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RESEARCH ARTICLE

MANAGEMENT OF ALCOHOLIC HEPATITIS THROUGH UNANI MEDICINE - A CASE STUDY

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ABSTRACT

The cases of Alcoholic Hepatitis are increasing day by day due to drinking alcohol habit in our society. Amale patient of age 56 years working as DGM in NTPC came with the complaint of abdominal heaviness, indigestion, alternate diarrhea and constipation, decreased appetite, nausea, tiredness and general weakness for six months. Patient was diagnosed a case of Alcoholic Hepatitis after taking history and required investigations specially LFT, USG W/A and Fibroscan. Patient was planned and treated by combination of Unani medicines formulations as described in texts of Unani system of medicine. With this unani treatment fibrosis of liver is decreased to normal level without producing any complications and side effects.

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INTRODUCTION

Alcohol consumption accounts for an estimated 3.8% mortality globally. In 1990, alcohol accounted for 3.5% of the global burden of disease, whereas tobacco accounted for 2.6%. (Rehm *et al.,* 2009). In 2003, 44% of all deaths from liver disease were attributed to alcohol (Yoon and Yi, 2007). Alcoholic liver disease (ALD) describes a spectrum of conditions ranging from reversible fatty liver to alcoholic hepatitis (AH), cirrhosis, and hepatocellular carcinoma (HCC). Alcoholic hepatitis is a distinct clinical syndrome caused by chronic alcohol abuse and carries a particularly poor prognosis with a 28-day mortality ranging from 30% to 50% (Maddrey *et al.,* 1978). Although alcoholic hepatitis is an acute condition, nearly 50% of patients with alcoholic hepatitis have established cirrhosis at the time of clinical presentation (O'Shea *et al.,* 2010).

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In alcoholic hepatitis patients several pro-inflammatory cytokines have been detected. In uncomplicated cases, histology of alcoholic hepatitis is characterized by neutrophilic infiltration (a marker of alcohol-induced hepatitis), ballooning degeneration of hepatocytes, spotty necrosis and fibrosis in the perivenular and perisinusoidal space of Disse (''chicken wire'' fibrosis), and Mallory hyaline inclusions (Purohit and Russo, 2002). The liver is the second largest organ in the body located on the right side of the body under the rib cage. The liver is responsible for processing what people eat and drink into nutrients that can be used readily by the body and also responsible for removing harmful substances from the blood. Alcohol can damage and even destroy the body's liver cells. The liver breaks down alcohol so that it can be removed from the body. Heavy drinking and its consequences have a significant impact on public health. 5% percent of the deaths occurring annually in the United States (approximately 100 000 per year) are either directly or indirectly attributable to alcohol abuse (Hoofnagle *et al.,* 1997). In 1994, approximately 7.4% of adult Americans met the DSM-IV criteria for the diagnosis of alcohol abuse and/or alcohol dependence (Grant *et*

al., 1994). More recent data suggest that 4.6% meet criteria for alcohol abuse and 3.8% for alcohol dependence (Grant *et al.,* 2006). The main cause of alcoholic hepatitis is excessive drinking over an extended period of time. Poisonous chemicals released by the breakdown of alcohol cause inflammation that can destroy liver cells. Irreversible scarring or cirrhosis is the final stage of alcoholic liver disease. Some factors can contribute to alcoholic hepatitis. People with other types of hepatitis are at an increased risk and should not drink.

Severe alcoholic hepatitis can occur without warning. This can lead to serious complications such as liver failure and even death. The signs and symptoms of severe alcoholic hepatitis include: Buildup of fluid in the upper body, confusion and behavior changes caused by a buildup of poisons in the body that are normally broken down and removed by the liver, liver and kidney failure. Signs and symptoms vary from person to person. It can also change depending on the severity of the disease and after recent periods of heavy drinking.

