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# Impact of Bacterial Blood Infections on Inflammatory Markers and Liver Function in Neonates with Pathological Jaundice in Erbil City

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## ABSTRACT

Blood infections can shift how the body works and make things get worse, turning a baby's yellow skin into a big health risk. The Neonatal Intensive Care Unit at Erbil Hospital's Department of Obstetrics and Gynecology treated 125 neonatal samples between January 2022 and June 2023. 50 neonatal samples with jaundice but no bacterial infection and 75 neonatal samples with jaundice and a confirmed bacterial blood infection (septicemia) made up the two groups of participants. Forty-three samples from a control group of healthy newborns were also included. Blood testing included WBC, hs-CRP, PCT, TRF, and liver enzymes: AST, GGT, and ALP. The showings were that WBC, hs-CRP, and PCT levels were high in babies with jaundice and septicemia, showing a big body fight response ($P < 0.05$). Babies with bacterial bugs had much lower TRF levels ($P < 0.01$) than those without, showing that the body fights changes in iron use in the body. Jaundiced neonates had higher levels of AST, ALP, and GGT according to liver enzyme testing; these values were more noticeable in those who also had blood infections, suggesting possible liver involvement. Babies with jaundice and bacterial infection had much higher white blood cell (WBC), hs-CRP, and PCT levels ($P < 0.05$). They also had lower transferrin (TRF) levels ($P < 0.01$). This shows body-wide infection and changes in how the body handles iron. Levels were liver enzymes, levels as jaundice groups: AST, GGT, ALP, and GUT, were high in both groups with jaundice, more so than in the control group.

**KEYWORDS**: Neonatal jaundice, septicemia, liver enzymes, inflammatory markers, newborn care.

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## 1. Introduction

Physiological jaundice is a usual thing, often seen on the baby's second or third day. It is the top reason for yellow skin on the first day after birth [1]. Characterized by yellowing of the skin and the whites of the eyes, representing about 50% of cases. However, only about 10% of newborns require phototherapy to treat jaundice. If the jaundice is pathological, it may appear on the first day of life, with total serum bilirubin (TSB) levels exceeding age-specific bilirubin charts or with a rapid increase in bilirubin levels. In certain instances, jaundice can last longer than three weeks, particularly in infants who are breastfed [2, 3]. The main reasons for baby jaundice can include biliary atresia; there may be other causes, issues with the child's nutrition,

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hydration, or erythrocyte lifetime [4]. Imaging examinations are essential for differentiating among various conditions affecting the extrahepatic bile ducts, including biliary atresia. This study attempts to use state-of-the-art diagnostic imaging techniques to identify various diseases and present some valuable information on the distinctive abnormalities of cholestasis in infants [5]. Mild jaundice in infants usually does not raise alarm; however, concerning congenital jaundice that is either severe or lasts too long can lead to a certain diagnosis and may result in intricate complications. There exists a relationship between congenital jaundice and systemic inflammation, which can cause such health complications [6]. Complex connections between congenital jaundice and blood inflammation can lead to results in high bilirubin levels, which can increase jaundice. Additionally, it can be observed that during inflammation, both acute and chronic, the body's inflammatory response alters the functioning of some liver enzymes required for bilirubin metabolism and numerous other vital elements of the blood [7]. Jaundice shows signs of many health woes, like blood cell breakdown, liver sickness, and some bugs. These problems are often linked with changes in certain blood counts and liver fluids [8, 9]. These findings point out that there is a link between blood swelling, shifts in liver enzymes and blood traits, and jaundice from causes other than liver causes. But a lot of parts of this link and its effects are still a puzzle [10]. Jaundice is considered an important contributor to the disease and mortality rate between women and infants worldwide, which affects 3–5% of pregnancies, especially in some regions in South Asia. During pregnancy, women experience different hormonal and physical changes [11, 12]. Blood infections with jaundice can occur and potentially lead to acute fatty liver conditions during pregnancy. Moreover, jaundice can be tied to many health issues, such as portal high blood pressure, viral liver disease, pregnancy, HELLP syndrome, being with a child with liver bile stop, and liver scarring with heavy sickness while with a child. It is key to note that some drugs used with a child can cause jaundice too [13]. Hepatitis in small kids can come from many viruses. These include cytomegalovirus (CMV), rubella, and the hepatitis viruses A, B, and C. Sometimes, we don't know which virus is to blame. Also, a not-so-common sickness called galactosemia happens when a newborn can't break down the milk sugar, galactose. This issue might cause jaundice, liver infection, and harm [14]. Jaundice may show up if there is too much bilirubin in the liver. This can happen if there's a block or the bile duct isn't made right due to biliary atresia. One clear sign of this is when a baby has very pale poop [15]. The study then intends to enhance knowledge about the impacts of congenital jaundice as well as systemic inflammation in terms of the blood markers, biomarkers, and enzymes of the liver. We are sure that finding out such deep works will give big thought into the ill health links with new jaundice and tied issues and may lead to better ways to find and treat them.

