

## Alcohol Induced Liver Cirrhosis and its Treatment

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### Abstract

The advent of liver diseases globally contributes to a major burden in terms of mortality rate and disease factor. The liver, which is a major complex organ, plays a vital role in the metabolism of the body. The diseases associated with the consumption of alcohol such as cirrhosis and cancer that can be diagnosed through various pathological and cytopathological methods. This review insights how the normally functioning liver cells become vulnerable and lead to the onset of various fatal disorders if left untreated. The alcohol, unlike other drinks, is directly absorbed from the stomach into the bloodstream and is further metabolized by the hepatocytes. The products of alcohol breakdown, such as aldehyde, are toxic to hepatocytes and thus cause liver injury, in cases of chronic alcohol consumption. This injury to the hepatocytes evolves over time and leads to liver cirrhosis. The only cure for severe liver cirrhosis is a transplant of the liver. On the whole, the widespread consumption of alcohol, its making, and effects on the hepatocytes on chronic consumption, which leads to alcoholic liver diseases reviewed.

**Keywords:** Hepatocytes; Fibrosis; Cirrhosis; Hepatitis; Hemochromatosis.

### Introduction

Alcohol is one of the most widely consumed beverages in the world and the traces of various form of alcohol and its consumption have existed throughout the history (Burnett, P. et al 1999). The traces of alcohol fermentation and its consumption go as far back as the Neolithic man (Sibbesson et al 2019). Alcohol has been prominent across time and generations due to the fact that it causes

exhilaration and provides a feeling of euphoria to the user. Various methods for the production and refining alcohol have been industrialized which work on the same old principle that the yeast breaks down the sugary product and yields ethanol as its by product. Now when it comes to the consumption of alcohol, youth and the elderly do not shy away from it. The remarkable ability of the alcohol to get absorbed without going through the regular digestion process and give an instant result to the brain; makes it hard to resist its consumption. Since, the alcohol does not undergo a normal digestion process and is directly absorbed in the blood, its break down and processing befalls on the liver (Cederbaum et al 2012).

The liver produces many chief enzymes and some hormones and plays a crucial role in the process of digestion of any and all food that we ingest. The liver acts as a filter for blood and all the blood passed through the liver via a dedicated circulation system called the hepatic portal system. Similar to all the consumed antibiotics and steroids which move to the liver for their breakdown, the alcohol also passes through the liver and gets broken down into simpler products (Tuma et al 2003). The two crucial steps that breakdown the alcohol is alcohol dehydrogenation in the presence of alcohol dehydrogenase (ADH) and acetaldehyde dehydrogenation in the presence of aldehyde dehydrogenase (ALDH). The dehydrogenation of acetaldehyde is a very crucial part of the alcohol metabolism as acetaldehyde is a major toxic metabolite and the longer it stays in the liver, the more harm it causes. The perpetual breakdown of alcohol in the liver causes the acetaldehyde to buildup in the liver and it causes irreparable damage to the hepatocytes. Acetaldehyde causes impairment in the functioning of key enzymes and also leads to DNA damage and thus promotes mutation in the hepatocytes. This promotes alcoholic liver disease and eventually the mutation in the hepatocytes due to DNA damage causes them to collect collagen and become fibrotic. The hepatocytes slowly harden over time and lose their

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ability to function normally. When the fibrosis continues, it leads up to cirrhosis of the liver where the normal functioning of the liver is completely lost. Cirrhosis can happen due to an ample number of reasons all of whom are worsened further by chronic use of alcohol and eventually this is referred to as alcoholic liver cirrhosis (Bellentani et al 1997).

