Section A-Research Paper



## Incidence of euthyroid sick syndrome in traumatic brain injury (TBI) patients

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## Abstract

**Background:** Euthyroid sick syndrome is a condition in which serum levels of thyroid hormones are low in patients who have a nonthyroidal systemic illness but who are euthyroid.

**Keywords:** Traumatic brain injury TBI, euthyroid sick syndrome (ESS), free T3, free T4, TSH

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**Introduction:** Traumatic brain injury (TBI) is an important cause of death and disability in young adults, and may lead to physical disabilities and long-term cognitive, behavioral, psychological and social defects. (1-3).

Substantial changes in serum levels of thyroid hormones have been described following many nonthyroidal illnesses, in particular after major surgery (4), burns (5) most of critical illness (6) and drug therapy (7). Decreased levels of  $T_3$  and free  $T_3$  is associated with increases in reverse T3 and normal TSH levels which known as euthyroid sick syndrome (ESS) have been observed in such conditions (8). This pattern is suggestive of a decrease in thyroid function of hypothalamic or pituitary origin as TSH levels remain normal despite low thyroid hormone levels.

Euthyroid sick syndrome (ESS) effects on patients suffering from traumatic brain injury (TBI) have received little attention. Moreover, there is limited evidence that serum levels of thyroid-related hormones might influence functional outcome in the acute phase of brain damage. However, the relationship is complex, and the relevance for functional outcome and the therapeutic interventions remain the subject of ongoing researches (9).

## **References:**

- Agha A, Rogers B, Mylotte D, Taleb F, Tormey W, Phillips J, Thompson CJ. Neuroendocrine dysfunction in the acute phase of traumatic brain injury. Clin Endocrinol 2004; 60(5):584-91.
- 2- Kaulfers AM, Backeljauw PF, Reifschneider K, Blum S, Michaud L, Weiss M, Rose SR. Endocrine dysfunction following traumatic brain injury in children. J Pediatr 2010; 157(6):894-9.
- 3- Bondanelli M, Ambrosio MR, Zatelli MC, De Marinis L, degli Uberti EC. Hypopituitarism after traumatic brain injury. European Journal of Endocrinology. 2005; 152(5):679-91.
- **4-** Kelly G. Peripheral metabolism of thyroid hormones: a review. Alternative medicine review. 2000; 5(4):306.
- Woolf PD. Hormonal responses to trauma. Critical care medicine. 1992 ; 20(2):216-26.
- 6- Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JI, Pessah-Pollack R, Singer PA, Woeber for the American Association of Clinical Endocrinologists and American Thyroid Association Taskforce on Hypothyroidism in Adults KA. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Thyroid. 2012; 22(12):1200-35.
- 7- Jeschke MG, Chinkes DL, Finnerty CC, Kulp G, Suman OE, Norbury WB, Branski LK, Gauglitz GG, Mlcak RP, Herndon DN. The pathophysiologic response to severe burn injury. Annals of surgery. 2008; 248(3):387.
- 8- Gereben B, Zavacki AM, Ribich S, Kim BW, Huang SA, Simonides WS, Zeold A, Bianco AC. Cellular and molecular basis of deiodinase-regulated thyroid hormone signaling. Endocrine reviews. 2008; 29(7):898-938.