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Case Report

A Rare Cause of Acute Hepatitis: Herpes Simplex Virus

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Abstract

HSV hepatitis is a rare cause of acute liver failure, accounting for 0.8% of all cases and only 2% of all viral hepatitis. It is mostly seen in immunocompromised individuals and pregnant women in the third trimester following orogenital HSV-1 or HSV-2 infection, but previous reports have shown up to 25% of cases in immunocompetent individuals. Due to the lack of specific clinical signs, diagnosis is often missed at presentation and if left untreated, it can lead to rapid progression to fulminant liver failure and multi-organ collapse.

Keywords: Acute hepatitis; Liver failure; X-ray; Pathology; Infections; HSV

Introduction

HSV hepatitis is a rare cause of acute liver failure, accounting for 0.8% of all cases and only 2% of all viral hepatitis. It is mostly seen in immunocompromised individuals and pregnant women in the third trimester following orogenital HSV-1 or HSV-2 infection, but previous reports have shown up to 25% of cases in immunocompetent individuals. Due to the lack of specific clinical signs, diagnosis is often missed at presentation and if left untreated, it can lead to rapid progression to fulminant liver failure and multi-organ collapse.

Case Presentation

The patient, who was followed up with a diagnosis of hypertension and did not use any medication other than candesartan 8 mg tablets, was hospitalized for the etiology and treatment of acute hepatitis. He has no history of smoking, alcohol, substance use or mushroom eating. Covid has become 3 doses of biontech. He had Covid 19 in December 2020. On physical examination, fever: 36.5 NDS: 80/min BP: 120/80 SS: 14/min consciously oriented PA Lung X-ray: normal X-ray findings Abdominal US: The liver is of normal size, with smooth contours. Parenchyma is homogeneous. No focal lesion was detected. Intrahepatic bile ducts are natural. The spleen size is normal. Parenchyma is homogeneous. Portal vein diameter is 11 mm and it is within normal limits. No significant thrombus was detected in the portal vein. Portal vein flow direction is hepatopedal. Portal vein diameter is normal and shows inspiratory changes. The diameter of the splenic vein is 8 mm, and the diameter of the superior mesenteric vein is 8 mm. Flow directions in these vessels are hepatopedal. Recanalization was not observed in the paraumbilical vein (Figure 1). HSM IGM: 25.5 Positive, 39.0 Positive (Tables 1 and 2).

In the pathology of liver biopsy; Microanatomy is largely preserved in the liver tissue, and there are 12 portal areas in total.

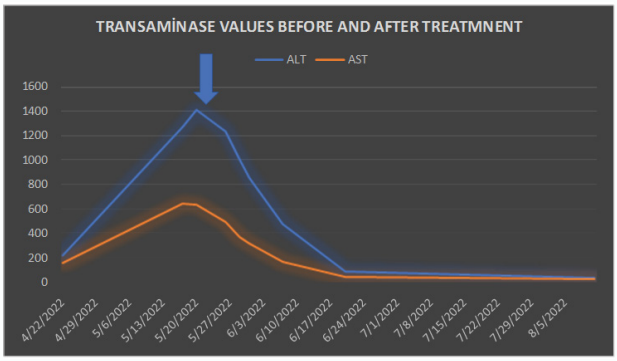

Figure 1: The course of transaminases during diagnosis and treatment. The blue arrow indicates the start of treatment.

Hydropic degeneration in hepatocytes, spotty and confluent necrosis foci, apoptotic bodies, congestion in sinusoids, prominent Kupffer cells, few mitotic figures, hyperchromasia in some hepatocyte nuclei, intranuclear inclusion-like structure, presence of perinuclear halo were observed in liver parenchyma. "Infectious Factors" were considered in the foreground.

After IV, acyclovir treatment was started, a decrease trend in transaminases started on the second day of the treatment, and clinical and laboratory improvement was detected in the patient after 15 days of treatment.

Discussion

Liver biopsy remains the only gold standard for the diagnosis of HSV hepatitis. However, the procedure is often not possible due to coagulopathy or ascites, and a diagnosis is often required based on laboratory tests and symptoms. In its pathology, hemorrhagic necrosis, inflammation, expanded ground glass nuclei with marginalized chromatin and intranuclear inclusion-like structure, perinuclear halo is observed [1].

Although oral and genital mucocutaneous lesions are useful in supporting the diagnosis in patients with suspected HSV hepatitis, they are absent in up to 50% of cases. Serological tests are widely used but carry a high rate of false negatives [2,3].

Other common features of HSV hepatitis in the absence of biopsy are >500 aminotransferases, fever, coagulopathy, encephalopathy, leukopenia, thrombocytopenia, and Acute Renal Failure (ARF).

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Table 1: The patient's biochemical examinations were as follows in the outpatient clinic, hospitalization and subsequent follow-ups.

WBC	HGB	PLT	ALT	AST	ALP	GGT	T.BİL	D.BİL	İNR
11.12	14.1	201	225	159	123	77	0.5	0.21	0.89
6.08	14.8	254	1273	643	166	134	0.94	0.51	1.06
6.72	14.4	267	1417	632	193	159	1.29	0.74	1.08
7.33	14	315	1242	491	206	145	1.33	0.78	1.07
6.54	13.5	250	1007	365	170	135	1.32	0.67	1.1
6.54	13.4	250	859	316	151	130	1.34	0.77	1.08
6.92	13.8	224	477	164	130	111	1.06	0.69	1.06
5.98	15	244	90	40	89	63	0.53	0.25	0.96
5.93	13.3	215	32	29	54	16	0.75	0.29	0.99

Table 2: The patient's biochemical examinations were as follows in the outpatient clinic, hospitalization and subsequent follow-ups.

HBSAG	NEGATİF
ANTİ HBS	NEGATİF
ANTİ HCV	NEGATİF
ANTI HBCIGM	NEGATİF
ANTİ HAV IGM	NEGATİF
ANTİ HEV	NEGATİF
ANTİ HBE	NEGATİF
ANTİ HBC TOTAL	NEGATİF
ANA	NEGATİF
ANTİ LKM1	NEGATİF
AMA- M2	NEGATİF
SERUPLAZMİN (MG/DL)	35
TRANSFERRİN SATURASYONU (%)	24

Although all these were not seen, the anicteric pattern of hepatitis was noted as a common point in the presentation of this disease [4].

It has been shown that early initiation of acyclovir (within 3 days) leads to better outcomes as it inhibits viral replication and prevents the spread of infection. Although data are scarce, intravenous foscarnet may be the agent of choice in cases of acyclovir-resistant herpetic infections [5]. Overall, the data also suggest that in high-risk patients with ALF of unknown etiology, acyclovir should be initiated empirically and await laboratory confirmation of the diagnosis. However, prospective data on empirical therapy in certain populations are lacking [6].

Conclusion

HSV hepatitis as a cause of fulminant liver failure is a factor that must be considered in the differential diagnosis, especially in high-risk patient populations. Although diagnosis by biopsy is the gold standard, HSV PCR with simultaneous elevation in aminotransferases may serve as substitution markers for diagnosis. HSV hepatitis should not be excluded due to the absence of mucocutaneous lesions, markers of systemic inflammatory reaction, ARF or anicteric hepatitis; however, these findings can be used to support a dubious diagnosis. Given the favorable side-effect profile of acyclovir, empiric treatment should be initiated early in admission.

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