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(CASE REPORT)

Anteropituitary insufficiency secondary to head trauma: A case report

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Abstract

Head injury (HIT) is a public health problem, post-traumatic pituitary deficits are a rare complication, the etiopathogenic mechanisms of pituitary insufficiency in HIT are variable, the most frequent deficiency in HIT isthat of GH and gonadotropins, hypothalamic-pituitary MRI is the key paraclinical examination; and management is multidisciplinary.

Keywords: Head trauma; Pituitary deficits; Hypothalamic-pituitary MRI; Multidisciplinary management

1. Introduction

Pituitary insufficiency is a polymorphous pathology on the clinical and etiological level. In our daily practice, it is most often of tumoral origin. Pituitary insufficiency is then linked either to a suffering of the different pituitary axes or to an interruption of the hypothalamo- hypophyseal control [1].

Pituitary insufficiency complicating head trauma is a very underestimated pathology. In the absence of associated diabetes insipidus. Hormone deficiency is most often not recognized until months or even years after the accident [2]. At the end of the 1980s, clinical presentations reported an association between head trauma (CT) and pituitary insufficiency, with about fifty cases published in the literature [1].

We report a case of anteropituitary insufficiency involving 3 axes complicating a severe head injury

Observation

He was a 25 year old patient, without any particular pathological history, victim of a severe head trauma following a road accident; causing a deep coma in him; the initial cerebral CT

scan had objectified: a left frontal parenchymal hematoma with left fronto-temporal extradural hematoma, several foci of hemorrhagic contusion with meningeal hemorrhage grade 4 of Fischer and subfalcoral involvement on the right side, hyposignal of the anteropophysis related to a bleeding. As well as multiple fractures of the frontal, temporal and midline bones (image 1).

During his hospitalization, the patient had presented episodes of hypoglycemia quantified at 0.4g/l for which a hormonal assessment was requested objectifying : thyroid insufficiency with TSH=3.5 uUi/l (1.27-4.2 uUi/l); T4 =8.8 pmol/l (12-22 pmol/l); gonadotropic insufficiency with FSH=0.67 ui/l (10.9-13.9 ui/l); LH=2.06 ui /l (6.9-10.3).

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Testosterone= 0.13 ng/ml (4-8 ng/ml); corticotropic insufficiency with cortsolemia =4.6 ug/dl (5-19 ug/dl); and prolactin =0.39 ng/m (2.6-18 ng/ml)

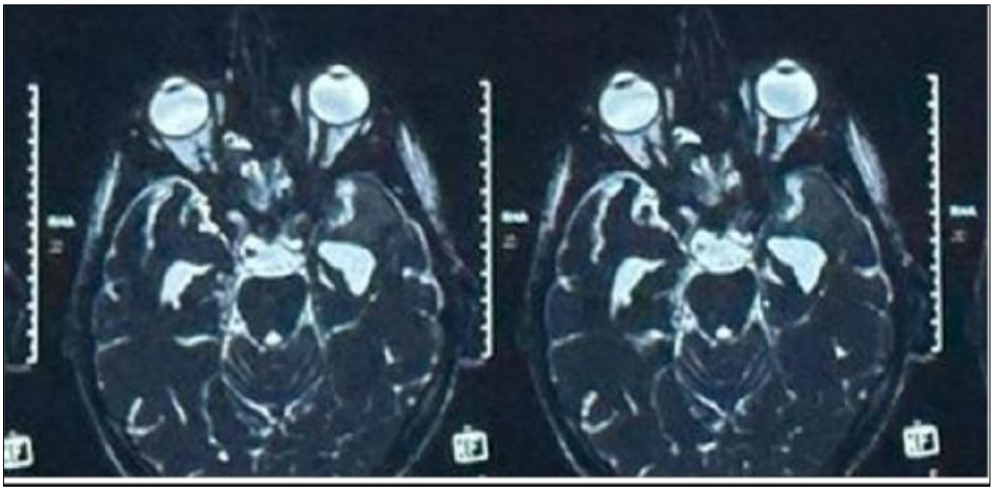

Figure 1 Cerebral CT scan

The patient was substituted with: L-thyroxine at a dose of 100ug/d; hydrocortisone: 20 mg/d with a good clinico-biological evolution

2. Discussion

Traumatic brain injury (TBI) is a public health problem (approximately 276,000,000 cases of moderate to severe TBI in the United States between 1995 and 2001), the long-term consequences of which are often unrecognized, particularly at the endocrine level [3].

