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Opinion Article

Energy Malnutrition Indicators in Chronic Liver Disease Patients

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DESCRIPTION

PEM is a negative prognosis factor in chronic liver illness and has been linked to sarcopenia, hepato-renal syndrome, esophageal gastric varicose vein rupture, abdominal fluid retention, and a lower Quality of Life (QOL). The Japanese Gastroenterological Society's treatment recommendations for liver cirrhosis characterize EM as having a npRQ 0.85%AC 95%, or FFA >660 Eq/L. The parameters that are used to diagnosis EM in all patients are challenging to assess, in contrast to PM, which may be diagnosed only based on albumin readings. As a result, EM is frequently misdiagnosed, especially in CLDs that are in the early stages, like chronic hepatitis and Child-Pugh cirrhosis. Indirect calorimetry measurement of the npRQ is a well-established technique for EM diagnosis. Due to increased energy consumption while at rest and hepatic atrophy, EM is linked to a decrease in glycogen storage in cirrhotic patients. During the early morning fasting period, the fat ratio rises while the sugar ratio falls. Reduced carbohydrate utilization efficiency is a result of increased insulin resistance as well as elevated blood levels of glucagon, catecholamines, and cortisol. %AC is a technique for assessing muscle mass based on body measurements and is used as a nutritional assessment parameter. In old age people, %AC is associated with measures of the total body's skeletal muscle mass made by dual-energy X-ray absorptiometry. For healthy elderly persons, a decline in AC is a sign of bad prognosis, while among older Japanese people; a decline in AC over time is linked to a decline in daily activities.

FFA makes up around 5% of all lipids, and its blood levels are controlled by liver uptake through the activity of the enzymes hormone-sensitive lipase and Lipoprotein Lipase (LPL). Plasma FFA levels and npRQ were associated (r=0.39, P 0.001), and 660 Eq/L of FFA predicted an npRQ of 0.85. FFA levels rise in cirrhotic patients due to impaired liver FFA processing and increased LPL activity. In cirrhotic patients, FFA is linked to the development of dementia and hepatic encephalopathy. FFA rises with fasting, smoking, ageing, growth hormone, and catecholamines, but it falls with nutritional intake, exercise, and hypoglycemic medication. Due to abnormal intestinal flora, bacterial translocation brought on by portal hypertension, and a decline in reticuloendothelial function, patients with cirrhosis are reported to have blood concentrations of cytokines that are much higher than those of non-cirrhosis patients. The primary cause of cytokine release is lymphocyte infiltration into the liver as a result of inflammation and liver injury. TNF- (Tumour Necrosis Factor), Interleukin (IL)-1, or IL-6, in particular, limit glucose oxidation and impede fat combustion and are linked to EM in cirrhotic individuals. Blood cytokine levels are anticipated to rise and the incidence of EM to rise in situations of inflammation and liver damage with high values of -GTP and transaminases. Guidelines like ESPEN and ASPEN have been proposed for the treatment of malnutrition in individuals with cirrhosis. After one month of food management, patients with EM displayed increased AC and decreased FFA. A decline in FFA and an improvement in QOL were seen in EM patients after receiving an LES. Compared to patients with an LES, cases without an LES had considerably higher FFA values in this study (453 307 vs. 278 359 Eq/L, P=0.031). The link between changes in FFA, %AC, therapeutic action, and prognosis requires more investigation. Child-Pugh grade, transaminase readings, and -GTP values were the EM predictors in patients with CLDs. This knowledge may aid in the selection of cases that are actively assessed for the existence of EM utilising precise diagnostic techniques as npRQ, %AC, and FFA.

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