Short Communication

Hepatitis B Reactivation in a Multiple Myeloma Patient with Resolved Hepatitis B Infection during Bortezomib Therapy: Case Report

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It has recently been reported that hepatitis B virus (HBV) reactivation in patients with hepatitis B surface antigen (HBsAg)-negative lymphoma during or after cytotoxic therapy occurs after the use of rituximab and stem cell transplantation for hematologic malignancies. However, clinical data on HBV reactivation in multiple myeloma patients have not been extensively reported. This is the first reported case of HBV reactivation in an HBsAg-negative myeloma patient treated with bortezomib (BOR) as salvage therapy and not stem cell transplantation. By closely monitoring HBV-DNA and early administration of entecavir, severe hepatitis was avoided and BOR therapy was continued. We suggest the importance of close monitoring of HBV-DNA for transplant-ineligible myeloma patients treated with BOR as salvage therapy. (J Clin Exp Hematopathol 52(1): 67-69, 2012)

Keywords: hepatitis B virus reactivation, multiple myeloma, bortezomib, entecavir

INTRODUCTION

Reactivation of hepatitis B virus (HBV) infection is a well-recognized complication in cancer patients with chronic HBV (hepatitis B surface antigen [HBsAg]-positive) undergoing cytotoxic chemotherapy, and prophylactic antiviral therapy before chemotherapy is recommended in such individuals.1,2 After the use of rituximab and stem cell transplantation for hematologic malignancies, HBV reactivation in patients with resolved HBV infection (HBsAg-negative and HBs antibody [anti-HBs]-positive and/or hepatitis B core antibody [anti-HBc]-positive) during or after cytotoxic therapy has been recently reported.1-5 Among lymphoma patients with resolved HBV infection treated with chemotherapy containing rituximab, 25% developed HBV reactivation.5,1 One-quarter of this group of 25% developed fulminant hepatic failure with an extremely high mortality rate.3 Clinical data on HBV reactivation in multiple myeloma (MM) patients have not been extensively reported. Some reports described HBV reactivation in myeloma patients with resolved HBV infection, but most studies reported HBV reactivation after autologous stem cell transplantation (ASCT).6-8

We report a case of HBV reactivation in a myeloma patient with resolved HBV infection during bortezomib (BOR) treatment as salvage therapy and successful management with close monitoring of HBV-DNA as well as early administration of entecavir (ETV).

CASE REPORT

A 72-year-old Japanese male who underwent a gastrectomy due to gastric ulcers at age 50 was diagnosed with monoclonal gammapathy of undetermined significance (MGUS) (IgG-κ, Bence-Jones protein-κ type) by a medical examination in 2005. He was regularly monitored without being given any therapy. However, MGUS transformed to symptomatic MM, and treatment (melphalan, prednisolone, and thalidomide) was started in October 2007. MM improved temporarily but became resistant to treatment, and anemia as well as renal disorders progressed. He was therefore admitted to our hospital for initiation of BOR therapy in January 2011. Physical examination revealed no abnormal findings. Imaging demonstrated no extramedullary lesions or pathologi-
Tanaka H, et al.

A patient treated with BOR as salvage therapy after ASCT, and a myeloma patient is HBsAg-negative and ineligible for ASCT. It is possible that fulminant hepatic failure might appear without close monitoring of HBV-DNA and early administration of ETV.

We administered a normal dose of BOR and DEXA to our patient. The myeloma was not refractory and a partial response was preserved. It is suggested that HBV reactivation can occur in other myeloma patients with resolved HBV infection treated with BOR as salvage therapy.

HBV reactivation in our patient developed immediately after increasing DEX. Steroid-containing regimen was shown not to be a risk factor for HBV reactivation in HBsAg-negative patients. In HBsAg-positive patients, however, steroid was most frequently associated with HBV reactivation.

It is important to check anti-HBs and anti-HBe as well as closely monitor HBV-DNA during BOR therapy even if a myeloma patient is HBsAg-negative and ineligible for ASCT. If HBV reactivation occurs, BOR therapy can be continued by closely monitoring HBV-DNA and by early administration of ETV.

DISCUSSION

BOR is a potent proteasome inhibitor that is currently used as therapy for myeloma. It is associated with a significant risk of reactivation of infection by the varicella zoster virus, and acyclovir prophylaxis is recommended. One report stated that BOR suppresses HBV proliferation in vitro. Two reports described HBV reactivation in patients receiving BOR therapy for myeloma. One study described a patient treated with BOR as salvage therapy after ASCT, and another study described an HBsAg-positive patient discontinuing lamivudine prophylaxis. Ours is the first report of HBV reactivation in a myeloma patient with resolved HBV treated with BOR as salvage therapy and not ASCT. It is possible that fulminant hepatic failure might appear without close monitoring of HBV-DNA and early administration of ETV.

REFERENCES


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