

Case Report

Pituitary apoplexy presenting as an intraventricular hemorrhage: An infrequent neurovascular emergency



Gabriel Torrealba-Acosta, MD^{a,b,*}, Emmanuelle Vargas-Valenciano, MD^c,
Katherine Escob-Bastos, MD^c, Juan Ignacio Padilla-Cuadra, MD^a

^a Neurocritical Care Unit, Department of Neurosciences, Hospital Rafael Ángel Calderón Guardia, Caja Costarricense de Seguro Social, San José, Costa Rica

^b Neurosciences Research Center, University of Costa Rica, Costa Rica

^c Division of Neurosurgery, Department of Neurosciences, Hospital Rafael Ángel Calderón Guardia, Caja Costarricense de Seguro Social, San José, Costa Rica

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ABSTRACT

Pituitary macroadenomas may present with a pituitary apoplexy, commonly with associated hemorrhage, but rarely large enough to show an intraventricular bleeding. This is a case report of a 66-year-old woman with a 3-month history of headache, hyporexia, asthenia and altered consciousness. First evaluations showed a large intraventricular hemorrhage on CT scan that was initially misdiagnosed for an aneurysmal rupture etiology but later confirmed to be a pituitary apoplexy from a large pituitary adenoma. Intraventricular hemorrhage due to a pituitary apoplexy could be mistaken for an aneurysmal origin thus warranting for a careful evaluation of the sellar region.

1. Introduction

In the clinical setting, the first manifestation of undetected pituitary macroadenomas could be a pituitary apoplexy (PA) with or without hemorrhage. Rarely it may be associated with an intraventricular bleeding and can be misdiagnosed as an aneurysmal subarachnoid hemorrhage. We describe a case of a PA presenting with a large intraventricular bleeding.

2. Case report

A 66-year-old woman was evaluated at our emergency department with a 3-month history of headache, hyporexia and asthenia, followed by periods of altered consciousness, disorientation and loss of sphincter control. She was described with somnolence but being able to follow simple commands. She had a slight pupillary asymmetry and was capable to move her four limbs. Although difficult to assess, due to her state of arousal; she seemed to have compromise of visual fields on direct confrontation. CT scan of the head revealed a large intraventricular hemorrhage filling the frontal and occipital horns of the left lateral ventricle, but also extending into the frontal horn of the right lateral ventricle, third ventricle, interpeduncular cistern, Sylvius fissure and sellar and supra-sellar spaces (Fig. 1). A complete blood count, white cell differential, and coagulation panel were normal. She had a mild hyponatremia and hypokalemia, and after multiple hormonal

assessments the results were compatible with a panhypopituitarism.

Albeit she had indirect signs of hydrocephalus in the CT scan, we preferred to defer the placing of a ventricular drain due to several reasons: first, we had the possibility to perform a strict clinical observation over the patient, thus allowing us to monitor her clinical evolution. Second, because initially we thought this could be a subarachnoid hemorrhage of an aneurysmal etiology, there was a putative increased risk of rupture if we performed a ventriculostomy. Finally, we would certainly reduce any risk of developing a procedure-related infectious process, if we refrained from doing it.

We performed an angiography on the patient that did not reveal an aneurysmal lesion; however, it did show severe vasospasm of the anterior communicating and both anterior cerebral arteries. The initial CT scan revealed a bifrontal hematoma with an estimated volume of 38 cm³ and prominent mass effect, hence warranting the need for a hematoma drainage. We considered that the transnasal-transphenoidal route for management of the intracranial hemorrhage would not allow us to have the proximal arterial control we could require. Furthermore, to drain this considerable hematoma, we would need an extended transplanum access in order to reach for the basal frontal region. Yet, none of us had sufficient experience with this surgical approach nor we had the ENT team available to aid us with it.

Moreover, there was still the possibility of a hematoma-related vasospasm masking an aneurysm or vascular malformation in the angiographic study. Thus, we resolved on pursuing a bifrontal approach

* Corresponding author at: Neurosciences Research Center, University of Costa Rica, San Pedro, Montes de Oca, San José Zip/Postal Code: 2060, Costa Rica.
E-mail address: doctortorrealba@gmail.com (G. Torrealba-Acosta).

