GAVIN PUBLISHERS

Journal of Urology and Renal Diseases

Tang W, et al. J Urol Ren Dis: JURD-166.

DOI: 10.29011/2575-7903. 000066

Case Report

# Hepatitis B Virus-Related Membranous Nephropathy Superimposed on Crescentic Nephritis Induced by ANCA Associated Vasculitis

**Song Wang[1], Hai-Jing Liu[2], Wen Tang[1*]**

[1]Division of Nephrology, Peking University Third Hospital, Beijing, China

[2]Department of Pathology, Peking University School of Basic Medical Sciences, Beijing, China

[*]**Corresponding author:** Wen Tang, Department of Nephrology, Peking University Third Hospital, 49 North Garden Rd, Haidian District, Beijing 100191, P.R.China. Tel: +861082265628; Fax: +861082265628; Email: tanggwen@126.com

**Citation:** Wang S, Liu H-J, Tang W (2017) Hepatitis B Virus-Related Membranous Nephropathy Superimposed on Crescentic Nephritis Induced by ANCA Associated Vasculitis. J Urol Ren Dis: JURD-166. DOI: 10.29011/2575-7903. 000066

**Received Date:** 12 November, 2017; **Accepted Date:** 04 December, 2017; **Published Date:** 11 December, 2017

## Abstract

We reported a case of acute renal failure and the patient was diagnosed to have ANCA associated vasculitis during admission. Renal pathological funding showed that the patients had hepatitis B virus-related membranous nephropathy superimposed on crescentic nephritis induced by ANCA associated vasculitis. Methylprednisolone pulse treatment and cyclophosphamide were given to the patient. The patient showed a decreased serum creatinine and remission of proteinuria without signs of recurrent of hepatitis B.

## Introduction

Some recent research has been focusing on the association between membranous nephropathy and crescentic nephritis [1]. According to this study, membranous nephropathy could be connected with crescentic nephritis by anti-GBM antibody, with anti-GBM antibody appeared before or after membranous nephropathy or detected at the same time [1]. Interestingly, some cases with membranous nephropathy and vasculitis glomerulonephritis have also been reported [2]. Membranous nephropathy is the most common form of HBV-related nephropathy [3]. To our knowledge, only two cases have been reported that HBV-related nephropathy transformed to crescentic nephritis [4,5]. In the present case report, we presented a case with HBV-related membranous nephropathy concurrent with crescentic nephritis induced by ANCA associated vasculitis.

## Case Report

A 64-year-old man was admitted to hospital because of malasia, and edema of lower extremities with elevated serum creatinine level of 255μmol/L(eGFR= 22ml/min/1.73m$^2$). The patient had been well until 1 month earlier, when edema of the lower extremities developed. Patient has a history of smoking and chronic pulmonary disease was diagnosis when admitted. The serum creatinine level was normal three months before admitting. There was no history of preceding infection especially hepatitis. His blood pressure was 150/90mmHg. Physical examination revealed sighs of pulmonary emphysema and pitting edema in both legs. Urinalysis showed 2+ protein and urinary sediment showed the 25-30 red cell and 10-15 blood cell per high power field. A complete blood count showed mild anemia with hemoglobin level of 100g/L. Serum albumin was 32g/L. The 24-hour urinary protein excretion was 5.3g. Serologic evaluation revealed HBsAb and HBcAb positive but both the HBsAg and HBeAg were negative. HBV DNA was lower than $10^3$ copies. Antibody to hepatitis C was negative. AST and ALT were normal. Perinuclear ANCA were positive with anti-MPO titer of 178.3 RU/ml and anti-PR3 was negative. Anti-GBM, anti-nuclear antibody, anti-DNA antibody were negative. ESR was 77mm/hr and CRP was 1.31g/L.

A percutaneous renal biopsy was performed which revealed atypical membranous nephropathy with HBsAg and HBcAg deposit and crescentic nephritis. In light microscopy, renal biopsy tissue contains 17 glomeruli, 1 of which are ischemic sclerosed. 2 cellular crescents and 9 fibrotic cellular crescents are observed. The glomerular basement membranes are diffusely thickened. The mesangium shows eosinophilic deposit material in mesangial area with mild and diffusely mesangial expansion. Endothelial proliferation is detected. In immunofluorescence, 5 glomeruli, IgG (++), IgA (+), IgM (+), C3 (+++), FRA (-), C1q (-) HBSAg (+),

HBcAg (+) deposit in mesangial area and capillary loop. In electron microscopy, the glomerular basement membranes are thickened due to subepithelial and encircled subepithelial electron dense deposits. The overlying foot processes are diffusely effaced.

