

# **Crescentic Glomerulonephritis Associated with Bacterial Endocarditis - Antibiotics alone may be Sufficient. a case report**

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### **Abstract**

Crescentic glomerulonephritis complicating the course of bacterial endocarditis carries a poor prognosis. Ideal treatment strategy is not clearly defined. In addition to antibiotic treatment, plasmapheresis and steroids have been used with variable results. Here we report a case of 40-year old female who was referred because of generalized body swelling and decrease urine output associated with low grade fever on and off for two to three months. She was diagnosed to have acute renal failure secondary to tricuspid valve endocarditis. Staph aureus was isolated from blood culture and renal biopsy showed crescentic glomerulonephritis. She received dialysis support and antibiotics and had complete recovery of renal function 6 weeks after initiation of therapy. Eradication of infection with antibiotics treatment may be sufficient for resolution of crescentic glomerulonephritis associated with infective endocarditis in some cases.

### **Introduction**

Infective endocarditis is a microbial infection of the endocardial surface of the heart and has been classified as acute or sub acute-chronic and chronic on the basis of the tempo and severity of the clinical presentation and the progression of the untreated disease.<sup>1</sup> Virchow described sys-

temic embolization for the first time as a complication of endocarditis.<sup>2</sup> Common renal diseases described in the literature since then are post infectious immune complex mediated glomerulonephritis (GN), a drug induced acute interstitial nephritis, embolic disease, focal necrotizing GN and crescentic glomerulonephritis.<sup>3-6</sup> The histologic findings in glomerulonephritis are similar to those in post-streptococcal glomerulonephritis or membrano-proliferative glomerulonephritis: hypercellularity and immune deposits in the glomerular capillary wall. In comparison to post streptococcal disease, the duration of antigenemia is often prolonged in patients with endocarditis or an infected shunt due to delay in diagnosis and treatment. Variable degree of renal dysfunction occurs in those who develop immune complex glomerulonephritis. In those who develop crescents, renal failure sufficiently severe to require dialysis may supervene. Management of such patients is problematic, as immunosuppression can hamper bacterial clearance and antibiotic treatment alone may not be sufficient to prevent fibrotic healing and irreversible renal failure. We report a case of crescentic GN with renal failure due to endocarditis who had complete recovery of renal function following treatment with antibiotics only.

## Case Report

A 40 year old female with no significant past medical history presented on 24 January 2002 with the complaint of decreased urine output for two weeks and generalized body swelling for one month. She had a history of low grade fever on and off associated with productive cough with scanty white/yellow sputum for 2-3 months. She denied any history of arthralgia, photosensitivity, sore throat, hematuria, epistaxis and hemoptysis. Past medical history was negative for diabetes, hypertension, tuberculosis or any other chronic illness. Physical examination showed a middle aged female of average height and built in mild to moderate distress. Her vital signs were as follows: Temperature 99°F, blood pressure 130/80 mmHg, pulse 96/min, RR 24/min. She had facial and pedal edema and her jugular pressure was increased. Examination of chest revealed bibasilar rales which were more prominent on right side. Rest of the physical examination was unremarkable.

Figure. glomerulus showing cellular crescent (x 200 silver stain).

Laboratory investigations showed haemoglobin of 6.99 gm/dl, white cell count of 27,700/cmm, platelet of 354,000/cmm. Urea was 141 mg/dl, creatinine 6.0 mg/dl, Na 125 mmol/lit, K 5.0 mmol/lit, and urinalysis showed 2+ albuminuria, blood ++, numerous RBCs /high power field with RBC casts. Ultrasound showed normal size kidneys and chest x-ray (CXR) showed peri hilar congestion with a questionable infiltrate at the right base. Patient was started on hemodialysis with ultrafiltration due to the presence of volume overload. Pan cultures were sent and on second day of admission patient was started on I/V amoxicillin + clavulanic acid. Blood cultures sent on day 1 and 2, showed methicillin sensitive Staph aureus. Echocardiogram was performed due to the presence of Staph aureus in blood which showed vegetations on tricuspid valve. Patient's serology which was sent on admission showed C3 of 0.446 g/lit (Normal range 0.55-1.20), C4 0.073 (Normal range 0.2-0.5), IgG 28.78 (Normal range 8.0-17.0) but anti

