# Immunotactoid Glomerulopathy in a Patient with Hepatitis C Viral Infection

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We present the case of a 55 year old African American male who was admitted for an acute right posterior middle cerebral artery ischemic infarction. Appropriate management of thrombotic stroke was provided. The patient was noted to have edema, hypoalbuminemia, hypercholesterolemia and 6.6 g proteinuria on a spot urine protein to creatinine ratio. His serum creatinine was normal. Work up for nephrotic syndrome was initiated. His past medical history is significant for chronic alcohol abuse, drug abuse, hypertension, and gout. Significant laboratory testing revealed: BUN 5, creatinine 0.98, glucose 91, total protein 5.8, albumin 1.1, positive for Hepatitis C with HCV antibody index >11.0 and HCV Quant by PCR 36,000 IU/mL, and a Lupus Russell viper venom time 1.7 consistent with a lupus inhibitor. Urinalysis showed 30 RBCs.

As part of the proteinuria work up, a renal biopsy was performed. The biopsy showed a membranoproliferative pattern with marked diffuse thickening of the glomerular basement membranes (GBM), focal increased glomerular lobulation with diffusely increased mesangial matrix and mesangial cells. Renal tubules were generally well-preserved with minimal tubular atrophy and with minimal interstitial fibrosis with patchy chronic inflammatory infiltrate. PAS stain highlighted the mesangial matrix and GBM thickening. Silver stain demonstrated thickened GMB with occasional spikes. A Congo red stain was negative. Immunofluorescence studies revealed “smudged” diffuse global IgG and C3 granular deposits with lambda clonality. Electron microscopy findings included large, 30-40nm in diameter, hollow microtubular subepithelial deposits with parallel arrangement and spike formation, producing a membranous pattern (Figure 1). GBM appeared variably thickened with focal areas with slightly increased electron density and mesangial hypercellularity.

In conclusion, we report a case of a patient with massive proteinuria, hypoalbuminemia, hypertension, hematuria, high HCV viral RNA load and a renal involvement with non-amyloid subepithelial microtubular deposits most consistent with immunotactoid glomerulopathy. The patient showed no diagnostic evidence of a lymphoproliferative disorder or monoclonal gammopathy. This represents, to our knowledge, the second report of immunotactoid glomerulopathy with HCV infection [1] as the only known source of immune stimulation. The role of the lupus inhibitor is unknown.

**References**:

[1] Markowitz GS, Cheng JT, Colvin RB. *et al*. (1998) *J Am Soc Nephrol Journal* **9**:2244.

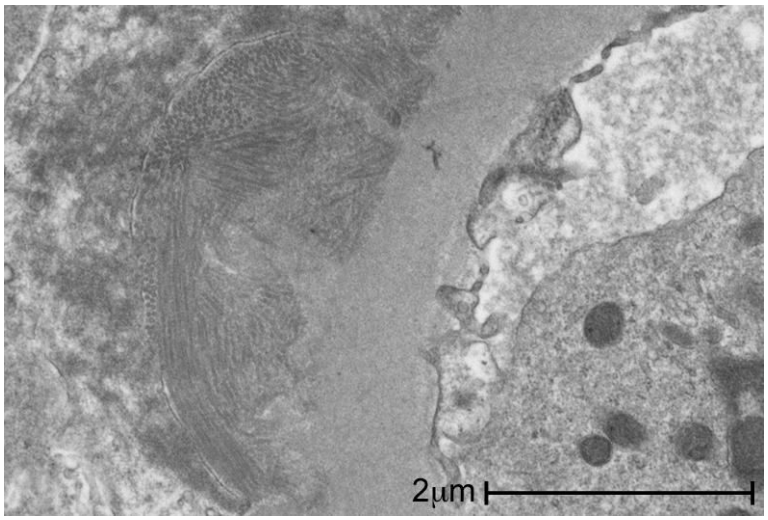


**Figure 1.** Subepithelial microtubular deposits with parallel arrangement and spike formation.