Case Report

Pleuro-Pulmonary Nocardiosis as Opportunistic Infection in a Patient with Chronic Hepatitis C under Combination Treatment with Pegylated Interferon, Ribavirin, and Boceprevir

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Nocardiosis is an infrequent but serious pulmonary infection caused by Gram-positive aerobic actinomycetes. In this paper, we report on a 48-year-old patient with pleuropulmonary nocardiosis and cirrhosis due to chronic hepatitis C virus infection treated with triple antiviral treatment complicated by prolonged neutropenia.

1. Introduction

Pleuropulmonary nocardiosis is an uncommon infection and may cause radiologic findings that vary from vague pulmonary infiltrates to cavitary lesions. It is found more commonly in patients with underlying lung disease and in immunocompromised patients. We describe the first case of this atypical bacterial infection in a patient with hepatitis C virus related liver cirrhosis undergoing antiviral therapy with pegylated interferon alpha, ribavirin, and boceprevir (Merck Sharp & Dome Ltd, Hertfordshire, United Kingdom) complicated by prolonged neutropenia.

2. Case Report

A 48-year-old Caucasian male was admitted to our inpatient liver clinic in October 2012. He was in poor general condition with ascites, fever (39.4°C), and a dry cough.

In 1997, the patient had been diagnosed with chronic hepatitis C (CHC) subtype 1b which was unresponsive to a 3-month course of interferon (IFN)-alpha monotherapy. In June 2007, he had been admitted to the internal medicine department because of portal decompensation following a period of higher alcohol consumption. CT scan at that time revealed, besides signs of cirrhosis, a right-sided nodular pulmonary lesion measuring $3 \times 2 \times 2$ cm, which was interpreted as posttuberculosis infiltration without structural changes of the bronchial architecture. Following abstinence from alcohol, liver function had stabilized from August 2008 onwards. Antiviral combination treatment with pegylated interferon (pegIFN) alpha-2a 180 μg given weekly and ribavirin (RBV) (600 mg/12 h) had been started in September 2009. Due to the rapid virological response with no detectable hepatitis C virus (HCV) RNA determined with the COBAS AmpliPrep/COBAS TaqMan HCV Test (lower limit of detection 15 IU/mL) at week 4 of treatment, the antiviral therapy had been shortened to 24 weeks. Three months after the end
In June 2012, a 4-week lead-in phase with pegIFN alpha-2a in combination with boceprevir was started. At week 8, HCV RNA was 67 IU/mL (limit of detection < 15 IU/mL) corresponding to a virological relapse.

**3. Discussion**

HCV is a leading cause of chronic liver disease and cirrhosis [1, 2]. Patients suffering from chronic hepatitis C (CHC) are usually immunocompetent but not able to eliminate HCV. Therapy of CHC has recently been improved through addition of direct-acting antiviral drugs such as boceprevir and telaprevir to the standard therapy consisting of pegIFN and RBV. Boceprevir is a NS3/4A protease inhibitor. The duration of boceprevir treatment depends on the virological response 8 weeks after commencing treatment [3, 4].

This report describes an atypical case of nocardiosis without structural modifications of the bronchial architecture in a patient with CHC and liver cirrhosis under antiviral combination therapy with pegIFN, RBV, and boceprevir. *Nocardia*, a Gram-positive bacterium, is a member of the order Actinomycetales. Nocardiosis represents a rare disease mainly affecting individuals with cellular immunodeficiency. This opportunistic pathogen induces disseminated infections with pulmonary, abdominal, bone marrow, and skin manifestations [5–9]. Patients with nocardiosis may present symptoms indistinguishable from those in patients with pulmonary infections of other etiologies. Furthermore, nocardiosis may be difficult to diagnose because of difficulties of identification in culture material. Due to the poor prognosis with a mortality of 15% in cases with delayed diagnosis, an early start of effective therapy is urgently required. Nocardia infections were recently observed in CHC liver transplant patients under immunosuppressive regimens [8, 9]. Therefore, it is tempting to speculate that in this case antiviral treatment-induced neutropenia may have favored the manifestation of Nocardia infection. In this context, it should be pointed out that in the RESPOND-2 clinical trial the addition of boceprevir to pegIFN/RBV increased the incidence of grade 3 neutropenia significantly (19 and 20% in boceprevir arms versus 9% with standard care) [3].

This paper is the first report on pleuro-pulmonary nocardiosis in a patient with HCV-related cirrhosis under triple antiviral combination therapy.
antiviral treatment including boceprevir complicated by prolonged neutropenia. In conclusion, opportunistic pathogens including Nocardia spp. should be considered for the differential diagnosis of pleuro-pulmonary infections of patients with prolonged neutropenia due to severe CHC treatment.

**Conflict of Interests**

The authors declare that there is no conflict of interests.

**References**


