

Case Report

Recurrent Exudative Pleural Effusion with Flare up of Chronic Hepatitis B Virus Infection.

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Abstract

We describe herein a young male patient who presented with exudative pleural effusion that appeared with flare up of chronic HBV infection and spontaneously recovered with the clinical and biochemical improvement of the hepatitis on two occasions five months apart. Other causes of hepatitis and exudative pleural effusion were excluded with appropriate investigations.

Keywords: Extrahepatic, ascites, bilirubin, hepatosplenomegaly, mantoux.

Case Report

A previously healthy 30 years old Sudanese man presented with right sided chest pain of two weeks duration. He described intermittent sharp chest pain over the right side of the chest and shortness of breath after minimal exertion. He denied having had fever, arthralgia or skin rash and had not experienced any nocturnal dyspnea, wheezing, cough, expectoration or haemoptysis and had no weight loss. He had taken paracetamol, but his symptoms continued. He gave history of jaundice ten years earlier.

On physical examination, he appeared weak and ill, moderately jaundiced. His temperature, pulse and blood pressure were normal, respiratory rate was 22 breaths per minute. Chest examination showed right sided pleural effusion while other systems were normal. Investigations revealed normal Hb, WBCs, blood sugar, RFT and electrolytes. His ESR was 95mm/hr. LFT showed: serum bilirubin 4.0, total serum protein 8.5, serum albumin 3.2, AST 89 (10-50), ALT 88 (10-50), ALP 93 (40-120). HBsAg +ve, HBcIgG +ve, HBc IgM-ve, HBeAg-ve, HBeAb-ve, HCV Abs -ve, HIV -ve.

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Abdominal ultrasonography: mild hepatosplenomegaly, no ascites. CXR: right sided pleural effusion (Figure1).



Fig1: Right side pleural effusion

Sputum for AAFB was negative three times. Mantoux test-ve. One liter of amber-colored pleural fluid was aspirated and the results of the analysis came as following: protein 6.8 gm/dl, sugar 108 mg/dl, WBCs count 260 mainly lymphocytes with no malignant cells. PCR for MTB in the blood and pleural fluid was - ve. The patient was planned for pleural biopsy but his jaundice and the pleural effusion rapidly and spontaneously started regressing and within three weeks of hospital stay the effusion regressed significantly (Figure2) and liver enzymes returned to normal levels (bil 0.8, protein 6.8, alb 3.9, AST 43, ALT 43, Alp 154).



Fig2: Significant regression of the effusion



Fig4: Complete clearance of the chest.

The patient was discharge home in a good condition without receiving any medication. Five months later the patient came back with jaundice and left side pleural effusion which was confirmed by chest-x-ray (figure3).



Fig3: Left side pleural effusion

Hb 14.1gm, TWBc 5.1, ESR 95mm/hr Pleural fluid was again exudative and negative for LE cells, Rh factor, malignant cells and AAFB. Echo was normal. LFTs: Total serum bilirubin 9.0, direct bilirubin 8, serum protein 7.5, serum albumin 4, AST 160 (10-50), ALT 195 (10-50), ALP 130 (40-120), INR 1.8, HBsAg and HBc IgG remained +ve. Patient again recovered rapidly and within six weeks his liver enzymes returned to near normal AST 55 (10-50), ALT60 (10-50), ALP 110 (40-120), and his pleural effusion resolved completely (figure4).

The markers for autoimmune hepatitis were negative on the two occasions and there were no clinical or biochemical evidence of any other concomitant disease.

The patient who is still under our follow up had no more flare up of his hepatitis and showed no clinical deterioration and still has no evidence of any other disease to explain his pleural effusion.

Discussion:

HBV is a multisystem disease. Although its main victim is the liver; other organs can be affected directly or indirectly¹⁻⁴. Nevertheless, the lungs were rarely affected. The presence of pleural effusion with HBV in the absence of ascites is very rare. Concomitant pleural effusion with acute HBV infection was 1st reported in 1971⁵. Since then less than 10 cases were reported^{6, 7}. Spontaneous clearance of exudative pleural effusions with seroconversion of HBsAg was reported before⁸. To the best of our knowledge only one case with recurrent isolated pleural effusions with exacerbations of the infection was reported in the English literature⁹. Similar to the case reported by Lee et al⁹, the effusion in our patient was exudative, recurrent and occurred with the acute exacerbation of the infection and disappeared with the remission of the infection, however, this case is peculiar in that the effusion recurred on the other side of the pleural cavity while the 1st side was completely clear.

The exact pathogenesis of concomitant exudative pleural effusion with exacerbation of chronic HBV infection remains a dilemma. Extrahepatic immune mediated manifestations of HBV infection including polyarteritis nodosa, glomerulonephritis and others are well known^{10, 11}. We think this presentation is a rare part of the clinical picture of HBV infection. It is very likely that the effusion is an immune reaction to the HBV infection itself as other causes of exudative pleural effusion were excluded and the spontaneous disappearance of the effusion with the clinical and biochemical remission of the hepatitis supports that.

Being an immunological reaction was suggested before and was supported with low complement levels in the effusion⁹. Nevertheless, because of the rarity of these cases the dilemma may persist for some time.

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