ARTICOLE

NUTRITIONAL SUPPORT IN NEUROSURGICAL ICU PATIENTS

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When considering nutritional therapy for the neurological/neurosurgical patient, the groups of patients to be included must be well defined. The neurological disorders can be seen into five main categories according to its cause; traumatic brain injuries, cerebrovascular accidents (haemorrhages, ruptured aneurisms/arteriovenous malformations), space occupying lesions, infection and chronic degenerative processes. These patients can also be classified regarding their acute and chronic illness of the neurological pathology. The causes of malnutrition often are multifactorial and can be divided into four phases, their antecedent nutritional status (elderly patients), the stress of acute illness, the surgical interventions/early post-operative period and long term care related to the degree of the neurological deficit.

The metabolic and physiologic functional alterations show a complex cascade. The metabolic aspect depends basically on the underlying disease process and stress is one of the most important causes and in the presence of neurosurgery the metabolic response becomes much more profound. Differences regarding their pathology and its location contribute to the severity. However there are similarities during the chronic period of the different diseases, the acute period stress response depends mostly to the severity of the illness. Some patients besides their neurological disease do pronounce systemic disorders such as organ failures which diverse the metabolic changes activating nutritional demand in a different way and influencing negatively the morbidity and mortality rates. The common seen metabolic aspects are hyperglycemia which reflects the severity and the outcome, altered gastrointestinal functions as intolerance and delayed gastric emptying and depressed immunity during the acute phase response.

The organism is governed by central nervous system (CNS) and its homeostasis is established by the hypothalamus and the brain stem. Most of the neurological pathologies affect the CNS and sometimes a very small injury in one of these important vital areas may contributes severely the outcome with a hypermetabolic clinical symptoms. All the pathological alterations of the organism generates primary a stress response via the brain and its network. But when the brain itself is affected it might not have the same ability to respond metabolically in the same way depending on the involved cerebral region. Abnormal counterregulatory responses might be seen with the malfunction of the hypothalamus leaving the brain stem on its own.

Besides axonal injuries CNS pathologies contribute also CSF biochemistry and cerebral blood flow leading to important clinical symptoms like high (heat gain) or low (heat loss) fever, hypoxia, hypercarbia, intense hypocarbia, fluid and electrolytes imbalance, hyperglycemia.

Heat loss is promoted by the control system activating a cholinergic neuronal system in the medial anterior hypothalamus which projects via relays in the brain stem and anterolateral grey matter of the spinal cord to cholinergic sympathetic nerve fibres making dilation of the skin vessels in the trunk and eccrine sweating. While heat gain is promoted by the control centre activating and adrenergic neuronal centre in the medial posterior hypothalamus. By similar medullary and spinal cord relays, it activates the sympathetic adrenergic neurons to conserve heat by causing vasoconstriction of skin and mucosal vessels, and piloerection. Nutrients are the key generator of heat via thermogenesis (diet induced thermonegesis) and makes an important role for the nutritional support evaluation. Over-feeding which will give raise to iatrogenic thermogenesis may cause an increase in body temperature in patients who have sustained the stress of the acute illness. The lower nutrient-induced thermogenesis and more positive energy balance, indicates a more efficient utilisation of nutrients. Some aminoacides like leucine and glycine are thermogenic not dependant upon the route of administration. Over-feeding must be avoided in neurological patients. The lower CO_2 production during TPN or EN , may be advantageous when respiratory function is compromised.

The metabolic response of the neurological patient is closely related to the pathology location and the clinical evolution of the disease. Studies have shown a pronounced hypermetabolism and hypercatabolism. There is marked increase in protein turnover, protein stores are broken, excess in urinary N excretion, excessive levels of counterregulatory hormones, significant increase in glucose utilization, gluconeogenesis.

In elderly patients suffering from cerebrovasvular accidents (CVA) there is no increase in resting energy expenditure (REE) and is generally less because of decreased activity and muscular tone subsequent to the CVA. But REE measurements done in our neurosurgical ICU by indirect calorimetry have shown a pronounced increase of metabolic requirement scaling between 20-30 % , while the aneurysms ruptures with subarachnoid heamorrage and the head traumas had the highest increases. Data show that starved head-injured patients lose sufficient nitrogen to reduce weight by 15 % per week.

