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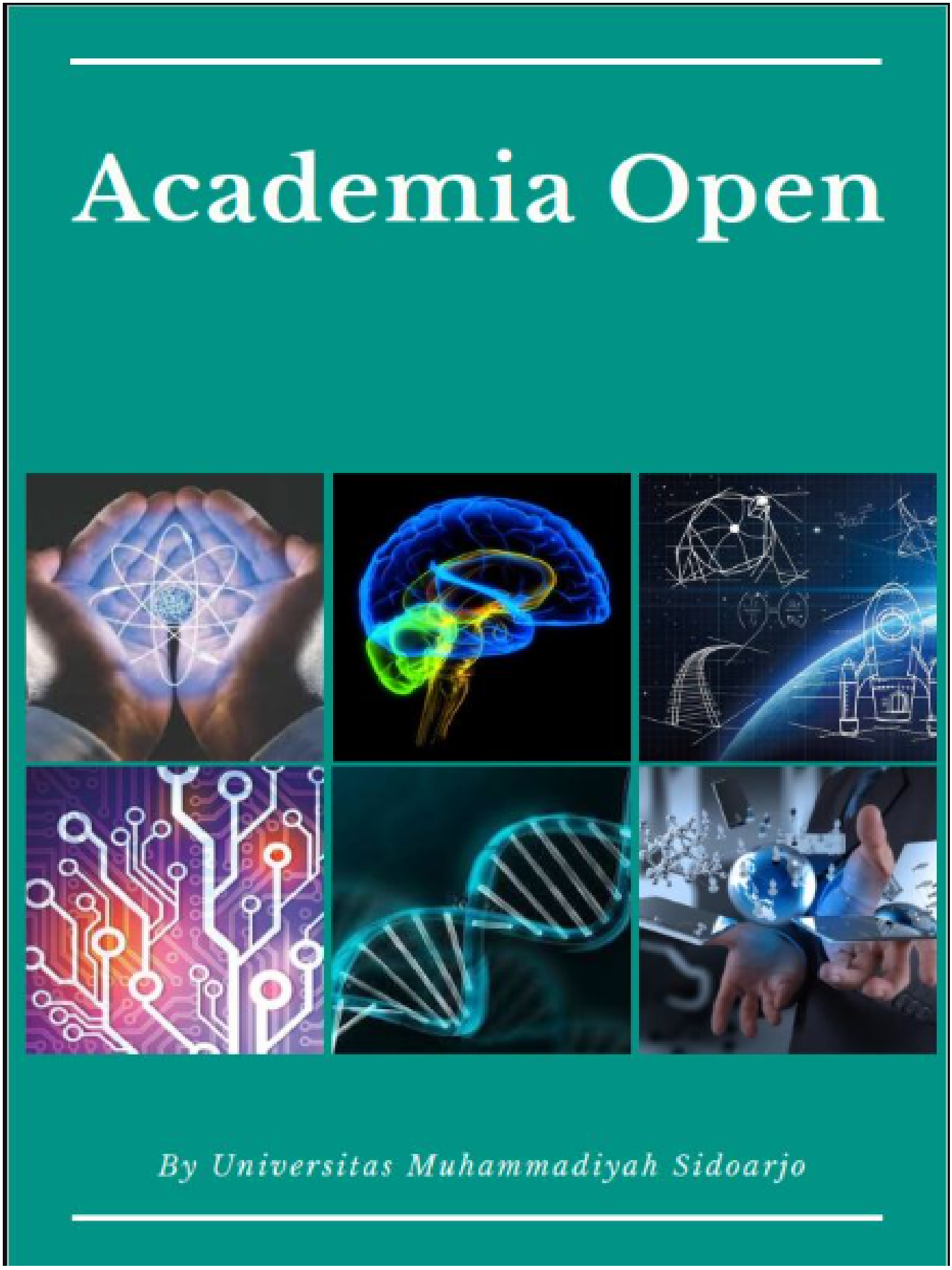

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Clinico-Immunobiochemical Markers of Destructive Cholecystitis: Penanda Klinis-Imunobiokimia pada Kolesistitis Destruktif

