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EFFECT ON LIVER FUNCTION TESTS IN SMOKERS & NON-SMOKERS

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ABSTRACT

Cigarette smoking is a major health concern in India. Cigarette smoking has well-documented negative effects on the lungs and cardiovascular system, but its impact on the liver is less clear. There are three ways that smoking damages the liver: cancer-causing, immunologic, and toxic (both directly and indirectly). Smoking produces cytotoxic chemicals that lead to increased inflammation and fibrosis. The act of smoking cigarettes is linked to an elevation in the synthesis of pro-inflammatory cytokines, including IL-1, IL-6, and TNF-α, which may cause damage to liver cells. Secondary polycythemia results in an increase in the amount of red blood cells and their turnover. This condition might potentially lead to oxidative stress in liver cells and an excess of iron in the body. Tobacco consumption can lead to liver injury, which can be detected through suspicious results on liver function testing. Increased amount of liver enzymes, such as gamma-glutamyl transpeptidase (GGT) and alkaline phosphates (ALP). are associated with it. While AST and ALT were unaffected by smoking, total protein, albumin, and GGT levels in the blood were. Hepatocyte hypoxia is a result of reduced oxygen levels in the bloodstream, which is a direct consequence of smoking.

KEYWORDS: *Sgpt, Sgot, Alp, Smoking, Liver Function, Tobacco.*

INTRODUCTION

Smoking is a major factor that affects the human health. In modern times smoking is still a huge problem because smoking can affect smokers and secondary persons as well. Every year more than 8 million people are killed and approximately 1.2 million are killed by secondhand smoke1.

Nicotine is a component of HTP smoke and it affects the adolescent brain and makes them addicted to it2. Other than nicotine it contains polycyclic aromatic hydrocarbons which are carcinogenic in nature3. Tobacco smoke contains approximately 4000 types of chemicals which may cause adverse effects on the body4.

A total of 28.5% of males and 2.1% of females had ever tried smoking. The percentage of people who smoked cigarettes was 47.5% in urban areas and 12.5% in rural areas. On the other hand, the percentage of people who smoked bidis was 51.7% in urban areas and 81.2% in rural areas each. Hookah was used by the remaining smokers either on its own or in conjunction with other smoking products5.

When a person comes in contact with smoke exhaled by a smoker is called secondhand smoke. SHS is very dangerous for pregnant women6.In healthy individuals SHS can cause NAFLD in this disease abnormal lipid content is deposited in the liver7. In research, it is demonstrated that in mice SHS can cause metabolic disease in the liver8.

SYSTEMIC EFFECT OF SMOKING

Smoking causes harm to almost all of the organs in the body. This leads to several ailments, lowers quality of life, and diminishes life expectancy. Smoking increases the risk of several ailments, some of which can be deadly, compared to non-smokers. However. Smoking-related medical disorders, while not lethal, can lead to years of chronic disease or other difficulties. Disease is:

- Asthma
- Immune system(impaired)
- Chronic rhinitis
- Sperm count reduced
- Sperm motility impaired
- Optic neuritis
- Sperm shape abnormalities increased
- Angina
- Back pain
- Neck pain41

LIVER FUNCTIONS

The liver is responsible for a diverse range of activities, including the breakdown of bilirubin, among other functions. bile secretion, the storage of minerals and vitamins, the metabolism of food, the detoxification of metabolic waste, and the regulation of vascular and hematologic processes. The liver's detailed function is as follows:

1. SECRETION OF BILE

The main and secondary bile acids are the precursors to the formation of bile salts in the liver. Conjugated bile acids, such as bile salts, throughout the digestive tract, play a significant part in the process of emulsification and the absorption of fatty proteins

2. BILIRUBIN METABOLISM

Bilirubin is a catabolic byproduct that is generated during the breakdown of aged erythrocytes. Mononuclear phagocytes, mostly located in the spleen and liver, capture and eliminate old red blood cells. The unconjugated bilirubin is transported from the plasma to the hepatocytes in the liver by the sinusoids. It forms a bond with glucuronic acid in liver cells to produce a water-soluble form of bilirubin known as conjugated bilirubin. Through conjugation, Bilirubin may change from a solubilizing in lipids substance to another form entirely by conjugation. And passes across cellular membranes into a compound that can dissolve in water and be expelled by bile. The deconjugation of conjugated bilirubin occurs when it reaches the distal ileum and colon, where bacteria are present. converting it into urobilinogen. The majority of urobilinogen is subsequently expelled via urine, while a little quantity is evacuated in feces.

3. FUNCTIONS RELATED TO BLOOD VESSELS AND BLOOD

Microorganisms and other particles are removed from the portal flow by kupffer cells, which are located in the sinusoids of the liver. The removal of intestinal pathogens and the prevention of infections are both significantly aided by the important function that kupffer cells perform. This is due to the fact that the liver receives all of the venous blood from the pancreas and the digestive tract. In addition, the liver is responsible for hemostatic functions. Blood clotting factors, fibrinogen, and thrombin are all produced by it. Additionally, the creation of extra clotting components necessitates the presence of vitamin K, which is a fat-soluble vitamin. As a result of the fact that bile salts are essential for the process of fat reabsorption, the capacity of the liver to create enough quantities of vitamin K is essential for its absorption15.

