SCHOOL OF MEDICINE

DEPARTMENT OF INTERNAL MEDICINE

BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMONG ALCOHOL ABUSERS ATTENDING REHABILITATION CENTRES IN DAR ES SALAAM.

By

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A DISSERTATION SUBMITTED IN (PARTIAL) FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF MEDICINE (INTERNAL MEDICINE) AT

KAIRUKI UNIVERSITY

2025

CERTIFICATION

The undersigned certifies that he has read and at this moment recommends for submission a Dissertation entitled "**BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMONG ALCOHOL ABUSERS ATTENDING REHABILITATION CENTRES IN DAR ES SALAAM**" in partial fulfillment of the degree of Masters of Medicine in Internal Medicine of Kairuki University.

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I, DR. JOYCE E. SABUKA, declare that this dissertation is my original work and has not been presented and will not be presented to any other university for a similar or any other degree award.

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Date 24/11/2025

ACKNOWLEDGMENTS

I thank Almighty God for granting me strength and good health throughout my life. I am deeply grateful to all who contributed to the completion of this dissertation.

Foremost, I extend my sincere appreciation to my advisor, Prof. Yassin Mgonda, for his steadfast support, insightful guidance, and invaluable feedback. I also thank my co-supervisors, Dr. Warles C. Lwabukuna and Dr. Alice Gwambegu, for their constructive critiques and encouragement, which greatly enhanced this work.

My gratitude goes to the Kairuki University Director of Postgraduate Studies and the Research Institute for their support. Special thanks to Mr. Nichodem S. Mgonzo and Mr. Lemmygius D. Balilemwa, the skilled laboratory technicians whose expertise ensured the accuracy of the study's findings.

I am profoundly thankful to my family for their unwavering love, patience, and support, which sustained me throughout this journey. I also appreciate the cooperation of the Muungano Recovery Community–MRC Centers, DAPO Recovery Houses, and Pillimissana Sober Houses for granting permission to conduct this study and assisting during data collection.

To the study participants, thank you for your willingness and cooperation. Lastly, I acknowledge the authors and scholars whose work informed and inspired my research.

To everyone who played a role in shaping this dissertation—your support is deeply appreciated.

DEDICATION

To my beloved sister, Dr. Hellen Sabuka, my devoted brother, Mr. Joseph Sabuka, and my cherished son, Elian Sabuka Emanuel, your unwavering support, boundless love, and unshakable belief in me have been my greatest wealth. This journey, in every sense, is owed to you.

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ABBREVIATIONS

AFLD	Alcoholic-fatty Liver Disease
ALD	Alcohol Liver Disease
ALP	Alkaline Phosphatase
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
AUD	Alcohol Use Disorder
GGT	Gamma-Glutamyl Transferase
HBV	Hepatitis B Virus
HCC	Hepatocellular Carcinoma
HCV	Hepatitis C Virus
HED	Heavy Episodic Drinking
HSCs	Hepatic Stellate Cells
INR	International Normalized Ratio
ROS	Reactive Oxygen Species
SNPs	Single Nucleotide Polymorphisms
TNF-α	Tumor Necrosis Factor Alpha
USA	United States of America
WHO	World Health Organization

OPERATIONAL DEFINITION

Alcohol abuse/Alcohol use disorder is defined as a pattern of drinking that causes physical or mental health harm or disrupts daily life within a year[1]. It involves consuming more than 21 units of alcohol per week for men and 14 units per week for women[2–4], each unit contains 10 grams of pure alcohol, with a specific gravity of 0.8[5] and diagnosis is often made using the CAGE questionnaire, a standardized and validated tool consisting of four questions designed to screen accurately for potential alcohol-related problems[3,6,7].

A rehabilitation center refers to a facility that provides services to help individuals recover from illnesses, injuries, or conditions that affect their physical, mental, or cognitive abilities[1].

Alcoholic Liver Disease refers to a condition caused by excessive alcohol consumption, leading to liver damage. It includes disorders such as fatty liver (hepatic steatosis), alcoholic hepatitis (liver inflammation), and alcoholic cirrhosis (severe liver scarring)[3].

An alcohol abuser is an individual currently engaged in excessive alcohol consumption and receiving care at rehabilitation centers in Dar es Salaam.

ABSTRACT

Background:

Alcoholic Liver Disease (ALD) is a significant public health concern in Tanzania, driven by high rates of Alcohol Use Disorder (AUD). ALD contributes substantially to cirrhosis, liver failure, and hepatocellular carcinoma, straining the healthcare system. Despite its impact, liver biochemical patterns such as Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Gamma-Glutamyl Transferase (GGT), bilirubin, and albumin remain poorly characterized among Tanzanian alcohol abusers, especially those in rehabilitation centers where mental health often overshadows physical assessments.

Objective: To determine the distribution of biochemical patterns of ALD among alcohol abusers attending rehabilitation centers in Dar es Salaam.

Methodology: A cross-sectional descriptive study was conducted from August 2024 to July 2025. A multi-stage, stratified sampling design was employed to select participants from rehabilitation centers in Dar es Salaam. Three centers were chosen using simple random and proportional stratified sampling, and participants were recruited through consecutive sampling. Socio-demographic and clinical data were collected using structured questionnaires. Venous blood samples were tested for ALT, AST, GGT, bilirubin, albumin, HBsAg, and anti-HCV using a Mindray BS-240 analyzer. Data were analyzed using SPSS version 23, with a p-value <0.05 considered statistically significant.

Results: Of 168 analyzed participants, the mean age was 37.8 years (±9.97), and 147 (87.5%) were male. Most had secondary 73 (43.5%) and university education 61 (36.3%).

Early alcohol initiation was common (median age: 14 years). Alcoholic hepatitis (AH) was diagnosed in 19 participants (11.3%). Elevated AST showed a statistically significant

association with higher income (p=0.011). ALT elevation was significantly more common in participants without AH than with AH (36 [24.2%] vs. 1 [5.3%], p=0.047. Conversely, GGT (9 [47.4%]) and AST (6 [31.6%]) were the most elevated markers in the AH group.

Conclusion: This study reveals an evolving Alcoholic Liver Disease profile in Tanzania, which increasingly affects educated, middle-aged, and affluent males. Early alcohol initiation and consumption of high-potency spirits contribute to liver injury. Elevated AST and GGT with normal ALT characterize AH, underscoring the limitations of single biomarkers and the need for integrated diagnostics using AST: ALT ratios and clinical assessment.

Recommendation: Integrate multimodal liver screening into rehabilitation and primary care services. Enforce stricter alcohol control policies, promote targeted public health education, and provide socioeconomic support. Longitudinal cohort studies are essential to understand ALD progression and guide tailored interventions.

CHAPTER ONE

1.0 INTRODUCTION AND BACKGROUND INFORMATION

Alcoholic Liver Disease (ALD) from excessive alcohol consumption poses a significant social and public health challenge globally, contributing substantially to the disease burden worldwide[4]. Alcoholic beverages contain ethanol, a psychoactive and toxic substance capable of leading to dependence[2]. Alcohol dependence, the most severe form of alcohol use disorder, is commonly referred to as "alcoholism"[3].

The risk associated with alcohol consumption varies depending on factors such as the amount consumed, drinking frequency, individual health status, age, sex, and other personal characteristics[3,5]. Even low levels of alcohol intake can pose health risks, but most alcohol-related harms arise from episodic or continuous heavy drinking[3,7].

Excessive alcohol consumption is linked to more than 200 diseases, injuries, and health conditions[3]. Excessive long-term alcohol use is a major cause of multisystem organ pathology. Hepatotoxicity is a predominant consequence, attributable to the liver's role as the chief organ for ethanol metabolism[8,9]. Chronic alcohol use progressively damages liver tissues, leading to conditions such as alcohol-fatty liver disease (AFLD), alcoholic hepatitis, fibrosis, and cirrhosis[8].

This study aims to assess the biochemical patterns of alcoholic liver disease among alcohol abusers attending rehabilitation centers in Dar es Salaam.

1.1.1 LIVER DISEASE

Liver diseases encompass a wide range of conditions that affect the structure and function of the liver. Liver diseases can be broadly classified into two categories: Acute liver disease, characterized by the rapid onset of liver dysfunction, often caused by viral hepatitis, drug toxicity, or ischemic injury. Acute liver disease typically resolves with appropriate treatment, and chronic liver disease involves persistent liver damage lasting over six months[10], often resulting from viral infections (e.g., hepatitis B and C), alcohol use, or non-alcoholic fatty liver disease (NAFLD). Chronic liver disease can progress to cirrhosis, liver failure, and hepatocellular carcinoma[11].

1.1.2 ALCOHOLIC LIVER DISEASE

Alcoholic liver disease (ALD) refers to liver damage caused by excessive alcohol consumption. It encompasses a spectrum of conditions ranging from steatosis and steatohepatitis to progressive fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). ALD also includes the unique presentation of alcohol-associated hepatitis (AH), which, in severe forms, may develop into acute-on-chronic liver failure[12–16].

Notably, while 90–100% of chronic heavy alcohol consumers develop alcoholic fatty liver (AFL), only 10–35% progress to alcoholic steatohepatitis (ASH), and even fewer (8–20%) develop alcoholic cirrhosis (AC). The variation in progression may be attributed to the interplay of genetic, epigenetic, and environmental factors[17]. ALD is considered one of the leading causes of liver disease-related mortality worldwide[18].

1.1.3 EPIDEMIOLOGY OF ALCOHOLIC LIVER DISEASE

Alcoholic Liver Disease is a global health issue that significantly contributes to morbidity and mortality. The epidemiology of ALD varies based on geographic, cultural, and socioeconomic factors, as well as patterns of alcohol use[19].

The World Health Organization (WHO) estimates that the highest prevalence of alcohol consumption and addiction occurs in Europe and the Americas and while the lowest levels are observed in the Eastern Mediterranean and Southeast Asia regions[19]. In 2018, approximately 2.3 billion people were current alcohol drinkers, representing about 43% of the global adult population[20].

Although overall alcohol consumption in Africa is lower compared to regions like Europe or the Americas, harmful drinking patterns, such as binge drinking, and alcohol-related health burdens are significant[2,3,21]. Countries such as South Africa, Nigeria, and Uganda report higher rates of alcohol-related diseases, injuries, and deaths. According to the WHO (Global Status Report on Alcohol and Health-2018), about 32% of the adult population (aged 15 and older) in Africa consumes alcohol, with a per capita alcohol consumption of 6.0 liters. Alcohol-related liver diseases contribute to 3.3% of global deaths from liver conditions[21]. Global data from 2019 indicates that the prevalence of Alcohol Use Disorder (AUD) in Africa ranges from 2.9% to 4.7% of the adult population. Higher rates are observed in Southern and Central African countries, with estimates around 6% for men and 2.5% for women in South Africa[22].

In Tanzania, the prevalence of AUD in 2016 was 6.8%, nearly twice the average for Africa. Additionally, alcohol use prevalence among the adult population is approximately

23.7%, with higher consumption rates among men (33.9%) compared to women (14.1%)[22].

These findings underscore the urgent need for stronger policies and interventions to address alcohol-related harms effectively.

1.1.4 PATHOPHYSIOLOGY OF ALCOHOLIC LIVER DISEASE

The pathogenesis of Alcoholic liver disease (ALD) follows a well-established sequence of stages, starting with simple liver steatosis, advancing to steatohepatitis (characterized by steatosis with inflammation), followed by cirrhosis (marked by advanced liver fibrosis), and, in severe cases, culminating in hepatocellular carcinoma (HCC)[23].

Alcohol (ethanol, the main component in alcoholic beverages) is a direct hepatotoxic and rapidly absorbed in the upper gastrointestinal tract, and is predominantly approximately 80% metabolized in the liver. Ethanol reaches the liver through the portal vein, and most of the ethanol is oxidized via alcohol dehydrogenase 1 (ADH1) into acetaldehyde in hepatocytes, causing direct cellular toxic effects. Chronic alcohol consumption induces the expression of a second ethanol-metabolizing enzyme, CYP2E1, which also converts ethanol into acetaldehyde (a toxic metabolite)and generates reactive oxygen species (ROS), resulting in further injury of hepatocytes[24], leading to steatosis as well as further inflammation, fibrosis, and ultimately liver cancer via lipid peroxidation and DNA damage[25].

