



Metabolic Disturbances in Children with Chronic Liver Disease

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Introduction:

Liver disease results in complex pathophysiologic disturbances affecting nutrient digestion, absorption, distribution, storage, and use. This article aimed to present a classification of metabolic disturbances in chronic liver disease in children?

Materials and Methods:

In this review study databases including proquest, pubmedcentral, scinedirect, ovid, medlineplus were been searched with keyword words such as" chronic liver disease" " metabolic disorder""children" between 1999 to 2014. Finally, 8 related articles have been found.

Results:

Metabolic disorder in this population could be categorized in four set: 1)carbohydrates, 2)proteins,3) fats and 4)vitamins.

1) Carbohydrates: Children with CLD are at increased risk for fasting hypoglycemia, because the capacity for glycogen storage and gluconeogenesis is reduced as a result of abnormal hepatocyte function and loss of hepatocyte mass.

2) Proteins: The liver's capacity for plasma protein synthesis is impaired by reduced substrate availability, impaired hepatocyte function, and increased catabolism. This results in hypoalbuminemia, leading to peripheral edema and contributing to ascites. Reduced synthesis of insulin-like growth factor (IGF)-1 and its binding protein IGF-BP3 by the chronically diseased liver results in growth hormone resistance and may contribute to the poor growth observed in these children.

3) Fats: There is increased fat oxidation in children with end-stage liver disease in the fed and fasting states compared with controls, which is probably related to reduced carbohydrate availability. The increased lipolysis results in a decrease in fat stores, which may not be easily replenished in the setting of the fat malabsorption that accompanies cholestasis. Reduced bile delivery to the gut results in impaired fat emulsification, and hence digestion. The products of fat digestion are also poorly absorbed, because bile is also required for micelle formation. In various liver diseases, there may be reduced bile production by inadequately functioning hepatocytes, reduced hepatocyte excretion into the bile canaliculus (as in PFIC), or obstruction to biliary flow. The circulating bile salt pool may be depleted secondary to treatment with binding agents, such as cholestyramine, which is often prescribed for pruritus in cholestatic patients. Pancreatic insufficiency may further exacerbate fat malabsorption in certain cholestatic liver diseases. Vitamins: Fat malabsorption occurs in cholestatic disorders, and one must also consider any accompanying fat-soluble vitamin and essential fatty acid deficiencies. Breastfed infants with CLD are at high risk for vitamin D and K deficiencies.

Conclusion:

The clinician should proactively evaluate, treat, and re-evaluate response to treatment of nutritional deficiencies. Because a better nutritional state is associated with better survival before and after LT, aggressive nutritional management is an important part of the care of these children.

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