CASE REPORT

Ischemic stroke in a patient with HBV and HDV related DCLD: A review report

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ABSTRACT

Stroke is a cerebrovascular accident to a part of brain that leads to brain injury and could be hemorrhagic or ischemic of type. It is a medical emergency that needs prompt treatment. Stroke is commonly caused in patients with risk factors like Obesity, Diabetes, Hypertension, dyslipidemias, cardiac disease, alcohol consumption, illicit drug use, sedentary life style or with a family history of stroke. Cirrhosis is a rare cause of ischemic stroke. Studies show that HBV has a protective role in preventing stroke.

More studies show HCV related DCLD with hemorrhagic stroke. In our case study, patient was HBV superinfected with HDV infection developed DCLD and had anticoagulant state and he developed ischemic without another risk factors. MRI Brain and MRS showed bilateral multiple infarcts. Patient however could not be given antiplatelet due to high INR and low platelet count. Patient deteriorated over next two days and collapsed but couldn't be revived

Key Words: *Hypertension; Dyslipidemias; Cerebrovascular; Cirrhosis*

INTRODUCTION

Hepatitis B virus is a double stranded DNA virus, and its infection is major health issue and is one of the common infection worldwide. About 3.5% of the populations is currently suffering from HBV infection globally. Most of the adult patients develop chronic hepatitis B without presenting with acute viral hepatitis. This virus is transmitted from person to person via infected blood, semen, household and infected syringes. It also has a vertical transmission from mother to child during birth of a HBV infected mother. Most of these patients have silent acute stage and 90% present with chronic hepatitis B. Incidence of infection is decreasing now a day due to availability of vaccination since end of 20th century. Treatment options for infected patients include antivirals including reverse transcriptase inhibitors that suppress the viral replication process. 5% of the infected patients develop chronic hepatitis B, 20% of those will develop cirrhosis and about few has the probability to develop hepatocellular CA. 1 million deaths are reported annually secondary to HBV infection. Cirrhosis is a serious condition that develops due to chronic liver injury that leads to fibrosis, scarring and nodular formation progressively leading to shrinkage of liver. This can occur due to many causes like in chronic hepatitis C i.e. 47%, chronic hepatitis B (20%), alcohol is responsible for 18% of the cause of cirrhosis.Other causes include autoimmune hepatitis, congenital disorders like PBC, alpha 1 antitrypsin deficiency, hemochromatosis, Wilson's Disease, congestive heart failure or Budd Chiari syndrome. It is end stage liver disease that could be compensated or decompensated. In patients with compensated cirrhosis they remain asymptomatic for most of the life. Only 5-7% show symptoms each year with a life expectancy of 9-12 years. In decompensated cirrhosis, there is very decreased life expectancy. Child Tourette Pugh score is calculated to estimate the life expectancy and MELD scoring is done to assess risk of mortality. Stroke can lead to major disability and functional impairment of different body parts. Among stroke, about 87% is ischemic stroke. Cirrhosis is a very uncommon risk factor for stroke development. Patients with decompensated cirrhosis, more commonly with HCV, if develop stroke, are more prone to develop hemorrhagic stroke. In our case, patient had no other risk factor to develop stroke except decompensated cirrhosis that was secondary to hepatitis B virus supeinfected with HDV infection, in highly anticoagulant state when he developed ischemic stroke.

CASE PRESENTATION

A 53 years old gentleman, product of non-consanguineous marriage and a non-smoker presented to us in Emergency department of Pakistan Atomic Energy Commission General Hospital in October, 2021 with the complaints of fever and abdominal pain for 5 days. Patient was known case of Hepatitis B infection for last 5 years, which was untreated and was initially uncomplicated. He later developed hepatitis D virus superinfection about 6 months back. Patient started developing progressive distension of abdomen about 3-4 months back when he was advised

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diuretics for ascites. Patient was in his usual state of health but progressively worsened over last 1-2 months with developing gross ascites, reduced appetite and generalized weakness and lassitude. Patient had a few visits a nearby clinic where he was advised symptomatic treatment. About 1-2 weeks before arriving at our Hospital, patient had an episode of hematemesis for which upper GI endoscopy and banding of grade 3 esophageal varices was done. Now he was having abdominal pain for 5 days associated with low grade undocumented fever. Pain was mild to moderate intensity, non-radiating and continuous. It was not associated with vomiting, constipation or urinary symptoms etc. There was no associated active hematemesis or melena or bleed. There was no history of encephalopathy. Patient had a past history of treated pulmonary TB about 30years back without any sequelae or complications. On examination, patient had a GCS of 15/15, with a BP of 105 mmHg /70 mmHg, pulse 80/min, RR 18/min, afebrile and SpO2 98% initially, jaundiced and mild pedal edema. Abdomen was distended and was mildly tender, bowel sounds were present. Chest had bilateral basal fine crepts. On auscultation of precordium, S1 and S2 sounds were audible without any added sounds. CNS and rest of the examination was unremarkable. Patient was admitted to ward and was empirically managed on the lines of HBV with HDV related DCLD and spontaneous Bacterial Peritonitis. Labs showed Hb of 8.0 mg/dl, platelet count of 88,000 with a WBC count of 12,000, LFTs and RFTs were deranged with bilirubin of 2.8, creatinine 2.7, Urea 270 mg/dl. Mildly raised inflammatory markers with CRP of 89, Albumin 2.56, INR 2.4 and APTT 35. Ascetics RE showed SAAG of1.9 and raised neutrophil count showing SBP, without any evidence of malignant cells on its cytology. Urinary sodium was low i.e 9 and AFP was 25 and rest of tumor markers were also negative. Patient was given antibiotics for SBP and superadded infections, with symptomatic treatment for ascites, prophylactic treatment for prevention of encephalopathy. Intravenous Human Albumin was also replaced. Patient had deranged RFTs secondary to Hepatorenal syndrome for which he was treated accordingly. He had a Child Class C Cirrhosis with MELD score of 20. Patient initially improved and was worked up for liver transplantation but after a day or two, he developed left lower limb weakness followed by weakness of left upper limb, however not associated with dysphagia, deviation of mouth, loss of consciousness or sudden drop of GCS.

