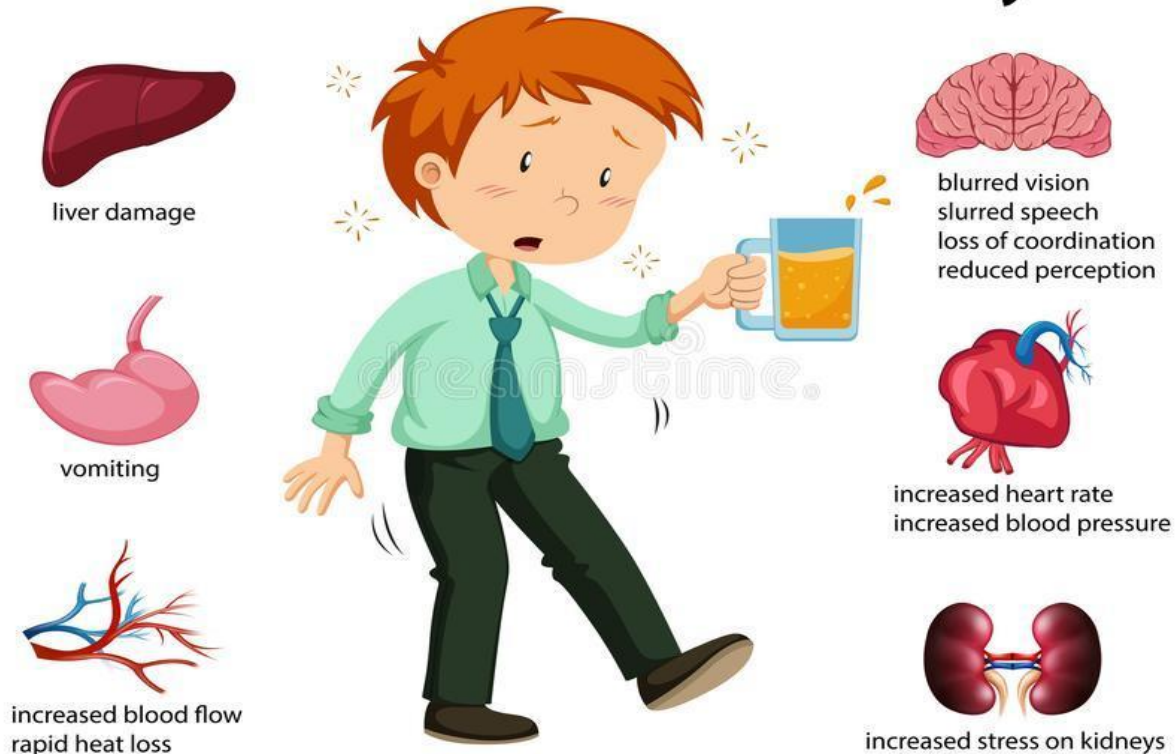


EFFECTS OF ALCOHOL ON THE BODY IN LIVER CIRRHOSIS

Effects of Alcohol on the Body



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CERTIFICATE

**This is to certify that Ms. Samruddhi Kiran Shinde has
successfully completed the seminar work on
EFFECTS OF ALCOHOL ON THE BODY IN LIVER CIRRHOSIS for
the academic year 2020-2021.**

College stamp

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Date: 10 November,2021

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I am extremely grateful to our principal , Dr. Prof. Mala Pandurang and our course coordinator, Mrs. Vinaya Vaishampayan for giving me all the support and facilities to carry out the seminar work. My heartfelt gratitude to our subject teacher, Ms. Madhura Ghaisas for her valuable guidance and suggestions in the preparation of entire seminar work.

I will be failing in my duty if I do not acknowledge the authors and the original researchers of the research articles referred for completion of the seminar work.

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INTRODUCTION:-

Alcoholic liver diseases comprises of a very broad spectrum of different liver diseases, which ranges from alcoholic hepatitis to the most common one which is liver cirrhosis. The common complication of the alcoholic liver disease is protein energy malnutrition (PEM) which causes sarcopenia. In this condition body loses its skeletal muscle mass and strength. Ethanol alters the signaling pathway in the body and it leads to decrease in muscle protein synthesis in type 2 muscle fiber. The European working group on sarcopenia has classified that it has 2 different categories 1 st is of aging related and the 2 nd one is on chronic disease like chronic liver disease etc.[1]

One of the most prevalent liver disorders is an alcoholic liver disease (ALD). It is a liver condition caused by long-term excessive drinking, which causes widespread liver cell necrosis. It generally starts off as fatty liver and can develop to alcoholic hepatitis, liver fibrosis, and cirrhosis, putting people, health and lives at jeopardy. In 2017, around 129,000 people died from alcoholic liver cancer throughout the world. In China, there was an unflatteringly high prevalence of

ALD (4.5 percent), affecting at least 62 million people. Long-term abstinence from alcohol can restore minor liver damage, but when the illness develops to the final stage of ALD, liver transplantation is the only viable therapy. However, because liver transplantation has certain drawbacks, such as the risk of donor immunological rejection and the high expense of the procedure, ALD has emerged as one of the most serious health issues.

Alcoholic liver cirrhosis (ALC) is a kind of ALD that progresses to the level of liver cirrhosis if not treated promptly and effectively. With the advancement of molecular biology and genetics, it is now widely assumed that liver cirrhosis and cancer are caused by a combination of variables. Environmental influences, genetic factors, and lifestyle choices all have a role. Consumption of alcoholic beverages is a substantial risk factor for ALC.[2]

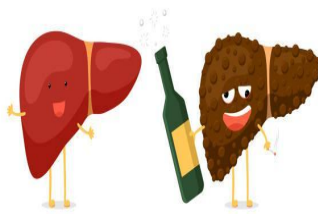
Cirrhosis is one of the leading cause of the world because the problem of alcohol consumption worldwide. Many studies took place on the consumption of alcohol with the meals and on the regularity of the same. As drinking with the meals and frequency of consumptions are correlated factor. The UK Million study states that in every woman born between 1935 and 1950 were the patients of cirrhosis because of their daily consumption incidence. The main aim of this study was to check whether the consumption of alcohol with the meals and the frequency of any drink had any risk of liver cirrhosis onset.[3]

80% of patients with liver cirrhosis were detected with cognitive impairment, and were associated with negative health outcomes but is under diagnosed in the clinical setting due to the lack of practical testing method. This single-center prospective observational study aimed to test the feasibility and prognostic utility of in-clinic cognitive assessment of patients with liver cirrhosis using the NIH Toolbox cognition battery (NIHTB).[4]

Alcoholic liver diseases are not only prevalent but also one of the common noninfectious liver disease worldwide. It is spread in very large spectrum which includes alcoholic steatosis. These diseases can only turn into cirrhosis when after the abstinence the patient continuous to drink, chances of getting cirrhotic in patients is 20%.As it is also the leading cause of mortality. When alcohol is indigested it is metabolized by the liver with a by-product such as reactive oxygen species (ROS) and acetaldehyde, which directly leads to liver injury.[5]

