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Short Report

Comparative study of serum 5' nucleotidase, alkaline phosphatase aminotransferases & bilirubin in alcoholic liver disease

Dr. Anil Batta

Associate Professor, Department of Biochemistry Baba Farid University of Health Sciences, FARIDKOT— 151203 (Punjab)

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ABSTRACT

Chronic alcohol use can lead to alcoholic hepatitis. The ALF warns that alcoholic hepatitis may last for years, causing progressive liver damage. The liver may be enlarged, tender, with nausea, fatigue, and jaundiced (yellow) skin and eyes as a result of hepatitis. Dilation of blood vessels beneath the skin's surface may cause spider veins and intense itching. Fluid accumulation around the liver and distention of major blood vessels can cause a hoarse voice and swelling. High blood pressure is common, and can damage the heart and kidneys. When healthy liver tissue is replaced by non-functioning scar tissue, the process of cirrhosis is underway. The normal passage of blood through the liver eventually becomes impossible, leading to the accumulation of toxins. Ammonia builds up in the blood, reaches the brain and the person feels and acts confused. A liver transplant may be necessary for survival. Liver cancer is a possible complication of alcohol-related liver problems, particularly of cirrhosis. Life-threatening congestive heart failure and internal bleeding are further complications of liver failure. With all above reasons & humanity in mind, I have tried my level best to bring this health hazard in a scientific mind to study. Since Alcoholic consumption has reached an alarming proportion I decided to take this research cum awareness to the mind of humanity at large so that my endeavor may bring the change in the thinking of people living in third world.

1. Introduction

Differential diagnosis of Alcoholic Liver Disease depends a lot on study of various enzymes. Development of serum enzymology has improved the ability to diagnose clinical course & treatment of patients with symptomatic & more so on asymptomatic cases of Alcoholic Liver Disease (ALD). For this purpose a bewildering array of enzyme assays is now available. It is like endoscope to the Surgeon & a step up in patient care. This enzyme assay was introduced six decade back with the usefulness of alkaline phosphatase in differential diagnosis of jaundice [1-5]. Alcoholic hepatitis describes liver inflammation caused by drinking alcohol. Though alcoholic hepatitis is most likely to occur in people who drink heavily over many years, the relationship between drinking and alcoholic hepatitis is complex. Not all heavy drinkers develop alcoholic hepatitis, and the disease can occur in people who drink only moderately Alcoholic hepatitis occurs when the liver is damaged by alcohol you drink [4,6-10]. Just how alcohol damages the liver — and why it does so only in a minority of heavy drinkers — isn't entirely clear. What is known is that the process of breaking down ethanol — the alcohol in beer, wine and liquor — produces highly toxic chemicals, such as acetaldehyde. These chemicals trigger inflammation that destroys liver cells. In time, web-like scars and small knots of tissue replace healthy liver tissue, interfering with the liver's ability to function. This irreversible scarring, called cirrhosis, is the final stage of alcoholic liver disease[10].

Because there are numerous liver diseases and a wide range of factors that can cause them, including viral infections, drugs and

* Corresponding Author : Dr. Anil Batta
MD (Biochemistry)Associate Professor,
Department of Biochemistry Baba Farid University of Health Sciences,
FARIDKOT—151203 (Punjab)
E.mail: akbattafarid@yahoo.co.in

environmental toxins, diagnosing alcoholic hepatitis can be challenging. In an effort to reach a diagnosis, your doctor may include one or more of the following steps [11-17].

Medical history and physical exam. Your doctor will ask you questions about your health history, including alcohol use, and conduct a physical exam.

Blood tests. These check for high levels of certain liver-related enzymes, such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT).

Ultrasound. Your doctor may use this noninvasive imaging test to view your liver and to rule out other liver problems.

Risk increases with time, amount Consumed Heavy alcohol use can lead to liver disease, and the risk increases with the length of time and amount of alcohol you drink. But because many people who drink heavily or binge drink never develop alcoholic hepatitis or cirrhosis, it's likely that factors other than alcohol play a role. These may include[18-20].

Genetic factors. Having mutations in certain genes that affect alcohol metabolism may increase your risk of alcoholic liver disease as well as of alcohol-associated cancers and other complications of heavy drinking.

Other types of hepatitis. Long-term alcohol abuse worsens the liver damage caused by other types of hepatitis, especially hepatitis C. If you have hepatitis C and also drink — even moderately — you're more likely to develop cirrhosis than if you don't drink. Increased blood pressure in the portal vein. Blood from your intestine, spleen and pancreas enters your liver through a large blood vessel called the portal vein. If scar tissue slows normal circulation through the liver, this blood backs up, leading to increased pressure within the vein (portal hypertension).

Enlarged veins (varicose). When circulation through the portal vein is blocked, blood may back up into other blood vessels in the stomach and esophagus. These blood vessels are thin walled, and because they're filled with more blood than they're meant to carry, they're likely to bleed. Massive bleeding in the upper stomach or esophagus from these blood vessels is a life-threatening emergency that requires immediate medical care.

Fluid retention. Alcoholic hepatitis can cause large amounts of fluid to accumulate in your abdominal cavity (ascites). Abdominal fluid may become infected and require treatment with antibiotics. Although not life-threatening in itself, ascites is usually a sign of advanced alcoholic hepatitis or cirrhosis.