Table 1. Comparison of clinical symptoms, lab investigations & Imaging technique

		Before Tt	After Tt			
		0 Day	**1 Month**	**2 Month**	**3 Month**	**6 Month**
1.Clinical Symptoms& Sign	Yellow coloration of urine, skin, sclera	+++	++	++	+	_
	Appetite	↓	↓	↓	↑	↑
	Nausea & vomiting	present	present	Absent	Absent	Absent
	Pain in upper abdomen	present	present	present	Absent	Absent
	Decreased weight	±	±	±	↑	↑
	Abdominal Swelling(Ascitis)	±	±	±	↓	↓
	Fatigue & Weakness	±	±	±	Absent	Absent
2.Lab Investigation	Hb gm%	11	_	_	_	11.6
	TLC	8100	_	_	_	7900
	ESR	62	_	_	_	15
	S.Bilirubin T	8.9	_	_	_	0.37
	SGOT	120	_	_	_	25
	SGPT	132	_	_	_	27
	SAP	164	_	_	_	88
	B. Urea	24	_	_	_	23
	S. Creatinine	0.9	_	_	_	1
	B Sugar F	140	_	_	_	107
	HbA1c	7.4	_	_	_	5.8
	TSH	2.5	_	_	_	1.79
3.Imaging Technique	X-Ray Chest PA view	WNL	_	_	_	_
	USG	Alcoholic hepatitis with hepatomegaly	Not done	Not done	Not done	Not significant abnormality
	Fibroscan	Ekpa M 27,7	-	Ekpa M 11,6	Ekpa M 11,1	Ekpa M 8,1

Inadequate absorption of nutrients from the intestine can also be a problem for this. Most people who suffer from alcoholic hepatitis are malnourished because drinking significant amounts of alcohol suppresses the appetite. As a result, most heavy drinkers get the majority of their daily calories from alcohol. Malnutrition can also contribute to liver disease. Other risk factors include:

Sex: Women may have a higher risk of developing alcoholic hepatitis, Obesity, Genetic factors, Race and ethnicity: African American and Hispanics may be at higher risk of developing alcoholic hepatitis. Binge drinking: Consuming five or more alcoholic drinks at one time can increase the risk of alcoholic hepatitis

Blood tests to determine alcohol hepatitis include: liver studies, cellular blood counts, bleeding times, electrolyte tests, tests for other chemicals in the body. An ultrasound whole abdomen, CT-scan of the liver is also used to show a more detailed view.

Fibro scan of liver to show the stiffness of liver. If other tests fail to provide a clear answer, a liver biopsy may be carried out. One of the most common signs of alcoholic hepatitis is jaundice, which causes yellowing of the skin and eyes. Additional symptoms that can occur include: Loss of appetite, nausea, vomiting, abdominal pain, fever, tiredness and weakness, weight loss. Alcoholic hepatitis is generally defined as either mild or severe. Mild alcoholic hepatitis can sometimes be reversed by giving up alcohol.

CASE STUDY

A 56 year old man, with complaints of yellowing of the skin and eyes, Loss of appetite, nausea, vomiting, stomach pain, tiredness and weakness, weight loss for six months and came to the family physician first time on April 17, advised allopathic medicine almost 2 months but no improvement was noted. Therefore the family physician referred this patient to higher centre (AIIMS). After 6 weeks of treatment the patient was suggested for liver transplantation. The patient and attendant became increasingly concerned as the condition is not improving. After suggesting liver transplantation patient movement has gone to traditional system of medicine, and ultimately came to the OPD of Majeedia Unani Hospital, SUMER, Jamia Hamdard under my direct supervision. He was also known case of Diabetes Mellitus with hypertension and was on treatment (Tab Janumet 50/1000 1 BD, Tab TWIN-TEL 40 1OD, Cap Urimax 1OD. His general health was poor but vital signs were stable at the time of visit in OPD and laboratory parameters were as follows: haemoglobin (Hb) 11.0 gm%, Total Leukocytes Count (TLC) 8100/µl, neutrophils 69%, lymphocytes 24%, eosinophils 7%, red blood cells (RBC_S) 4.05 million cells/µl, Platelets 1.75 lacs/Cumm, erythrocyte sedimentation rate (ESR) 62 mm/hr. Blood sugar (F) 140 mg/dl, Blood sugar (PP) 181 mg/dl, HbA1c 7.4, serum bilirubin total 8.9mg/dl, SGOT 120 IU/L, SGPT 132 IU/L, SAP 164 IU/L. Serum protein total 7.9, Serum cholesterol total 170 mg/dl, serum triglyceride 140 mg/dl, HDL 44 mg/dl, blood urea 24mg/dl, serum creatinine 0.9 mg/dl, serum uric acid 7.2 mg/dl, TSH 2.5 mIU/L.

