Alcohol abuse and the digestive system

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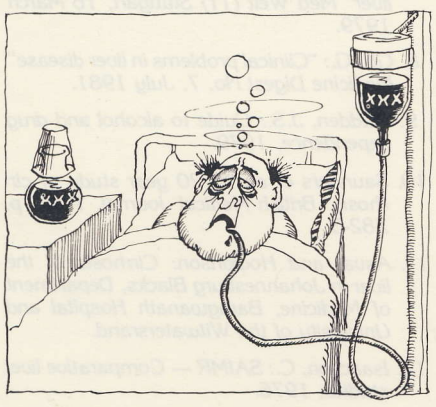
Alcohol abuse is an important cause of disease in the gastro-intestinal tract, with the liver and pancreas at particular risk.

I speak as a Clinician, not as a Pathologist, and have been for many years closely associated both with the illness and with the alcoholic in my clinical practice in the Primary Care situation.

There, one sees at first hand, by meeting "The hidden alcoholic in our midst" (1), the ravages of acute and chronic alcohol abuse, and hears the first faltering appeal of the alcoholic victim for help.

At present I am witnessing daily the emergence of "the White man's alcoholic problem" (2) in the urban Black patient.

The most frequent primary symptoms of the alcohol abuser are upper abdominal pain — with or without vomiting. This, of course, is not surprising, since alcohol enters the body by the mouth, and has to pass through the mouth, pharynx and



oesophagus before it reaches the stomach from where it is absorbed into the whole system.

Alcohol is a powerful pharmacological agent and an irritant to all those mucous membranes which respond by becoming overactive and thickened giving the clinical picture of chronic pharyngitis and oesophagitis which we see so often.

In the stomach itself, strong liquor inhibits gastric secretion except for mucous, and produces the well known inflammatory changes of "gastric catarrh" or gastritis — the usual cause of "morning sickness" in males!

On the other hand, the weaker alcoholic beverages stimulate gastric secretion, and precipitate gastric erosion and ulceration, as well as gastric bleeding in susceptible people.

More than 20% of patients with established histories of alcohol abuse develop peptic ulceration, and may present with perforation or haematemesis (3).

This should always be borne in mind in dealing with such cases, and the drinking history properly delineated before surgery.

In simple words, the stomach rebels at the repeated deluges of alcohol descending on it and reacts by the phenomena of gastritic and peptic ulceration which lead to a distaste for food (4).

The drinker ceases to eat properly, and then the resultant complications of undernourishment and vitamin deficiency both add their weight to the already problematic situation.

In addition, even if the alcohol abuser does eat meals of an adequate nutritional content, the malfunctioning digestive tract cannot absorb many of the essential nutrients.

Vitamin B — the whole complex — is one of these nutrients, and as alcohol increases the body's need for Vitamin B, while itself only supplying rapidly expendible energy, deficiencies soon manifest themselves and we see the angular stomatitis, and the scaling of the lower lip, the buccal oedema, the "raw beef" and later atrophic tongue — all well known clinical entities, diagnostic of **Pellagra** (5).

The stomach absorbs alcohol totally **and rapidly** because of the blood-vessel dilatation induced by the irritant effect — (well known socially as that lovely feeling of warmth — "the warming of the cockles of the heart"!) so the next organ to be deluged with alcohol is the liver.

The Liver is responsible for the metabolism of most of the alcohol delivered to it from the gut. This obligatory oxidation process of such a substantial metabolic load is accomplished by the liver enzymes at the expense of other cellular metabolic requirements, and thus the general body lipid and CHO metabolism is disturbed and the oxidation of fatty acids inhibited, leading to a build up of FAT in the liver cells — **the fatty infiltration of liver** common to all heavy drinkers.

The "fatty liver" patient may not appear to be ill, but the liver is always moderately enlarged and tender (6).

At this stage, the changes are reversible and abstinence from alcohol can be a life-saving measure, but if the drinking continues, a **fatty liver hepatitis** develops which is the tone precursor of cirrhosis of the liver — which is a fatal disease (7).

Acute Alcoholic Hepatitis occurs frequently on or around pay day in the urban areas with alarming frequency. Alcohol is second only to the virulent viruses as a cause of this potentially lethal condition (8).

