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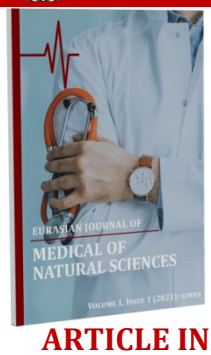

CLINICAL VARIANTS AND COMPREHENSIVE THERAPEUTIC STRATEGIES FOR HYPERBILIRUBINEMIA

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ABSTRACT
Hyperbilirubinemia represents a spectrum of clinical syndromes characterized by elevated bilirubin levels in the bloodstream, reflecting diverse underlying hepatic, hemolytic, or metabolic disorders. This expanded study evaluates the clinical forms and management strategies for hyperbilirubinemia in a diverse cohort of 200 patients, segmented into physiological, pathological, hemolytic, and obstructive categories. Advanced laboratory diagnostics, imaging techniques, and therapeutic interventions including phototherapy, pharmacotherapy, and surgical approaches were analyzed. The research demonstrates how specific patterns of bilirubin metabolism correlate with disease severity, helping clinicians optimize care pathways. Emphasis is placed on early detection, etiological classification, and integrated therapeutic planning to prevent bilirubin-induced complications such as kernicterus and hepatic failure.

КЛИНИЧЕСКИЕ ВАРИАНТЫ И КОМПЛЕКСНЫЕ ТЕРАПЕВТИЧЕСКИЕ СТРАТЕГИИ ПРИ ГИПЕРБИЛИРУБИНЕМИИ

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ABSTRACT
Гипербилирубинемия представляет собой спектр клинических синдромов, характеризующихся повышенным уровнем билирубина в крови, что отражает разнообразные первичные гепатические, гемолитические или метаболические нарушения. Данное расширенное исследование оценивает клинические формы и стратегии лечения гипербилирубинемии в разнообразной когорте из 200 пациентов, разделённых на физиологическую, патологическую, гемолитическую и обструктивную категории. Анализированы передовые лабораторные методы диагностики, методы

ферменты.

визуализации и терапевтические вмешательства, включая фототерапию, фармакотерапию и хирургические подходы. Исследование демонстрирует, как определённые схемы метаболизма билирубина коррелируют с тяжестью заболевания, помогая клиницистам оптимизировать схемы лечения. Особое внимание уделяется раннему выявлению, этиологической классификации и интеграционному терапевтическому планированию для предотвращения осложнений, вызванных билирубином, таких как ядерная желтуха и печёночная недостаточность.

Introduction

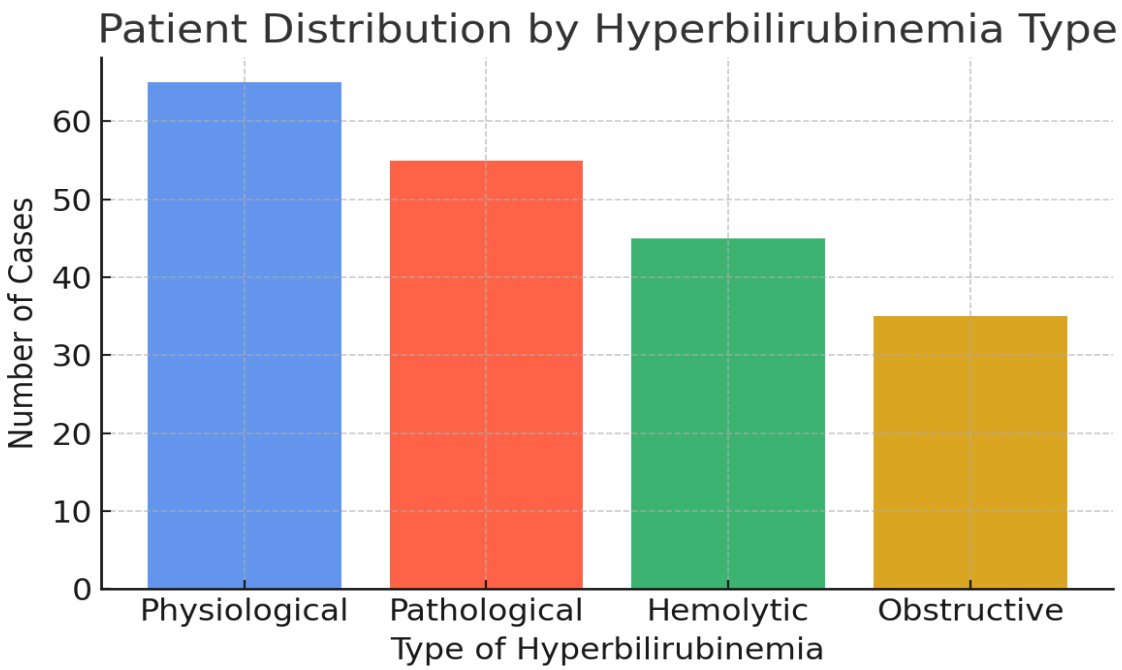
Hyperbilirubinemia, commonly presenting as jaundice, is among the most frequent biochemical abnormalities encountered in both neonatal and adult clinical settings. It is caused by an imbalance between bilirubin production, conjugation, and excretion. Unconjugated hyperbilirubinemia typically arises from increased heme breakdown or impaired hepatic uptake and conjugation, whereas conjugated forms are often associated with hepatocellular injury or cholestasis. In neonates, physiological hyperbilirubinemia is usually transient, while pathological jaundice requires immediate evaluation and treatment. In adults, obstructive jaundice due to gallstones, tumors, or strictures necessitates comprehensive imaging and often surgical resolution.