On examination, he had power of 2/5 in left upper and lower limb progressively became 0/5 with hyperreflexias and left plantar was up going. However, GCS remained 15/15. CT Brain was done that was normal, having no evidence of bleed or infarct. MRI Brain was done that showed multiple abnormal hyper intense signal intensity areas of variable sizes in deep white matter of bilateral cerebral hemispheres, isointense to gray matter. No perilesional edema was noted. Magnetic Resonance Spectroscopy was also done to confirm the findings of MRI, showing the evidence of multiple infarcts. Lipid profile was normal with a cholesterol of 50 mg/dl and TG of68 and normal thyroid function tests. Patient was not given antiplatelet as he had low platelet count of 64,000 with high INR of 3.2. ECHO was normal showing no clot and EF 60%. Patient was managed symptomatically, given good nursing care with physiotherapy, FFPs transfused but patient further deteriorated, became drowsy, hypotensive and gross ascites and pulmonary edema despite therapeutic ascetic tap. He was then shifted to intensive care unit, where he was given vasopressors but his clinical lab parameters worsened that after 1 week of admission, he had cardiac arrest when he could not be revived back despite managing on full ACLS protocolat zero second. Death was declared.

DISCUSSION

In our case, patient had no other risk factors for developing stroke. Patient had HBV with HDV infection with highly anticoagulant state with INR >2.5 and low platelet count with severe liver dysfunction, when he developed ischemic stroke. Most of the studies have showed that HBV infected patients have a low risk of stroke and thus has a protective role. Cirrhosis and stroke are two independent causes of major morbidity and mortality around the world. The patients with cirrhosis who develop stroke have many other risk factors for stroke development e.g. diabetes, hypertension, dyslipidemia or cardiac diseases etc. Studies have been done that show a positive relation between cirrhosis and stroke however hemorrhagic has more probability to occur than the ischemic stroke. Hemorrhagic stroke has a prevalence of 0.8-34.3% while that of ischemic stroke is 0.8-6.5% [1].

Many studies have been done that proclaims cirrhosis to be an important cause of stroke with a prevalence of 2.175% and about 1.11% in those without cirrhosis [2]. Likewise, HCV infection is also reported to have a relation with stroke occurrence. A study was done in which the patients had HCV infection who developed stroke had lower levels of triglycerides and cholesterol but a higher levels of inflammatory markers i.e. CRP, ESR and fibrinogen [3]. Another study done in Taiwan concluded HCV infection to be an independent risk factor for stroke. However, study did not mention whether they are more prone to develop hemorrhagic or ischemic stroke [4].

Stroke is not very common in patients with cirrhosis with a prevalence of about 9.0% of unspecific stroke but there is more risk to develop intracranial hemorrhage or Sub arachnoid hemorrhage [5]. Another study also proclaimed intracranial hemorrhage and SAH have probability to develop than ischemic stroke. The cause of cirrhosis was not studied [2]. A study in Korea was done on the causes of cirrhosis in patients developing stroke. Alcohol was found to be the important cause of cirrhosis, other atients had diabetes, hypertension, metabolic syndrome and cardiac arrhythmias. Only two patients out of 23 had no other risk factor. Majority of these were child Pugh B (39%) and Child Pugh C (26%). Out of these two, one had cirrhosis secondary to hepatitis C and the other developed cirrhosis due to chronic alcoholism. However, all the patients had normal liver function except one who had INR >1.7 [6] The risk of stroke in cirrhosis was found to be more in female patients than male patients [7]. Among the patients developing ischemic stroke with cirrhosis were very old aged, had low AST, ALT and PT, high platelet count, high triglycerides, high WBCs but had very low HBV infection [8]. Thus stroke could be an important cause of cirrhosis, however the

cause is unknown. Most of the studies report alcohol and HCV infection to be a major cause of cirrhosis leading to stroke. Among it, hemorrhagic stroke has more prevalence. Comparison of HBV and HCV infection for the stroke incidence showed HCV to be an independent risk factor but HBV infection has very low chance of developing stroke [9]. A study concluded that risk of stroke in HBV infected patients is less than patients with other comorbidities [10]. Studies done in various countries showed that HBV infection has an inverse relation with the prevalence of stroke. A largest study done that obtained similar results about the risk of stroke from both western and eastern countries. HBV infection reduces risk of stroke by 22% than uninfected patients [11,12].

CONCLUSION

Hepatitis B virus infection is one of the risk factor for ischemic stroke. It is rare for ischemic stroke to occur in hepatitis B virus infection but is an independent risk factor for stroke. More studies needs to be done whether to thrombolysis or to perform thrombectomy in such patients. Patients admitted with hepatic encephalopathy and comatose state must have a CT Brain done to rule out stroke

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