Acetaldehyde is then oxidized into acetate via acetaldehyde dehydrogenase (ALDH). Acetate is converted into acetyl-coenzyme A (CoA), contributing to fatty acid and triglyceride synthesis. Alcohol, in part through epigenetic changes, increases the

expression of genes involved in lipogenesis, while genes involved in fatty acid transport and oxidation are suppressed. Alcohol also increases the ratio of reduced nicotinamide adenine dinucleotide (NAD) to oxidized NAD (NADH/NAD+) in hepatocytes, which further reduces mitochondrial β-oxidation. Alcohol can increase fatty acid mobilization in adipose tissue and the intestine, which will lead to hepatic accumulation of fatty acids and increased hepatic steatosis. Fatty liver is the initial and most common histologic response to hepatotoxic stimuli, including excessive alcohol ingestion. The accumulation of fat within the perivenular hepatocytes coincides with the location of alcohol dehydrogenase, the major enzyme responsible for alcohol metabolism. Continuing alcohol ingestion results in fat accumulation throughout the entire hepatic lobule[11]. Pro-inflammatory cytokines are believed to play a role in hepatic damage associated with alcoholic hepatitis. Increased gut permeability allows endotoxins to enter the bloodstream, triggering the release of cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1, IL-2, and IL-8 from immune cells. These cytokines, released into the portal circulation, contribute to inflammation, activation of the hepatic stellate cells (HSCs), and the development of liver fibrosis[26].

1.1.5 CLINICAL FEATURES OF ALCOHOLIC LIVER DISEASE

In the early stages, patients with ALD exhibit subtle or no abnormal physical findings[27]. It is usually not until the development of advanced liver disease that the stigmata of chronic liver disease become apparent[28].

Symptoms and Signs include Tiredness, Malnutrition, and sarcopenia[27]. Abdomen: abdominal discomfort, hepatomegaly, splenomegaly, caput medusae, ascites with weight gain, abdominal pain, and shortness of breath. Skin: spider angioma[12,27], palmar

erythema[12], jaundice, ecchymoses, Eyes: icteric sclerae. Hands: Dupuytren's contracture[27]. Face: rhinophyma, Reproductive system: gynecomastia[11], gonadal atrophy, loss of libido, amenorrhea and Neurologic signs including Peripheral neuropathy, Alcohol withdrawal: tachycardia, agitation, tremor, seizures, delirium, Hepatic encephalopathy[12,27]; asterixis, forgetfulness, inversion of sleep/wake pattern, altered consciousness, confusion, lethargy, coma[11], and Wernicke-Korsakoff syndrome; Ocular abnormalities, Mental status changes, Incoordination of gait, and trunk ataxia[29].

1.1.6 DIAGNOSIS OF ALCOHOLIC LIVER DISEASE

Diagnosis of alcoholic liver disease (ALD) involves detailed history, CAGE scoring, clinical findings, and laboratory investigations, particularly elevated transaminase levels. Imaging and liver biopsy may aid in unclear cases, with histological features confirming the diagnosis[30]. The amount of alcohol intake could be registered by using standard drink units[31]. Early ALD is usually discovered during routine health examinations when liver enzyme levels are found to be elevated[32].

The diagnosis of early ALD (alcoholic fatty liver disease) in patients with AUD requires the presence of hepatic steatosis, an AST level elevated above ALT, and a serum bilirubin concentration of less than 3 mg/dL. The measurements of GGT, ALT, AST levels, and mean corpuscular volume (MCV) are the most frequently used biochemical markers for early detection of ALD[33]. Patients with simple hepatic steatosis can present with normal liver function tests[23,29] and /or elevations in aspartate aminotransferase (AST) and alanine aminotransferase (ALT), which are typically mild, with AST predominating. γ-Glutamyl transpeptidase (GGT) may be elevated, whereas serum bilirubin and International Normalized Ratio (INR) generally remain within normal limits[33].

Alcoholic Hepatitis reveals neutrophilia, hyperbilirubinemia (>50 µmol/L (>2.92 mg/dL)), serum levels of AST greater than twice the upper limit of normal range, AST >50 IU/ml, although rarely above 300 IU/ml[11], with an AST/ ALT ratio typically greater than 1.5. The ratio of aspartate aminotransferase (AST) to alanine aminotransferase (ALT) level is generally greater than 2, which is due to reduced hepatic ALT activity and increased hepatic mitochondrial aspartate aminotransferase. Ratios greater than 3 are highly suggestive of ALD[27].

In severe cases, prolonged prothrombin time, increased international normalized ratio (INR), hypoalbuminemia, and decreased platelet count are frequently observed[31,33].

Imaging techniques, such as abdominal ultrasound and transient elastography, assist in assessing liver condition[30]. A liver biopsy may aid in unclear cases, with histological features to evaluate the extent of liver damage and confirm the diagnosis[27].

1.1.7 MANAGEMENT OF ALCOHOLIC LIVER DISEASE.

Identifying both modifiable risk factors, such as heavy drinking and metabolic syndromes, and non-modifiable risk factors, like genetic predispositions, is essential for effective disease management[34]. Additionally, assessing and treating alcohol use with behavioral therapy and pharmacotherapeutic approaches, providing nutritional support, and optimizing liver disease modifiers form the cornerstone of management[34,35].

Early intervention improves disease outcomes. While the stage of fibrosis significantly impacts survival outcomes, abstinence from alcohol greatly enhances survival rates for both compensated and decompensated ALD[36]. Therefore, abstinence remains the core clinical principle in managing ALD to improve patient prognosis and survival[35,36] even after severe and moderate Alcoholic Hepatitis[37].

Corticosteroid therapy, including prednisolone, has been widely used for moderate to severe alcoholic hepatitis, provided there are no contraindications. This is due to its effectiveness in suppressing the immune response and reducing proinflammatory cytokines. The use of intravenous N-acetylcysteine as an adjunct to corticosteroids is strongly recommended in patients with severe alcoholic Hepatitis[37].

Furthermore, nutritional deficiencies in vitamins and minerals are prevalent in Alcoholic Liver Disease (ALD) and may contribute to its pathogenesis. Therefore, a comprehensive nutritional evaluation and aggressive intervention with oral or enteral supplementation are imperative in the management of alcoholic hepatitis and advanced ALD[9,37].

In advanced cases, liver transplantation is considered the final treatment option[9]. However, for patients with severe alcoholic hepatitis who do not respond to corticosteroids and are ineligible for early liver transplantation, those with four or more organ failures may benefit from palliative therapy[37].

This study focuses on assessing the biochemical markers of ALD among alcohol abusers in Dar es Salaam rehabilitation centers. Liver biochemical markers, including ALT, AST, ALP, GGT, bilirubin, and albumin, are essential in diagnosing and monitoring liver damage. Their specific patterns provide insights into the severity and type of liver injury, aiding in clinical diagnosis, monitoring, and treatment[38].

1.2 PROBLEM STATEMENT

Alcohol consumption as a social beverage serves both as a source of enjoyment and a facilitator of social interactions and trust among individuals[39]. However, its excessive use can lead to serious health problems, including liver diseases (such as alcohol use disorder), kidney diseases, cardiovascular problems, mental health disorders, and an increased risk of injuries and accidents[37].

Alcohol abuse is accountable for 6.9 % and 2.0% of the global burden of disease for males and females, respectively[3]. In Tanzania, where alcohol use disorder (AUD) rates are twice the African average, excessive alcohol consumption is a major public health concern[40]. In Dar es Salaam, 2023 data shows that over one-third (37.3%) of the population exhibits drinking patterns suggestive of alcohol abuse[15].

Genetic, environmental, social, political, and economic factors play complex roles in excessive alcohol use, varying depending on the socioeconomic status, political and cultural setting. Several contributors to the development of ALD, including genetic predisposition**,** mental health issues such as depression and anxiety, trauma or stress**,** cultural acceptance of alcohol use**,** easy availability and affordability of alcohol, and poor regulation of alcohol consumption. These factors collectively lead to higher consumption rates[4,41,42].

If untreated ,ALD can cause progressive liver damage, resulting in cirrhosis**,** liver failure, and increased risk of liver cancer**,** thereby straining healthcare systems and elevating morbidity and mortality rates[37].In 2016, alcohol use disorder (AUD) accounted for 50% of liver disease deaths among individuals aged 15 and older. AUD also contributes to high healthcare costs due to increased hospital admissions for alcohol-related conditions[15].

Many patients are diagnosed with advanced stages, where treatments can only manage symptoms rather than cure the condition[40,43,44].

Biochemical markers, such as liver enzymes (ALT, AST, and GGT), bilirubin, and albumin, are essential for diagnosing and monitoring liver diseases[38]. However, their patterns among alcohol abusers in Tanzania remain poorly characterized.

Alcohol abusers are often taken to rehabilitation centers for treatment and recovery[1]. Tanzanian rehab centers face significant barriers to liver function testing, with less than 20% routinely conducting LFTs due to cost constraints[45]. Additional systemic challenges such as inadequate infrastructure, staff training gaps, and low patient awareness—further limit ALD screening[46], highlighting critical deficiencies in biochemical monitoring for at-risk populations.

Data suggest that most rehabilitation facilities primarily focus on mental health counseling and psychiatric medications[47], often neglecting routine investigations or check-ups of key biochemical markers such as ALT, AST, GGT, bilirubin, and albumin[48]. Consequently, many alcohol abusers remain undiagnosed and unaware of their liver health. This gap in interpreting biochemical markers may hinder early detection of liver dysfunction, which is vital for effective management and preventing disease progression.

This study aimed to address this gap by evaluating changes in biochemical markers, identifying characteristic patterns, and developing targeted interventions for ALD among alcohol abusers attending rehabilitation centers in Dar es Salaam.

1.3 RATIONALE

This study addresses a critical knowledge gap by defining the biochemical patterns of Alcoholic Liver Disease (ALD) among Tanzanian alcohol abusers in rehabilitation centers. The identification of population-specific biomarkers will enhance the early detection and monitoring of ALD, improving clinical decision-making and patient outcomes[18].

In practice, these findings are pivotal for developing context-appropriate screening tools. Integrating this approach into rehabilitation centers will facilitate early detection and enable targeted interventions, such as lifestyle modifications or specialist referrals[21].

The evidence findings will also support crucial policy reforms, including stricter alcohol regulations and the integration of routine liver screening into national addiction care protocols[17].

Ultimately, by identifying at-risk profiles and promoting early intervention, this research contributes directly to preventing advanced liver disease, thereby alleviating the significant burden of ALD on Tanzania's healthcare system[4,49].

1.4 RESEARCH QUESTIONS

These research questions were

I. How are liver enzymes distributed among alcohol abusers attending rehabilitation facilities in Dar es Salaam?
II. What is the magnitude of Alcoholic Hepatitis among alcohol abusers attending rehabilitation centers in Dar es Salaam?

III. How is the duration of alcohol consumption correlated with levels of GGT, ALT, AST, albumin, and bilirubin among alcohol abusers attending rehabilitation centers in Dar es Salaam?

1.5 OBJECTIVES

1.5.1 BROAD OBJECTIVE

To determine the distribution of biochemical patterns of ALD among alcohol abusers attending rehabilitation centers in Dar es Salaam.

1.5.2 SPECIFIC OBJECTIVES

I. To determine the prevalence of Alcoholic Hepatitis among alcohol abusers attending rehabilitation centers in Dar es Salaam

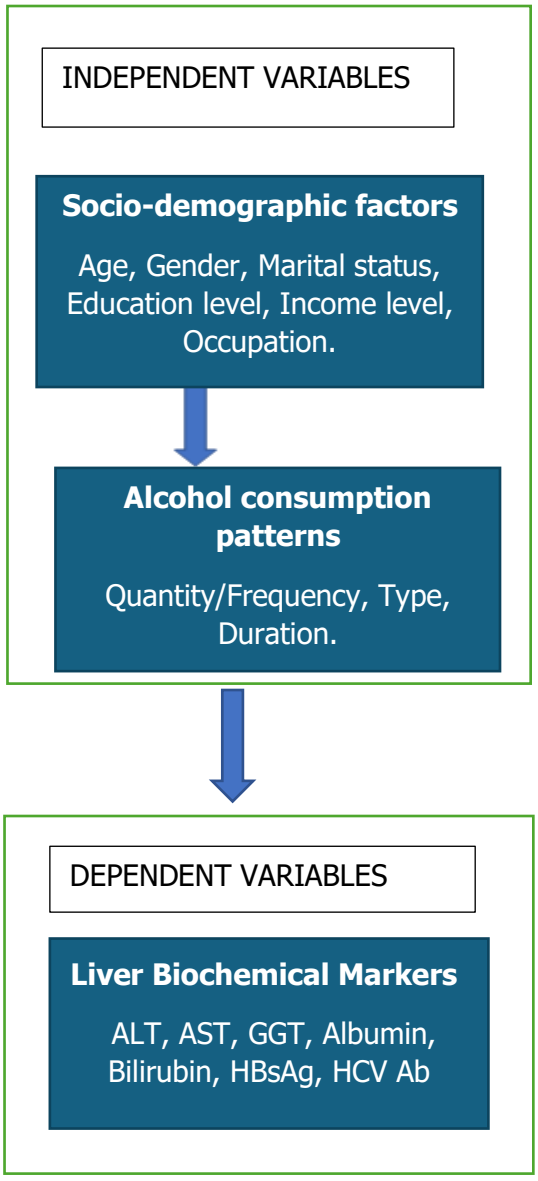
II. To determine the distribution of GGT, ALT, and AST levels by age and gender among alcohol abusers attending rehabilitation centers in Dar es Salaam.

