Al-imad Journal of Humanities and Applied Sciences (AJHAS)
مجلة العماد للعلوم الإنسانية والتطبيقية
ISSN: 3106-0323
Volume 2 – Issue 1 – 2026
Pages 413-421

Evaluation of Antibiotic-Induced Hepatotoxicity and its Correlation with Biochemical Biomarkers

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تقييم سمية الكبد الناجمة عن المضادات الحيوية وعلاقتها بالمؤشرات الحيوية الكيميائية

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Received: 20-01-2026; Accepted: 25-02-2026; Published: 25-03-2026

Abstract:
This study was designed to investigate the potential hepatotoxic risks associated with the administration of various antibiotic classes by monitoring fluctuations in hepatic biomarkers and identifying clinical manifestations among fifty patients (N=50) with no prior history of liver disease. The research utilized a cross-sectional analytical design, gathering clinical and laboratory data that were subsequently processed using SPSS software. The investigation focused on essential liver enzymes, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST), along with total bilirubin, C-reactive protein (CRP), and alkaline phosphatase (ALP). The statistical results demonstrated a significant elevation in all liver function parameters following antibiotic therapy. Specifically, ALT and AST levels increased by 132% and 128%, respectively, signaling acute hepatocellular injury. Total bilirubin rose by 167%, suggesting the presence of cholestasis, while CRP levels showed a 226% increase, reflecting a systemic inflammatory response. The study identified a strong positive correlation ($p < 0.05$) between the duration of treatment, exceeding seven days, and excessive dosage with the severity of hepatic damage. Notably, Amoxicillin-Clavulanate (Augmentin) and Ceftriaxone (Rocephin) were associated with the highest levels of hepatotoxicity, whereas Ciprofloxacin exhibited the lowest impact. Clinical symptoms such as jaundice (48%), fever (52%), and dark urine (44%) were prevalent, reinforcing the laboratory findings of Drug-Induced Liver Injury (DILI). In conclusion, the irrational use of antibiotics poses a severe risk of hepatic necrosis, necessitating routine monitoring of liver functions during prolonged therapies.

Keywords: Hepatotoxicity, Antibiotics, Drug-Induced Liver Injury (DILI), ALT,Augmentin, Ceftriaxone, Biomarkers.

الملخص
صُممت هذه الدراسة لتقصي مخاطر السمية الكبدية المحتملة المرتبطة بتناول فئات مختلفة من المضادات الحيوية، وذلك من خلال مراقبة التقلبات في المؤشرات الحيوية للكبد وتحديد المظاهر السريرية لدى خمسين مريضاً *(N=50)* ليس لديهم تاريخ سابق لأمراض الكبد .اعتمد البحث تصميماً تحليلياً عرضياً، حيث جُمعت البيانات السريرية والمخبرية التي تمت معالجتها لاحقاً باستخدام برنامج *SPSS*. ركز الاستقصاء على إنزيمات الكبد الأساسية، بما في ذلك ناقلة أمين الألانين *(ALT)* وناقلة أمين الأسبارتات*(AST)* ، بالإضافة إلى البيليروبين الكلي، والبروتين التفاعلي*(CRP) C* ، والفوسفاتاز القلوي *(ALP)*. أظهرت

