Trauma of the Pituitary Region

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The frequency of pituitary hormone deficiency after head trauma has been debated for a long time. Nevertheless, today it has become accepted that traumatic brain injury (TBI) can generate gonadotropin and somatotropin deficiency. Diabetes insipidus is frequent in the early phase, but permanent diabetes insipidus is much rarer than anterior pituitary dysfunction. Diagnosis is confirmed by axial T1WI, which best demonstrates the absence of posterior pituitary bright spot (Fig. 61.1). Severity of TBI seems to be an important risk factor for developing post-traumatic hypopituitarism, which has also been described. Post-traumatic pituitary deficiency is more frequently observed in young adults, involved mainly in motor vehicle accidents, than in the elderly, who are more commonly involved in falls. Pathophysiology of post-traumatic pituitary deficiency includes pituitary or hypothalamus hemorrhage or necrosis, vascular damage to the long hypophyseal portal system, hypoxic insult, shearing axonal injury, or direct mechanical insult to the pituitary gland, the pituitary stalk, or the hypothalamus.

Literature dealing with MRI aspects of pituitary trauma is scarce. Focal changes in the pituitary gland (hemorrhage/hemorrhagic infarction), swollen gland with bulging superior margin, and heterogeneous signal intensities in the anterior lobe have been described. Section of the pituitary stalk is rarely visible. Recently, it has been proposed that DWI could be interesting for the evaluation of the pituitary gland after trauma and that the ADC could be a marker to predict pituitary function.

Demonstration of basal skull fractures, particularly of the sellar region, must draw attention to and prompt the search for a pituitary dysfunction (Fig. 61.2). A long delay between initial trauma and development of hormone deficiencies is not infrequent. For medicolegal reasons, the peculiar pattern of some skull-base fractures seen as late as 20 years after brain injury must be recognized. Fractures of the planum sphenoidale, for instance, heal with a blistering appearance of the sphenoid sinus, mimicking a meningioma osteoma. In such cases it could be of the utmost importance to be able to analyze the initial radiological files (Fig. 61.3). The most common late radiological pattern of post-traumatic hypopituitarism is shrinkage of the sellar content and, consequently, a partial or complete empty sella. T1 hypointensity of the pituitary gland can been observed (Fig. 61.4). Fibrotic tissue may occupy the bottom of the sella: marked T2 hypointensity and strong postgadolinium enhancement differentiate fibrosis from residual normal pituitary tissue (Fig. 61.5). Pituitary failure may coexist with apparently normal pituitary MRI.

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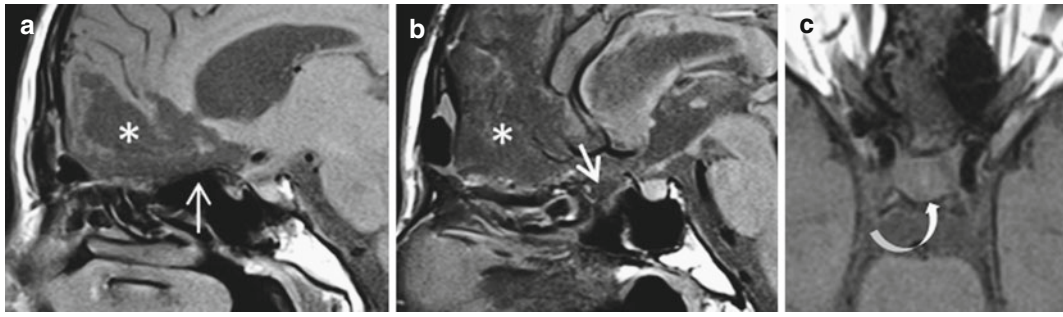

Fig. 61.1 Post-traumatic diabetes insipidus and gonadotropic deficit in a 17-year-old girl persisting 2 years after a severe traffic accident. (**a**, **b**) Mid- and parasagittal T1WIs. Severe post-traumatic alterations of the frontal lobe (*asterisk*). Post-traumatic blistering (*thin arrow*) and displaced fracture (*thick arrow*) of the planum sphenoidale on two consecutive sections. (**c**) No ADH storage demonstrated on axial T1WI (*curved arrow*)