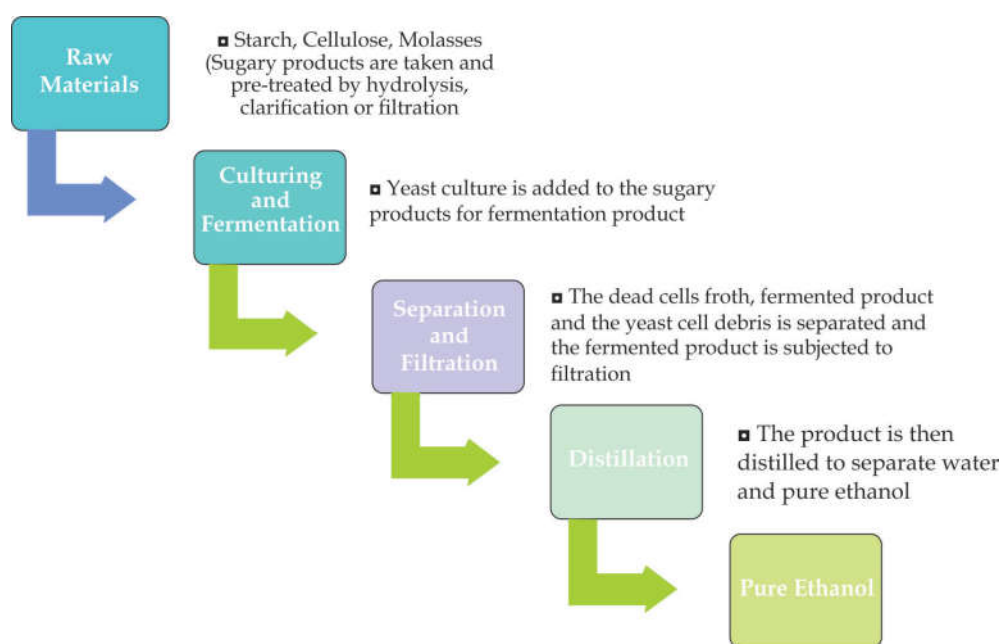
### Alcohol

Alcohol is a fermented drink that is formed when the yeasts breakdown the sugars of various foods. Most of the foods that contain high amounts of sugars (such as grapes, barley, apples, potatoes, etc.) can be used to make alcohol. Alcohol is classified as sedative drug and low amounts of alcohol act as stimulants and induce the feelings of euphoria and loquacity. Being sedative in nature, alcohol

represses the function of central nervous system, which indicates that consuming too much of alcohol in one go can cause respiratory depression and drowsiness. Depending on the concentration of alcohol in the blood, it affects all the organs differently (Swift et al, 2003).

### The Conventional Production of Alcohol

Every brewery that ferments alcohol beverage has its own scheme which is patented. The use of different species to ferment sugars varies from industry to industry thus makes the taste of the beverage different from the other. The quality of



**Figure 1.** The Steps in Alcohol Fermentation. The step by step process of making of alcohol. The process starts from a raw material that has high content of sugar, then the yeast is added to it. As the yeast digests the sugar, it releases ethyl alcohol which is then filtered and distilled out to make the consumable ethanol.

the beverage depends on how well the yeast cells ferment the sugars and the distillation of the same is another crucial step. The aging plays an essential role in its quality as it is quoted that the older the alcohol the better is the taste and quality.

The process of obtaining the final product as alcohol might vary from industry to industry but the core chemistry of the making remains invariable.

The process begins with some grains or fruits that have high sugar content in them. Then yeast

is added to the batch and it begins to digest the sugars in present and in turn releases ethanol and carbon dioxide as the by-products of that digestion. After the alcohol content in the batch reaches to about 14%, the yeast dies and thus the fermentation stops. After the death of the yeast, we are left with a batch that has ethanol, water and debris of dead yeast cells. This fermented content is then filtered to obtain a mixture of ethanol and water, which is then distilled using steam distillation process to obtain pure consumable ethanol (Figure 1).

