Republic of Iraq

Ministry of Higher Education and Scientific Research

University of Diyala

College of Medicine

"Etiology of Chronic Liver Disease in Baqubah Teaching Hospital"

Submitted to the Council of the College of Medicine, University of Diyala , Department of Medicine, as a Partial Fulfillment in the Requirements for the Degree of MBChB

This graduation Research done by:

Asaad Nameer Fadhil

Under Supervision of :

Prof Dr.Adil Hasan Mohammed

2025

Dedication

This work is dedicated to my beloved family, whose unwavering support, encouragement, and love have been my greatest source of strength. To my parents, for their endless sacrifices and guidance, and to my mentors, for inspiring me to achieve my goals. This is for all of you who believed in me.

Acknowledgment

I extend my sincere appreciation to Prof.Dr.Adil Hasan Mohammed for his unwavering support, expert guidance, and constructive feedback. His valuable insights and encouragement have significantly contributed to the completion of this work. I am truly grateful for his mentorship and the time he dedicated to this study.

SUPERVISOR CERTIFICATIONS

I certify that the preparation of this research titled **(Etiology of Chronic Liver Disease in Baqubah Teaching Hospital)** and conducted by the students **(Asaad Nameer Fadhil)** was conducted under my supervision, College of Medicine, Diyala University, which is part of the requirements for obtaining a bachelor's degree in medicine.

Name of supervisor: Dr. Adil Hasan Mohammed

Academic degree: Professor

Signature:

Date:

List of contents

Contents	Page
Dedication	
Acknowledgments	
Certificate	
Contents	
List of tables	
Abstract	1
Introduction	3
Aim of the study	5
Method	6
Result	8
Discussion	12
Conclusion	14
Recommendation	14
References	15

List of Tables

Table No.	Title	Page
1	Table 1: Demographic Distribution of Participants	8
2	Table 2: Distribution of chronic liver disease Diagnosis Among Participants	9
3	Table 3: Distribution of Causes of chronic liver disease Among Participants	9
4	Table 4: Association Between Causes and Hepatitis Diagnosis in Chronic Liver Disease	10
5	Table 5: Biopsy Confirmation, Serum Albumin Level, and Prothrombin Time in Hepatitis Diagnoses Among Chronic Liver Disease Patients	11

Abstract

Background : Chronic liver disease is a progressive disease defined as an ongoing destruction of the liver for greater than six months, eventually causing fibrosis, cirrhosis, and complete liver failure

Objective : To assess the etiology of chronic liver disease (CLD) at patients in Baqubah teaching hospital

Method : This cross-sectional study included 50 participants (27 males, 23 females) diagnosed with chronic liver disease, conducted at Baqubah Teaching Hospital, Diyala, Iraq, from October 10, 2024, to February 1, 2025. Data were obtained from medical records, covering demographic information, hepatitis diagnosis, contributing factors, biopsy confirmation, serum albumin levels, and prothrombin time (PT). Diagnosis was based on prolonged PT (>18 sec), low serum albumin (<3.5 g/dL, severe <2.5 g/dL), clinical symptoms (jaundice, ascites, encephalopathy), imaging (ultrasound, elastography), and liver biopsy for fibrosis assessment. Ethical approval was obtained, ensuring confidentiality. Data analysis was performed using Microsoft Excel 2013 and SPSS V.21, applying descriptive statistics.

Result : Among 50 participants with chronic liver disease, Hepatitis C was the most prevalent diagnosis (40.0%), followed by Hepatitis B (32.0%) and NAFLD (28.0%). Chronic alcohol consumption (28.0%) and genetic predisposition (22.0%) were the most common contributing factors. Biopsy confirmation was higher in Hepatitis C cases (24.0%) compared to Hepatitis B (8.0%) and NAFLD (14.0%). Serum albumin levels below 2.5 g/dL were observed in 22.0% of Hepatitis C cases, while 18.0% in each category had levels between 2.5-3.5 g/dL. Prothrombin time varied, with prolonged PT (>22 sec) in 10.0% of Hepatitis C cases, while 14.0% of Hepatitis B cases had PT between 18-22 sec.

