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ASSESMENT OF LIVER FUNCTION TESTS IN CIGARETTE SMOKER

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Abstract

Although cigarette smoking is known risk factors for systemic diseases, little is known about how they affect liver function, especially in Pakistani people. The purpose of this study was to evaluate the biochemical impacts of tobacco use on bilirubin, alkaline phosphatase (ALP), and alanine aminotransferase (ALT), three indicators of liver function, in adult smoking users. Over the course of six months, Sarhad University of Science and Information Technology in Peshawar used a cross-sectional comparative design. Purposive sampling was used to select 79 participants, who were identified as current tobacco users and ranged in age from 15 to 75. Automatic analyzers were used to measure the serum levels of bilirubin, ALP, and ALT. SPSS v25.0 was used to analyze the data, with a significance threshold of $p < 0.05$. According to the findings, 41 people had elevated ALT levels (>56 U/L), with the age group of 36 to 55 having the highest frequency. Seventy-three participants had elevated ALP levels, which may indicate hepatocellular or cholestatic stress. Only four of the 75 people had elevated bilirubin levels; the rest had levels within normal ranges (0.1–1.2 mg/dL). These results imply that tobacco use whether smoked is linked to notable changes in liver enzymes, especially elevated ALT and ALP, which indicate biliary dysfunction and hepatocellular damage. After quitting smoking, the biochemical alterations might be reversible and seem to be dose-dependent. This study emphasizes the necessity of routine liver function monitoring among smoking and users, as well as the need for greater awareness of tobacco's hepatotoxic potential. Tobacco cessation-focused public health initiatives may be essential in averting chronic x liver problems. These findings have significant ramifications for clinical practice and health policy, particularly in areas like Khyber Pakhtunkhwa, where tobacco products are widely used in Pakistan.

INTRODUCTION

The liver is the largest internal organ in the human body and a true biochemical powerhouse. It is primarily located under the diaphragm in the right upper quadrant of the abdomen. This essential organ, which weighs around 1.4 kg in an average adult, is a dynamic, multifunctional hub that is required for maintaining metabolic balance, getting rid of harmful chemicals, making essential proteins, and storing key energy reserves. Its vital role as a link between digestion and

systemic circulation is highlighted by its unique dual blood supply, which includes blood from the portal vein, which is rich in nutrients and toxins, and blood from the hepatic artery, which is rich in oxygen. The liver's amazing, if limited, potential for regeneration allows it to recover from injury, even though persistent insults may cause severe, sometimes permanent damage that affects almost every other system in the body (Trefts *et al.,* 2017).

The liver is anatomically separated into two main lobes (left and right), which are further separated into eight functionally distinct segments according to biliary and vascular drainage patterns. This classification is essential for contemporary surgical resection. Hepatic lobules are microscopic hexagonal functional units that make up each segment. Each lobule has a central vein at its core and portal triads with branches of the hepatic artery, portal vein, and bile ductile at its periphery. Hepatocytes, the main parenchymal cells of the liver, form cords that radiate outward from the central vein, with specialized capillaries known as sinusoids dotted throughout. The fenestrated endothelial cells that line sinusoids are home to resident macrophages called Kupffer cells, which are essential for phagocytosing pathogens and blood debris. Hepatic stellate cells (HSCs), which store vitamin A and become activated during fibrosis, are located in the "Space of Disse," which is the area between the sinusoidal endothelium and hepatocytes that facilitates effective material exchange. Bile, produced by hepatocytes, flows in the opposite direction to blood, entering bile canaliculi between hepatocyte membranes, eventually draining into the bile ducts within the portal triads (Poisson *et al.,* 2017).

The blood supply to the liver is particularly intricate and essential to its operations. The portal vein brings in about 75% of its blood flow, which includes absorbed nutrients, hormones from the gut, bacterial products, and possible toxins from the entire digestive system. The remaining 25% enters through the hepatic artery, supplying blood that is rich in oxygen and necessary for the high metabolic demands of the organ. Within the sinusoidal network, where fenestrated endothelium and slow flow allow for the close exchange of substances between hepatocytes and blood, these two blood streams mix. Blood flows into the central veins after passing through the sinusoids, and then converges into the hepatic veins before emptying into the inferior vena cava. A vital "first-pass effect" for both nutrients and medications is that substances absorbed from the gut are processed by the liver before entering the systemic circulation thanks to this complex vascular arrangement (Eipel *et al.,* 2010).