III. To determine the distribution of GGT, ALT, and AST levels by socioeconomic characteristics (occupation, income status) among alcohol abusers attending rehabilitation centers in Dar es Salaam.

IV. To determine a correlation of GGT, ALT, and AST levels with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam.

V. To determine a correlation of albumin and bilirubin levels with the duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam.

Conceptual Framework



CHAPTER TWO

2.0 LITERATURE REVIEW

Alcoholic Liver Disease (ALD) is a major public health issue, contributing substantially to the global disease burden and premature mortality[50]. It accounts for 5.1% of all diseases and injuries worldwide and is one of the primary causes of liver cirrhosis and liver transplantation globally[51,52]. ALD frequently remains undiagnosed until it advances to decompensated cirrhosis, which may lead to a substantial underestimation of the actual prevalence of compensated ALD[53].

The global prevalence of alcoholic liver disease (ALD) is estimated to be 3.5% in the general population, 26.0% among individuals who engage in hazardous drinking, and 55.1% among those with alcohol use disorders[54]. An alternative study reported a global prevalence of 4.8%[55]. However, the prevalence and mortality due to ALD vary across different settings[54]. The majority of European countries have recorded a prevalence of ALD far beyond the global average. For instance, Italy had an ALD prevalence of 16.1%, followed by Sweden (14%) and the United Kingdom (7.2%)[55] according to a study conducted in 2023. Consequently, the prevalence of ALD in the Western world is roughly estimated to be about 6%, almost twice the global average, according to a study done in 2019[56].

In the United States of America (USA), it was estimated that between 2019 and 2040, over 1 million people could die from alcohol-related disease, with 35% of deaths occurring in individuals under 55 years old. However, an intervention capable of achieving a 3.5% annual decrease in high-risk drinking could reverse adverse trends and lower alcohol-related liver disease deaths by as much as 30% by 2040[57]. Nonetheless, this might not be feasible because, despite the availability of cost-effective interventions to reduce alcohol abuse, many countries are not giving the issue the attention it deserves[58]. The prevalence of ALD

varies greatly due to disparities in alcohol consumption trends, habits, and access to healthcare. For instance, the highest prevalence of ALD was recorded in Uganda (11%) according to a study done in 2023[55].

Definitive laboratory tests for diagnosing ALD are lacking, and unlike non-alcoholic liver disease, ALD often presents in advanced stages[59]. Early-stage Alcoholic Liver Disease (ALD) may be diagnosed in individuals with Alcohol Use Disorder (AUD) who present with hepatic steatosis on ultrasonography, a biochemical profile characterized by elevated liver enzymes with an AST-to-ALT ratio >1, serum bilirubin levels <3 mg/dL, and no evidence of alternative etiologies for liver dysfunction[29,33].

Physical examination in ALD often reveals mildly tender hepatomegaly that resolves with abstinence. Laboratory findings typically show minimal aminotransferase elevations (AST > ALT), elevated GGT, and normal bilirubin and INR[60]. Notably, some studies have reported hyperbilirubinemia in patients with various forms of liver disorders. A significant increase in both unconjugated and conjugated serum bilirubin levels has been observed in alcoholic patients[61]. Among biomarkers, serum GGT is the most sensitive and commonly used marker for alcohol consumption[61,62].

Studies indicate that the diagnostic modality significantly influences estimated ALD prevalence, with imaging yielding the highest rate (7.0%), followed by biochemistry (3.8%) and transient elastography (1.3%)65. In contrast, prevalence estimates for alcohol-associated cirrhosis are consistent between imaging and elastography[63].

Only 10–20% of heavy drinkers develop advanced liver disease, suggesting that other factors, including genetic, behavioral, and environmental influences, may contribute. While the risk of ALD increases with alcohol intake, individual susceptibility varies widely. Women are more vulnerable at lower levels of ethanol intake, a phenomenon attributed to

physiological factors such as increased body fat percentage and diminished gastric alcohol dehydrogenase activity.

For example, the intake of 40–80 grams of ethanol per day in males and 20–40 grams per day in females over 10–12 years generally predicts more severe ALD cases[42,64,65].

Although the effect of age on ALD progression remains unclear, older adults tend to be more vulnerable and exhibit greater ethanol-induced impairments than younger individuals[66].

Dietary fat acts as both a macronutrient and a dietary modifier for ALD. The risk of liver injury is further exacerbated in individuals with elevated body mass index (BMI), those who smoke, and concomitant use of hepatotoxic agents (medications and illicit substances)[67], which contribute to synergistic hepatotoxic mechanisms[68].

Alcohol abuse also exacerbates the progression of hepatitis C (HCV) and hepatitis B (HBV) infections[60–62].

Genetic and epigenetic determinants play a pivotal role in the onset and progression of Alcoholic Liver Disease (ALD). Key insights from genome-wide association studies implicate variants in genes for alcohol-metabolizing enzymes, cytokine production, and antioxidant activity in modulating disease risk[69].

In Tanzania, there has yet to be a comprehensive investigation into the liver biochemical profiles of individuals with alcohol use disorders receiving treatment at rehabilitation centers. While general biomarkers for Alcoholic Liver Disease (ALD) have been extensively documented in broader contexts, their specific characteristic patterns in Tanzanian rehabilitation settings remain inadequately explored. The absence of localized biochemical data hinders both understanding and timely identification of ALD among these high-risk population[59,60]. Therefore, this study seeks to elucidate and characterize these biochemical

patterns to enhance early detection and facilitate more effective clinical management. By addressing this critical knowledge gap, the study aims to provide valuable insights into the biochemical profiles associated with ALD in this underserved population.

CHAPTER THREE

3.0 METHODOLOGY

3.1. Study design

This is a cross-sectional descriptive study, which was conducted among alcohol abusers attending rehabilitation centers in Dar es Salaam.

3.2. Study area

The study was conducted at rehabilitation centers in Dar es Salaam. It is located at 6°48′ South and 39°17′ East on the eastern coast of East Africa[70]. According to the 2022 census, it has a population of 5,383,728[71] and is a significant city for industrial, business, government activities, and education. Dar es Salaam comprises five municipalities, namely, Kinondoni (3), Ilala (1), Temeke (0), Ubungo (0), and Kigamboni (9), with a total of 13 rehabilitation centers. These include Hope Again Rehabilitation Centre, Safe Haven Sober House, Mwananyamala Hospital Rehab Centre, Muhimbili National Hospital Rehab Centre, Muungano Recovery Community-MRC Centers, DAPO Recovery Houses, Drug-Free Center, Awaited Rehab Centre, and Pillimissana Sober Houses. They are receiving patients from all five municipalities in Dar es Salaam and across Tanzania.

3.3. General population

Alcoholics in Dar es Salaam.

3.4. Target population

Alcohol abusers attending rehabilitation centers in Dar es Salaam.

3.5. Study population

Selected alcohol abusers attending rehabilitation centers in Dar es Salaam and accepted to participate in the study.

3.6. Eligibility criteria

3.6.1. Inclusion criteria:

I. Current alcohol abusers diagnosed with Alcohol Use Disorder (AUD) based on the CAGE tool and receiving rehabilitation care services.

II. Fully informed consent.

3.6.2. Exclusion criteria:

I. Alcohol abusers with viral liver infections, such as hepatitis B or C infections.

The coexistence of alcohol abuse and chronic hepatitis B or hepatitis C infection exhibits a synergistic effect on hepatotoxicity, significantly accelerating the progression to cirrhosis and the onset of its decompensating complications[60–62].

3.7. Sample size estimation

Using the Kish Leslie formula with an 11% proportion, derived from a study on the prevalence of ALD in Uganda[55], ensured the validity of results and minimized bias by employing a similar proportion.

$$\boldsymbol{n = \frac{z^2 p(1-p)}{e^2}}$$

Whereby;

$\boldsymbol{n}$ = Projected sample size for this study

$\boldsymbol{z}$ = 1.96 corresponding to 95% CI

e = Margin of error is 5% (0.05),

$\boldsymbol{p}$ = expected proportion with the characteristic of interest. For this study p-value = 11%

$\boldsymbol{n}$ = $(1.96)^2$ x 0.11 x (1 - 0.11) / $(0.05)^2$ = 150

The estimated minimum sample size was 150.

3.8. Sampling procedure

The study used a multistage sampling technique across five municipalities in Dar es Salaam (Ilala, Ubungo, Temeke, Kinondoni, and Kigamboni). First, one district was selected by simple random sampling, and then three rehabilitation centers were chosen as strata. A total of 172 participants were enrolled using proportional stratified sampling, as shown in Table 1, with participants recruited consecutively from each stratum based on the given formula:-

$$\text{Number of participants selected from a center} = \frac{\text{Total alcohol abusers attending in a Centre} \times \text{Sample size (150)}}{\text{Total population attending in a center}}$$

Table 1: Number of participants selected from each rehabilitation center

NAME OF REHABILITATION CENTER	TOTAL ALCOHOL ABUSERS ATTENDING IN A CENTRE	TOTAL POPULATION ATTENDING IN A CENTER	NUMBER OF PARTICIPANTS SELECTED FROM A CENTER
MRC KIGAMBONI	200	280	107
DAPO RECOVERY HOUSE	90	228	59
PILIMISSANA SOBER HOUSE	8	200	6
TOTAL			**172**

3.9. Data collection.

A structured data collection tool, comprising the CAGE questionnaire[31], was used. This tool assesses the lifetime occurrence of the following: **C**ut down: Have you ever felt you should cut down on your drinking? **A**nnoyed: Have people annoyed you by criticizing your drinking? **G**uilty: Have you ever felt guilty about drinking? And **E**ye-opener: Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?

Two or more positive responses were indicative of alcohol abuse and dependence[10,11]. The CAGE tool is particularly effective in studies of ALD due to its high diagnostic accuracy, its identification of critical scores for different treatment settings, and its ability to stratify patients' risk levels. This facilitates early intervention and reduces healthcare costs associated with alcohol abuse, making it a practical tool for both clinical and research settings[4,72].

Data Collection Procedures: Data were systematically gathered throughout the study period using a structured questionnaire (Appendices I and II), adapted from a previous study[73]. The principal investigator and research assistant (a trained nurse) collected detailed information on sociodemographic characteristics, alcohol consumption patterns (including CAGE screening), clinical history, and physical examination findings.

Sociodemographic variables included gender, age, residence, marital status, educational level, employment status, and monthly income, categorized according to Tanzania Revenue Authority (TRA) guidelines (Monthly Income from TRA, last accessed 19/06/2025). https://www.tra.go.tz/page/income-tax-for-individuals

Clinical variables included both family and personal history of liver disease, signs of liver dysfunction, and patterns of alcohol use. A standardized physical examination checklist was used to evaluate signs of liver disease, such as jaundice, hepatomegaly, ascites, and spider angiomas[74]**.** All collected data were systematically recorded in the questionnaire for further analysis.

Following written consent from participants, a research assistant (trained laboratory technician) drew a venous blood sample of at least 2.5mls**.** A tourniquet was applied 3-4 inches above the antecubital fossa, the area was cleaned with an alcohol swab, and a 5cc

syringe needle was inserted into the vein. Once the blood was drawn, the tourniquet was released, the needle was removed, and pressure was applied with gauze to stop the bleeding. The needle was then disposed of in a sharps container, a bandage was applied to the puncture site, and the blood sample was placed in yellow tubes labeled with the patient's information[75].

The samples were transported in refrigerated conditions (2°C to 8°C) to the laboratory for serum biochemical testing within 8 hours and were analyzed immediately upon arrival using the Mindray BS-240 clinical chemistry analyzer, manufactured by Shenzhen Mindray Biochemical Electronics in 2019 (China). This analyzer employed photometric detection with a halogen-tungsten lamp to measure the absorbance of sample-reagent mixtures at specific wavelengths, based on Beer-Lambert's law[76]. It utilized various assay methods, including endpoint, kinetic, and turbidimetric techniques. Kinetic assays tracked absorbance changes over time to assess enzyme activity (e.g., ALT, AST, ALP)[77].

The specific tests for this study included ALT, AST, GGT, bilirubin, and albumin, as well as HBsAg (Hepatitis B Surface Antigen) and HCV antibody tests (Hepatitis C Virus Antibody), which were measured and reported by the research assistant (trained laboratory technician)[12].

Alcoholic Liver Disease diagnosis criteria[78].