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Abstract

General Background: Gallstone disease and its inflammatory complications remain a major concern in hepatobiliary surgery, with destructive cholecystitis frequently associated with severe postoperative morbidity. **Specific Background:** Progression from chronic calculous cholecystitis to acute purulent-destructive forms involves systemic inflammatory activation, metabolic disturbances, and immune dysregulation measurable through clinical, laboratory, biochemical, and immunological parameters. **Knowledge Gap:** However, early identification of patients at high risk of complications based on integrated clinico-immunobiochemical indicators has not been sufficiently systematized. **Aims:** This study aimed to determine the prognostic value of clinical and laboratory markers and to formulate preventive recommendations for early risk assessment in destructive cholecystitis. **Results:** In 111 patients, acute purulent-destructive cases demonstrated higher total cholesterol (7.81 ± 0.12 mmol/L), elevated LDL (3.44 ± 0.10 mmol/L), reduced HDL (0.89 ± 0.04 mmol/L), and markedly increased proinflammatory cytokines TNF-α (68.5 ± 2.74 pg/mL) and IL-6 (24.8 ± 1.12 pg/mL) compared with chronic cholecystitis and controls; imaging revealed more frequent gallbladder wall destruction, biliary hypertension, and ascites, accompanied by greater postoperative complication rates. **Novelty:** The study integrates cytokine profiling, lipid parameters, and instrumental findings into a unified clinico-immunobiochemical framework for early complication prediction. **Implications:** Comprehensive assessment of these markers supports personalized preventive strategies, optimized surgical planning, and timely identification of high-risk patients, thereby improving clinical decision-making and perioperative safety in destructive cholecystitis.

Keywords: Destructive Cholecystitis, Cytokine Profile, TNF-α, Interleukin-6, Lipid Metabolism

Key Findings Highlights:

1. Acute purulent cases presented markedly higher proinflammatory mediator concentrations compared with chronic forms and controls.
2. Dyslipidemia characterized by raised total cholesterol and LDL with decreased HDL distinguished severe disease groups.
3. Combined laboratory and imaging indicators enabled earlier detection of patients prone to postoperative complications.

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Introduction

Over the past decades, the incidence of gallstone disease (GSD) has increased significantly, reaching up to 40% in the structure of gastrointestinal diseases [1]. According to WHO data, cholelithiasis is diagnosed in more than 20 million patients, with an annual increase of approximately 1 million new cases [2].

In hepatobiliary surgery, considerable importance is attached to improving methods of hemostasis and biliostasis, since their effectiveness directly affects the incidence of postoperative complications [3,4]. Intraoperative and postoperative bleeding during cholecystectomy in destructive inflammation remains a serious clinical problem [5,6].

The need for blood transfusion increases the risk of complications such as acute renal failure, thrombosis, thromboembolism, myocardial infarction, and acute post-transfusion lung injury [7]. In addition, transfusions may induce immunosuppression, thereby increasing the likelihood of nosocomial infections, including sepsis and postoperative wound infection [8,9].

Alongside the risk of bleeding, the prevention of hospital-acquired infections remains an urgent challenge, given that purulent-inflammatory complications after cholecystectomy occur in 6–15% of cases [10].

Objective: To identify clinical and laboratory markers that allow early prediction of the risk of complications in destructive cholecystitis and to develop recommendations for their prevention.

Materials and Methods

As part of the study, a comprehensive examination was performed in 111 patients aged 21–88 years who were treated at the 7th City Clinical Hospital of Tashkent during 2023–2025. All patients had purulent-inflammatory complications of destructive calculous cholecystitis of various types, often in combination with choledocholithiasis and peritonitis.

The study was conducted in two stages and included 111 patients with gallstone disease. The first group consisted of 88 patients with acute purulent-destructive cholecystitis, while the second group included 23 patients with chronic calculous cholecystitis. In addition, 20 conditionally healthy volunteers were enrolled for immunobiochemical comparative analysis.

The diagnostic evaluation of patients comprised clinical and instrumental examinations, laboratory, biochemical, and immunological studies, as well as consultations with related specialists in order to clarify concomitant diseases.

Immune status was assessed based on cytokine levels (TNF-α, IL-6) using Cytokine test kits (Saint Petersburg, Russia) and ElisaKid kits (China).

The inclusion criteria for this study were a confirmed diagnosis of gallstone disease and patient age over 18 years.

The exclusion criteria included acute myocardial infarction; the presence of HIV/AIDS; severe autoimmune diseases (systemic lupus erythematosus, dermatomyositis, autoimmune thyroiditis, etc.); acute or chronic renal or hepatic failure; peptic ulcer disease; oncological diseases; and acute psychiatric disorders.

