

Obesity and Hepatic Steatosis in the Pediatric Patient

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The epidemiological transition, which has taken place worldwide during the last 30 years, has modified the nutritional profile of the population, since obesity and non-communicable diseases predominate today in the face of malnutrition and communicable diseases. Secondary alterations of metabolic and cardiovascular order have occurred at earlier ages of the population [1]. Among the complications of obesity, we mainly have resistance to insulin, diabetes mellitus, dyslipidemia, hypertension and grade [2] liver. Some of these complications can be grouped into what we now call the metabolic syndrome [2,3].

The frequency of Hepatic Steatosis (HS) is 3 to 36.9% in the general population. It is more prevalent in patients in the fourth to sixth decades of life, male, Hispanic or Metabolic Syndrome (MS). In pediatric patients, steatohepatitis is becoming the leading cause of referral to a specialist; 42% of overweight/obese Mexican schoolchildren have elevated ALT. During puberty, there is physiological resistance to insulin, which in obese puberty can trigger the development of diabetes mellitus (DM) 2/HS [4,5].

Within the definitive diagnosis it is required the accomplishment of the liver biopsy, procedure of high cost and risk. Imaging techniques are extremely important for the diagnosis, although it lacks the ability to distinguish fibrosis, but it does provide information on the presence of disease progression. Ultrasonography is defined as a good, low-risk, risk-free diagnostic method with a sensitivity of 89%, 93% specificity, for detecting fatty liver. Magnetic resonance imaging identifies severe HS, but its capacity to detect mild HS is scarce, this is of high cost and risk of radiation to the patient [6-9].

Therapeutic alternatives are aimed at modifying lifestyle, diet and the use of drugs, which together have an impact on the pathophysiology of the disease, especially in insulin resistance and metabolic syndrome [6-9].

The importance of timely detection of HS is based on avoiding a more serious affection that is nonalcoholic steatohepatitis since it causes inflammation of the liver cells as well as degrees of fibrosis and cirrhosis. Hepatic cirrhosis has been reported in 10-year-old pediatric patients [7], which tells us about the installation of fatty liver from an early age. The evolution is variable in each case being able to establish as predictors of progress in liver damage the degree of obesity and insulin resistance.

It is a priority to detect and establish risk factors in the pediatric population in order to contribute to the development of measures for the prevention and management of this disease, thereby improving the quality of life of these patients, limiting the damage in the medium and long term decrease public health costs.

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