IJNU Case Report

Int J Nephrol Urol, 2009; 1(1): 69 - 71

Treatment of Hepatitis B Virus-Associated Acute Glomerulonephritis with Lamivudine: Case Report

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Abstract

Lamivudine has been successfully used in the treatment of acute hepatitis B virus (HBV) infection and HBV-associated polyarteritis nodosa. There are few such reports in patients with HBV-related nephropathy. We report our experience with a patient with acute glomerulonephritis and HBV infection. Lamivudine therapy resulted in the complete resolution of renal disease and subsequently the remission of viral infection.

Keywords: Glomerulonephritis, Chronic Hepatitis B, Lamivudine

Introduction

Glomerulonephritis (GN) is an infrequent but well-described complication of chronic hepatitis B virus (HBV) infection (1). The exact pathogenetic mechanisms by which certain individuals with chronic HBV infection develop GN are unknown (2). However, several in vitro investigations have supported a role for hepatitis B surface antigen (HBsAg) (2).

Lamivudine has been shown to be a potent inhibitor of HBV replication (3, 4). Little has been reported about the efficacy of lamivudine in the treatment of chronic hepatitis B in the setting of renal involvement (5). In children with HBV-associated membranous nephropathy, lamivudine has also been reported to induce remission of nephrotic syndrome in two case reports (6, 7).

Herein, we report our experience with a patient who had chronic hepatitis B with superimposed GN.

Case report

A 68-year-old man was referred to Valiasr hospital for acute renal failure (raised serum creatinine). The patient had abdominal pain for five days, which was mild, vague and non-radiating. The pain was not aggravated by deep breaths and movement and did not relate to the fat food. There were nausea, vomiting, anorexia and malaise. The past medical history was unremarkable. At physical exam, blood pressure was 140/80 mmHg, pulse rate 74/min, respiratory rate 17/min and body temperature 37 °C. Laboratory examination revealed a hemoglobin of 9.3 g/dl, white blood cell count of 7000/mm^3, platelet count of 246000/mm^3. Initial creatinine was 13.9 mg/dl (which then raised to 25 mg/dl), blood urea nitrogen 144 mg/dl, potassium 5.6 mEq/l, albumin 3.1 gr/dl, aspartate aminotransferase 17 U/L, alanine aminotransferase 18 U/L, alkaline phosphatase 315, cholesterol 205 mg/dl.

Prothrombin and partial thromboplastin times

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Received: 12 Mar 2009
Revised: 28 Mar 2009
Accepted: 20 Apr 2009

were 13.4 and 32, respectively. The urinalysis showed a mild proteinuria and hematuria. Examination of the urine sediment under polarized microscope revealed many red blood cells (RBCs with up to 80% dysmorphic figures) and 3-4 RBC cast per high power field (hpf). Daily protein excretion was 50 mg. On the ultrasonography at longitudinal view, both kidneys were swollen with diffusely enlarged echogenic cortex. Kidneys' lengths were 11.5 and 10.5 cm.

Subsequently, following tests were performed to exclude secondary causes of GN: antinuclear, anti-dsDNA, anti-glomerular basement membrane, cytoplasmic-antineutrophil cytoplasmic, and perinuclear- antineutrophil cytoplasmic antibodies, VDRL, cryoglobulin, as well as anti-hepatitis C and anti-HIV antibodies, all of which were negative. Tuberculin skin test was unrevealing. Urine and serum electropheresis was normal. C3 and C4 complements and CH50 were also within their normal limits.

However, HBsAg and anti-HBc IgG antibody were positive. HBeAg was negative. Subsequently, HBV DNA was detected in serum with polymerase chain reaction (PCR). To exclude diagnosis of polyarteritis nodosa, magnetic resonance angiography of the renal and mesenteric arteries was performed that was unrevealing. A diagnosis of HBV-associated acute GN was made and adjusted-dose of 100 mg/day lamivudine was initiated.

Following one week of oral lamivudine administration, renal function dramatically improved. The serum creatinine level decreased from 25 mg/dl to 1.6 mg/dl and RBC cast and dysmorphic RBCs disappeared from the urine sediment. The patient was discharged in a favorable clinical condition and was recommended to continue lamivudine for the rest of his life. On a follow-up one month later, the patient was doing well, and HBV DNA was not detected on quantitative PCR.

Discussion

Lamivudine has been shown in a randomized trial to be effective in the treatment of chronic HBV infection (8). It has also been used in the treatment of acute HBV infection (9). Treatment with lamivudine was also found successful in adults with HBV-associated polyarteritis nodosa (10),

and in combination with other antiviral agents in HIV-associated nephritic syndrome (11). However, in HBV-associated nephropathy, the duration of lamivudine therapy is still controversial. Notably one study showed that this type of nephropathy may progress even after anti-HBe seroconversion (12). Indeed, Tang et al. reported a relapse of nephrosis following two years of complete remission when lamivudine treatment was withdrawn (3). Hence, it seems reasonable to continue lamivudine for as long as possible after initial remission. Lamivudine is considered safe and effective for HBsAg positive patients. Interestingly, in a HBV-related cirrhotic patient with membranoproliferative GN, Wen and Chen showed that 3-month lamivudine therapy resulted in resolution of renal disease (13).

We have shown that lamivudine therapy was successful in a patient with HBV-associated acute GN and resulted in a complete resolution of condition in one week. Based on the previous reports, we suggest that lamivudine therapy should be continued in such patients even after initial remission of the viral infection in order to prevent possible relapses of renal disease. However, this has to be confirmed with further studies.

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