Results and Discussion

Under our observation were 111 patients aged 21 to 88 years (mean age 49.3 ± 1.59 years) with purulent-inflammatory complications of destructive calculous cholecystitis.

Age analysis of patients with complicated cholecystitis revealed a predominance of younger patients (≤45 years) — 42.3% (n=47; mean age 32.2 ± 1.0 years), reflecting the significant impact of the disease on the working-age population. The middle-aged (45–59 years) and elderly (60–74 years) groups accounted for 28.8% (n=32; mean age 51.8 ± 0.9 years) and 18.9% (n=21; mean age 63.5 ± 0.9 years), respectively, maintaining a high risk of complications. The oldest group (75–90 years) represented 9.9% (n=11) and requires increased attention due to age-related changes and comorbidities.

In the study, 111 patients were examined and divided into two groups: the main group (n=88) with acute purulent-destructive cholecystitis and the comparison group (n=23) with chronic calculous cholecystitis.

In the main group, younger patients (18–44 years) were more frequent — 44.3% compared to 34.8% in the comparison group ($P<0.05$). The mean age in this category was lower (32.5 ± 1.07 vs. 37.4 ± 1.67 years; $P<0.05$), reflecting a trend toward earlier development of destructive forms of cholecystitis and the possible influence of active inflammatory and immune processes in the working-age population (Fig. 1).

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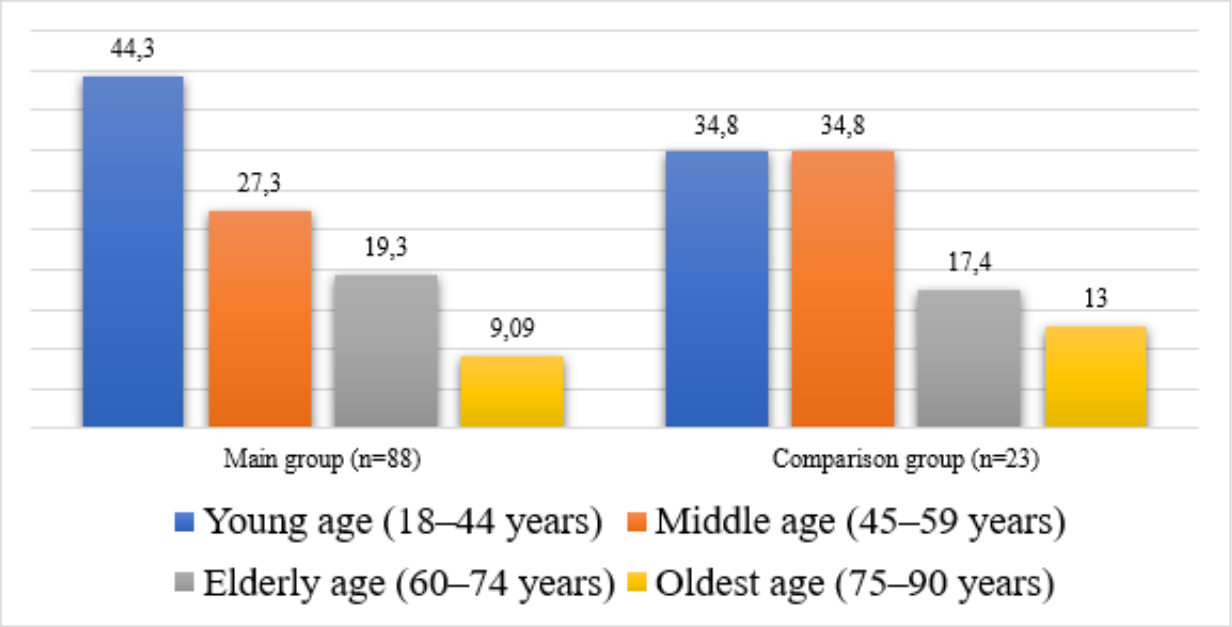

Figure 1. **Fig. 1. Age distribution of patients in the main and comparison groups [11]**

Systemic and cholestatic signs manifested differently. General weakness was observed in 100% of patients in the comparison group and 90.9% in the main group (P>0.05), reflecting the severity of systemic inflammation. Cholestatic symptoms (jaundice, changes in urine color, abdominal bloating, and bitter taste in the mouth) were significantly more frequent in the main group (P<0.05), whereas loss of appetite was almost three times more common in the comparison group (13.0% vs. 4.55%; P<0.05), indicating a more prolonged or complicated course of the disease.

