JMS 2023; Vol (6):198-202
UDC: 616.155.294:[616.36-002-02:578.891
https://doi.org/10.55302/JMS2363198s

THROMBOCYTOPENIA ASSOCIATED WITH HEPATITIS B VIRUS INFECTION- A CASE REPORT

Haris Sulejmani[1], Nenad Bogdanovski[1], Ivana Zafirova[1], Svetlana Krstevska Balkanov[2]
[1]Medical Doctor
[2] Clinic for Haematology, Faculty of Meidicne, Ss Cyril and Methodius University in Skopje, North Macedonia

Abstract

Two thirds of people with acute hepatitis B have an asymptomatic or subclinical infection that goes undetected. Progressive liver infection (counting cirrhosis and HCC) can be anticipated to develop in one quarter to one third of individuals who obtain infection within the first few years of life. While the exact mechanism underlying the correlations between HBV and various skin problems is still unknown, theories usually put up include virally related,immune-related mechanisms or direct harm brought on by viral replication. The most frequent cutaneous HBV infection symptom is acute urticaria. HBsAg positive was significantly and independently related with incidence thrombocytopenia in a large cohort research of an essentially healthy population, suggesting that mechanisms other than portal hypertension may underlie thrombocytopenia in healthy HBV carriers.

We presented a case of Hepatitis B virus infection associated with thrombocytopenia.The patient was 53 years old man who was admitted in our clinic due to a maculo-papular rash predominantly in lower extremities after consuming alcohol without elevated temperature and accompanying symptoms.Lab test was performed and there were detected elevated liver enzymes and lower count of platelets.

Key words:Hepatitis B virus,Thrombocytopenia,rash

Introduction

Today, an estimated 260 million people worldwide are chronically infected by HBV.The vast majority of these people will not have issues, but 15% to 40% will develop significant complications, such as cirrhosis or HCC, and many will die early. The virus is 50 to 100 times more contagious than HIV and 10 times more contagious than HCV.A high viral count suggests an increased risk of transmission not just from mother to child, but also following needlestick contact and in the context of family contact. Two thirds of people with acute hepatitis B have an asymptomatic or subclinical infection that goes undetected. Progressive liver infection (counting cirrhosis and HCC) can be anticipated to develop in one quarter to one third of individuals who obtain infection within the first few years of life. Before the onset of symptoms or an increase in serum aminotransferase levels, HBsAg emerges in serum 2 to 10 weeks after HBV exposure. After 4 to 6 months, HBsAg generally stops being detectable in acute self-limited hepatitis.

The development of chronic HBV infection is implied by the persistence of HBsAg for more than 6 months. Anti-HBs lasts a lifetime and gives long-term immunity in the majority of patients.Anti-HBc, which predominately belongs to the IgM class during acute infection, is often detectable for 4 to 6 months following an acute bout of hepatitis, with rare exceptions lasting up to 2 years.

Exacerbations of chronic hepatitis B may make IgM anti-HBc detectable, and it can even be utilized as a stand-in for viral replication in progress. People with chronic hepatitis B infection and those who recover from acute hepatitis B both have anti-HBc of the IgG type. [1]

While the exact mechanism underlying the correlations between HBV and various skin problems is still unknown, theories usually put up include virally related immune-related mechanisms or direct harm brought on by viral replication. The most frequent cutaneous HBV infection symptom is acute urticaria. According to earlier research, 15.6% more ladies than males (13.8%) developed urticarial rash, which affected 14.5% of HBsAg carriers. [2]

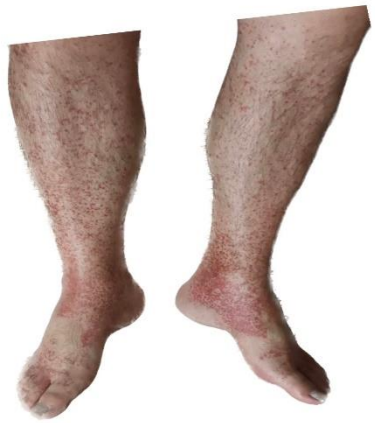
Vasculitis and essential mixed cryoglobulinemia seemed to be the most common skin lesion in chronic hepatitis B patients (53.3%), subsequently followed by papular changes, rashes, and Gianotti-

Crosti syndrome. Skin cancer and Henoch-Schönlein purpura were uncommon. The most common spontaneous skin lesion in those with chronic hepatitis B was vasculitis. After vaccination and IFN, lichen planus and lupus-like lesions were most common. [3]

HBsAg positive was significantly and independently related with incidence thrombocytopenia in a large cohort research of an essentially healthy population, suggesting that mechanisms other than portal hypertension may underlie thrombocytopenia in healthy HBV carriers. [4]

Case Report

A 53 year old man Z.I came to our clinic complaining of maculo-papular (photo nr.1) confluent rash on both lower limbs after consuming alcohol.It gives information that similar changes have appeared 5-6 times last year but,they spontaneously resolved.Elevated temperature and accompanying symptoms were not registered.



