Bacterial meningoencephalitis that was actually a pituitary apoplexy

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ABSTRACT

Introduction: *Pituitary apoplexy is a syndrome that occurs as a result of an ischemic or hemorrhagic lesion in the pituitary gland, leading to a deficiency of pituitary hormones. It manifests in the form of neurological deterioration with thunderclap headache as the main symptom, with meningeal irritation being an infrequent manifestation.* ***Methods:*** *We present the case of a 53-year-old woman with a history of prolactin-producing madroadenoma that began with headache, nausea and impaired level of consciousness. Incomplete hypopituitarism with normal cortisol level is detected. Cerebrospinal fluid (CSF) is consistent with an aseptic pleocytosis unresponsive to antibiotic therapy. It is associated with oculomotor paresis and a cranial MRI reveals bleeding in the pituitary adenoma with involvement of the cavernous sinus.* ***Results:*** *the initial suspicion is bacterial meningoencephalitis due to fever, stupor and CSF with pleocytosis, although no microorganism is identified and there is no response to antibiotics. CSF from stroke shows aseptic pleocytosis due to meningeal irritation of the subarachnoid space from bleeding and necrosis of the gland. Hypopituitarism can be partial or complete, selective deficiency being more frequent. ACTH deficiency deserves special attention due to the morbidity and mortality that adrenal failure entails. Ophthalmoparesis translates involvement of the cavernous sinus due to an increase in sellar pressure.* ***Conclusions:*** *We emphasize the importance of having a suspected diagnosis of apoplexy in case of an acute neurological condition, to direct the pertinent investigations with hormonal determination and thus initiate early replacement therapy and a neurosurgical approach if necessary; requiring a multidisciplinary management.*

Key words: *pituitary apoplexy, pituitary adenoma, sterile meningitis, hypopituitarism, cavernous sinus, acute headache.*

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INTRODUCTION

Pituitary apoplexy is a rare entity that can be severe if treatment is not received adequately[1]. Its cumulative incidence is inaccurately described and varies between 0.6 to 10% [2]. It is a syndrome resulting from a pituitary insult resulting from infarction or hemorrhage in the gland[3]; more than 60-80% of cases are spontaneous, although there are some risk factors such as diabetes mellitus, anticoagulant therapy, and postpartum hemorrhage[4]. The clinical spectrum includes headache, fever, nausea, altered consciousness, visual deficit, and ophthalmoplegia1. It must be considered in the differential diagnosis of a patient with sudden neurological decline. Meningeal irritation is an uncommon sign that may delay diagnosis due to confusion with a nervous system infection. We present the following case, given the few reports of pituitary apoplexy mimicking bacterial meningoencephalitis[1,5].

METHOD

We present a case of a 53-year-old woman with a history of high blood pressure, anorexia nervosa, and a prolactin-secreting pituitary macroadenoma, treated with cabergoline and thyrotropin. The patient arrived with headaches, vomiting, epileptic seizures, and hemodynamic instability at the emergency room. Infectious parameters on the laboratory tests were normal. A CT brain scan showed a pituitary macroadenoma without any signs of complications. Hormonal determination revealed an incomplete hypoputitarianism with a TSH, FSH, LH, IGF-1, and PRL deficiency, with normal cortisol. Four days after admission, the patient began with stupor, fever, and meningeal irritation signs. A lumbar puncture was then performed, revealing intracranial hypertension (25 cmH2O). Cerebrospinal fluid (CSF) analysis shows pleocytosis (1060 cells, 88% neutrophils), hyper protein (135 mg/dl) and hypoglycorrhachia (35 mg/dl). Empirical therapy was started with Ceftriaxone (2g/12h), Ampicillin (2g/4h), Vancomycin (1g/8h), associating Dexamethasone (4mg/8h), and Levetiracetam (1g/12h). Despite this, there was no improvement, requiring admission to the ICU. The torpid evolution forced to change to Metronidazole (500mg/6h) and Linezolid (600mg/12h). No microorganisms were isolated in two CSF cultures and four blood cultures. A cranial MRI showed signs of pituitary adenoma hemorrhage **(fig.1)**. Antibiotic therapy was interrupted after ten days due to a minimal response, although the evolution with support therapy was satisfactory. Paresis of the III and VI cranial nerves were detected **(fig. 2)**. Neurosurgical evaluation deferred the intervention until the