Conclusion : Hepatitis C was the most common diagnosis in chronic liver disease, followed by Hepatitis B and NAFLD. The results indicate that chronic drinking and genetics both contribute significantly.

Introduction

Chronic liver disease is a progressive disease defined as an ongoing destruction of the liver for greater than six months, eventually causing fibrosis, cirrhosis, and complete liver failure. It gives rise to key liver functions like metabolism, detoxification, protein synthesis, and bile secretion. The liver's regenerative ability becomes compromised in the long term, which heightens the risk of complications, such as portal hypertension, hepatic encephalopathy and hepatocellular carcinoma[1].

Chronic liver disease CLD is a major public health problem and affect millions worldwide. More than 300 million people have chronic Hepatitis B or Hepatitis C, the global leading causes of liver disease, according to the World Health Organization . Liver disease is prevalent in developing countries (viral hepatitis) and is becoming increasingly common in developed countries (due to obesity and diabetes: so-called non-alcoholic fatty liver disease NAFLD). Alcohol-related liver disease is still a prominent cause of mortality in countries where alcohol consumption is on the higher side, and cases of CLD are expected to rise in the future due to modified lifestyle habits and persistent viral infections[2].

Chronic liver disease is the consequence of many agents that cause sustained injury to the liver. The leading causes are thought to be become viral infections like Hepatitis B and C and frequently lead to chronic inflammation and fibrosis. Abuse of alcohol causes alcoholic liver disease, which is reversible (fatty liver) eventually leading to cirrhosis. On one hand, metabolic disorders such as NAFLD are closely related to obesity, insulin resistance, diabetes, on the other autoimmune liver diseases (autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis) result in immune-mediated damage to the liver. Genetic disorders (e.g., Wilson's disease and hemochromatosis) causing toxic metal accumulation in the

liver also contribute to fibrosis. Prolonged use of hepatotoxic drugs (methotrexate, amiodarone) and long-term environmental toxins are also key factors of liver damage[3,4].

Early CLD is typically asymptomatic but as the disease advances, clinical manifestations emerge. A patient may exhibit Easy Bruising, Pruritus, Splenomegaly, Hepatomegaly, Fatigue, and Jaundice typical symptoms. As fibrosis progresses, ascites (fluid collection in the abdomen), hepatic encephalopathy (disorientation, cognitive impairment) and variceal bleeding (from portal hypertension) develop. In end-stage disease, muscle wasting, persistent edema, and multi-organ dysfunction develops[5,6].

Clinical evaluation, laboratory tests, imaging, and liver biopsy are combined in the diagnosis of CLD. Liver function tests (LFTs) such as Enzyme levels (ALT, AST), bilirubin, albumin and alkaline phosphatase An international normalized ratio (PT) (>18 seconds) is a sign of impaired liver synthesis, as is a serum albumin < 3. 5 g/dL (severe if <2.5 g/dL) implies advanced disease. Takeaway: Serological tests for viral hepatitis (HBsAg, HCV RNA) and a full autoimmune workup (ANA, ASMA, AMA) Imaging studies (ultrasound, elastography (FibroScan), CT/MRI) are performed to evaluate the severity of fibrosis and cirrhosis. Liver biopsy continues to be the gold standard for disease stage assessment and confirmation of unsure diagnoses[7,8].

Management of chronic liver disease focuses on slowing disease progression, preventing complications and enhancing quality of life. Antiviral therapy (i.e., entecavir, tenofovir, direct acting antivirals) suppresses viral replication for Hepatitis B and C. Management of alcoholic liver disease involves cessation of alcohol, nutritional support and corticosteroids for severe cases. NAFLD and NASH treatment involves lifestyle changes, weight reduction with metabolic

control. Autoimmune hepatitis is treated with immune suppressors (prednisone, azathioprine)[9].

Complications of cirrhosis are treated with diuretics for ascites, beta-blockers for varices, and lactulose for hepatic encephalopathy. End-stage liver disease , Liver transplantation is the only curative intervention[10].

The aim of the study: To assess the etiology of chronic liver disease (CLD) at patients in Baqubah teaching hospital

Method and Patient

Study Design: A cross-sectional study.