Picture 1: Macular-papular rash.

Lab tests was performed,HGB 16.30 (normal value 11.00-18.00 g\Dl),WBC 7.66 (normal value 4.00-10.50 10^3\Ul), elevated AST recorded: 333; (normal value 10-34 U\L), ALT: 385 (normal value 10-45 U\L),indirect bilirubin 13.3 (normal value below 13.6 umol\L);direct bilirubin 12.1 (normal value below 6.8 umol\L;)total bilirubin 25.4 (normal value below 25.4 umol\L); mild thrombocytopenia 125 (normal value above 150 10^9\L),hemostasis was in normal ranges INR1,15(normal values 1.00-3.00); aPTT 26.2 sec (normal values 24-35);.D-dimer test was high 813 (normal value 0-500 ng\ml).Other laboratory findings such as viral,tumor and serum markers were all negative except HBsAg,AntiHBc,AntiHBe REACTIVE (positive).

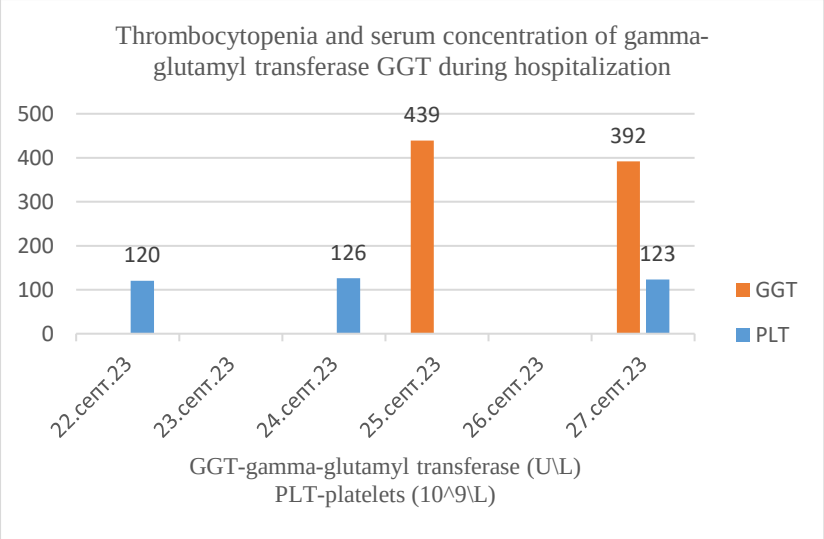
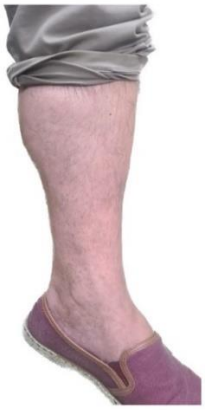


Figure 1. Progression of selected laboratory parameters:gamma-glutamyl transpeptidase and platelets

Physical examination of the abdomen - the abdomen was soft on palpation without painful tenderness.EHO of abdomen was also performed;Liver, nonhomogeneous structure with cirrhotic changes, fine-grained appearance with fibrosis and without convincing signs of a visible defect in the parenchyma. Gallbladder,lien and pancreas was normal. The right kidney was in order, the left kidney had one cortical cyst with and a smaller one without obstacles in urodynamics. No large lymph nodes, no ascites was seen in the abdomen.