Perhaps the most distinctive function of the liver is metabolism. It serves as a central processing unit for proteins, fats, and carbohydrates. After a meal, the liver quickly absorbs glucose through insulin-dependent processes and GLUT2 transporters, turning it into glycogen for storage (glycogenesis). It maintains blood glucose levels for the brain and other essential organs during fasting by breaking down glycogen (glycogenolysis) and creating new glucose from precursors like lactate, glycerol, and amino acids (gluconeogenesis). Lipid metabolism includes the liver's oxidation of fatty acids for energy, synthesis of triglycerides and cholesterol, packaging of lipids into verylow-density lipoproteins (VLDL) for export, and production of ketone bodies as an alternative fuel during prolonged fasting. The majority of circulating plasma proteins, such as albumin (which is essential for oncotic pressure and transport), clotting factors (I, II, V, VII, VIII, IX, X, XI, XIII), fibrinogen, and transport proteins like transferrin and ceruloplasmin, are synthesized by the liver. Additionally, it breaks down amino acids, including the urea cycle, which turns toxic ammonia into urea for safe renal excretion (Roth *et al.,* 2012).

Another crucial hepatic function is detoxification, which mostly takes place in two stages inside hepatocytes. The cytochrome P450 (CYP450) enzyme superfamily plays a major role in phase I reactions, which include oxidation, reduction, or hydrolysis and frequently increase a compound's reactivity or water solubility. More hazardous 3 intermediates may occasionally be produced during this phase. In Phase II reactions, hydrophilic groups such as glucuronic acid,

sulfate, glutathione, or amino acids are attached to the molecule or its Phase I metabolite by enzymes, which greatly increases the water solubility for excretion in the kidneys or biliary system. Through these pathways, the liver effectively eliminates drugs, alcohol, environmental pollutants, and endogenous waste products (bilirubin, hormones like estrogen and aldosterone). This is demonstrated by the metabolism of bilirubin, which is produced when heme breaks down in senescent red blood cells. Hepatocytes absorb insoluble unconjugated bilirubin, conjugate it with glucuronic acid, and then expel it as bile. Jaundice results from impairment at any stage (Zanger & Schwab, 2023).

METHODOLOGY

Study Design

This study employed a cross-sectional comparative design to evaluate liver function biomarkers (ALT, ALP, and bilirubin) among cigarette smokers. The design allowed for the assessment of biochemical differences between exposed and non-exposed individuals at a single point in time.

Study Place

The current study was conducted within the chemical pathology department of Institute of Kidney (IKD) located in Peshawar Hayatabad.

Study Duration

The study was carried out over a period of six months, from January 2025 to June 2025, encompassing participant recruitment, sample collection, laboratory testing, and data analysis.

Sample Size

A total of 79 participants were included in the study. These individuals were categorized based on age and tobacco use status (cigarette smokers).

Sampling Technique

A non-probability purposive sampling method was used to recruit participants who met the inclusion criteria and were willing to participate. This technique ensured the selection of individuals with relevant exposure to tobacco products.

Sample Selection Criteria

Inclusion Criteria

Adults aged 15 to 75 years 2) Individuals who are current cigarette smokers 3) Participants who provided informed consent 4) Individuals with no known history of chronic liver disease

Exclusion Criteria

Individuals with known liver disorders (e.g., hepatitis, cirrhosis))

1 Alcohol consumers)

2 Patients on hepatotoxic medications)

3 Individuals with comorbid conditions affecting liver function (e.g., diabetes,)

4 cancer).

Sample Collection and Processing Venous blood samples (5 mL) were collected from each participant under aseptic)1 conditions. Samples were processed using centrifugation to separate serum.)2 Serum levels of ALT, ALP, and bilirubin were measured using automated)3 biochemical analyzers following standard protocols.

