#1986 - A case report of C1q Nephropathy	
Authors and Affiliations	Hormat Rahimzadeh, Assistant Professor of Nephrology, Sina Hospital, Tehran University of Medical Sciences. Mehdi Kashani, Sina Hospital, Tehran University of Medical Sciences Ziba Aghsaei Fard, Assistant Professor of Nephrology, Sina Hospital, Tehran University of Medical Sciences
Corresponding Author and email	Hormat Rahimzadeh. h-rahimzadeh@sina.tums.ac.ir
Body	**Introduction**: C1q nephropathy is a rare glomerular disease and such as IgA, IgM nephropathy is an immune complex-mediated disease. C1q nephropathy was first described in 1982 by Jones, but reported in detail and named in 1985 by Jennette.The main clinical manifestations of C1q nephropathy are nephrotic syndrome, proteinuria and hematuria. Immunofluorescence examination shows mesangial C1q deposition is the main diagnostic basis. C1q nephropathy usually occurrs in older children and young adults, however, we introduce a 48-year-old man who was diagnosed as C1q nephropathy and had complete remission after steroid drugs treatments. **Case report**: The patient was a 48-year-old woman who was admitted to the hospital with lower extremity and scrotal edema from one month ago. He also had generalized weakness and fatigue. He was also suffering from chronic headaches that was diagnosed with migraines headache and was controlled at that time. . There was no dyspnea or urinary symptom. He didn’t mentioned any other problems. On examination, her weight was 65 kg, blood pressure 130/80 mmHg, respiratory rate 18/min, and pulse 80 beats/min. Urinalysis revealed proteinuria (3+) , hematuria (2+) and RBC cast. . The 24 h proteinuria was 6616 mg. His serum creatinine was 1.1 mg/dL, blood urea was 40 mg/dL, albumin 2.5 g/dL, cholesterol 242 mg/dL and hemoglobin 14 g/dL. Serological and

secondary tests were negative.Renal ultrasonography showed normal-sized kidneys with maintained corticomedullary differentiation.

After a few days he showed gross hematuria and creatinine rising to 2.5 mg/ dL. we performed a kidney biopsy.In all glomeruli, mesangial matrix expansion and hypercellularity were seen. Four glomeruli showed global endocapillary hypercellularity. No significant inflammatory cell infiltration, crescent, necrosis or duplication were not identified. Immunofluorescence test revealed full-house pattern staining without IgA. The staining of Clq was 2+ in distribution, which had strong mesangial and GBM pattern. Electron microscopic examination demonstrated mesangioprolifrative glomerulonephritis with mesangial and few subendothelial electron dense deposits.

Intravenous methylprednisolone was started and followed by 1mg/kg prednisolone daily and cellcept 1000 mg every 12 hours. His edema decreased and after two weeks, our patient was discharged.

After three month, edema was completely recovered and serum creatinine was 1.3 mg/dl.

Conclusion:

C1q nephropathy is a poorly understood disease with distinctive immunopathologic manifestations.Although responsiveness to steroid and immunosuppressive drugs is poor, but our patient had good response.

References

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