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ORIGINAL ARTICLE

Assessment of histological and immunohistochemical features in fatal acute fulminant hepatitis E

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ABSTRACT:

Background: Hepatitis E is a viral liver disease caused by the hepatitis E virus (HEV). The present study was conducted to evaluate histological and immunohistochemical features in fatal acute fulminant hepatitis E.**Materials & Methods:**Postmortem liver biopsies of 28 patients with fulminant hepatitis E of both genders with formalin-fixed paraffin-embedded tissue was sectioned at 2 to 4 µm thickness and stained with hematoxylin and eosin, reticulin, and Masson's trichrome stains.Immunohistochemistry was performed on formalin-fixed, paraffin-embedded liver sections. **Results:** Out of 28 patients, males were 15 and females were 13. Necrosis was seen in 27, ballooning degeneration in 26, councilman bodies in 6, pseudo-rosettes in 17, steatosis in 19, plasma cells in portal inflammation in 4, interface hepatitis in 27, Kupffer cell prominence in 26, bile ductular proliferation in 24 and bile duct damage in 14 patients. **Conclusion:** Due to the prevalence of HEV genotypes, histological alterations in HEV infection may differ geographically, and CD8+ cells may contribute to HEV-induced liver damage.

Keywords: Kupffer cell, ballooning degeneration, hepatitis E

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INTRODUCTION

Hepatitis E is a viral liver disease caused by the hepatitis E virus (HEV). It is primarily transmitted through the consumption of contaminated water or food, particularly in areas with poor sanitation.[1] The virus can also be transmitted through the fecal-oral route, and in some cases, through blood transfusions or organ transplants.Hepatitis E usually resolves on its own within a few weeks to months and does not typically cause chronic infection.[2] However, it can be more severe in pregnant women, leading to a higher risk of complications such as liver failure, and it can also be more severe in people with pre-existing liver disease.Symptoms of hepatitis E can include jaundice (yellowing of the skin and eyes), fatigue, abdominal pain, nausea, vomiting, and fever. However, some people infected with HEV may not experience any symptoms.[3]

There are four known HEV genotypes; genotypes 1 and 2 do not infect animals but instead cause sickness in endemic areas in humans.[4] On the other hand, genotypes 3 and 4 primarily affect elderly people who also have other coexisting disorders, and they mostly infect animal species with sporadic zoonotic transmission to humans, mostly in places where HEV infection is not endemic.Liver biopsy is an invasive technique that carries a risk of internal bleeding and is generally not recommended during acute viral hepatitis.[5]This means that there is little literature on the histological alterations in the liver caused by HEV infection, with only sporadic small case series being published. The majority of these were from wealthy nations with low rates of HEV infection.[6]The present study was conducted to evaluate histological and immunohistochemical features in fatal acute fulminant hepatitis E.

MATERIALS & METHODS

The present study was conducted on postmortem liver biopsies of 28 patients with fulminant hepatitis Eof both genders.

Data such as name, age, gender etc. was recorded. Paraffin blocks of liver biopsies were retrieved from our archives. Formalin-fixed paraffin-embedded tissue was sectioned at 2 to 4 µm thickness and stained with hematoxylin and eosin, reticulin, and Masson's trichrome stains. Immunohistochemistry was performed on formalin-fixed, paraffin-embedded liver sections.Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 28		
Gender	**Males**	**Females**
Number	15	13

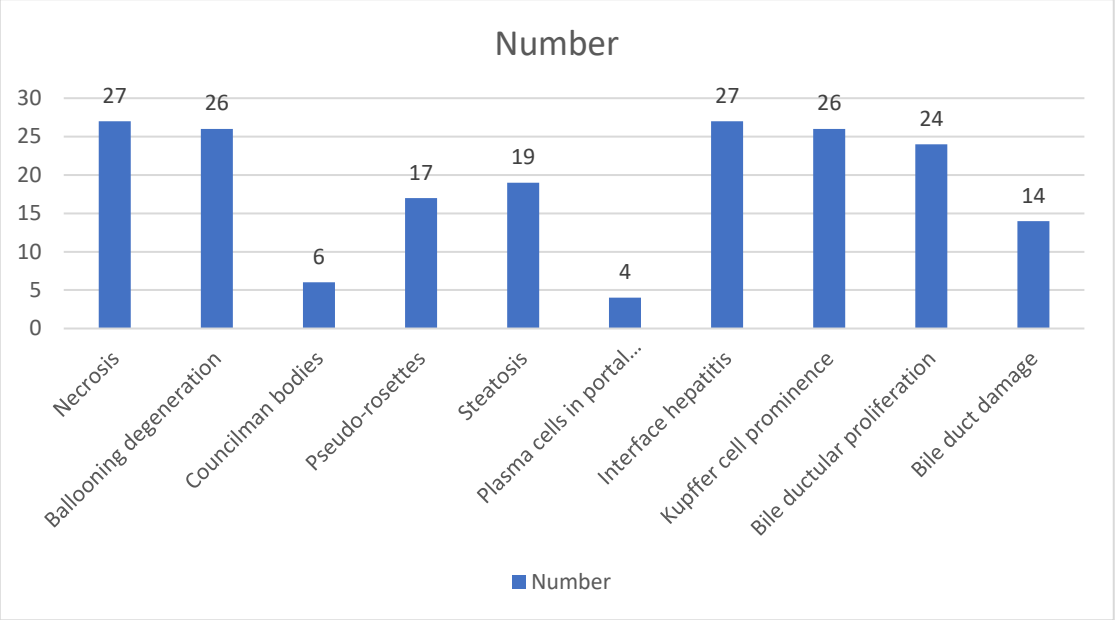
Table I shows that out of 28 patients, males were 15 and females were 13.