Malnutrition is common in patients with ALD, which is caused by the replacement of energy from food to alcohol (7.1 kcal per gramme of alcohol) and gastrointestinal disturbances of nutrient utilization. Alcohol metabolism causes many problems including in reduction of nicotinamide adenine dinucleotide (NADH / NAD⁺) ratio and by altering the redox state which interrupts the TCA cycle and suppresses lipid, oxidation in the body; which results in energy supply disruption and alteration of the intermediary metabolites.[5]

This particular report gives the information of the demerits and also the merits of alcohol and cirrhosis relation in every scientific part with logical reasoning and point of views. It contains topics such as mortality, and how obesity can alter the effects of alcohol, brain dysfunctioning etc. it includes some experimental based papers to provide more dynamic study as they had used different methods for detailed results. Nutritional therapy and management of alcohol in the body is also stated in this report.



METHODOLOGY

There were numerous researches done for the effects of alcohol on the body in liver cirrhosis. As we all know that too much of ethanol in the body has its adverse effects so to understand and get to its root this report is done. Here we can see the correlation between alcohol and liver cirrhosis. I choose this specific topic to find out the various reasons and causes of the same.

Many experimental based research papers are used to get an exact overview of the problem . Papers from past 10 years that is from 2009-2021 have been the part of this report. Key words to find every paper was “liver cirrhosis”, “Alcohol effects”, “Alcoholic cirrhosis” , “Liver diseases”, . The sites which helped me to find the precise set of papers were “Pubmed”, “Wiley online library”, “Research gate”. The same kind of experiments or some invalid treatments were excluded, from the study and each and every patients were asked for the consent before the experiments such as Liver transplantation’ or Stem cells operations. The information collected is all pure and true on scientific terms. There was no misuse or copying or any mischief was done with the original publications. Most of the experiments took place in United States and Japan from here many experimental based research studies were done and deep core information was gained and they are also added in this report with all due respect to the authors.

As learning about an organ such as liver is fascinating but it becomes more interesting to get a knowledge about it in its adverse condition such as liver cirrhosis. While searching and reading those papers I came across to many of the different crisis faced due to alcohol on brain, and how it eventually causes hepatocellular carcinoma. For some people it gets easy to live after operations and transplantation but for rest of the patients it was another battle to fight against their own body. The results we collected here were well justified and in diagramatic formats so it became easier to understand the statistics about the outputs of the experiments. Overall the research articles which are included in this report contains a very descriptive point of views of the effects because some are given by professionals itself and some are from the specialists, Doctors and done with proper understanding of the topic and detailing of work.

REVIEW OF LITERATURE

Alcoholism is defined as the consumption of alcoholic drinks in such a way that the drinker is continually injured or causes harm to others. A majority of physicians, although not all, see alcoholism as an addiction and a disease. The injury might be bodily or emotional, as well as social, legal, or financial.

Alcohol use disorders have been consistently associated with co-occurrent depressive disorders. Further more in-depth studies focused mainly on heavy use and examined alcohol use indicators independently of each other (e.g. quantity, frequency) It may be that specific patterns of alcohol use differ qualitatively in their depressive symptomatology.

Some major effects on the body are as follows:-

Brain:

Alcohol disrupts the brain's communication connections, altering the way it appears and functions. These disturbances can alter mood and behaviour, as well as making it difficult to think clearly and move in a coordinated manner.

Heart:

Drinking a lot over a long time or too much on regular occasion can damage the heart, causing problems including:

- Cardiomyopathy – Stretching and drooping of heart muscle
- Arrhythmias – Irregular heart beat
- Stroke
- High blood pressure

Liver:

Heavy drinking damages the liver and can cause a number of issues and inflammations, including: • Steatosis (fatty liver)

- Hepatitis caused by alcohol
- Fibrosis and Cirrhosis are two diseases that affect the liver.

"There is a clear scientific consensus that alcohol drinking can cause numerous forms of cancer," according to the National Cancer Institute. Consumption of alcoholic drinks is listed as a known human carcinogen by the US Department of Health and Human Services' National Toxicology Program. According to 2009 data, cancer fatalities in the United States accounted for 3.5 percent of all deaths (about 19,500 deaths were alcohol related).

Oral cavity, throat, and laryngeal malignancies; esophageal cancer, especially esophageal squamous cell carcinoma. Furthermore, it has been shown that those who inherit a defect in an enzyme that metabolises alcohol have a much higher risk of esophageal squamous cell carcinoma if they consume alcohol.

- Liver cancer.
- Breast cancer: Studies have consistently found an increased risk of breast cancer in women with increasing alcohol intake. Those who drink roughly one drink per day had a 5 to 9% increased risk of breast cancer than women who do not drink at all.
- Colorectal cancer.

Immune System: Drinking too much can weaken the immune system, making one's body a much easier target for disease. Chronic drinkers are more likely than non-drinkers to get illnesses such as pneumonia and TB. Even up to 24

hours after being intoxicated, drinking a lot on a single occasion reduces your body's ability to fight diseases.

- **STRAIGHT TO THE HEAD-** Thirty seconds after the first sip, alcohol rushes into the brain. It slows down the chemicals and pathways that our brain cells use to send messages. That alters the mood, slows down the reflexes, and throws off one's balance. Someone furthermore might can't think straight, which one will not recall later, because it becomes a struggle to store things in memory.

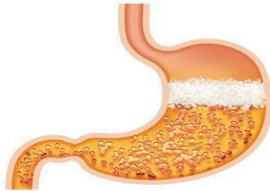


- **BRAIN SHRINKS-** If one drink heavily for a protracted time, booze can affect how the brain looks and works. Its cells start to alter and even get smaller. An excessive amount of alcohol can actually shrink the brain. And that'll have big effects on the ability to think, learn, and remember things. It also can make it harder to stay a gentle vital sign and control one's motor movements.



- **STOMACH ACIDITY-** Booze irritates the liner of the stomach and makes the digestive juices flow. When enough acid and alcohol build up, people get

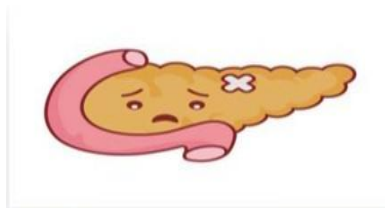
nauseated and eventually will bestow. Years of heavy drinking can cause painful sores called ulcers in the stomach. And high levels of stomach juices mean one won't feel hungry. That's one reason long-term drinkers often don't get all the nutrients they need from the diet. Alcohol alters the metabolism in the body.