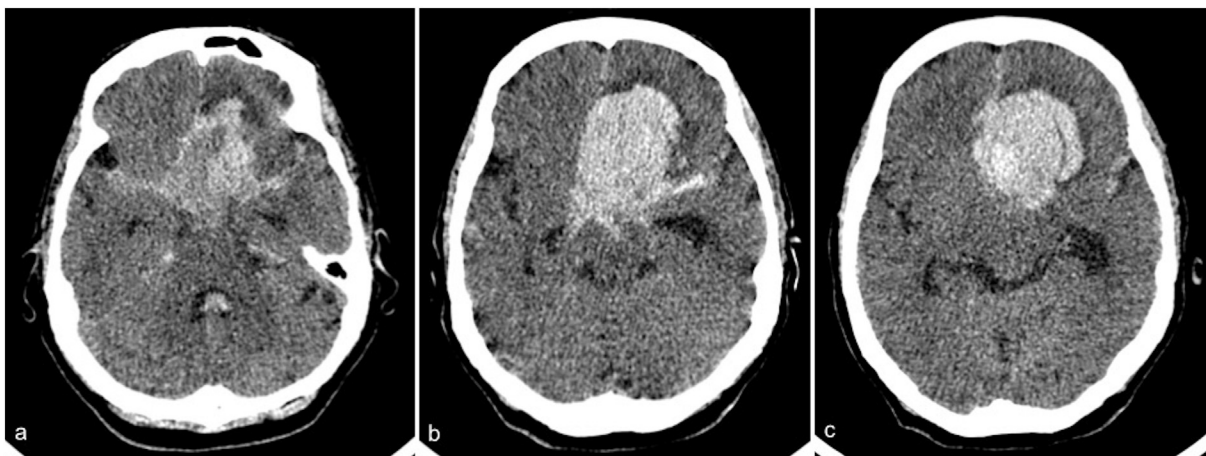


Fig. 1. Axial head CT scan showing an intraparenchymal hemorrhage originating from a pituitary apoplexy from a pituitary adenoma, that involves the left rectal gyrus and extends into the Sylvian and suprachiasmatic cisterns.

where we could eventually occlude the anterior superior sagittal sinus, through an interhemispheric subfrontal access, in the event of coming across a midline aneurysm. Likewise, we anticipated for an increased intracranial pressure that could also benefit from a bifrontal craniectomy.

Intraoperatively, we drained the hematoma uncovering a whitish friable mass with a discernible border. We were able to remove part of the lesion without any complications. However, during the surgery the extension of the edema hindered any further decompression in the perisellar area (Fig. 2). We deemed urgent to drain the hematoma, decompress the area and to obtain a biopsy of the observed lesion (Fig. 3). Nonetheless, any tumoral remnant could be later on intervened with a neuronavigated procedure probably through a transphenoidal route. After surgery, we confirmed the pituitary apoplexy and subsequent imaging studies never showed a progression of the ventricular dilation; rather a gradual resolution of the ventricular hemorrhage, precluding the clinical need for a ventriculostomy.

The patient later was transferred to the Neurocritical Care Unit where she had an adequate convalescence time. We recently saw her at the outpatient clinic. She still has some residual executive cognitive deficits, yet showing an almost complete recovery of her visual fields. She is currently under hormonal replacement therapy without any clinical manifestations of hypopituitarism.

3. Discussion

PA was first described in 1898 [1] and clinically defined as a syndrome that included: headache, vomiting, ophthalmoplegia and visual disturbances [2]. Also, several endocrine functions may be compromised due to concomitant panhypopituitarism [3]. Although local hemorrhage is common, intraventricular bleeding is mainly seen in autopsy and rarely it will present clinically or radiologically. Expansion of local intra-sellar bleeding towards the subarachnoid space may cause clinical manifestations similar to meningitis or a subarachnoid hemorrhage, which might be considered as differential diagnoses [3].

