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RESEARCH ARTICLE

Study on Alcoholic Withdrawal Score, with Questionnaire Based Session Conducted on Acute and Chronic Alcoholic Liver Disease Patients

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ABSTRACT

Alcohol liver disease is damage to the Liver and its function due to alcohol abuse. It occurs after years of heavy drinking and by through which cirrhosis can occur and which leads to the final phase of Alcoholic liver disease. It not only occurs in heavy drinkers but also there is a chance of getting liver disease go up the longer of been drinking and more alcohol consumption. A study was observational, prospective and descriptive; and was carried out one hundred and nine patients [n=109] who were with suffering from an Alcoholic liver disease, to determine the alcohol withdrawal score and there symptoms involved after they were kept on alcohol withdrawal therapy. An observational, prospective and randomized study was conducted in the hospital from March 2014-March 2016. Questionnaire based session with 10 scaled questions were framed according to CIWA (assessment and management of alcohol withdrawal) and the score was noted with their symptoms occurrence after the alcohol cessation plan. CIWA score with moderate severity were found to be highest. 7 patients out of 33 patients in severe category of CIWA score were admitted in the hospital with alcohol withdrawal syndrome and psychological disturbances. Clinical Institute Withdrawal Assessment of Alcohol Scale (CIWA) helps clinicians assess and treat potential alcohol withdrawal.

KEYWORDS

Alcohol Liver Disease, Alcohol Withdrawal Therapy, Alcohol Withdrawal Score, Duration of Alcohol Intake

INTRODUCTION

Definition

Alcohol liver disease mainly refers to liver damage caused by excess intake of alcohol which includes the fatty liver, alcohol hepatitis, and chronic hepatitis with liver fibrosis or cirrhosis. The liver is one of the large lobed glandular organs in the abdomen of vertebrates.

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It also performs multiple functions, such as the production of proteins and the enzymes and its metabolic functions, and the regulation of cholesterol and blood clotting in the body. Because the liver is the primarily responsible for alcohol metabolism, it is especially vulnerable to alcohol-related injury.

Alcoholic liver disease (ALD) is a most serious and potentially fatal consequence of drinking alcohol. ALD mainly undergoes three conditions: Fatty liver (i.e., steatosis), which is the most common alcohol-induced liver disorder and is marked by the excessive accumulation of fat inside the liver cells. Alcoholic hepatitis it is the

inflammation and also a severe injury of the liver in which the body's immune system responds to and causes liver damage. In cirrhosis, normally in the body, the liver cells are replaced by mainly scar tissues such as like fibrosis and by which consequently the liver is unable to perform many of its normal functions in the body. Cirrhosis and alcoholic hepatitis often leads to morbidity and mortality. Mainly this article examines the diagnosing and treating of ALD and the complications of the disease.[1]

Epidemiology

Alcoholic liver disease is a major medical complication of alcohol abuse. Liver cirrhosis is the major cause in the Western world, with schistosomal infection being the major cause of portal hypertension in the developing countries. Alcohol mostly accounted for 80% of all liver cirrhosis cases which were observed in general hospitals in the UK. Alcoholic cirrhosis is more increasingly seen in countries such as Japan and India which has traditionally a low prevalence of the disease.

The safe limits for alcohol intake are mainly controversial. The Guidelines which were recommended by the Royal College of Physicians advise a weekly limit of 21 units (210 g) of alcohol in men and were as in women 14 units. In 1994 the office of population Censuses and Surveys General Household survey were found that 27% of men and 13% of women in the UK were exceeding these limits. A recent prospective Italian study in 6334 subjects showed that the risk threshold for developing ALD is 30 g ethanol/day and this risk increases with increasing by its daily intake. Women had greater susceptibility to ALD in the range of 3-8 drinks daily.

The pattern of drinking was also as important as ALD which is been increased in those who drink without accompanying food and also in those who drink multiple different alcoholic Beverages. It is well known that food delays gastric emptying and intestinal absorption of alcohol and thus intake of the food before drinking will decrease the rise of blood alcohol concentrations in the body. The absorption of alcohol is lower when consumption low concentration beverages such as beer compared with high concentration spirits.

Major Classification of ALD

- Alcoholic fatty liver (Steatosis)
- Acute alcoholic hepatitis
- Alcoholic cirrhosis

Alcoholic Fatty Liver (Steatosis)

Steatosis is invariable if and so the consumption exceeds 80g of alcohol per day. A large proportion of the cytoplasm is affected to hepatocytes and is occupied by a single large triglyceride occlusion but the liver function is often normal. It is as well as a reversible with abstinence but may progress to cirrhosis if excess alcohol intake persists. Deaths due directly to the fatty liver are rare and are usually caused by acute liver failure or a fat embolism.