Assessment of concomitant pathology revealed that cardiovascular diseases were the most common, occurring in 59.1% of the main group and 52.2% of the comparison group (P>0.05). Respiratory disorders were more frequent in the main group (41.0% vs. 26.0%; P<0.05), reflecting more pronounced comorbidity and a possible increased risk of postoperative complications (Fig. 2).

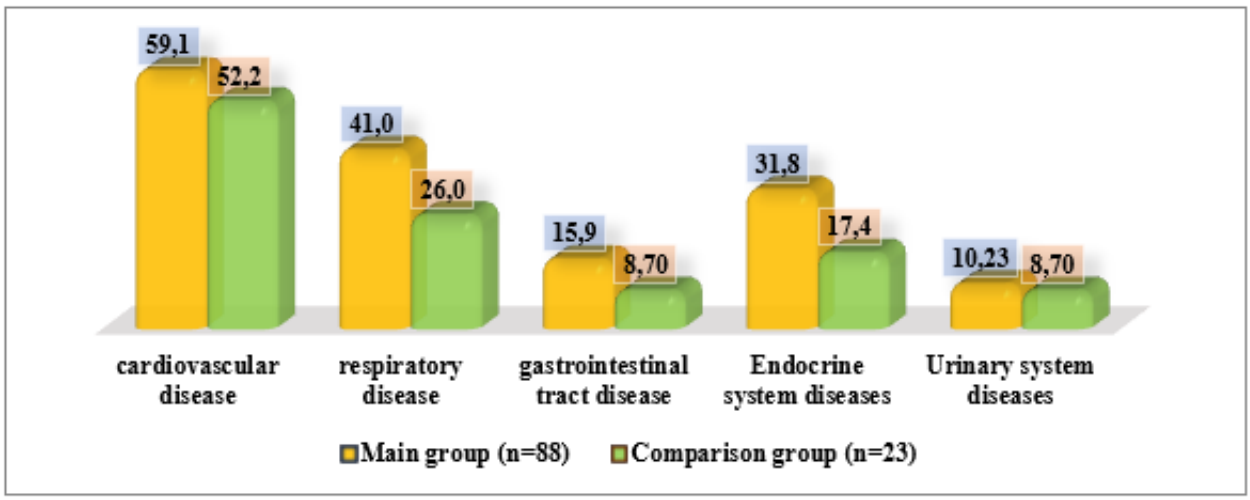

Figure 2. **Fig. 2. Concomitant pathology in the examined patient groups, (%), (P≤0. 05) [12]**

The total cholesterol level in the main group was 7.81 ± 0.12 mmol/L, significantly higher than the control (4.23 ± 0.09 mmol/L; P<0.001). In the comparison group, the level was also above normal — 6.55 ± 0.15 mmol/L (P<0.05). Moreover, the level in the main group exceeded that of the comparison group by 11%, reflecting a more pronounced hypercholesterolemia.

The LDL level in patients of the main group reached 3.44 ± 0.10 mmol/L, which was significantly higher than the control (2.67 ± 0.08 mmol/L; P<0.001) and 17% higher than in the comparison group (2.94 ± 0.11 mmol/L; P<0.05). HDL was reduced to 0.89 ± 0.04 mmol/L (control — 1.33 ± 0.05 mmol/L; P<0.001), while in the comparison group it was 1.09 ± 0.06

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mmol/L (P<0.05) and remained higher than in the main group (P<0.05) (Fig. 3).

