

## Association of IgA Nephropathy with Hepatitis C

SKK Malik\* , Dhruv Kant Mishra\*\*

\* Assistant Professor, PG Dept. Of Medicine, Subharti Medical College, Meerut

\*\* Assistant Prof., PG Dept. Of Medicine, Subharti Medical College, Meerut

Corresponding Author: SKK Malik

---

**Abstract:** Case report of 21 years old male who presented with fatigue, oliguria and generalised edema. Further investigations revealed proteinuria in the tune of 6gm/day and microscopic hematuria. Renal biopsy was done which gave impression of Mesangioproliferative glomerulonephritis with IgA deposits suggestive of IgAN (Berger disease). To assess the cause of IgA nephropathy, screening was performed which was normal except for Hepatitis C positive with genotype '3'. Patient was put on oral Daclatasvir and sofosbuvir which led to marked reduction in proteinuria after 12 weeks of treatment. It was assumed that Hepatitis C manifested as IgAN. Therefore this case highlights the possibility of association between Hepatitis C and IgAN (Berger's disease).

---

Date of Submission: 11-08-2018

Date Of Acceptance: 25-08-2018

---

### I. Introduction

Hepatitis C virus is a major cause of chronic liver disease. Moreover HCV infection has been related with immunologically mediated renal disease.<sup>(1-5)</sup> In addition, in chronic liver disease of any etiology, mesangial glomerular deposits of IgA can appear resulting in lesions similar to those observed in idiopathic IgA nephropathy (Berger's disease). We report a patient with Hepatitis C who was diagnosed as having Berger's disease and its marked improvement with Daclatasvir and sofosbuvir therapy.

### II. Case Report

A 21 years old male patient presented to us with history of fatigue, generalised edema and oliguria since 3.5 months. The patient was apparently well with no history of any renal disease or any history suggestive of any connective tissue disorder. The patient was not on any medication prior to this.

On examination, he was average built with good general condition with pulse rate of 86/min and blood pressure of 132/88 mmHg. Except for dependent pitting edema, rest of the systemic examination was unexceptional.

His reports were as follows- Haemoglobin of 10.1g/dl and WBC count of 5600 and DLC was normal. His General blood picture showed Normocytic, normochromic anaemia. Lipid profile showed serum triglyceride level of 211mg/dl and cholesterol level of 310 mg/dl. Kidney function test showed serum creatinine 0.8 and blood urea 22mg/dl. In liver function test, bilirubin (T) was 0.19mg/dl, ALT of 56mg/dl, AST of 39mg/dl, ALP of 139mg/dl, total protein of 3.9mg/dl and albumin was 1.9mg/dl. Urine report showed proteinuria in the range of 3+ and 10-12 red blood cells. His 24hr urinary protein was 6gm. Ultrasound (whole abdomen) was normal with normal size of kidneys and maintained corticomedullary differentiation.

He was also found out to be Hepatitis C positive with viral load of 3,246,800 copies and genotype '3'. No signs of Portal hypertension was present and upper GI endoscopy was normal as well as the liver biopsy.

His immunologic tests were also done which included ANCA, dsDNA, ANA, and cryoglobulins which were all negative. C3, C4 and CH50 levels were normal.

Finally, renal biopsy of the patient was done with immunofluorescence staining. The report showed Mesangioproliferative glomerulonephritis with IgA deposits which was suggestive of IgAN (Berger's disease)

After all the workup was completed, he was started on oral Daclatasvir 60mg/day and Sofosbuvir 400mg/day for 12 weeks for hepatitis C treatment. His reports were reassessed at 6 weeks and then at 12 weeks at completion of treatment. The reports showed remarkable improvement i.e. 24hr urinary protein was reduced to 222mg/day and later to 32mg/day at the completion of treatment. There was no adverse event documented during the entire period of treatment.