Bruising and bleeding. Alcoholic hepatitis interferes with the production of proteins that help your blood to clot. As a result, you may bruise and bleed more easily than normal.

Jaundice. This occurs when your liver isn't able to remove bilirubin — the residue of old red blood cells — from your blood. Eventually, bilirubin builds up and is deposited in your skin and the whites of your eyes, causing a yellow color.

Hepatic encephalopathy. A liver damaged by alcoholic hepatitis has trouble removing toxins from your body — normally one of the liver's key tasks. The buildup of toxins can damage your brain, leading to changes in your mental state, behavior and personality (hepatic encephalopathy). Signs and symptoms of hepatic encephalopathy include forgetfulness, confusion and mood changes, and in the most severe cases, coma.

Scarred liver (cirrhosis). Over time, the liver inflammation that occurs in alcoholic hepatitis can cause irreversible scarring of the liver (cirrhosis). Cirrhosis frequently leads to liver failure, which occurs when the damaged liver is no longer able to adequately function.

Liver biopsy. In this procedure, a small sample of tissue is removed from your liver and examined under a microscope. Liver biopsy usually involves inserting a long, thin needle through your skin and into your liver in order to draw out a sample of tissue.

Malnutrition. Many people who drink heavily are malnourished, either because they eat poorly — often substituting alcohol for food — or because alcohol and its toxic byproducts prevent the body from properly absorbing and metabolizing nutrients, especially protein, certain vitamins and fats. In both cases, the lack of nutrients contributes to liver cell damage. Recommendation of using gut-cleansing antibiotics for the treatment of hepatic encephalopathy is outdated and not evidence-based. Although neomycin has been used to treat hepatic encephalopathy for many years, no controlled studies have found it to be effective, as compared with standard treatment alone. A randomized trial2 comparing neomycin at a dose of 6 g per day with placebo showed no difference in outcomes between the two study groups. A downside to using neomycin is its association with ototoxicity and Nephrotoxicity Metronidazole, vancomycin,3 and rifaximin4 have shown some effectiveness in small clinical trials, but convincing evidence of effectiveness is lacking, and these antibiotics may cause alterations in gut flora that may lead to the development of bacterial overgrowth syndromes.

2. Material and Methods

Present study included a total of one hundred cases. Out of this forty cases comprised normal healthy individuals with no history of taking alcohol. These were attendants & relatives who came along with the admitted patients of alcoholic liver disease or came to the OPD of Govt. Medical College & Hospital. These were examined thoroughly i.e. any disease which can affect liver. So pregnancy & other disorders that can lead to rise in 5'Nuceotidase (5' NT),Alkaline phosphatase (ALP), aminotranferases (AST/ALT) & serum bilirubin.

For the study group sixty patients suffering from ALD were admitted in Rajindta Hospital (GMC,Patiala).Detailed history was taken & examined completely.All this was recorded recorded on a special performa.Each case was investigated as under:-

Haemoglobin, TLC/DLC, bleeding clotting time, Urine examination for bile salts & pigments, Fasting Blood sugar, Blood urea.

2.1. Collection of serum

About 10 cc of blood was collected by venepuncture using disposable & sterilized syringes & needles. It was collected in dry & clean test tubes at & centrifuged at 3000 rpm for ten minutes to separate serum. It was used to study above mentioned parameters.

2.2. Various facts to construct the study & control Groups

1. Age in all varied between 20—65 yrs.
2. All were males in the study Group
3. Socioeconomic Status was mixed.
4. Diet was normal middle class .
5. Routine investigations were done by routine tests.

3. Results and Discussion:

3.1. Observations

5'Nuucleotidase found to be a useful parameter to guess the effect of alcohol on the Liver. ALP was again found to be a useful parameter in cases of ALD. Both the above parameters were found to be highly significant when obstruction was there & serum bilirubin was raised (Table 1&2).

Values of AST were though significant were only to a limited extent. Values of ALT were found out to be highly significant as compared to level of AST. Bilirubin level in the serum was adjudged as significant when obstruction was significant (Table 1&2).

Table1. Analysis of various parameters in control group

Investigation	Range	Mean +
5'NT	2—8	5.00 1.69
ALP	3—10	6.42 2.19
AST	4—12	7.55 2.37
ALT	5—12	9.15 2.42
Bilirubin	0.4—0.8	0.57 0.166

Table 2. Statistical analysis of serum levels in control & study Group

Instigation & Groups	Range	Mean	S.D	S.E	't'value	Significance
Control	2—8	5-0	+i.69	-------	-------	-------
ALD						
5'NT	5----17	10.28	+3.76	1.44	3.66	High
ALP	8--15	9.57	+2.32	0.94	3.35	High
AST	11--55	20.85	+14.30	5.41	2.45	High
ALT	7--25	17.53	+7.53	2.87	2.73	High
Bilirubin	o.6—3.6	1.54	+1.33	0.42	2.30	High

4. Conclusion

Thus it is concluded that enzyme 5'NT in ALD contributed as an encouraging boost along with other laboratory findings & clinical data. 5'NT was superior to ALP as far as it's sensitivity & specificity is concerned. While elevated levels of AST/ALT signify the extent of hepatocellular damage,5'NT specifically signifies the biliary tract obstruction or cholestasis.but used as a solitary index, it doesn't measure up to expectations .

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He is presently working as Associate Professor Baba Farid University of Health Sciences, Faridkot (Punjab).

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