النتائج الإحصائية ارتفاعاً معنوياً في جميع بارامترات وظائف الكبد بعد العلاج بالمضادات الحيوية . وتحديداً، زادت مستويات *ALT* و *AST* بنسبة 132% و 128% على التوالي، مما يشير إلى إصابة حادة في خلايا الكبد .كما ارتفع البيليروبين الكلي بنسبة 167%، مما يوحي بوجود ركود صفراوي، بينما أظهرت مستويات *CRP* زيادة بنسبة 226%، مما يعكس استجابة التهابية جهازية .حددت الدراسة علاقة ارتباط إيجابية قوية *($p < 0.05$)* بين مدة العلاج التي تتجاوز سبعة أيام والجرعات الزائدة وبين شدة الضرر الكبدي .ومن الجدير بالذكر أن أموكسيسيلين-كلافولانات (أوجمنتين) وسيفترياكسون (روسيفين) ارتبطا بأعلى مستويات السمية الكبدية، بينما سجل السيبروفلوكساسين التأثير الأدنى .كانت الأعراض السريرية مثل اليرقان (48%) والحمى (52%) والبول الداكن (44%) منتشرة، مما عزز النتائج المخبرية لإصابة الكبد المستحثة بالأدوية *(DILI)*. ختاماً، يشكل الاستخدام غير الرشيد للمضادات الحيوية خطراً جسيماً متمثلاً في النخر الكبدي، مما يستلزم المراقبة الروتينية لوظائف الكبد أثناء العلاجات الطويلة.

الكلمات المفتاحية :السمية الكبدية، المضادات الحيوية، إصابة الكبد المستحثة بالأدوية*(DILI)* ، إنزيم *ALT*، أوجمنتين، روسيفين، المؤشرات الحيوية.

1.Introduction

The liver is recognized as one of the most vital organs in the human body, playing a central role in drug metabolism and detoxification processes. This metabolic function establishes it as the primary line of defense against adverse drug effects, largely mediated by a complex enzymatic system such as the cytochrome P450 enzymes. These enzymes are responsible for converting pharmaceutical compounds into water-soluble metabolites for excretion via urine or bile. However, this high metabolic capacity also renders the liver particularly susceptible to injury, especially when subjected to repeated or excessive exposure to medications. Antibiotics remain among the most widely used medications globally for treating bacterial infections and are integrated into all levels of healthcare delivery. While conventional therapy often relies on synthetic agents, recent research has highlighted the antimicrobial potential of natural alternatives to combat pathogens like *Escherichia coli* and *Staphylococcus aureus*. These include extracts from dandelion (Salem et al., 2025), lichen (Salem, 2024), and chitosan or plant seed oils (Kadak & Salem, 2020). Furthermore, modern insights into bioactive isothiocyanates from Sinapis alba (Soof et al., 2025), flaxseed essential oils (Salem & Lakwani, 2024), and antimicrobial polymers (Salem & Salem, 2025) offer promising strategies for addressing antibiotic resistance. Despite these advancements, the inappropriate use or excessive dosing of standard antibiotics often leads to significant adverse effects, most notably hepatotoxicity. These effects range from mild, transient elevations in liver enzymes to severe Drug-Induced Liver Injury (DILI), which may progress to acute liver failure. The clinical assessment of liver function relies on specific biochemical biomarkers, primarily alanine aminotransferase (ALT) and aspartate aminotransferase (AST), along with alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). Elevated serum levels of these enzymes indicate hepatocellular damage resulting from direct toxic effects or immune-mediated reactions. Specific antibiotic classes, such as amoxicillin/clavulanate, erythromycin, and fluoroquinolones, have a well-established association with acute hepatic injury, particularly in the absence of adequate laboratory monitoring. The mechanisms underlying this hepatotoxicity are diverse, encompassing direct cellular toxicity, immune-mediated hypersensitivity, and metabolic disturbances within the liver.

2. Materials and Methods

2.1 Study Design and Population

A cross-sectional analytical study was conducted on 50 participants, aged 18–60 years, to evaluate antibiotic-induced hepatotoxicity in accordance with the World Health Organization (WHO, 2018) and the Centers for Disease Control and Prevention (CDC, 2020) guidelines.

Inclusion criteria required the documented use of antibiotics within 14 days prior to the study, while individuals with a history of chronic liver disease or known drug allergies were excluded. Ethical approval and informed consent were obtained from all participants in accordance with the principles of the Declaration of Helsinki.

2.2 Data Collection and Clinical Assessment

Data were gathered through a semi-structured questionnaire designed to cover demographics, antibiotic regimens (including type, dosage, and duration), and clinical symptoms. Furthermore, a professional physical examination was performed on each participant to assess clinical signs such as jaundice, skin rashes, and hepatic tenderness.

