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COPPER STATUS AND INTESTINAL FAILURE ASSOCIATED LIVER DISEASE IN PATIENTS ON HOME PARENTERAL NUTRITION FOR CHRONIC INTESTINAL FAILURE

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Background and aims

Recent studies in patients with Non Alcoholic Fatty Liver Disease suggest that Cu deficiency may play a role in the development of liver steatosis, being Cu a co-factor of enzymes that neutralize oxidative stress in the liver.(1) The aim of this study is to assess Cu status in adult patients on home parenteral nutrition (HPN) for chronic intestinal failure (CIF) and its association with liver steatosis due to intestinal failure associated liver disease (IFALD).

Methods

Patients cared at the Center for Chronic Intestinal Failure of a third level hospital were included in the study from January 1, 2020 until December 31, 2021. The following were assessed: Cu daily supplementation with HPN, Cu serum concentration, ceruloplasmin, zinc, ferritin, C reactive protein (CRP). Liver steatosis was assessed by liver ultrasound and categorized as mild, moderate, severe. Statistics: median (IQR), non-parametric tests.

Results

108 patients, F 58%, age 50.7(29.2) years, Short Bowel Syndrome 67%; low serum Cu: 66%; Liver Steatosis 41% (mild: 56%, moderate: 36%, severe: 8%). No significant association was observed between Cu and ferritin or zinc, and between serum CRP and liver steatosis. Low Cu and high ferritin were associated with the presence of severe liver steatosis (p= 0.009; p=0.037, respectively).

Conclusions

In this population, a marginal biochemical Cu deficiency has been detected, despite intravenous supplementation. Cu deficiency may contribute to the development of IFALD-steatosis.

References

Vinicius S Nunes, et al. Distinct phenotype of non-alcoholic fatty liver disease in patients with low levels of free copper and of ceruloplasmin. Arq Gastroenterol Jul-Sep 2020;57(3):249-253.