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Early enteral nutrition positively influences endocrine function in traumatic brain injury patients

Dear Editor,

Recent data provide evidence supporting the provision of early enteral nutrition (EEN) (within 24-48h) after admission to intensive care unit (ICU)¹. Moreover, sufficient data demonstrate the influence of EEN on the deregulated endocrine system (ES) of traumatic brain injury (TBI) patients. The ES is directly or indirectly affected by TBI², while it participates in the metabolic and immunologic response following trauma. A recently published open-labeled randomized study investigated the effect of EEN on the ES of TBI patients³. The effect of the onset of nutrition on the pituitary (thyroid-stimulating hormone; TSH), thyroid (free-triiodothyronine/f-T₃, free-thyroxine/f-T₄), gonadal (testosterone-males), and adrenal (cortisol) hormones were investigated on day 6 and day 12 after admission to the ICU. The mortality rate was similar in both groups (p=0.693). However, the duration of ICU stay tended to be longer in control patients with delayed enteral feeding (DEF) (p=0.06). The levels of TSH, f-T₃, f-T₄, and testosterone of DEF patients declined and cortisol concentration increased in comparison to the levels of the day of admission. Notably, the changes of hormonal values were less pronounced in the EEN group.

Overall, deficiency in hormones released via the hypothalamus-pituitary-thyroid-axis results in typical symptoms of hypothyroidism⁴. In traumatic patients the so-called "low T₃ syndrome" is supposed to counteract the catabolic processes appearing in severe illness. These data suggest that the provision of EEN to TBI patients can lead to a diminished drop of thyroid hormones; the latter may decrease the need to consume endogenous substrate storages, and therefore, intense down-regulation of TSH and thyroid hormones may no longer be required. Moreover, EEN clearly reduced increase in cortisol levels, which is very important, since injured patients with high cortisol levels show the highest risk of mortality⁵. TBI leads to hypogonadotropic hypogonadism and lowered testosterone levels in critically ill male polytrauma patients⁶. Of note, by decreasing the decline of testosterone, EEN contributes to the counteracting response against catabolic processes induced by TBI.

It is uncertain if the hormonal changes initiate the metabolic changes or they are the effect of it (thus, being a "marker" of increased catabolism or decreased anabolism). Taken together, an early onset of feeding (EEN) may exert beneficial effects on the hormonal profile of TBI patients (and also to other parameters, such as early-onset ventilator associated pneumonia reduction), possibly contributing to a better clinical outcome. Further studies are warranted to elucidate the mechanisms by which feeding is affecting the hormonal system in TBI patients.

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