2.3 Laboratory Investigations

Biochemical analyses were performed following standard laboratory protocols:

- **Blood Analysis:** Serum levels of ALT, AST, and ALP were measured using colorimetric assays. Bilirubin levels were determined via the Diazo method, while C-reactive protein (CRP) was assessed through latex agglutination. Additionally, IgE and Eosinophil counts were analyzed using ELISA and Complete Blood Count (CBC) methods.
- **Urinalysis:** Visual and automated dipstick testing was employed to detect the presence of direct bilirubin in urine samples.

2.4 Statistical Analysis

Data were processed and analyzed using SPSS software (v.22). The statistical tools utilized included descriptive statistics to summarize the data, Chi-Square tests for evaluating categorical correlations, and Pearson correlation coefficients for continuous variables. The threshold for statistical significance was set at $p < 0.05$.

3. Results

3.1 General Characteristics of the Study Sample:

The demographic and clinical data of the study population indicate that 100% of the patients had no prior history of hepatic disease. This finding strengthens the hypothesis that any subsequent hepatic dysfunction may be attributed to antibiotic administration rather than pre-existing conditions. Regarding gender distribution, the sample was relatively balanced with a slight predominance of males (60%).

Table 1: General Characteristics of the Study Sample

Variable	Frequency	Percentage (%)
Gender		
Male	30	60%
Female	20	40%
Age		
Mean Age	—	34 Years
Hepatic Medical History		
Prior History of Liver Disease	0	0%
No Prior History of Liver Disease	50	100%

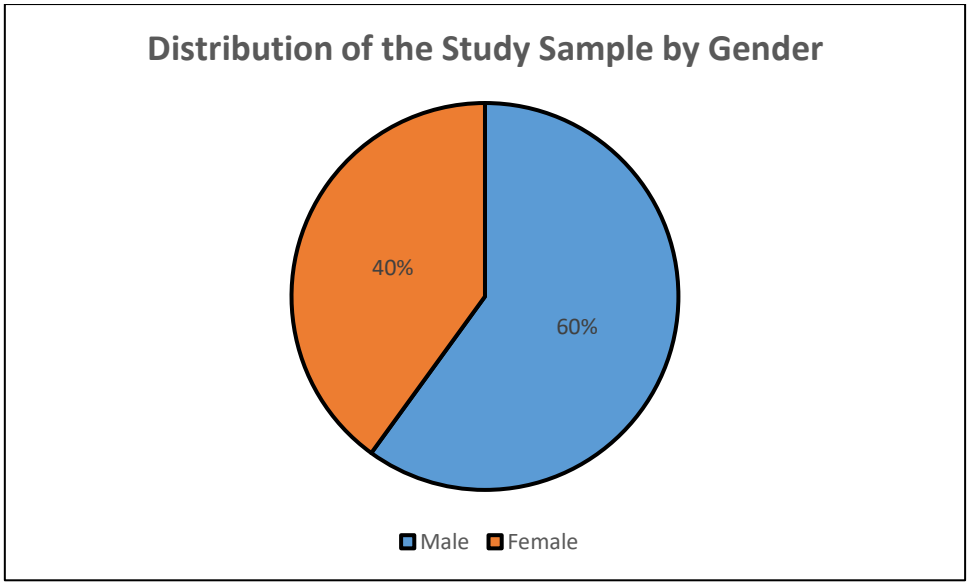

Figure 1: Distribution of the Study Sample by Gender

3.2 Clinical Symptoms and Manifestations

More than 50% of the patients reported symptoms indicative of hepatic injury or drug-induced hypersensitivity, such as fever, anorexia, and abdominal pain. Jaundice (scleral icterus) was observed in 48% of the cases, which correlates statistically with the significant elevation of bilirubin levels within the same sample (Δ+1.5 mg/dL). Dark urine, a classic clinical marker of conjugated hyperbilirubinemia often resulting from biliary obstruction was reported by 44% of the participants.