In alcoholic hepatitis[62] : AST>ALT (usually AST >2X upper limit), ALT may be normal despite significant pathology; so normal levels do not exclude ALD[61,67,79]. Aminotransferase levels are typically <300 IU/L in ALD cases[62,79]. AST/ALT Ratio ≥2:1: strongly suggestive (≥3: highly indicative)[78] of alcoholic hepatitis[62,80]. While AST/ALT <2: does not exclude ALD, and AST/ALT <1: may suggest alcoholic steatosis[67].

GGT is often elevated due to enzyme induction by alcohol[61,67,74,79] .GGT is not alcohol-specific—cannot be used alone for diagnosis. GGT ≥2× normal and AST/ALT ≥2:1 strongly suggests alcohol-related liver damage[62], and increases diagnostic sensitivity of alcoholic hepatitis[61,80].

Serum bilirubin may be significantly elevated,>3 mg/dL[11] in severe acute alcoholic hepatitis and is a marker of hepatocellular dysfunction[61,74].

In severe cases, patients have been showing increased prothrombin time and INR, decreased serum Albumin, and platelet count[74].

**Assessing Comorbid Conditions[61,62]; ** Hepatitis B virus (HBV) screen via HBsAg and Hepatitis C virus HCV: Screen with anti-HCV antibody.

Kairuki Hospital's normal levels are as follows[73,80]; ALT: 0-41 IU/L, AST: 0-41 IU/L, GGT: Male 8.0-38 IU/L; Female 5.0-27 IU/L, Albumin: 3.5–5.3 g/dL, Total Bilirubin: 2.0-21.0 µmol/L, HBsAg Test (Hepatitis B Surface Antigen): Negative and HCV Antibody Test (Hepatitis C Virus Antibody): Negative

3.10 Study variables

3.10.1 Independent variables

I. Socio-demographic characteristics (age, gender, marital status, education level income level, occupation etc.)
II. Alcohol consumption (quantity and frequency, type of alcohol consumed and duration of drinking habits)

3.10.2 Dependent variables

I. Changes in biochemical markers; serum ALT, AST, GGT, albumin, bilirubin levels, HBsAg and HCV Antibody Test.

3.11 Data analysis

The data collected were entered into Microsoft Excel and cleaned for errors before analysis. Data analysis was conducted using the Statistical Package for Social Sciences (SPSS) version 23. Summary statistics (means, medians, and standard deviations) were calculated and presented in tables and figures. Chi-square tests were used to assess the association between independent categorical variables (for example, gender, marital status, education level, income status, occupation, and alcohol consumption pattern) and the dependent categorical variable (Alcoholic liver disease) among alcohol abusers. All categorical data, such as gender, level of education, family history of liver disease, alcohol consumption patterns, and continuous variables that were transformed into categorical variables, were summarized using absolute counts and percentages. Spearman rank correlation was used to assess the correlation between two continuous variables by determining the correlation coefficient (r). Factors were determined by odds ratios, 95% confidence intervals, and a p-value threshold of less than 0.05, which was considered statistically significant.

3.12 Dissemination of Results

The research findings will be disseminated to the KU Library, the Ministry of Health, the Diabetes Society of Tanzania, and the Association of Physicians of Tanzania through research reports, scientific conferences, and publications.

CHAPTER FOUR

4.0 ETHICAL CONSIDERATION

Ethical clearance was obtained from the KU Institutional Research and Ethics Committee, managers of the selected rehabilitation centers approved to conduct the study.

Before data collection, the purpose, objectives, and procedures of the study were clearly explained to all participants. They were informed about the slight pain associated with blood sample withdrawal and assured that the discomfort would subside immediately after the procedure. Psychological discomfort was mitigated by establishing a supportive and confidential environment.

Voluntary informed consent forms were provided for participants to read and sign. For those unable to read or write, the consent forms were read to them, and a thumbprint was accepted as a signature alternative.

To ensure confidentiality, each participant was assigned a serial number linked to their identification, known only to the principal investigator. No personal identifiers were recorded on the data collection forms.

All participant-related information and study data were kept strictly confidential. Hard copies of data collection tools were stored securely in a locked cupboard accessible only to authorized personnel, while electronic records were stored on password-protected computers with access restricted to users possessing valid login credentials.

Participants were informed of their right to withdraw from the study at any time without impacting their access to services in the study setting. The principal investigator was responsible for addressing all questions and concerns raised by participants.

Regular progress reviews were conducted, and health and safety protocols were followed throughout the study. Participants exhibiting abnormal findings suggestive of liver disease were informed, counseled, and referred to the hospital for further specialist management[61].

CHAPTER FIVE

5.0 RESULTS

5.1. Socio-demographic characteristics of the study participants.

A total of 172 participants were enrolled, four were excluded due to hepatitis B or C coinfection, leaving 168 for analysis with a mean age of 37.8 years (±9.97). Regarding the gender of the participants, the majority were male (87.5%), and most resided in various districts of Dar es Salaam, with Kinondoni (27.4%) being the most represented. Most participants were single (60.1%), followed by married (26.2%). In terms of education, 43.5% had secondary education, and 36.3% had university education, while a small proportion had no formal education (3.6%). The largest employment group was self-employed individuals (57.7%), followed by those employed (26.2%) and unemployed (16.1%). The details are shown in table 2.

Table 2: Demographic characteristics of alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168)

Variable	Categories	Frequency	Percent
Residence	Kigamboni	13	7.7
	Kinondoni	46	**27.4**
	Ilala	21	12.5
	Temeke	20	11.9
	Ubungo	21	12.5
	Out of Dar	47	28.0
Gender	Male	147	**87.5**
	Female	21	12.5
Marital status	Single	101	**60.1**
	Married	44	26.2
	Divorced	18	10.7
	Widow	5	3.0
Education	No Education	6	3.6
	Primary	28	16.7
	Secondary	73	**43.5**
	University	61	**36.3**
Employment	Employed	44	26.2
	Self Employed	97	**57.7**
	Unemployed	27	16.1
Age*		**37.8 ± 9.97**	

* Data expressed in mean ± standard deviation

5.2. Clinical characteristics of study participants.

The distribution of clinical characteristics of the participants was assessed, and found that most of the study participants began drinking between the ages of 15–20 (49.4%), with daily drinking reported by 57.1%. Spirits were the most commonly consumed alcohol (62.5%). While only 6% reported having previously been diagnosed with liver disease, symptoms like appetite loss (61.9%), weight loss (57.1%), and fatigue (47%) were frequent, and yellowish skin discoloration was rare (3%). Physical examination signs such as ascites, spider angiomas, and jaundice were absent, while the median liver span was 13cm. Median alcohol use duration was 14 years, with a median of 4 months of sobriety (Table 3).

Table 3. Clinical characteristics of alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168)

Variables	Categories	Frequency	Percent
Drinking start age (year)	<15	19	11.3
	15-20	83	49.4
	21-30	56	33.3
	>30	10	6.0
Alcohol consumption/week	Daily	96	57.1
	4-6 times	28	16.7
	2-3 times	32	19.0
	Less often	12	7.1
Alcohol type	Beer	55	32.7
	Spirit	105	62.5
	Local	8	4.8
Ever diagnosed with liver disease	Present	10	6.0
	Absent	158	94.0

Clinical symptoms			
Fatigue	Present	79	47.0
	Absent	89	53.0
Yellowish skin discoloration	Present	5	3.0
	Absent	163	97.0
Abdominal pain	Present	137	81.5
	Absent	31	18.5
Nausea & vomiting	Present	67	39.9
	Absent	101	60.1
Appetite loss	Present	104	61.9
	Absent	64	38.1
Weight loss	Present	96	57.1
	Absent	72	42.9
Family history of liver disease	Present	13	7.7
	Absent	155	92.3
Physical examination findings			
Liver status	Normal	148	88.1
	Shrunken	1	0.6
	Enlarged	19	**11.3**
URQ tenderness	Present	3	1.8
	Absent	165	98.2
Palmar erythema	Present	4	2.4
	Absent	164	97.6
Paraclinical data			
Alcohol duration (year) *		**14**	9 to 20
Sober duration (month) *		**4**	2 to 7
Liver span (cm) *		**13**	11.3 to 14

*, data expressed in median (interquartile range); URQ, right upper quadrant.

5.3. Prevalence of Alcoholic Hepatitis among alcohol abusers attending rehabilitation centers in Dar es Salaam.

Of 168 participants evaluated for alcoholic liver disease, 11% had alcoholic hepatitis (AH) based on biochemical markers (GGT > 2 × ULN and AST: ALT ratio≥2) (Figure 1).

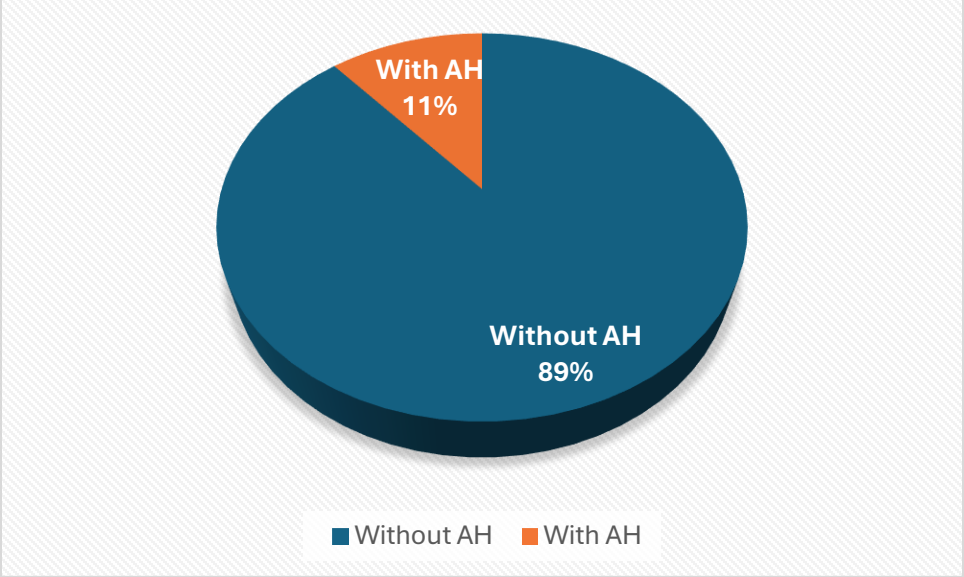


Figure 1: Prevalence of alcoholic hepatitis among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168). Abbreviation: AH, alcoholic hepatitis.

5.4. The distribution of ALT, AST, and GGT levels by age and gender of the study participants.

The study indicated that most youth and adults had normal ALT levels. A chi-square test yielded a value of 3.580 with a p-value of 0.167. Similarly, gender-based analysis shows that most males and females maintain normal ALT levels, and the chi-square test (χ^2 = 0.837, p = 0.36) indicates that the analysis of ALT levels across age and gender revealed no statistically significant associations (P > 0.05) (Table 4).

Table 4: Distribution of ALT across age and gender among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		ALT levels (IU/L)		χ^2	P-value
		Normal	Elevated		
Age groups	Youth	10 (**76.9**)	3 (23.1)	3.580	0.167
	Adults	121 (**78.6**)	33 (21.4)		
	Elderly	0 (0.0)	1 (**100)**		
Gender	Male	113 (**76.9**)	34 (23.1)	0.837	0.36
	Female	18 (**85.7**)	3 (14.3)		

ALT, Alanine Aminotransferase

Regarding the distribution of AST levels across age and gender, most youth and adults had normal AST levels. A chi-square test yielded a value of 3.726 with a p-value of 0.208. Similarly, among genders, both males and females predominantly have normal AST levels, and the chi-square ($\chi^2 = 0.081$, $p = 0.776$) confirms that age and gender were not statistically significantly associated with AST levels (Table 5).

Table 5: Distribution of AST across age and gender among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		AST levels (IU/L)		χ^2	P-value
		Normal	Elevated		
Age groups	Youth	10 (**76.9**)	3 (23.1)	3.726	0.208
	Adults	122 (**79.2**)	32 (20.8)		
	Elderly	0 (0.0)	1 (**100**)		
Gender	Male	116 (**78.9**)	31 (21.1)	0.081	0.776
	Female	16 (**76.2**)	5 (23.8)		

AST, Aspartate Aminotransferase

Contrary to the low prevalence of ALT and AST elevation across age and gender, a moderately higher prevalence of elevated GGT levels was noted among participants. Across participants' age, the youth (46.2%), adults (56.5%), and elderly (100%) had elevated GGT levels. On the other hand, 57.1% of males and 47.6% of females had GGT levels elevated. However, the Chi-square test showed that there were no statistically significant associations between GGT levels with either age group ($\chi^2 = 1.704$, $p = 0.682$) or gender ($\chi^2 = 1.651$, $p = 0.511$) (Table 6).