Picture 2*:*Normal skin without macular-papular rash,after therapy administration

Discussion

Thrombocytopenia can be caused by a reduction in platelet production, platelet sequestration, or platelet destruction. Acute liver failure patients may have thrombocytopenia for a variety of causes, including reduced thrombopoietin (TPO) production, splenic sequestration, or, in rare cases, consumptive coagulopathy. Thrombocytopenia is common in patients with acute liver failure. This is a rare instance of thrombocytopenia coupled with concomitant diagnoses of HBV. [5]

Hepatitis B virus (HBV) is a DNA virus that infects the liver. HBV has been discovered in a range of extrahepatic tissues, including the kidney, thyroid, pancreas, bone marrow, and others, in

addition to the liver. By infiltrating these tissues, HBV might cause significant consequences. Pancytopenia is one of the most prevalent consequences, particularly thrombocytopenia, which causes life-threatening bleeding. The etiology of thrombocytopenia, however, is unknown, and therapy is exceedingly challenging. HBV has been shown to have a tight association with platelets. By stimulating the monocyte-macrophage system and the immune system, HBV can directly infect bone marrow, restrict platelet formation, and accelerate platelet death. Platelets can deliver anti-fibrotic chemicals to the liver, improving its functions and reducing hepatic fibrosis. [6]

Pharmacological medications such as lamivudine and corticosteroids were used to treat the patient's condition.

Current hepatitis B treatment options are divided into two distinct groups, immune modulator medications these are interferon-type medications that support the immune system fight the hepatitis B virus. They are administered as a shot over a period of 6 months to a year. Antiviral medicines these are medications that prevent or inhibit the replication of the hepatitis B virus, reducing inflammation and liver damage. These are normally taken as a pill once a day for at least a year. [7]

In individuals with chronic hepatitis B, lamivudine treatment can restore antiviral T cell responses. It is uncertain if the recovery of T cell responsiveness is long-lasting and remains during treatment, and whether the increase in viremia that occurs after medication discontinuation might return a state of T cell unresponsiveness. [8]

In patients with chronic or resolved HBV infection who are undergoing chemotherapy or immunosuppressive medication, entecavir appears to be more beneficial than lamivudine in avoiding HBV reactivation and HBV-related hepatitis. [9]

Tenofovir and entecavir are recommended over lamivudine in long-term immunosuppressive therapy due to their lower resistance profile. Corticosteroids may boost HBV expression via a glucocorticoid-responsive element found in viral genoma and accelerate viral replication in individuals receiving these medicines. Despite the increase in viral replication, the major damage rarely appears during the period of maximal immunosuppression and typically develops after the immunosuppressive therapy is discontinued, during the stage of immune reconstitution, when the body's immune system has the ability to eliminate the hepatitis B-infected hepatocytes, resulting in liver disease. [10]

Conclusion

Strong relationships between HBsAg positive and thrombocytopenia were consistently detected across the globe. HBsAg positive was highly and independently related with incidence thrombocytopenia in many research studies. The patient's disease was treated with pharmacological drugs such as lamivudine and corticosteroids.

References

1. L.J. Mark Feldman. Lawrence S. Friedman. Sleisenger and Fordtran’ s Gastrointestinal Liver DiseasePatophysiology Diagnosis Management, in Gastrointestinal Liver Disease, Philadephia, Elsiver, 2021, pp.1216-1277.
2. J.W.Yeh, H.S Yang. Dermatological disease associated with Hepatitis B virus infection. Dermatologica Sinica, pp142-150. 2020.
3. D.L.D.Ioana Grigorescu.Sponatneous antiviral induced cutaneous lesions in chronic hepatitis B infection.WJG. 2014:15860-66.
4. Y.CJ.J-Joo. Hepatitis B infection is associated with an increased incidence of thrompocytopenia in a healthy adults without cirrhosis.Journal of Viral Hepatits. 2016: 253-8.
5. DM Cloe Nobuhara. Immune thropmocytponeia associated with hepatitis B virus autoimmune hepatits and recovery of platelet count following liver transplantation. Hindawi case report in Transplantation, 2021; 1-5.
6. Human Jiang. Relationship between hepatitis B virus infection and platelet production and dysfunction. Plateles, 2021:212-218
7. Trearment options for Hepatitis B [online].Avialable:https// www.herb.org/treatment and managment/treatment

8. Carolina Boni. Transient restorationof anti-viral T cell responses induced bu lamivudine therpay in chronic hepatitis B. Jof hepatology,2003; pp595-605.
9. Chung Yang. Meta analysis of prpophylactic enecavir or lamivudine agains hepatitis B virus reactivation. Annals of Hepatology, 2016; pp501-511.
10. CFR Pilar Lopez- Serrano. Hepatitics B and immunosuppressive therapies for chronic inflammatory dieseas. When and how to apply prophylaxis, with a special focus on corticosteroid therapy. World Journal of Hepatology; 2015: pp 539-547