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Post-traumatic pituitary apoplexy: Case presentation and review of literature

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ABSTRACT

Pituitary apoplexy is a dramatic condition that can occur spontaneously or triggered by various precipitating factors. Head trauma is a rare but well-recognized cause of apoplectics events. We present the case of an 81-year-old woman, with negative past medical history and under antiplatelet agents, who experienced an isolated VI cranial nerve palsy 24 h after a mild head trauma. Early brain CT revealed an unknown pituitary lesion without signs of intralesional bleeding. Only late brain MRI imaging revealed pituitary apoplexy together with a subarachnoid hemorrhage. After aggravation of neurological condition the patient, undergo endoscopic transsphenoidal decompression of cranial nerves with rapid deficits improvement. Our aim is to share our experience and to propose the first critical review of all cases of post-traumatic pituitary apoplexy described in literature. We also try to suggest some management advice for post traumatic pituitary apoplexy.

1. Introduction

Pituitary apoplexy (PA) is a rare event characterized by a rapid expansion of a pituitary adenoma after a hemorrhage or ischemia, and occurs in about 14–22% of patients [1]. Male-to-female ratio is 2:1, and most cases present in the V or VI decades of life [11]. PA is a dramatic condition that can be sometimes life threatening for the patient [9]. 70–81% of patients with PA had negative medical history for pituitary adenoma [9,15]. PA can be asymptomatic and recognizable only after performing neuroimaging, and is therefore called subclinical or subacute apoplexy [14]. Pituitary adenomas can develop silently, enlarge and then suddenly become symptomatic after triggering events such as head trauma, radiation therapy, sudden changes in intracranial pressure, dopamine agonists administration, hormone stimulation tests, lumbar puncture or spinal anesthesia [1,5,13]. Some authors argue that pituitary apoplexy is more prone to occur in large pituitary tumors (e.g. macroadenoma) whereas microadenomas (<1 cm in diameter) are less susceptible to bleeding [14]. Studies showed than about 70% of pituitary apoplexy occurred in nonfunctioning adenoma [10,15]. Furthermore, there is no evidence that a defined histological type of pituitary tumor is more susceptible to apoplectic events [3,15].

To date, only 12 cases of post-traumatic pituitary apoplexy are described in literature. In this report we want to share our experience in the management of post traumatic PA and propose the first critical review of all cases described in literature.

2. Case presentation

We report the case of an 81-year-old woman, which accidentally fell, in October 2015, from about 1.5 m height with subsequent mild head trauma and blunt contusion on her occipital bone. She was under lifetime prophylaxis with antiplatelet drugs. After she was admitted to the Emergency Department she underwent a brain CT scan which highlighted a left frontal contusion with subarachnoid hemorrhage (SAH), together with the evidence of an undiagnosed pituitary lesion (Fig. 1a, b). Neurological examinations were negative. Twenty-four hours later, she developed VI c.n. (cranial nerve) palsy with no evidence of diplopia or visual acuity loss. Visual field defects were not documented on manual visual field testing. A new brain CT scan showed that the pituitary lesion had enlarged in size, with presence of hyperdense foci, signs of potential intralesional hemorrhage (Fig. 1c). Imaging was compatible with pituitary apoplexy. A comprehensive hormone panel was performed which confirmed mild hypopituitarism (Prolactin 1.7 ng/ml, T3 1.93 mIU/ml, serum cortisol 2.9 ng/ml) and brain MRI (magnetic resonance) scan showed bilateral subdural hematoma together with left temporal and frontal contusions as well as an intrasellar-suprasellar lesion, of 30 mm × 20 mm size, with signs of recent bleeding (Fig. 1d). Brain MRI imaging evidenced how the sellar lesion exerted compression on chiasm and optic nerves, which were dislocated. In the first hours after PA diagnosis the only neurological sign manifested was the VI c.n. palsy and, considering patient's age and comorbidities, we adopt a “wait and see” approach. The patient's clinical conditions were constantly monitored and hormone replacement therapy was administered. After 4 days the patient complained of progressive loss of visual acuity (01/10 on right eye (R.E.) and 2/10 on left eye (L.E.)) and temporal hemianopsia was confirmed by Goldman visual field