4. METABOLISM OF NUTRIENTS

The main location where glucose and protein are converted into fat is the liver. Triglycerides make up the majority of the absorbed fat via lacteals in the intestinal villi, which makes its way to the liver via the lymphatics. The liver has two ways to break down triglycerides: either it uses them to produce adenosine triphosphate (ATP), which is a metabolic energy source, or it disperses them throughout the body as lipoproteins. Lipoproteins are transported from the circulation to adipose cells, where they are stored. It is via the phospholipid and cholesterol manufacturing processes that the liver creates bile salts, steroid hormones, and components of the plasma membrane, among other unique chemicals. With the exception of gamma-globulin, the liver produces albumins, globulins, and other proteins found in plasma. Not only does the liver produce non-essential amino acids, but it also produces blood enzymes including AST, ALT, LDH, and ALP15.

Sugars and carbs: In response to low blood sugar (hypoglycemia), the liver releases glucose into the bloodstream. In response to high blood sugar (hyperglycemia), after glucose is absorbed by the liver, it may be converted to fat (gluconeogenesis) or stored as glycogen (gluconeogenesis). At the end of the glycogen depletion process, the liver may produce glucose from glycerol and amino acids15.

5. DETOXIFICATION OF METABOLIC SYSTEMS

Both exogenous and endogenous chemicals, such as pharmaceuticals, as well as foreign molecules and hormones are altered by the liver in order to lessen the toxicity or biological activity of these compounds. The hepatocytes are damaged when these end products are produced as a result of excessive consumption of alcohol over a prolonged duration. Acetaldehyde causes harm the mitochondria of cells, and the presence of excess hydrogen encourages the storage of fat. This is the manner in which alcohol hinders the liver's capacity to operate15.

6. STORING MINERALS AND VITAMINS

The process of storing minerals and vitamins, the liver is responsible for storing particular nutrients and vitamins, such as iron and copper, when they are taken in excess. The liver then releases these minerals and vitamins when they are required. It is possible for the liver to store vitamins B12, D, and A for a good number of months or even years. Vitamin K and vitamin E are also stored in the liver. The liver retains iron by binding it with proteins to produce ferritin, and then releases it when it is required for the creation of red blood cells15.

7. THE LIVER'S IMMUNOLOGICAL ROLE

The main hematopoietic organ at certain phases of fetal development is the liver, which also maintains this role after delivery. From local hematopoietic stem cells, it may create all leukocyte lineages. Hemopoietic stem cells along other hematopoietic cells make up the portal tract of the liver. Cells in the liver participate in both the adaptive and innate immune responses15.

LIVER TESTS

During the review of liver function tests, the following markers are commonly discussed: SGPT, SGOT 5'-nucleotidase, ALP, GGT, TB, conjugated bilirubin, and unconjugated bilirubin, as well as PT, INR, LDH, total protein, globulins, and albumin are some of the other tests that are performed. In addition to providing useful insights into the region of liver injury, these tests may also aid in establishing a differential diagnosis by providing information about the pattern of elevation16.

1. ALT

The kidney, muscles, heart, and liver are all places where you might find the enzyme known as alanine aminotransferase17. Significant quantities of it, on the other hand, are discovered in the liver. The rise in the concentration of ALT may be attributed to any kind of harm that occurs to this component of body. Patients who suffered from hepatitis C have a higher incidence of it. In a recent research, it was shown that drinking coffee and tea lowers the likelihood of having an elevated ALT level15. The ALT might be within the usual range of up to 40 U/L17.

2. AST

A wide variety of organs contain the enzyme aspartate aminotransferase, including the liver and heart. There are a lot of biological processes that this enzyme aids in facilitating. A high AST level in the blood may indicate hepatocyte damage. Up to 40 IU/L is considered typical for AST17.

3. ALP

Placenta, small intestine mucosal epithelia, bones, liver, and kidneys all contain ALP in their proximal convoluted tubules. Limpid transit in the colon and bone calcification are both regulated by it. The liver is the primary source of serum ALP activity, with 50% of the activity originating from bone18.

An increase in ALP levels may also be caused by metastases to the liver or bones. Amyloidosis, granulomatous liver disease, infections, infiltrative liver disease, and pus are among the additional disorders that may cause an elevation of ALP. Mildly elevated ALP values may be the result of cirrhosis, hepatitis, or congestive heart failure19.

4. GGT

The microsomal enzyme GGT is found in many different organs and tissues, including the intestines, pancreas, biliary epithelial cells, kidneys, and liver cells. Additionally, it is present in the membranes of cells, where It has a function in the metabolism of glutathione and is important for delivering peptides into cells. activity; nevertheless, larger quantities of this activity have been seen in renal tissue18.

5. BILIRUBIN

The breakdown of heme-containing proteins, notably hemoglobin, results in the production of bilirubin, which is a pigment in bile that is orange-yellow in color. The digestion of heme results in the formation of biliverdin, which is subsequently converted into unconjugated or indirect bilirubin (UCB). The UCB molecule is insoluble in water and forms a bond with albumin in the bloodstream. UCB is made water-soluble (direct bilirubin) by the addition of glucuronic acid in the liver, which is the process that occurs. Subsequently, it is either eliminated by the bile or reabsorbed into the bloodstream, where it undergoes filtration by the kidneys and is expelled via urine.

