

# Hepatobiliary System Involvement in Neonatal Sepsis: A Clinical and Morphological Study

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**Annotation:** Neonatal sepsis remains a leading cause of mortality in the neonatal period and is frequently accompanied by multiple organ dysfunction. The hepatobiliary system plays a crucial role in metabolic regulation, immune response, and detoxification; therefore, its involvement reflects the severity and systemic nature of septic injury. However, clinical and morphological characteristics of hepatobiliary damage in neonatal sepsis have not been sufficiently systematized.

**Keywords:** newborns, sepsis, liver, cholestasis, hepatobiliary system, pathomorphology.

## INTRODUCTION

Neonatal sepsis remains one of the leading causes of morbidity and mortality worldwide, particularly among preterm and low birth weight infants, despite significant advances in neonatal intensive care and antimicrobial therapy [1,2,3]. The high incidence of fatal outcomes is largely associated with the rapid development of systemic inflammatory response syndrome and multiple organ dysfunction, which determine the severity and prognosis of the disease [5,6]. The liver plays a central role in metabolic homeostasis, immune regulation, synthesis of acute-phase proteins, and detoxification processes. In neonatal sepsis, the hepatobiliary system becomes one of the primary target organs affected by hypoxia, endotoxemia, cytokine-mediated injury, and microcirculatory disorders [7,8]. Hepatic dysfunction in septic newborns not only reflects the severity of systemic inflammation but also contributes to the progression of multiple organ failure and adverse outcomes [9]. Morphological studies have demonstrated that liver involvement in sepsis is characterized by a wide spectrum of pathological changes, including hepatocellular dystrophy, intrahepatic cholestasis, sinusoidal congestion, inflammatory infiltration, and necrotic lesions [4]. In newborns, these alterations are often more pronounced

due to morphofunctional immaturity of hepatic tissue, underdeveloped enzymatic systems, and insufficient adaptive responses to hypoxic and inflammatory stress[ 10].

Intrahepatic cholestasis is considered one of the most characteristic manifestations of septic liver injury in the neonatal period. It develops as a result of impaired bile formation and excretion caused by inflammatory mediators, endothelial dysfunction, and microvascular thrombosis[11,12].The presence of cholestatic syndrome in neonatal sepsis has been associated with prolonged critical illness, increased risk of complications, and unfavorable prognosis. Despite the recognized clinical importance of hepatic involvement in neonatal sepsis, clinical and morphological correlations of hepatobiliary system lesions remain insufficiently systematized, especially in autopsy-based studies. Most available publications focus on clinical and biochemical markers of liver dysfunction, while detailed morphopathological analysis of liver and biliary tract lesions in fatal cases of neonatal sepsis is limited [ 13,14]. This gap restricts the understanding of tanatogenesis and reduces the diagnostic value of postmortem examinations. Therefore, a comprehensive clinical and morphological assessment of hepatobiliary system lesions in newborns who died from sepsis is highly relevant. Such an approach allows for a deeper understanding of pathogenetic mechanisms of hepatic injury, improves the accuracy of postmortem diagnosis, and contributes to the identification of prognostic markers of severe septic course. Moreover, the obtained data may serve as a scientific basis for early clinical and laboratory monitoring of liver function in high-risk newborns and for the development of preventive and therapeutic strategies aimed at reducing neonatal mortality.

**Objective.** To analyze the clinical and morphological characteristics of hepatobiliary system lesions in newborns who died from sepsis.

**MATERIALS AND METHODS.** A retrospective clinical and morphological study was conducted on 46 autopsy cases of newborns who died from sepsis. Clinical and anamnestic data, laboratory findings, and intensive care parameters were analyzed. Macroscopic and histological examinations of the liver and biliary system were performed using standard histological techniques with hematoxylin and eosin staining. The frequency and distribution of morphological changes were evaluated and correlated with clinical indicators of disease severity.

