CASE REPORTS

# An unrecognized endocrinology emergency masquerading as a hypertensive emergency: A can't miss diagnosis

Yanerys Agosto Vargas*[1], Sharon Velez Maymi[1], Paola Mansilla Letelier[1], Luis Raul Hernandez-Vazquez[1], Samayra Miranda Rodriguez[1], Michelle Mangual García[1], José Rodríguez León[2], José H. Martínez[1], Madeleine Gutierrez[1], Maria de Lourdes Miranda [1]

[1] *Department of Endocrinology, San Juan City Hospital, San Juan, Puerto Rico*
[2] *Department of Pathology, University of Puerto Rico, Medical Science Campus, San Juan, Puerto Rico*

**Received:** September 28, 2016 **Accepted:** November 29, 2016 **Online Published:** December 11, 2016
**DOI:** 10.5430/crim.v4n1p21 **URL:** https://doi.org/10.5430/crim.v4n1p21

## ABSTRACT

Pituitary apoplexy secondary to sellar tumors is a rare entity that carries a high mortality rate. It could be secondary to infarction or hemorrhage of the pituitary gland. The incidence remains unclear, most are reported in men between the ages of 50 to 60. In the majority of times, apoplexy is idiopathic in nature, without a clear discernible cause. However, there are multiple risk factors associated with this entity, such as systemic hypertension, among others. There are few cases of pituitary apoplexy caused by infarction of a pituitary macroadenoma. We present this case of pituitary apoplexy secondary to infarction of a nonfunctional pituitary adenoma in a young woman, with a fortunate resolution.

**Key Words:** Pituitary apoplexy, Pituitary macroadenoma, Hypertension

## 1. BACKGROUND

Pituitary apoplexy secondary to sellar tumors is a rare entity that carries a high mortality rate.[1–3] It may be caused by acute hemorrhages and/or infarction of a previously existing pituitary adenoma.[2] Pituitary apoplexy may be the first clinical finding associated with a pituitary tumor, including both functional and nonfunctional macroadenomas, with the latter being more prevalent.[4] The incidence of pituitary apoplexy remains unclear.[5] However, the majority of cases are reported in men, with a prevalence of 3%-20% of all pituitary tumors.[5] Rapid enlargement of a pituitary mass leads to compression of adjacent structures, presenting a sudden development of headache, visual disturbances, cranial nerves involvement, mental status changes and endocrine dysfunction.[1,2,4,6] We present this case of a pituitary apoplexy secondary to infarction of a nonfunctional pituitary adenoma in a young woman.

## 2. CLINICAL COURSE

A 38-year-old woman, Gravida 3 Partum 0 Abortum 3 (G3P0A3), menarche at 12-year-old regular cycle, with history of obesity, diabetes mellitus type 2, uncontrolled arterial hypertension and dyslipidemia, all diagnosed within a one year time frame, treated with metformin 500 mg daily, glipizide 2.5 mg daily, hydrochlorothiazide 12.5 mg daily, nifedipine 10 mg every 12 hours and losartan 100 mg daily,

*Correspondence: Yanerys Agosto Vargas, MD; Email: yankiav@gmail.com; Address: Yanery's Agosto Vargas Urb. Las Haciendas Calle Vereda Verde # 15 Canóvanas, PR 00729, Puerto Rico.

