Gastrintestinal Manifestation and Alcoholic Liver Disease

Aswad Al Obeidy (FICMS) *

Summary:

Background: *Alcohol remains the single most significant cause of liver disease throughout the Western World, responsible for between 40 and 80% of cases of cirrhosis in different countries. Many of the factors underlying the development of alcoholic liver injury remain unknown, and significant questions remain about the value of even very basic therapeutic strategies.*

Patients and Methods: *A total number of 113 patients with ALD attending the Gastroenterology and Hepatology teaching hospital between December 2001 and December 2003 were studied for the gastrointestinal, esophagogastroduodenoscopic manifestation of alcoholic liver disease.*

Results: *The most common presenting symptom was jaundice (62.8%), anorexia, weight loss (39.8-54%) followed by hematemesis and malena (46.9%) and encephalopathy (40.7%). The pattern of the bowel motion was predominantly diarrhea (47.8%), constipation (16.8%). The most common physical finding was hepatomegaly in (70.8%) followed by jaundice and ascites. The most common EGD finding was esophageal varieses in (77%), PHT gastropathy (52%), hemorrhagic gastritis (10.6%), GERD and lax cardia (8.8-15%) and esophageal candidiasis (4.4%).*

Conclusion: *In Iraqi patients with ALD, the most common clinical manifestations were jaundice and hepatomegaly. The pattern of the bowel motion was predominantly diarrhea. The most common EGD finding was esophageal varieses, hemorrhagic gastritis was prevalent and esophageal candidiasis may be a manifestation.*

***Key* wards:** Alcoholic liver disease, hemorrhagic gastritis, diarrhea.

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Introduction

The association of alcohol with cirrhosis was recognized by Matthew Baillie in 1793. The incidence of cirrhosis among alcoholics is about 10-15 %(1) The liver injury requires 80 gm of ethanol daily for 10-20yrs(2). There are currently four major theories concerning the mechanism by which alcohol damage the liver:(1) Centrilobular hypoxia (3). (2) Neutrophil infiltration and activation (4). (3) Inflammatory cell infiltration and activation.(5) (4) Antigenic adduct formation. (6).

The laboratory parameters that are most useful in predicting the severity of alcoholic liver injury are bilirubin level, prothrombin time (PT), and albumin level. The first two have been used to formulate a discrimination function (DF), defined as 4.6 x (PT-control in second)+bilirubin (mg/dL); when the result is greater than 32, a mortality rate of 50% can be predicted within one month (7). The mainstays of treatment for ALD are: 1.Abstinence.2. Nutritional Supplement.

* *Gastroenterology & Hepatology Teaching Hospital, Baghdad, Iraq*

3.Antiinflammatorydrugs.(Glucocorticoids)(8).
4.Antioxidants. 5.LiverTransplantat The prognosis of patient with alcoholic liver disease (ALD) depend upon several variables including: The clinical severity of liver injury at diagnosis, the extent of irreversible liver damage at diagnosis and the subsequent drinking behavior. Patient with fatty liver or equivalent have had the best outcome (70% to 80% survival rate at 4 to 5 years); those with alcoholic hepatitis or cirrhosis, an intermediate outcome (50% to 75% survival rate at 4 to 5 years); and those with cirrhosis combined with alcoholic hepatitis, the worst outcome (30% to 50% survival rate at 4 to 5 years) (9).

Patients and Methods:

A total number of 113 patients with ALD attending the Gastroenterology and Hepatology Teaching Hospital between December 2001 and December 2003 were studied. The inclusion criteria were consumption 80 grams of ethanol or its equivalent for 10-20 years with evidence of liver disease with absence of another etiology. Each patient was interviewed, detailed history, general medical examination was done and a study protocol paper was filled. Esophagogastroduodonoscopy (EGD), abdominal ultrasonography and blood sample were taken for liver function, serum iron, total iron binding capacity, serum ferritin, copper,

cerolop!asmin, lipid, hematological and coagulation profile, virological and immune markers.

The aim of the study was to determine the gastrointestinal manifestation, the pattern of the bowel motion and esophagoduodenogastric (EGD) finding of alcoholic liver disease.

Results:

Of the 113 alcoholic patients attending the Gastroenterology and Hepatolgy Teaching Hospital between December 2001 and December 2003. The most common presenting symptom was jaundice (62.8%), anorexia, weight loss (39.8-54%) followed by hematemesis and malena (46.9%) and encephalopathy (40.7%). The pattern of the bowel motion was predominantly diarrhea (47.8%), constipation (16.8%). Fever in the absence of demonstrable infection in (16.8%) as shown in table (1).

Moderate disease was defined by bilirubin level >5mg/dl and severe disease by bilirubin level >5mg/dl and PT>4 seconds prolonged.

The most common physical finding was hepatomegaly in (70.8%) followed by jaundice and ascites in (62.8%) as shown in table (2).