Given that of perinuclear ANCA was positive and anti-MPO titer was high, the pathological diagnosis of HBV-related membranous nephropathy with crescent nephritis induced by ANCA associate vasculitis was made. In view of the crescentic nephritis and the acute deterioration of the renal function, intravenous pulse methylprednisolone (500 mg/day for three days) was given to the patient. The patient response well to this therapy with serum creatinine significantly declined. Another two rounds of pulse methylprednisolone (500 mg/day for three days ) (seven days between the first and second round and three days between the second and third round of methylprednisolone) were given and oral methylprednisone 48mg daily were initiated consequently. Intravenous cyclophosphamide 800mg per month was given. After seven months of treatment, the serum creatinine of patient decreased from maximum 372μmol/L (eGFR=14 ml/min/1.73m$^{2)}$ to 189μmol/L (eGFR=32 ml/min/1.73m$^{2)}$ and remained stable. 24 hours urinary total protein was 1.4 to 2.5g and serum albumin was 35.1g/L. No sigh of recurrence of Hepatitis B were detected with the liver function remained stable and HBV DNA was lower than $10^3$ copies. ANCA was negative.

## Discussion

Crescents are rare in primary membranous nephropathy. Recent study pointed out that in some patients with membranous nephropathy and crescents, the crescentic lesion may be due to anti-glomerular basement membrane antibodies or antineutrophil cytoplasmic antibodies-related pauci-immune glomerulonephritis [1,6]. On the other hand, membranous nephropathy is the most common form of HBV-related membranous nephropathy [3]. Two cases with HBV-related membranous nephropathy have been reported to transform to crescentic nephritis [4,5]. However, in those cases, ANCA were negative. To our knowledge, our case is the first one that was reported to have HBV-related membranous nephropathy as well as crescentic nephritis induced by ANCA associate vasculitis.

Almost all cases of hepatitis B-associated nephropathy are of membranous nephropathy and a few patients with mesangial proliferative glomerulonephritis have also been described [7]. Clinically, most patients with HBV-related nephropathy present with proteinuria or the nephrotic syndrome and the majority of patients have normal renal function at time of presentation. In our case, the patients presented with nephritic syndrome but rapid deteriorate of renal function with the pathologic funding showed no sign of mesangial proliferative glomerulonephritis but crescentic nephritis. After immunosuppressant treatment, the patients renal function was stable, and proteinuria were remission, which indicated that the AAV were the predominant culprit of renal damage of this patient. However, in view of the deposition the HBV antigen in the kidney, HBV-DNA and hepatic enzymes were monitored. The patient's liver function remained stable during the follow-up. In conclusion, to our knowledge, this is the first case that has been reported to have Hepatitis B virus-related membranous nephropathy superimposed on crescentic nephritis induced by ANCA associated vasculitis. Our finding extends of the spectrum of coexist forms of HBV induced membranous nephropathy and crescentic nephritis.

**This study was supported by**

Grant from Chinese Society of Nephrology (No. 1405046058), Fund of Peking University Third Hospital (76496-03) and Fund from Peking University (BMU20160584)

## References

1. Basford AW, Lewis J, Dwyer JP, Fogo AB (2011) Membranous nephropathy with crescents. J Am Soc Nephrol 22: 1804-1808.
2. Tse WY, Howie AJ, Adu D, Savage CO, Richards NT, et al. (1997) Association of vasculitic glomerulonephritis with membranous nephropathy: a report of 10 cases. Nephrol Dial Transplant 12: 1017-1027.
3. Kleinknecht C, Levy M, Peix A, Broyer M, Courtecuisse V (1979) Membranous glomerulonephritis and hepatitis B surface antigen in children. The Journal of pediatrics 95: 946-952.
4. Li PK, Lai FM, Ho SS, Wong KC, Lui SF, Lai KN (1992) Acute renal failure in hepatitis B virus-related membranous nephropathy with mesangiocapillary transition and crescentic transformation. Am J Kidney Dis 19: 76-80.
5. Taskapan H, Oymak O, Dogukan A, Ozbakir O, Utas C (2000) Transformation of hepatitis B virus-related membranous glomerulonephritis to crescentic form. Clinical nephrology 54: 161-163.
6. James SH, Lien YH, Ruffenach SJ, Wilcox GE (1995) Acute renal failure in membranous glomerulonephropathy: a result of superimposed crescentic glomerulonephritis. J Am Soc Nephrol 6: 1541-1546.
7. Venkataseshan VS, Lieberman K, Kim DU, Thung SN, Dikman S, et al. (1990) Hepatitis-B-associated glomerulonephritis: pathology, pathogenesis, and clinical course. Medicine 69: 200-216.