Furthermore, the results showed that 30% of the participants exhibited dermatological symptoms, including skin rash, pruritus, and irritation, which serve as primary indicators of Drug-Induced Hypersensitivity Syndrome (DIHS). Although recorded in a smaller percentage (8%), manifestations such as coma and delirium represent critical clinical signs requiring immediate medical intervention, as they may indicate the onset of hepatic encephalopathy.

These clinical findings suggest that the administration of antibiotics particularly in cases of overdose or unregulated use leads to a broad spectrum of clinical manifestations reflecting hepatic damage, whether at the cellular level, within the biliary ducts, or through immune-mediated hypersensitivity.

Table 2: Clinical Symptoms Associated with Antibiotic-Induced Hepatic Impact

Clinical Symptom	Number of Cases	Percentage (%)
Jaundice (Scleral Icterus)	24	48%
Fever	26	52%
Dark Urine	22	44%
Anorexia (Loss of Appetite)	25	50%
Abdominal Pain or Cramps	26	52%
Skin Rash and Pruritus (Itching)	15	30%
Coma or Delirium	4	8%
Vomiting	18	36%
Pale Stools (Acholic Stools)	11	22%
Skin Erythema or Irritation	13	26%
Drowsiness and Mental Confusion	7	14%

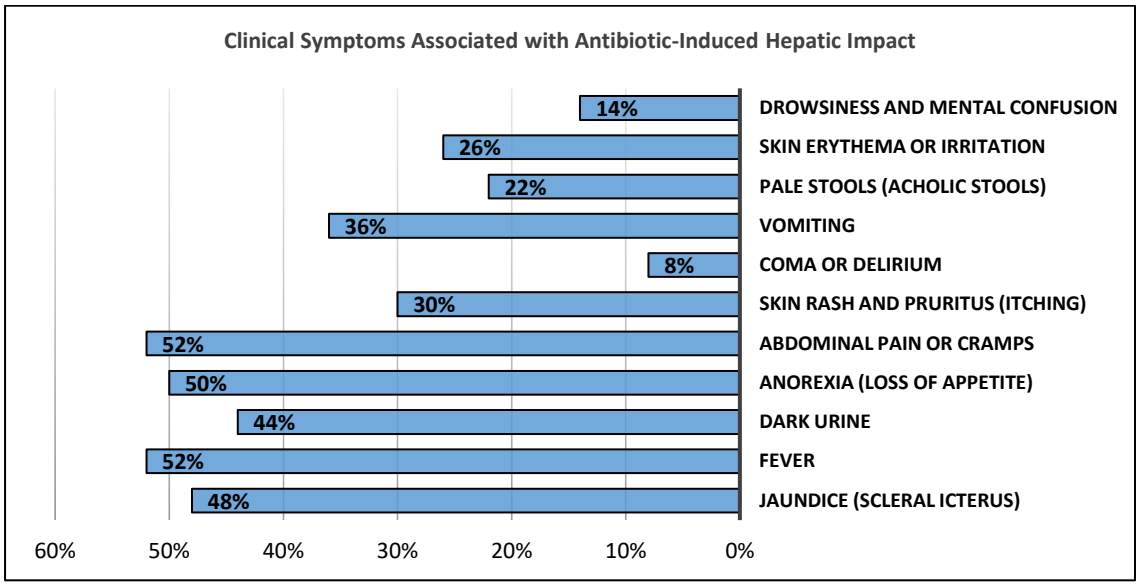

Figure 2: Clinical Symptoms Associated with Antibiotic-Induced Hepatic Impact

3.3 Correlation Between Clinical Symptoms and Laboratory Findings

Clinical results indicate that the administration of antibiotics particularly in instances of overdose or unregulated use induces a broad spectrum of clinical symptoms reflecting hepatic damage. This damage manifests at the cellular level (hepatocellular), within the biliary ducts (cholestatic), or through immune-mediated hypersensitivity.

