

Hubungan asupan nutrisi dengan metabolisme energi dan estimasi Kebutuhan energi pada penderita sirosis hati = The relationship between nutritional intake, energy metabolism and the estimation of energy requirements of decompensated liver cirrhotic patients

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Abstrak

ABSTRAK

Tujuan: Mengetahui status metabolisme penderita SHD rawat inap di rumah sakit, dan memperoleh rumus untuk menentukan kebutuhan energi yang sesuai dengan status metabolisme penderita.

Tempat: Bagian Gizi dan Bagian Penyakit Dalam Rumah Sakit Sumber Waras, Jakarta.

Metode Penelitian didisain Cross Sectional, pada 49 subyek SHD laki-laki atau perempuan 30-80 tahun yang diseleksi secara konsekutif, dan 40 kontrol sehat yang diseleksi secara random dari karyawan RSSW. Data REE diperoleh dari hasil pemeriksaan konsumsi O₂ dan CO₂ yang dikeluarkan tubuh, dengan mempergunakan Kalorimeter Indirek. Asupan makanan dicatat selama 3 hari berturut-turut sebelum pemeriksaan REE. Data antropometri (LLA, TLLBK, KAOLA, TB, dan BB) dan pengumpulan urin dilakukan satu hari sebelum pemeriksaan REE, pengambilan darah untuk pemeriksaan IGF-1 dan GH dilakukan setelah pemeriksaan Kalorimetri Indirek. Uji Statistik: Univariat mempergunakan tes Kormogorov Smimov, Bivariat mempergunakan uji t tidak berpasang, uji Mann Whitney dan Korelasi Pearson. Uji multivariat mempergunakan uji regresi linier ganda.

Hasil dari 49 subyek SHD yang masuk RS karena komplikasi: hematemesis (34,69%), malaria (46,94%), ikterus (55,1%), dan yang terbanyak asites (87,76%). Dari jumlah tersebut 67,35% tergolong Child C, sisanya Child B. Ditemukan 63,27 % subyek SHD mengeluh mual dan 75,52% anoreksia. Rerata asupan energi subyek SHD secara bermakna lebih rendah dari kontrol sehat ($1282,04 \pm 229,85$ vs $1448,71 \pm 325,56$; $p = 0,006$), dan mempunyai korelasi dengan derajat penyakit. Proporsi asupan terhadap kebutuhan energi subyek SHD hanya mencapai $79,49\% \pm 17,60\%$ REE. Proporsi asupan terhadap kebutuhan energi lebih besar pada subyek SHD yang tanpa keluhan mual dan anoreksia. Penelitian ini menemukan 73,57 % dari subyek SHD dalam keadaan malnutrisi, dan 58,26 % diantaranya (42,86% total subyek SHD) dalam keadaan muscle wasting (AOLA pada persentil < 5) dan menunjukkan korelasi dengan asupan energi ($p=0,007$). Meningkatnya mobilisasi lemak dan oksidasi substrat lemak ditandai oleh TLLBK pada lebih dari 67% subyek SHD pada persentil < 15 dan $RQ = 0,7 \pm 0,08$, serta menunjukkan korelasi yang bermakna dengan kurangnya asupan energi ($p = 0,005$). Meningkatnya mobilisasi lemak dan lipolisis diduga mempunyai hubungan dengan rendah IGF-1 dan tingginya GH dalam darah. Walaupun oksidasi lemak diduga untuk mencegah berlanjutnya katabolisme otot, penelitian ini menunjukkan katabolisme otot berlanjut, hal ini ditandai dengan; imbang nitrogen negatif, rasio NUU/K.AOLA subyek SHD bermakna lebih tinggi dari kontrol sehat, dan RQ sekitar 0,43-0,86. Namun, pada penelitian ini tidak didapatkan perbedaan yang bermakna REE subyek SHD dengan kontrol sehat; hal ini disebabkan oleh menurunnya

massa otot, dan meningkatnya oksidasi substrat lemak sehingga konsumsi oksigen dan REE rendah. Data menunjukkan rasio REE/K.AOLA bermakna lebih tinggi dari kontrol sehat. Keadaan ini menunjukkan subyek SHD dalam kondisi hipermetabolisme disertai penyimpangan metabolisme yang dapat ditandai oleh berlanjutnya mobilisasi lemak; oksidasi substrat lemak (tak sempurna); dan oksidasi substrat protein berlangsung bersama. Asupan nutrisi, komposisi tubuh, dan status metabolisme penderita SHD telah diidentifikasi merupakan acuan penting untuk menentukan REE. Dengan menggunakan variabel; rerata asupan energi, komposisi tubuh (TB, BB, AOLA, dan lainnya) dan derajat penyakit (skor Child-Pugh, albumin, dan NUU) sebagai variabel independen. Melalui uji regresi linier ganda (metode STEPWISE) penelitian ini menemukan 3 variabel merupakan determinan kuat REE yaitu TB, AOLA dan kadar albumin. Dan uji tersebut diperoleh persamaan model yang merupakan model REE estimasi SHD yang reliabel, sehingga dapat direkomendasikan sebagai rumus estimasi REE atau kebutuhan energi penderita SHD yaitu :
kebutuhan energi = $-270,40 + 17,26 * AOLA - 217,83 * Albumin + 11,42 * TB$.