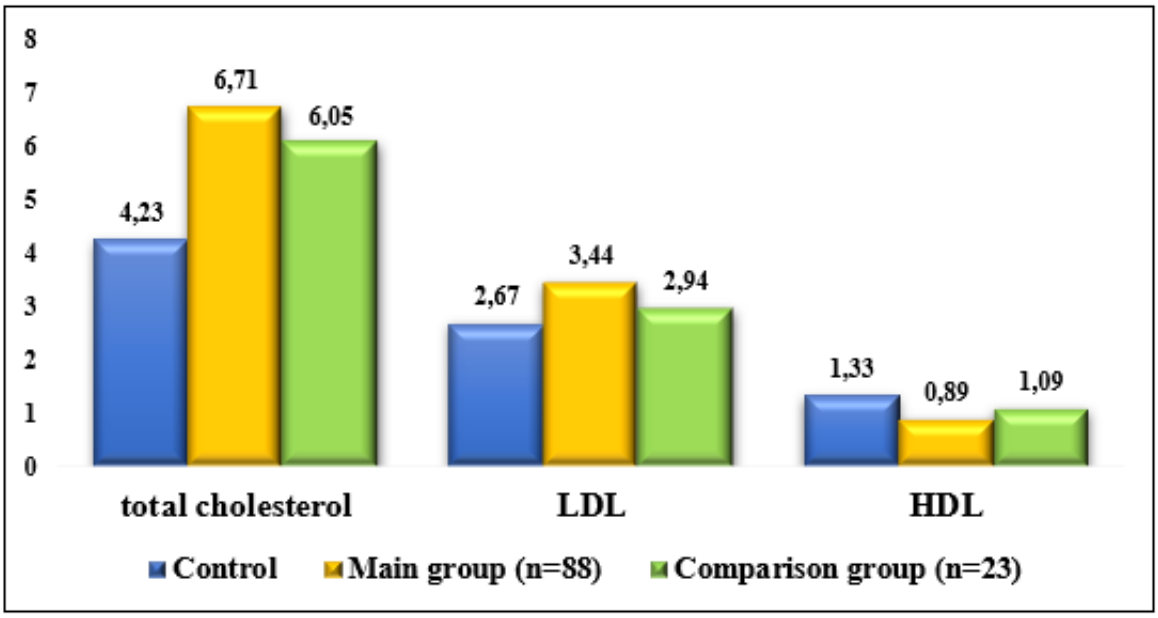

Figure 3. **Fig. 3. Lipid profile in the examined groups (mmol/L), (P≤0. 05) [13]**

Ultrasound and MRI revealed that patients in the main group more frequently exhibited destructive changes of the gallbladder wall, biliary hypertension, and reactive ascites (P<0.01), whereas the comparison group was dominated by an obstructive-mechanical component, including choledocholithiasis with increased common bile duct diameter (P<0.05).

In the main group, laparoscopic cholecystectomy with drainage was performed more frequently (P<0.01), whereas in the comparison group, staged interventions with ERCP and EST predominated. The incidence of postoperative complications (hyperthermia, infections, local complications) was higher in acute purulent-destructive cholecystitis, which determined the choice of surgical strategy depending on the severity of inflammation.

The TNF-α level in acute purulent-destructive cholecystitis was significantly elevated compared to the control (68.5 ± 2.74 pg/mL vs. 21.4 ± 0.96 pg/mL; P<0.001), whereas in patients with chronic cholecystitis it was 37.3 ± 1.85 pg/mL (P<0.01). In the main group, TNF-α exceeded the levels in the comparison group by almost 1.8 times (P<0.01) (Fig. 4).