- **DIARRHOEA AND HEARTBURN-** Alcohol relaxes the muscle that keeps acid out of the esophagus, the tube that connects the mouth and stomach. It can also cause one's small intestine and colon to get irritated, too. Hard drinking can lead to diarrhea, which can turn into a long-term problem later on.



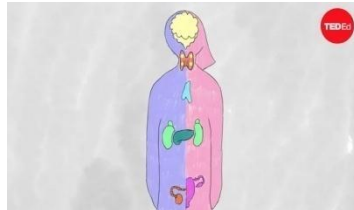
- **PANCREAS GET DAMAGED-** Alcohol can cause damage to the pancreas, which means one's body won't be able to make the insulin which body needs to help break down food. It also makes one more likely to get pancreatic cancer, which is a serious type of cancer.



- **CHANGES THE BODY TEMPERATURE-** Alcohol can make people feel more flushed and toasty, but it can also the your temperature to drop if someone is drinking too much. Stress hormones release stress hormones that narrow blood vessels, so it doubles the work of heart and it has to pump harder to push blood through the walls.



- **HORMONE HAVOC-** Alcohol can throw the hormones out of whack - in women, that can knock the periods off cycle and cause problems getting pregnant. In men, it can mean trouble getting an erection, a lower sperm count and shrinkage of testicles.



- **HEARING LOSS-** Alcohol affects the hearing too, but no one's sure exactly how, and more depth researches are taking place to ensure the same. It could be that it messes with the part of one's brain that processes sound. Or it might damage the nerves and hairs in drinkers inner ear that help to hear. Long-term drinkers often have hearing loss.



When a person consumes large amounts of alcohol over a long period of time, the body begins to replace good liver tissue with scar tissue. This illness is known as alcoholic liver cirrhosis by doctors. Your liver will cease working correctly as the illness continues and more of your good liver tissue is replaced with scar tissue.

Loss of muscular tone is one of the symptoms of alcoholic liver cirrhosis (atrophy), Easily bruised, Appetite loss and weight loss The skin and eyes get a yellowish colour (jaundice), Edema (swelling and fluid accumulation) in the legs and

abdomen (ascites), Bleeding in your mouth (mouth bleeds) or vomiting blood are both signs of a serious problem. Erythema (patchy red skin on the palms of your hands), Confusion, memory loss, poor concentration, and mental fog.[15]

The risk factors of ALC (alcoholic liver cirrhosis) are:-

- **Mortality-** The prevailing clinical impacts of malnutrition and sarcopenia are well established in the cirrhotic liver disease following with mortality and morbidity. This is due to the patients with cirrhosis shows very low level of physical activity and also has altered exercise physiology with impairment in aerobic capacity. Aerobic exercise improves cardiorespiratory fitness, which also turns down the risk of all cause mortality in the general population. Carrying out physical activities has shown to improve the muscle strength and functional ability in frail older adults and even it increase functional capacity in chronic kidney disease. So in a same way the concern regarding the safety of exercise in patients with cirrhosis at an early age stage showed an impending progress in the past; so the main core of this review article is to evaluate the alteration in physical capacity which was observe in cirrhosis and potential benefits of exercise intervention.[6]

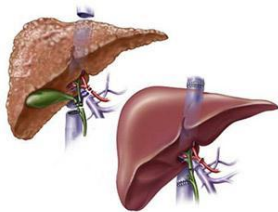
Major cause of mortality in the developed world is represented by colorectal cancer (CRC). In many cases, the cancer develops along side with adenoma-carcinoma sequence. Recently most of the studies were focusing only on the liver disease in its last stages (cirrhosis) as a risk factor for colon.[7]

In the year 2010, cirrhosis reckoned for more than 493,300 deaths (156,900 female and 336,400 male deaths) and for more than 14.5 million DALYs (disability-adjusted life year) (around 4.1 million DALYs (millions for women and 10.4 million for males). In addition, in total 82,000 deaths (around 14,000 female and 66,000 male deaths) and 2.1 million DALYs were due to alcohol-induced liver cancer only.[8]



- **Transplantation:-** The relation between or the causes of the prevalence of RD,PVT & PVT/RD in liver transplantation (LT) was very difficult or poorly understood by the researchers. [15]

The specific retrospective study encompassed the consecutive liver diseases patients (LDPs) of all the etiologies evaluated for liver transplantation from a signal institution and a control group of liver- healthy as part of the German CRC screening programme, patients (LHPs) undergo colonoscopy. In multivariable and univariable settings like adjusting for age, gender, BMI and number of colonoscopies, the rates of adenomas, CRC, polyps were analyzed. Difference between LDPs and LHPs and cirrhotic and non-cirrhotic hepatopathy were also assessed.

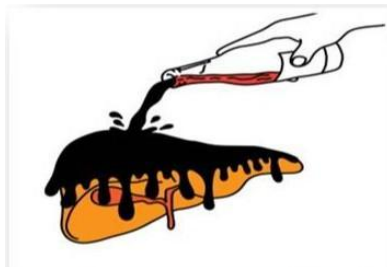


- **Obesity:-** This particular study was seeking to inform a potential treatment gap by describing the safety and efficacy of pharmacologic weight loss in patients with advanced liver disease. Obesity has very advanced risk factor on the patients having liver fibrosis, and however it also has surgical risks of bariatric surgery in cirrhosis treatment recommendations are currently limited to lifestyle interventions. The benefits of weight loss in patients with obesity were well established and extended to the patients with cirrhosis and other liver morbidities having additional unique consideration. hence, treatment of obesity in this patient population is limited by increased operative risks of

bariatric surgery, and the complexity of nutrition management and weight loss in patients having sarcopenia and elevated BMI.[9]



- **Risk of Alcohol relapse:-** The HRAR score had no relationship with the incidence of recidivism or harmful relapse. The significant risk factor for recidivism and harmful relapse were that in recidivism that the positive history of treatment for psychological diseases, absence of a marital history and noncompliance with clinic visits after transplantation and smoking after transplantation and on the other hand in harmful relapse the factors are no marital history, noncompliance with clinic visits after LT , living alone before LT. Abstinence for six months before to LT had no effect. Abstinence for 24 months or more reduced the risk of detrimental recurrence by 3.3 percent, although the difference was not statistically significant. Alcohol relapse following transplantation is still a serious problem, and transplanted patients' drinking habits should be monitored on a regular basis. [8,10]



- **Risk of Sarcopenia with obesity:-** Patients with Alcoholic liver cirrhosis (ALC) were the younger ones and had a lower risk of sarcopenia despite of having advanced stage liver impaired disease. And among the 181 patients there were 64 patients who were diagnosed with ALC. The danger prevalence of sarcopenia was seen increasingly alongside of advanced age in patients with ALC. According to the multivariate analysis it was examined that in the older

age (having no disease or stage/liver function reserve and alcohol/consumption) was an independent factor and strongly associated with sarcopenia in patients having ALC. [1]

