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Case report

Hepatitis B virus-related post-infectious glomerulonephritis: a case report

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Abstract

Hepatitis B virus infection is an uncommon cause of acute glomerulonephritis. We present a case of acute glomerulonephritis revealing a chronic viral hepatitis B. A 45 year-old man was admitted in the nephrology department of Hassan II university hospital (Fez, Morocco) for nephritic syndrome with advanced acute kidney injury. The investigations have revealed viral hepatitis B with a positive HBV-DNA and Others viral serology tests were negative. Renal biopsy showed a diffuse and global endocapillary proliferation without extra-capillary proliferation; and global deposits of C3 and Ig G On immunofluorescence. There wasn't any other infectious cause. We thus retained the diagnosis of hepatitis B virus-associated acute glomerulonephritis. He was given entecavir and corticosteroids. Three months later, the evolution was marked by the normalization of renal function, negativity of proteinuria and HBV DNA became undetectable. There was no relapse of glomerulonephritis and HBV viral load was still negative after one year follow-up.

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Introduction

Hepatitis B virus (HBV) infection is an important health problem especially in developing countries. HBV can be complicated by acute glomerulonephritis , this association remains controversial. The optimal therapy is undefined, although in several studies, the antiviral drugs and the immunosuppressive therapy have been tried for those patients [1]. We report the case of a patient admitted in nephrology with acute glomerulonephritis revealing a chronic viral hepatitis B.

Patient and observation

A 45 years old male, he had uncomplicated type 2 diabetes for at least 7 years treated by insulin. He was admitted in June 2011 in the nephrology department of Hassan II university hospital (Fez, Morocco) for nephritic syndrome with advanced acute kidney injury.

The clinical exam has showed a conscious patient , hypertensive at 220/120 mm Hg, the heart rate was at135b/mn, the FR=25c/mn. He was oliguric with lower limbs edema and crackles on pulmonary auscultation. The dipstick test was positive for proteinuria (+) and microscopic hematuria (+ +). The serum creatinine was more than 1000 mmol/l, urea at 38mmol/l, without electrolytes disturbances. Proteinuria was at 2g per day. He has an anemia at 10g/dl without thrombocytopenia or leucopenia. Immunological tests (ANCA and anti-MBG) were negative and the complement C3 was consumed. Viral hepatitis B serology was positive (HBs+ Ag, HBe+Ac, HBe- Ag and HBc+ Ac (IgG+IgM). HBV-DNA was positive by PCR with a viral load of 284ui/ml. liver cytolysis was absent. Others viral serology tests (HVC, HVD, HIV) were negative. The abdominal ultrasound was normal and the fibroscan wasn't showed liver fibrosis. According to international recommendations [2,3]. The diagnostic of HBV inactive carrier has been retained.

Renal biopsy was performed to identify the causes of this nephritic syndrome, which showed a diffuse and global endocapillary proliferation without extra-capillary proliferation or endo-extra membranous deposits. There were global glomeruli deposits of C3 and Ig G on immunofluorescence. In conclusion, the post-infectious glomerulonephritis diagnosis related to Hepatitis B virus was retained because of the presence of HBV replication and the absence of others infectious causes.

The patient has received a strong antiviral treatment by Entecavir 0.5 mg per week adapted to renal function. 15 days later, he has received methylprednisolone bolus 10mg/kg/day (three consecutive days) relayed by oral prednisone 0.5mg/kg/day under cover of antiviral treatment. In parallel, the patient required hemodialysis for advanced acute kidney injury.

One month later, evolution was marked by the improvement of renal function and negativity of viral load. The patient was weaned off dialysis sessions, and we have increased gradually the dosage of entecavir. Renal function became normal after 3 months. Corticotherapy was stopped at six months while antiviral therapy was continued. There was no relapse of glomerulonephritis and HBV viral load was still negative after one year follow-up.

Discussion

The most common type of GN related to HBV are membranous glomerulonephritis, membranoproliferative glomerulonephritis, mesangial proliferative GN and minimal change disease [4]. The post infectious GN remains a rarely association to HBV. The first association was described by Combes et al in a patient with acute hepatitis B infection complicated by membranous nephropathy (MGN) .

The pathogenesis of HBV-associated GN is not clear .The immune-complex basis is the most likely mechanism of this disease, according to HBsAg and HbeAg deposits in the glomerular [3] and the consommation of complement in HBV-GN.

In most reports, diagnosis of HBV-GN has been based on persistence of circulating HBV or HBV-DNA, absence of other causative agents, and presence of HBV specific antigen or viral genome in the glomerulus [5]. In this case, despite of absence HBV specific antigen in the glomerulus, the diagnostic of post-infetious GN related to HVB was retained on the presence of a positive viral load and the absence of other infectious causes. The relatively fast therapeutic response after one month from the start of treatment (antiviral treatment and corticosteroids) was also an other endpoint. The treatment of HBV-GN is not well defined, various therapeutic strategies have been tried. HBV-GN is treated by antiviral drugs associated to corticosteroids or even immunosuppressive [6, 7]. The antiviral therapy like lamivudine and entecavir allows the clearance of HBV and improve renal disease [8]. The efficacy of INF on HBV-GN has not been confirmed [9].

Zheng et al have showed, in meta-analysis of 12 clinical trials, that the association of antivirals with immunosuppressants agents is an effective and safe therapy for HBV-GN cases (83% of remission) [10]. We have opted for a powerful antiviral treatment given the severity of acute kidney injury and the risk of hepatitis B relapse under corticosteroids. So, one year later, there was no relapse of glomerulonephritis and HBV viral load was still negative.

Conclusion

HBV- post infectious glomerulonephretis is an uncommon association. We reported the case of post infectious GN related to HBV infection retained on the presence of a positive viral load and the absence of other infectious cause. The treatment with anti-viral agents and corticotherapy was effective to promote the disease remission.

Competing interests

The authors declare no competing interests.

Authors' contributions

Arrayhani mohamed is major contributors to writing the manuscript and to the conception and design as well as drafting the article for publication Fatima Zahra Batta and Souad Dahri were doctors responsible for this patient El youbi randa: revising the article critically for important intellectual content. Sqalli Tarik Houssaini : contributed also to design as well as final approval of the revision. Nourdin Aqodad: gastroenterologist who monitors with us this case. All the authors have read and approved the final version of the manuscript.

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