Table II Histology findings in postmortem liver biopsies

Parameters	Number	P value
Necrosis	27	0.81
Ballooning degeneration	26	
Councilman bodies	6	
Pseudo-rosettes	17	
Steatosis	19	
Plasma cells in portal inflammation	4	
Interface hepatitis	27	
Kupffer cell prominence	26	
Bile ductular proliferation	24	
Bile duct damage	14	

Table II shows that necrosis was seen in 27, ballooning degeneration in 26, councilman bodies in 6, pseudo-rosettes in 17, steatosis in 19, plasma cells in portal inflammation in 4, interface hepatitis in 27, kupffer cell prominence in 26, bile ductular proliferation in 24 and bile duct damage in 14 patients. The difference was significant (P< 0.05).

Graph I Histology findings in postmortem liver biopsies



DISCUSSION

Preventive measures for hepatitis E include practicing good hygiene, such as washing hands thoroughly with soap and water, and avoiding consuming water or food that may be contaminated.[7,8] In regions where hepatitis E is endemic, vaccination may be recommended, particularly for pregnant women and travellers to affected areas.[9]Treatment for hepatitis E typically focuses on supportive care to relieve symptoms, such as rest, adequate hydration, and medications to manage nausea and vomiting.[10] In severe cases, hospitalization may be necessary, particularly if there is a risk of liver failure. However, there is no specific antiviral therapy for hepatitis E.[11]The present study was conducted to evaluate histological and immunohistochemical features in fatal acute fulminant hepatitis E.

We found thatout of 28 patients, males were 15 and females were 13. Agrawal et al[12]studied the histological features and the type of inflammatory infiltrate in liver biopsies of patients with acute fulminant hepatitis E. They retrieved postmortem liver biopsies of 11 Indian patients with fulminant hepatitis E, and compared these with biopsies from seven patients with fulminant hepatitis B. Biopsies from acute fulminant hepatitis E showed varying degrees of hepatocyte necrosis, mixed portal and lobular inflammation, accompanied by bile ductular proliferation, lymphocytic cholangitis, Kupffer cell prominence, cholestasis, apoptotic bodies, pseudo-rosette formation, steatosis, and presence of plasma cells in portal tracts. Interface hepatitis was more frequent in acute hepatitis B than in acute hepatitis E. These findings differ from those reported in cases with autochthonous hepatitis E in Europe. On immunohistochemistry, lymphocyte infiltrate consisted predominantly of CD3+ T cells in both hepatitis E and hepatitis B; these cells contained a predominant cytotoxic (CD8+) cell subpopulation in

81.8% of cases with hepatitis E and in 50% of cases with hepatitis B.

We observed thatnecrosis was seen in 27, ballooning degeneration in 26, councilman bodies in 6, pseudo-rosettes in 17, steatosis in 19, plasma cells in portal inflammation in 4, interface hepatitis in 27, kupffer cell prominence in 26, bile ductular proliferation in 24 and bile duct damage in 14 patients. Peron et al[13]reported a series of 11 patients with sporadic acute hepatitis E and needle liver histology. Hepatitis E was diagnosed based on elevated transaminases (>10 upper limit normal) and the presence of specific serum antibodies (immunoglobulin-G class, present in all 11 patients) and/or viral RNA detection in serum and/or stools. Acute hepatitis lesions were observed in all cases with marked necro-inflammatory activity in nine patients. Confluent necrosis was present in five cases. Anisocaryosis and Kupffer's cell aggregates with siderosis were observed in most of the 11 patients. Cholangitis was frequent (9/11 cases). Cholestasis was observed in eight cases. Pseudo-glandular pattern was present in only one case but without zonal repartition. Characteristic pathological signs of acute hepatitis E were severe intralobular necrosis, polymorph inflammation, and acute cholangitis with numerous neutrophils.

Malcolm et al[14] in their study four patients were serologically positive for HEV; three had no traditional risk factors. Liver histology of the three autochthonous (locally acquired) cases showed portal tracts expanded by a severe mixed polymorph and lymphocytic inflammatory infiltrate, with a geographical distribution of polymorphs at the interface and lymphocytes centrally. Moderate to severe interface hepatitis and cholangiolitis were present. There was a striking acinar mixed inflammatory infiltrate made up of polymorphs, lymphocytes and macrophages; frequent apoptotic hepatocytes, focal necrosis, cholestatic rosettes and zone 3 canalicular and cytoplasmic bilirubinostasis were noted. Significant steatosis, megamitochondria and Mallory bodies were not present. There was no evidence of iron, copper or alpha(1)-antitrypsin accumulation. By contrast, the histology of the imported case of HEV infection showed less intense portal and acinar inflammation, no cholangiolitis and no geographical distribution of the portal inflammatory infiltrate.

The shortcoming of the study is small sample size.

CONCLUSION

Authors found that due to the prevalence of HEV genotypes, histological alterations in HEV infection may differ geographically, and CD8+ cells may contribute to HEV-induced liver damage.

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