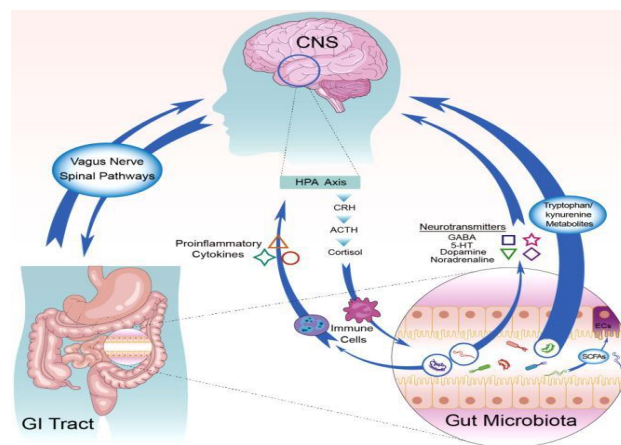
The lethal combination of sarcopenia and obesity in cirrhosis is associated with higher rate of morbidity and mortality than either condition. The reason of its complexity is the pathogenesis of sarcopenic obesity in cirrhosis is multifactorial and also has multiple disruptions in the muscle and liver-adipose tissue axis. Management of sarcopenic obesity was focusing on how to increase muscle mass and strength +- carefully and they monitored the weight reduction using nutrition as a key, exercise and pharmacologic therapies too.[11]



- **Malnourishment:** Many alcoholic drinkers are malnourished, either because they ingest too little of essential nutrients for example carbohydrates, proteins, and vitamins or because alcohol and its metabolism restricts the preventing those nutrients from being adequately absorbed, digested, and used by the body. As a result, alcoholics usually have protein and vitamin deficiencies, notably vitamin A, which can lead to liver damage and other significant alcohol-related illnesses. [12]

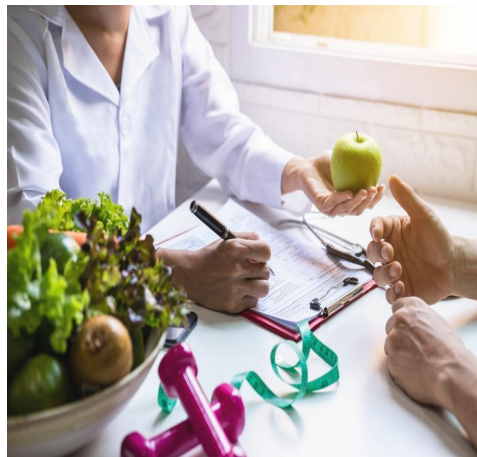


- Intestinal and Brain dysfunction and malabsorption :-** The stomach and, to a lesser extent, the duodenum and jejunum absorb alcohol by diffusion. Chronic alcohol intake can lead to intestinal fibrosis and an overgrowth of aerobic and anaerobic microbes, which can lead to functional and morphological changes in the small bowel. [18] Cirrhotic patients [have] dysfunctional livers that cannot remove poisons from the blood stream, and these poisons are able to move into the brain and disrupt normal function," says Dr Richard Branson, chairman of the European Commission's Committee for Medicinal and Biologics in Europe. In cirrhotic individuals, 482 genes exhibited elevated expression and 643 genes showed decreased expression. "These genes are critical in controlling cell death and how particular cells in the brain communicate with one another," explains Dr. Mayfield. The study suggests widespread effects in many cellular pathways related to cirrhosis in the alcoholic group.[14]



Some management tactics to stop the adverse alcohol effects in the body

- **Nutritional Therapy**:- Malnutrition in cirrhosis is significantly common because there is low energy into it due to absorption and altered energy metabolism researches indicate that energy intake should be increased but also some exercise or else the weight loss would cause the problem again so there should be an adequate protein intake; 1.2-1.5kg protein per day should be targeted for the patients with cirrhosis, and in older and non-cirrhotic patients intake 25-30g per meals Smaller meals contain 50g of complete carbohydrates and a protein source, which improves nitrogen balance, lowers skeletal muscle proteolysis, and boosts muscle mass and quality of life. When attempting to reconcile this more frequent eating with weight loss, dietitian guidance is crucial. [11]



- **Prevention**:- You should restrict your alcohol consumption to avoid alcohol poisoning. Limit yourself to one alcoholic beverage each hour. If you or a companion is drinking, keep track of how much you consume and how quickly you consume it. Moderation is always essential. Drinking games can

put participants under pressure to binge drink. Never consume alcoholic beverages when taking prescription drugs.



CONCLUSION

After classification by risk factor, there was no difference in patient outcomes between alcoholic liver cirrhosis and non-alcoholic liver disease disease

recipients. The risk of PVT, RD, and concurrent PVT/RD was rising in liver transplant recipients, particularly those with NAFLD. Regardless of the rationale for LT, recipients with PVT, RD, or concurrent PVT/RD had worse short- and long-term results than patients without those risk factors.

During this investigation, the ALDH2 A allele (G/A + A/A genotypes) was found to increase the risk of developing alcoholic liver cirrhosis in Hakka people in southern China. More study with larger samples is needed to corroborate their findings. In order for the findings to raise the current data and give important information for future study, a lot of review papers show that by diminishing physical capability, the chances of cirrhosis were clearly associated to their negative results. It also indicates that specific types of exercise should be advised to people with cirrhosis only after sufficient safety assessment.

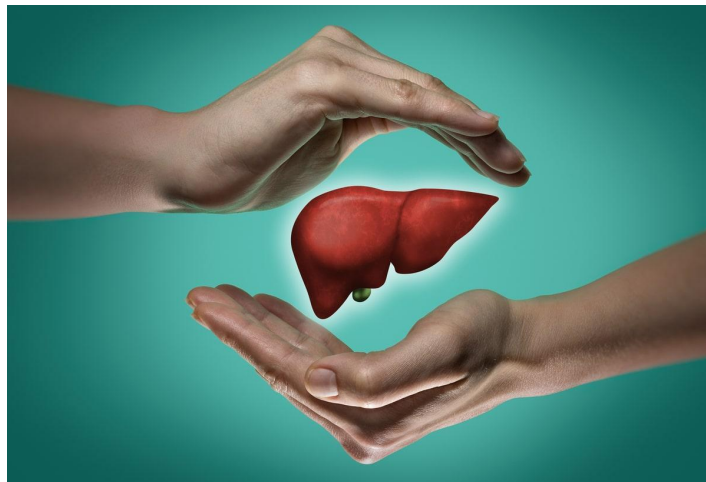
Acute malnutrition is highly prevalent in cirrhosis; it decreases overall calorie intake and, when paired with the disruption of a complicated metabolic system, resulting in significant loss of musculus mass and function. As a result, individuals with cirrhosis have the problem of low respiratory quotients, as evaluated by indirect calorimetry. As a result, it decreases hepatic glycogen storage drive, resulting in improper use of body fat and protein stores for gluconeogenesis in a capacity that mimics hunger. Cirrhotic individuals have an extra increase in BMR. Cirrhotic patients also have a higher baseline flow rate.