X-Ray chest PA view shows normal study. Ultrasonography whole abdomen shows: Alcoholic Hepatitis with splenomegaly. The first Fibroscan of liver was Ekpa M 27.7on 15.07.2017.

Treatment method: After diagnosing the alcoholic hepatitis, patient is advised to stop the alcohol consumption in any form. Unani treatment (started from 27 July 2017) given in this case consisted of:

1) Dawa-e-Mujarrab50ml in the morning before breakfast,
2) Heptagreen 50ml in the evening.
3) A mixture of aqueous extracts of Mako (*Solanumnigrum*) 50 ml + Kasni (*Cichoriumintybus*) 50 ml + Birinjasif (*Achilleamillefolium*) 50 ml twice daily.
4) Cap Jigreena two tablets twice daily
5) Cap D. W. 2BD in the morning.
6) Q. Podina. 2BD twice daily after meal.

Patient was also advised to continue anti-diabetic and anti-hypertensive treatment because patient was known case of DM with HTN and was on allopathic treatment.

Patient was also advised for:

1. Regimental therapy (*Ilaj-bil-Tadbeer*) like walking and exercise.
2. Diet: Fat free diet.
3. Alcohol (stopped)

RESULTS AND DISCUSSION

Some improvement in all symptoms was noted only taking one month treatment and advised to continue all medicines and come in OPD for follow up time to time. The second fibroscan of liver was Ekpa M 11.6on 21.10.2017. The third fibroscan of liver was Ekpa M 11.1on 20.01.2018. The fourth fibroscan of liver was Ekpa M 8.1on 21.04.2018. The second time LFT were as follows: serum bilirubin total 3.0 mg/dl, SGOT 90 IU/L, SGPT 82 IU/L, SAP 114 IU/L. Laboratory parameters after taking treatment were as follows: haemoglobin (Hb) 11.6 gm%, Total Leukocytes Count (TLC) 7900/µl, neutrophils 68%, lymphocytes 26%, eosinophils 6%, red blood cells (RBC_S) 4.21million cells/µl, Platelets 1.68 lacs/Cumm, erythrocyte sedimentation rate (ESR) 15 mm/hr. Blood sugar (F) 107 mg/dl, Blood sugar (PP) 151 mg/dl, HbA1c 5.8, serum bilirubin total 0.37mg/dl, SGOT 25 IU/L, SGPT 27 IU/L, SAP 88 IU/L. Serum protein total 7.8, Serum cholesterol total 143 mg/dl, serum triglyceride 127 mg/dl, HDL 43 mg/dl, blood urea 23mg/dl, serum creatinine 1.0 mg/dl, serum uric acid 7.0 mg/dl, TSH1.79µIU/L. Ultrasonography whole abdomen shows: No significant abnormality.

After completing the six months of Unani treatment yellow colouration of the skin and sclera diminished, appetite is increased, Nausea and Vomiting is subsided, pain in upper abdomen, weight is increased (Table 1).

CONCLUSION

This case study was concluded that Unani treatment was beneficial for Alcoholic Hepatitis because it normalize the LFT from deranged LFT. Liver is decreased in size and inflammation due to alcohol is subsided. The median stiffness is decreased to the normal level from 27.7 Ekpa M. Significant improvement was noted in all symptoms after six months of treatment. This Unani treatment can be proposed for alcoholic hepatitis cure on the basis of above such observation.

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