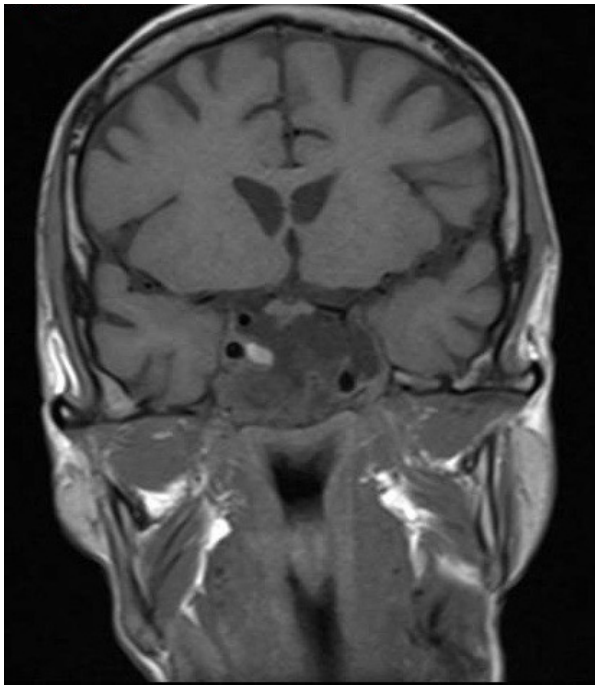

Figure 1. RM craneal 1.5 T.
Coronal section MRI T1 sequence, showing bleedingwithin the macroadenoma, lateralized to the right.

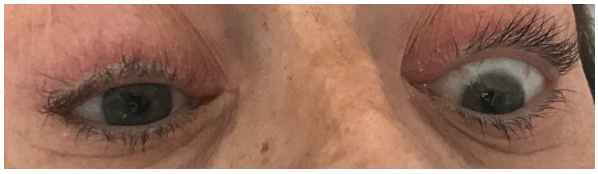

Figure 2. Ophthalmoparesis of the VI and III left cranial nerves. Ophthalmoparesis of the lateral rectus, inferior rectus, superior rectus, and inferior oblique.

resolution of the medical situation. At discharge, the hypopituitarism was corrected.

RESULTS

In this case, the symptoms and the CSF initially led us to suspect bacterial meningitis. Even more so with the determination of normal cortisol, although she presented with incomplete hypopituitarism. The minimal antibiotic response, sterile cultures, and hemodynamic instability misled us into an alternative diagnosis. The brain MRI showed a pituitary hemorrhage affecting the cavernous sinus that was not seen on the admission brain CT[6].

The clinical syndrome was compatible with meningeal irritation as an atypical presentation of pituitary apoplexy, resulting from blood and necrotic tissue that extended to the subarachnoid space, which can mimic a subarachnoid hemorrhage or meningoencephalitis with aseptic pleocytosis.

The pathophysiology of pituitary apoplexy is attributed to increased intrasellar pressure, compressing healthy pituitary tissue and its vascular supply. This high pressure would compromise the cavernous sinus laterally [7,8,9], the optic chiasm and hypothalamus superiorly, and inferiorly may cause a CSF leakage with consequent chemical meningitis.

