Rapidly progressive glomerulonephritis following Chlamydia pneumoniae pneumonia in a child

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Abstract
Acute renal failure following Chlamydia pneumoniae pneumonia is rarely reported in adults. We present an observation in a 6-year-old child, with hydronephrosis of 3 degrees to the right and the left uninhabited lodge from birth, who had C. pneumoniae pneumonia treated with macrolide antibiotics for a period of 10 days, without any other nephrotoxic drug, in particular nonsteroidal anti-inflammatory drugs. The etiological investigations showed positive C. pneumoniae antibodies, reduced serum concentrations of C3 complement. No uveitis was noted. The diagnosis was rapidly progressive glomerulonephritis after Chlamydia pneumoniae infection and hemolytic anemia in a child with only one kidney. C. pneumoniae pneumonia should be considered a differential diagnosis of community-acquired pneumonia, especially in cases of poor response to conventional antibiotic therapy. It may be associated with tubulointerstitial nephropathy and/or glomerulonephritis whose severity varies in children as in adults. Early and effective treatment of C. pneumoniae infection with macrolide antibiotics usually provides favorable progression of renal function.

Introduction
We report an unusual case of a 6 years old sicilian boy with only one kidney affected by progressive glomerulonephritis after Chlamydia pneumoniae infection and hemolytic anemia. We had treated with macrolide antibiotics for a period of 10 days with seriated serological surveys that documented a progressive decrease in antibody titer. After 2 months, in a visit to the surgery of pediatric nephrology, our patient had diffuse petechial lesions. Blood tests showed thrombocytopenia and therefore has been practiced therapy with immunoglobulins and corticosteroids, with resolution of symptoms. We also conducted the examination of the bone, in the suspicion of a Fanconi anemia, but it was negative.

Case summary
Our patient is a third son, born at term by normal pregnancy with neonatal phenomena reported in the norm. Breastfeeding for one month, after artificial. Weaning from the fourth month. Psychomotor development and growth in height-weight standard. On the second day of life, after picelic dilation detected during fetal ultrasound, ultrasound scan was performed and showed a picture of hydronephrosis of 3 degrees to the right and the left uninhabited lodge. Thus the child came to undergo surgery to reduce hydronephrosis to the right. He comes to our attention at age 4, when in the course of an acute febrile episode, presents gross hematuria. The tests performed showed severely impaired renal function, hypocomplementemia (C3), a positive direct Coombs test and frankly pathological urine, with a carpet of red blood cells and proteinuria. The history and clinical findings allow us to affirm that our patient had a hyperacute glomerulonephritis with chronic renal failure and hemolytic anemia.

The immunological process triggered by infectious cause has been documented by hypocomplementemia (C3) and by a positive direct Coombs test. Because hyperacute onset of glomerulonephritis with acute renal failure and severe anemia with positive direct Coombs test we performed steroid boluses followed by oral steroid therapy. Furthermore, the investigations have documented a positive for chlamydia pn. So we have practiced therapy with macrolide for ten days with serological surveys that had documented a progressive decrease in antibody titer.

Discussion
Chlamydia pneumoniae, a cause of respiratory tract infection [1], was recognized as the third Chlamydia species in 1986 [2]. In addition to respiratory diseases, the organism has...
been linked with atherosclerosis and related clinical manifestations such as coronary heart disease, carotid artery stenosis, claudication and stroke [3, 4]. Recent evidence suggests C. pneumoniae organisms can survive and multiply within, not only macrophage, but also polymorphonuclear neutrophils (PMN) [5]. There is a possibility that persistent infection of C. pneumoniae within PMN may lead to autointimmy, and plays a role in enhancement of the process of ANCA production. In addition, C. pneumoniae infected macrophages adhere to the endothelium and migrate to the subendothelium in atherosclerotic lesion. These processes result in the release of cytokines and growth factor synthesis, which up-regulate endothelial cell adhesion molecules, leading to increased leucocyte adhesion [6]. These components, including macrophage infiltration, cytokine release, up-regulation of adhesion molecules and leucocyte adhesion, are indispensable to the pathogenesis of MPO-ANCA-associated glomerulonephritis (GN) [7]. Furthermore, C. pneumoniae produces chlamydial heat shock protein (cHSP) in infected cell macrophages and elicits a hypersensitivity reaction of the host, resulting in severe endothelial injury [8]. It is likely that the focal inflammatory reaction would be strongly enhanced resulting in necrotizing vasculitis in the presence of circulating ANCA, the production of which may be the result of interactions between T cells and B cells activated by microbial superantigens.

In our case, we assumed the association between C. pneumoniae infection and the development of MPO-ANCA-associated GN. The presence of positive IgM ab against C. pneumoniae is closely associated with the development of MPO-ANCA-associated GN, while the presence of positive IgG and IgA ab against C. pneumoniae as a risk factor don't reach statistical significance. In addition, the serological pattern of increased IgM and IgA titres has been suggested to indicate active infection and chronic persistence of active infection, respectively. IgG titres in the absence of IgM or IgA titres may be a serological marker of an older, inactive infection. Thus, active or chronically active rather than inactive C. pneumoniae infection may increase the risk of MPO-ANCA-associated GN. There is a possibility that C. pneumoniae influences the pathogenesis of MPO-ANCA-associated GN, in fact there is a significantly higher prevalence of active C. pneumoniae infection in patients with MPO-ANCA-associated GN [9]. This case represents another interesting facet of this infection, in fact the hypothesis that C. pneumoniae is aetiologically involved in MPO-ANCA associated GN is of particular therapeutic relevance, because this is a potentially eradicable infectious agent. For these reasons an early diagnosis is important considering that the tubulointerstitial nephropathy and/or glomerulonephritis subsequent to infection can be slowed from an early and effective treatment with macrolide antibiotics.

References

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