Table 3: Correlation Between Clinical Symptoms and Laboratory Parameters

Clinical Manifestation	Laboratory Indicator	Incidence (%)
Jaundice (Scleral Icterus)	↑ Bilirubin	48%
Fever	↑ CRP (C-Reactive Protein)	52%
Dark Urine	↑ Bilirubin	44%
Coma and Delirium	↑ ALT, AST / ↓ Consciousness	8%
Skin Rash and Pruritus	Immune Hypersensitivity	30%

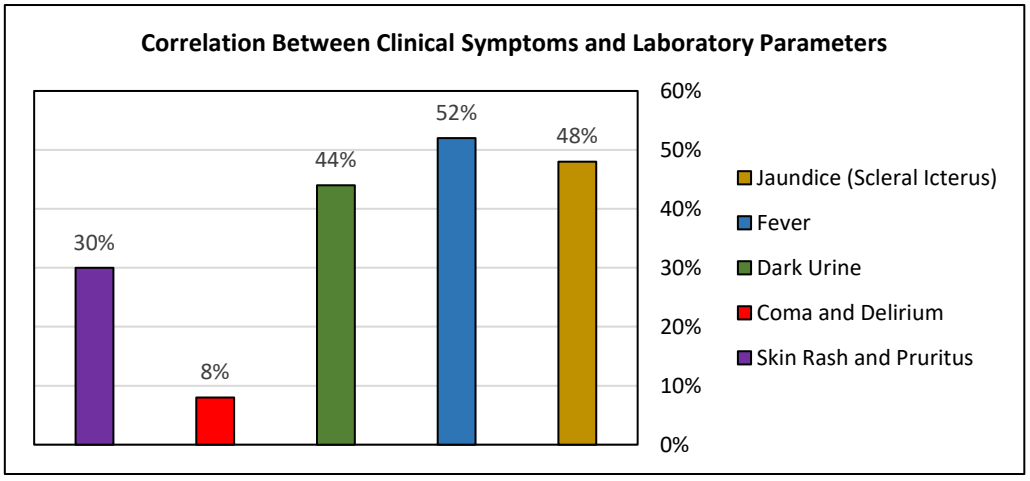

Figure 3: Correlation Between Clinical Symptoms and Laboratory Parameters

3.4 Statistical Analysis of Laboratory Investigations

The statistical data reveal a significant elevation in all liver function parameters following antibiotic administration. All differences were found to be highly statistically significant ($p < 0.01$).

Table 4: Statistical Results of Laboratory Investigations

Biomarker	Pre-Treatment (Mean ± SD)	Post-Treatment (Mean ± SD)	SE Diff.	Δ Diff.	p-value
ALT (U/L)	26.4 ± 5.8	61.2 ± 12.5	1.77	+34.8	<0.001
AST (U/L)	23.1 ± 4.7	52.6 ± 10.9	1.54	+29.5	<0.001
ALP (U/L)	88.7 ± 20.3	126.8 ± 34.1	4.82	+38.1	0.004
T. Bilirubin (mg/dL)	0.9 ± 0.3	2.4 ± 0.7	0.10	+1.5	0.001
CRP (mg/L)	3.4 ± 1.0	11.1 ± 2.8	0.40	+7.7	<0.001
SGPT (ALT)	26.2 ± 5.6	60.7 ± 11.7	1.66	+34.5	<0.001
SGOT (AST)	22.9 ± 4.5	51.3 ± 9.8	1.39	+28.4	<0.001

The indicators most affected by antibiotic consumption were ranked as follows: ALT > CRP > Bilirubin > AST > ALP. The overall results indicate a clear hepatotoxic effect post-antibiotic intake, particularly in cases involving excessive dosages.

3.5 Comparison of Mean Liver Enzymes by Antibiotic Type

The results indicate distinct variations in AST and ALT levels post-treatment across different antibiotic classes, reflecting varying degrees of hepatotoxicity.