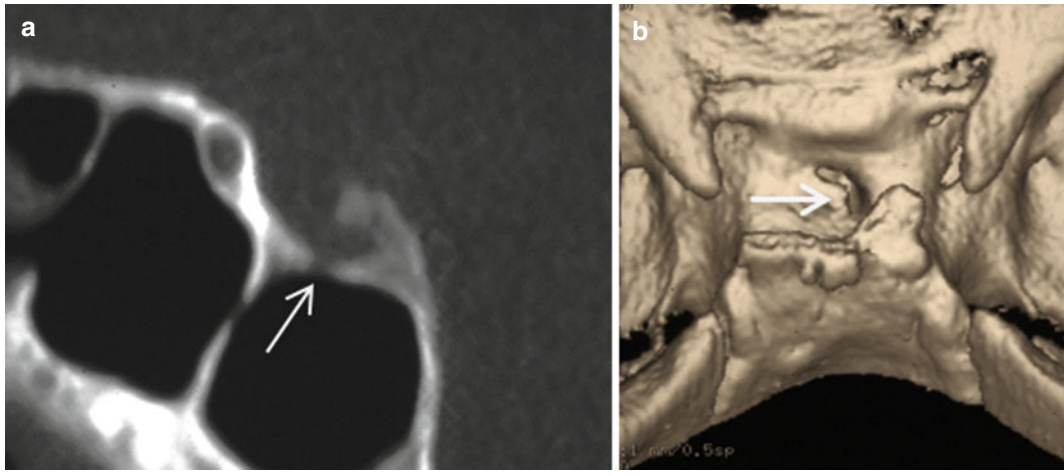

Fig. 61.2 Sella turcica fracture (*arrow*) diagnosed 1 year after head trauma in an 18-year-old patient with gonadotropin deficiency. (**a**) Sagittal CT. (**b**) 3D reformatted image

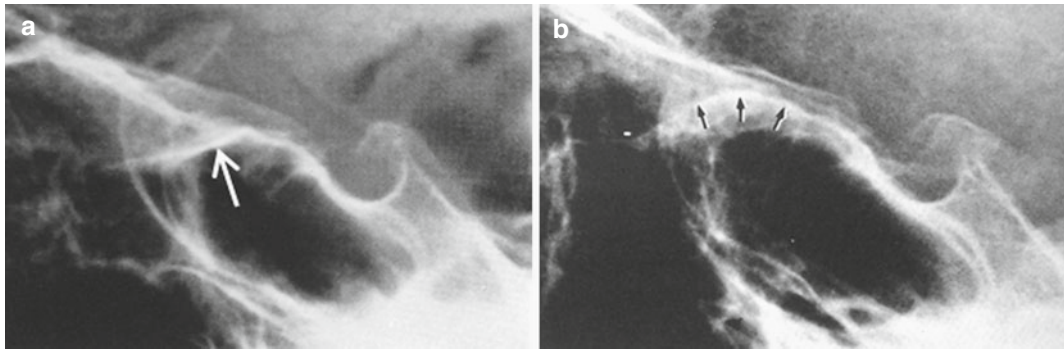

Fig. 61.3 Post-traumatic blistering of the planum sphenoidale in a 25-year-old man. (**a**, **b**) Sagittal radiographs. At the time of trauma (**a**), the fracture of the planum was not recognized and the planum was flat (*arrow*). Seven years later (**b**), the patient presents with transient blunt vision. A blistering of the planum sphenoidale (*small arrows*) is described and a presellar meningioma is first suspected, then eliminated by further examinations. Final diagnosis was post-traumatic blistering mimicking an osteoma of a meningioma of the planum sphenoidale

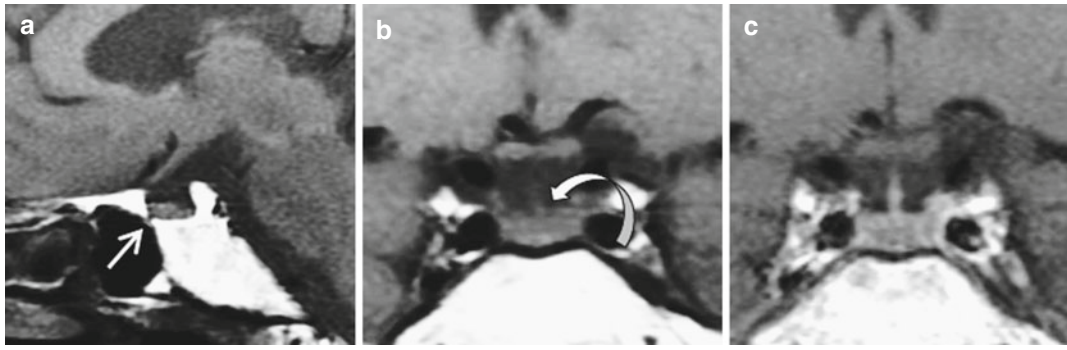

Fig. 61.4 GH deficiency seen 35 years after severe head trauma in a 45-year-old-man. (**a**) Sagittal T1WI. Fracture of the sella turcica; the sphenoid sinus is weakly pneumatized. (**b**, **c**) Coronal T1 and CET1WIs. T1 hypointensity of the pituitary gland; normal postgadolinium enhancement

Fig. 61.5 Anterior pituitary deficit in a 38-year-old woman 3 years after traumatic brain injury. No diabetes insipidus. (**a**) Sagittal CT at the time of the accident. Fracture of the tuberculum sellae and effusion of the sphenoid sinus. (**b**–**d**) Sagittal T1, T2, and CE TIWIs 3 years later. The sella is quite empty; ADH ectopic storage at the stalk level (*arrow*). T2 hypointense fibrotic tissue at the sellar floor with strong enhancement after contrast (*curved arrows*)

Further Reading

Klose M, Stochholm K, Janukonyté J et al (2015) Patient reported outcome in posttraumatic pituitary deficiency: results from. The Danish National Study on posttraumatic hypopituitarism. Eur J Endocrinol 172(6):753–762

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Popovic V, Aimaretti G, Casanueva FF et al (2005) Hypopituitarism following traumatic brain injury. Growth Horm IGF Res 15(3):177–184