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Spontaneous reactivation of hepatitis B infection causing fulminant hepatic failure; induction of ciprofloxacin highly suspected of hepatotoxicity in a patient with suspected salmonellosis

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Objectives

Chronic hepatitis B virus (HBV) infection is a dynamic state of interaction between HBV, hepatocytes, and the immune system. Reactivation flares may occur due to multiple causes such as after immunosuppressive therapy, chemotherapy, during postpartum period, and rarely spontaneously. This is due to immune-mediated destruction of HBV-expressing cells following withdrawal of immunosuppressive effect. We report a case of fulminant hepatic failure in a previously unrecognized HBV-positive female while using ciprofloxacine with suspected salmonella infection.

Case

An unconscious 22-year-old woman was hospitalized with the diagnosis of hepatic encephalopathy. A week ago; symptoms of upper respiratory tract infection, and in recent days; nausea, vomiting, diarrhea complaints began, then her consciousness gradually deteriorated. She had received ciprofloxacin 1000mg daily for 5 days.
In admittion, she was no verbal response, but responsive to painful stimuli, and had a continuous fever about 39-40C0, hyperpnea, tachypnea, hypotension (90\60 mmHg), conspicuous fever-pulse discordance, mild diarrhea, icteric skin and scleras, dry skin, decreased turgor-tonus, hepatomegaly (about 217mm in long axis). In laboratory investigation, leukocyte count was 17600/µL, hemoglobin 10,3g\dL, platelet count 118000/µL, aspartate-aminotransferase (AST) 1170U\L, alanine-transaminase(ALT) 2620U\L, direct-bilirubin 12,1g\dL, indirect-bilirubin 7,1g\dL, alkaline-phosphatase (ALP) 441U\L, gamma-glutamyl transferase (GGT) 240U\L, lactate dehydrogenase (LDH) 805U\L, albumin 2.8g\L, prothrombin time (PT) 63,3sec, INR 4,9, HBsAg positive, anti-HBc IgM and IgG positive, HBeAg negative, anti-HBe positive, HBV-DNA positive, Gruber-Widal agglutination (TH 1\100, TO 1\100) positive, however blood and stool culture negative, respectively.

After her drugs were terminated and supportive care was given, she was intubated, then followed in intensive-care unit. The patient's consciousness improved a few days later, and her transaminases showed gradual decline to normal values within 2 months. Other etiologies of fulminant hepatic failure were ruled out, except acut on chronic HBV infection, suspected salmonella infection and ciprofloxacin-induced fulminant hepatic failure.

Conclusions

Spontaneous reactivation is an unusual cause of hepatic decompensation in patients with chronic HBV infection, but may lead to fulminant hepatic failure. Liver involvement is common but acute hepatitis is very rare and has an excellent prognosis in Salmonella infections. We didn’t think acute salmonella hepatitis in our cases. Because, laboratory findings were supporting acute hepatitis B, as well as, although clinical features were resembling, laboratory findings weren’t sufficient for the diagnosis of salmonellosis and response to ciprofloxacin was inadequate. HBeAg-negativity and anti-HBe-positivity were also suggestive for acute on chronic hepatitis B. But we believe that intervening salmonellosis suspicious and ciprofloxacin use may play a triggering role for hepatic decompensation in our case, based on published a few cases indicate that idiosyncratic drug reaction can be with the short term use of ciprofloxacin. In conclusion, although ciprofloxacin is generally considered a well-tolerated and safe drug, clinical practitioners should be aware of its potential hepatotoxicity.