Table 6: Distribution of GGT across age and gender among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		GGT levels (IU/L)			χ^2	P-value
		Low	Normal	Elevated		
Age groups	Youth	0 (0.0)	7 (53.8)	6 (**46.2**)	1.704	0.682
	Adults	3 (1.9)	64 (41.6)	87 (**56.5**)		
	Elderly	0 (0.0)	0 (0.0)	1 (**100**)		
Gender	Male	2 (1.4)	61 (41.5)	84 (**57.1**)	1.651	0.511
	Female	1 (4.8)	10 (47.6)	10 **(47.6)**		

GGT, Gamma-Glutamyl Transferase

5.5. The distribution of ALT, AST, and GGT levels by employment and monthly income of the study participants.

ALT elevation was highest among employed participants, but employment status showed no statistically significant association ($\chi^2 = 1.531$, $p = 0.465$). By income, elevated ALT was most common in the higher middle-income group (35.7%) and lowest in the highest income group (9.1%), with no significant difference ($\chi^2 = 7.389$, $p = 0.06$). The details are shown in Table 7.

Table 7: Distribution of ALT across employment and monthly income among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		ALT levels (IU/L)		χ^2	P-value
		Normal	Elevated		
Employment	Employed	32 (**72.7**)	12 (**27.3**)		
	Self Employed	76 (**78.4**)	21 (**21.6**)	1.531	0.465
	Unemployed	23 (**85.2**)	4 (**14.8**)		
Monthly income (Tsh)	Less than 270,000	21 (77.8)	6 (22.2)		
	270,000-519,000	63 (81.8)	14 (18.2)		
	520,000-1,000,000	27 (64.3)	15 (**35.7**)	7.389	0.06
	Above 1,000,000	20 (90.9)	2 (**9.1**)		

ALT, Alanine Aminotransferase

The distribution of AST levels across employment status showed no statistically significant differences (P = 0.345), with slightly higher elevations among employed (25.0%) and self-employed (22.7%) participants compared to the unemployed (11.1%). There is a statistically significant association between monthly income and elevated AST levels (p=0.011), where participants earning above 520,000 Tsh. exhibited notably higher proportions of elevated AST. The details are shown in Table 8.

Table 8: Distribution of AST across employment and monthly income among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		AST levels (IU/L)		χ^2	P-value
		Normal	Elevated		
Employment	Employed	33 (75.0)	11 (**25.0**)	2.131	0.345
	Self Employed	75 (77.3)	22 (**22.7**)		
	Unemployed	24 (88.9)	3 (**11.1)**		
Monthly income (Tsh)	Less than 270,000	25 (92.6)	2 (7.4)	11.216*	**0.011**
	270,000-519,000	65 (84.4)	12 (15.6)		
	520,000-1,000,000	27 (64.3)	15 **(35.7)**		
	above 1,000,000	15 (68.2)	7 **(31.8)**		

AST, Aspartate Aminotransferase

GGT elevation was common across all employment and income levels, but neither employment status ($\chi^2 = 6.001$, $p = 0.199$) nor income ($\chi^2 = 5.129$, $p = 0.527$) showed statistically significant associations (Table 9).

Table 9: Distribution of GGT across employment and monthly income among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Variable		GGT levels (IU/L)			X^2	P-value
		Low	Normal	Elevated		
Employment	Employed	1 (2.3)	12 (27.3)	31 (**70.5**)	6.001	0.199
	Self Employed	2 (2.1)	46 (47.4)	49 (50.5)		
	Unemployed	0 (0.0)	13 (48.1)	14 (51.9)		
Monthly income (Tsh)	Less than 270,000	0 (0.0)	15 (55.6)	12 (44.4)		
	270,000-519,000	1 (1.3)	29 (37.7)	47 (**61.0)**	5.129	0.527
	520,000-1,000,000	1 (2.4)	16 (38.1)	25 (59.5)		
	Above 1,000,000	1 (4.5)	11 (50.0)	10 (45.5)		

GGT, Gamma-Glutamyl Transferase

5.6. Correlation of ALT, AST, and GGT levels with duration of alcohol consumption

Figure 2 illustrates the relationship between the duration of alcohol consumption (years) and serum ALT levels (IU/L)**.** Upon analysis there was a negative correlation between the duration of alcohol consumption and ALT levels (r = –0.041, P = 0.599); however, the association was not statistically significant.

There was a positive correlation between the duration of alcohol consumption and AST levels (r=0.132, p=0.087), however, the association was not statistically significant as illustrated in Figure 3.

Also, there was a negative correlation between the duration of alcohol consumption and GGT levels (r = -0.050) (p = 0.520), however, the association is not statistically significant. The detail is shown in Figure 4

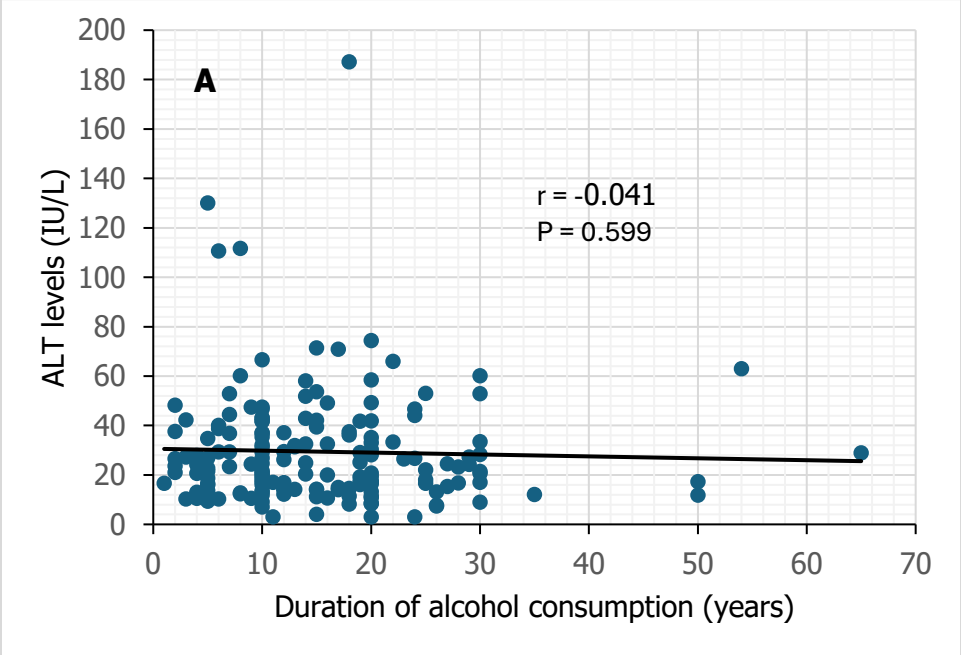


Figure 2. Correlation of ALT (A), levels with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168). Abbreviations: ALT, alanine aminotransferase

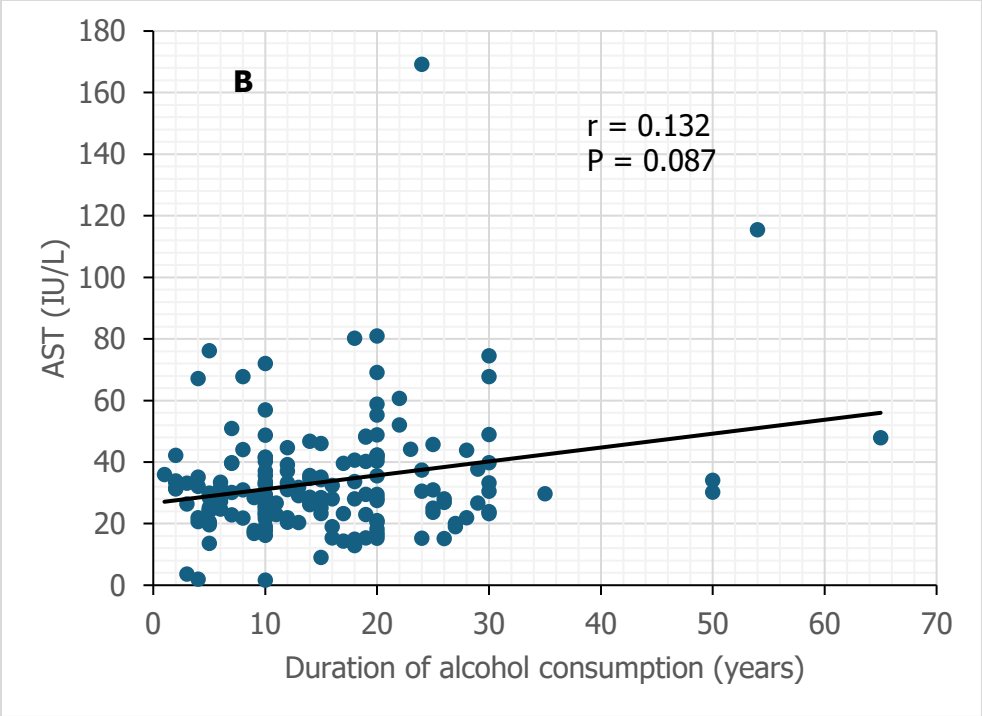


Figure 3. Correlation of AST (B) levels with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168). Abbreviations: AST, Aspartate aminotransferase

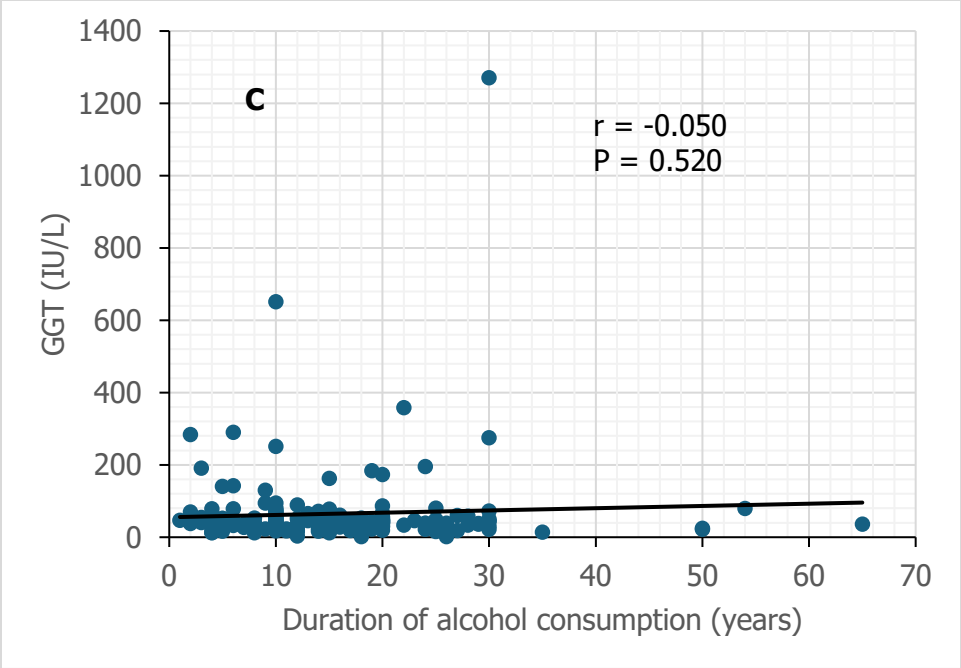


Figure 4. Correlation of GGT(C) levels with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168). Abbreviations: GGT, gamma-glutamyl transferase.

5.7. Correlation of serum Albumin and Total bilirubin with duration of alcohol consumption.

Figure 5 illustrates a revealed a weak, negative correlation between duration of alcohol consumption and serum albumin levels (r = -0.146, p = 0.059), a positive correlation was also observed between the duration of alcohol consumption and serum bilirubin level (r = 0.120, p = 0.122) (Figure 6), However, the correlations were not statistically significance.