## 2. Materials and Methods

### 2.1. Study Patients:

The research involved 125 infants diagnosed with neonatal pathological jaundice and was conducted from January 2022 to June 2023. It was a study conducted in the Neonatal Intensive Care Unit of the Obstetrics and Gynecology Department at Erbil Hospital. The patients were split into two groups: 75 cases with proven bacterial blood inflammation based on hospital laboratory data and 50 cases with jaundice but no bacterial infection. Also, we had 43 healthy babies as a control group. Their families agreed by signing papers, and the hospital's ethics group said yes to the study. They made sure that everyone joined in of their own will.

### 2.2. Sample collection:

Fasting venous blood samples were obtained from each infant patient in the early morning after admission. The collected blood was placed in K2-

EDTA anticoagulant tubes for routine blood analysis. Serum samples were acquired by centrifuging self-coagulated venous blood at 3000 revolutions per minute [16] and were utilized for assays such as high-sensitivity C-reactive protein (hs-CRP), procalcitonin (PCT), white blood cells (WBC), transferrin (TRF), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP). Routine blood testing was performed by using a fully automated hematology analyzer, the SYSMEX XS-1000i. PCT and TRF tests were performed on a fully automated Roche E601 analyzer, which employs the electrochemiluminescence immunoassay method, was utilized. Tests for hs-CRP, TRF, and liver enzyme tests for ALT, AST, GGT, and ALP were conducted using a fully automated biochemistry analyzer (Siemens ADVIA 2400). Original reagents provided with the instruments were utilized, and operational protocols were strictly adhered to. Quality control measures met all specified requirements. The reference ranges for the tested markers were as follows: WBC, (15.00 to 20.00) x10^9; hs-CRP, (0.00 to 5.00) mg/L; PCT, (0.00 to 0.25) ng/mL; TRF, (28.60 to 51.90) micrometers; AST, (0.00 to 50.00) U/L; GGT, (0.00 to 50.00) U/L; ALP, (36.00 to 150.00) U/L [17, 18].

### 2.3. Statistical analysis

The work on data processing and checking it all out was done with SPSS 22.0 (from Chicago, IL, USA). We did the t-test and $\chi^2$ test for the stats work. All the numbers we got were shown as an average with the usual ups and downs (mean ±s). To see how different groups matched up, an own t-test for just that group was used, and the $\chi 2$ test helped see differences in counts. We also did the Pearson test to see how things were linked to one another. A result was seen as good if the p-value was under 0.05.

## 3. RESULTS AND DISCUSSION

The results presented in Table 1 compare the levels of inflammation markers (WBC, TRF, hs-CRP, and PCT) in the peripheral blood samples among three groups: patients with neonatal pathological jaundice associated with bacterial pathogenic infection, patients with jaundice without blood inflammation, and healthy infants. The info shows that babies with jaundice and blood infection had much higher marker levels than both the only-jaundice group and the well babies. The change was big enough to matter in tests ($P < 0.05$). Also, babies with bugs in them had way lower TRF levels than those with jaundice but no bugs ($P < 0.01$). This study shows how blood swelling affects many blood cells in babies with jaundice. The yellow-skinned babies who also had blood swelling showed high levels of hs-CRP, WBC, and PCT. This hints at a still-active swell, which might make the jaundice worse. On the other hand, the low TRF levels seen in the group with a bacterial infection might be linked to abnormal iron use, often seen with swelling issues. These points show how key it is to check swelling signs in babies with jaundice to look for possible complications and make good care plans. The data in **Table 1** show big changes in blood details among three sets of people: those with jaundice who also have a blood infection, those with just jaundice, and a group with no infections. All these changes are key, with P values less than 0.05. The numbers we looked at are white blood cell count (WBC) shown in 10^9/L, procalcitonin (PCT) in ng/mL, high-sensitivity C-reactive protein (hs-CRP) in mg/L, and total bilirubin (TRF) in μm. It seems there's a tie between high WBC counts and the chance of getting blood infections and jaundice. The group with both problems had much higher WBC counts than those who just had jaundice or were in the control group. A severe inflammatory response associated with jaundice is also suggested by the significantly higher hs-CRP readings in the group with jaundice and blood infection. Likewise, PCT levels, which are usually suggestive of bacterial infections, are noticeably elevated in this group. On the other hand, TRF levels peak in the group with jaundice but no infection. This fits with how jaundice often shows

up with high bilirubin levels caused by a sick liver. The shown P values (0.014, 0.001, 0.004, 0.004) point out how big the statistical significance of the changes in WBC, hs-CRP, PCT, and TRF levels is across the groups. These low P values suggest that the observed differences are highly unlikely to be due to chance, illustrating distinct patterns in blood parameters between the groups, which emphasize the impact of blood infection on inflammatory markers and the effect of jaundice on bilirubin levels.