Table 1: Symptoms of ALD

	Group 1 (n=43) Mild		Group 2 (n=26) Moderate		Group 3 (n=44) Severe		Overall (n=113)	
Symptoms	N	%	N	%	N	%	N	%
Hematemesis and Malena	16	37.2	12	46.2	25	56.8	53	46.9
Anorexia	20	46.5	15	57.7	26	59.1	61	54.0
Weight loss	17	39.5	8	30.8	12	27.3	45	39.8
Fever	6	14.0	6	23.1	7	15.9	19	16.8
Encephalopathy	5	11.6	10	38.5	27	61.4	46	40.7
Diarrhea	17	39.5	11	42.3	27	61.4	54	47.8
Constipation	9	20.9	3	11.5	10	22.7	19	16.8

Table 2: Signs of ALD

	Group 1 (n=43) Mild		Group 2 (n=26) Moderate		Group 3 (n=44) Severe		Overall (n=113)	
Signs	N	%	N	%	N	%	N	%
Hepatomegally	31	72.1	21	80.8	26	59.1	80	70.8
Jaundice	22	51.2	17	65.4	32	72.7	71	62.8
Ascites	18	41.9	17	65.4	36	81.8	71	62.8
Splenomegally	17	39.5	10	38.5	32	72.7	57	50.4
Spider naevi	10	23.3	8	30.8	18	40.9	37	32.7
Palmar erythema	12	27.9	11	42.3	21	47.7	45	39.8
Testicular atrophy	4	9.3	3	11.5	7	15.9	14	12.4
Dupuyteren contracture	5	11.6	4	15.4	8	18.2	17	15.0
Gynecomastia	2	4.7	2	7.7	8	18.2	11	9.7
Parotid enlargement	8	18.6	6	23.1	14	31.8	28	24.8

Table (3): EGD Finding of ALD

	Overall (n=113)	
	N	%
Esophageal varices	87	77.0
PHT gastropathy	59	52.2
Duodenal erosion	26	23.0
Peptic ulcer	17	15.0
Lax cardia	17	15.0
Gastric erosion	15	13.3
Fundal varices	15	13.3
Hemrragic gastritis	12	10.6
Prolapse gastropathy	11	9.7
GERD	10	8.8
Esophageal candidiasis	5	4.4

The most common EGD finding was esophageal varieses in (77%), PHT gastropathy (52%), hemorrhagic gastritis (10.6%), GERD and lax cardia (8.8-15%) and esophageal candidiasis (4.4%) as in table (3).

Discussion:

In this study the most common clinical manifestations of ALD were jaundice (62.8%) and hepatomegaly (70.8%). Liver enlargement can be detected in more than 75% of patients who are actively drinking and observed consistently at all stages of liver injury. Hepatomegaly is related in part to the accumulation of fat within liver cells. Hepatocytes swelling, rather than steatosis is believed to the major cause of liver enlargement in moderately to severe ALD. (10, 11) The frequency of jaundice increased as the disease progresses. (12) Unusually large proportion exhibit hepatic encephalophathy (40.7%) even in patients with mild disease (11.6%). Chedid and colleagues have confirmed this finding: They reported a 19% frequency of encephalophathy and a 28% frequency of PHT in patients whose liver biopsy results indicated only fatty liver. (13) PHT has been reported in patients with hepatic steatosis (14) and may be related to compression of the hepatic sinusoids by enlarged hepatocytes. (15) Fever in the absence of demonstrable infection in 17% suggests a possible contribution by ethanol-induced cytokines. (16)

The pattern of the bowel was predominantly diarrhea (47.8%). Diarrhea with steatorrhea can be related to decreased biliary excretion of bile salts, pancreatic insufficiency and to a direct, toxic effect of alcohol on the intestinal mucosa, reversible intestinal abnormalities in alcoholics include malabsorption of folic acid, thiamine, nitrogen, B12 and B2.Decreased output of HCO3, amylase thiamin, and chemotrypsin in response to secretin. (17) The most common OGD finding was esophageal varieces (77%), PHT gastropathy (52.2%) followed by duodenal erosion (23%), hemorrhagic gastritis (10.6%), GERD, lax cardia (8.8-15%) and esophageal candidiasis (4.4%). Acute and chronic alcohol intake produces both mucosal damage and motor dysfunction in gastrointestinal tract. Esophagitis and gastritis are common features of both acute and chronic excessive alcohol intake. Motor abnormalities include a reduction in lower esophageal sphincter pressure and decreased esophageal peristalsis. Alterations in small intestinal function also occur with mucosal changes to duodenal and jejunal villi and these may lead to a mild degree of malabsorption. (18) *H pylori* infection and gastritis can reduce gastric ADH activity; whether they promote the development of ALD is not known.. (19)

In conclusion: In Iraqi patients with ALD, the most common clinical manifestations were jaundice and hepatomegaly. The pattern of the bowel motion was predominantly diarrhea. The most common EGD finding was esophageal varieses, hemorrhagic gastritis was prevalent and esophageal candidiasis may be a manifestation.

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