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What's all the fuss about fatty livers?

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With obesity hitting the headlines so called Non-alcoholic fatty liver disease or NAFLD is getting increasing attention. In fact NAFLD has become the commonest cause of chronic liver disease in children. So what does fatty liver disease mean to my child?

A minor degree of fat storage in the liver is not rare and can be found in healthy individuals and in many medical conditions. It can be a non-specific response of the liver to any change of environment, be very mild or only seen temporarily in intercurrent infections and acute weight loss. It can also be a consistent finding in some medical conditions like hepatitis C or Wilson's disease. Because it is also common in over-nutrition and obesity, the "obesity epidemic" (WHO) has led to much debate as to how relevant this excess fat storage is for the liver.

It is important to understand that the liver responds differently to excess fat storage in different individuals and hence different forms of such "fatty livers" exist. The definitions for the different forms of fatty livers are based on the examination of liver tissue by a pathologist: The

term "(simple) steatosis" describes an excess of fat in the liver. Because a little bit of fat is quite common the term is only used if the pathologist sees more than 5% of liver cells with fat droplets. The degree of steatosis is graded in mild, (< 30% of the liver cells), moderate, (30-60% of the liver cells), and severe, (> 60% of the liver cells) steatosis. In obesity livers can respond to this fat challenge with inflammation or scarring and the pathologist can find additional microscopic features that are otherwise known to occur from toxic damage by alcohol. Hence the term "Non-alcoholic fatty liver disease" was coined to account for these changes in the tissue that are suggestive of alcoholic damage but in fact due to obesity.

NAFLD spans a whole spectrum of microscopic findings ranging from mild steatosis to inflammation or "degeneration" (sick looking cells) to varying degrees of "fibrosis" (scarring) (Fig. 1). If degeneration or fibrosis are present in the microscopic examination this can be taken as a sign of aggressive liver disease that may progress to end stage liver disease and cirrhosis at some stage. For

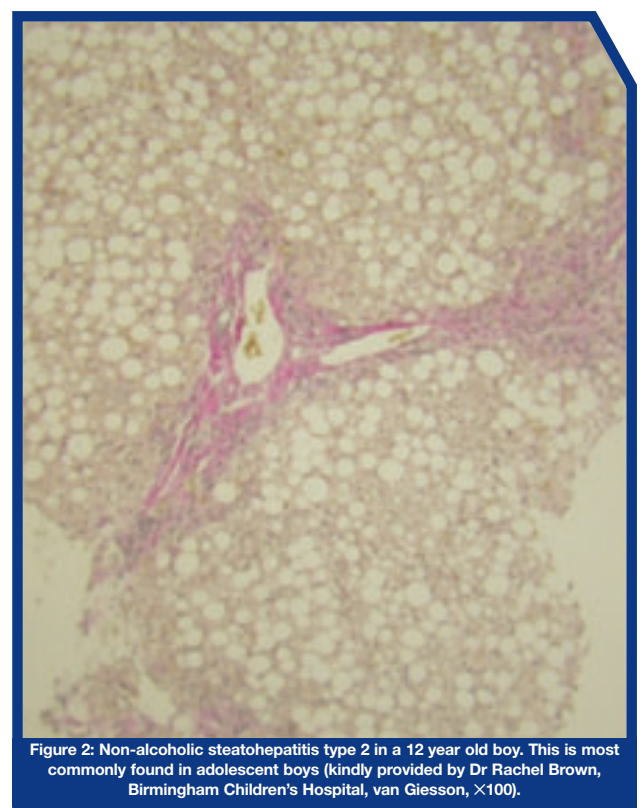
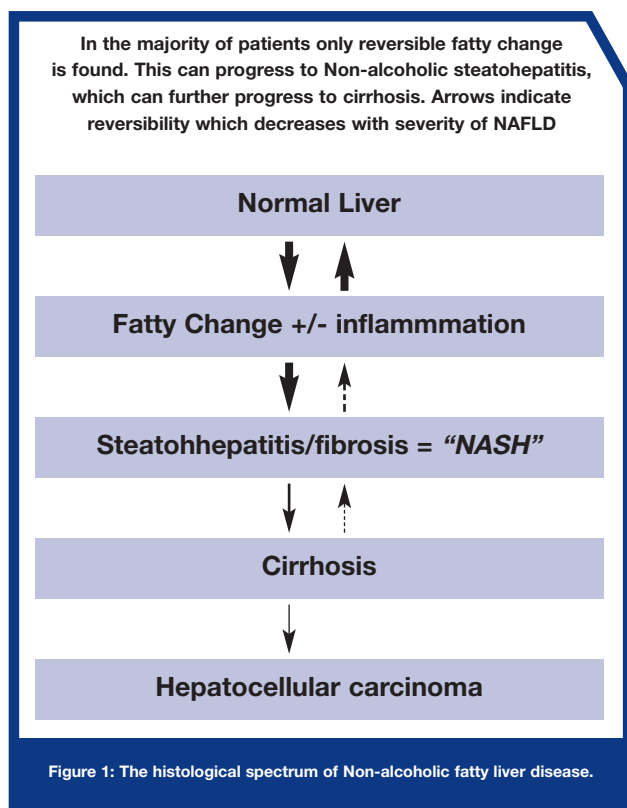
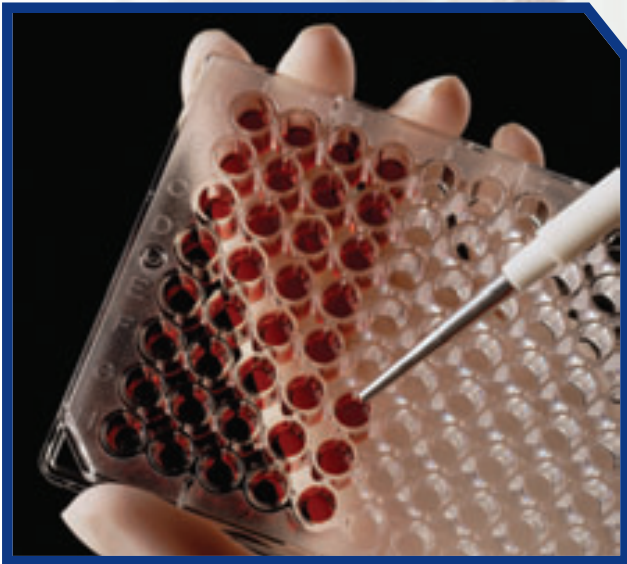


