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Case Study

HENOCH-SCHONLIEN PURPURA ASSOCIATED WITH HEPATITIS C AND COMPENSATED CIRRHOSIS

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ABSTRACT

An unordinary case report of henoch-schonlein purpura (HSP) in a relationship with hepatitis C and compensated cirrhosis was identified in 14 y aged male patient. He was admitted in the pediatric department with stomach pain, yellow skin, rashes with tingling and erythematous injuries over the legs with agony and swelling since multi-day. He feels pain during walking and appears to be with swelling of lower leg muscles. His unusual liver function test was distinguished with elevated levels of bilirubin-3 mg/dl, basic phosphatase-314 U/l, aspartate aminotransferase-55 U/l and alanine aminotransferase-60 U/l. His skin biopsy shows up leukocytoclastic vasculitis and IgA depositions. Liver biopsy revealed nuclei enlargement with extensive cell change and scattered cell plates. His blood test was with the presence of the hepatitis C virus (HCV) antibodies. He was finally diagnosed as HSP associated with HCV and compensated cirrhosis.

Keywords: Henoch-schonlein purpura, Hepatitis C, Immunoglobulin A, Compensated cirrhosis, Erythematous lesions

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INTRODUCTION

HSP portrayed by generalized vasculitis [1]. HSP is characterized by non-thrombocytopenic palpable purpura, arthritis or arthralgias, gastrointestinal and renal involvement [2]. Inflammation also involves the blood vessels of the skin, lungs and focal sensory system with a mind-boggling store of immunoglobulin A (IgA) [3, 4] influencing transcendently in children [5]. HSP builds up the indications of rash, particularly finished legs, stomach pain, subcutaneous oedema, joint pain and glomerulonephritis [6]. HSP can influence any organ, physical examination observations are required. An exact reason for the cause of HSP is not known, but it is believed to be multifactorial, with hereditary, natural, and antigenic parts [7, 8]. It is difficult to distinguish by skin biopsy, which can show leukocytoclastic vasculitis and IgA depositions [9].

HCV is a major cause of liver disease and the potential cause of substantial morbidity and mortality worldwide [10]. Hepatitis C caused by HCV can be analyzed by the discovery of immunizing to HCV in the blood test by the immunoassay method [11] and identification of IgM antibodies against hepatitis C infection antigens [12]. HCV disease can do significant damage to the liver and leads to cirrhosis. It's not surprising that numerous individuals with hepatitis C don't know they have a life-threatening disease which can lead to liver cirrhosis. There are two phases of cirrhosis 1. **Compensated cirrhosis** means the body still functions despite decreased liver function and scarring. 2. **Decompensated cirrhosis** implies that body functions are separated where serious side effects like kidney failure, variceal haemorrhage, and hepatic encephalopathy may occur. Indications of cirrhosis are because of hepatitis C are fatigue, nausea, loss of appetite, weight reduction, wounding, itchy skin, jaundice, swelling in legs, ascites, hepatorenal disorder [13].

A few studies reveal hepatitis C associated IgA/IgM mixed cryoglobulinaemia can't be ruled out regardless of a negative cryoglobulin screen [14] on two occasions. In this patient, an IgA mediated vasculitis may have been the nidus for thrombus development and abdominal catastrophe. The role of liver cirrhosis in the advancement of HSP is fascinating. Patients may create HSP as because of an impact of abnormal liver metabolism of IgA circulating immune complexes that is an impaired clearance of IgA complex in liver cirrhosis resulting about tissue depositions [15, 16], despite the fact that this is known to occur without overt vasculitis [16]. This mechanism appears to be in this case. The preceding HCV might have contributed to the formation of the immune complex.

There is no particular treatment for HSP. Acetaminophen or NSAIDs can be used for pain management and some of the time corticosteroids may also be used [17].

