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CHANGES IN ARTERIAL BLOOD PRESSURE IN PATIENTS WITH VIRAL HEPATITIS: CLINICAL AND PATHOPHYSIOLOGICAL PERSPECTIVES

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Abstract

Viral hepatitis is a widespread infectious disease affecting the liver parenchyma and leading to systemic metabolic and hemodynamic disturbances. Although hepatic injury is primarily associated with abnormalities in bilirubin metabolism, coagulation, and inflammation, alterations in arterial blood pressure (BP) remain under-investigated. This study aims to assess arterial BP changes in patients with acute and chronic viral hepatitis, evaluate contributing mechanisms, and identify clinical correlations with liver dysfunction. A total of 120 patients were examined and divided into hepatitis A, B, and C groups. Blood pressure, biochemical parameters (ALT, AST, bilirubin), and markers of hepatic synthetic function were recorded. Results demonstrated a significant trend toward reduced systolic and diastolic BP in moderate and severe hepatitis, whereas mild hepatitis showed stable or slightly increased BP

levels. The decrease in BP correlated with hypoalbuminemia, systemic vasodilation mediated by nitric oxide, and reduced vascular resistance. Hypertensive responses were found in patients with cholestatic variants and sympathetic overactivation. Understanding BP patterns in hepatitis is crucial for early management, prevention of circulatory collapse, and better clinical outcomes.

Keywords

Viral hepatitis; arterial blood pressure; hypotension; hepatic dysfunction; vasodilation; nitric oxide; portal hypertension; systemic circulation

Introduction

Viral hepatitis (A, B, C, D, E) represents one of the most common infectious diseases worldwide and significantly contributes to global morbidity. The liver plays a central role in maintaining vascular tone through albumin production, metabolism of vasoactive substances, and regulation of systemic blood volume.

Arterial blood pressure abnormalities—particularly hypotension—are often overlooked in hepatitis patients. This hemodynamic change may emerge due to:

- Decreased systemic vascular resistance
- Excessive nitric oxide (NO) production
- Reduced plasma oncotic pressure (hypoalbuminemia)
- Autonomic dysfunction
- Adrenal insufficiency in severe hepatitis
- Fluid imbalance due to vomiting, diarrhea, or ascites

Although hypotension is more characteristic of advanced hepatic dysfunction, hypertension may occur in cholestatic hepatitis or during sympathetic excitation.

This study investigates BP changes in hepatitis patients and identifies underlying mechanisms associated with disease severity.

Materials and Methods

Study Population

A total of **120 patients** (18–65 years old) diagnosed with viral hepatitis were enrolled. They were divided into the following groups:

- Group A: Hepatitis A (n = 30)
- Group B: Hepatitis B (n = 45)
- Group C: Hepatitis C (n = 45)

Clinical Assessment

- Systolic and diastolic blood pressure measurements
- Heart rate
- Edema and ascites evaluation
- Neurological status
- Presence of dehydration

BP was measured three times daily and averaged.

Laboratory Tests

- ALT, AST
- Total and direct bilirubin
- Albumin levels
- Prothrombin index
- Electrolytes (Na^+, K^+)
- Creatinine, urea

Severity Classification

- Mild
- Moderate
- Severe hepatic injury based on WHO clinical guidelines

Statistical Analysis

SPSS 26 was used; $p < 0.05$ considered significant.

Results

1. Blood Pressure Trends

- **Mild hepatitis:**
  - BP remained normal or slightly elevated (120–130/75–85 mmHg).
  - 15% of patients showed transient sympathetic-mediated BP rise.
- **Moderate hepatitis:**
  - 60% patients showed mild hypotension (95–110/60–70 mmHg).
  - BP reduction correlated with increased bilirubin and ALT levels.
- **Severe hepatitis:**
  - Marked hypotension (80–90/50–60 mmHg) observed in 72% of patients.
  - Associated with hepatic encephalopathy and coagulopathy.

2. Correlating Factors

- **Hypoalbuminemia (<32 g/L):** showed strong association with low BP ($p < 0.001$).
- **High NO levels and systemic vasodilation:** patients had lower systemic vascular resistance.
- **Electrolyte imbalance:** hyponatremia significantly reduced BP ($p = 0.02$).

• **Cholestatic hepatitis:** 18% showed increased BP due to sympathetic activation and RAAS stimulation.

3. Hepatitis Type Comparison

• Hepatitis A showed milder BP changes.

• Hepatitis B and C showed more pronounced hypotension, especially in chronic active forms.

Discussion

The study confirms that arterial BP changes are a significant but under-recognized aspect of viral hepatitis. Hypotension is more common and is mainly driven by:

1. Systemic vasodilation

In severe hepatitis, excess NO and inflammatory mediators decrease vascular resistance, causing “warm shock–like” hypotension similar to sepsis.

2. Reduced oncotic pressure

Liver injury impairs albumin synthesis, reducing plasma volume and promoting hypotension.

3. Autonomic dysfunction

Hepatitis may alter sympathetic–parasympathetic balance, contributing to unstable BP.

4. Adrenal suppression

Severe hepatitis reduces cortisol synthesis, leading to reduced vascular tone and BP.

Hypertension Mechanism

Although rare, some hepatitis patients develop hypertension due to:

- Cholestasis
- Pain or anxiety
- Sympathetic nervous system activation
- RAAS system overactivity

These findings emphasize the need for regular BP monitoring in hepatitis patients to prevent complications such as syncope, renal hypoperfusion, and hepatic shock.

Conclusion

Arterial blood pressure changes are clinically important indicators of disease severity in viral hepatitis. Hypotension is more prevalent and correlates with inflammatory activity, hypoalbuminemia, and vasodilatory mechanisms. Hypertension occurs in specific cholestatic or autonomic states. Monitoring BP is essential for timely intervention, fluid management, and improved outcomes in hepatitis patients.

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