A specific examination drew our attention to the scarcity of cirrhosis-specific data in this area. Because they provide us with a framework for the diagnosis and therapy of sarcopenic obesity, we may extrapolate from the broader literature as well as the existing cirrhosis-specific findings. Clinical studies were required to obtain data to support management recommendations for diet, exercise, and pharmaceutical interventions.

Before evaluating alcoholic patients, most liver programmes demand a 6-month period of sobriety. The aim behind this technique was to allow illness regression in patients who have recently abstained from alcohol and to confirm adequate alcohol counselling. There is no agreement on what constitutes an alcohol relapse. Some experts consider any drinking to be a relapse, whilst others

consider heavy drinking to be a relapse. During the pre- and post-liver transplant period, psychosocial efforts should be directed at avoiding and addressing modifiable risk factors such as obesity and cigarette smoking. A recent proof-of-concept research discovered that quitting smoking following a liver transplant has a protective impact on the development of neoplasia.

Many alcoholics are malnourished to varying degrees, both primary and secondary. Primary malnutrition occurs when alcohol substitutes other nutrients in the diet, resulting in total nutritional deficiency. Excessive alcohol drinking may impair these alcoholics' capacity to absorb and utilise the nutrients they do ingest. Drinkers who consume more than 30% of their calories from alcohol may consume less protein, fat, and vitamins A, C, and B. Consumption of fat-soluble vitamins, vitamin C, and thiamine (vitamin B1) may fall short of the RDAs.



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ABSTRACT

1. Clinical Characteristics of sarcopenia in patients with alcoholic liver cirrhosis-Sarcopenia frequently develops in patient with liver cirrhosis (LC). Ethanol reduces muscle protein synthesis and accelerates proteolysis. However, the relationship between heavy alcohol consumption and sarcopenia remains controversial. This study aimed to investigate the characteristics and prevalence of sarcopenia among patients with alcoholic LC (ALC) in real-world clinical settings.
2. Association between aldehyde dehydrogenase 2 gene rs671 G>A polymorphism and alcoholic liver cirrhosis in southern Chinese Hakka population -Alcoholic liver cirrhosis (ALC) endangering people's health. The association between aldehyde dehydrogenase 2 (ALDH2) gene polymorphisms and ALC is not clear. To analyze the relationship between *ALDH2* and ALC among Hakka population in southern China.
3. Alcohol drinking patterns and liver cirrhosis risk: analysis of the prospective UK Million Women Study -Alcohol is a known cause of cirrhosis, but it is unclear if the associated risk varies by whether alcohol is drunk with meals, or by the frequency or type of alcohol consumed. Here we aim to investigate the associations between alcohol consumption with meals, daily frequency of consumption, and liver cirrhosis.
4. Impaired Cognition Predicts the risk of hospitalization and death in cirrhosis -Cognitive impairment, detected in up to 80% of patients with liver cirrhosis, is associated with negative health outcomes but is underdiagnosed in the clinical setting due to the lack of practical testing method. This single-center prospective observational study aimed to test the feasibility and prognostic utility of in-clinic cognitive assessment of patients with liver cirrhosis using the NIH Toolbox cognition battery (NIHTB).

5. Metabolomic Analysis uncovers Energy supply disturbance as an underlying Mechanism of the development of alcohol Associated liver cirrhosis
-Alcohol-associated liver disease (ALD) is caused by alcohol metabolism's effects on the liver. The underlying mechanisms from a metabolic view in the development of alcohol-associated liver cirrhosis (ALC) are still elusive. We performed an untargeted serum metabolomic analysis in 14 controls, 16 patients with ALD without cirrhosis (NC), 27 patients with compensated cirrhosis, and 79 patients with decompensated ALC. We identified two metabolic fingerprints associated with ALC development (38 metabolites) and those associated with hepatic decompensation (64 metabolites) in ALC. The cirrhosis-associated fingerprint (eigenmetabolite) showed a better capability to differentiate ALC from NC than the aspartate aminotransferase-to-platelet ratio index score. The eigenmetabolite associated with hepatic decompensation showed an increasing trend during the disease progression and was positively correlated with the Model for End-Stage Liver Disease score. These metabolic fingerprints belong to the metabolites in lipid metabolism, amino acid pathway, and intermediary metabolites in the tricarboxylic acid cycle. *Conclusion:* The metabolomic fingerprints suggest the disturbance of the metabolites associated with cellular energy supply as an underlying mechanism in the development and progression of alcoholic cirrhosis.
6. Exercise physiology in Cirrhosis and the potential benefits of exercise intervention -Reduction in muscle mass is a highly prevalent phenomenon in cirrhosis and is now well-documented to be associated with significant morbidity and mortality. Research into muscle loss in cirrhosis remains limited by an ongoing poor understanding of its relationship with muscle function, physical activity, and aerobic capacity. Alterations in exercise physiology have been documented in studies of individuals with cirrhosis that provide important information on physical function that is not captured by simple quantification of muscle mass. Despite expert consensus recommending regular exercise in end-stage liver disease to maintain muscle mass and function, there is little evidence guiding clinicians as to which form of exercise or delivery mechanism is most effective. It also remains unproven whether any specific intervention can alter clinically relevant outcomes. This review article

summarizes the available literature regarding the changes in exercise physiology observed in cirrhosis, the associated impact on physical capacity, and the results of existing trials that examine the potential benefits of exercise delivery in patients with cirrhosis, particularly pertaining to their impact on exercise physiology.

7. Chronic Liver disease promotes lesions of the colorectal adenoma carcinoma sequence, independent of liver cirrhosis -Research increasingly focuses on identifying individuals at greater risk of colorectal cancer (CRC) to enhance colonoscopy screening efficacy. The objective of this article is to determine associations between chronic liver disease and lesions along the colorectal adenoma-carcinoma sequence.
8. Trends in the management and burden of alcoholic liver disease -Alcoholic liver disease (ALD) is the most prevalent cause of advanced liver disease in Europe and is the leading cause of death among adults with excessive alcohol consumption. There is a dose-response relationship between the amount of alcohol consumed and the risk of ALD. The relative risk of cirrhosis increases in subjects who consume more than 25 g/day. The burden of alcohol-attributable liver cirrhosis and liver cancer is high and is entirely preventable. Health agencies should develop population-based policies to reduce the prevalence of harmful and/or hazardous alcohol consumption and foster research in this field to provide new diagnostic and therapeutic tools. Disease progression of patients with ALD is heavily influenced by both genetic and environmental factors.
9. Safety and efficacy of pharmacologic weight loss in patients with cirrhosis -Obesity poses unique risks in patients with advanced liver fibrosis; however, given surgical risks of bariatric surgery in cirrhosis treatment recommendations are currently limited to lifestyle interventions. This study seeks to inform a potential treatment gap by describing the safety and efficacy of pharmacologic weight loss in patients with advanced liver disease.
10. Risk factors for alcohol relapse after liver transplantation for alcoholic cirrhosis in Japan. First published -Alcoholic liver cirrhosis (ALC) is an established indication for liver transplantation (LT). Most LT procedures in Japan are living donor liver transplantation (LDLT) because of an extreme shortage of deceased