Bilirubin is a commonly used biochemical diagnostic for people with liver impairment and associated conditions. Nonetheless, bilirubin is not To accurately diagnose liver function, test findings must be carefully interpreted, since they might be sensitive or specific markers.

Long-term smoking can induce oxidative imbalance in the body, It can cause depletion in the vitamin level like vitamins A and C. The Level of CRP is higher in the smokers. Although most of the bad effects of smoking are reversible means condition is improved after quitting smoking but some parameters remain high after quitting smoking like CRP9.

DISEASE OF THE LIVER

1. NAFLD

Despite its previous obscurity, non-alcoholic fatty liver disease (NAFLD) has rapidly emerged as the predominant cause of chronic liver disease worldwide. Currently, most people think that NAFLD is a catch-all word for a bunch of different diseases that involve metabolic potential dangers (especially being overweight and having type 2 diabetes) and steatosis in over 5% of hepatocytes, excluding chronic liver diseases caused by excessive alcohol consumption20.

Being overweight and having insulin resistance (IR) may slow down lipid metabolism and cause inflammation that can speed up the advancement of non-alcoholic steatohepatitis, cirrhosis (HCV), and eventually mortality. In clinical practice, biochemical testing reveals that individuals with NAFLD have high triglycerides, raised LDL, and lowered HDL21.

Nevertheless, the pathophysiology of NAFLD is still unknown, which has impeded its treatment. The "first-hit" is IR and hepatic steatosis caused by excess fatty acids, according to early studies. The "second-hit" is the injury, inflammation, fibrosis, and other pathological alterations that hepatocytes ultimately undergo as a result of oxidative stress and lipid peroxidation22.

2. OXIDATIVE STRESS

Typically, DNL changes excess crabs to fatty acids. So, hepatocytes store these triglycerides (TGs) formed when esterification transforms these fatty acids. When the body's energy levels drop, TG uses β-oxidation to make up the difference. Inflammation and oxidative stress may result from elevated FFA levels in the liver, which can influence β-oxidation and mitochondrial activity. The inflammatory response is significantly mediated by reactive oxygen species (ROS) 23.

3. LIVER CIRRHOSIS

Many chronic liver illnesses progress to cirrhosis, the end stage of the disease. In Western countries, cirrhosis is most often caused by alcoholism, severe hepatitis C virus infection, and fatty liver disease, but different people may have different reasons. In spite of this, chronic hepatitis B accounts for the highest number of cases of liver cirrhosis in the Asia-Pacific area. Alternatively, cirrhosis of the liver is most often caused by chronic hepatitis B in the Indo-Asian region.

Some pathological symptoms are shared by all cases of liver cirrhosis, but there are several potential causes of cirrhosis. Hepatocyte death and degeneration, fibrotic tissue and regenerative nodular replacement of liver parenchyma, and liver dysfunction are all hallmarks of this condition. Fibrosis, a precursor to cirrhosis. This is a crucially important pathogenic phase in the progression of all chronic liver disorders that ultimately result in cirrhosis24.

4. LIVER CANCER

More than one million people are expected to pass away as a result of liver cancer by the year 2030, according to predictions provided by the World Health Organization. Liver cancer makes up the fourth top cause of mortality worldwide that is connected to cancer26.

Alcohol use over an extended period of time, infections with the hepatitis virus, as well as more recently, nonalcoholic fatty liver syndrome are the key risk factors for liver cancer. Iron excess, smoking, obesity, and diabetes have all been associated to the development of NAFLD27.

5. JAUNDICE

Bilirubin metabolism is a sophisticated and intriguing demonstration of the body's ability to discard, regenerate, and recycle essential materials. Jaundice is a warning indication of systemic dysfunction. Jaundice, like pain, is a compelling reason to see a doctor. Jaundice is commonly linked with hepatitis by nonclinicians; however its causes can range from benign to dangerously malignant. Patients may have a variety of idiopathic or nosocomial diseases that contribute to their icteric state. This study outlines the phases of bilirubin metabolism, lists the causes of bilirubin derangement, and investigates patient factors and medications that might lead to hyperbilirubinemia and jaundice.

CHARACTERISTICS OF SMOKERS

In smokers there is many characters are present which distinguish from nonsmokers, these are:

- They drink more coffee and tea than the average person.
- They have a higher alcohol intake.
- They become tired during exercise.
- The total TLC, DLC, hematocrit, serum Ige levels, and platelet count all indicate a little increase in their values.
- In addition, their immune systems are less robust.
- They have lower amounts of leukocyte ascorbic acid.
- They have lower serum albumin levels than nonsmokers.
- Their levels of uric acid in the blood are reduced.
- The SGPT has been raised to the SGOT level.
- A decrease in the generation of PGI2, It is a powerful substance that widens blood vessels, generated by the cells lining the blood vessels, and prevents blood platelets from sticking together.
- They possess a greater quantity of pulmonary macrophages, which are linked to a distinct metabolic pathway.
- Based on the results of the lung function test, they have abnormal findings.
- Tobacco smoking stimulates hepatic microsomal enzymes and interacts with several medications.
  - i) Propranolol-assisted first pass clearance
  - ii) Propoxyphene reduces analgesia,
  - iii) Benzodiazepines reduce sedation,
  - iv) Beta blockers are known to lower overall blood pressure and heart rate.
  - v) Chlorpromazine lowers serum levels,
  - vi) Oral estrogens boost liver clearance, and haloperidol lowers serum concentrations.
  - vii) Heparin promotes quicker clearance.
  - viii) Insulin absorption is limited due to cutaneous vasoconstriction

MATERIALS AND METHODS

Research Approach: Quantitative

Research Design: Case-Control Study

Variables under study: Age, smokers, nonsmokers.