Table 1. The impact of neonatal jaundice and blood inflammation on certain blood parameters

| Group | Transferrin TRF (µM) | White Blood cells WBC (x109/L) | high-sensitivity C-reactive protein hs-CRP (mg/L) | procalcitonin PCT (ng/mL) |
|---|---|---|---|---|
| **Jaundice and blood infection** | 11.73±1.03[a] | 26.03±3.01[a] | 18.43±1.32[a] | 5.39±2.54[a] |
| **Jaundice without blood infection** | 40.56±3.65[b] | 12.11±2.71[b] | 4.86±0.55b[c] | 0.42±0.54[b] |
| **Control** | 4.76±3.76[c] | 4.042c | 4.64[c] | 5.08c[a] |
| **P- Value** | 0.004 | 0.014 | 0.001 | 0.004 |

[a,b,c] $P<0.05$, compared with the control group

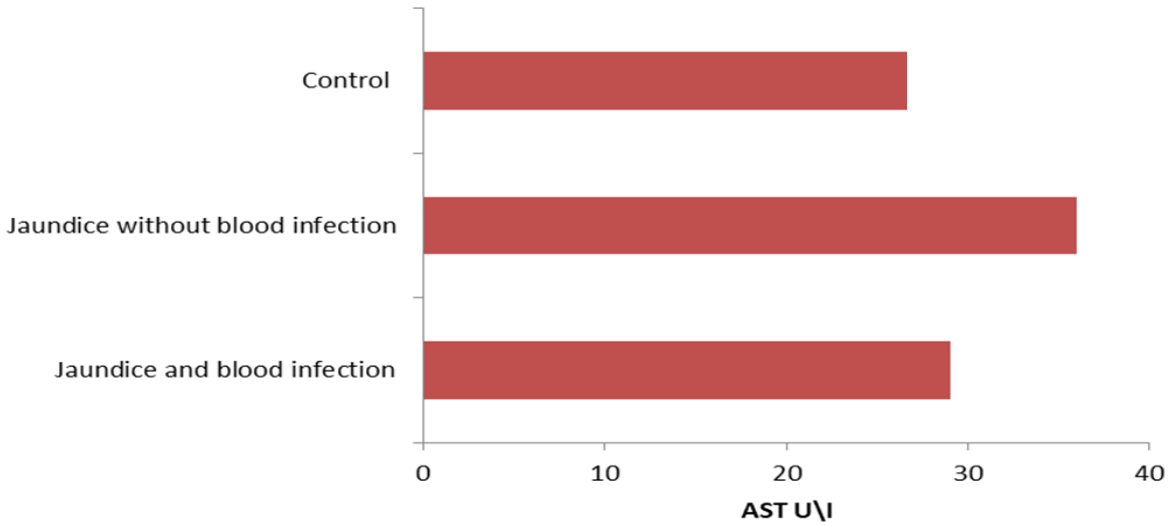


Figure 1: The effect of congenital jaundice and blood inflammation on the enzyme Aspartate aminotransferase (AST), LSD 5%=1.204.

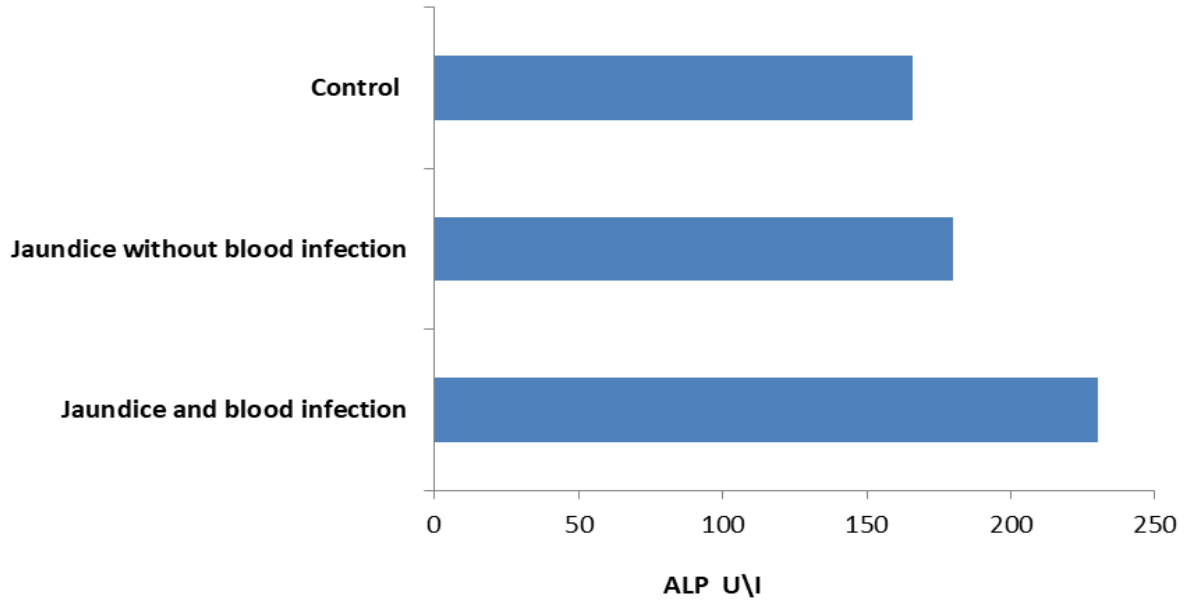


Figure 2: The effect of jaundice and blood inflammation on the enzyme Alkaline phosphatase(ALP), LSD 5%=2.650.


