Effect of alcohol on liver enzyme (sGOT)

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Abstract

This study was aimed to estimated effect of alcohol on liver enzyme (GOT) , long term alcohol consumption can cause alcoholic liver disease It leads to develope alcoholic hepatitis and cirrhosis, Most of the liver damage caused by alcohol is attributed to alcohol metabolism and by products of that metabolism, Liver injury may be caused by direct toxicity of alcohol by products and also by inflammation that is induced secondarily by these same compounds, One half of heavy drinkers develop alcoholic hepatitis or cirrhosis.

Introduction:

Alcohol is clear drink that is made from corn, barley, grain, rye, or beverage containing ethyl, ethyl alcohol (ethanol) refers to the intoxicating ingredient found in wine, beer, and hard liquor. [1]

Blood alcohol concentration can rise up to 20 minutes after having drink. Alcohol is absorbed it leaves the body in three ways via kidneys, lungs, and liver[2].

How alcohol made? Beer and wine made by adding yeast to a substance that contains sugar, The yeast starts the formation process, which turns sugar into ethyl and carbon dioxide gas [2,3].

Another type of alcoholic beverage is called a distilled beverage. That is made from fermented grain mash or fermented juice. Distilled beverages include drinks such as whiskey, vodka, liqueurs, gin, rum and other beverages [3,4].

When alcohol is consumed, it passes from the stomach and intestine into the blood, a process referred to absorption [3]. Alcohol is then metabolized by enzymes, which are body chemicals that break down other chemicals. In the liver, an enzyme called alcohol dehydrogenase (ADH) mediates the conversions of alcohol to acetaldehyde. Acetaldehyde is rapidly converted to acetate by other enzymes and is eventually metabolized to carbon dioxide and water [5]. Alcohol also is metabolized in the liver by the enzyme cytochrom p450iiE1(cyp2E1) , which may be increased after chronic drinking [10].

Most of the alcohol consumed is metabolized in the liver , but the small quantity that remains and metabolized permits alcohol concentration to be measured in breath and urine . The liver can metabolized only ascertain amount of alcohol per hour, regardless of the amount that has been consumed. The rate of alcohol metabolism depends, in part, on the amount of metabolizing enzymes

in the liver, which varies among individuals and appears to have genetic determinants.

In general, after the consumption of one standard drink, the amount of alcohol in the drinker's blood (blood alcohol concentration, or BAC) peaks within 30 to 45 minutes. (standard drink is defined as 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of 80-proof distilled spirits , all of which contain the same amount of alcohol.) , alcohol is metabolized more slowly than it is absorbed. Since the metabolism of alcohol is slow, consumption needs to be controlled to prevent accumulation in the body and intoxication [6,7].

AST (transaminases) this enzyme is associated with inflammation and / or injury to liver cells, a condition known as hepatocellular liver injury. Damage to the liver typically results in a leak of AST into the blood stream. Because AST is found in many other organs beside the liver , including the kidneys , muscles and the heart , having a high level of AST does not always (but often does) indicate that there is a liver problem. For example, even vigorous exercise may elevate AST levels in the body.

The normal ranges for AST around (15 – 40 IU/) respectively [8] .

The level of transaminases may be much higher than if the alcohol had not been consumed. Following the same reasoning , if the liver was damaged years before – by excessive alcohol use – the results of a blood test done today may be normal , but a damaged liver may still be present[6]. With moderate drinking, the liver can process alcohol fairly safely. However, heavy drinking overtaxes the liver resulting in serious consequences. A liver clogged with fat causes liver cells to become less efficient at performing their necessary tasks, resulting in impairment of a person's nutritional health. Fatty liver is the first stage of liver deterioration in heavy drinkers, and interferes with the distribution of oxygen and nutrients to the liver's cells. If the condition persists long enough, the liver cells will die, forming fibrous scar tissue (the second stage of liver deterioration, or fibrosis). Some liver cells can regenerate with good nutrition and abstinence, however in the last stage of deterioration, or cirrhosis, the damage to the liver cells is the least reversible. And these process leads to elevate of GOT level [9,10].

Materials and methods:-

The chemical materials and the company provider;-

AST & ALT kit [RANDOX]

Blood from alcohol abuse patients.

Laboratory instrument & company provider:-

1-centrifuge 122593, German, Hettich

2-water bath 790792, German, MeMert

3-spectronic – 20 3300166456, American

Enzymes source:-

Sample of blood taken form 20 patients in different age of alcohol abuse and the doses range between (1 – 7) days, the blood put on nonheparinized test tube, then the serum separated by centrifuge on 3000 circle/minute for 15 minutes.

