#### **EDITORIAL**

## FATTY LIVER: YET ANOTHER SILENT KILLER

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"Ultra sound is normal apart from a fatty liver", a not uncommon ultrasound report which is frequently dismissed by the physician as "normal" or casually attributed to obesity since the word 'fatty liver' is preceded by 'ultrasound is normal'. The fact is that it may herald a condition as serious as hepatitis B or C and a condition which can as devastating as hepatitis B or C.

Fatty liver can be either alcoholic or non-alcoholic in aetiology. Worldwide prevalence of non-alcoholic fatty liver has been reported to range from 9–7% in the general population, and in the United States 30% of the general population and as high as 90% of the morbidly obese are affected by non-alcoholic fatty liver. <sup>1</sup>

The prevalence of "Fatty Liver" in Asia ranges from 12–24%<sup>2</sup> of the general population with most estimates within the range of 6–14%.<sup>3</sup> Presence of obesity, diabetes specifically type-II, high triglycerides and low HDL are considered to have strong association with non-alcoholic fatty liver. Men are more prone to get non-alcoholic fatty liver, and the risk increases as age advances. Women are particularly at risk after menopause.<sup>3</sup> Non-alcoholic fatty liver may indicate a significant metabolic problem with effects reaching outside the liver. In the liver itself cryptogenic cirrhosis (no cause attributable) is increasingly thought to be due to Non Alcoholic Steatohepatitis (NASH).

Fatty liver can simply be a non-alcoholic fatty liver disease, in which there is an accumulation of triglycerides and other lipids in liver cells. In some cases, this may be accompanied by hepatic inflammation and liver cell death. When this inflammation and liver damage is present it is known as NASH.<sup>4</sup>

Unfortunately the difference between simple non-alcoholic fatty liver and NASH is only made through a liver biopsy. An apparently well patient can hardly be motivated to undergo a traumatic procedure especially in our society.

Why does it occur? The exact aetiology is unknown. Probable mechanisms include decreased fatty acid oxidation, increased delivery of fatty acids to the liver, increased endogenous fatty acid synthesis and deficient export of triglycerides as very-low density lipoprotein from the liver. Non-alcoholic fatty liver is associated with insulin resistance. Patients are frequently obese and have features of the metabolic syndrome including

obesity, hypertension, diabetes mellitus type II and dyslipidemia.<sup>5</sup>

It is imperative for a clinician to be aware of NASH and to identifypeople with fatty liver because non-alcoholic fatty liver or NASH is usually asymptomatic and is often detected while investigating metabolic syndrome or during abdominal ultrasound advised for some other problem. Some patients may complain of vague upper abdominal discomfort, tiredness and malaise. Attention is frequently drawn towards it by coming across high alanine transaminase (ALT) levels in routine screening tests.

Confirming the diagnosis is often difficult as it may need serial liver biopsies. Patients are usually unwilling for these invasive investigations. Non-invasive investigations like the fibro-scan have largely proved inaccurate. NASH may thus remain undiagnosed and the cryptic process of fibrosis goes on undetected.<sup>4</sup> Hence the patients are usually diagnosed when overt liver changes have set in.

What to do if non-alcoholic fatty liver is picked up early? Ignoring it as something insignificant is unfortunate. Though nothing dramatic is visible in short term in such cases, a major morbidity can be averted in the future. Diabetes, ischemic cardiovascular cerebro-vascular disease, the debilitating effects of obesity along with its accompanying complications can be postponed if not averted altogether.

Some interventions are promising though it has been demonstrated that normalizing ALT has not always corresponded to reversal of histological abnormalities. Lifestyle modifications like dietary changes with low calorie low fat diet, a gradual (not crash!) weight loss in obese patients and aerobic exercise for 45 minutes, five days a week may be helpful in halting or reversing the pathological changes in liver.

Pharmaceutical agents like vitamin E, Statins, Glitazones and Metformin have all shown results though unfortunately the results have not shown the consistency to be recommended for use as standard practice. What remains beyond dispute is that diet and exercise are a very cost effective way of reducing morbidity from this largely underestimated problem.<sup>4</sup>

Non-alcoholic fatty liver in apparently normal subjects should be given due attention and life-style modifications or pharmacological therapy or both should be started so as to avoid and prevent the long-term consequences. Patient education in this regard is highly important so that they take these apparently benign changes on ultrasound as an important warning sign.

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