Figure 3: The effect of jaundice and blood inflammation on the levels of The Enzyme Gamma-Glutamyl transferase peptidase (GGT), LSD 5%=3.650.

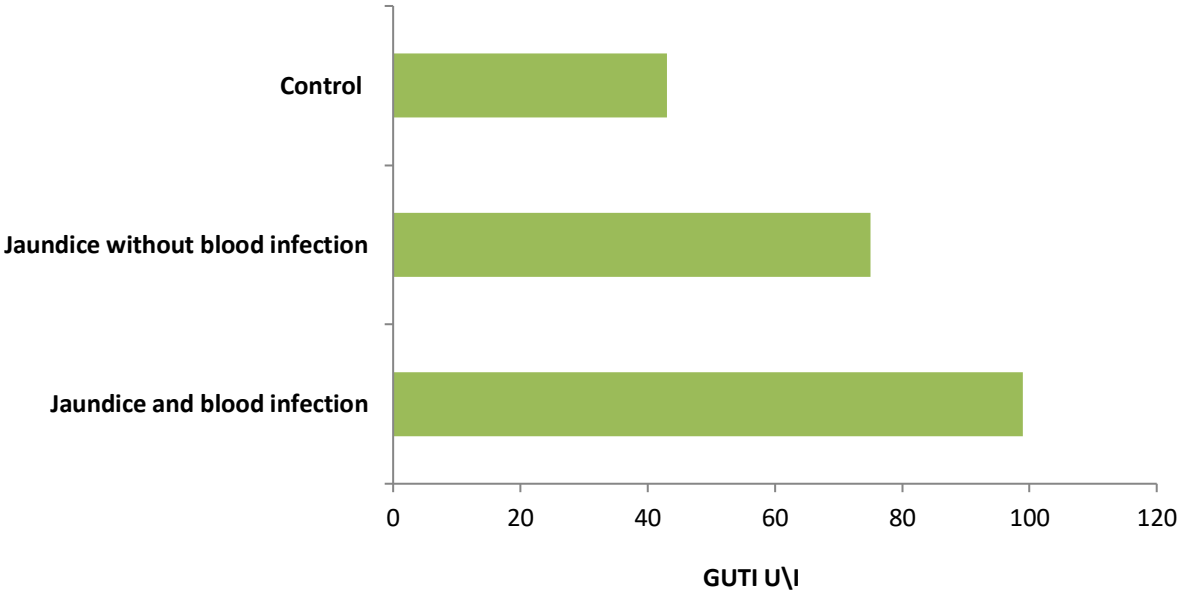


Figure 4: The effect of jaundice and blood inflammation on the levels of the glucuronosyl transferase enzyme (GUTI), LSD 5%=2,976.

### 3.1. The effect of jaundice and blood inflammation on liver enzymes

The results depicted in Figure 1 demonstrate the effect of neonatal jaundice, either concurrent with blood inflammation or without, in comparison to healthy infants, on the liver enzyme aspartate aminotransferase (AST).

The results are shown in Figures 1, 2, 3, and 4, which present the liver enzyme levels (AST, ALP, GGT, and GUTI) for cases of jaundice with or without blood inflammation and the control group of healthy infants. Compared to the control group, the comparison aims to understand the relationship between liver enzymes and jaundice, with or without blood inflammation. The AST levels are relatively higher in both the jaundice with blood infection group and the jaundice without blood infection group compared to the control group. This suggests potential liver injury or stress in both conditions associated with jaundice, with the highest level being 36 units/L (jaundice without blood infection) and the lowest being 29 units/L (jaundice with blood infection). ALP levels are significantly greater in both jaundice groups than in the control group; they peak at 230 units/L in cases of inflammation-accompanied jaundice and 180 units/L in cases of non-inflammatory jaundice. The liver and bile ducts can play a part in jaundice, as high ALP levels often link with liver and bile duct issues. Like this, both groups with jaundice have higher GGT levels than the group with no jaundice; for jaundice with redness, the level is 155 units/L, and for jaundice without redness, it is 120 units/L. The possible involvement of the liver and biliary system in jaundice is further supported by the fact that elevated GGT levels are frequently linked to liver and bile duct disorders. The research indicates that individuals with jaundice absence of blood inflammation, exhibited glucuronosyl transferase enzyme levels at 75 units/L, whereas those with inflammation showed elevated levels of 99 units/L/L. The likelihood of liver and biliary system involvement in jaundice rises with the increased levels of AST, ALP, and GGT observed in both groups when contrasted with the control group. This big jump in enzyme levels, way above the normal range, might mean a blood infection is helping jaundice start. These facts point out how the biliary

