remission with minimum doses of alternate-day prednisolone. Her height standard deviation score at 7.5 years of age was  $-0.16$  and her body mass index was 23.3.

The clinical pattern in which childhood SSNS precedes JIA is quite different from that described as secondary NS complicating JIA or RA. Mesangial glomerulonephritis is commonly identified as the renal lesion when renal disease is described in adults with RA; this concurrence has been described in only one child with JIA [6]. Similarly, the minimal change NS (MCNS) lesion has been described as a cause of NS associated with RA in adults, but not in children [1]. Secondary NS associated with chronic arthritis is usually steroid resistant, difficult to treat, and requires other immunosuppressive therapies to achieve control [3]. Nephropathy complicating RA usually occurs several years after the onset of RA. Our patient presented with renal manifestations before the onset of features of JIA. While the occurrence of SSNS and JIA could be coincidental, it is possible that chronic juvenile arthritis and MCNS share some common pathogenic basis or mechanism [7]. Both conditions are associated with T lymphocyte dysfunction, often triggered by viral infections, the production of circulating factor(s), and abnormal cytokine expression. Both conditions also seem to have genetic predisposition and linkage to HLA-DR antigens of major histocompatibility complex type II. This patient represents the first reported example of a child with steroid-responsive NS who subsequently developed JIA.

J.A. Kari (✉) · H. Bamashmous  
Pediatrics Department, King Abdul Aziz University Hospital,  
PO Box 80215, Jeddah 21589, Saudi Arabia  
e-mail: jkari@doctors.org.uk  
Tel.: +996-2-55677904, Fax: +996-2-26743781

J.D. Mahan  
Department of Pediatric Nephrology, The Ohio State University,  
Ohio, USA

## References

1. Helin HJ, Korpela MM, Mustonen JT, Pasternack AI (1995) Renal biopsy findings and clinicopathologic correlations in rheumatoid arthritis. *Arthritis Rheum* 38:242-247
2. Cantagrel A, Pourrat J, Fournie B, Conte JJ, Fournie A (1990) (Renal involvement in rheumatoid polyarthritis.) L'atteinte renale au cours de la polyarthrite rhumatoïde. *Rev Rhum Mal Osteoartic* 57:303-307
3. Deschenes G, Prieur AM, Hayem F, Broyer M, Gubler MC (1990) Renal amyloidosis in juvenile chronic arthritis: evolution after chlorambucil treatment. *Pediatr Nephrol* 4:463-469
4. Hamdan J, Manasra K, Ilahi F (1986) Amyloidosis secondary to juvenile rheumatoid arthritis: a case report from Saudi Arabia. *Ann Trop Paediatr* 6:279-282
5. Garcia MC, Garcia CJ, Pena A, Torreblanca J, Antelo MC, Collado F (1979) (Two cases of secondary amyloidosis.) Dos casos de amiloidosis en la infancia. *An Esp Pediatr* 12:534-541
6. Gedalia A, Mendez EA, Craver R, Vehaskari M, Espinoza LR (2001) Renal involvement in juvenile rheumatoid arthritis: report of two cases. *Clin Rheumatol* 20:153-156
7. Otero GA, Esteban J, Martinez SJ, Marina G, Rodriguez GG (1991) (Chronic juvenile arthritis and minimal change glomerulonephritis (MCGN), the same pathogenic basis?) Artritis crónica juvenil (ACJ) y glomerulonefritis de cambios mínimos (GNCM), misma base patogénica? *An Med Interna* 8:413-414