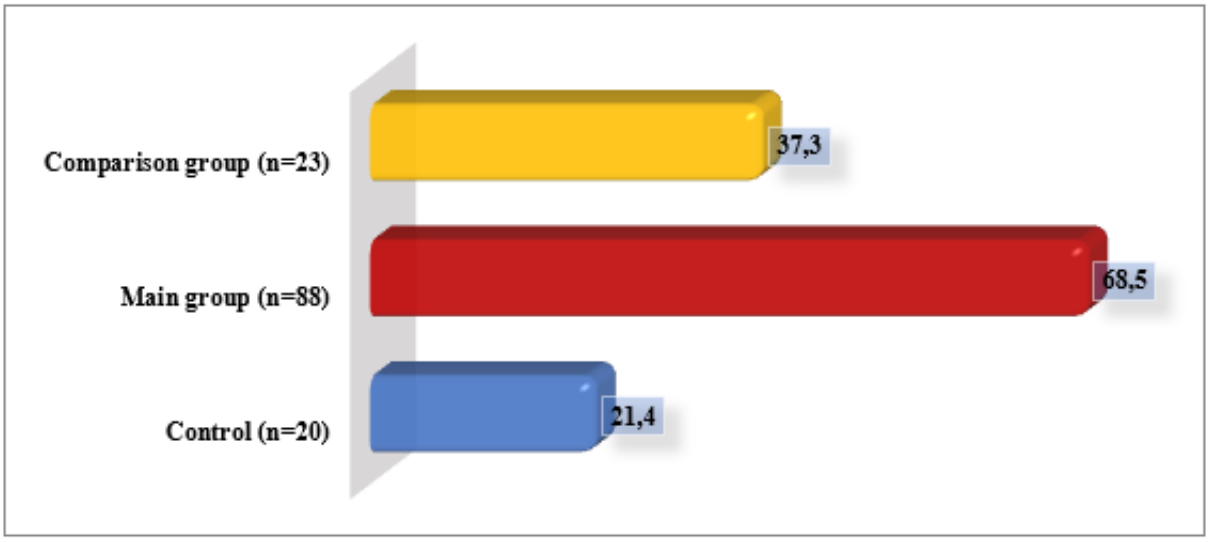

Figure 4. **Fig. 4. Tumor necrosis factor (TNF- α) levels in the examined groups (pg /mL), (P≤0. 05) [14]**

The IL-6 level in acute purulent-destructive cholecystitis was significantly elevated compared to the control (24.8 ± 1.12 pg/mL vs. 5.12 ± 0.24 pg/mL; P<0.001), whereas in patients with chronic cholecystitis it was 13.3 ± 0.76 pg/mL (P<0.01). In

the main group, IL-6 exceeded the levels in the comparison group by almost 1.9 times (P<0.01) (Fig. 5).

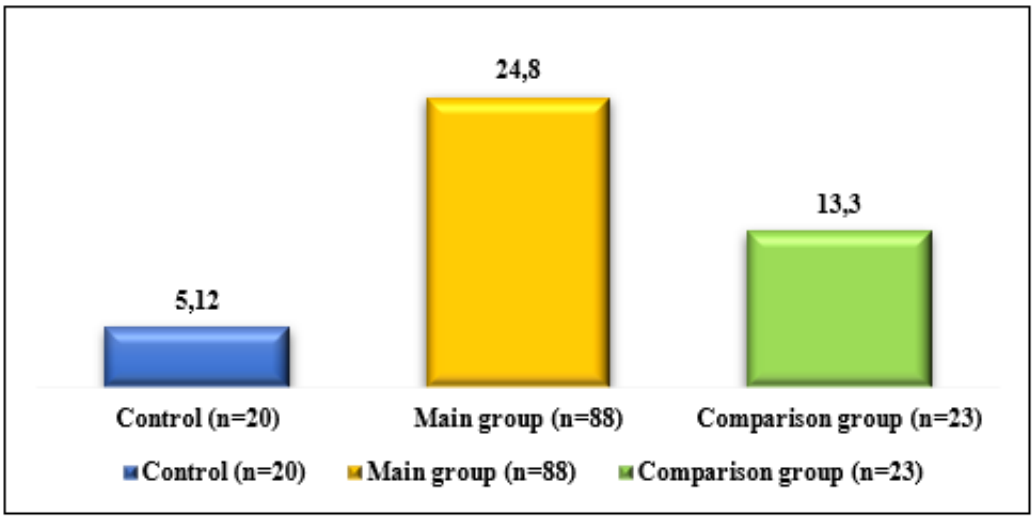

Figure 5. **Fig. 5. IL-6 levels in the examined groups (pg /mL), (P≤0. 05) [15]**

Conclusion

The conducted study demonstrated that the progression of chronic calculous cholecystitis to an acute purulent-destructive process is a multistage pathogenetic phenomenon, accompanied by activation of the systemic inflammatory response, cytokine imbalance with predominance of proinflammatory mediators, and suppression of Th1 immune mechanisms. Endothelial dysfunction, microcirculatory disturbances, impaired fibrinolytic activity, and depletion of antioxidant defenses contribute to tissue ischemia and destructive changes in the gallbladder wall.

Clinical, laboratory, immunobiochemical, and instrumental indicators (including TNF-α, IL-6, lipid profile, ultrasound, and MRI findings) allow for the early identification of patients at high risk of complications and can be used to develop individualized preventive strategies and optimize surgical management.

Thus, a comprehensive assessment of clinical, laboratory, and immunobiochemical markers provides effective prediction of disease severity, timely identification of patients at risk for complications, and enhances postoperative safety in destructive cholecystitis.

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