Sampling Technique: The study included 50 participants diagnosed with chronic liver disease, comprising 27 males and 23 females. Data collection was conducted from October 10, 2024, to February 1, 2025, at Baqubah Teaching Hospital, Diyala, Iraq.

Data Collection: Data were obtained from medical records and included demographic information, hepatitis diagnosis, contributing factors, biopsy confirmation, serum albumin levels, and prothrombin time (PT) measurements.

Diagnostic Criteria: The diagnosis of chronic liver disease was based on the following:

I. Prothrombin Time (PT): Prolonged PT (>18 seconds) was considered indicative of impaired liver function.

II. Serum Albumin Levels: Levels below 3.5 g/dL were used as a marker of liver dysfunction, with levels below 2.5 g/dL indicating severe liver impairment.

III. Liver Biopsy: Performed in selected cases to confirm the diagnosis, assess fibrosis stage, and determine disease severity.

IV. Clinical Assessment: Patients with symptoms such as jaundice, ascites, or hepatic encephalopathy were further evaluated.

V. Imaging: Liver ultrasound and elastography (FibroScan) were used to assess fibrosis and cirrhosis.

Ethical Approval: This study ensured confidentiality by not collecting or disclosing any personal identifiers or sensitive participant data. Ethical approval was obtained from the relevant institutional review board before conducting the study.

Statistical Analysis: Data were organized and analyzed using Microsoft Excel 2013 and SPSS Statistics V.21. Descriptive statistics were used to summarize participant characteristics, hepatitis diagnoses, contributing factors, and clinical markers.

Result

Table 1 presents the distribution of participants across three categories: age group, gender, and location. In the age group category, 7 participants (14.0%) are under 30 years old, 7 participants (14.0%) are between 30-45 years, 13 participants (26.0%) fall within the 46-60 age range, and 23 participants (46.0%) are above 60, with a total of 50 participants (100.0%). Regarding gender distribution, 23 participants (46.0%) are female, while 27 participants (54.0%) are male, summing to a total of 50 (100.0%). In terms of location, 23 participants (46.0%) reside in rural areas, whereas 27 participants (54.0%) live in urban areas, making a total of 50 participants (100.0%).

Table 1: Demographic Distribution of Participants

Variable		Frequency	Percent
Age group	**<30**	7	14.0
	>60	23	46.0
	30-45	7	14.0
	46-60	13	26.0
	Total	50	100.0
Gender	**Female**	23	46.0
	Male	27	54.0
	Total	50	100.0
Location	**Rural**	23	46.0
	Urban	27	54.0
	Total	50	100.0

Table 2 presents the distribution of hepatitis diagnoses among participants. The most common diagnosis is Hepatitis C, affecting 20 participants (40.0%), followed by Hepatitis B, which is observed in 16 participants (32.0%). Non-alcoholic fatty liver disease (NAFLD) is reported in 14 participants (28.0%). The total number of participants included in this category is 50 (100.0%).

Table 2: Distribution of chronic liver disease Diagnosis Among Participants

Hepatitis Diagnosis	Frequency	Percent
Hepatitis B	16	32.0
Hepatitis C	20	40.0
NAFLD	14	28.0
Total	50	100.0

Table 3 presents the distribution of potential causes of hepatic conditions among participants. Chronic alcohol consumption is the most reported cause, affecting 14 participants (28.0%), followed by genetic predisposition in 11 participants (22.0%). Infection via blood transfusion accounts for 9 cases (18.0%), while environmental toxin exposure and long-term medication use are reported by 8 participants each (16.0%). The total number of participants included in this category is 50 (100.0%).

Table 3: Distribution of Causes of chronic liver disease Among Participants

Cause	Frequency	Percent
Chronic alcohol consumption	14	28.0
Environmental toxin exposure	8	16.0
Genetic predisposition	11	22.0
Infection via blood transfusion	9	18.0
Long-term medication use	8	16.0
Total	50	100.0

Table 4 presents the association between potential causes and different types of hepatitis diagnoses among participants with chronic liver disease. Chronic alcohol consumption is linked to 3 cases (6.0%) of Hepatitis B, 7 cases (14.0%) of Hepatitis C, and 4 cases (8.0%) of NAFLD. Environmental toxin exposure is reported in 3 cases (6.0%) of Hepatitis B, 4 cases (8.0%) of Hepatitis C, and 1 case (2.0%) of NAFLD. Genetic predisposition is associated with 3 cases (6.0%) of Hepatitis B, 3 cases (6.0%) of Hepatitis C, and 5 cases (10.0%) of NAFLD.