RESULT

Table 4.1 Shows the Serum ALT Level in different Age Group

Table 4.1 presents the distribution of serum ALT levels across three age groups. Elevated ALT levels (>56 U/L) were observed in 41 out of 79 participants, with the highest prevalence in the 36–55 age group (20 individuals). This suggests a possible age-related increase in hepatocellular stress among tobacco users. The 15–35 and 56–75 age groups showed similar distributions, indicating that liver enzyme elevation is not confined to older individuals.

Table 4.2 Shows the ALP Level in different Age Group

Serum ALP Level in Cigarette Smokers				
		Serum ALP Level		
		44-147 U/L	Above 147 U/L	Total
Age of Participant	15-35 years	1	22	23
	36-55 years	4	36	40
	56-75 years	1	15	16
Total		6	73	79

Table 4.2 show a striking elevation in ALP levels among tobacco users, with 73 out of 79 participants exceeding the normal range (>147 U/L). The 36–55 age group again showed the highest number of elevated cases (36 individuals), followed by the 15–35 and 56–75 age groups. Only 6 participants had ALP levels within the normal range, indicating widespread cholestatic or hepatocellular dysfunction in this population.

Table 4.3 Shows the Bilirubin Level in different Age Group

Serum Bilirubin Level in Cigarette Smokers				
		Serum Bilirubin		
		0.1 - 1.2 mg/dl	Above 1.2 mg/dl	Total
Age of Participant	15-35 years	21	2	23
	36-55 years	39	1	40
	56-75 years	15	1	16
Total		75	4	79

Table 4.3 ALT and ALP, bilirubin levels remained within normal limits (0.1–1.2 mg/dL) for the majority of participants (75 out of 79). Only 4 individuals showed elevated bilirubin levels, distributed across all age groups. This suggests that while liver enzymes are significantly affected by tobacco use, bilirubin may be less sensitive or slower to reflect hepatic impairment in this context.

Serum ALT Level in Cigarette Smokers :

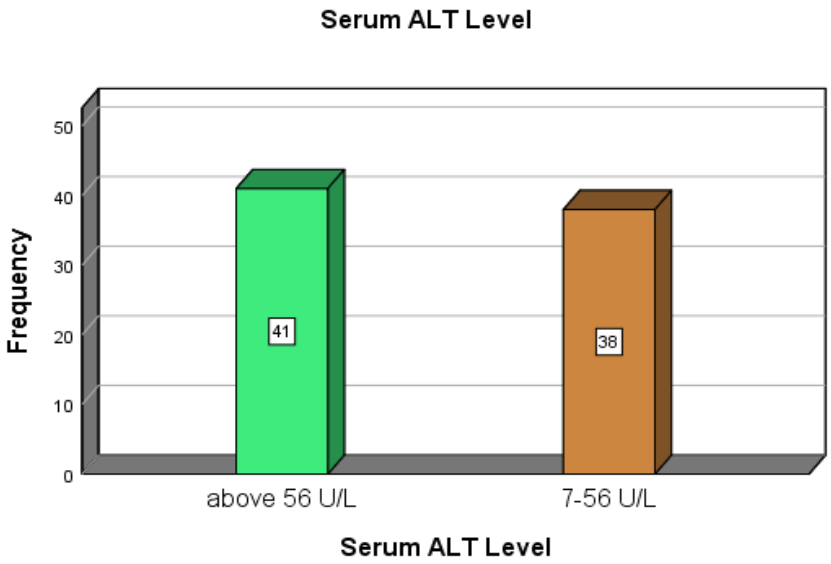

Figure no 1 Shows the ALT Level in Cigarette Smokers

This figure visually represents the distribution of ALT levels among participants. It highlights the predominance of elevated ALT values in the 36–55 age group, reinforcing the trend observed in Table 1. The graphical format aids in identifying age-related patterns of hepatocellular injury.

