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A Prospective Clinical Study on Hypoxic Hepatitis in Surgical Intensive Care Unit

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Abstract: ***In spite of tremendous progress in surgery, sepsis still remains the main cause of death in surgical intensive care unit. Detoxifying liver dysfunction which occurs in sepsis has a significant impact on mortality in this set of patients. Our prospective study focusses on the clinical, biochemical, and hemodynamic profile of hypoxic hepatitis in surgical intensive care units in tertiary care academic institution. Possible pathophysiology, prognosis and recommendations are also discussed.***

Keywords: sepsis, liver dysfunction, pathophysiology, prognosis

1. Introduction

Though there are tremendous achievements in recent surgical era, sepsis remains the main threat which is the lead cause of mortality in post operative patients in surgical intensive care unit. The novel detoxification function of liver which nullifies enormous toxic products gets augmented in sepsis. This liver dysfunction which occurs in sepsis has a significant impact on mortality in this set of patients. Our prospective study focuses on the clinical, biochemical, and hemodynamic profile of hypoxic hepatitis in surgical intensive care unit in tertiary care academic institution.

Aim and Objectives

1) To study the clinical, hemodynamic and biochemical profile of hypoxic hepatitis in septic shock due to underlying condition.
2) To study the prognosis of hypoxic hepatitis in septic shock and discuss the pathophysiology and treatment of hypoxic hepatitis.

2. Materials and Method

2.1. Study type

Observational

2.2 Study design

Non randomized prospective study

2.3. Study group

Over two years (2018-19) 32 patients with sepsis due to underlying surgical pathology presenting with hypoxic hepatitis in surgical intensive care unit of the govt mohan kumarangalam medical college hospital, Salem, Tamil Nadu were enrolled in this study as data source. Individuals who fulfilled the inclusive criteria and exclusive criteria were enrolled in this study.

2.4. Inclusive criteria

a) Any age and any sex.
b) Patients with sepsis due to underlying surgical pathology.
c) Dramatic increase in serum aminotransferase activity reaching at least 20 fold of upper normal.

2.5. Exclusion criteria

a) Not willing to give consent.
b) Underlying known liver pathology.
c) Viral or drug induced hepatitis.

2.6 Study method instituted

Patients presenting with sepsis due to any underlying surgical pathology and presenting with symptoms and signs of hypoxic hepatitis were enrolled in non randomized manner. An elementary hemodynamic evaluation including vitals assessment, arterial blood gas analysis, serum amino transferase level and other liver function test with serial assessment were done. Ultrasound scan abdomen and hepatitis viral markers were done to exclude the organic liver pathology, Non randomized selection of all eligible patients were included in this study for a period of two years. Results were documented as per clinical examination, hemodynamic and biochemical examination.

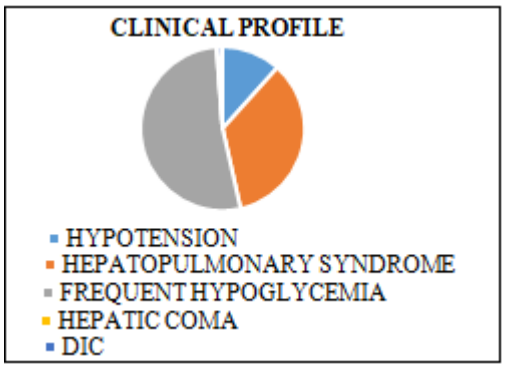
2.7. Follow up

Patients were followed up weekly for 3 months with biochemical investigations every fortnightly and documented.