It may be a mild illness with slight jaundice, general malaise, enlarged liver and disordered liver function

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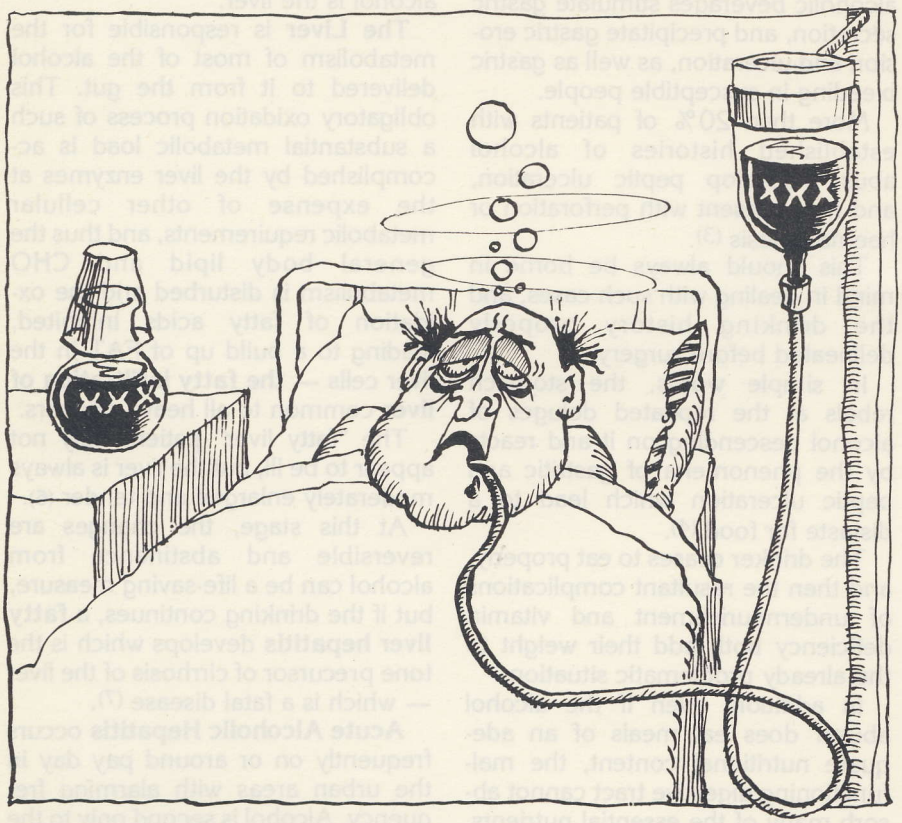
tests (especially the Transaminases) or it can be severe and fatal, with cellular necrosis, and the clinical presentation of enlarged and acutely tender liver, with fever, leucocytosis, jaundice and even liver failure, and signs of portal hypertension (9).

In 1976 the SAIMR (JHM) reported that at autopsy the livers of Blacks were now riddled with fatty change, in place of the high Iron (Fe) content of the pre 1962 livers (12).

This undoubtedly will lead to an increasing incidence of cirrhosis in the future.

Cirrhosis of the liver is a late result of alcohol abuse, and one report estimates that at least 2/3 of the patients presently attending hospitals with cirrhosis, owe their uncomfortable invalidism to alcohol taken in excess in earlier years.

It may be a finely nodular "portal cirrhosis", or a coarsely nodular post necrotic cirrhosis; both types may present at routine examinations, or in patients undergoing treatment for alcoholism.



It mostly enters the Primary Care scene with jaundice, portal hypertension, ascites or oedema (the latter being the presenting symptom in 10 - 15% of cases), and neuro-psychiatric phenomena (drowsiness, slurred speech and flapping tremor).

There is total gastro-intestinal tract involvement with anorexia, nausea vomiting, gastric and oesophageal (varices) bleeding, diarrhoea and generalised abdominal pain and tenderness.

The prognosis is very poor, and the most frequent cause of death is from gastro-intestinal massive haemorrhage. Even in the late stages complete abstention from alcohol improves the survival rate.

A recent study showed that alcohol is the commonest cause of cirrhosis in the livers of Johannesburg Blacks (11), and again an SAIMR survey found that 1/3 of all White alcoholics show signs of **primary liver cancer** at autopsy (12).

Further, **Pancreatitis,** both acute, and chronic is yet another consequence of alcoholic abuse. Its incidence is becoming increasingly common, and in South Africa alcoholism is now recognised as the commonest cause of acute pancreatitis (13). Alcohol-induced pancreatitis is an inflammatory process which leads to cell necrosis; the patient is **ill** with severe upper abdominal pain, nausea and vomiting, fever and jaundice, and often a glycosuria (5).

These acute abdominal symptoms often lead to emergency surgery being performed which may well be fatal.

Recurrent attacks after drinking bouts lead to calcification within the pancreas, or cyst formation (9). The damage leads to duct obstruction, and eventually to a total failure of pancreatic function with a steatorrhoea, diabetes, and often an obstructive jaundice.

In fact, a vicious cycle develops from the pancreatic failure as the consequent diabetes mellitus leads to excessive thirst (14), which in the alcoholic requires assuaging with more and more drinking!

Suffice it to say that should alcohol abuse not be arrested, either gastric, liver or pancreatic failure or cancer, or all four will ensure an early death.

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