nuclear antibodies, Hepatitis B surface antigen (HbsAg), Hepatitis C virus antibodies (Anti HCV) were negative. Renal biopsy was performed which showed upto six glomeruli, five of them having diffuse extra capillary epithelial proliferation forming crescents as shown in Figure. All the crescents were cellular. Diffuse severe degree of acute tubular necrosis (ATN) and mild mixed inflammatory cell infiltration in the interstitium were noted. Immunofluorescence showed granular membranous positivity of IgG, IgM, C3 and C1q while IgA was negative. Electron microscopy (EM) revealed large intramembranous electron dense deposits. Repeat CXR performed on 3<sup>rd</sup> hospital day showed cavities lesions in the lungs, one on each side. Due to the presence of Staph bacteremia and vegetation, it was decided not to give any immunosuppressive therapy and to continue with antibiotics only. Fever subsided after 2 weeks and antibiotics were continued for total of 4 weeks. Patient gradually had recovery in renal function after requiring seven sessions of hemodialysis and was discharged on 26th hospital day with a serum creatinine of 2.5mg/dl. On her outpatient follow up, 4 weeks after discharge, she had a completely normal renal function with a serum creatinine of 0.6 mg/dl.

## Discussion

Infective endocarditis can present as apparently primary renal disease. The clinical manifestations are similar to those in other forms of acute glomerulonephritis. There is variable degree of hypertension and renal insufficiency.<sup>3-6</sup> Urine examination shows haematuria, red cell and other cellular casts. Complement levels are reduced in both conditions, indicating activation of complement pathway. A variety of organisms may be involved in those developing glomerulonephritis, Staph aureus in acute infective endocarditis, Strep viridans in sub acute infective endocarditis and Staph epidermidis in shunt nephritis. Majumdar in his series of biopsies performed for acute renal failure due to endocarditis found acute glomerulonephritis in 9 out of 20 patients, a acute tubular necrosis (ATN) in 4/20, acute tubulo-interstitial nephritis 5/20 and pre-existing glomerular disorders in remaining patients.<sup>7</sup> There was a lack of immunoglobulin and complement deposits in glomeruli in patients with vasculitic lesions, but not in other types of glomerulonephritis. Neugarten and others reported focal glomerulonephritis in 8% of patients and diffuse glomerulonephritis in 14% with glomerular crescents in half of each type.<sup>8</sup> Conlon et al found increase age, history of hypertension, thrombocytopenia, presence of staphylococcus aureus and prosthetic valve infection as the independent risk factors for acute renal failure in patients with endocarditis.<sup>9</sup>

Although antibiotic prophylaxis and treatment have reduced the incidence of sub acute bacterial endocarditis

and concomitant glomerulonephritis, *Staph aureus* has become the major cause of acute bacterial endocarditis with a high incidence of glomerulonephritis. Patients with infective endocarditis who presented with advanced renal failure usually have poor prognosis.<sup>7</sup>

Effective strategies to treat rapidly progressive glomerulonephritis associated with infective endocarditis have not been established. In the majority of patients with infective GN, immune complexes are considered to participate in the progression of glomerular abnormalities<sup>10</sup> and steroid therapy, immunosuppressive therapy, or plasmapheresis have been performed in addition to antibiotic therapy. Orfila et al reported a case of infective endocarditis induced crescentic GN in whom there was complete recovery with antibiotics<sup>11</sup> only but there have been case reports in which antibiotics alone showed no efficacy.<sup>8</sup>

We report a case of infective endocarditis induced crescentic GN who despite of her sub-acute course presented with *Staph bacteremia* and advanced renal failure. She was treated with antibiotics only keeping in view the risk of immunosuppressive therapy. Advanced renal failure on presentation is associated with both failure of antibiotic therapy to eradicate infection and failure to recover renal function<sup>3</sup>, which is in contrast to recovery of renal function, normalization of serum complement level, and remission of other clinical features of glomerulonephritis in patients with mild to moderate renal impairment. Favourable clinical course of the patients treated with antibiotics only is thought to be due to the gradual and small amount of immune complex deposits that do not induce severe glomerular damage<sup>11</sup> which was not the case in our patient

who despite presentation with advanced renal failure, both clinically and histologically responded completely to antibiotics. Reduction and disappearance of bacterial antigens with antibiotic treatment may well have allowed the natural clearance of immune complexes and accompanying glomerular inflammation without much residual scarring. An analogy can be drawn with post streptococcal GN with superimposed crescents formation which has been shown to resolve spontaneously without any treatment.

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