3. Results

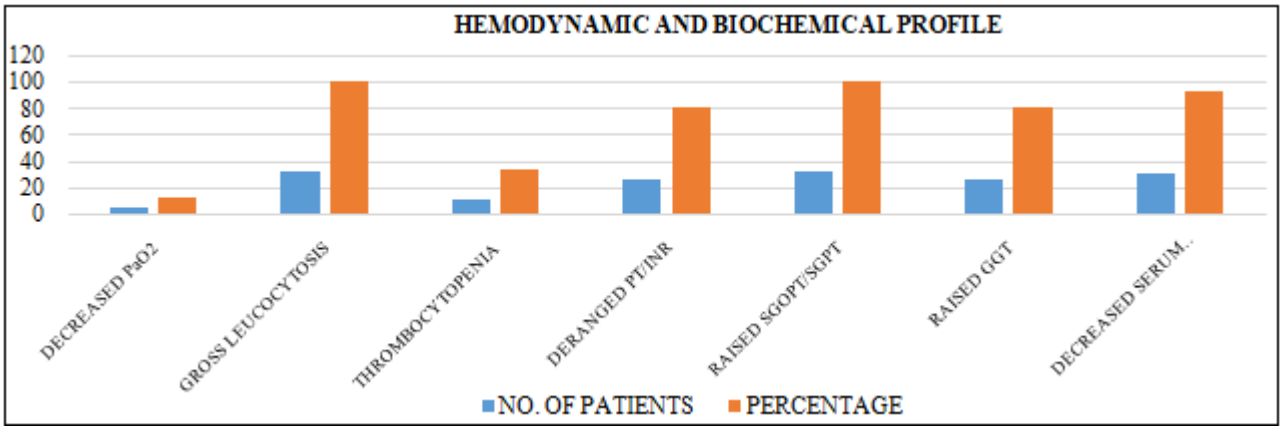
a) Clinical profile:

S.No	*Clinical Signs*	*No. of Patients*	*Percentage*
1.	Hypotension	4	12.5
2.	Hepatopulmonary syndrome	12	37.5
3.	Frequent hypoglycemia	18	56.25
4.	Hepatic coma / hepato renal syndrome	1	0.3
5.	Dic / bleeding diasthesis	1	0.3



b) Hemodynamic and biochemical profile

S.No	Parameter	No. of Patients	Percentage
1.	Decreasedpao2	4	12.5
2.	Gross leucocytosis and elevated ESR	32	100
3.	Thrombo-cytopenia	11	34
4.	Deranged PT/INR	26	81
5.	Raised SGOT/SGPT	32	100
6.	Raised GGT	26	81
7.	Decreased serum proteins	30	93