Since 1979 PA has been reported on computed tomographic findings associated with intraventricular hemorrhage [4]. It has been explained as occurring because of a suprasellar extension of the tumor bleeding through the floor of the third ventricle [5].

Most of pituitary adenomas presenting with a PA are defined as non-functioning; however, the syndrome can also be originated from secreting tumors. Accordingly, this syndrome has been shown also in patients with clinical manifestations of Cushing disease, acromegaly and prolactinomas [6,7,8].

Hemorrhage within the pituitary tumor as well as hemorrhagic extension to the ventricles, may be the result of three main mechanisms: disproportionate growth in relation to blood flow, concomitant use of anticoagulation therapy or pharmacological inhibition of the adenoma [8]. Although this rare presentation is mainly related to primary pituitary tumors, metastases of a distant tumor may also cause it.

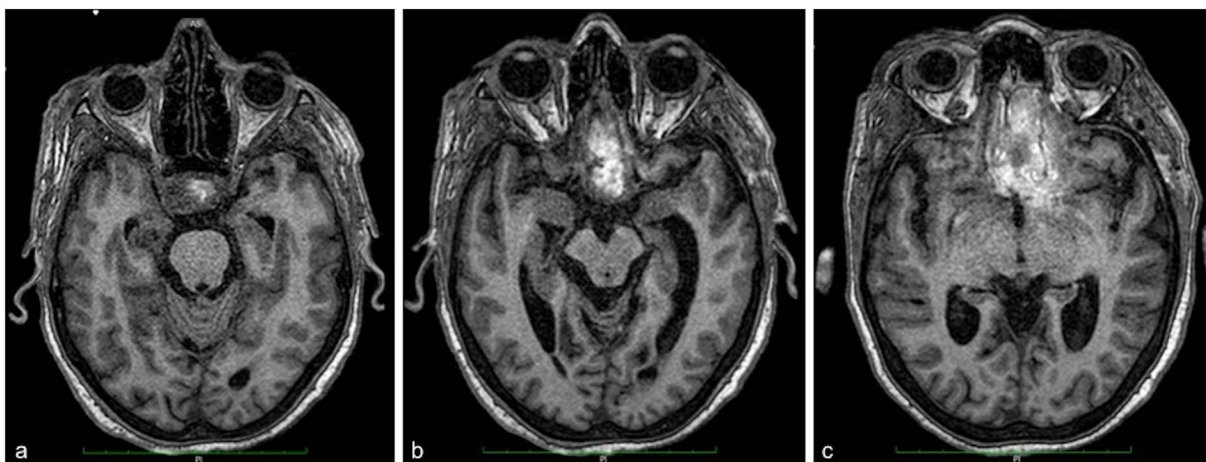


Fig. 2. Post-operative axial T1WI MRI showing a hyperintense lesion involving the hypophysis and extending into the interhemispheric region and left rectal gyrus.

