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Editorial

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Non-alcoholic fatty liver disease: Another factor requiring aggressive risk management?

Non-alcoholic fatty liver disease (NAFLD) is now thought to possibly be another feature of the metabolic syndrome on the grounds of its correlation with visceral obesity, insulin resistance, hyperlipidaemia and type 2 diabetes (Marchesini et al, 2005). As it has been proposed that the metabolic syndrome is a significant predictor of cardiovascular disease, it may be hypothesised that people with NAFLD may demonstrate a greater cardiovascular risk than people without NAFLD, even at a risk that is potentially greater than that seen in people with the individual risk factors of the metabolic syndrome.

Indeed, markers of inflammation and endothelial dysfunction are increased in people with NAFLD, as is carotid artery intima–media thickness (Targher et al, 2005), when compared with matched control individuals or with patients with the metabolic syndrome but without NAFLD. Moreover, a recent study (Targher et al, 2006) has suggested that the presence of cardiovascular disease is increased in people with type 2 diabetes who have NAFLD, in association with an increased prevalence of the metabolic syndrome, as compared with people with diabetes but without NAFLD. Thus, these data firstly suggest that there is a high prevalence of NAFLD in people type 2 diabetes and secondly indicate that NAFLD may well be a manifestation of the metabolic syndrome. While strong associations have been noted between NAFLD and cardiovascular disease, causality remains to be established, as does the joint effect of NAFLD and the metabolic syndrome on cardiovascular mortality (Targher et al, 2006).

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Likewise, it needs to be established whether NAFLD makes a contribution to cardiovascular disease that is independent of its links with metabolic syndrome. Consequently, further prospective studies will be required to elucidate the long-term effects of NAFLD. It also remains to be established whether the detection of NAFLD in people with type 2 diabetes will contribute further to the assessment and management of cardiovascular risk over and above individual metabolic syndrome components.

For the diagnosis of NAFLD over and above liver function tests, it is proposed that ultrasonography is by far the commonest modality used in clinical practice and demonstrates good sensitivity and specificity, particularly in moderate and severe steatosis (Joseph et al, 1991). Studies (e.g. Saadeh et al, 2002) have suggested that more than 33 % hepatic fat infiltration on liver biopsies is optimal for ultrasound detection of steatosis, whereas sensitivity maybe reduced in those with 33 % or less. Thus, the authentication of significant NAFLD should be relatively uncomplicated in clinical practice. While longer-term prospective studies are required to draw firm conclusions, in the era of aggressive management of cardiovascular risk in people with type 2 diabetes it may well be appropriate to give consideration to NAFLD per se.

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