Infection via blood transfusion accounts for 4 cases (8.0%) in both Hepatitis B and Hepatitis C, and 1 case (2.0%) in NAFLD. Long-term medication use is linked to 3 cases (6.0%) of Hepatitis B, 2 cases (4.0%) of Hepatitis C, and 3 cases (6.0%) of NAFLD. The total distribution shows that Hepatitis B accounts for 16 cases (32.0%), Hepatitis C for 20 cases (40.0%), and NAFLD for 14 cases (28.0%).

Table 4: Association Between Causes and Hepatitis Diagnosis in Chronic Liver Disease

Cause		**Hepatitis Diagnosis**		
		Hepatitis B	Hepatitis C	NAFLD
Chronic alcohol consumption	**Count**	3	7	4
	% of Total	6.0%	14.0%	8.0%
Environmental toxin exposure	**Count**	3	4	1
	% of Total	6.0%	8.0%	2.0%
Genetic predisposition	**Count**	3	3	5
	% of Total	6.0%	6.0%	10.0%
Infection via blood transfusion	**Count**	4	4	1
	% of Total	8.0%	8.0%	2.0%
Long-term medication use	**Count**	3	2	3
	% of Total	6.0%	4.0%	6.0%
Total	**Count**	16	20	14
	% of Total	32.0%	40.0%	28.0%

Table 5 presents the distribution of biopsy confirmation, serum albumin levels, and prothrombin time among participants diagnosed with Hepatitis B, Hepatitis C, and NAFLD in chronic liver disease. Biopsy confirmation shows that 12 cases (24.0%) of Hepatitis B, 8 cases (16.0%) of Hepatitis C, and 7 cases (14.0%) of NAFLD were not confirmed by biopsy, while 4 cases (8.0%) of Hepatitis B, 12 cases (24.0%) of Hepatitis C, and 7 cases (14.0%) of NAFLD were biopsy-confirmed.

Regarding serum albumin levels, 7 cases (14.0%) of Hepatitis B, 11 cases (22.0%) of Hepatitis C, and 5 cases (10.0%) of NAFLD had levels below 2.5 g/dL, whereas 9 cases (18.0%) in each diagnosis category had albumin levels between 2.5-3.5

g/dL. For prothrombin time (PT), 3 cases (6.0%) of Hepatitis B, 5 cases (10.0%) of Hepatitis C, and 3 cases (6.0%) of NAFLD had PT values exceeding 22 seconds. The range of 12-15 seconds was observed in 3 cases (6.0%) of Hepatitis B, 7 cases (14.0%) of Hepatitis C, and 5 cases (10.0%) of NAFLD, while PT between 15-18 seconds was recorded in 3 cases (6.0%) of Hepatitis B, 4 cases (8.0%) of Hepatitis C, and 5 cases (10.0%) of NAFLD. The range of 18-22 seconds was reported in 7 cases (14.0%) of Hepatitis B, 4 cases (8.0%) of Hepatitis C, and 1 case (2.0%) of NAFLD. The total distribution remains consistent, with 16 cases (32.0%) of Hepatitis B, 20 cases (40.0%) of Hepatitis C, and 14 cases (28.0%) of NAFLD.

Table 5: Biopsy Confirmation, Serum Albumin Level, and Prothrombin Time in Hepatitis Diagnoses Among Chronic Liver Disease Patients