It has been described many equations for the measurements of the requirement of caloric intake. Measuring a patient energy requirement at least once by indirect calorimetry is important, because the degree of metabolism predicts how easily a patient will be underfed or overfed. Particularly surveys of nutritional support have reported discripancies between a prescribed intake and actual delivery in ICU.

The easy way which has a narrow range of error deviation is the measurement with the body weight (25-35 kcal/kg) formula. A lower energy intake, 20-25 kcal/kg is adequate in obesity; patients with a normal bodyweight need 25-30 kcal/kg for maintenance; underweight patients or patients hypercatabolic state or head trauma patients may require a higher energy intake with 35-40 kcal/kg actual bodyweight.

Once the cardiovascular system is stabilized, nutrition should be provided as early as possible in order to prevent the development of malnutrition during the course of the illness. Enteral nutrition should be preferred within the tolerability and of the gastrointestinal tract. Less risk of hyperglycemia, low risk of infection and low cost are the advantages of enteral nutrition. The protein intake must be at least 1g/kg and the composition of the amino acids (nonessential and essential) must be well defined for the best protein synthesis of the liver. Nutrition is not an exact science and what we must considered the most is realability on clinical experience accepting the individualized tailored nutrition regimes. There is no sufficient data to support a treatment standart , but ASPEN (2009) and ESPEN (2008-9) clinical guidelines are the best tools as recommendations for the metabolic and nutritional benefits of the patients.

As a conclusion, nutrition support in the neurosurgical/neurological patient's estimated requirements need to be adjusted according to the underlying neurological disorder , the metabolic alterations, the clinical evolution and manifestations of the disease. Localisation of the cerebral pathology is important (Secondary Brain Injury), metabolic aspects must be defined, stress response is disturbed, factors other than the cerebral pathology might interact nutritional approach , high fever makes detrimental clinical status, hypoxia, hypo/hyper hypocarbia must be corrected, appropriate caloric requirement has to be given, protein delivery is of vital importance, if enteral nutrition is not indicated parenteral nutrition must be administered.

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DISFUNCȚIA MULTIPLĂ DE ORGANE ÎN SEPSIS-UL CHIRURGICAL SEVER

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Actualitatea problemei

Mortalitatea în sepsisul chirurgical sever (disfuncția de organe indusă de infecție sau hipoperfuzie tisulară) şi în şocul septic (hipotensiunea arterială refractară resuscitării cu lichide şi asociată cu disfuncție de organe sau hipoperfuzie tisulară) rămâne inacceptabil de înaltă în majoritatea centrelor de tratament al sepsisului.

Rezultatele tratamentului în sepsisul chirurgical sever, la fel ca şi în infarctul miocardic acut sau atacul cerebral acut, sânt influențate de oportunitatea şi adecvanța terapiei intensive complexe şi specifice, administrate în primele ore de apariție a sindroamului caracteristic SIRS / MODS sau MOSF.

Este ştiut că la leziunile tisulare produse de agenții mecanici, chimici sau bacterieni organismul raspunde inițial printr-un mecanism nespecific de aparare, care este inflamatia. Mesagerii fiziologici ai raspunsului inflamator sunt citokinele, în principal TNF-alfa, interleukinele (IL-l şi IL-6), interferonul şi CSF. Efectorii celulari ai raspunsului inflamator sunt: polimorfonuclearele, monocitele, macrofagelee si celulele endoteiiale. Aceste celule activate duc la sinteza şi secretia de noi citokine şi, de mediatori inflamatori secundari (prostaglandine, leucotriene, tromboxani, factorul activator al trombocitelor, radicalii liberi de oxigen, oxid nitric, proteaze). Acestă activare a celulelor endoteliale şi prezenta cito-kinelor duce la activarea cascadei coagularii care tinde să izoleze aria inflamatorie.

Pierderea controlului local sau exagerarea reacției inflamatorii se identifică clinic cu Sindromul de Raspuns Inflamator Sistemic (Sistemic Inflamator Response Syndrome - SIRS).

Definirea unor termeni şi sindroame

SIRS poate fi inițiat atât de cauza neinfecțioasă (traumatisme, intoxicații, reactii autoimune), cât şi de infectii (virusi, bacterii, protozoare, ciuperci şi al.).

Conform ultimilor întruniri internaționale de consens in domeniu Sepsis-ul este definit ca SIRS, în care infecția este dovedită.