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Short Communication

Non Alcoholic Fatty Liver Disease and Increased Intima Media Thickness as Markers of Subclinical Atherosclerosis in Severe Obesity

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Metabolic Syndrome (MS) and overweight/obesity incidence rates are both sharply rising. According to the widely held belief, being overweight has less severe but yet comparable health effects as being obese. Throughout all age ranges and the range of minor and significant increases in body weight, the probability of death from all exerted a profound influence including both men and women [1].

Even so, obesity is a major contributor to the development of non-alcoholic fatty liver disease (NAFLD) [2], which can range from "simple" fatty liver disease (FL) to the more severe version i.e. non-alcoholic steatohepatitis (NASH), both of which are defined by a steady buildup of triglycerides in the tissue of the liver. Low-grade inflammation in adipose tissue is one of the pathways that leads to insulin resistance (IR) [3]. An key predictor of metabolic risk is the architecture of abdominal fat redistribution, namely visceral adipose tissue (VAT) and subcutaneous abdominal adipose tissue (SAAT). The most significant contributing determinant for the development of hepatic steatosis in adult obese patients is typically believed to be visceral fat, which is better linked with MS [4], however some researchers disagree [5].

For the examination of central obesity, both SAAT and VAT can be simply and frequently assessed using ultrasonography (US) [6]. Additionally, US is frequently used to identify NAFLD, with lower expenses and often without patient to radiation exposure. In the spectrum of NAFLD, it is practically difficult to differentiate clearly between FL and NASH without the need of a liver biopsy, which is not always done due to ethical and technological considerations. Because of this, we commonly refer to hepatic steatosis (HS).

Non-alcoholic fatty liver disease (NAFLD) was previously thought to have a clinically benign appearance. However, in recent years, several clinical and pathological manifestations have been associated with NAFLD [7]. In keeping with this, a strong relationship has been documented between insulin resistance (IR) and liver steatosis (LS) in obese as well as lean subjects [8]. IR/hyperinsulinemia has been shown to be a risk factor for atherosclerosis [9].

These findings have clinical relevance because endothelial dysfunction is an early step in the atherosclerotic process. In the lack of conventional atherosclerotic risk factors, it has recently been shown that IR is an independent predictor of poor endothelium-dependent vasodilation in persons who appear healthy [10].

Moreover, it is widely known that IR is a pathogenetic component of NAFLD [11]. In practice, without regard to body mass index (BMI), fat distribution, or glucose tolerance. Marchesini., *et al*. [12] shown that IR was commonly related with the development of NAFLD in a

large cohort of individuals. At present, there is some evidence for the detection of clinically evident atherosclerotic damage in subjects with NAFLD [13,14] but few data are available about a possible association between NAFLD and endothelium-dependent vasodilation [15,16], particularly in patients with obesity and severe obesity.

Since there is a direct correlation between increased IMT, conventional cardiovascular risk factors, and clinically evident cardiovascular disease (CVD), the common carotid artery's (CCA) IMT is regarded as a proxy marker of subclinical atherosclerosis [17,18]. Observational studies have shown distinct connections between carotid IMT, obesity, and abdominal adiposity [19,20].

The interrelationships of endothelial function, carotid IMT, and sonographically determined visceral obesity and liver steatosis in severely obese patients have not been well reported. Even more, biochemical pathways of such interactions have not been elucidated yet.

Disclosure Statement

The authors declare that there are no conflicts of interest.

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