donors. Social circumstances enabling LDLT could be favorable for preventing relapse. The aims of this retrospective study were to analyze the outcomes of LDLT for ALC and to evaluate risk factors for relapse in this cohort. One hundred ninety-five subjects underwent LT [LDLT (n = 187), deceased donor LT (n = 5), or domino LT (n = 3)] for ALC in Japan from November 1997 to December 2011. Risk factors for alcohol relapse and the impact of relapse on outcomes were analyzed for 140 patients after the exclusion of 26 patients who died in the hospital and 29 patients without information about alcohol relapse.

11. Sarcopenia Obesity in cirrhosis-The confluence of 2 prognostic titan – Sarcopenia and obesity are 2 major health conditions with a growing prevalence in cirrhosis. The concordance of these 2 conditions, sarcopenic obesity, is associated with higher rates of mortality and impact on the metabolic profile and physical function than either condition alone. To date, there is little consensus surrounding the diagnostic criteria for sarcopenia, obesity or as a result, sarcopenic obesity in patients with cirrhosis, cross-sectional imaging, although the most accurate diagnostic technique, has practical limitations for routine use in clinical practice. Management strategies are focused on increasing muscle mass and strength.
12. Relationship between Nutrition Alcohol use and liver disease-Many alcoholics are malnourished, either because they ingest too little of essential nutrients (e.g., carbohydrates, proteins, and vitamins) or because alcohol and its metabolism prevent the body from properly absorbing, digesting, and using those nutrients. As a result, alcoholics frequently experience deficiencies in proteins and vitamins, particularly vitamin A, which may contribute to liver disease and other serious alcohol-related disorders. Furthermore, alcohol breakdown in the liver, both by the enzyme alcohol dehydrogenase and by an enzyme system called the microsomal ethanol-oxidizing system (MEOS), generates toxic products such as acetaldehyde and highly reactive, and potentially damaging, oxygen-containing molecules. These products can interfere with the normal metabolism of other nutrients, particularly lipids, and contribute to liver cell damage. Nutritional approaches can help prevent or ameliorate alcoholic liver disease. For example, a complete balanced diet can

compensate for general malnutrition. Administration of antioxidants (e.g., precursors of the endogenous antioxidant glutathione) can help the body eliminate reactive oxygen molecules and other reactive molecules generated from abnormal lipid breakdown. New agents currently are being studied as promising nutritional supplements for alcoholics with liver disease.

13. Nutrition in alcohol related liver disease-Physiopathology and management
-Malnutrition encompassing both macro- and micro-nutrient deficiency, remains one of the most frequent complications of alcohol-related liver disease (ArLD). Protein-energy malnutrition can cause significant complications including sarcopenia, frailty and immunodepression in cirrhotic patients. Malnutrition reduces patient's survival and negatively affects the quality of life of individuals with ArLD. Moreover, nutritional deficit increases the likelihood of hepatic decompensation in cirrhosis. Prompt recognition of at-risk individuals, early diagnosis and treatment of malnutrition remains a key component of ArLD management. In this review, we describe the pathophysiology of malnutrition in ArLD, review the screening tools available for nutritional assessment and discuss nutritional management strategies relevant to the different stages of ArLD, ranging from acute alcoholic hepatitis through to decompensated end stage liver disease.
14. Brain functioning in liver cirrhosis -Sustained exposure to alcohol can cause scarring and dysfunction of the liver, referred to as cirrhosis. Heavy alcohol use can also cause brain damage. An examination of gene expression in the frontal cortex has found that brain function is even more impaired in cirrhotic than non-cirrhotic alcoholics.
15. The prevalence of portal vein thrombosis (PVT), renal dysfunction (RD), and simultaneous PVT/RD in liver transplantation (LT) is poorly understood. We analyzed the prevalence of PVT, RD, simultaneous PVT/RD, and the outcomes of adult recipients of LT for nonalcoholic fatty liver disease (NAFLD) and alcoholic liver disease (ALD) between 2006 and 2016 in the United States. We found that the prevalence of PVT (7.2% → 11.3%), RD (33.8% → 39.2%), and simultaneous PVT/RD (2.4% → 4.5%) has increased significantly over the study period (all *P*-values <0.05). NAFLD patients had a higher proportion of PVT (14.8% vs. 9.2%), RD (45.0% vs. 42.1%), and simultaneous PVT/RD (6.5% vs.

3.9%; all P -values <0.05). 90-day mortality was 3.8%, 6.3%, 6.8%, and 9.8% for PVT(-)/RD(-), PVT(-)/RD(+), PVT(+)/RD(-), and PVT(+)/RD(+) recipients, respectively ($P < 0.01$). 5-year survival was 82.1%, 75.5%, 74.8%, and 71.1% for PVT(-)/RD(-), PVT(-)/RD(+), PVT(+)/RD(-), and PVT(+)/RD(+) recipients, respectively ($P < 0.05$).