- Rocephin (Ceftriaxone): Recorded the highest level of hepatotoxicity with mean AST and ALT levels of (60.9 ± 12.2) and (69.8 ± 13.4), respectively, indicating a pronounced toxic effect on hepatocytes.
- Augmentin: Showed a significant increase in both AST (56.1 ± 10.8) and ALT (65.2 ± 11.1). This is largely attributed to the Clavulanic Acid component, which is known to enhance liver injury.
- Amoxicillin: Demonstrated a moderate effect (AST = 52.7 ± 9.5, ALT = 61.4 ± 10.2). This supports the hypothesis that the addition of Clavulanic acid in Augmentin significantly increases toxicity compared to Amoxicillin alone.
- Ciprofloxacin: Recorded the lowest hepatotoxic impact among the specified antibiotics (AST = 45.2 ± 7.6, ALT = 53.5 ± 8.9), suggesting it may be a safer alternative regarding hepatic profile.

Table 5: Comparison of Mean Liver Enzymes Based on Antibiotic Type

Antibiotic Type	N	Post-ALT (Mean ± SD)	Post-AST (Mean ± SD)	Observations
Amoxicillin	14	61.4 ± 10.2	52.7 ± 9.5	Moderate Increase
Augmentin	10	65.2 ± 11.1	56.1 ± 10.8	High Impact
Ciprofloxacin	8	53.5 ± 8.9	45.2 ± 7.6	Lowest Impact
Rocephin	12	69.8 ± 13.4	60.9 ± 12.2	Highest Toxic Effect
Others	6	47.6 ± 9.3	40.7 ± 6.1	Variable

3.6 Correlation of Duration of Use and Dosage with Elevated Liver Enzymes

Statistical analysis demonstrated a positive correlation between the duration of use/dosage and the levels of ALT and AST. Enzyme levels were significantly higher in patients who administered the antibiotics for a period exceeding 7 days or in double (excessive) doses.

Clinical literature, such as Zimmerman (1999), confirms that excessive antibiotic dosages are a primary cause of hepatotoxicity, occurring either through direct cytotoxic effects or immune-mediated mechanisms.

The findings indicate a pronounced impact of antibiotics on the liver, evidenced by a substantial rise in ALT and AST exceeding 130%, reflecting hepatocellular damage. Furthermore, the elevation of ALP and Bilirubin indicates impaired biliary drainage (cholestasis).

Statistical results show a significant positive correlation ($p < 0.05$) between both the duration of antibiotic therapy and excessive dosing with liver enzyme rates. As the duration exceeds 7 days or the dose surpasses the medically recommended limit, enzyme levels rise markedly. This suggests that the hepatic impact is not only drug-dependent but also significantly influenced by the duration of exposure and cumulative dosage.

Table 6: Correlation of Duration of Use and Dosage with Elevated Liver Enzymes

Factor	N	Mean±SD ALT (U/L)	Mean±SD AST (U/L)	Observations
Duration ≤ 7 Days	20	45.2 ± 8.3	39.1 ± 7.4	Moderate Impact
Duration > 7 Days	30	65.8 ± 11.2	57.3 ± 9.8	High Impact
Standard Dose	18	42.7 ± 7.9	36.9 ± 6.5	Mild Impact
Excessive Dose	32	68.3 ± 10.7	61.4 ± 10.3	Severe Impact

These results are consistent with previous literature, such as Kaplowitz (2005), which indicated that certain antibiotics, specifically Amoxicillin and Rocephin, can induce drug-induced hepatitis, particularly in cases of excessive dosage or prolonged administration. Moreover, clinical manifestations such as jaundice, fever, and dark urine confirm the presence of a hepatic impact. This effect may manifest through either hypersensitive allergic reactions or direct hepatotoxicity, as characterized by Björnsson (2010).