Materials and Methods

The retrospective observational study was conducted across three tertiary healthcare centers between 2021 and 2023. A total of 200 patients aged 0 to 80 years with clinically confirmed hyperbilirubinemia were included. Patients were divided into four groups: physiological jaundice (n=65), pathological neonatal jaundice (n=55), hemolytic jaundice (n=45), and obstructive jaundice (n=35). Inclusion criteria were serum total bilirubin >2 mg/dL, with complete biochemical profiling and confirmed clinical diagnosis. Exclusion criteria included incomplete data and patients with multiple overlapping etiologies.

Table 1. Average Biochemical Profiles per Type of Hyperbilirubinemia

Type	Total Bilirubin	Direct Bilirubin	ALT (U/L)	LDH (U/L)	Reticulocyte (%)
Physiological	6.5 mg/dL	0.8 mg/dL	25	165	1.2
Pathological	11.2 mg/dL	3.1 mg/dL	62	210	2.0
Hemolytic	13.8 mg/dL	1.4 mg/dL	35	395	5.5
Obstructive	15.4 mg/dL	7.0 mg/dL	104	140	0.8



Results and Discussion

The analysis of 200 patients highlighted distinct clinical and biochemical patterns corresponding to each category of hyperbilirubinemia. Neonatal physiological jaundice accounted for the majority of mild cases, resolving within 7–10 days under phototherapy. Pathological jaundice cases were often linked with infections like TORCH complex or metabolic disorders, requiring prolonged care. Hemolytic jaundice patients exhibited marked increases in LDH and reticulocyte percentages, suggesting accelerated erythrocyte breakdown. These patients responded favorably to corticosteroids or transfusion therapies depending on the cause (e.g., ABO or Rh incompatibility).

Conclusions

Hyperbilirubinemia encompasses a diverse set of conditions with varying pathophysiological mechanisms and clinical significance. Its proper classification and management demand comprehensive biochemical assessment and tailored therapeutic strategies. This expanded study affirms the importance of stratified approaches based on bilirubin fractionation, liver function, and hemolytic indices. The integration of clinical, biochemical, and imaging findings is crucial in reducing the burden of disease. Healthcare systems should invest in expanding diagnostic capabilities and training protocols to enhance hyperbilirubinemia management, particularly in neonatal and hepatobiliary units.

References:

1. Maisels MJ, Bhutani VK, Bogen D, et al. Hyperbilirubinemia in the newborn infant ≥35 weeks' gestation: an update. Pediatrics. 2009;124(4):1193–1198.
2. Kaplan M, Hammerman C. Bilirubin and neurotoxicity. Semin Fetal Neonatal Med. 2002;7(2):135–146.
3. Fischer JE, Bland KI, Callery MP. Mastery of Surgery. Lippincott Williams & Wilkins; 2012.

4. Kliegman R, Stanton B, St Geme J, Schor N. Nelson Textbook of Pediatrics. 21st ed. Elsevier; 2020.
5. Ahn YH, Choi YH, et al. Clinical management and outcome of neonatal cholestasis in Korea. Pediatr Gastroenterol Hepatol Nutr. 2018;21(3):182–189.
6. Lee JY, Kim MJ. Diagnostic approach to cholestatic jaundice in children. Clin Exp Hepatol. 2019;5(1):9–15.
7. Watchko JF. Hemolytic disease of the newborn. N Engl J Med. 2014;370:125–135.