system can mess up. They also make clear the strong link between jaundice and problems with liver function. This study's results match well with those from Mutar *et al*. (2025). They found that high liver enzyme levels point to poor liver function because of disorders and infection in the liver [19]. Neonatal congenital jaundice usually comes from high amounts of bilirubin, a yellow color that comes from hemoglobin, crucial for moving oxygen in red blood cells. When these cells fall apart, the body makes new ones to replace them. The liver is key in dealing with the old cells, but if it can't work right, bilirubin can go up in the system, making the skin and the white parts of the eyes turn yellow [20]. Jaundice can show many signs of health issues that cause blood problems or swelling. This leads to high levels of bilirubin. The condition happens when the liver can't deal with all the bilirubin by mixing it with glucuronic acid [21, 22]. Additionally, the counting of white blood cells is frequently used as a basic bacterial infection detection during a routine blood test. These cells contribute to the immune response of the body [23]. In clinical tests, they are especially relevant for assessing infections in neonates, partly to determine the seriousness of the illness and partly to assess the state of the immune response. Another important inflammation marker is the C-reactive protein (CRP), which the liver produces during acute inflammatory processes. While nearly all healthy individuals experience low levels of CRP, it is very often measured in clinical cases to follow the development of bacterial infection alongside jaundice since both aid in the diagnosis of infant illnesses [24,25]. Because of its ability to pick up on very low CRP concentrations, high-sensitivity (hs-CRP) represents one of the most advanced tools for detecting subtle infections [26]. Hs-CRP is acknowledged as a product of gene expression with a sensitive response in inflammation reaction, exerting a more rapid increase in its levels, therefore often preceding any visible clinical signs such as fever [27]. Patients who have jaundice caused by bacterial bugs show much higher levels of hs-CRP. This fact makes hs-CRP a key sign for spotting bacterial bugs. Also, in looking after very young babies, testing hs-CRP is a main way to tell if an infection is from a bacteria or a virus. This helps in correctly finding out if a newborn's jaundice comes from bacterial reasons [28].

The way bilirubin is broken down needs liver-helping stuff. A key bit in this is a thing called uridine diphosphate glucuronosyltransferase (UGT). It helps make bilirubin easier to mix with other stuff. In small kids, this bit does not work the same as in big people. This can make them get jaundice [29]. Jaundice may show up when there are problems that stop the liver from working as it should. This could happen because of liver sickness or harm. These troubles can change liver enzyme amounts and mess up how bilirubin is dealt with [30]. In children suffering from severe illnesses, jaundice can be associated with blood infections. Therefore, hyperbilirubinemia connected to sepsis becomes a crucial consideration for patients with serious health issues who exhibit notable increases in bilirubin levels [31]. In a similar study, the group with bacterial infections showed significantly elevated levels of White Blood Cells (WBC), high-sensitivity C-reactive Protein (hs-CRP), and Procalcitonin (PCT) compared to the non-infected group, while Transferrin (TRF) levels were lower in the infected individuals [32]. There are no hard rules for finding cholestasis from sepsis; doctors work this out by checking the person's past health, what they find in checks, and lab test outcomes. People with cholestasis due to sepsis usually show signs like jaundice and other signs tied to infection, but sometimes jaundice shows up alone, with no other signs [33]. It is not very common for these patients to have itching and stomach pain. Hepatomegaly might be seen, and transaminase and serum alkaline phosphatase (SAP) are often a bit high. Conjugated bilirubin levels range from 2 to 10 mg/dl. A key point in our case was the sole high levels of conjugated bilirubin, without any increase in transaminases, SAP, or Gamma-Glutamyl Transferase (GGT) [34]. Sepsis-linked cholestasis most often ties to belly infections due to Gram-negative bacteria. Yet, it can also link to staph sepsis