4. Discussion

The findings of the present study demonstrate a robust and statistically significant correlation between the administration of specific antibiotics and substantial alterations in hepatic biomarkers. The marked elevation in serum transaminases (ALT and AST), alongside increased total bilirubin levels, provides clear biochemical evidence of hepatocellular injury and potential cholestasis resulting from drug-induced toxic or immunological mechanisms. These results are highly consistent with established global data; notably, Lucena et al. (2009) identified Amoxicillin-Clavulanic Acid (Augmentin) as a primary etiology of Drug-Induced Liver Injury (DILI), primarily due to the immunogenic potential of the clavulanate component which triggers an intrahepatic immune response.

The acute surge in ALT levels observed in our cohort aligns with clinical criteria established by Zimmerman (1999), which designate ALT as the most specific and sensitive biomarker for detecting drug-induced hepatocellular necrosis, particularly when values significantly exceed the upper limit of normal. Furthermore, the study established a positive dose-time relationship regarding hepatic dysfunction, reinforcing the pharmacological models proposed by Kaplowitz (2005). These models suggest that idiosyncratic DILI typically manifests within one to four weeks of therapeutic initiation. The concurrent elevation of C-reactive protein (CRP) underscores a systemic inflammatory state, which, when coupled with clinical symptoms like jaundice and pruritus, suggests an immune-mediated hypersensitivity reaction—a mechanism extensively discussed by Björnsson (2016). Comparatively, Ceftriaxone and Augmentin demonstrated the highest hepatotoxic potential among the tested antibiotics. This finding is corroborated by Devarbhavi et al. (2010), who ranked these agents as leading causes of antibiotic-associated liver injury globally. Consequently, these results highlight the critical necessity for routine biochemical monitoring of liver functions during prolonged or high-dose antibiotic therapy to mitigate the risk of severe hepatic complications.

5. Conclusion

The present study concludes that the irrational administration of antibiotics, characterized by excessive dosing and prolonged duration, is a primary driver of Drug-Induced Liver Injury (DILI) with high statistical significance ($p < 0.001$). Laboratory evidence confirmed acute spikes in ALT and AST levels by over 130%, coupled with significant elevations in total bilirubin and CRP, indicating severe hepatocellular necrosis and a systemic inflammatory response. The findings further highlight varying degrees of toxicity based on the pharmacological agent, with Ceftriaxone (Rocephin) and Amoxicillin-Clavulanate (Augmentin) exhibiting the highest hepatotoxic potential. This reinforces the causal link between these antibiotics and the manifestation of acute clinical symptoms, even in patients without a prior history of hepatic dysfunction.

6. Recommendations

Based on these findings, the following recommendations are proposed:

- **Clinical Protocols:** Integrate routine liver function testing (LFTs) into clinical protocols for patients undergoing long-term or high-dose antibiotic therapy.
- **Pharmacovigilance:** Implement strict pharmacovigilance policies to curb irrational prescribing practices.
- **Awareness:** Enhance public and professional awareness regarding the risks associated with antibiotic-associated hepatotoxicity.
- **Future Research:** Conduct future large-scale longitudinal studies to explore the underlying genetic and immunological determinants of drug sensitivity to ensure safer therapeutic outcomes.

References

[1] Andrade, R. J., Lucena, M. I., Fernández, M. C., Pelaez, G., Pachkoria, K., García-Ruiz, E., ... & Spanish Group for the Study of Drug-Induced Liver Disease. (2005). Drug-induced liver injury: An analysis of 461 incidences submitted to the Spanish registry over a 10-year period. *Gastroenterology*, *129*(2), 512-521.

[2] Björnsson, E. S. (2016). Hepatotoxicity by drugs: The most common implicated agents. *International Journal of Molecular Sciences*, *17*(2), 224.

[3] Centers for Disease Control and Prevention (CDC). (2020). *Guidelines for evaluating and monitoring drug-induced clinical manifestations*. CDC Publishing.