### *Liver: Major gland in the body*

The liver serves as both endocrine and exocrine gland in the body. It produces proteins that are responsible for clotting of blood at the site of an injury. It releases hormones like IGF-1 into the blood stream and at the same time, helps in digestion of food and breakdown of fatty acids and lipid molecules by producing bile juice. It produces bile juice all day long but since bile is needed only four to five times a day, during digestion, it stores the bile in a small pouch-like structure called the gall bladder. Liver is connected to two major blood vessels for blood supply, namely hepatic artery and portal vein. The main function of the liver is to filter the blood coming from the stomach and detoxify it before it is circulated to other organs of the body. It is responsible for the metabolism of antibiotics and steroids and serves as the primary site for their breakdown. The cells of the liver are completely renewed every two years and they also have a remarkable capability to heal wounds and recover from significant injuries. This ability of hepatocytes to heal efficiently is what makes the liver a perfect site to deal with blood toxins since the cells repair quickly after the damage and don't show signs of fatigue. The damage to the DNA of hepatocytes is also minimal, so long as they are not subjected to the toxins repeatedly and chronically (Ozougwu, J, 2017).

### *Metabolism of alcohol in the liver*

Despite of the fact that, some of the alcohol reaches to the lungs and brain, the key metabolism of alcohol, which is principally ethanol, happens in the liver. Ethanol, unlike carbohydrates and fats that are ingested in the diet but can also be synthesized in the body, is a foreign substance and cannot be synthesized in the body. Moreover, very little alcohol can be disposed of by the kidneys and lungs. The most suitable way that outlines and finally eliminates the alcohol content from the body is the oxidation of alcohol. Contrary to the carbohydrate and fat oxidation that can take place in all tissues, the alcohol oxidation is only limited to the liver as it contains the crucial enzymes for the same (Cederbaum A. I, 2012).

The primary alcohol metabolism proceeds in four main steps. In the first step the alcohol is broken down into acetaldehyde with the help of the enzyme called alcohol dehydrogenase (ADH). The ADH acts as a generalized electron remover not only from alcohol but also from various

other compounds. This removal of hydrogen from the alcohol converts it to acetaldehyde and then the enzyme acetaldehyde dehydrogenase (ALDH) converts acetaldehyde into acetic acid. The acetic acid then eventually breaks down into carbon dioxide and water and passes out of the liver (Figure 2). The harmful metabolic effects of alcohol are mainly related to the first two steps of the alcohol metabolism.

### *Diseases Associated with Liver*

#### *Viral diseases*

The liver is vulnerable to many types of diseases that impair its function and cause general discomfort in the body. The hepatocytes are prone to many types of viruses and genetic disorders. The prominent viruses that affect the liver are of hepatitis. Some forms of hepatitis go away with time, such as hepatitis A and some other forms of hepatitis remain in the liver for a long time, such as hepatitis C. Jaundice is also a form of liver disease that is generally a precursor or indication of a bigger problem in the liver. The basic symptom of jaundice is the yellowing of skin, nails and sclera of the eyes. This happens because in jaundice, the liver cells are unable to process the bilirubin and thus the bilirubin begins to leak in the blood stream causing the yellowness of the skin and other parts. Jaundice is either just a short-lived disorder that is cleared over a span of few weeks or jaundice is a symptom of a much larger cellular damage in the liver. It is often seen as the first symptoms of liver cell damage in most cases (Tandon, B. N. et al 1987).

Viral diseases of the liver mainly comprise of various forms of hepatitis, ranging from hepatitis A all the way up to hepatitis E and G. These diseases have a similar attack mechanism in which the virus affects the hepatocytes and hinders their function. The most prominent and commonly occurring viruses are of hepatitis A, B and C. All the forms of viruses cause acute viral hepatitis. Hepatitis A virus (HAV) causes a short-term hepatitis that is generally cleared by the body's immune system and does not require and heavy medical treatment (Emini et al 1985). Viruses of hepatitis B and hepatitis C are capable of causing chronic viral hepatitis that, in some cases, might last a life time. Other forms of viral hepatitis that infect liver are hepatitis D, E and G.

Hepatitis D virus is not harmful on its own and is usually inert in the body. The virus requires

HBV to survive in the body and also requires a surface antigen that is made by HBV to infect the liver cells. This virus is more commonly active in the individuals who have chronic hepatitis B as these individuals have a proper viral load of HBV and this makes the survival of HDV possible. Individuals having chronic hepatitis D along with chronic hepatitis B are very difficult to treat and this combined infection speeds up the process of liver scarring and the individual is at a greater risk of developing liver cirrhosis later in life (Braga. et al 2006).