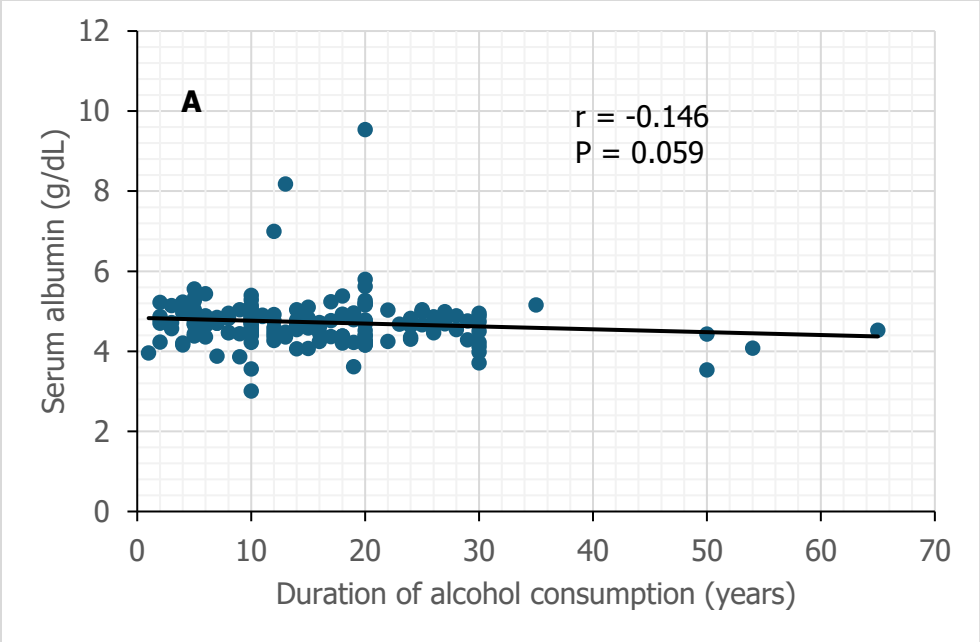


Figure 5. Correlation of serum Albumin (A) with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

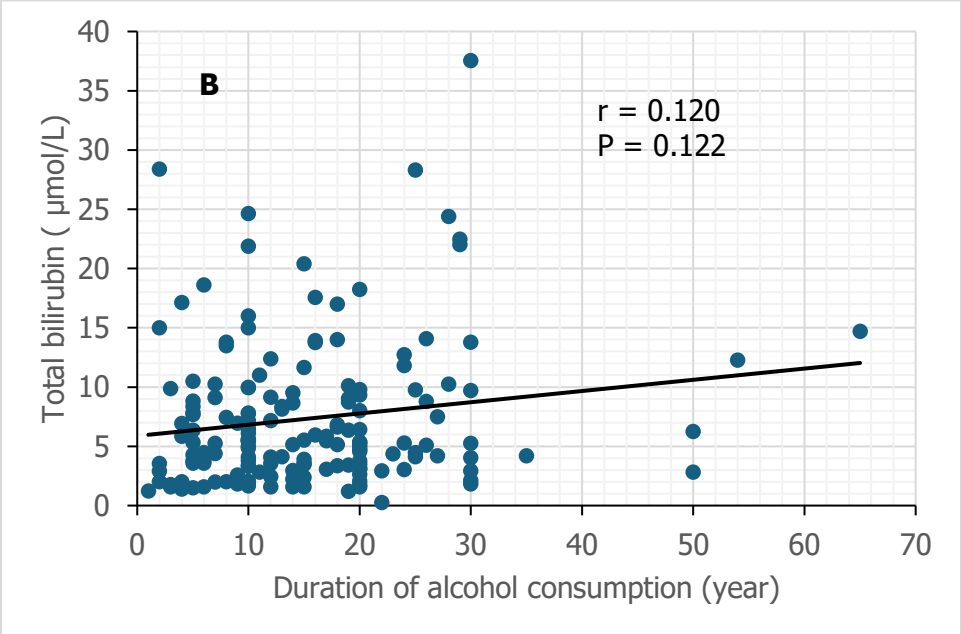


Figure 6. Correlation of Total bilirubin (B) with duration of alcohol consumption among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

5.8. The association between Alcoholic hepatitis and liver biochemical markers

The association between alcoholic hepatitis (AH) and liver biochemical markers was described in Table 10. Serum ALT levels were found to be elevated in participants without AH compared to those with AH, $\chi^2 = 3.5$, $p = 0.047$ was statistically significant. While GGT (47.4%) and AST (31.6%) were the most commonly elevated markers in those with AH, when comparing elevated AST, GGT, Bilirubin, and albumin levels in participants with AH and without AH, the differences were not statistically significant.

Table 3. Association between Alcoholic hepatitis with liver biochemical markers among alcohol abusers attending rehabilitation centers in Dar es Salaam (N =168).

Biochemical markers		Alcoholic hepatitis (AH)		χ^2	P-value
		With AH	Without AH		
ALT	Normal	18 (94.7)	113 (75.8)	3.5*	**0.047**
	Elevated	1 **(5.3)**	36 **(24.2)**		
AST	Normal	13 (68.4)	119 (79.9)	1.31	0.247
	Elevated	6 **(31.6)**	30 (20.1)		
GGT	Normal	10 (52.6)	64 (43.0)	0.64	0.468
	Elevated	9 **(47.4)**	85 (57.0)		
Total Bilirubin	Normal	17 (89.5)	143 (96.0)	1.57	0.225
	Elevated	2 (10.5)	6 (4.0)		
Albumin	Low	0 (0.0)	1 (0.7)	0.13	0.887
	Normal	19 (100.0)	148 (99.3)		

Abbreviations: ALT, alanine aminotransferase; AST, Aspartate aminotransferase; GGT, gamma-glutamyl transferase.

CHAPTER SIX

6.0 DISCUSSION

This study examined the biochemical patterns of Alcoholic Liver Disease (ALD) among alcohol abusers attending rehabilitation centers in Dar es Salaam, revealing a significant male predominance (87.5%). This aligns with broader African and global data indicating higher rates of alcohol consumption and ALD risk among men [44,60,81,82].

The average participant age was 37.8 ± 9.97 years, consistent with findings by Kabwama et al., who identified the 30–50-year age group as most affected in Uganda. This middle-aged demographic represents a critical target for alcohol-related public health interventions[82].

Contrary to earlier reports linking ALD primarily to lower education levels[84], this study found that 43.5% of participants had secondary education and 36.3% held university degrees. This trend mirrors recent evidence showing increased hazardous drinking among educated and higher-income populations, consistent with Manthey et al.'s findings[85]. Such findings suggest that excessive alcohol use is no longer confined to low socioeconomic groups but is rising among urban, educated individuals—likely influenced by lifestyle and cultural shifts. Future prevention strategies should therefore include these evolving at-risk groups.

Early initiation of alcohol consumption was also notable, with 48.7% of respondents beginning between ages 15–20 and 11.3% before 15. These findings support the neurobehavioral vulnerability model of adolescence[86], and highlight the urgent need for preventive measures such as stricter enforcement of underage drinking laws, school-based education, and parental awareness programs.

Spirits were the most commonly consumed beverages (62.5%), and daily drinking was reported by 57.1% of participants. These patterns are consistent with Seitz et al.[87] and Jayathilaka et al[88], who linked high-ethanol beverages to oxidative liver injury and increased ALD prevalence in low- and middle-income settings. Regulatory interventions such as taxation on spirits, advertising restrictions, and limiting access to high-potency beverages are therefore warranted.

Employed participants exhibited the highest prevalence of elevated liver enzymes (ALT 27.3%, AST 25.0%, GGT 70.5%), though these associations were not statistically significant. This finding aligns with Finnish population-based data showing that occupational status has less influence on liver injury than drinking patterns and lifestyle[89]. Conversely, the high rate of self-employment (57.7%) supports Lee and Merali's finding that financial instability may drive excessive alcohol use[90], underscoring the need for interventions that integrate economic and mental health support.

Unlike global patterns linking ALD mainly to poverty[84], a statistically significant association emerged between elevated AST levels and higher-income earners ($p = 0.011$), consistent with findings by White et al[85]. While this suggests shifting disease patterns toward more affluent groups, it also raises concerns about underdiagnosis among lower-income populations with limited healthcare access, as reported by Mhando et al[91]. Given AST's limited liver specificity, this result should be interpreted with caution[89].

Overall, the study confirms the limited standalone diagnostic value of liver biomarkers (ALT, AST, GGT, bilirubin, albumin). Elevated GGT across all groups reaffirms its sensitivity as an alcohol exposure marker, though it lacks specificity[67]. A notable finding was the statistically significant association between normal ALT levels and Alcoholic Hepatitis (AH), consistent

with the classical pattern of alcohol-induced hepatocellular injury, where pyridoxine deficiency and mitochondrial dysfunction suppress ALT release, resulting in a high AST: ALT ratio, a hallmark of AH[27].

Although correlations between biochemical markers and duration of alcohol use were not statistically significant, the observed patterns (decreasing ALT, GGT, and albumin with rising AST and bilirubin), suggest progressive hepatic dysfunction[24,97]. This contrasts with Walter et al[98], who reported cumulative increases in all liver enzymes with longer drinking duration. The present findings emphasize hypoalbuminemia and hyperbilirubinemia as late-stage indicators[65], of hepatic decompensation[27,33], reinforcing previous observations by Narro et al[72]. They also support Malakouti et al.'s recommendation for broader screening frameworks that incorporate drinking patterns, genetic susceptibility, and nutritional status[63].

The prevalence of Alcoholic Hepatitis (AH) in this study (11%) falls within the 10–35% range reported in high-income countries and slightly below the 20% observed in U.S. treatment centers[65]. This variation likely reflects demographic and clinical differences, including a younger population with shorter drinking histories and earlier disease stages[96]. Similar to global data from Niu et al.[55], these results underscore the growing burden of AH among Tanzanian alcohol abusers.

Elevated GGT (47.4%) and AST (31.6%) among AH participants reflect well-established biochemical patterns of alcohol-induced hepatocellular injury[61,80].When considered alongside other liver biochemical markers, these results reinforce the value of comprehensive diagnostic approaches that combine clinical assessment with biomarker panels, particularly the AST: ALT ratio and GGT, for improved diagnostic accuracy and disease staging staging[63,81,85].

Collectively, these findings highlight the clinical necessity of integrating biochemical monitoring into alcohol rehabilitation and primary care settings. Routine liver function testing could enhance early detection, disease staging, and timely therapeutic intervention.

From a prevention perspective, it is crucial to incorporate systematic liver health screening into alcohol rehabilitation and treatment programs for early detection and appropriate management. The likelihood of AH underdiagnosis due to limited laboratory capacity further underscores the urgent need to strengthen diagnostic infrastructure and train healthcare providers in recognizing early biochemical signs of alcohol-related liver dysfunction.

CHAPTER SEVEN

7.0 STUDY STRENGTHS, LIMITATIONS, AND MITIGATIONS

This study, conducted among alcohol abusers attending rehabilitation centers in Dar es Salaam, has several notable strengths. It targets a clearly defined, clinically relevant population selected through rigorous sampling methods, which enhances the validity and relevance of the findings. Additionally, trained personnel administered the validated CAGE questionnaire to ensure dependable assessment of alcohol abusers. A key methodological strength is the inclusion of multiple biochemical markers, including albumin, bilirubin, ALT, AST, and GGT, to improve ALD detection, providing a more comprehensive approach than relying on a single marker. Furthermore, the study addresses an important regional research gap by providing localized data from Kigamboni District, an area with high-capacity rehabilitation centers serving alcohol-dependent individuals across Tanzania. The findings offer crucial evidence to inform liver disease screening and management policies in Tanzania, emphasizing the importance of multi-marker strategies for high-risk populations.

While the study provides valuable insights, it is not without its limitations. The cross-sectional design inhibits the ability to assess the progression of Alcohol-Related Liver Disease (ALD) over time. Furthermore, the small convenience sample drawn from rehabilitation centers may introduce selection bias and limit the statistical robustness of the findings. The reliance on self-reported alcohol consumption data carries inherent risks of underreporting, influenced by recall bias, social stigma, and the variability associated with traditional home-brewed alcoholic beverages.

The diagnostic capabilities of the study were also constrained by the absence of a control group and the lack of advanced diagnostic tools, such as Fibro Scan or other imaging

methods, which hampers both the accuracy of staging and the generalizability of the results. Additionally, potential confounding factors, including nutritional status, comorbid conditions, and genetic influences, were not evaluated, which may have affected the observed outcomes.

To address underreporting in self-reported alcohol intake, efforts were made to include concise questions with short recall periods in a privacy-focused and comfortable setting; however, discrepancies in measurements may still occur.

CHAPTER EIGHT

8.0 CONCLUSION AND RECOMMENDATION

8.1 CONCLUSION

This study highlights the changing epidemiological and biochemical landscape of Alcoholic Liver Disease (ALD) among alcohol abusers in Tanzania. The findings show that excessive alcohol consumption and its liver-related consequences are no longer limited to traditionally vulnerable groups but are increasingly common among educated, economically active adults and higher-income populations, with a persistent male predominance. This signals an epidemiological shift that requires a reorientation of public health messaging and intervention strategies. The study also emphasizes that harmful drinking behaviors, such as early initiation of alcohol use and a preference for high-potency spirits, continue to be significant contributors to liver injury.

The study also highlights the limitations of relying solely on liver biomarkers for diagnosing and staging ALD, a concern underscored by the frequent occurrence of Alcoholic Hepatitis with normal ALT levels. Although biochemical indicators like elevated AST and GGT or a high AST: ALT ratio are still clinically relevant, their diagnostic value is best when combined with clinical evaluation and contextual factors. The observed biochemical patterns indicate progressive liver dysfunction with prolonged alcohol consumption, emphasizing the need for ongoing monitoring and comprehensive screening.

Collectively, these study findings reveal crucial insights with direct implications for public health strategies and clinical management. They call for a shift in how we approach ALD, requiring a multifaceted strategy that considers changing risk profiles, closes diagnostic

gaps, addresses social determinants of health, and incorporates comprehensive liver health monitoring into preventive and rehabilitative care systems.