Figure 2: Non-alcoholic steatohepatitis type 2 in a 12 year old boy. This is most commonly found in adolescent boys (kindly provided by Dr Rachel Brown, Birmingham Children's Hospital, van Giesson, X100).



this aggressive subgroup of NAFLD the term non-alcoholic steatohepatitis (NASH) is used. Only few children with NASH and established liver cirrhosis have been described in the world literature and this extreme end of the spectrum is probably very rare in childhood or adolescence and may only become a clinical problem in early adulthood. However we do not yet know how rapidly the condition progresses in children and adolescents because NASH in children and adults can behave differently. The microscopic findings in children and adults vary and this has led to a further classification into NASH type 1 (adult form) and NASH type 2 which is the form often seen in children and adolescents. (Fig. 2).

We still do not know exactly how frequent NAFLD is. An ultrasound scan will often suggest a degree of fat in the liver and some clinicians feel that if one looks hard enough by ultrasound this can be found in every obese child. Because such ultrasound findings are so common and limited to the suggestion of steatosis, only children with abnormal liver function tests should be investigated further. Probably 10% of all obese children have abnormal liver function tests and some of these are at risk to have NASH. The risk to develop NASH rises when a body becomes generally unwell from obesity and a patient develops the so called "metabolic syndrome". In the "metabolic syndrome" the obese body with a large amount of fat stores has to produce more and more insulin to control the blood sugar, so called "insulin resistance" occurs and a pre-diabetic state develops.

Other typical features of the metabolic syndrome are high blood pressure or a fat distribution in the central areas of the body with an increased waist circumference.

Overweight or obese children with persistently abnormal liver function tests and suspected NAFLD should have a medical review and need further investigations. It is paramount to confirm a specific diagnosis for these children and treatable conditions need to be identified. Examples for conditions that can mimic NAFLD are Wilson's disease, which naturally also has extra fat storage in the liver or autoimmune hepatitis (in up to one third of all patients with NAFLD autoantibodies can be found and only further tests exclude autoimmune hepatitis). A liver biopsy is part of such an assessment and will also yield additional prognostic information such as the microscopic features of NASH or the degree of scarring. The timing of a liver biopsy can vary and depend on the initial assessment of a child. Some children with features of other medical conditions like positive autoantibodies or an enlarged spleen will need a complete assessment for chronic liver disease immediately as opposed to children without such findings. In the latter case children and families will be given lifestyle advice to lose weight or to hold the current weight and "grow out of the obesity". If this is achieved over a period of i.e. one year the liver function tests will normalise and no further investigations are necessary.

Current best therapy of NAFLD is centred on increased physical activity and healthy eating. It is important that these lifestyle changes are planned around the daily routine and are taken up by the entire family. Sometimes over-enthusiasm is exhausting and only lasts for a month or two, followed by a rebound and extra weight gain. Families need to find out which sport suits them best and learn to walk more rather than use transport. However in children dietary changes are not focussed on weight loss as this might impair growth of a developing child and would need medical guidance. It is more important to get into the habit of healthy eating and to discover the pleasures of "slow food". We need to get away from accepting crisps as staple diet and more often enjoy the "5 a day". However because this advice is so much easier for prevention of obesity than as therapy we need to continue to investigate other therapeutic options. We need studies and interventional trials and hopefully will continue to learn how to prevent NAFLD from progression to NASH and advanced liver disease.