Hepatitis E (HEV) resembles to HAV in terms of disease symptoms and the virus is mainly contaminated by water. Hepatitis E, just like hepatitis A, is self-limiting and often goes away on its own once the disease has completed its course (Balayan, M. S. 1997).

All of these viral hepatitis diseases are contaminable and are transferred through air, water, food, fecal matter, urine, infected needles, and shared needles such as in case of drug addicts. Most of the forms of hepatitis have a vaccine available to prevent the disease however, the hepatitis C virus has no vaccine till today and the treatment options available for HCV are limited to anti-viral medications. This is one of the reasons why the HCV is hard to treat and often becomes chronic.

## 2. Genetic Diseases

The genetic diseases in the liver result in the predisposition of liver cirrhosis and cause lifelong problems as they start from an early age and are mostly incurable because the liver cells have a genetic defect in them and today's science is not advanced enough to treat every cell individually. The possible solution for a genetic liver disease is a liver transplant in which a new liver that is free of any kind of genetic disorder is placed in the patient (T. Taddei et al 2008).

Alpha-1 Antitrypsin deficiency or AAT is a genetic disorder in which the production of AAT is not possible as the gene that codes for its production is not available. The protein is mainly synthesized by the liver cells and it inhibits pro-inflammatory proteases such as neutrophil elastase thus protecting the lungs from proteolytic damage. The AAT deficiency generally presents itself as lung disorders. The severity of the disease ranges from chronic hepatitis to liver cirrhosis. If the disease progresses further, it may lead to total liver failure.

The disease progression is slow but the individuals with AAT deficiency have a higher chance of developing hepatocellular carcinoma. Transplant is not a viable solution for the AAT deficiency as the liver damage is due to the presence and accumulation of AAT mutant polymers and not due to the lack of the AAT circulating in the body (S. K. Brode et al 2012).

*Cystic fibrosis* is a genetic disorder that mainly affects lungs and causes respiratory insufficiency. The pancreas and pulmonary system is also affected by this disorder (D. Debray et al 2011). It is a frequent autosomal recessive disorder that is lethal, if left untreated. The symptoms of the disease begin to appear in the first decade of the life but strangely only a few patients develop a liver disease. The disease depends upon the altered activity of cystic fibrosis transmembrane regulator (CFTR) chloride channel on the apical membrane of colangiocytes. This causes the bile flow to alter and is then followed by colangiocyte-induced inflammatory response and proliferation of stellate cells and this gives rise to periportal fibrosis and cholangitis. Now the cystic fibrosis related liver disease is slowly progressive but, in some cases, it may rapidly result in portal hypertension. The diagnosis methods for cystic fibrosis are not accurate however the biopsy can help but the patchy distribution of the fibrosis affects the sensitivity of the biopsy as well (N. Ovchinsky et al 2012).

*Wilson's disease*, the genetic disorder with a fairly common occurrence in the population. This disorder manifests itself towards the second decade of the life and the neurological disorders also begin shortly thereafter, if the disease is left untreated. This disorder mainly depends on the mutations in the gene encoding ATP7B Cu translocase. This protein is mainly made by the hepatocytes and is responsible for maintaining the levels of copper in the liver. The lack of ATP7B Cu activity causes the hepatocytes to accumulate copper in the liver. The effects of Wilson's disease range from mild hepatomegaly to severe liver cirrhosis to liver failure. The patients often show brain function alterations and psychotic symptoms which are reversible with adequate therapy. Other treatment options include liver transplant in which the new liver is genetically free of errors in the gene and the liver produces ATP7B Cu properly.

Hemochromatosis is an autosomal recessive hereditary disease and it is characterized by excessive buildup of iron in the liver. This condition is fairly common and manifests itself in the fourth and fifth decade of life. The damage to



the liver in hemochromatosis happens mainly due to iron-induced lipid peroxidation that happens in hepatocytes and causes their death. Kupffer cells are activated and begin to produce cytokines, which stimulate hepatic stellate cells to synthesize collagen, and lead to cirrhosis. The cases of hepatocellular carcinoma are quite common in the patients with hemochromatosis (B. R. Bacon et al 2011).