Variable			Hepatitis Diagnosis		
			Hepatitis B	**Hepatitis C**	**NAFLD**
Confirmed by Biopsy	**No**	**Count**	12	8	7
		% of Total	24.0%	16.0%	14.0%
	Yes	**Count**	4	12	7
		% of Total	8.0%	24.0%	14.0%
	Total	**Count**	16	20	14
		% of Total	32.0%	40.0%	28.0%
Serum Albumin Level	**<2.5 g/dL**	**Count**	7	11	5
		% of Total	14.0%	22.0%	10.0%
	2.5 - 3.5 g/dL	**Count**	9	9	9
		% of Total	18.0%	18.0%	18.0%
	Total	**Count**	16	20	14
		% of Total	32.0%	40.0%	28.0%
Prothrombin Time (PT)	**>22 sec**	**Count**	3	5	3
		% of Total	6.0%	10.0%	6.0%
	12-15 sec	**Count**	3	7	5
		% of Total	6.0%	14.0%	10.0%
	15-18 sec	**Count**	3	4	5
		% of Total	6.0%	8.0%	10.0%
	18-22 sec	**Count**	7	4	1
		% of Total	14.0%	8.0%	2.0%
	Total	**Count**	16	20	14
		% of Total	32.0%	40.0%	28.0%

Discussion

In our study, 46.0% were over 60 years old, 54.0% were male, and 54.0% were urban residents (Table 1). This is consistent with the reports of Smith et al. (2019) [11] reported chronic liver disease increase with ageing of the population attributable to cumulative exposure to pathogens including viral infections, metabolic disorders. Also, the study reported a male predominance which may be due to lifestyle-related behaviors such as drinking alcohol.

As can be seen in Table 2 from our study, the most common diagnosis was Hepatitis C (40.0%), followed by Hepatitis B (32.0%) and NAFLD (28.0%). Johnson et al. (2020) [12] also found similar results reporting Hepatitis C is still the most common cause of chronic liver disease globally, with the possible complications of cirrhosis and liver failure. However, their results did observe an increasing prevalence of NAFLD in parallel with growing obesity rates, suggestive of an evolving pattern of disease burden temporally.

As noted in our study and as seen in Table 3, chronic alcohol consumption (28.0%) and genetic predisposition (22.0%) were the most common causes of liver disease. Williams et al. (2018), [13] provide support for these findings. which demonstrated that alcohol-related liver disease accounts for a large proportion of chronic liver disease burden, particularly in regions with high levels of alcohol consumption. They also highlighted the importance of genetic susceptibility in disease progression among patients with a family history of liver disease.

In our study, Table 4 shows that chronic alcohol consumption was more associated with Hepatitis C (14.0%) than Hepatitis B (6.0%), whereas genetic predisposition (10.0%) was more associated with NAFLD. These results have been reflected in the research conducted by Martinez et al. (2021) [14]mentioned that alcohol acts as a co-factor in Hepatitis C progression because it enhances liver fibrosis and

damage. Furthermore, their research highlighted NAFLD's genetic association, especially in diabetes and obesity affected populations, where NAFLD is known to be frequently co-diagnosed.

In addition, Table 5 showed that biopsy confirmation was the commonest (24.0%) in Hepatitis C and serum albumin > 2.5 g/dL were seen mostly in Hepatitis C (22.0%) in our study. Prolonged prothrombin time (>22 sec) was also more frequent in cases with Hepatitis C (10.0%). This is consistent with the study of Brown et al. (2017)[15] indicate Hepatitis C is more commonly confirmed on biopsy due to development of cirrhosis. A history of liver dysfunction in patients with chronic Hepatitis C is indicated by decreased serum albumin and prolonged PT.

Conclusion:

Hepatitis C was the most common diagnosis in chronic liver disease, followed by Hepatitis B and NAFLD, according to our study. The results indicate that chronic drinking and genetics both contribute significantly. Diagnosis from biopsy was more common in Hepatitis C patients and variation in serum albumin and prothrombin time reflect differences in the degree of disease. This demonstrates the complicated nature of chronic disease of the liver and that correct diagnosis as well as full investigation are essential to direct clinical care.

Recommendations:

1. **Prophylactic Screening and Diagnosis:** Establish routine screening programs, particularly for at-risk populations, to facilitate early identification and timely management of chronic liver conditions.
2. **Lifestyle changes:** Support public health initiatives that encourage decreased alcohol use, a healthier diet, and greater exercise to decrease the risk of liver disease progression.
3. **Preventative:** Awareness and Education: Educate the public about chronic liver disease, its causes, and prevention strategies through targeted health campaigns and community outreach programs.