and lung infections. More risks for cholestasis from sepsis are being born too soon, how bad and how long the sepsis lasts, feeding through IV, and other liver diseases. If performed, liver biopsies usually show minimal or no inflammation, prominently featuring intrahepatic cholestasis, though such biopsies are seldom required for diagnosis [35,36]. Currently, there are no specific pharmacological treatments to alter the course of the cholestatic process. Right now, there are no drugs that can change how cholestasis works. So, the main goal of treatment is to get rid of the infection with strong drugs to kill germs and surgery to remove fluid if needed, plus taking care of the patient in other ways. Some drugs can cause cholestasis or harm the liver, so we have to be careful or not use them. These drugs include acetaminophen, drugs called NSAIDs that lower pain and swelling, sodium valproate, and rifampicin. On the side of treatment, there are a few ways that might help, based on stories that they worked. These include enteral nutrition, ursodeoxycholic acid, and glycine. Additionally, agents such as nitric oxide (NO) donors, for example, molsidomine, N-acetyl cysteine, corticosteroids, anti-TNF agents, and methods like extracorporeal liver support have also been cited for their potential benefits in treatment regimens [37,38]. Current Research. Still, there is no set rule to determine if bad liver function due to bad blood flow is for sure. It often leans on past health info, how one feels, and lab tests. People with this issue often show yellow skin and signs of a bad infection. But sometimes, just yellow skin can be the only sign [39]. It is infrequent for these patients to experience itching or abdominal pain. Hepatomegaly might be observed, and levels of transaminases and serum alkaline phosphatase (SAP) tend to be moderately raised. A distinctive finding is conjugated bilirubin levels ranging from 2 to 10 mg/dl. Notably, an unusual feature in certain cases includes elevated conjugated bilirubin without a corresponding rise in transaminases, SAP, or gamma-glutamyl transferase (GGT) [40]. Most times, sepsis-linked cholestasis starts from inside-belly bugs from Gram-negative bacteria. But it can also link with staph infections and lung problems. Other risk points for cholestasis from sepsis include being born too soon, how bad and long the infection is, tube feeding, and other liver health issues. Although liver biopsy might reveal mild to absent inflammation and pronounced intrahepatic cholestasis as key histological features, this procedure is seldom required for diagnosis [26]. Current Treatment of Cholestasis. Right now, no exact drugs exist for cholestasis. It's all about stopping infections using strong drug care. Sometimes, surgery helps too, along with extra help and care. It’s key to stay away from or use with care drugs like acetaminophen, these include enteral nutrition, ursodeoxycholic acid for improving bile flow, glycine for its hepatoprotective properties, nitric oxide donors like molsidomine for vascular modulation, N-acetyl cysteine for oxidative stress reduction, corticosteroids for inflammation control, anti-TNF agents for targeting inflammatory pathways, and extracorporeal liver support systems as a temporary support mechanism for liver function [41,42]. Newborns with severe jaundice caused by blood group incompatibility were studied. Childhood jaundice caused by neonatal hepatitis and biliary atresia was found to be exacerbated by bacterial infection if it lasted for more than a month [43]. Bacterial infections lead to jaundice in different ways. Some studies show more breakdown of red blood cells and liver cell death in bad cases. Antibiotics work well in treating jaundice that comes from bacterial infections, with jaundice often going away within 72 hours after starting the treatment [44].

## CONCLUSION

This study shows how much blood infections affect newborns' yellow skin. It finds high levels of swelling signs (WBC, hs-CRP, PCT) and poor iron control (low TRF levels) in babies with yellow skin and also with germ infections. Raised liver markers (AST, ALP, and GGT) play a big part in liver issues, telling us how bad the strain from the infection is.

These points make it clear that close watch and care of blood levels and liver health in young ones with yellow skin is very important. This study looks at how jaundice and blood infections team up. It shows new ways to find and fix these issues. The aim is to cut risks and improve baby health when dealing with these problems.

## Conflict of interest

There are no conflicting interests, according to the authors.

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## Ethics approval

This study was reviewed and approved by the Research Ethics Committee at the University of Anbar https://www.uoanbar.edu.iq/English/CMS.php?ID=89.

## REFRENCES

1. Young, Hun, Choi., Seul, Bi, Lee., Yeon, Jin, Cho., Seung, Hyun, Lee., Su, Mi, Shin., Jung, Eun, Cheon. (2022). (Jaundice in Young Children). Journal of the Korean Society of Radiology, 83 5(5):966-978. doi: 10.3348/jksr.2022.0091

2. Daniel, Alagille., A., Estrada., Michelle, Hadchouel., M., Gautler., Michel, Odièvre., Dommergues, Jp. (1987). Syndromic paucity of interlobular bile ducts (Alagille syndrome or arteriohepatic dysplasia): Review of 80 cases. The Journal of Pediatrics, 110(2):195-200. doi: 10.1016/S0022-3476(87)80153-1

3. Yanli, L. I. U., Xiuhua, S. U. N., Yaqiong, W. A. N. G., Cuihong, X. I. N. G., Li, L. I., & Shiying, Z. H. O. U. (2021). Evaluation of associated markers of neonatal pathological jaundice due to bacterial infection. Iranian Journal of Public Health, 50(2), 333.

4. Dong T, Chen T, White RA, et al. (2018). Meconium microbiome associates with the development of neonatal jaundice. Clin Transl Gastroenterol, 9(9): 182

5. Jiao Y, Jin Y, Meng H, et al. (2018). An analysis on treatment effect of blue light phototherapy combined with Bifico in treatingneonatal hemolytic jaundice. Exp Ther Med, 16(2): 1360–1364

6. Li Y, Zhong X, Cheng G, et al. (2017). Hs-CRP and all-cause, cardiovascular, and cancer mortality risk: A meta-analysis. Atherosclerosis, 259: 75–82.