### Drug Induced Liver Injury (DILI)

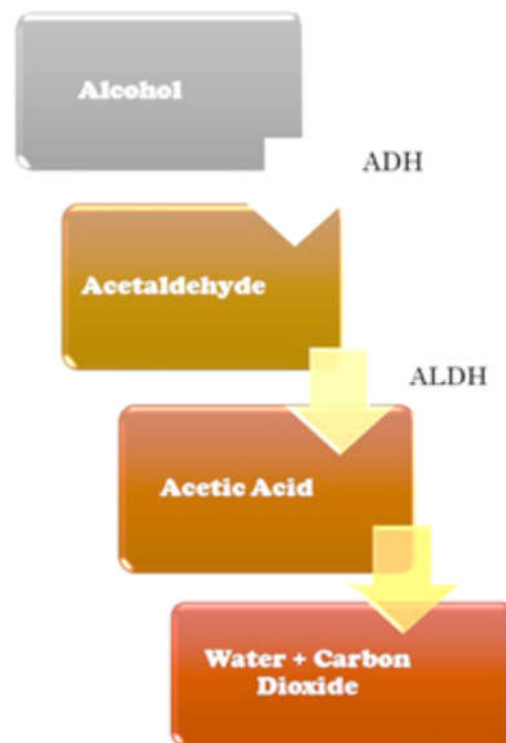
As the name suggests, the drug induced liver injury happens when the cells of the liver are affected by certain drugs that cause them to die during metabolism. This type of injury happens in response to every class of drugs. The complications arrive in cases where the liver is already damaged and the heavy dosage of medications can worsen the damage further. Paracetamol (acetaminophen) is one of the most prominent drugs in case of DILI. Other classes of the drugs that can damage the liver and cause hepatotoxicity (DILI) are anti-cancer drugs, anesthetics, cardiac medications and antibiotics. Cellular damage is repaired once the medications are stopped. Most of the cases of DILI are resolved as soon as the medications are discontinued. Some clinical symptoms may include jaundice and hepatitis. Most classes of steroids also cause drug induced liver injury (Agarwal, Vijay K et al 2010).

### Alcohol induced liver injury

The metabolism of alcohol (as mentioned in figure 2) produces aldehyde as a breakdown product. This aldehyde is then later converted to acetic acid which is further converted to carbon dioxide and water.

This acetaldehyde, however short lived, causes fast cellular damage and up to a certain level, the hepatocytes are able to recover from that damage and maintain a healthy function (Weathermon et al 1999). The problem arises when the individual consumes alcohol at a scale so large that it is too hard for the hepatocytes to recover from and the constant breakdown of alcohol to aldehyde and its slow excretion makes the aldehyde accumulate in the hepatocytes and the hepatocytes begin to mutate and collect fat globules, leading to fatty liver disease. The fatty liver disease that is mediated by the consumption of alcohol is called alcoholic fatty liver disease. This disease then progresses further