8.2 RECOMMENDATIONS

Based on these findings, it is recommended that routine liver function testing and comprehensive biomarker assessment be integrated into rehabilitation and primary healthcare services to facilitate early detection and proper staging of Alcoholic Liver Disease. Strengthening diagnostic capacity through improved laboratory infrastructure and clinician training is essential to address underdiagnosis and enhance case management. Public health policies should enforce stricter control over the sale and marketing of high-potency alcoholic beverages, including taxation on spirits and the reinforcement of age restrictions to curb early alcohol initiation.

Furthermore, community- and school-based education programs should be expanded to raise awareness of the health risks associated with heavy and early alcohol consumption, particularly among adolescents and urban populations. Addressing the socioeconomic and psychosocial factors that drive harmful drinking, such as unemployment, financial instability, and stress, through integrated support and empowerment initiatives is crucial for sustainable prevention.

Finally, further large, longitudinal, multi-center cohort studies are needed to better characterize ALD progression and determinants of Alcoholic Liver Disease across Tanzanian and similar low-resource contexts, thereby informing evidence-based interventions and policies.

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APPENDICES

APPENDIX I: QUESTIONNAIRE (ENGLISH VERSION)

TITLE: BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMONG ALCOHOL ABUSERS ATTENDING REHABILITATION CENTRES IN DAR ES SALAAM.

1. Date of data collection _____

2. Study site. _____

3. Residence area. _____

SECTION A: SOCIO-DEMOGRAPHIC DATA

1.0 Age ☐

2.0 Gender ☐ Male ☐ Female

3.0 Marital Status ☐ Single ☐ Married ☐ Divorced ☐ Widowed

4.0 Education Level

☐ No formal education ☐ Primary school ☐ Secondary school ☐ Higher education

5.0 Employment Status ☐ Employed ☐ Self-employed ☐ Unemployed

6.0 Monthly Income (in TZS):

☐ Low-income: <270,000 ☐ Lower-middle-income: 270,000- 519,000

☐ Upper-middle-income: 520,000-760,000 ☐ High-income: 761,000-1,000,000

☐ >1,000,000

SECTION B: CAGE TOOL

7.0 Have you ever felt you should Cut down on your drinking? ☐ Yes ☐ No

8.0 Have people Annoyed you by criticizing your drinking? ☐ Yes ☐ No

9.0 Have you ever felt Guilty about drinking? ☐ Yes ☐ No

10.0 Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (Eye-opener)? ☐ Yes ☐ No

SECTION C: ALCOHOL CONSUMPTION PATTERNS

11.0 At what age(years) did you start drinking alcohol?

☐ Below 15 ☐ 15–20 ☐ 21–30 ☐ Above 30

12.0 How often do you consume alcohol?

☐ Daily ☐ 4–6 times per week ☐ 2–3 times per week ☐ Once a week ☐ Less often

13.0 What type of alcohol do you primarily consume?

☐ Beer ☐ Spirits (e.g., whiskey, vodka) ☐ Wine ☐ Traditional brews/local/others

14.0 On average, how many drinks do you consume per session?

☐ 1–2 ☐ 3–5 ☐ 6–8 ☐ More than 8

SECTION D: CLINICAL HISTORY

15.0 Have you been diagnosed with any liver-related condition before?

☐ Yes (specify: _____) ☐ No

16.0 Do you experience any of the following symptoms? ☐ Fatigue

☐ Jaundice (yellowing of eyes/skin) ☐ Abdominal pain or swelling

☐ Nausea or vomiting ☐ Loss of appetite ☐ Weight loss

17.0 Do you have a family history of liver disease?

☐ Yes (specify: _____) No

SECTION E: PHYSICAL EXAMINATION FINDINGS

18.0 Abdominal Findings.

☐ Liver Status; Enlarged/ Shrunken/Normal ☐ Liver span(cm) _____

☐ Ascites (fluid accumulation) ☐ Tenderness in the upper right quadrant ☐ None

19.0 Skin Findings ☐ Jaundice ☐ Spider angiomas ☐ Palmar erythema ☐ None

Section F: PARACLINICAL DATA

20.0 Biochemical tests.

a. ALT (Alanine Aminotransferase): _____ IU/L

b. AST (Aspartate Aminotransferase): _____ IU/L

c. Albumin: _____ g/dl

d. GGT (Gamma-Glutamyl Transferase): _____ IU/L

e. Total Bilirubin: _____ mg/dl

f. HBsAg Test _____

g. HCV Antibody Test _____

APPENDIX II: DODOSO (KISWAHILI)

MFUMO WA KIBIOKEMIKALI WA UGONJWA WA INI UNAOHUSIANA NA MATUMIZI YA POMBE KWA WATUMIAJI WA POMBE WANAOHUDHURIA KITUO CHA REHABU DAR ES SALAAM.

1. Tarehe ya ukusanyaji wa data _____
2. Jina la kituo cha urejeshaji (rehabu). _____
3. Utambulisho. _____

SEHEMU A: TAARIFA ZA KIJAMII

1.0 Umri ☐

2.0 Jinsia ☐ Mwanaume ☐ Mwanamke

3.0 Hali ya Ndoa ☐ Binafsi ☐ Ndoa ☐ Talaka ☐ Mfiwa

4.0 Kiwango cha Elimu

☐ Hakuna elimu rasmi ☐ Shule ya msingi ☐ Shule ya sekondari ☐ Elimu ya juu

5.0 Hali ya Ajira ☐ Aliyeajiriwa ☐ Kujiajiri ☐ Hana ajira

6.0 Kipato cha Kila Mwezi (kwa TZS): ☐ Kipato cha chini: chini ya 270,000

☐ Kipato cha kati cha chini: 270,000–519,000 ☐ Kipato cha kati cha juu: 520,000–760,000

☐ Kipato cha juu: 761,000–1,000,000 ☐ Zaidi ya 1,000,000

SEHEMU B: ZANA YA CAGE

7.0 Ulishawahi kuhisi kwamba unapaswa kupunguza unywaji wa pombe?

☐ Ndio ☐ Hapana

8.0 Watu wamekuwa wakikukasirisha kwa kukosoa unywaji wako?

☐ Ndio ☐ Hapana

9.0 Ulishawahi kujihisi na hatia kutokana na kunywa pombe? ☐ Ndio ☐ Hapana

10.0 Ulishawahi kunywa asubuhi ili kutuliza wasiwasi au kuondoa hangover (kama tiba ya haraka)? ☐ Ndio ☐ Hapana

SEHEMU C: MWELEKEO WA MATUMIZI YA POMBE

11.0 Ulianza kunywa pombe ukiwa na umri gani?

☐ Chini ya miaka 15 ☐ 15–20 ☐ 21–30 ☐ Zaidi ya miaka 30

12.0 Unakunywa pombe mara ngapi? ☐ Kila siku ☐ Mara 4–6 kwa wiki

☐ Mara 2–3 kwa wiki ☐ Mara moja kwa wiki ☐ Mara chache

13.0 Aina gani ya pombe unakunywa zaidi?

☐ Bia ☐ Vileo (mfano: whisky, vodka) ☐ Divai ☐ Pombe za kienyeji

14.0 Kwa wastani, unakunywa vinywaji vingapi kwa kikao?

☐ 1–2 ☐ 3–5 ☐ 6–8 ☐ Zaidi ya 8

SEHEMU D: HISTORIA YA KLINIKI NA DALILI

15.0 Je, umewahi kugunduliwa kuwa na tatizo lolote la ini?

☐ Ndio (eleza: _____) ☐ Hapana

16.0 Je, unakumbana na dalili zozote kati ya zifuatazo?

☐ Uchovu ☐ Njano (manjano ya macho/ngozi) ☐ Maumivu ya tumbo au uvimbe

☐ Kichefuchefu au kutapika ☐ Kukosa hamu ya kula ☐ Kupungua uzito

17.0 Je, kuna historia ya ugonjwa wa ini kwenye familia yako?

☐ Ndio (eleza: _____) ☐ Hapana

SEHEMU E: MATOKEO YA UCHUNGUZI WA KIMWILI

18.0 Matokeo ya Uchunguzi wa Tumbo ☐ Hali ya ini ; Kubwa/ Dogo/ Kawaida

☐ Ukubwa wake (cm)_____ ☐ Mkusanyiko wa maji tumboni (ascites)

☐ Maumivu upande wa juu kulia wa tumbo ☐ Hakuna

19.0 Matokeo ya Ngozi ☐ Njano ☐ Vidonda vya mishipa midogo ya damu (spider angiomas) ☐ Mwekundu wa viganja vya mikono (palmar erythema) ☐ Hakuna

SEHEMU F: VIPIMO VYA MAABARA

20.0 Uchunguzi wa kibiokemia

a. ALT (Alanine Aminotransferase): _____ IU/L
b. AST (Aspartate Aminotransferase): _____ IU/L
c. Albumini: _____ g/dl

d. GGT (Gamma-Glutamyl Transferase): _____ IU/L

e. Jumla ya Bilirubini: _____ mg/dl

f. HBsAg Test _____

g. HCV Antibody Test _____

APPENDIX III: CONSENT FORM (ENGLISH VERSION)

TITLE: BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMONG ALCOHOL ABUSERS ATTENDING REHABILITATION CENTRES IN DAR ES SALAAM.

I **Dr. Joyce Elias Sabuka**, a resident in the Department of Internal Medicine, would like to conduct the named study above as a necessary requirement for fulfillment of my post-graduate studies.

Your participation is required to acquire necessary information regarding your health to be used as data in this study.

The study aims to determine the biochemical pattern of alcoholic liver disease among alcohol abusers attending rehabilitation centers in Dar es Salaam. Findings from this study shall be helpful in the recommendation of early screening of liver disease among alcohol abusers thus early intervention before the development of further complications.

Those adults with alcohol abuse, who will meet the inclusion criteria, will be recruited into the study and will be interviewed using a questionnaire, which will include their social demographic characteristics and physical examination.

Blood tests for liver function tests will be taken. Blood will be tested for liver enzymes. There will be slight pain in the venipuncture for blood sample collection.

Study findings will not be released to any unauthorized person.

The participant will not be asked for any fee/money and will be free to withdraw at any time during the study.

People to contact in case of questions or problems.

Prof Y. Mgonda, chairperson of the Department of Internal Medicine, Phone no:0754277554

Dr. Joyce Sabuka, Postgraduate student at Kairuki University, Phone no: 0788939337

I……..have read/been told of the contents of this form and understood its meaning. Hence, I agree to participate in this study.

Signature …….. (Participant),

Signature……..

Date……..

APPENDIX IV: FOMU YA IDHINI (SWAHILI VERSION)

KICHWA CHA UTAFITI: MFUMO WA KIBIOKEMIKALI WA UGONJWA WA INI UNAOHUSIANA NA MATUMIZI YA POMBE KWA WATUMIAJI WA POMBE WANAOHUDHURIA KITUO CHA REHABU DAR ES SALAAM.

Mimi, **Dkt. Joyce Elias Sabuka**, Mwanafunzi wa shahada ya uzamili ya Magonjwa ya ndani katika Chuo kikuu cha Kairuki. Ninafanya utafiti ulioainishwa hapo juu kama sharti la lazima la kutimiza masomo yangu ya uzamili.

Ushiriki wako unahitajika ili kupata taarifa muhimu zinazohusiana na afya yako zitakazotumika kama data katika utafiti huu.

Lengo la utafiti ni kubaini mfumo wa kibaokemikali wa ugonjwa wa ini unaohusiana na matumizi ya pombe kwa watumiaji wa pombe wanaohudhuria vituo vya kubadilisha tabia jijini Dar es Salaam. Matokeo ya utafiti huu yatakuwa na manufaa katika kutoa mapendekezo ya uchunguzi wa mapema wa ugonjwa wa ini miongoni mwa watumiaji wa pombe ili kuwezesha hatua za mapema kabla ya matatizo makubwa kuibuka.

Watu wazima wenye matumizi mabaya ya pombe, watakaokidhi vigezo vya kuingizwa kwenye utafiti, watajumuishwa katika utafiti huu na watahojiwa kwa kutumia dodoso ambalo litajumuisha taarifa zao za kijamii na kidemografia pamoja na uchunguzi wa kimwili.

Vipimo vya damu kwa ajili ya kuchunguza vimeng'enya vya ini vitachukuliwa. Kutakuwa na maumivu kidogo wakati wa kuchomwa sindano ya kuchukua sampuli ya damu.

Matokeo ya utafiti hayatakabidhiwa kwa mtu yeyote asiyeidhinishwa.

Mshiriki hatatakiwa kutoa ada au pesa yoyote na atakuwa huru kujiondoa wakati wowote wa utafiti.