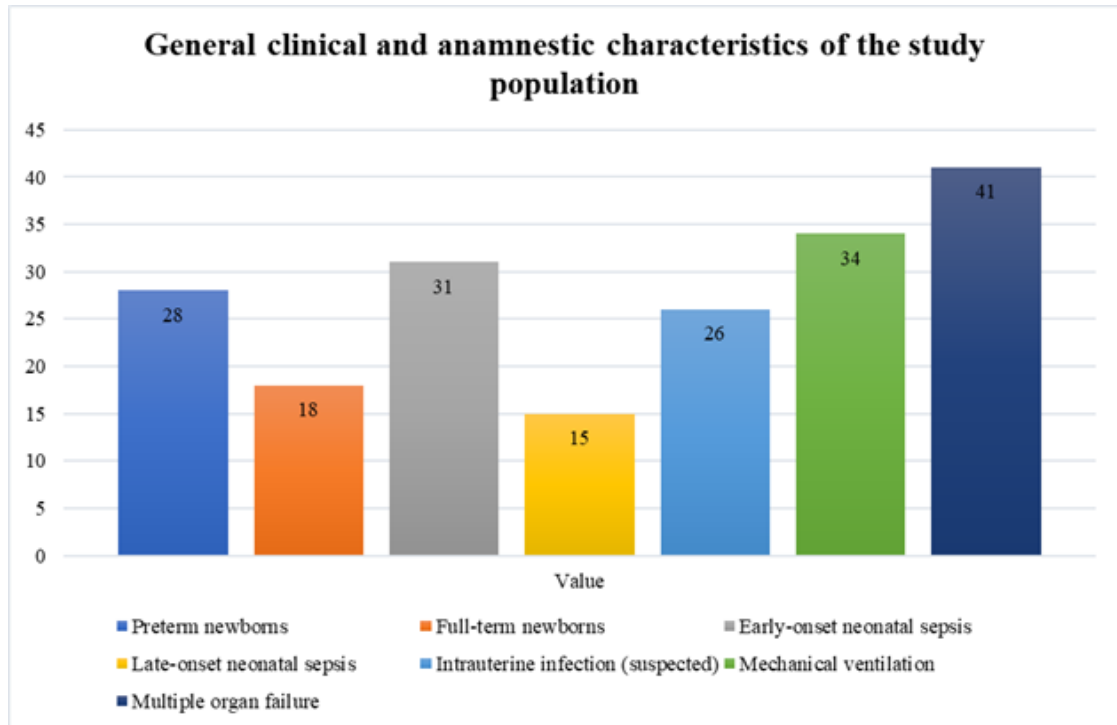
## RESULTS

The study included 46 newborns who died from sepsis, with gestational age ranging from 24 to 41 weeks. The majority of cases were preterm infants, accounting for 28 cases (60.9%), while full-term newborns comprised 18 cases (39.1%), indicating a predominance of septic fatal outcomes among infants with morphofunctional immaturity of organs and systems. According to the timing of infection onset, early-onset neonatal sepsis predominated and was diagnosed in 31 newborns (67.4%), highlighting the significant role of antepartum and intrapartum factors in the development of the septic process. Late-onset neonatal sepsis was observed in 15 cases (32.6%) and typically developed following prolonged stay in the neonatal intensive care unit. Signs of intrauterine infection or clinical suspicion thereof were identified in 26 newborns (56.5%), confirming the high frequency of transplacental and ascending infection as one of the leading pathogenetic factors of neonatal sepsis. Mechanical ventilation was required in 34 infants (73.9%), reflecting the severity of the clinical condition and the presence of pronounced respiratory failure characteristic of a generalized infectious and inflammatory process in the neonatal period. Multiple organ failure was diagnosed in the vast majority of newborns-in 41 cases (89.1%)-indicating the systemic nature of sepsis and confirming its leading role in the tanatogenesis of fatal outcomes.

**Table 1. General clinical and anamnestic characteristics of the study population (n = 46)**

No.	Indicator	Value
1.	Gestational age	24–41 weeks
2.	Preterm newborns	28 (60.9%)

3.	<b>Full-term newborns</b>	<b>18 (39.1%)</b>
4.	<b>Early-onset neonatal sepsis</b>	<b>31 (67.4%)</b>
5.	<b>Late-onset neonatal sepsis</b>	<b>15 (32.6%)</b>
6.	<b>Intrauterine infection (suspected)</b>	<b>26 (56.5%)</b>
7.	<b>Mechanical ventilation</b>	<b>34 (73.9%)</b>
8.	<b>Multiple organ failure</b>	<b>41 (89.1%)</b>