and the liver begins to form scars and collect

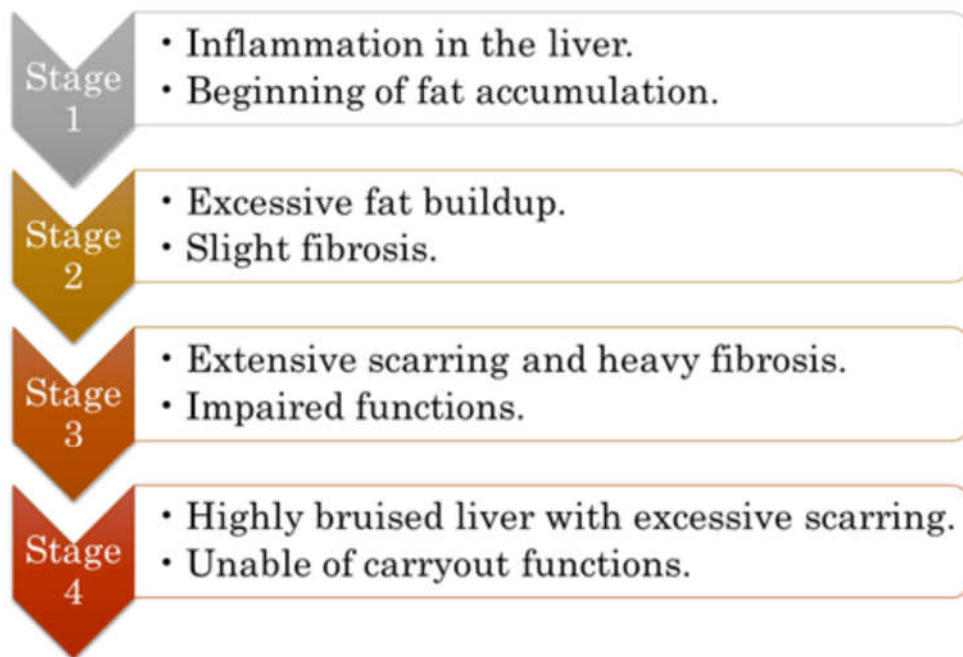


**Fig. 2** The conversion of alcohol in the liver. Hepatocyte breaks the alcohol down by the above-mentioned steps. ADH turns alcohol to aldehyde and then ALDH turns aldehyde to acetic acid. Acetic acid is then slowly removed from the body in the form of water and carbon dioxide.

collagen (scar tissue), which leads to hardening of the liver and ultimately causes liver cirrhosis.

### Liver Cirrhosis

The hepatocytes have a remarkable ability to regenerate and repair the damage to a certain point. The metabolism of alcohol by the hepatocytes leaves them in a vulnerable state. The hepatocytes breakdown the alcohol to acetaldehyde and this acetaldehyde is a toxic substance that actually harms the hepatocytes in the long run. The hepatocytes repair the damage that happens to them during alcohol metabolism, but slowly and gradually the genetic makeup of the hepatocytes begins to alter and the hepatocytes begin to collect connective tissue and collagen fiber in them to resist the perpetual damage that is happening to them. This collection of collagens in the hepatocytes leads to the hardening of the cells and this stage is called fibrosis and the fibrosis is a natural reaction of the cells to prevent further scarring in the liver. This fibrosis is the first stage of extensive liver damage and the continuation of fibrosis in the liver leads up to liver cirrhosis (Tsochatzis et al 2014).



**Fig. 3** Stages of Liver Cirrhosis. Different stages of liver cirrhosis change the structure of liver and affect its ability to function. In the beginning stage, the liver starts to inflame and accumulate fat. This does not affect much on the functioning of the liver but the structure of the liver changes. In the last stage, the structure of the liver is completely altered and the all the main functions are impaired to a point where the only treatment is a liver transplant.

Cirrhosis of the liver can be prevented, especially if the root cause of the cirrhosis is alcohol and not some other disease (Verrill et al 2009). Cirrhosis takes a long time to manifest and doesn't just happen overnight. The symptoms of liver damage become clear right from the beginning and if the alcohol ingestion is stopped after the indication of liver disease, the chances of cirrhosis due to alcohol become almost negligible. The alcoholic liver disease progresses in various stages and each of those stages have a defined structure of the liver damage. The disease begins with the liver cells collecting fat globules in them and then the disease progresses if the alcohol consumption is not stopped. After the fat accumulation, the liver begins to develop wounds since the fat molecules are soft and get damaged easily. These wounds heal and form scar tissues that are made of collagen and this stage is termed as fibrosis and this is where the liver begins to lose many of its functions and the body begins to show significant symptoms of liver damage. Eventually, the scarring of the liver cells continues, there comes a point where most of the surface of the liver is covered in scar tissue and the liver turns from a soft meaty chunk to a hard and rigid mass of cells. In the fibrosis stage, the liver cells barely function and the liver struggles to keep up with body's nutrition demands and this is the reason why most of the patients with liver disease are malnourished. What follows