7. Chen, L. J., Chen, P. J., Yang, S. F., & Chen, J. Y. (2022). Causative organisms and antimicrobial susceptibility in jaundiced infants with significant bacteriuria. Journal of the Chinese Medical Association, 85(4), 514-518.

8. Hansen, T. W. R. (2021). The epidemiology of neonatal jaundice. Pediatric Medicine, 5(18), 18-18.

9. Pavlovic Markovic, A., Stojkovic Lalosevic, M., Mijac, D. D., Milovanovic, T., Dragasevic, S., Sokic Milutinovic, A., & Krstic, M. N. (2022). Jaundice as a diagnostic and therapeutic problem: A general practitioner's approach. Digestive Diseases, 40(3), 362-369.

Chand N, Sanyal AJ (2007) Sepsis-induced cholestasis. Hepatology 45: 230-241. Link: https://goo.gl/tMx5BD

10. Yu, Y., Choi, J., Lee, M. H., Kim, K., Ryu, H. M., & Han, H. W. (2022). Maternal disease factors associated with neonatal jaundice: a case–control study. BMC Pregnancy and Childbirth, 22(1), 247.

11. Ambalavanan N, Waldernar A. In: Nelson textbook of pediatrics. 19th ed. Kliegman RM, Staton B, Geme J, Schor NF, Behrman RE, editors. Philadelphia: WB Saunders; 2011. Jaundice and hyperbilirubinemia in newborn; p. 603.

12. Harb, A., Yassine, V., Ghssein, G., Salami, A., & Fakih, H. (2023). Prevalence and clinical significance of urinary tract infection among neonates presenting with unexplained

hyperbilirubinemia in Lebanon: a retrospective study. Infection & Chemotherapy, 55(2), 194.

13. Chen CJ, Lo YF, Huang MC, Chung RL, Tang RB, Wu KG. A model for predicting risk of serious bacterial infection in febrile infants younger than 3 months of age. J Chin Med Assoc. 2009; 72:521–526.

14. Chiou FK, Ong C, Phua KB, Chedid F, Kader A. Conjugated hyperbilirubinemia presenting in first fourteen days in term neonates. World J Hepatol. 2017; 9:1108–1114

15. Omar, C., Hamza, S., & Mariam, R. (2011). Urinary tract infection and indirect hyperbilirubinemia in newborns. North American journal of medical sciences, 3(12), 544.

16. Shahian M, Rashtian P, Kalani M. Unexplained neonatal jaundice as an early diagnostic sign of urinary tract infection. Int J Infect Dis. 2012;16: e487–e490

17. Özcan M, Sarici SÜ, Yurdugül Y, Akpinar M, Altun D, Özcan B, Serdar MA, Sarici D. Association between early idiopathic neonatal jaundice and urinary tract infections. Clin Med Insights Pediatr. 2017; 11:1179556517701118.

18. Kemper AR, Newman TB, Slaughter JL, Maisels MJ, Watchko JF, Downs SM, Grout RW, Bundy DG, Stark AR, Bogen DL, Holmes AV, Feldman-Winter LB, Bhutani VK, Brown SR, Maradiaga Panayotti GM, Okechukwu K, Rappo PD, Russell TL. Clinical practice guideline revision: management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2022;150: e2022058859.

19. Mutar, T. F., Jasim, R. A., Noori, S. S., Alrawi, D. D., Mohammed, O. M., & Saeed, B. I. (2025). Evaluation of inflammatory and biochemical indicators in dialysis patients infected with hepatitis B virus Acta Microbiol. Bulg. 2025; 41(02). https://doi.org/10.59393/amb25410207.

20. Anirban, Mandal, Amitabh, Singh. (2016). Isolated Conjugated Hyperbilirubinemia in Seriously Ill Children – Don't Forget Sepsis! 2(1):010-012. doi: 10.17352/2640-7612.000009.

21. Yanli, Liu., Xiuhua, Sun., Yaqiong, Wang., Cuihong, Xing., Li, Li., Shiying, Zhou. (2021). Evaluation of Associated Markers of Neonatal Pathological Jaundice Due to Bacterial Infection. Iranian Journal of Public Health, 50(2):333-340. doi: 10.18502/IJPH.V50I2.5394

22. Ijaz, Ahmed., Musa, Hassan., Kaynat, Khalid., M., Khan. (2022). Seropositivity of Hepatitis B Surface Antigen among Jaundice Children. Pakistan Journal of Medical and Health Sciences, 16(5):928-930. doi: 10.53350/pjmhs22165928

23. Yameny, A. Characteristics of peripheral Leukocyte in moderate infection of COVID-19. *Journal of Bioscience and Applied Research*, 2021; 7(4): 216-222. doi: 10.21608/jbaar.2021.251237

24. Ghenu, M. I., Dragoş, D., Manea, M. M., Ionescu, D., & Negreanu, L. (2022). Pathophysiology of sepsis-induced cholestasis: A review. JGH Open, 6(6), 378-387.