The first case of pituitary deficit after CT was described in 1918. Other observations have subsequently confirmed the possibility of deficits following CT. In 2000, S. Benvenga et al. reported 367 cases of pituitary deficits secondary to CT, based on studies published between 1970 and 1998. All the patients, mostly young men, had a gonadotropic deficit, while corticotropic and thyroid deficits affected about 50% of them. One third of the patients had diabetes insipidus [4].

Autopsy studies have shown frequent hemorrhagic or necrotic hypothalamic lesions (23.2 to 59% of patients). Involvement of the pituitary stalk (5.9 to 27.4%) or the anterior lobe of the pituitary gland (3.8 to 22%) is less common. However, 14 to 74% of patients have no obvious hypothalamic-pituitary morphological abnormality [4].Morphological study (hypothalamic- pituitary MRI) in the context of CT is rare, in one review [4], it was performed in 20% of patients, and was abnormal in only 32.9% of cases. Another study carried out by Schneider et al. whose objective was to study the MRI or the pituitary scanner performed in 22 patients with CT, showed hypothalamic-pituitary morphological abnormalities in 80%, with hypopituitarism. Only 29% of patients with normal pituitary function [5].

The clinical manifestations after head injury is variable depending on the severity of the injury and the age of the patient; The most common deficiency in head injury is GH and gonadotropins, followed by ACTH and TSH (table 1, 2) [6].

Table 1 Seven retrospective series with brain damage post-traumatic and secondary hypopituitarism [6]

Publication	Patients	Suivi / Tests	Lignées déficitaires %
Agha et al. 2004	N=50 TBI 12F/38H 37 ± 14 ans	Phase aiguë Glucagon ITT GHRH-GHRP6	GH 18 LH-FSH 12 ACTH 16 TSH 2 DBT insip 2.6
Dimopolou et al. 2004	N=34 TBI 27H/7 F 36 ± 16 ans	0-12 mois	GH 9 LH-FSH 24 ACTH 24
Aimaretti et al. JCEM 2005	N=70 TBI	3 -12 mois GHRH / Arginine	GH 3---11 LH-FSH 17---11 ACTH 8,5---7 TSH 6 PRL élevée 4 DBT insip 4---2,8
Schneider et al. 2006	N =78 TBI 52H/26 F 36 ans	GHRH-Arginine Synacthen	GH 9---10 LH-FSH 32---21 ACTH 19---9 TSH 8---3
Tanriverdi et al. 2006	N=52 TBI 43H/9F	0-12 mois GHRH-Arginine	GH 19---37 LH-FSH 38---8 ACTH 10---19 TSH 5,8---5,8 PRL élevée 7,8 DBT insipid ?
Klose et al. 2007	N=46 TBI 39 (19-63)ans	0-12 mois ITT/Synacthen/ GHRH-Arginine	GH 11 LH-FSH 2 ACTH 6 TSH 2 DBT insip 2
Tanriverdi et al. 2008	N=30 TBI 25H/5F 37 ± 2 ans	0-12-36 mois GHRH-GHRP6 Synacthen	1an 2ans GH 43---17 LH-FSH 3,3---0 ACTH 20---6,6 TSH 6,6---0

Table 2 Seven retrospective series with posttraumatic brain damage and secondary hypopituitarism [6]