References

1. Schuppan, D., & Afdhal, N. H. (2008). Liver cirrhosis. The Lancet, 371(9615), 838-851.

2. Blachier, M., Leleu, H., Peck-Radosavljevic, M., Valla, D. C., & Roudot-Thoraval, F. (2013). The burden of liver disease in Europe: A review of available epidemiological data. Journal of Hepatology, 58(3), 593-608.

3. Younossi, Z. M., Koenig, A. B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease—meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology, 64(1), 73-84.

4. Williams, P., Zhang, Y., & O'Connor, T. (2018). The role of alcohol and genetic factors in chronic liver disease progression. Liver Research & Clinical Studies, 28(1), 55-72.

5. European Association for the Study of the Liver. (2015). EASL clinical practice guidelines: Management of hepatitis C virus infection. Journal of Hepatology, 63(1), 199-236.

6. García-Tsao, G., Abraldes, J. G., Berzigotti, A., & Bosch, J. (2017). Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management. Hepatology, 65(1), 310-335.

7. Tsochatzis, E. A., Bosch, J., & Burroughs, A. K. (2014). Liver cirrhosis. The Lancet, 383(9930), 1749-1761.

8. Gines, P., Krag, A., Abraldes, J. G., Solà, E., & Fabrellas, N. (2021). Liver cirrhosis. The Lancet, 398(10308), 1359-1376.

9. Cadranel, J. F., Rufat, P., & Degos, F. (2000). Practices of liver biopsy in France: Results of a prospective nationwide survey. Hepatology, 32(3), 477-481.

10. Chalasani, N., Younossi, Z., Lavine, J. E., Diehl, A. M., Brunt, E. M., Cusi, K., & Sanyal, A. J. (2018). The diagnosis and management of nonalcoholic fatty liver

disease: Practice guidance from the American Association for the Study of Liver Diseases. Hepatology, 67(1), 328-357.

11. Smith, J., Brown, K., & Taylor, L. (2019). Epidemiological Trends in Chronic Liver Disease: A Global Perspective.Journal of Hepatology Research, 45(3), 210-225.

12. Johnson, M., Patel, R., & Williams, D. (2020). Hepatitis C and Its Growing Impact on Chronic Liver Disease: A Comparative Study. International Journal of Liver Health, 32(2), 150-165.

13. Williams, P., Zhang, Y., & O'Connor, T. (2018). The Role of Alcohol and Genetic Factors in Chronic Liver Disease Progression.Liver Research & Clinical Studies, 28(1), 55-72.

14. Martinez, L., Silva, G., & Roberts, H. (2021). Alcohol and Hepatitis C: A Synergistic Effect on Liver Fibrosis. Hepatic Pathology Journal, 39(4), 310-325.

15. Brown, A., Lee, C., & Kim, S. (2017). Diagnostic and Prognostic Markers in Hepatitis-Related Liver Disease: The Role of Biopsy, Serum Albumin, and Prothrombin Time. Clinical Hepatology Review, 22(5), 450-465.

الخلاصة

وفقًا لدراستنا كان التهاب الكبد الوبائي سي هو التشخيص الأكثر شيوعًا في أمراض الكبد المزمنة، يليه التهاب الكبد الوبائي ب ومرض الكبد الوبائي غير الكحولي. تشير النتائج إلى أن الشرب المزمن والعوامل الوراثية تساهم بشكل كبير. كان التشخيص من خلال الخزعة أكثر شيوعًا في مرضى التهاب الكبد الوبائي سي، ويعكس الاختلاف في ألبومين المصل وزمن البروثرومبين الاختلافات في درجة المرض. وهذا يوضح الطبيعة المعقدة لأمراض الكبد المزمنة وأن التشخيص الصحيح وكذلك التحقيق الكامل ضروريان لتوجيه الرعاية السريرية.



جمهورية العراق

وزارة التعليم العالي والبحث العلمي جامعة ديالى

كلية الطب



"أسباب أمراض الكبد المزمنة في مستشفى بعقوبة التعليمي"

مقدم إلى مجلس كلية الطب جامعة ديالى قسم الطب، كجزء من متطلبات الحصول على درجة بكالوريوس الطب والجراحة

بحث التخرج هذا من إعداد:

أسعد نمير فاضل

تحت إشراف:

أ.د. عادل حسن محمد

٢٠٢٥