25. Alabd, S., Yameny, A. C-Reactive Protein as a Prognostic Indicator in COVID-19 mild infection Patients. *Journal of Medical and Life Science*, 2021; 3(2): 38-43. doi: 10.21608/jmals.2021.240126

26. Chand, N., & Sanyal, A. J. (2007). Sepsis-induced cholestasis. Hepatology, 45(1), 230-241.

27. Geier A, Fickert P, Trauner M (2006) Mechanisms of disease: mechanisms and clinical 23. Implications of cholestasis in sepsis. Nat Clin Pract Gastroenterol Hepatol 3: 574-585. Link: https://goo.gl/kUxFlw

28. David, M., Danks, P., E., Campbell, J., F., Connelly. (1965). An aetiological study of neonatal jaundice in a children's hospital*.

Journal of Paediatrics and Child Health, 1(4):193-201. doi: 10.1111/J.1440-1754. 1965.TB02527. X.

29. Gilroy RK, Mailliard ME, Gollan JL (2003) Gastrointestinal disorders of the critically ill. Cholestasis of sepsis. Best Pract Res Clin Gastroenterol 17: 357- 367. Link: https://goo.gl/EMWCh

30. Kalas, M. A., Chavez, L., Leon, M., Taweesedt, P. T., & Surani, S. (2021). Abnormal liver enzymes: A review for clinicians. World journal of hepatology, 13(11), 1688.

31. Chen, H. L., Wu, S. H., Hsu, S. H., Liou, B. Y., Chen, H. L., & Chang, M. H. (2018). Jaundice revisited: recent advances in the diagnosis and treatment of inherited cholestatic liver diseases. Journal of biomedical science, 25, 1-13.

32. Van Der Woerd, W. L., Houwen, R. H., & van de Graaf, S. F. (2017). Current and future therapies for inherited cholestatic liver diseases. World journal of gastroenterology, 23(5), 763.

33. Henkel, S. A., Squires, J. H., Ayers, M., Ganoza, A., Mckiernan, P., & Squires, J. E. (2019). Expanding etiology of progressive familial intrahepatic cholestasis. World Journal of Hepatology, 11(5), 450.

34. Ansong-Assoku, B., Shah, S., Adnan, M., & Ankola, P. (2024). Neonatal jaundice. StatPearls.

35. Mouleeswaran, K. S., Varghese, J., & Reddy, M. S. (2023). Atlas of Basic Liver Histology for Practicing Clinicians and Pathologists. Springer.

36. Mouleeswaran, K. S., Varghese, J., & Reddy, M. S. (2023). Pediatric Liver Pathology. In Atlas of Basic Liver Histology for Practicing Clinicians and Pathologists (pp. 25-46). Singapore: Springer Nature Singapore.

37. Bsirini, C., Katerji, R., Lee, E. J., & Gonzalez, R. S. (2022). Liver histology in septic patients: is it all about ductular cholestasis? Archives of Pathology & Laboratory Medicine, 146(11), 1329-1337.

38. Yameny, A., Alabd, S., Mansor, M. MiRNA-122 association with TNF-α in some liver diseases of Egyptian patients. *Journal of Bioscience and Applied Research*, 2023; 9(4): 212-230. doi: 10.21608/jbaar.2023.329927

39. Quelhas, P., Jacinto, J., Cerski, C., Oliveira, R., Oliveira, J., Carvalho, E., & Dos Santos, J. (2022, October). Protocols of investigation of neonatal cholestasis—a critical appraisal. In Healthcare (Vol. 10, No. 10, p. 2012). MDPI.

40. Hong, J. Y., F Sato, E., Hiramoto, K., Nishikawa, M., & Inoue, M. (2007). Mechanism of Liver Injury during Obstructive Jaundice: Role of Nitric Oxide, Splenic Cytokines, and Intestinal Flora. Journal of clinical biochemistry and nutrition, 40(3), 184–193. https://doi.org/10.3164/jcbn.40.184

41. Fernández, J., Angeli, P., Trebicka, J., Merli, M., Gustot, T., Alessandria, C., ... & Arroyo, V. (2020). Efficacy of albumin treatment for patients with cirrhosis and infections unrelated to spontaneous bacterial peritonitis. Clinical gastroenterology and hepatology, 18(4), 963-973.

42. Yameny, A., Alabd, S., Mansor, M. Serum TNF-α levels as a biomarker in some liver diseases of Egyptian patients. *Journal of Medical and Life Science*, 2023; 5(1): 1-8. doi: 10.21608/jmals.2023.329303

43. Ng, D., & Fan, W. Q. (2023). Antibiotic Therapy in Moderate to Late Preterm Infants with Presumed Early Onset Sepsis is Associated with Aberrations to Feeding Tolerance and Hyperbilirubinemia.

44. Crismale, J. F., & Friedman, S. L. (2020). Acute liver injury and decompensated cirrhosis. Medical Clinics, 104(4), 647-662.