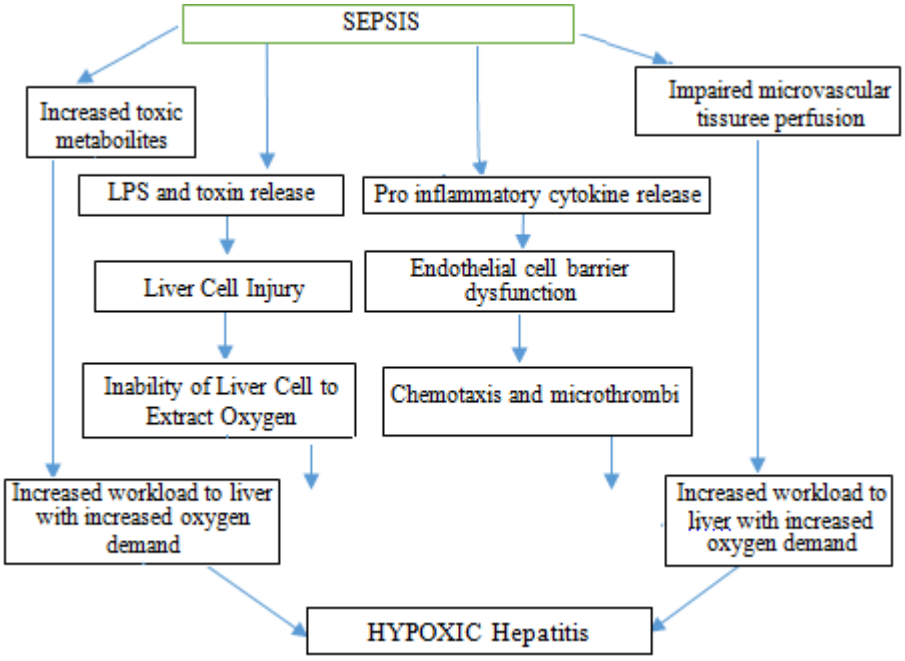


In our study we noticed 93 % hypoxic hepatitis patients initially presented with hypoproteinemia and then presented with raised AST which was proportionately higher than raised ALT. GGT was raised less proportionately than above two enzymes. More than 50 % presented with frequent hypoglycemic episodes. Less than 1% proceeded to DIC probably due to fulminant hepatic failure. 6 patients out of 32 died of septic shock and all others recovered without any remnant liver cell dysfunction in the follow up.

4. Discussion

Sepsis associated hypoxic hepatitis is usually contributed by systemic or microcirculatory disturbance. Our results clearly show that hypoxic hepatitis occurs not only in hypotension setting but also considerably in sepsis, patients with normal hemodynamic vitals. This clearly shows that the hypoxic hepatitis in sepsis is not merely due to decreased cardiac output to liver, but because of combined factors listed below.

- Sepsis poses a scenario of increased toxic metabolites reaching liver for detoxification and hence more demand for oxygen but not sufficient to meet the high demand.
- LPS and toxins and inflammatory cytokines induced injury to liver cells leading to inability of liver cells to extract oxygen.
- Microvascular tissue perfusion in sepsis is usually uncoupled from systemic circulation. This lead to decreased blood flow velocity in liver sinusoids.
- Increased proinflammatory cytokines usually causes dysfunction of endothelial cell barrier which elicits a significant chemotaxis of leucocytes and platelets causing subsequent micro thrombi. This further aggravates the liver issue ischemia and damage. Morbidly increased WBC count, increased ESR, deranged PT INR in all our cases support this point.



Also our study has emphasized that hypoalbuminemia precedes hypoxia hepatitis in majority cases. Leak of transaminases is characteristic of all cases with a predominance of AST over ALT followed by GGT. Hypoxic hepatitis progressed to fulminant liver failure in one patient in our study and one patient presented as disseminated intravascular coagulopathy. Frequent episodes of hypoglycemia occurred in sepsis only after liver derangement. Liver biopsy was not done in any of our patients as hypoxic hepatitis is purely based on clinical, hemodynamic and biochemical manifestation, histological confirmation is not mandatory. In our study, we also noted that the treatment of underlying sepsis, replacement of toxic secondary bile acids with ursodeoxycholic acid, management of microvascular tissue perfusion dysfunction and endothelial cell barrier dysfunction using simvastatin or atorvastatin perfectly reversed the liver dysfunction without any sequela in majority of patients. So, a thorough understanding of the pathophysiology of hypoxic hepatitis helps in perfect management of the patient, hence better prognosis. Also our study observed early enteral feeding of hemodynamically stable patients helped in early recovery of hypoxic hepatitis due to modulation of bile acids. Therefore we document the following recommendations.

5. Recommendations in hypoxic hepatits in sepsis

1) Early enteral feeding for better modulation of bile acids.
2) Substitution of toxic secondary bile acids with ursodeoxycholic acid.
3) Starting prophylactic and therapeutic statins in sepsis to prevent microvascular complications to reduce liver inflammation.
4) Perfect glucose monitoring and adequate glucose supply as necessary.
5) Avoidance of hepatotoxic drugs.
6) Foremost of all, treatment of underlying sepsis.

6. Conclusion

Perfect knowledge about pathophysiology, clinical profile, hemodynamic and biochemical profile of hypoxic hepatitis in sepsis, helps a surgeon to manage the condition perfectly and also to prevent mortality and dreadful complications to facilitate early recovery of the patient.

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Author Profile

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