Acute Alcoholic Hepatitis

In alcoholic hepatitis mainly the hepatocyte ballooning causes due to increased intracellular water accumulation. The Mallory's hyaline bodies are perinuclear eosinophilic inclusion bodies which are probably condensed and disorganized the fragments of the cytoskeletal framework of the hepatocyte. They are not specifically for alcoholic hepatitis as they are also seen in Wilson's disease and primary biliary cirrhosis. It has been estimated that 15-20years of age group people who consumes excessive drinking is been to developed alcoholic hepatitis. Thus there is prominent cholestasis. It is more severe in females and also in Northern Europeans and is unrelated to patterns of drinking or type of alcohol drink.

Alcoholic Cirrhosis

It is the most severe form of alcoholic liver injury and is usually of the micro nodular type. Mainly the risk is increased in continuous drinkers compared to binge drinkers. In this, the Collagen deposition can be pericellular, and around central veins (central hyaline sclerosis). Then latter is often associated with rapid progression to cirrhosis and is more severe in women. Collagen

bridges eventually develop between central veins and portal tracts isolating groups of hepatocytes which form the regeneration of the nodules. Survival for patients is 60%-70% at one year and 35%-50% at five years.[2]

Pathophysiology

Alcohol is metabolized in the liver through 2 main pathways:

Alcohol Dehydrogenase

Cytochrome P-450 2E1

1. In the liver, Alcohol is metabolized to acetaldehyde by alcohol dehydrogenase, which then is metabolized to acetate by the mitochondrial enzyme acetaldehyde dehydrogenase. Alcohol dehydrogenase and acetaldehyde dehydrogenase reduce nicotinamide adenine dinucleotide (NAD) to NADH (reduced form NAD). Excessive NADH in relation to NAD inhibits gluconeogenesis and increases fatty acid oxidation, which in turn promotes fatty infiltration in the liver.

2. The cytochrome P-450 2E1 pathway which also metabolizes the alcohol and generates free radicals through the oxidation of NADPH (reduced form of nicotinamide adenine dinucleotide phosphate) to NADP. Chronic alcohol use up-regulates cytochrome P-450 2E1 and produces, more free radicals.

Chronic alcohol exposure also activates the third site of metabolism: Hepatic macrophages, which produces TNF-alpha and induce the production of reactive oxygen species in the mitochondria.

Alcoholic people are usually deficient in antioxidants, such as Glutathione and vitamin E. Therefore, oxidative stress promotes hepatocyte necrosis and apoptosis in these patients. Free radicals can also induce lipid peroxidation, which can cause inflammation and fibrosis. The alcohol metabolite acetaldehyde, when bound to cellular protein, produces antigenic adducts and induces inflammation. Alcohol also affects the barrier function of the intestinal mucosa, Producing Endotoxaemia, which leads to hepatic inflammation.[3]

Symptoms

Early Symptoms: Abdominal pain, fatigue, Diarrhea.

Advanced Symptoms: As the liver becomes more damaged, more obvious and serious symptoms can develop, such as.

- Yellowing of the skin and white of the eyes (jaundice)
- Swelling in the legs, ankles, and feet, due to a build-up of fluid (edema)
- Swelling in the abdomen, due to a build-up of fluid known as ascites
- Fever
- Significant weight loss
- Insomnia
- Vomiting blood and black, tarry stools due to internal bleeding
- Weakness and muscle wasting

Diagnosis

Maintenance of a high clinical suspicion

- ✓ **Clinical features:** Overweight/obesity, Hypertension, Insulin resistance (type II DM), Hepatomegaly, Acanthosis nigricans.
- ✓ **Serum biomarkers:** Transaminases ALT and ASP, Total Bilirubin, Pro inflammatory marker, Pro fibrinogen markers, cytokines, Hepatocyte apoptosis marker, Lipid Profile, Serum autoantibodies.
- ✓ **Imaging techniques:** Abdominal ultrasound, Magnetic resonance imaging, computerized Tomography, Transient Elastography.
- ✓ **Liver biopsy:** Inflammation, Fibrosis, Cellular infiltration.[4]

Alcohol Withdrawal Syndrome: Alcohol withdrawal syndrome is the symptom that occurs when a heavy drinker suddenly stops or significantly reduces their alcohol intake through which a combination of physical and emotional symptoms can be experienced by the person.

Table1: Alcohol Drinking Level and Risk

	Men Daily	Women Daily	Men Weekly	Women Weakly
Very high risk	20+ drinks	15+ drinks	80+ drinks	60+ drinks
High risk	13-19 drinks	9-14 drinks	50-79 drinks	40-59 drinks
Medium Risk	8-12 drinks	6-8 drinks	30-49 drinks	25-39 drinks
Low risk	5-7 drinks	4-5 drinks	15-29 drinks	8-24 drinks
No risk	3-4 drinks	2-3 drinks	14 or fewer	7 or fewer
Healthy	1-2 drinks	1 drinks	1-14 drinks	1-7 drinks

The main cause of alcohol withdrawal syndrome is excessive drinking excites and irritates the nervous system by drinking daily the body becomes dependent on alcohol over time when this happens the central nervous system can no longer adapt easily to the lack of alcohol. Thus when u suddenly stops drinking or significantly reduces the amount of alcohol it can cause alcohol withdrawal syndrome.