Watu wa kuwasiliana nao endapo kuna maswali au matatizo:

Prof. Y. Mgonda, Mwenyekiti wa Idara ya Tiba na magonjwa ya ndani,

Mawasiliano :0754277554

Dr Joyce Sabuka, Mwanafunzi wa shahada ya uzamili ya Magonjwa ya ndani ,

Chuo kuku cha Kairuki, Mawasiliano: 0788939337

Mimi, ……, nimesoma/nimeelezwa kuhusu maudhui ya fomu hii na kuelewa maana yake. Hivyo, nakubali kushiriki katika utafiti huu.

Sahihi: ……(Mshiriki)

Sahihi: ……

Tarehe: …….

APPENDIX V: KAIRUKI UNIVERSITY INSTITUTION REVIEW ETHICAL COMMITTEE REPORT.

KAIRUKI UNIVERSITY (KU)

70 Chwaku Street,
Mikocheni,
P.O BOX 65300,
Dar es Salaam,
Tanzania.

Tel: +255-22-2700021/4
Fax: +255-22-2775591
Email: irec@ku.ac.tz
Website: www.ku.ac.tz

Ref. No. KU/IREC/27.10/573 **12 June, 2025**

Dr. Joyce Elias Sabuka,
Kairuki University,
70 Chwaku Street,
Mikocheni,
P. O. Box 65300.
Dar es Salaam, Tanzania.

RE: ETHICAL CLEARANCE CERTIFICATE FOR CONDUCTING HEALTH RESEARCH

I am pleased to inform you that the research titled: **Biochemical Pattern of Alcoholic Liver Disease among Alcohol Abusers Attending Rehabilitation Centres in Dar es Salaam (Sabuka, J. E., 2025)** has been granted ethical approval.

This approval is in effect for one year from the above date.

- Any changes in the procedures should be reported to the Institutional Research Ethics Committee.
- Significant changes will require the submission of a revised request for ethical approval.
- You will be required to submit **a study progress report** every six months.

Permission to publish your findings should be sought from the National Institute for Medical Research (NIMR) before submission to a publisher and not concurrently.

CHAIR PERSON

Name: Prof. Frederick Kaijage

Signature: _____

SECRETARY

Name: Prof. Columba Mbekenga

Signature: _____

DIRECTOR POSTGRADUATE STUDIES AND RESEARCH INSTITUTE
KAIRUKI UNIVERSITY
P. O. Box 65300, DSM

APPENDIX VI: PERMISSION LETTERS FOR DATA COLLECTION

KAIRUKI UNIVERSITY (KU)

70 Chwaku Street
Regent Estate – Mikocheni
P.O. Box 65300,
Dar es Salaam
Tanzania

Tel: +255-22-2700021/4
Email: secvc@ku.ac.tz
Website: www.ku.ac.tz

Ref. No. KU/PT/30.5/595

12th June 2025

Managing Director,
DAPO Recovery House,
Kigamboni, Dar es Salaam.

Re: LETTER OF INTRODUCTION FOR DR. JOYCE ELIAS SABUKA (MMed Part II – INTERNAL MEDICINE).

The above named is a MMed postgraduate student specialising in Internal Medicine.
As part of fulfilling her MMed programme, she plans to undertake a study titled, **"Biochemical Pattern of Alcoholic Liver Disease among Alcohol Abusers Attending Rehabilitation Centres in Dar es Salaam".** This study was reviewed and has been granted with an ethics approval No. **KU/IREC/27.10/573** by the KU Institutional Research Ethics Committee that will be valid for one year with effect from 11th June 2025.

This letter serves to introduce **Dr. Joyce Elias Sabuka** who will be conducting her study at your rehabilitation centres, please accord her with the needed support.
Thank you for your support and cooperation in developing human resources for health in our country.

Regards,

DIRECTOR POSTGRADUATE STUDIES
KAIRUKI UNIVERSITY
P. O. Box 65300, DSM
AND RESEARCH INSTITUTE

Professor Naboth Mbemba
Ag. Director Postgraduate Studies & Research Institute

c. c. Prof. Yassin Mgonda, Head, Department of Internal Medicine, KU

General Contact:
Email: info@ku.ac.tz
Contact: +255 659 371 234

Academic:
Email: dvc-academics@ku.ac.tz
Contact: +255 716 999 151

Admissions:
Email: admissions@ku.ac.tz
Contact: +255 769 724 636
+255 659 371 234

PILLIMISSANAH FOUNDATION SOBER HOUSE

P.O BOX 36259, KIGAMBONI-DAR ES SALAAM
Email: pillimythamaleek@gmail.com
Mob: +255 715 744464, +255757 858563

18/06/2025.

Professor Naboth M

Ag. Director, Postgraduate Studies

Kairuki University

P.O. Box 65300

Dar es Salaam, Tanzania

Dear Professor Naboth,

RE: ACCEPTANCE OF DR. JOYCE ELIAS SABUKA'S RESEARCH STUDY

I hope this letter finds you in good health and high spirits.

On behalf of Pilimisanah Foundation Sober House, I wish to acknowledge with gratitude the receipt of your letter introducing Dr. Joyce Elias Sabuka, a postgraduate student in Internal Medicine at your esteemed institution.

We are pleased to accept Dr. Sabuka's proposed research titled "Biochemical Pattern of Alcoholic Liver Disease among Alcohol Abusers Attending Rehabilitation Centres in Dar es Salaam." We recognize the importance of this study and its potential to contribute meaningfully to the understanding and management of substance use-related health conditions.

Our team is committed to supporting Dr. Sabuka throughout the course of her research. She will be granted the necessary access and cooperation to facilitate the smooth execution of her study within our centers.

We appreciate the role that Kairuki University continues to play in advancing health research and human resource development in Tanzania. We look forward to a fruitful collaboration.

Should you require any further information or clarification, please do not hesitate to contact us.

Yours sincerely,

Kassim Mabula Dede

Assistant Managing Director

Pilimisanah Foundation Sober House

Phone: 0715 744464

Email: pillimythamaleek@gmail.com

MUUNGANO RECOVERY COMMUNITY
Dir: +255683620421
Tel: +255755088941
Email: mrcsober@gmail.com
Address: P.O.Box 79149,Dar es salaam.

Ref No: MRCTZ/FIELD/2025/12

Date: 14th june, 2025

KAIRUKI UNIVERSITY (KU)
REGENT ESTATE
P.O.Box 65300,
DAR ES SALAAM.

REF: STUDY APPROVAL

Kindly adhere to the subject above and reference letter No.KU/PT/30.5/597 dated 12th june2025.

With this letter, The organization has granted **Dr. Joyce Elias Sabuka** the opportunity undertake your study titled Biochemical pattern of alcohol liver diseases among alcohol abusers attending rehabilitation programs in our centers that contributes to partial fulfillment her MMed in internal medicine.

Regards,
Hamis Ajib
MANAGER

Dapo Recovery House
Telephone Number: +255 673 204 332
Dar Es Salaam – Tannzania.

Director of Post Graduate
Prof. Nabort Mbembati
Prof. Yassin Mgonda
S.L.P 65300
Dar Es Salaam -Tanzania

YAH: KUKUBALI DR. JOYCE ELIAS SABUKA KUJA KUFANYA RESEARCH YA INI KWA WATAWALIWA POMBE.

Husika na kichwa cha habari hapo juu.

Sisi Dapo Recovery House asasi inayohusika na huduma za utengemao kwa waraibu wa dawa za kulevya pamoja na pombe, tumeridhia Dr Joyce Sabuka kuja kwenye kituo chetu kufanya research kwa watawaliwa wa pombe maana inaonyesha asilimia kubwa ya watawaliwa wa pombe hupata ugomjwa wa ini na figo.

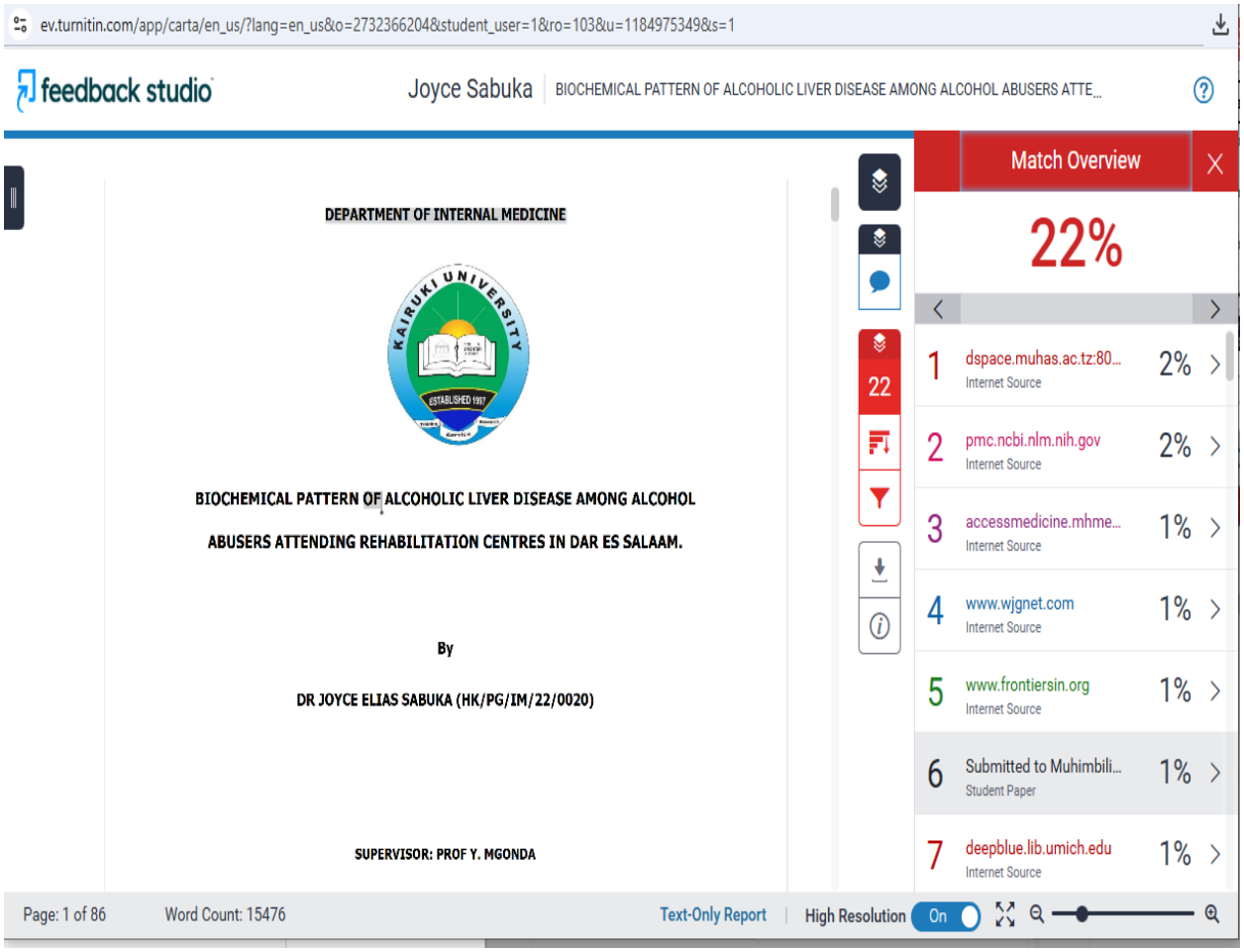
Hivyo basi tumekubali na huduma hii ya Dr. Joyce Sabuka kuja kituoni kwetu kuanzia tarehe 13 June 2025.

Ahsante sana.

Wako katika huduma

Surah Nyembo
Managing Director
Dapo Recovery House

APPENDIX VII: TURNITIN PLAGIARISM REPORT



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Assignment title:	Research Proposal
Submission title:	BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMON...
File name:	DESSERTATION_FINAL_REPORT_FOR_DR._JOYCE_E._SABUKA-dr...
File size:	5.49M
Page count:	86
Word count:	15,476
Character count:	93,812
Submission date:	24-Aug-2025 10:52PM (UTC+0200)
Submission ID:	2732366204

SCHOOL OF MEDICINE

DEPARTMENT OF INTERNAL MEDICINE

BIOCHEMICAL PATTERN OF ALCOHOLIC LIVER DISEASE AMONG ALCOHOL ABUSERS ATTENDING REHABILITATION CENTRES IN DAR ES SALAAM.

By

DR JOYCE ELIAS SABUKA (HK/PG/IM/23/9020)

SUPERVISOR: PROF F. MGONDA

CO SUPERVISOR: DR MARLEB CHARLES AND DR ALICE SWAMBOSO

A DISSERTATION SUBMITTED IN (PARTIAL) FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF MEDICINE (INTERNAL MEDICINE) AT KAIRAKI UNIVERSITY

2030