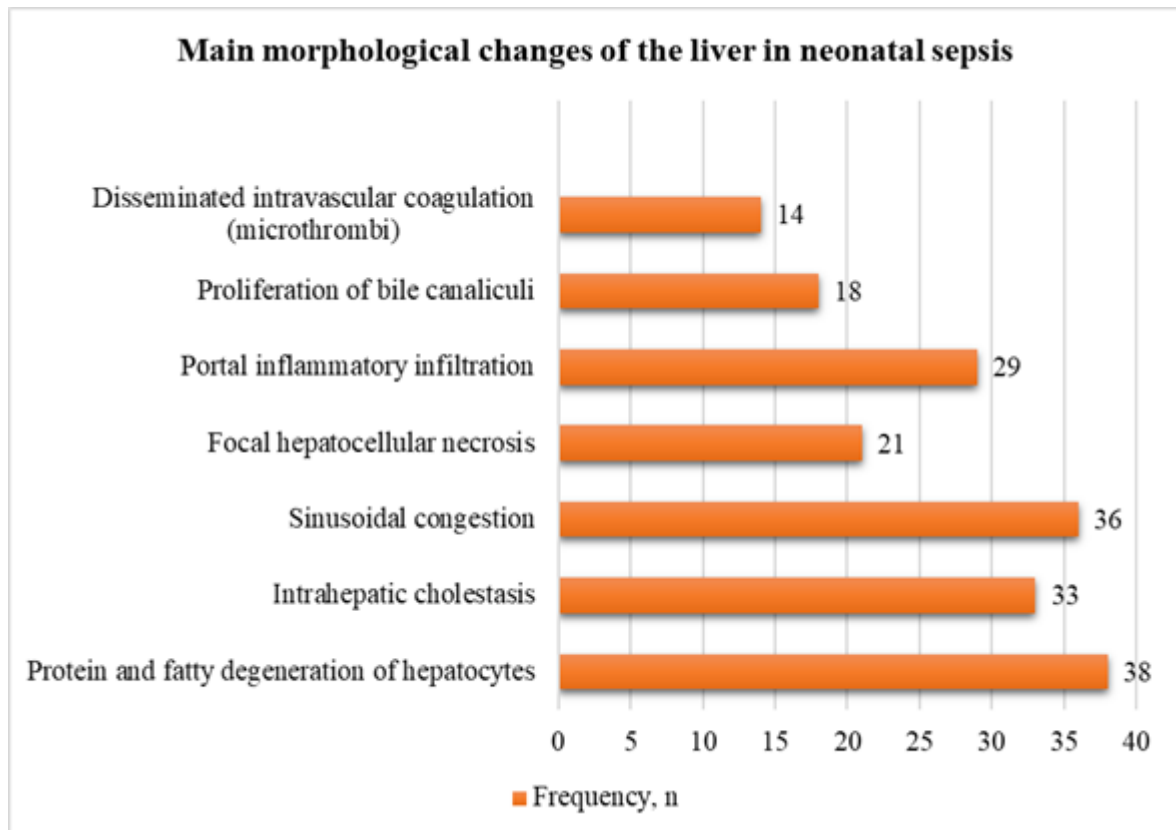


The high frequency of multiple organ dysfunction underscores the critical extent of involvement of vital organs, including the hepatobiliary system, in the pathological process. In addition, autopsy liver tissue samples were examined microscopically during postmortem examination in order to clarify and confirm the diagnosis of sepsis.

Histological examination of liver tissue revealed a high prevalence of dystrophic, cholestatic, inflammatory, and microcirculatory alterations. Protein and fatty degeneration of hepatocytes was the most frequent morphological finding, observed in 38 cases (82.6%), indicating severe metabolic impairment of hepatic parenchyma under septic conditions. Sinusoidal congestion was identified in 36 cases (78.3%) and was characterized by marked dilatation and blood stasis within hepatic sinusoids, reflecting pronounced disturbances of intrahepatic microcirculation. Intrahepatic cholestasis was detected in 33 newborns (71.7%), manifested by the accumulation of bile pigments in hepatocytes and bile canaliculi, which suggests functional insufficiency of bile excretion associated with septic injury. Portal inflammatory infiltration was observed in 29 cases (63.0%) and consisted predominantly of lymphocytes and macrophages, indicating activation of inflammatory and immune responses within portal tracts. Focal hepatocellular necrosis was identified in 21 cases (45.7%), reflecting irreversible cellular damage caused by hypoxia, endotoxemia, and inflammatory mediators. Proliferation of bile canaliculi was noted in 18 cases (39.1%) and represented a reactive compensatory response to cholestatic changes and biliary obstruction. Signs of disseminated intravascular coagulation, manifested by the presence of fibrin microthrombi within hepatic microvessels, were detected in 14 cases (30.4%), confirming the involvement of coagulation disorders in the pathogenesis of hepatic damage in neonatal sepsis. Overall, the observed morphological changes demonstrate that liver involvement in neonatal sepsis is characterized by a combination of parenchymal degeneration, cholestasis, inflammatory infiltration, necrosis, and microvascular thrombosis, reflecting the systemic nature of septic injury and its role in the development of multiple organ failure (Table 2).

**Table 2. Main morphological changes of the liver in neonatal sepsis (n = 46)**

No.	Morphological feature	Frequency, n (%)
1.	Protein and fatty degeneration of hepatocytes	38 (82,6 %)
2.	Intrahepatic cholestasis	33 (71,7 %)
3.	Sinusoidal congestion	36 (78,3 %)
4.	Focal hepatocellular necrosis	21 (45,7 %)
5.	Portal inflammatory infiltration	29 (63,0 %)
6.	Proliferation of bile canaliculi	18 (39,1 %)
7.	Disseminated intravascular coagulation (microthrombi)	14 (30,4 %)



## CONCLUSION

In conclusion hepatobiliary system lesions in neonatal sepsis are polymorphic in nature and include cholestatic, dystrophic, inflammatory, and necrotic changes. The severity of morphological alterations correlated with the duration of sepsis, gestational age, and the degree of multiple organ failure. The identified changes confirm that hepatobiliary system involvement in neonatal sepsis results from the combined effects of hypoxia, endotoxemia, and systemic inflammation. Cholestatic syndrome in newborns is predominantly intrahepatic in origin and correlates with the severity of sepsis and the duration of the critical condition. The obtained data are consistent with current concepts regarding the liver as a target organ in multiple organ dysfunction syndrome and emphasize the necessity of early clinical and laboratory assessment of hepatobiliary system function in high-risk newborns.

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