Tuberculous peritonitis after treatment for chronic hepatitis C

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A 71-year-old man was admitted to our Department with weakness, low-grade fever, and weight loss (approximately 10 kg) during the last 3 months. The patient had received antiviral therapy for chronic hepatitis C in another clinic (pegylated interferon-2a 180 mcg/week plus ribavirin 1000 mg/day) for 4 months, but this regimen had been discontinued 2 months before his admission due to the development of severe leukopenia (neutrophil count 450/mm³). On admission, the patient was febrile (37.5°C), with abdominal distension and lower limb edema. Admission laboratory findings included: hemotocrit: 36.4%, white blood cells: 3.1x10⁹/µL (neutrophils: 61%, lymphocytes: 21%), platelet count: 110,000/µL, albumin: 3.0 g/dL, alanine aminotransferase: 104 IU/L [upper limit of normal (ULN)<40], alkaline phosphatase: 270 IU/L (ULN<120), γ-glutamyl transferase: 386 IU/L (ULN<38), bilirubin: 4.6 mg/dL (direct: 2.95 mg/dL), INR: 1.2, and C-reactive protein (CRP): 12 mg/L. Abdominal computed tomography showed cirrhotic liver with splenomegaly and ascites without other abnormal findings, and cardiac ultrasound was normal. Analysis of ascitic fluid revealed: leukocytes: 800/mm³ (neutrophils: 500/mm³), serum-ascites albumin gradient (SAAG) >1.1 g/dL, protein: 2.6 g/dL. With the diagnosis of spontaneous bacterial peritonitis (SBP), the patient received ceftriaxone 2 g intravenously/day, but three days later, new paracentesis of ascitic fluid showed poor response to antibiotic therapy (leukocytes: 1000/mm³, neutrophils: 800/mm³). At this time, ceftriaxone was changed to imipenem 2 g/day intravenously. After 2 days, new paracentesis revealed that the number of neutrophils in ascitic fluid remained unchanged and the patient was still febrile. All cultures of ascitic fluid were negative for the common bacteria. Further evaluation of ascites was decided. Although tuberculin skin test and Ziehl-Neelsen staining of the ascitic fluid were both negative, amplified Mycobacterium tuberculosis direct test (Gen-Probe) of the fluid was intensively positive [10⁸ relative light units (RLU) (>500,000 RLU indicates active infection from Mycobacterium tuberculosis)]. Computed tomography of the chest showed no indication for pulmonary tuberculosis. With the diagnosis of peritoneal tuberculosis (TBP), the patient was commenced on isoniazid 300 mg, rifabint 600mg and ethambutol 2 gr per day for 2 months and continued with isoniazid, and rifampicin for another 4 months. He showed good response with normal ranges of CRP one month after initiation of anti-tuberculosis therapy. The patient was followed up in the clinic, and, fortunately, without adverse events during anti-tuberculosis therapy.

Patients with cirrhosis are considered at higher risk for development of TBP [1]. Analysis of ascitic fluid in TBP usually reveals lymphocytic ascites with SAAG <1.1 g/dL [2]. However, similar to our case, patients with concomitant cirrhosis may have neutrophil predominance and SAAG >1.1 g/dL in up to 40% and 71% of cases, respectively [2]. In our patient, we were able to establish the diagnosis of TBP based on Gen-Probe, which is a quick, specific, but expensive test for the activity of tuberculosis.

In the literature, few patients with pulmonary tuberculosis during or after discontinuation of antiviral therapy for hepatitis C virus (HCV) infection have been described [3]. Although tuberculosis has been associated with the development of interferon-induced neutropenia/lymphopenia, recent studies have found that other factors, such as age over 50 years and diabetes mellitus, are significantly associated with the occurrence of infections in patients under anti-HCV therapy (our patient was older than 50 years) [4,5]. To our knowledge, this is the first case report of TBP associated with antiviral therapy against HCV infection.

References


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