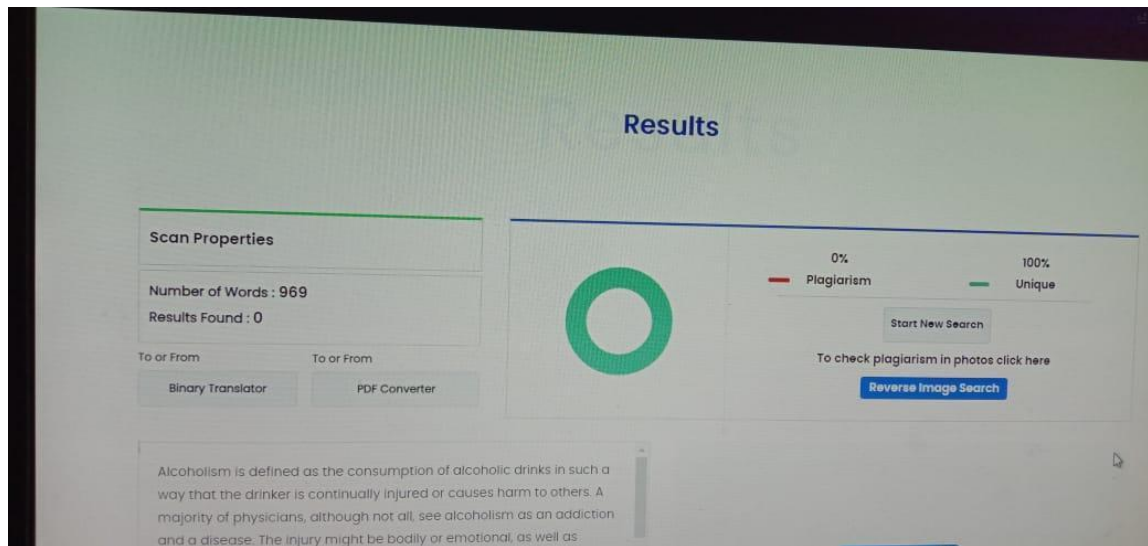
16. Mesenchymal stem cell therapy for advanced liver cirrhosis -Mesenchymal stem cell (MSC) transplant may offer an alternative to liver transplantation in patients with end-stage liver disease. However, its efficacy remains uncertain. MSC was performed on a 50-year-old male with decompensated (Child-Turcotte-Pugh grade C) alcoholic liver cirrhosis due to an absence of donors for adult-deceased and living-related liver transplantation. Autologous bone marrow-derived MSCs were harvested from the patient and cultured using standard protocols. The MSCs were subsequently re-administrated into the liver via hepatic intra-arterial infusion on two separate occasions. After infusion, there was an improvement in biochemical parameters (serum total bilirubin, serum albumin), and a reduction of diuretic use for ascites for up to 8 weeks. However, all biochemical and clinical parameters deteriorated on long-term follow-up without any further infusions. The patient eventually succumbed to his disease. MSC transplantation may have a clinical benefit on adult patients with end-stage liver cirrhosis, but this appears to be transitory.
17. Liver Cirrhosis contributes to the disorder of gut microbiota in patients with hepatocellular carcinoma-Gut microbiota (GM) of patients with liver cancer is disordered, and syet no study reported the GM distribution of liver cirrhosis-induced HCC (LC-HCC) and nonliver cirrhosis-induced HCC (NLC-HCC). In this study, we aimed to characterize gut dysbiosis of LC-HCC and NLC-HCC to elucidate the role of GM in the pathogenesis of HCC.
18. Guidelines on nutritional management in Japanese patients with liver cirrhosis from the prospective of preventing hepatocellular carcinoma-The Japanese Nutritional Study Group for Liver Cirrhosis (JNUS) was assembled in 2008 with the support of a Health Labor Sciences Research Grant from the Ministry of Health, Labor and Welfare of Japan. The goal of the study group was to propose new nutritional guidelines for Japanese patients with liver cirrhosis (LC), with the aim of preventing hepatocellular carcinoma. Methods: Between

2008 and 2010, the member investigators of JNUS conducted various clinical and experimental studies on nutrition on LC. These included anthropometric studies, a questionnaire study on daily nutrient intake, clinical trials, experimental studies using animal models, re-evaluation of previous publications and patient education. Over this 3-year period, the group members regularly discussed the nutritional issues related to LC, and a proposal was finally produced.

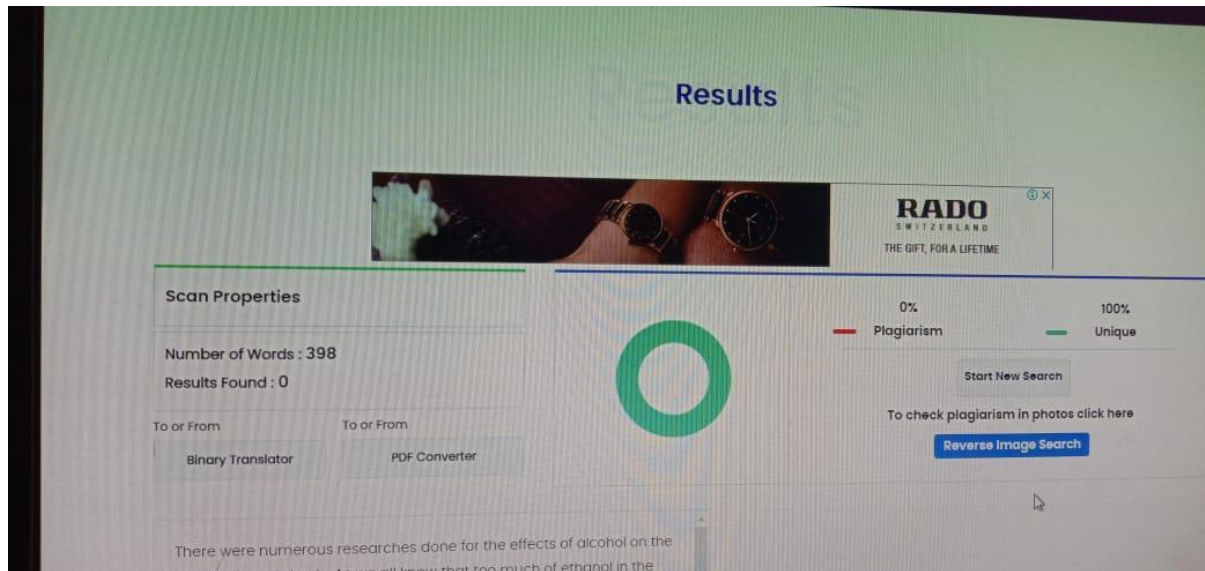
19. Drinking habits as cofactors of risk for alcohol induced liver damage- The Dionysos Study is a cohort study of the prevalence of chronic liver disease in the general population of two northern Italian communities. It included 6917 subjects, aged 12-65 (69% of the total population). The aim of this part of the study was to examine the relationship of daily alcohol intake, type of alcoholic beverage consumed, and drinking patterns to the presence of alcohol induced liver damage in an open population. Patients and methods: 6534 subjects, free of virus related chronic liver disease and participating in the first cross-sectional part of the study, were fully examined. Each subject underwent: (a) medical history and physical examination, (b) evaluation of alcohol intake using an illustrated dietary questionnaire, and (c) routine blood tests. More invasive diagnostic procedures were performed when indicated.
20. Overweight and obesity worsens the liver damaging effects of alcohol- Led by the University of Sydney's Charles Perkins Centre, the study looked at medical data from nearly half a million people and found having overweight or obesity considerably amplified the harmful effects of alcohol on liver disease and mortality.