Study Setting: The current investigation was carried out in Uttar Pradesh's Gonda district. The trial will last for six months.

Target Population: Smoking Population

Inclusions:

- Age 18 to 60 years
- A group of 100 male individuals who have never engaged in smoking.
- There are 100 males who have been smoking for duration of 3 years or more.

Exclusion:

- Those who will be limiting their diet.
- Individuals who are under the influence of medication are known to change their LFT profiles.
- Individuals who have a known history of drinking alcohol.
- People who have disorders that are known to change their LFT profile such as Liver cirrhosis, Hepatitis, and Jaundice.
- Due to the lower prevalence of smoking among women in our nation, this research will only focus on males.

Sample Size:

The study will consist of a sample size of 100 individuals who smoke and 100 individuals who do not smoke.

Sample Techniques:

- Simple random sampling

RESULTS

The table 3.1 presents the distribution of 200 individuals according to age group, control group, and smoking duration. In the control group, the highest percentage is observed in the 20–30 years age group (15.5%), followed by 40–50 years (14.5%), indicating more non-smokers or baseline individuals are younger and middle-aged. In case group-2 (smokers for more than 3 years), the proportion increases with age, with the highest in 50–60 years (11.5%) and 40–50 years (11%), suggesting long-term smoking is more prevalent among older individuals. In contrast, case group-3 (smokers for less than 3 years) shows the highest percentage in the 20–30 years group (14.5%), with very low percentages in older groups. Overall, the data indicates that younger individuals are more likely to be recent smokers, while older individuals tend to have a longer duration of smoking, highlighting a clear relationship between age and smoking duration.

Table 3.1: Distribution of Control, Age Group and Duration of Smokers

Age group in years	Control group-1		Case group-2 (More than 3 years)		Case group-3 (Less than 3 years)	
	N=200					
	Patient count	Percentage %	Patient count	Percentage %	Patient count	Percentage %
20-30	31	15.5	5	2.5	29	14.5
30-40	21	10.5	15	7.5	4	2
40-50	29	14.5	22	11	1	0.5
50-60	19	9.5	23	11.5	1	0.5

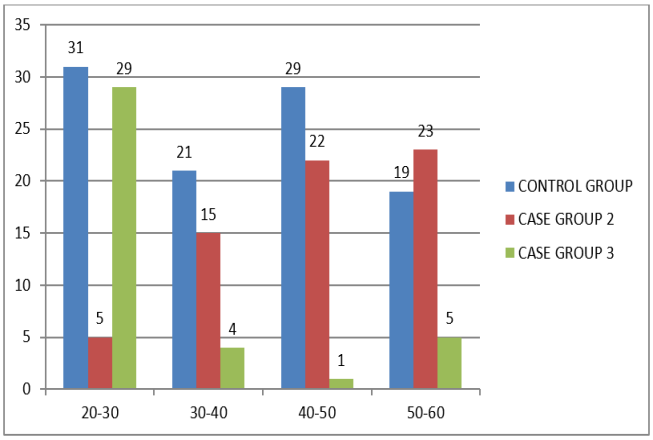

Figure 3.1: Distribution of Control, Age Group and Duration of Smokers

The table 3.2 compares liver function parameters between smokers and non-smokers (N=100 each) based on normal and high ranges. Among smokers, elevated levels are more common across all enzymes. SGOT shows 38% in the high range among smokers compared to 20% in non-smokers. Similarly, SGPT is elevated in 34% of smokers versus 17% of non-smokers. ALP levels are also higher in smokers (41%) compared to non-smokers (11%), indicating a notable difference. However, bilirubin levels remain almost similar in both groups, with only 3% smokers and 4% non-smokers showing elevated values.

Overall, the data suggests that smoking is associated with increased liver enzyme levels, particularly SGOT, SGPT, and ALP, which may indicate liver stress or damage. Non-smokers generally maintain normal enzyme levels more frequently. The minimal difference in bilirubin suggests that smoking has less impact on this parameter. These findings highlight the potential harmful effects of smoking on liver function and emphasize the importance of avoiding smoking to maintain liver health.