MATERIAL AND METHODS

Study Design, Setting and Study Population

The present study was observational, prospective and descriptive; and was carried out in Narayana Hrudayalaya-Malla Reddy Hospital in Hyderabad, Telangana, India and the study was conducted in between March 2014 and March 2016. One hundred and nine patients [n=109] were collected who were with suffering from an Alcoholic liver disease**.**

Inclusion Criteria

All the patients were considered based on their disease condition (ALD). The patients were kept on alcohol withdrawal therapy and they were advised to come for regular checkup, for measuring alcohol withdrawal assessment scores (CIWA-Clinical institute withdrawal assessment for alcohol)**.**

Exclusion Criteria

Pediatric, Gynecology was excluded in this study.

Questionners Based Session

Alcohol withdrawal scoring.

Data Collection

Medical case sheets, drug charts, and their laboratory investigations were recorded and were analyzed. Demographics [Age, Sex], Chief complaints, Medical History, Medication prescribed [Dose, Route of administration, Frequency, Indication, Therapy duration, Marketing categories [Generic/Branded] were collected.

Ethical Considerations

The study was done using WHO guidelines only after obtaining approval from institutional research and ethics committee.

RESULT

Table 2: Gender distributions

Gender	No. of patients	Percentage
Male	96	88%
Female	13	12%

Table 3

	No. of Patients	Percentage
Acute	16	14.65
Chronic	93	85.3%

Table 4: Age distribution

Age (years)	No. of Patients	Percentage
Below 25	19	17.4%
25-50	24	22%
51-75	48	44%
Above 75	18	16.6%

Table 5: Disease condition

Disease	No. of patients
1. Fatty liver	9
2. Acute alcoholic hepatitis	16
3. Chronic alcoholic hepatitis	7
4. Liver fibrosis	46
5. Alcoholic/Hepatic Cirrhosis	24
6. Hepatic Steatosis	2
7. Portal triaditis	3
8. Congestive liver	2

Table 6: Liver weights

Liver size	Male	Female
1.<999 gms	31	3
2.1000-1500 gms	52	10
3.1501-2000 gms	9	-
4.2001-2500 gms	4	-

Table 7: Liver function test (LFT's) score

LFT'S/Levels	Mild increase	Patients	Moderate increase	Patients	Severely increased	Patients
1.Bilirubin						
a)Total(0.0-1.4mg/dl)	>1.5	31	>2.0	65	>2.5	13
b)Direct(0.0-0.3mg/dl)	>0.5	34	>1.0	63	>1.5	12
c)Indirect(0.2-1.2mg/dl)	>1.5	31	>2.0	62	>2.5	16
2.Gamma glutamyl transferase*(N=0-55U/L)	56-65	59	66-75	43	>77	7
3.Aspartate aminotransferase (N=10-50 IU/L)	51-75	29	75-100	68	>100	12
4.Alanine aminotransferase (N=5-42 IU/L)	45-65	30	65-85	79	>85	-
5.Alkaline phosphate (N=44-147IU/L)	150-169	42	170-189	-	>190	-

Table 8: Alcohol intakes per week

Risk levels in Men	No. of male patients	Risk levels in Women's	No. of female patients
Low risk(15-29 drinks)	69	Low risk (8-24drinks)	13
Medium risk(30-49 drinks)	36	Medium risk (25-39drinks)	
High risk(50-79drinks)	4	High risk (40-59drinks)	
Very high risk(80+drinks)	-	Very high risk (60 + drinks)	

Table 9: Duration of alcohol intake in years

Duration in years	No. of patients
5years	9
5-10 years	16
10-15years	7
15-20years	53
>20years	24

Table 10: Alcohol withdrawal therapy Score (Clinical institute withdrawal assessment)

CIWA score	Severity levels	No. of patients
1) Less than 10	Mild	4
2) 10-20	Moderate	72
3) >21	Severe	33

DISCUSSION

In our study population number of patients (n=109) taken, who were put on alcohol withdrawal therapy after alcohol cessation plan. Questionnaire based session with 10 scaled questions were framed according to CIWA (assessment and management of alcohol withdrawal) and the score was noted with their symptoms occurrence after the alcohol cessation plan. CIWA score with moderate severity were found to be highest. 7 patients out of 33 patients in severe category of CIWA score were admitted in the hospital with alcohol withdrawal syndrome and psychological disturbances.

CONCLUSION

From this study, it may be concluded that Cirrhosis, Chronic hepatitis, and Steatosis are the common liver diseases in chronic alcoholic patients. These diseases are more commonly seen in healthy individuals and if it is not detected early some of these conditions may lead to a serious outcome in which it needs liver transplantation. Hence steps should be taken for the early detection and treatment with alcohol withdrawal therapy. Clinical Institute Withdrawal Assessment of Alcohol Scale (CIWA) helps clinicians assess and treat potential alcohol withdrawal.

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