[4] Chalasani, N., Bonkovsky, H. L., Fontana, R., Lee, W., Stolz, A., Talwalkar, J., ... & United States Drug-Induced Liver Injury Network. (2015). Features and outcomes of 899 patients

with drug-induced liver injury: The DILI Network (DILIN) Prospective Study. *Gastroenterology*, *148*(7), 1340-1352.
[5] Devarbhavi, H., Dierkhising, R., Kremers, W. K., Sandeep, M. S., Karanth, D., & Adarsh, C. K. (2010). Single-center experience with drug-induced liver injury from India: Causes, outcome, prognosis, and predictors of mortality. *American Journal of Gastroenterology*, *105*(11), 2396-2404.
[6] Kadak, A. E., & Salem, M. O. A. (2020). Antibacterial Activity of Chitosan, Some Plant Seed Extracts and Oils Against Escherichia coli and Staphylococcus aureus. Alinteri Journal of Agriculture Sciences, 35(2), 144-150. doi: 10.47059/alinteri/V35I2/AJAS20086
[7] Kaplowitz, N. (2005). Idiosyncratic drug hepatotoxicity. *Nature Reviews Drug Discovery*, *4*(6), 489-499.
[8] Larrey, D. (2000). Drug-induced liver diseases. Journal of Hepatology, 32, 77-88.
[9] Lucena, M. I., Andrade, R. J., Kaplowitz, N., García-Cortes, M., Fernández, M. C., Romero-Gomez, M., ... & Spanish Group for the Study of Drug-Induced Liver Disease. (2009). Phenotypic characterization of drug-induced liver injury: The influence of age and sex. *Hepatology*, *49*(6), 2001-2009.
[10] McKee, T., & McKee, J. R. (2017). *Biochemistry: The molecular basis of life* (6th ed.). Oxford University Press.
[11] Navarro, V. J., & Senior, J. R. (2006). Drug-related hepatotoxicity. New England Journal of Medicine, 354(7), 731-739.
[12] Salem, M. O. A., & Lakwani, M. A. (2024). Determination of chemical composition and biological activity of flaxseed (Linum usitatissimum) essential oil. Journal of Biometry Studies, 4(2), 91-96.
[13] Salem, M. O. A., Ahmed, G. S., Abuamoud, M. M. M., & Rezgalla, R. Y. M. (2025). Antimicrobial activity of extracts of dandelion (Taraxacum officinale) against Escherichia coli and Staphylococcus aureus: Mechanisms, modern insights, and therapeutic potential. Libyan Journal of Medical and Applied Sciences, 37-40.
[14] Salem, M. O. A., Antimicrobial Activity of Aqueous Methanolic Extract of Lichen (Usnea barbata) Against Escherichia coli and Staphylococcus aureus. (2024). Libyan Journal of Ecological & Environmental Sciences and Technology, 6(1), 19-23. https://doi.org/10.63359/j8639d64
[15] Salem, M., & Salem, I. (2025). Antimicrobial polymers: Mechanisms of action and applications in combating antibiotic resistance. Al-Imad Journal of Humanities and Applied Sciences (AJHAS), 12-15.
[16] Soof, S. A., Sauf, M. A., Salim, A. A., & Salem, M. O. A. (2025). GC-MS Quantification of Bioactive Isothiocyanates in Sinapis alba Essential Oil and Validation of Rapid Bactericidal Kinetics Against Clinically Relevant Pathogens. Scientific Journal for Publishing in Health Research and Technology, 1(2), 86-93. https://doi.org/10.65420/sjphrt.v1i2.20
[17] Tujios, S., & Fontana, R. J. (2011). Mechanisms of drug-induced liver injury: From bedside to bench. Nature Reviews Gastroenterology & Hepatology, 8(4), 202-211.
[18] World Health Organization (WHO). (2018). *Safety monitoring of medicinal products: Guidelines for laboratory biomarkers*. WHO Press.
[19] Zimmerman, H. J. (1999). *Hepatotoxicity: The adverse effects of drugs and other chemicals on the liver* (2nd ed.). Lippincott Williams & Wilkins.