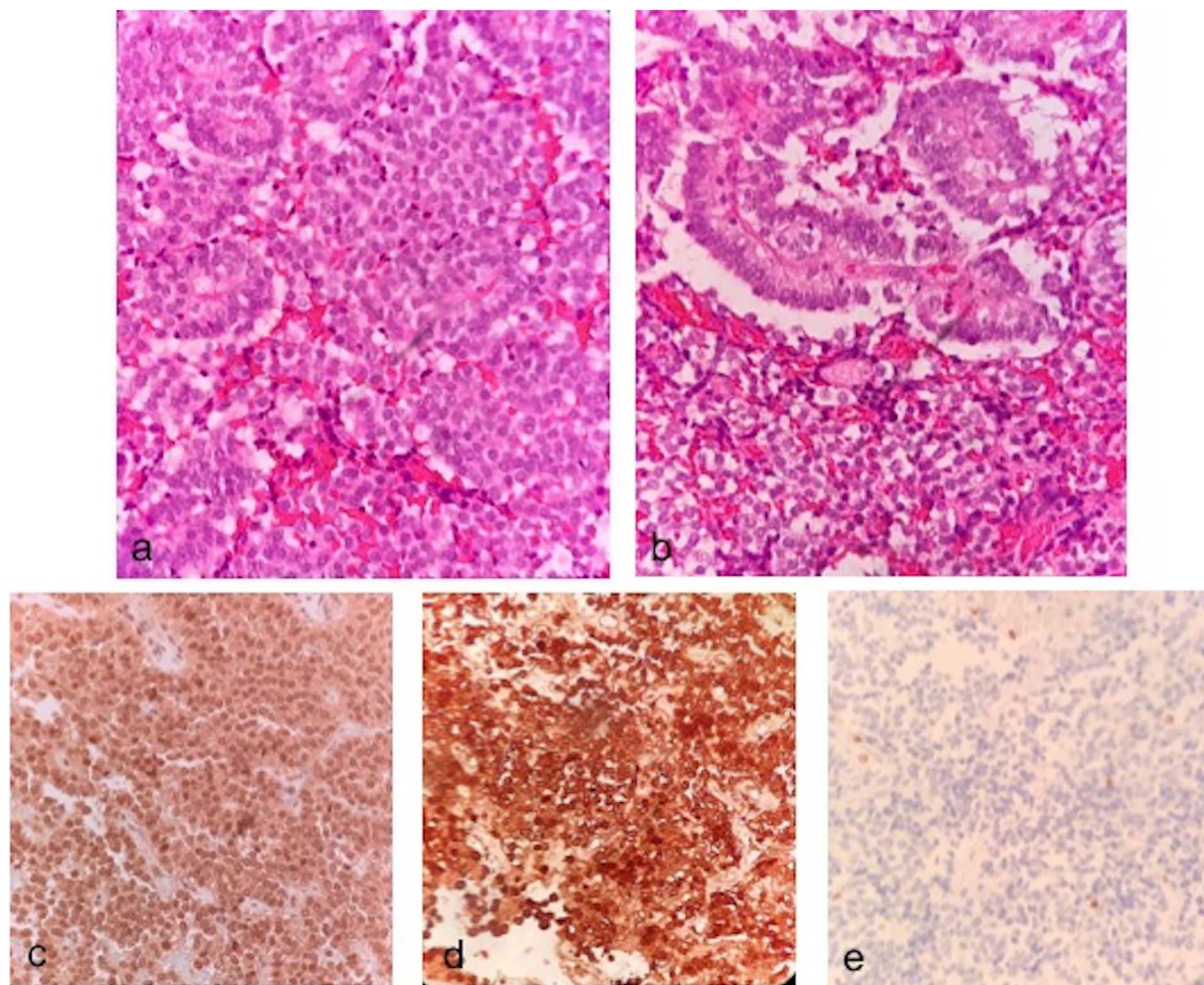


Fig. 3. Histologic appearance of pituitary adenoma (H & E, $\times 10$) (a, b) showing sheets and papillae composed of round cells with spherical and monomorphic nuclei with dense and regular chromatin. Tumor cells stained positive for prolactin (c) and growth hormone (d) on immunohistochemistry, with a Ki-67 index of approximately 1% (e).

For example: metastases from bladder tumors and renal cell carcinomas have been described affecting the pituitary, leading to vascular collapse of the gland [9,10].

Finally, because, PA can co-exist with cerebral aneurysms in up to 7.4% of subjects, an angiographic study must be performed even if the subarachnoid hemorrhage is thought to be due to a PA [11].

4. Conclusion

The case here reported showed how the clinical presentation and neuroimaging misled the diagnosis of a PA because of the subarachnoid and intraventricular hemorrhage findings. In this setting, a negative angiographic report for aneurysm must be followed by cerebral MRI, with a careful evaluation of the sellar region. If apoplexy is confirmed, an urgent decompression surgery must be a priority.

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Conflicts of interest

Gabriel Torrealba-Acosta: none.
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