Serum Bilirubin Level in Cigarette Smokers:

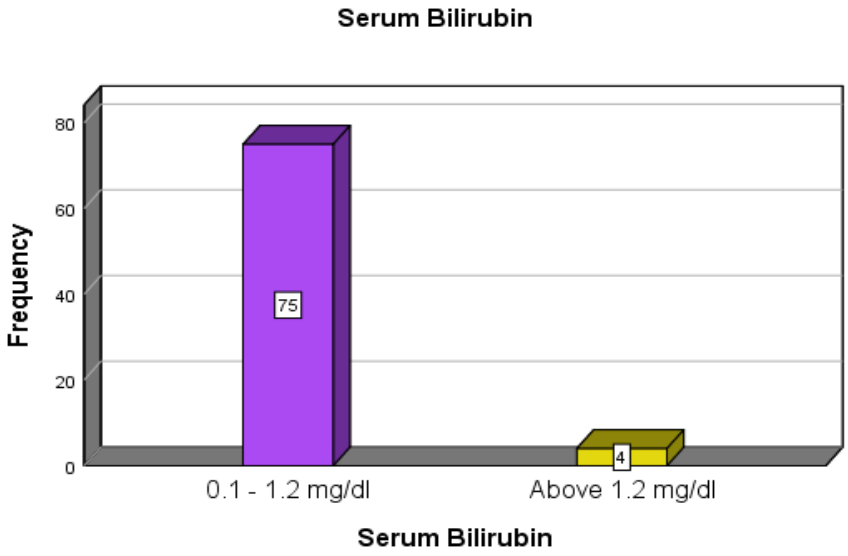

Figure no 2 Shows the Bilirubin Level in Cigarette.

The figure illustrates the bilirubin levels across age groups, showing that most participants fall within the normal range. The minimal elevation observed supports the conclusion that bilirubin is less affected by tobacco use compared to ALT and ALP.

Serum ALP Level in Cigarette Smokers:

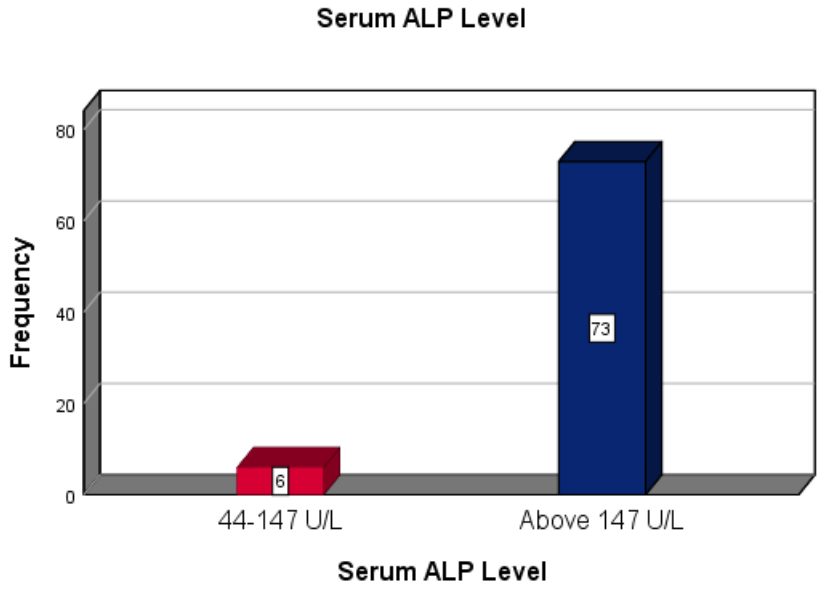

Figure no 3 Shows the Serum Alp Level in Cigarette Smokers.

This figure displays the ALP level distribution, emphasizing the high prevalence of elevated values among all age groups, particularly 36–55 years. The visual reinforces the data from Table 2, suggesting a strong association between tobacco use and cholestatic liver stress.

DISCUSSION

The current study set out to assess how smoking cigarettes using affects the biomarkers of liver function in adult Pakistanis, specifically bilirubin, alkaline phosphatase (ALP), and alanine aminotransferase (ALT). The findings indicate that liver enzyme levels, particularly those of ALT and ALP, are significantly altered in tobacco users, and age-related trends suggest that hepatic stress increases with time. These results contribute to the growing body of evidence linking tobacco use to hepatocellular damage and biliary dysfunction. Elevated ALT is a crucial measure of hepatocellular integrity and typically denotes liver cell damage. 41 out of 79 participants in the study had ALT levels above the normal threshold (>56 U/L), with the highest frequency occurring in the 36–55 age group. This pattern suggests that long-term exposure to tobacco toxins may lead to progressive liver damage, particularly in middle-aged individuals. Further evidence that tobacco users may suffer from hepatic stress at a relatively young age, perhaps as a result of prolonged or high-intensity exposure, comes from the fact that younger participants (15–35 years old) had elevated ALT. These findings are in line with past studies that demonstrated smoking weakens hepatocyte membranes and increases serum ALT levels by causing inflammation and oxidative stress (Zahran *et al.*, 2018).