Table 3.2: Showing correlation of liver parameters in smokers vs. nonsmokers

Parameters	Smokers		Nonsmokers	
	N=100		N=100	
	Normal range	High range	Normal range	High range
SGOT	62	38	80	20
SGPT	66	34	83	17
ALP	59	41	89	11
Bilirubin	97	3	96	4

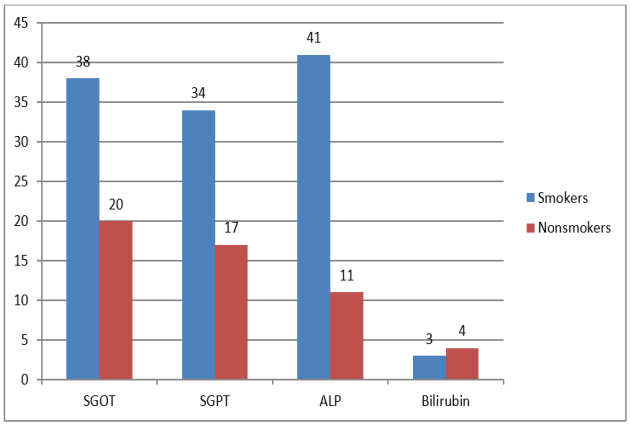

Figure 3.2: Showing correlation of liver parameters in smokers vs. nonsmokers

Table 3.3 compares clinical and biochemical parameters between smokers of more than 3 years (N=65) and less than 3 years (N=35), along with their statistical significance (p-value).

The mean age in both groups is almost similar (38.67 vs 38.24 years) with a high p-value (0.869), indicating no significant age difference. Similarly, liver enzymes SGOT and SGPT show very close mean values in both groups with p-values of 0.791 and 0.657 respectively, suggesting no statistically significant difference based on duration of smoking.

However, ALP shows a noticeable difference, with higher levels in long-term smokers (283.22) compared to short-term smokers (246.82), and a p-value of 0.0355. Since this is less than 0.05, it is statistically significant, indicating that prolonged smoking may affect ALP levels.

Total bilirubin levels are almost identical in both groups (0.54 vs 0.53) with a non-significant p-value (0.828). Overall, only ALP shows a significant association with smoking duration, while other parameters do not.

Table 3.3: Comparison of Variables among the Smoker (Smoker More Than 3 Year) and Smoker (Less Than 3 Year)

Variables	Greater than 3 year Smoker [Mean ± SD] N=65	Less than 3 year Smoker [Mean ± SD] N=35	P-Value	Conclusion
Age in years	38.67±12.38	38.24±12.59	0.869	Non significant
SGOT	35.41±11.85	34.75±11.90	0.791	Non significant
SGPT	35.93±12.78	34.77±11.75	0.657	Non significant
ALP	283.22±74.64	246.82±92.90	0.0355	Significant
Total bilirubin	0.54±0.22	0.53±0.22	0.828	Non significant

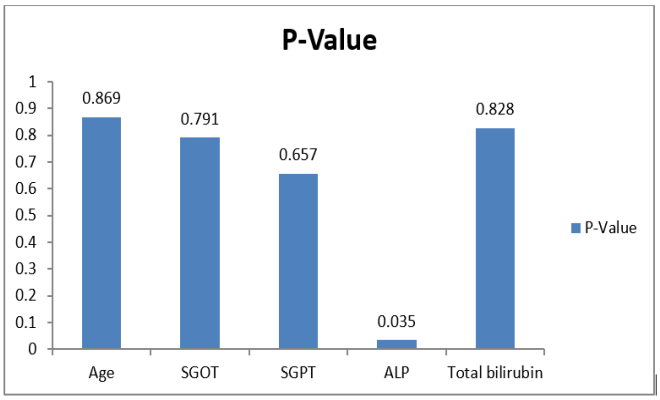

Figure 3.3: Comparison of Variables among the Smoker (Smoker More Than 3 Year) and Smoker (Less Than 3 Year)

Table 3.4 compares liver function parameters between smokers and non-smokers using mean ± standard deviation, along with p-values to assess statistical significance.

For SGOT, smokers (35.20) have higher levels than non-smokers (32.07), with a p-value of 0.0431, indicating a statistically significant difference. Similarly, SGPT levels are higher in smokers (35.69) compared to non-smokers (32.31), and the p-value (0.0374) confirms significance. ALP also shows a notable increase in smokers (250.49) versus non-smokers (210.80), with a significant p-value of 0.0347.

In contrast, total bilirubin levels are slightly lower in smokers (0.54) compared to non-smokers (0.59), but the p-value (0.101) is greater than 0.05, indicating no significant difference.

Overall, the findings suggest that smoking has a significant impact on liver enzymes (SGOT, SGPT, and ALP), indicating possible liver stress or damage, while bilirubin levels remain unaffected.

Table 3.4: Statistical Comparison of Liver Parameters in Smokers & Nonsmokers

Parameters	Smokers [Mean ± SD]	Nonsmokers [Mean ± SD]	P-Value	Conclusion
SGOT	35.207±11.956	32.075±9.681	0.0431	Significant
SGPT	35.691±12.950	32.311±9.618	0.0374	Significant
ALP	250.49±93.03	210.80±78.86	0.0347	Significant
Total Bilirubin	0.54± 0.22	0.59± 0.21	0.101	Non Significant

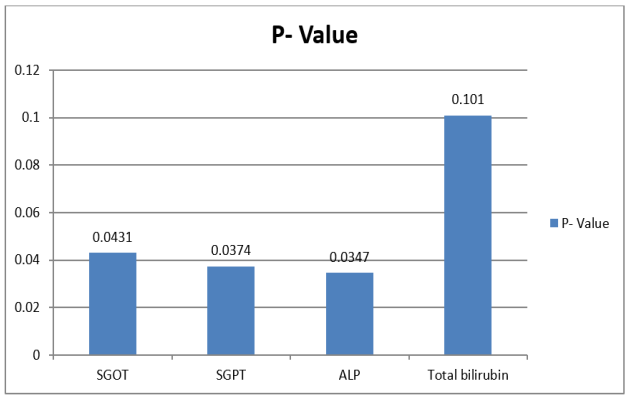

Figure 3.4: Statistical Comparison of Liver Parameters in Smokers & Nonsmokers

DISCUSSION

In the present study, 100 smokers and 100 nonsmokers were included. The study focused on examining the specific impacts of smoking on liver function. Variables that are included are age, duration of smoking, and weight.

