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Prevalence and study of neuroendocrine deficits in a series of 75 patients following traumatic brain injury

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**Introduction:** Clinical research studies over the last 15 years have reported a significant burden of hypopituitarism in survivors of traumatic brain injury (TBI). However, these endocrine anomalies remain underdiagnosed.

**Patients and methods**: We are studying data from a series of 75 TBI patients. They had a basal hormonal test, and dynamic confirmation tests if necessary. Somatotropic deficiency was defined by growth hormone (GH) remaining under 3 μg/l on two dynamic tests, with glucagon or insulin. Gonadotropic deficiency was defined in men by a total testosterone lower than 10 nmol/l without peripheral cause. Gonadotropic deficiency was defined in non-menopausal women with amenorrhea, and low estradiol in absence of elevated FSH. Gonadotropic deficiency was defined in postmenopausal women by inappropriately low FSH and LH. Corticotropic deficit was defined by a basal cortisol less than 180 μg/l, without stimulation by a glucagon, insulin or synachten test. Thyrotrophic deficit was defined as low free T4 without compensatory elevated TSH. Hyperprolactinemia was defined as prolactin greater than 15 μg/l, TSH less than 2 mUI/l, and absence of interfering medication. Prolactin deficiency was defined by prolactin under 4 μg/l. Diabetes insipidus was defined by the need to use vasopressin to correct a hydro-electrolytic disorder.

**Results:** The prevalence of neuroendocrine disorders in this series is 37% (28/75). Hypopituitary patients had a mean age of 41±15 years (19M/9F). TBI patients with endocrine deficiencies had significantly higher BMI (29±4.4 vs 25±4.4 kg/m2, *P*<0.001) and lower IGF1 (134±54 vs 161±57 ng/ml, *P*=0.04) than TBI patients without hormonal deficiencies. A first glucagon test was abnormal in 22/43 patients, a second insulin or glucagon test was abnormal in 6/12 patients. The biological explorations found: somatotropic deficits (17/28), gonadotropic deficits (13/28), corticotropic deficits (16/28), thyrotrophic deficits (4/28), hypoprolactinemia (3/28), hyperprolactinemia (2/28), and diabetes insipidus (1/28). Pituitary MRI objective: interruption of stem (1 case), aspect of empty sella (2 cases), hypothalamic hemorrhage (1 case). Hormonal substitution improved quality of life: data is still under analysis.

**Conclusion**: Searching for a neuroendocrine deficit is necessary during the assessment of a patient with TBI. We suggest an approach to the diagnosis of post-traumatic hypopituitarism in routine clinical practice. More importantly, this search should also be integrated into medical insurance expertise. Therefore, it is of great importance to evaluate the pituitary function and take appropriate hormone replacement in TBI patients with apparent clinical symptoms and hormonal disturbances.