Enzyme assay:-

The principle of measured activity of AST (GOT) depend on colorimetric method.

$$\alpha\text{-oxoglutarate} + \text{L} - \text{Aspartate} \xrightarrow{\text{GOT}} \text{L} - \text{glutamate} + \text{oxaloacetate}$$

AST(GOT) is measured by monitoring the concentration of oxaloacetate formed with 2,4 – dinitrophenyl - hydrazine.

Solutions:-

Determination of activity enzyme used the following solution

Solution	Concentration mmol/L
R1= GOT Buffer Phosphate Buffer L – Aspartate α - oxoglutarate	 100 mmol/L 100 mmol/L 2 mmol/L
R2=2,4- DiNitro phenyl hydrazine	2.O mmol/L
R3=Sodium hydroxide Pyruvate standard Pyruvate	 2 mmol/L

The procedure

1- Determine the enzyme activity (GOT) on serum by kit procedure

a-Sample

add (0.5 ml) from R1 (GOT Buffer) on test tube , then add (0.1 ml) from sample (serum) to the same test tube , then the test tube put in water bath for (30 minutes) on 37C°

Then add (0.5 ml) of the R2 (2,4-DiNitro phenyl hydrazine) without water bath for (20 minutes) on(20-25) C°

Then add (5ml) from R3(NaOH) after 5 minutes calculate the absorption of sample

b-Reagent Blank

Add (0.5 ml) from R1(GOT Buffer) on test tube , then add (0.5 ml) from R2(2,4-DiNitro phenyl hydrazine) with (0.1ml) from distill water without water bath for 20 minutes on (20-25) C°

Then add (5 ml) from R3(NaOH) by concentration after dilution (0.4N)

After 5 minutes calculate the absorption of Reagent Blank.

2- read the absorption to each sample on wave length 545 , then calculate the result to define the enzyme activity by use the following equation :-

(IU/L)GOT activity = Test – control

T = serum absorption , C= spectronic absorption

3- compare the result of the samples with the normal value of GOT[16].

Results and discussion:

the study discuss the effect of alcohol consumption on liver enzyme activity (SGOT) in humans by taking blood sample from 20 drinkers of different age group and measuring the enzyme level which shows the following results:-

10 of them show increase in enzyme activity five of them markedly increased.

9 of them shows no change in enzyme activity.

1 of them in border line.

In comparisim with normal value of RANDOX kit (up to 12 U/l).

Details about result s shown in table (1-1)

Patient	Age/Y	SGOT U/L
1	25	17
2	30	15
3	60	30
4	40	10
5	52	11
6	30	13.5
7	35	14.5
8	38	12
9	28	8.5
10	25	7.5
11	40	8.1
12	37	7.5
13	34	14.5
14	24	11.5
15	26	9.5
16	50	35
17	45	65
18	50	25
19	35	10
20	55	78

Large proportion of heavy drinkers develop serious alcoholic liver disease , most alcoholic liver damage is attributed to alcohol metabolism , liver injury may be caused by direct toxicity of metabolic by product of alcohol as well as by inflammation induced by these by product[10,11] .

As alcohol broken down in the liver a number of potentially dangerous by product are generated such as acetaldehyde and highly reactive molecules called free radicals. Perhaps more so than alcohol itself these product contribute to alcohol induced liver damage [12] .

The liver is one of the largest organs in the body it has not only considerable reserves but also the ability to regenerate itself.

Consequently symptoms of liver damage may not appear until damage to the organ is quite extensive [13].

Epidemiological studies suggest that a threshold dose of alcohol must be consumed for serious liver injury to become apparent[15].

For men:-

This dose amount to 600 kg taken chronically over many years, an intake can be achieved by consuming approximately 72 oz of beer, 1 L of wine, 8 oz distilled spirits daily for 20 years [12,14].

For women:-

The threshold dose is one fourth to one half that amounts.

Heavy drinking refers to daily intake; heavy long term alcohol consumption clearly plays a major role in the development of alcohol related liver damage.

Yet no more than one half of heavy drinkers develop alcoholic hepatitis or cirrhosis [12,14].

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تأثير الكحول الاثيلي على أنزيم الكبد (GOT)

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الخلاصة

الدراسه تهدف الى تقييم تأثير الكحول الاثيلي على الكبد وعلى أنزيم ال(GOT) ، حيث أثبتت النتائج المختبريه أن المرضى الذين يتعاطون الكحول بكميات كبيره ولفترات طويله يسبب أمراض الكبد الكحوليه ويؤدي الى تشمع الكبد كحاله نهائيه للمرض الناتج عن اصابة الخلايا الكبديه بالسميه الناتجه عن العمليه الايضيه التي يتم بها معالجة الكحول في الكبد بعد تعاطيه، واثبتت الدراسه ان نصف المرضى الذين يتعاطون الكحول بشكل كبير ولفترات طويله يصابون بالتهاب الكبد وتشمعه.