liver fibrosis is the last stage of liver disease that is called liver cirrhosis and in this there is no hope for the liver to repair as the hepatocytes have lost all their healing ability and they barely perform any function. The cirrhosis is characterized by big areas of scar tissues on the surface of the liver, but the scars are not limited only to the surface of the liver, they cover the whole mass of the liver and also restrict all the blood vessels in the liver that goes on to create portal hypertension in the hepatic portal system and varices in the esophagus. The last stage of liver cirrhosis is called end stage liver disease (ESLD) and this is fatal without a transplant (Figure 3). The liver loses all its ability to function and the body shows significant symptoms of the liver cirrhosis. The general symptoms of liver cirrhosis that can be diagnosed physically are weakness, ascites, edema, easy wounding, slowed healing of the wounds, perpetual jaundice, mental confusion also known as hepatic encephalopathy, low immunity and multiple digestive disorders. Other symptoms that are used to diagnose liver cirrhosis chemically, include lack of clotting factors in the blood, peritonitis, lack of albumin protein in the blood, abnormally high levels of bilirubin in the blood which is also called jaundice. Imaging options available for diagnosis of liver cirrhosis include ultrasound, computerized tomography (CT Scanning), Magnetic Resonance Imaging (MRI) and fibroscan. All of these methods give an accurate

idea of the shape of liver and its ducts and all these methods are used to predict the extent of damage in the liver. One final test that is considered to be the most accurate out of all the known tests is biopsy. Biopsy is done to check the state of the cells. In this, a piece of liver tissue is obtained using the needle (needle biopsy) and then it is examined in the lab to give the exact idea of the liver damage (Murata, K. et al 1984).

Biopsy has certain limitations to it too. Since the liver is already damaged, it might not stop bleeding after the biopsy sample is taken. The internal bleeding may not stop and can cause complications later. Another limitation of biopsy is that it takes cells from one area of the liver, so we may not be able to know the state of the whole liver just by taking sample from one zone (Bravo et al 2001).

### Treatment of Liver Cirrhosis

Liver is a complex gland and as such is hard to treat. The treatment of the liver disease is further complicated by the fact that the functions of the liver cannot be outsourced to any machine or organ like we can do in the case of kidneys by dialysis or in case of lungs by a ventilator. The liver is at the center of the digestive system and as such any treatment of the liver tends to affect the whole body. Furthermore, the patient cannot be prescribed high doses of antibiotics as the liver will not metabolize them. This makes the treatment of infections like peritonitis difficult.

**Table 1.** Liver disease and their treatment. Types of liver diseases and the treatments that are possible to cure them by the use of modern medicine.

Type of Liver Disease	Treatment
Jaundice	Plenty of Rest
Hepatitis A, B,C,D,E	Antiviral medicines and antibiotics
.	Plenty of Rest for the liver to recover
Fatty Liver Disease	Refraining from alcohol High Protein Diet
Auto-Immune Conditions	Immunosuppressants
Genetic Diseases	Lifestyle Changes
Liver Cirrhosis (ESLD)	Liver Transplant
Liver Cancer	Liver Transplant

The cures for liver disease vary with the stage of the disease (Table 1). There is no known cure for end stage liver disease (ESLD) aside from a liver transplant. The stages prior to ESLD have known cures but none of them are effective to a large extent and do not help the liver fully recover. The liver

cirrhosis, therefore, has no known treatment to heal it completely or even partially.

### Liver Transplant

The liver transplant, is an exchange of old diseased liver with a new functioning liver so that the patient's life can be prolonged. The transplant in itself is a complex process but what follows is not a pleasant site either. The whole life of the survivor is uprooted and the living standard is impacted and diminished severely. The survivor cannot do much of anything after it. They are limited to bed rest and lose most of the human interaction as the immunosuppressants that are prescribed to them weaken their immune system and contact with another non-sterile human can cause them to develop severe infections. The infections will also be hard to get rid of because of the compromised immune system and the lack of antibiotics prescription. So, a minor infection to a normal functioning human can be life threatening to a transplant survivor. The rate of survival for a transplant patient has significantly increased and is now at 75% at one year, however sometimes the transplanted liver may fail due to some underlying cause that was not diagnosed previously. In some cases, the disease may return, especially if it was viral (B.J. Veldt et al 2002).