It is revealed that SGOT, SGPT, and total bilirubin of smokers of more and less than 3 years are not statistically significant (P >5). But ALP of smokers of more and less than 3 years are statistically significant (P<5). The SGOT, SGPT, and ALP in comparison to smokers and nonsmokers are statistically significant (P<5). However, bilirubin is not statistically significant in comparison to smokers and nonsmokers.

In support of these clinical findings Abdul Razaq et al proposed a mechanism that connect a link that this might be related to nitrosative stress, A scenario in which the human body is unable to neutralize and eliminate very active nitrogenous molecules, such as nitrous oxide, because the production of these compounds surpasses the capacity of the body to do so. One sensitive biomarker of liver injury is serum aminotransferases (Rochling, 2001). Lipid peroxidation, which is caused by cigarette smoking, is responsible for the damage that occurs to the bio membrane of the liver42.

This study examined whether smoking may induce a shift in serum liver enzyme levels SGOT and SGPT. In addition to aldehydes, hydrogen cyanide, lead, and cadmium, cigarette smoke contains a substantial amount of gaseous compounds. These gaseous chemicals consist of, among other things, carbon monoxide (CO), nitrogen dioxide (NO2), and nitric oxide (NO). In addition, it has a high concentration of free radicals that may be discovered by analysis. SGOT and SGPT are highly concentrated in cells of the heart and liver. Cigarette smoke causes lipid peroxidation, damaging the liver and heart membranes. Smokers have higher levels of SGOT and SGPT in their blood than non-smokers due to enzyme leakage. Our investigation found that SGOT and SGPT activities were elevated39,(Ramamurthy et al.)

Studies have found a link between excessive smoking and higher levels of SGPT and SGOT. Cigarette smoking increases the amount of chemicals in the body, leading to permanent liver damage and elevated liver enzymes. In 2017, Jabbar, D.K., and Abdul Hasan, H.K did a study. The usage of tobacco has been associated with elevated concentrations of numerous biochemicals, such as VEGF, TNF-a, IL-1, and IL-6, growth factor-b, and angiotensin II. In addition to lowering glutathione levels and causing free radical lipid peroxidation, smoking is responsible for activating the NADPH oxidase system. This results in inflammation of the hepatocytes, cell damage, initiation of the stellate cells, and activation of the mesangial cells, all of which contribute to the creation of fibrosis causing mediators, matrix metalproteinases, and extracellular proteins. Iron accumulation is another factor that may lead to fibrosis. In addition to El Zayadi30.

The effects of smoking include an increase in the amounts of carboxyhemoglobin and a reduce in the capacity of RBCs to carry oxygen; It finally results in a deficiency of oxygen in the tissues. Hypoxia stimulates the synthesis of erythropoietin, leading to an increase in bone marrow disorder. The oxidative stress of liver cell is caused by secondary high concentration of RBCs as well as a rise in bulk of erythrocytes (S. M. Rutledge and others)

Current research found that those who smoked cigarettes had a considerably greater level of blood SGPT and SGOT activity compared to the group that served as the control. In the course of my experiment, I discovered that smokers' plasma exhibited significant increases in SGPT and SGOT activity (P<0.05). As a result, my research indicates that those who smoke is at a greater risk than people who do not smoke. Recent studies by Durar Kamil Jabbar et al., Sara Ismaeel et al, have also shown that smoking can cause a rise in SGPT and SGOT levels. In my study, out of 100 smokers, 38 individuals have high levels of SGOT, and 34 individuals out of 100 have high levels of SGPT. In the nonsmoker group, 20 individuals have a high level of SGOT and 17 have SGPT.

Cigarette smoking changes the hematological system by increasing eosinophil, basophil, monocyte, lymphocyte, platelet, and macrophage levels In addition to this; it raises the quantities of RBC and hemoglobin in the blood. Nicotine, the most important of the components that are found in cigarettes, works to stimulate the release of hormones, which results to the accumulation of blood components, an increase in the stickiness of blood vessels, and the aggregation of platelets and blood cells. (Pankaj et al 2014).

Young adult smokers' livers are relatively robust, although smoking can still cause high SGPT and SGOT levels. SGPT & SGOT levels slight to moderate increase. Metabolism is normally higher, and the liver has a greater ability to recover, but the combined stress of smoking and lifestyle variables can cause a significant enzyme increase.

Middle-aged smokers frequently see a considerable rise in SGPT and SGOT levels. SGPT & SGOT levels moderate to high increase. Prolonged exposure to tobacco toxins, potential onset of metabolic syndrome, increased likelihood of co-morbidities like hypertension or diabetes, which can exacerbate liver damage.

In older individuals, the body's ability to heal and regenerate declines, making the liver more vulnerable to smoking-related harm. This age group had the greatest rise in SGPT and SGOT values, indicating considerable liver stress and possible chronic liver disease.