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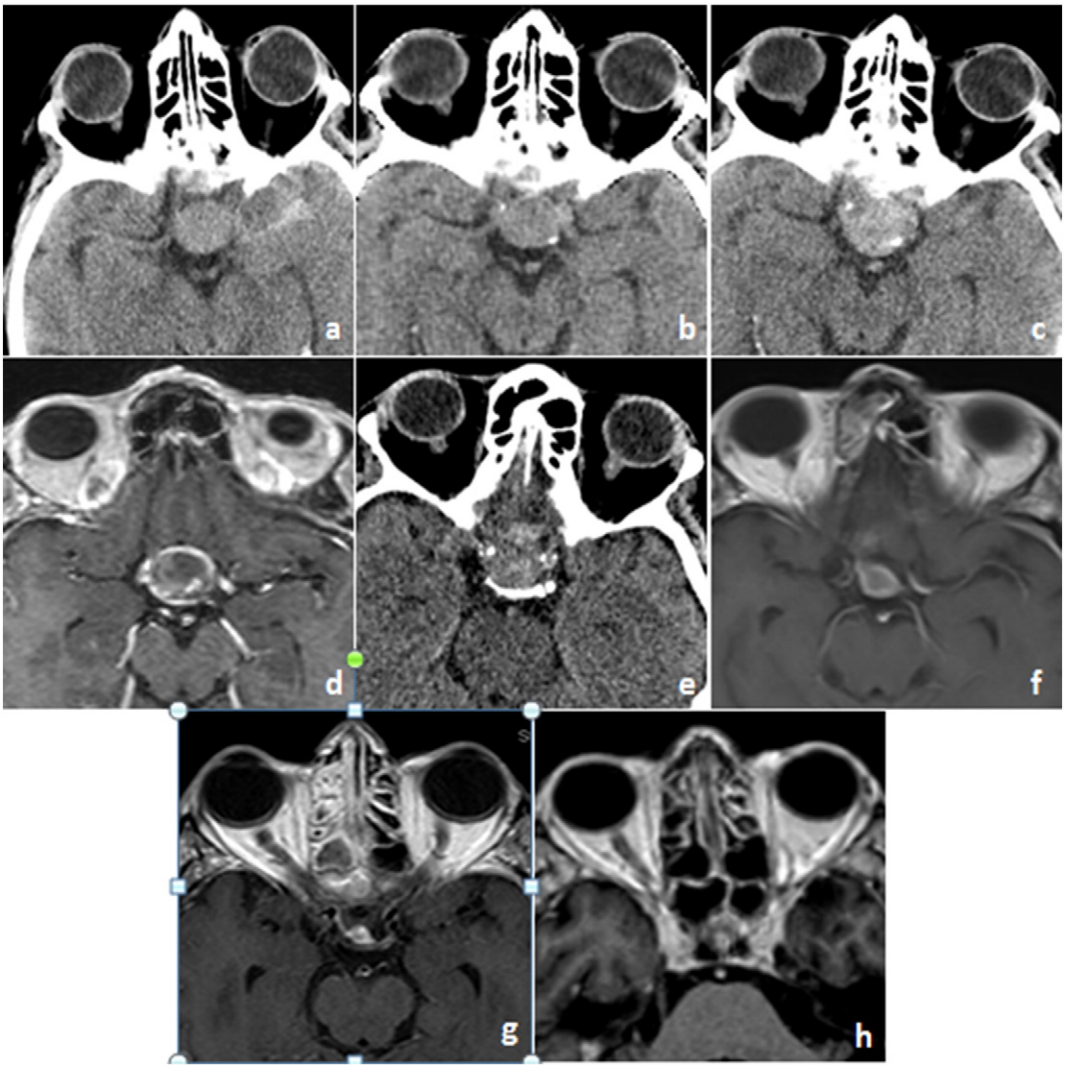

Fig. 1. Early after head trauma axial brain CT scan (a) and 6 h later brain CT scans (b); 24 h axial brain TC scan; recognition of pituitary apoplexy and first manifestation of symptoms (c); 36 h later T1 contrast enhanced axial brain MRI scan: aggravation of neurological condition (d) and 48 h later post-apoplexy CT axial scans (e). Postoperative axial T1 contrast enhanced brain MRI axial scans (f). Early (g) and long term follow up T1 contrast enhanced brain MR axial scan (h).

examination. At that point, surgical treatment was mandatory (Fig. 1d, e). The patient underwent endoscopic trans-sphenoidal surgery in order to decompress optic nerves (Fig. 1f). Histological and immunohistochemical examinations showed extensive necrosis with fibrosis and inflammatory granulation infiltration. The lesion express diffusely synaptophysin together with Ki-67 in 3% of cells, whereas ACTH, prolactin, CKCAM5.2, FH, TSH, GH were not expressed. At that point, the pituitary apoplexy was confirmed. After surgical decompression, visual acuity progressively improved. The patient underwent brain MRI imaging that documented a satisfactory decompression of chiasm and optic nerves. Clinical evaluation at 30 and 60 days documented recovery of visual acuity (4/10 in R.E. and 4–5/10 in L.E.) as well as of visual field impairment. Long-term MRI follow-up did not document any further complications (Fig. 1g, h).

3. Discussion

In 1932 Van Wagenen reported the first two cases of post-traumatic pituitary apoplexy and since 1983 no other cases have been described [2]. In addition, Dr. Harvey Cushing described a similar case in which the pathological examination documented a pituitary tumor with extensive blood infiltration in a patient died after head trauma [2]. In our review of literature, including our case report, we identified 13 post-traumatic pituitary apoplexy cases from 1983 to 2016 (Table 1). 9 patients were men, with a male to female ratio of 2:1, which is also the reported ratio for PA in literature. Age ranged from 30 to 85 with a mean age of 58.7 years. All patients were unaware of the presence of the pituitary tumor. In 2 cases patients complained of symptoms compatible with GH over secretion and in one case symptoms of increased serum prolactin levels were manifest.

To date the pathophysiological mechanism underlying post traumatic pituitary apoplexy has not been established [4]. The pituitary gland is perfused by its portal venous system, which passes down the hypophyseal stalk. It is well described how the particular pattern of vascular supply contributes to the occurrence of pituitary apoplexy [3]. Dandy et al. suggest that pituitary massive hemorrhagic infarcts can be consequent to traumatic shearing forces that cause the destruction of the pituitary stalk and lead to the consequent blockage of pituitary portal vascularization [2]. Several authors support that if the trauma involves the occipital region, as occurred in 9 cases (Table 1), shearing-rotational forces are more susceptible to tear the fragile junction between the intra-sellar portion, that is relatively firm, and the supra-sellar portion, that lies freely within the supra-sellar cistern [1]. Others authors argue that after a head trauma an intralesional hemorrhage can occur, such event alter the venous vascularization pattern encouraging ischemic-hemorrhagic occurrences. This hypothesis can explain the