The most common form of liver transplant is the Orthotopic Liver Transplant (OLT). In this, a whole piece of liver is taken from the donor and is fixed in the receiver's abdomen. This is preferred in the patients that have low chances of survival and are unable to qualify for other methods of liver transplant. This OLT method is generally performed with a dead donor. The donor is either fully dead or brain dead. The transplant procedure is initiated immediately after the donor's life functions cease. The liver is taken out and relocated into the receiver's body (B.J. Veldt et al 2002).

Other methods used in liver transplant are living donor transplant and split transplant. Both of these methods require only a portion of the liver to be transplanted into the donor. In split donation, the liver is taken from a dead donor and is split in half. Both the halves are then relocated to different organ receivers and the liver then grows to full volume inside the receiver. In living donor transplant, a part of healthy liver of a living donor is taken and is relocated in the receiver's body. The liver then grows to original volume on its own inside the receiver. In case of the donor, the liver grows back to its original volume within a matter of weeks. This is possible only due to the liver's remarkable

capability to regenerate quickly. The survivors are given immunosuppressants to prevent the body from rejecting the organ. These medications are generally taken for life and the patient requires regular follow ups to keep track of the progress or deterioration of the liver transplant. The success rate of the liver transplant has increased in the last two decades. Out of 10 transplant patients, 5 survive as long as 20 years after the transplant. This is a huge progress as compared to only 4 out of 10 surviving in the 1990s (Kilpe VE et al, 1993).

The transplant in itself is gruesome for the patient and requires many precautions and tests which include matching the blood type, CT Scanning, Color Doppler, Liver Function Tests, Body Type, MELD Score. The Model of End Stage Liver Disease (MELD) is a score that is used to rank people on the basis of severity and urgency of the liver transplant. This score is used to rank patients on the national waiting list for organ donors. The MELD score is decided on the basis of the blood tests. The MELD score may increase as the patient gets more ill. This allows the sickest patient to receive the earliest treatment.

## Discussion

The alcoholic liver diseases are generally of three types depending on the frequency of consumption of alcohol. Alcoholic fatty liver disease, alcoholic hepatitis and alcoholic liver cirrhosis. These diseases are generally preventable by controlled use of alcohol or a total abstinence from it. The fatty liver disease is entirely reversible and the liver usually recovers from this within weeks, once the alcohol consumption is stopped. The alcoholic hepatitis is also a preventable disease and this is also a rare form of hepatitis that occurs in the individuals consuming large amounts of alcohol for a long period of time (chronic alcohol consumption). The reversibility of the disease depends on its severity and level of restraint of the patient. The reversibility of alcoholic hepatitis is variable and largely depends upon the speed of regeneration of hepatocytes and the abstinence from alcohol once the disease has been diagnosed. The chances of long-term survival in the patients who stop alcohol consumption are higher than the ones who continue to drink even after the disease has been diagnosed. Alcoholic liver cirrhosis is a chronic condition with a bad prognosis. In this, the patient's chance of long-term survival are really thin and the condition is irreversible. The only treatment known for alcoholic liver cirrhosis,

besides the general precautions, is a liver transplant where a new liver is placed in the patient.

## Conclusion

Alcoholic liver cirrhosis, at its core, is a form of liver cirrhosis that is promoted due to excessive and chronic use of alcohol. The alcoholic liver cirrhosis, just like any other disease, has multiple stages and the death of the patient is entirely preventable in this case if the disease is diagnosed early. The simple solution for preventing alcoholic liver cirrhosis, is refraining from excessive use of alcohol. The four stages of alcoholic liver cirrhosis are classified on the basis of extent and severity of damage. In which Stage 1 is mild liver disease that is cured on its own, provided that the individual doesn't complicate the situation further by consuming alcohol and Stage 4 is the lethal stage where the damage to the liver is so severe that the only option out is a liver transplant. Liver transplant is the only possible solution for the end stage liver disease (ESLD). This process is fairly complex and reduces the quality of life of the individual.

## Declaration

The authors declare that they have no conflicts of interest.

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