The elevation of liver enzymes in smokers can be attributed to oxidative stress and inflammatory responses triggered by toxic compounds in tobacco smoke[10,6].

These compounds lead to hepatocyte damage and increased permeability of liver cell membranes, resulting in leakage of enzymes into the bloodstream[11,12].

The findings of this study are consistent with previous research highlighting the adverse effects of smoking on liver health[5,13,14].

CONCLUSION

Cigarette smoking has been shown to have an impact on liver function, as shown by the concentration of liver enzyme secretion in blood serum, according to the results. The secretion of the liver enzymes SGOT and SGPT may be affected by tobacco smoking, which may have an effect on the activities of the liver. When compared to those who do not smoke, smokers are more likely to get liver damage. Smokers have a spike in their SGPT and SGOT levels that varies with their age. Those who are younger have had a little increase, those who are middle-aged have experienced a more substantial increase, and those who are old have experienced the biggest amounts. When relate to non-smokers, smokers have greater levels of ALP in their bodies to begin with. A significantly lower amount of bilirubin is discovered in smokers when compared to the level seen in nonsmokers.

More and more research is linking smoking to poorer clinical outcomes, liver cell carcinoma development, and the onset and progression of liver disease. A greater understanding of smoking's negative impact and plan to motivate and guide patients in quitting should be developed by hepatologists. Although many medical professionals are aware of this situation, they do not routinely recommend their patients to programs that help them quit smoking. outcomes from clinical studies including NAFLD, fibrosis, and HCC should be considered with the possibility that active smoking might influence those outcomes. Because smoking is associated with worse outcomes for liver transplant recipients, more hospitals should make quitting smoking a mandatory requirement for patients. Research in the future should look at the link between liver-related consequences and measurable smoking markers such urine nicotine levels.

The impact of smoking on liver parameters is small, and neither smokers nor nonsmokers differ significantly from one another in terms of albumin levels, AST and ALT enzyme activity.

REFERENCES

1. Pan, E.H., Wu, J., Hu, C., & Global Burden of Disease. (2019). *Global Burden of Disease*. Washington, DC: Institute of Health Metrics and Evaluation (IHME). Accessed 17 July 2021.
2. Upadhyay, S., Rahman, M., & Johanson, G. (2023). Heated tobacco products: Insights into composition and toxicity. *Toxics*, 11, 667.
3. Li, Y., & Hecht, S.S. (2022). Carcinogenic components of tobacco and tobacco smoke: A 2022 update. *Food and Toxicology*, 165, 17–18.
4. Wipfli, H.L., & Samet, J.M. (2010). Second-hand smoke worldwide disease toll. *The Lancet*, 377, 101–102.
5. Jindal, S.K., Aggarwal, A.N., Chaudhry, K., et al. (2006). Tobacco smoking in India. *The Indian Journal of Chest Diseases and Allied Sciences*, 48, 38–40.
6. Zain, M., Arifin, M., & Mokhtar. (2023). Preventive interventions to reduce second-hand smoke exposure among non-smoking pregnant women: A systematic review. *IMJM*, 22(2), 12–19.
7. Yuan, H., Yao, J., & Martins, M. (2009). Second-hand smoke stimulates lipid accumulation in the liver by modulating AMPK and SREBP-1. *Journal of Hepatology*, 51(3), 535–547.
8. Tomasi, S., Yoon, J.I., & Besaratinia, A. (2020). Secondhand smoke induces liver steatosis through deregulation of genes involved in hepatic lipid metabolism. *International Journal of Molecular Sciences*, 21, 1296.
9. Yanbaeva, D., Dentener, M., et al. (2007). Systemic effects of smoking. *CHEST*, 131(5), 1557–1566.
10. Djordjevic, M.V., & Doran, K.A. (2009). Nicotine content and delivery across tobacco products. Springer, 192, 71–74.
11. World Health Organization. Bidi rolling is an occupational health hazard: WHO study. Available at: https://www.who.int/india/news/feature-stories/detail/bidi-rolling-is-an-occupational-health-hazard--who-study
12. Srivastava, A., & Srivastava, M. (2017). Assessment of risk factors associated with chronic obstructive pulmonary disease: A clinical study. *Journal of Advanced Medical and Dental Sciences Research*, 5(1), 140.
13. Wipfli, H.L., & Samet, J.M. (2010). Second-hand smoke worldwide disease toll. *The Lancet*, 377, 101–102.
14. Djordjevic, M.V., & Doran, K.A. (2009). Nicotine content and delivery across tobacco products. Springer, 192, 71–74.
15. Ozougwu, J.C. (2017). Physiology of the liver. *International Journal of Research in Pharmacy and Biosciences*, 4(8), 13–24.
16. Premkumar, M., & Anand, A.C. (2021). Tobacco, cigarettes and the liver: The smoking gun. *Journal of Clinical and Experimental Hepatology*, 11(6), 700–712.
17. Nicoll, D. (2007). Therapeutic drug monitoring and laboratory reference ranges. In S.J. McPhee & M.A. Papadakis (Eds.), *Current Medical Diagnosis and Treatment* (46th ed., pp. 1767–1775). McGraw-Hill.
18. Mauro, P., Renze, B., & Wouter, W. (2006). Enzymes. In C.A. Burtis, E.R. Ashwood, & D.E. Bruns (Eds.), *Tietz's Textbook of Clinical Chemistry and Molecular Diagnostics* (4th ed., pp. 604–616). Elsevier.
19. Rosalki, S.B., & McIntyre, N. (1999). Biochemical investigations in the management of liver disease. In *Oxford Textbook of Clinical Hepatology* (2nd ed., pp. 503–521). Oxford University Press.
20. Chalasani, N., Younossi, Z., Lavine, J.E., Diehl, A.M., Brunt, E.M., Cusi, K., Charlton, M., & Sanyal, A.J. (2012). The diagnosis and management of non-alcoholic fatty liver disease: Practice guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. *Hepatology*, 55, 2005–2023.