Hypopituitarism contributes to morbidity and mortality in pituitary apoplexy, which may be partial or total, being the ACTH deficiency the most relevant, with the consequent adrenal insufficiency[10]. The most common presentation is partial hypopituitarism with ACTH deficiency in 40-100% cases, TSH deficiency in 25-80%, gonadal deficiency in 60-100%, and prolactin deficiency 6-40%2. The published case series reveals that pituitary function is not usually assessed at the beginning of symptoms.

Therefore, the pillars in pituitary apoplexy are an early clinical diagnostic suspicion, appropriate tests to confirm it, endocrine substitution therapy, and surgical decompression when indicated[11].

Substitution therapy should address hormonal deficits, emphasizing corticosteroids, with the recommended dose being Hydrocortisone 50 mg every six hours[2].

CONCLUSIONS

We highlight the need to suspect the diagnosis of pituitary apoplexy in patients with a sudden neurological deterioration and thunderclap headache, even more so with the presence of a pituitary adenoma, despite a normal blood cortisol level. We must know that the CSF analysis may mask this entity, mistaking us with a meningeal infection. Finally, remember that MRI is the ideal imaging modality for the sellar region. An early diagnosis reduces morbidity and mortality from 100% to 6.7%[1,5] with a multidisciplinary approach involving endocrinology, neurosurgery, and ICU is essential.

REFERENCES

1. Davinder S Jassal, Gregory McGinn, John M Embil. Pituiraty Apoplexy masquerading as meningoencephalitis. Headache. 2004 Jan;44(1):75-8. doi: 10.1111/j.1526-4610.2004.04013.x.
2. Rita N. Nawar, MD, Dima AbdelMannan, MD, Warren R. Selman, MD, and Baha M. Arafah, MD. Pituitary tumor aplopexy: a review. Journal of Intensive Care Medicine. Volume 23 Number 2 March/April 2008 75-90. 2008 Sage Publications 10.1177/0885066607312992 http://ves.sagepub.com hosted at http://online.sagepub.com
3. Wakai S, Fukushima T, Teramoto A, Sano K. Pituitary apoplexy: its incidence and clinical significance. J Neurosurg. 1981; 55:187-193.
4. Sally Murad-Kejbou and Eric Eggenberger. Pituitary apoplexy: evaluation, management, and

prognosis. Current Opinion in Ophtalmology 2009, 20: 456-461. Department of Neurology and Ophtalmology, Michigan State University, East Lansing Michigan, USA.

5. Wen-Yi Huang, Yu-Yi Chien, Chia-Lun Wu, Wei-Chieh Weng, Tsung-I Peng, Hsien-Chih Chen. Pituitary adenoma apoplexy with initial presentation mimicking
6. bacterial meningoencephalitis: a case report. Am J Emerg Med. 2009 May; 27(4):517.e1-4. doi: 10.1016/j.ajem.2008.08.004.
7. Tosaka M, Sato N, Hirato J, et al. Assessment of hemorrhage in pituitary macroadenoma by T2-weighted gradient echo MR imaging. Am J Neuroradiol 2007; 28:2023-2029.
8. Kim S, Lee K, Kim S. Cranial nerve palsies accompanying pituitary tumour. J Clin Neurosci 2007; 14:1158-1162.
9. Yang S, Lee K, Lee K, et al. Pituitary apoplexy producing internal carotid artery compression: a case report. J Korean Med Sci 2008; 23:1113-1117.
10. Netter F. Atlas of human anatomy. 3rd ed. New Jersey: Icon Learning Systems; 2003.
11. Bonicki W, Kasperlik-Zaluska A, Koszewski W, Zgliczynski W, Wislawski J. Pituitary apoplexy: endocrine, surgical and oncological emergency. Incidence, clinical course and treatment with reference to 799 cases of pituitary adenomas. Acta Neurochir (Wien). 1993;120:118-122.
12. Onesti ST, Wiskiewski T, Post KD. Clinical versus subclinical pituitary apoplexy: presentation, surgical management, and outcome in 21 patients. Neurosurgery 1990; 26(6):980-6.

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