Bile duct function and cholestasis are associated with ALP, another significant liver enzyme. 73 out of 79 participants had ALP levels above the normal range (>147 U/L), with the age group of 36 to 55 having the highest prevalence. Although it can also be an indication of biliary obstruction, hepatic inflammation, or alterations in bone metabolism, elevated ALP in the context of tobacco use is most likely a sign of hepatic stress. Cigarette smoking have a substantial impact on biliary function, as evidenced by the widespread rise in ALP seen across all age groups. This is consistent with research by (Al-Mustafa et al., 2015).

The age-wise distribution of abnormal liver enzymes provides additional insight into the

onset of hepatic effects. The 36–55 age group consistently had the highest number of elevated ALT and ALP cases, suggesting that middle-aged individuals may be more susceptible due to cumulative toxic effects or longer exposure times. The 15–35 age group also showed significant increases, indicating that hepatic stress occurs earlier in life for younger tobacco users. These findings demonstrate the importance of early intervention and regular liver function monitoring for tobacco product users of all ages. The biochemical alterations observed in this study are most likely caused by the toxic nicotine, polycyclic aromatic hydrocarbons, and reactive oxygen species found in tobacco. These chemicals induce oxidative stress, lipid peroxidation, and inflammatory responses in hepatic tissues, which results in enzyme leakage into the bloodstream. Snuff is absorbed through the oral mucosa and contains similar toxic substances, which can cause systemic toxicity even though it is smokeless. Similar enzyme elevations between snuff users and smokers in this study show that smokeless tobacco is not a safer alternative and presents significant hepatic risks. The implications of these findings for public health are significant. Tobacco use is still widespread in Pakistan, particularly in regions like Khyber Pakhtunkhwa where socioeconomic and cultural factors contribute to the prevalence of use. Hepatic dysfunction brought on by tobacco use may be an underappreciated health burden in these communities, based on the elevations in liver enzymes that have been observed. Regularly screening tobacco users for liver function biomarkers may help identify hepatic stress early and prevent the onset of chronic liver disease. By emphasizing the hepatotoxic potential of both smoking and snuff use, public health campaigns should also dispel the myth that smokeless tobacco is safe, Whether liver enzyme increases can be reversed after quitting smoking is another important consideration. Studies have shown that ALT and ALP levels can progressively return to normal after stopping smoking, signifying hepatic recovery (Lee et al., 2017).

CONCLUSION

This study demonstrates that cigarette smoking are associated with significant elevations in liver enzymes, particularly ALT and ALP, among adult individuals in Pakistan. These biochemical changes reflect hepatocellular and biliary stress and are more pronounced in middle-aged users. Bilirubin levels remain largely unaffected, suggesting selective hepatic impact. The findings highlight the need for routine liver function monitoring, public health education, and targeted cessation interventions to address the hepatotoxic risks of tobacco use. Given the high prevalence of tobacco consumption in Pakistan, these measures are essential for reducing the burden of liver-related morbidity and promoting long-term health.

RECOMMENDATIONS

Based on the findings of this study, several key recommendations can be proposed to address the hepatic risks associated with cigarette smoking:

1. Routine Liver Function Screening for Tobacco Users Healthcare providers should incorporate regular liver function tests (LFTs), including ALT, ALP, and bilirubin, into clinical assessments of individuals who smoke or use cigarette. Early detection of enzyme abnormalities can facilitate timely intervention and prevent progression to chronic liver disease.
2. Public Health Campaigns on Hepatic Risks of Tobacco Awareness programs should emphasize the liver-related consequences of both smoking and smokeless tobacco use. Messaging should challenge the misconception that snuff is a safer alternative, highlighting its comparable hepatotoxic effects.

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