Kesimpulan Pada penderita SHD, keadaan hipermetabolisme tidak dapat ditentukan hanya dengan indikator REE. Hipermetabolisme pada subyek SHD menjadi nyata dengan menentukan REE/K.AOLA dan NUU/K.AOLA. Nasib oksidasi makronutrien pada SHD berbeda dengan pada starvasi. Pada subyek SHD terjadi rangsangan mobilisasi lemak, oksidasi substrat lemak, katabolisme protein otot, dan oksidasi substrat protein secara bersama. Keadaan yang membuktikan adanya penyimpangan metabolisme. Dengan uji regresi linier ganda (metoda STEPWISE), AOLA, albumin dan tinggi badan ditemukan sebagai determinan kuat dari REE atau kebutuhan energi pada penderita SHD rawat inap di RS.

ABSTRACT

Objective: To study the metabolic status of the Decompensated Liver Cirrhotic (DLC) patients who were hospitalized, and to formulate the equation of energy requirements equal to their metabolic status.

Places: The Department of Nutrition and the Department of Internal Medicine at Sumber Waras Hospital (SWH), Jakarta.

The cross sectional study was carried out on 49 DLC subjects, aged 30-80 years, selected consecutively, and on 40 healthy control subjects, selected at random, from SWH staff. The REE data was determined by assessing the Oz consumption (V02) and CO2 production (VCO2] using an Indirect Calorimeter. Food intake was recorded for 3 consecutive days before determining REE. The anthropometrics data (AC, TSF, C.AMA, Height and Weight) and urine samples were assessed one day prior to determining REE. The blood samples for determining IGF-1 and GH were taken after the Indirect Calorimetric assessment (REE data).
The statistical tests: Univarian (using Kormogorof-Smimov), Bivarian (using unpaired T-tests, Mann-Whitney and Pearson Correlation), Multivariate (using multiple linear regression).

Results The 49 DLC subjects were hospitalized mainly due to complications of ascites (87.76%); many also suffered with hematemesis (34.69%), melena (46.94%), or icterus (55.1%). Of the 49 subjects, 67.35% were classified as Child C, the rest were Child B. The subjective findings were nausea (63.27%) and/or anorexia (75.52%). The mean energy intake of DLC subjects was significantly lower than the control (healthy volunteers) (1282.74 ± 229.85 vs. 1448.71 ± 325.56; p = 0.006), and had a correlation to the degree of disease, Their intake had only been 79.49 ± 17.60% of REE. The proportion of food intake to energy requirements was

larger in the DLC subject who had no symptoms of nausea and anorexia. This study has proved that 73.57% of DLC subjects had malnutrition, and 58.26 % of them (42.86 % of all DLC subjects) were in a muscle wasting condition (the percentile of AMA < 5). It showed a correlation to a decrease in the energy intake ($p = 0.007$). The increase of fat mobilization and lipid substrate oxidation were shown by the DLC subjects' TSF of more than 67% with a percentile of less than 15 and the mean RQ = 0.7 ± 0.08 . This also had a significant correlation to a decrease in the energy intake ($p = 0.005$). The increase of fat mobilization and lipolysis was assumed to have a correlation with the low level of blood IGF-1 and the high levels of blood GH. The increase of lipid substrate oxidation was assumed to prevent the subsequent of muscle catabolism, however this study showed that the process of muscle catabolism does not end, which was marked by a negative nitrogen balance, a significantly higher the UNUIC.AMA than the control and a RQ of 0.43 - 0.86. In this study, there was no significant difference between the REE of the DLC subjects and the control; this was due to the decrease of muscle mass and the increase of lipid substrate oxidation. This caused a low V02 consumption and a low REE. This study showed REEIC.AMA of the DLC subjects was significantly higher than the control. This condition indicated that the subjects were hyper metabolic with several abnormalities in metabolism such as: continued stimulation causing lipid mobilization from adipose tissue; incomplete oxidation of fatty acid and protein substrate oxidation running together. Energy intake, body composition, the metabolic status of DLC patients was an important reference for the identification of the REE. By using variables which influenced REE, i.e. the mean energy intake, body composition (height, weight, C.AMA, etc.) and the degree of disease severity (Child-Pugh score, albumin, and UNU), which were tested by the multiple linear regression of STEPWISE method, the equation model has been formulated and tested The final equation for estimating energy requirement is:

Energy requirements = $-270.40 + 17.26 \cdot \text{AOLA} - 217.83 \cdot \text{Albumin} + 11.42 \cdot \text{Height}$.

Conclusion RITE is not the only indicator of hyper metabolism in DLC patients. Hyper metabolism can be identified in DLC patients using REEIC.AMA and UNUIC.AMA. This study has proved abnormalities in metabolism of DLC patients as follows: continued stimulation causing lipid mobilization from adipose tissue; oxidation of fatty acid; muscle protein catabolism; and protein substrate oxidation running together. Through multiple linear regression analysis (the STEPWISE method), AMA, albumin level and height have been found as strong determiners of REE or determiners of energy requirements for DLC subjects.