Publication	Patients	Suivi / Tests	Lignées déficitaires %
Kelly 2000	N=22 TBI (18H/4F)	ITT	GH 24 LH-FSH 90 ACTH 60 TSH 50 PRL ↑ 45 DBT insip 30
Richard 2001	N=93 TBI (75H/18 F)	Biologie de base	GH non étudié LH-FSH 28 ACTH 0 TSH 1 PRL non étudié DBT insip 1
Lieberman et al. 2001 JCEM 2001	N=70 TBI N=32 SAH	6m-20 ans Glucagon/ LDopa test	GH 15 LH-FSH 0 ACTH 7 TSH 3 DBT insip 0
Bondanelli et al. 2004	N=50 TBI 10F/40M	???	GH 20 LH-FSH 14 ACTH 0 TSH 10 DBT insip ?
Agha et al. 2004 JCEM	N=102 TBI 17F/85H	6 à 36 mois Glucagon ITT Arg/GHRH	GH 18-10 LH-FSH 12 ACTH 22 TSH 1 PRL ↑ 12 DBT insip ?
Aimaretti Clin Endocrinol 2004	N=100 TBI (69H/31F)	3 mois GHRH/ Arginine	GH 21 LH-FSH 17 ACTH 8 TSH 5 DBT insip 4
Leal Cerro et al. 2005 Clin Endoc 2005	N=170 TBI (99H/14F)	<50 mois GHRH- GHRP6 ITT, Ou Glucagon	GH 5,8 LH-FSH 17 ACTH 17 TSH 6,4 DBT insip 5,8

Abbreviations : ITT : insulin tolerance test, GHRH : Growth hormone releasing hormone, GHRP6 : Growth hormone related peptide 6, Arg : Arginine test, DBT insip : Diabète Insipide.

Prospective studies have been carried out with the aim of evaluating pituitary function at 3 months, 6 months, 1 year and even 3 years after the trauma. During the first year, the prevalence of deficits was stable, but cases of recovery or, on the contrary, worsening of pituitary function one year after the trauma have been observed, especially in the case of an isolated deficit. The frequent gonadotropic deficit at the time of the check-up carried out 3 to 6 months after the CT

is usually transient, not observed at one year, which is in favour of a more often functional than organic impairment (Table 3, 4) [7].

Table 3 Prevalence of pituitary deficits 3 or 6 months after trauma [8,9,10].

	Agha et al. n = 48	*Aimaretti et al.* n = 70	*Schneider et al.* n = 78
≥ 1 déficit (%)	ND	22,2	36
Déficits multiples (%)	ND	9,9	4,2
Déficit isolé (%)	ND	12,8	31,8
Somatotrope (%)	10,4	20	10
Gonadotrope (%)	12,5	11,4	21
Corticotrope (%)	18,7	7,1	9
Thyréotrope (%)	2	5,7	3
Hyperprolactinémie (%)	12,5	5,7	14
Diabète insipide (%)	6,2	2,8	0

Table 4 Prevalence of pituitary deficits 1 year to 3 years after trauma [8,9,10]

	Agha et al. n = 48	*Aimaretti et al.* n = 70	*Schneider et al.* n = 78
Délai post-traumatisme	6 mois	3 mois	3 mois
Âge (ans)	37 ± 14	39,3 ± 2,4	35,7 ± 13,8
Pourcentage d'hommes	76	71	83
≥ 1 déficit (%)	ND	32,8	56
Déficits multiples (%)	ND	11,4	10,2
Déficit isolé (%)	ND	21,4	45,8
Somatotrope (%)	12,5	22,8	9
Corticotrope (%)	19	8,5	19
Gonadotrope (%)	23	17,1	32
Thyréotrope (%)	2	5,7	8
Hyperprolactinémie (%)	15	4,2	20,5
Diabète insipide (%)	8	4,2	0

The practical management of post CT pituitary insufficiency must be multidisciplinary (anesthesiologist, neurosurgeon, neurologist and endocrinologist). According to international recommendations, gonadotropic, corticotropic, thyrotropic or somatotropicdeficits should be substituted [7].

3. Conclusion

Pituitary insufficiency is a complication to look for after any moderate to severe head trauma, which is confirmed by an expert consensus that recommends a systematic exploration of pituitary function in all patients with moderate to severe head trauma and toimprove the management of these patients, thus underlining the importance of a multidisciplinary management.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

No conflict of interest.

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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