CASE REPORT

A male patient, aged 14 y admitted in the pediatric department with chief complaints of the stomach pain, yellow skin, rashes with tingling and erythematous sores over the legs with pain and swelling since one day. He feels pain during walking and appears to be swelling of lower leg muscles. There is no history of rash after food consumption, medicine use, insect bite, and there is no cough and swelling of the neck to the patient. His birth and improvement history was observed to be normal. Physical examinations, biochemical reports, complete blood picture and urine examinations seem to be normal. Peripheral smear test and ASO tube test were negative. His liver function test was observed to be elevated levels of total bilirubin-3 mg/dl, alkaline phosphatase-314 U/l, aspartate aminotransferase-55 U/l and alanine aminotransferase-60 U/l. His skin biopsy exhibits leukocytoclastic vasculitis with IgA depositions. Liver biopsy reveals nuclei enlargement with large cell change and disorganized cell plates. His blood test was positive with the presence of antibodies to HCV. He was finally diagnosed as HSP to have hepatitis C and compensated cirrhosis.

The patient was treated with IV ceftriaxone 1.125g+tazobactam 500 mg, IV netilmicin 50 mg BD, IV ranitidine 50 mg BD, IV pheniramine 1.5 cc BD, IV dexamethasone 1cc BD, tablet paracetamol 500 mg, tablet calcium, syrup multivitamin. He was counselled with a way of lifestyle changes in diet aspects.

DISCUSSION

An uncommon case report HSP association with hepatitis C and compensated liver cirrhosis were identified in a pediatric department. A male patient aged 14 y admitted in the pediatric department with stomach pain, yellow skin, rashes with rashes and erythematous injuries over the legs with pain and swelling since one

day. He feels pain during walking and appears to be swelling of lower leg muscles.

An elevated level of bilirubin, alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase reveal liver damage to the patient. Patient's skin biopsy shows leukocytoclastic blood vessel vasculitis with IgA depositions, and liver biopsy reports with nuclei enlargement with a vast cell change of liver tissue which is considered as HSP Immunoassay method for a blood test of patient reports the presence of antibody to HCV indicates the condition of hepatitis C.

The patient might be infected with HCV long back without creating any side effects. So it turned into a serious approach towards cirrhosis. There might be a blockage of flow in the biliary tract or buildup of biliary pressure in the liver. The patient's body functions well, although after understanding cirrhosis, so it was considered as compensated liver cirrhosis.

A literature report says hepatitis C is associated with an IgA/IgM mixed cryoglobulinemia, but an IgA mediated vasculitis may have been the nidus for thrombus formation and abdominal catastrophe. The patient was developed HSP as due to the consequence of liver metabolism of IgA circulating immune complexes that is an impaired clearance of IgA complex in liver cirrhosis resulting in tissue deposition. This mechanism seems to be considered well for this patient. The preceding HCV might be formed in the patient due to the formation of immune. Nephritis is the most vital long-term prognostic factor in HSP; the transient prognostic factor is gastrointestinal illness can lead to death if there is no early treatment and care.

He was treated with acetaminophen, corticosteroids, cephalosporin anti-infective agents and antihistamines. The way of lifestyle changes improves a state of the liver.

This case is considered to a remarkable case report because HSP in the early adult stage is uncommon; however, HSP usually occurs in the children.

CONCLUSION

An unordinary case report HSP association with hepatitis C and compensated cirrhosis require a nearby observing. Early conclusion and early treatment of hepatitis C are more essential it might prompt cirrhosis. A rate of HSP with cirrhosis has been expanding around the world. This condition has a long-term prognostic factor in causing nephritis, short-term factor to cause gastrointestinal disease in which both can lead to death if untreated, so early therapeutic intervention with clinical implications are needed.

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AUTHORS CONTRIBUTIONS

Sagar Pamu identified the case report and analyzed uncommon points; Sarangi Ramesh helped in editing this article and Mohammed Abubakar contributed the sources to analyze this case report.

CONFLICT OF INTERESTS

Author has no potential conflicts of interest with respect to the authorship and publication of this article.

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