### III. Discussion

HCV has been associated with multiple extrahepatic manifestations, including cryoglobulinemia, glomerulonephritis, skin disorders (porphyria cutanea tarda and lichen planus), arthritis, a sicca-like syndrome, Mooren's corneal ulcer, and lymphoproliferative disorders. It has been shown that in humans, the principal renal manifestation of chronic hepatitis C infection is development of membranoproliferative glomerulonephritis (MPGN), most often associated with cryoglobulinemia.<sup>[6]</sup>

Indeed, it is now regarded that HCV infection is the pathogenetic basis for the great majority of cases that were previously thought to be essential mixed cryoglobulinemia.<sup>[7,8]</sup> The renal manifestations are usually associated with long-standing (i.e., a greater than 10-year history) HCV infection. HCV glomerular disease has been most frequently identified in men; this disease process is rare in children.

Meanwhile, IgAN is the most common pattern of glomerulonephritis identified in all parts of the world where renal biopsy is widely practiced. The initiating event in the pathogenesis of IgAN is the mesangial deposition of IgA, which is predominantly polymeric IgA of the IgA1 subclass (pIgA1).

However, nephrotic-range proteinuria is uncommon, occurring in only 5% of patients with IgAN, and is more commonly seen in children and adolescents.

Thus, IgAN with hepatitis C is rare. Immune complex deposition as an extrahepatic manifestation of hepatitis C is well-known.<sup>[9,10]</sup> However, in these instances, MPGN has been the most common type. There have been isolated case reports of hepatitis C with IgAN.<sup>[11]</sup>

### References

- [1]. Johnson R, Crotch D, Yamabe H, Hart J, Uaccli C, Hastwell P, Couser W, Corey L, Wener M, Aljeis C, Willson R: Membranoproliferative glomerulonephritis associated with hepatitis C virus infection. *N Engl J Med* 1993;328:465-470.
- [2]. Rollino C, Roeatello D, Oiaehino O, Basolo B, Piccoli O: Hepatitis C virus infection and membranous glomerulonephritis. *Nephron* 1991;59: 319-320.
- [3]. Gonzalo A, Bãrcena R, Mampaso F, Zea A, Ortuno J: Membranoproliferative glomerulonephritis and hepatitis C virus infection. *Nephron* 1993;63:475-476.
- [4]. Doulelepont JM, Adler M, Willems M, Durez P, Yap SH: Hepatitis C infection and membranoproliferative glomerulonephritis. *Lancet* 1993;341:317.
- [5]. Pasquariello A, Ferri C, Moriconi L, C'ivita LL, Longombardo G, Lombardini F, Greco F, Zignego AL: Cryoglobulinemic membranoproliferative glomerulonephritis associated with hepatitis C virus. *Am J Nephrol* 1993;13:300-304.
- [6]. Kamar N, Izopet J, Alric L, Guilbeaud-Frugier C, Rostaing L: Hepatitis C virus-related kidney disease: An overview. *Clin Nephrol* 2008;69:149-60.
- [7]. Stehman-Breen C, Willson R, Alpers CE, Gretch D, Johnson RJ: Hepatitis C virus-associated glomerulonephritis. *Curr Opin Nephrol Hypertens* 1995;4:287-94.
- [8]. D'Amico G, Fornasieri A: Cryoglobulinemic glomerulonephritis: A membranoproliferative glomerulonephritis induced by hepatitis C virus. *Am J Kidney Dis* 1995; 25:361-9.
- [9]. Komatsuda A, Imai H, Wakui H, Hamai K, Ohtani H, Kodama T, *et al.* Clinicopathological analysis and therapy in hepatitis C virus-associated nephropathy. *Intern Med* 1996;35:529-33.
- [10]. Enr'iquez R, Rodr'iguez JC, Sirvent AE, Amor'os F, Ardoy F, Alvarez M, *et al.* Prevalence of hepatitis B and C markers in glomerular diseases. *Scand J Urol Nephrol* 2007;41:535-8.
- [11]. Gonzalo A, Navarro J, Bãrcena R, Quereda C, Ortuno J: IgA nephropathy associated with hepatitis C virus infection. *Nephron* 1995;69:354.