21. Younossi, Z.M., Koenig, A.B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease: Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*, 64, 73–84.
22. Basaranoglu, M., Basaranoglu, G., & Senturk, H. (2013). From fatty liver to fibrosis: A tale of "second hit". *World Journal of Gastroenterology*, 19, 1158–1165.
23. Farzanegi, P., Dana, A., Ebrahimpoor, Z., Asadi, M., & Azerbaijani, M.A. (2019). Mechanisms of beneficial effects of exercise training on non-alcoholic fatty liver disease (NAFLD): Roles of oxidative stress and inflammation. *European Journal of Sport Science*, 19, 994–1003.
24. Liaw, Y.F., Leung, N., Kao, J.H., Piratvisuth, T., Gane, E., Han, K.H., Guan, R., Lau, G.K., & Lucarini, S. (2008). Asian-Pacific consensus statement on the management of chronic hepatitis B: A 2008 update. *Hepatology International*, 2, 263–283.
25. Asrani, S.K., Larson, J.J., Yawn, B., Therneau, T.M., & Kim, W.R. (2013). Underestimation of liver-related mortality in the United States. *Gastroenterology*, 145, 375–382.
26. World Health Organization. (2020). *Projections of Mortality and Causes of Death, 2016–2060*. Geneva, Switzerland: World Health Organization.
27. Singal, A.G., & El-Serag, H.B. (2015). Hepatocellular carcinoma from epidemiology to prevention: Translating knowledge into practice. *Clinical Gastroenterology and Hepatology*, 13(12), 2140–2151.
28. Pessione, F., Ramond, M.J., Nalpas, B., et al. (2001). Cigarette smoking and hepatic lesions in patients with chronic hepatitis C. *Hepatology*, 121–125.
29. Chen, Z.M., Liu, B.Q., Boreham, J., Wu, Y.P., Chen, J.S., & Peto, R. (2003). Smoking and liver cancer in China: Case-control comparison of 36,000 liver cancer deaths versus 17,000 cirrhosis deaths. *International Journal of Cancer*, 106–112.
30. El-Zayadi, A.R. (2006). Heavy smoking and liver. *World Journal of Gastroenterology*, 12, 6098–6101.
31. Younossi, Z.M., Koenig, A.B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease: Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*, 64(1), 73–84.
32. Ampuero, J., Aller, R., Gallego-Durán, R., et al. (2020). Significant fibrosis predicts new-onset diabetes mellitus and arterial hypertension in patients with NASH. *Journal of Hepatology*, 73(1), 17–25.
33. Chiolero, A., Faeh, D., Paccaud, F., & Cornuz, J. (2008). Consequences of smoking for body weight, body fat distribution, and insulin resistance. *American Journal of Clinical Nutrition*, 87(4), 801–809.
34. Altamirano, J., & Bataller, R. (2010). Cigarette smoking and chronic liver diseases. *Gut*, 59(9), 1159–1162.
35. Ismaeel, S., & Sdek, G. (2021). Effect of cigarette smoking in some liver function tests. Department of Environmental Science and Health, College of Science, University of Salahaddin, Erbil, Kurdistan Region, Iraq.
36. Iluz-Freundlich, D., Zhang, M., Uhanova, J., & Minuk, G.Y. (2020). The relative expression of hepatocellular and cholestatic liver enzymes in adult patients with liver disease. *Annals of Hepatology*, 19(2), 204–208.
37. Alsalhen, K.S., & Abdalsalam, R.D. (2014). Effect of cigarette smoking on liver functions. *International Current Pharmaceutical Journal*, 3(7), 291–295.
38. Wannamethee, S.G., & Shaper, A.G. (1990). Cigarette smoking and serum liver enzymes: The role of alcohol and inflammation. *Journal of Clinical Epidemiology*, 47, 1258.
39. Ramamurthy, V., & Raveendran, S. (2012). Biochemical changes of cigarette smokers and non-cigarette smokers. *International Journal of Advanced Life Sciences (IJALS)*, 1, 68–69.
40. Rutledge, S.M., & Asgharpour, A. (2020). Smoking and liver disease. *Gastroenterology and Hepatology*, 16(12), 617–625.
41. Mohammed, E.T.E. (2014). Assessment of serum total cholesterol level and aspartate transaminase activity among Sudanese cigarette smokers, 14–17.
42. Alsalhen, K.S., & Abdalsalam, R.D. (2014). Effect of cigarette smoking on liver functions: A comparative study conducted among cigarette smokers and non-smokers.

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