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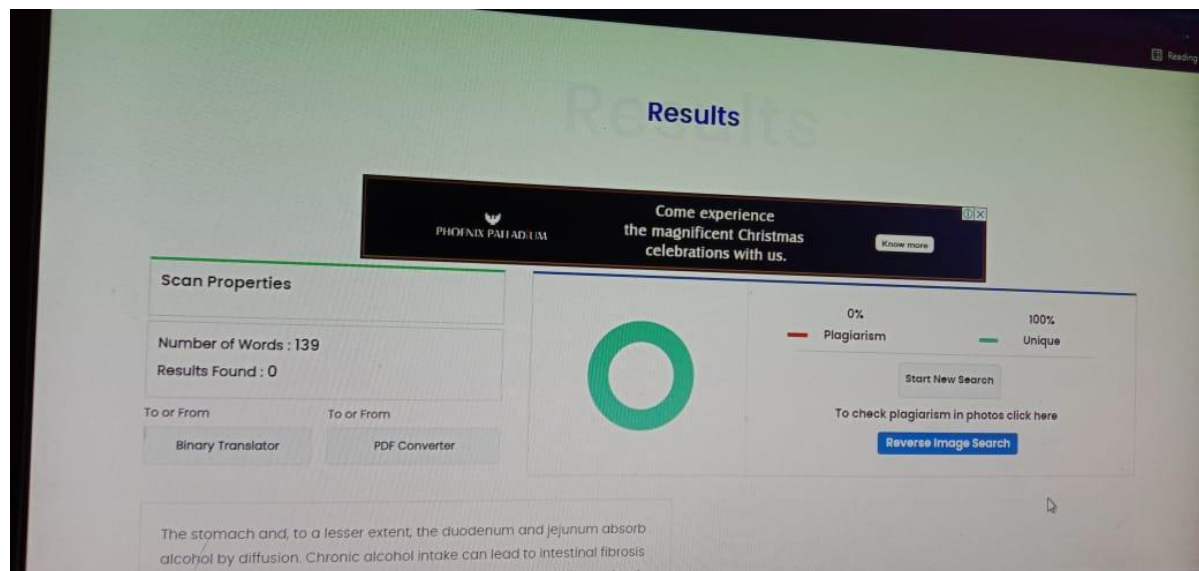
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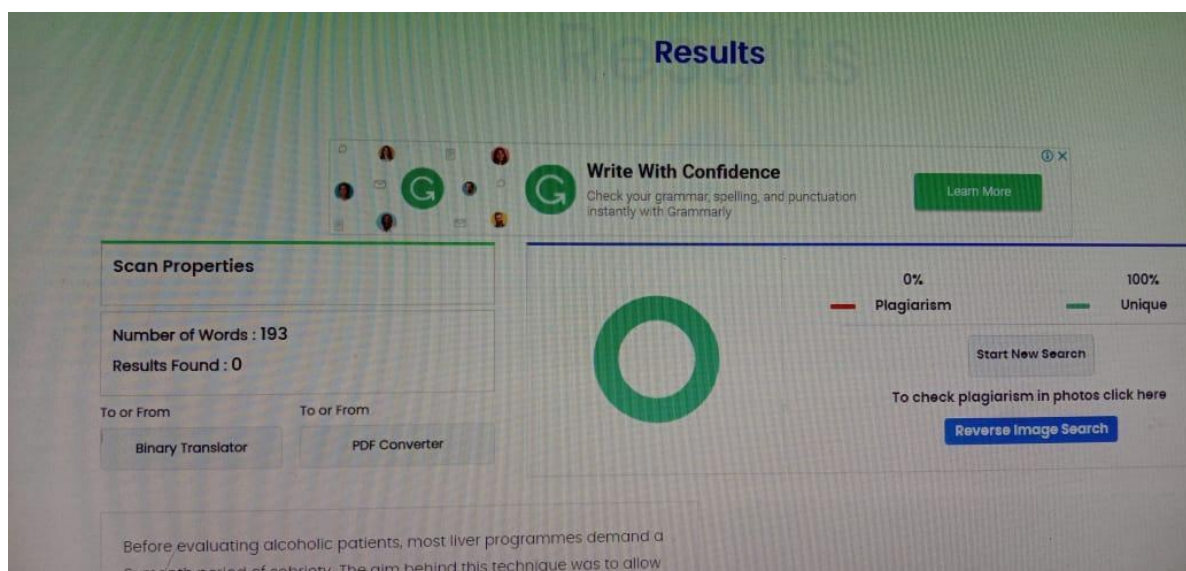
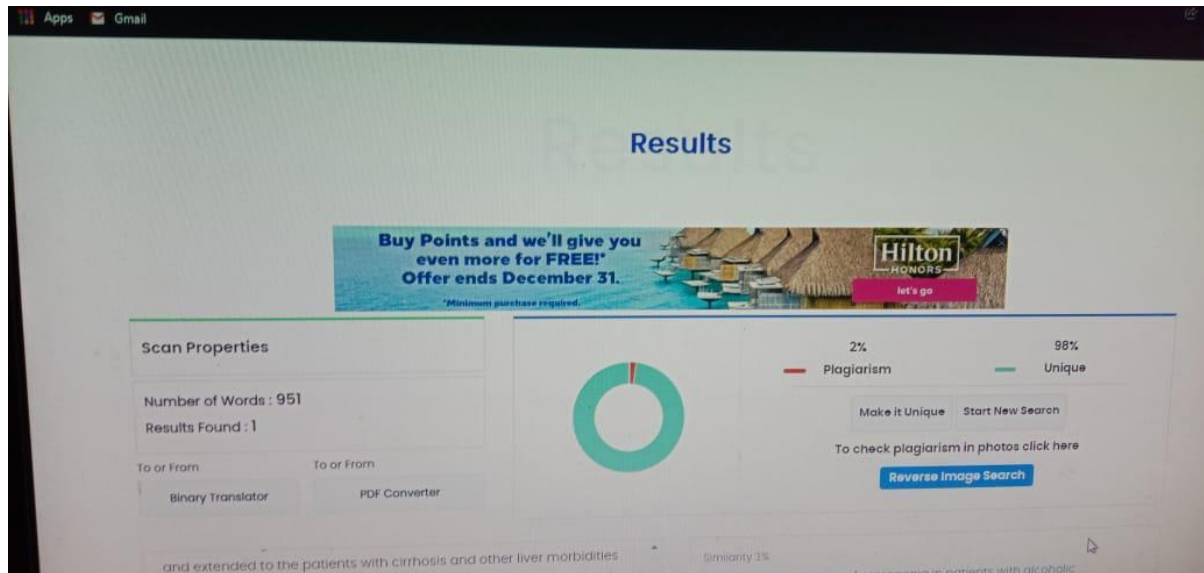
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Alcoholic liver diseases comprises of a very broad spectrum of different liver diseases, which ranges from alcoholic hepatitis to the most common one which is liver cirrhosis. The common complication

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After classification by risk factor, there was no difference in patient outcomes between alcoholic liver cirrhosis and non-alcoholic liver disease disease recipients. The risk of PVT/BD and concurrent PVT/BD

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• Nutritional Therapy:- Malnutrition in cirrhosis is significantly common because there is low energy into it due to absorption and altered energy metabolism researches indicate that energy intake should be increased