who presented to the emergency room (ER) complaining of frontal headaches, with associated nausea and vomiting, over the day preceding evaluation. Patient described her headache as oppressive and generalized in nature, rated 8/10 in intensity, non-radiating, without any provocative or palliative factors, nor any associated neurological symptoms, loss of consciousness or blurred vision. She had unquantified episodes of non-bloody emesis. Upon initial evaluation at the ER, she appeared acutely ill. Vital signs revealed markedly elevated blood pressure at 217/107 mmHg, with heart rate at 90 beats per minutes (bpm) and temperature at 36.7°C. Physical examination was remarkable for a non-focal neurological exam, with negative meningeal signs, preserved strength and sensation. Basic laboratories revealed mild leukocytosis with neutrophilic predominance, renal dysfunction with increased creatinine levels, significantly elevated blood glucose, but without acidosis or elevated osmolality, and negative cardiac enzymes. However based on patient's presentation, a presumptive diagnosis of hypertensive emergency with central nervous system as target was established. Intravenous infusion of clevidipine was promptly initiated. Symptomatic management of her nausea and vomiting was provided with intramuscular promethazine 25 mg and intravenous ondansetron 8 mg. She was then admitted to the Coronary Care Unit for further treatment and evaluation. CT scan of the head reported a 2.5 cm × 2.8 cm pituitary adenoma, without signs of intracranial hemorrhage (see Figure 1). As her condition allowed, antihypertensive medications were optimized to clonidine 0.3 mg PO every 6 hours, hydralazine 100 mg PO three times daily and hydrochlorothiazide 50 mg PO daily, leading to improvement of blood pressure. However, despite initial improvement, patient developed right eye ptosis with associated diplopia, anisocoria slightly reactive to light, blurred vision and persistent headaches during the second day of admission. Brain MRI/MRA showed an enhancing suprasellar mass with possible invasion into the right cavernous sinus and mass effect upon the optic chiasm (see Figures 2, 3). Biochemical endocrine evaluation, including Thyroid Stimulating Hormone (TSH), Insulin like growth factor 1 (IGF-1), Prolactin, Follicular stimulating hormone (FSH), cortisol, Adrenocorticotrophic hormone (ACTH), aldosterone were almost normal (see Table 1). Due to the aforementioned symptoms and MRI/MRA findings, Neurology and Ophthalmology services were consulted and agreed with physical findings that correlates with mass effect most likely due to optic chiasm compression. According to Ophthalmology service third nerve palsy was caused by optic chiasm compression. Neurosurgery service was also consulted and recommended a transphenoidal surgical approach for tumor resection. Histopathology reported an infarcted pituitary adenoma with extensive ischemic necrosis (see Figure 4). Reticulin stain revealed complete disruption of the reticulin fiber network, which supported the diagnosis of pituitary apoplexy. After surgery, patient achieved full recovery of her vision without residual deficits. She was eventually discharged home on optimized antihypertensive treatment.

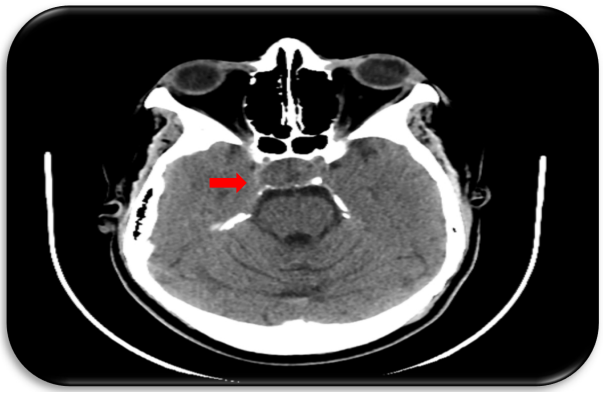

**Figure 1.** Head CT scan. Pituitary adenoma 2.5 cm × 2.8 cm, without signs of intracranial hemorrhage

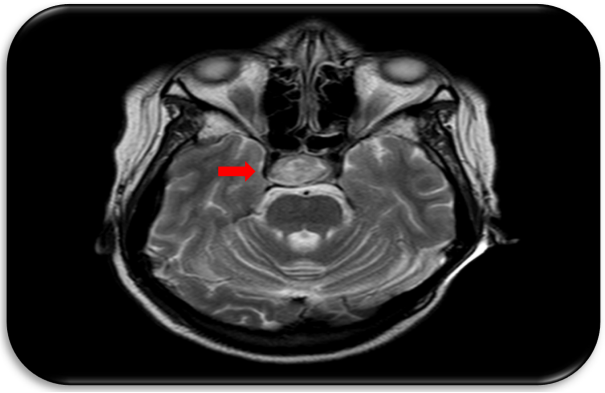

**Figure 2.** Brain MRI. Enhancing suprasellar mass 2.2 cm × 1.9 cm × 2.5 cm with possible invasion into the right cavernous sinus and mass effect upon the optic chiasm

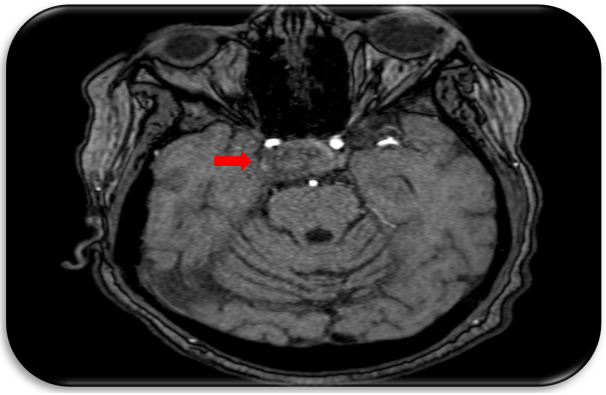

**Figure 3.** Brain MRA. Nine millimeters (9 mm) Outpouching suggested arising from the proximal A2 segment of the right anterior cerebral artery


ISSN 2332-7243 E-ISSN 2332-7251

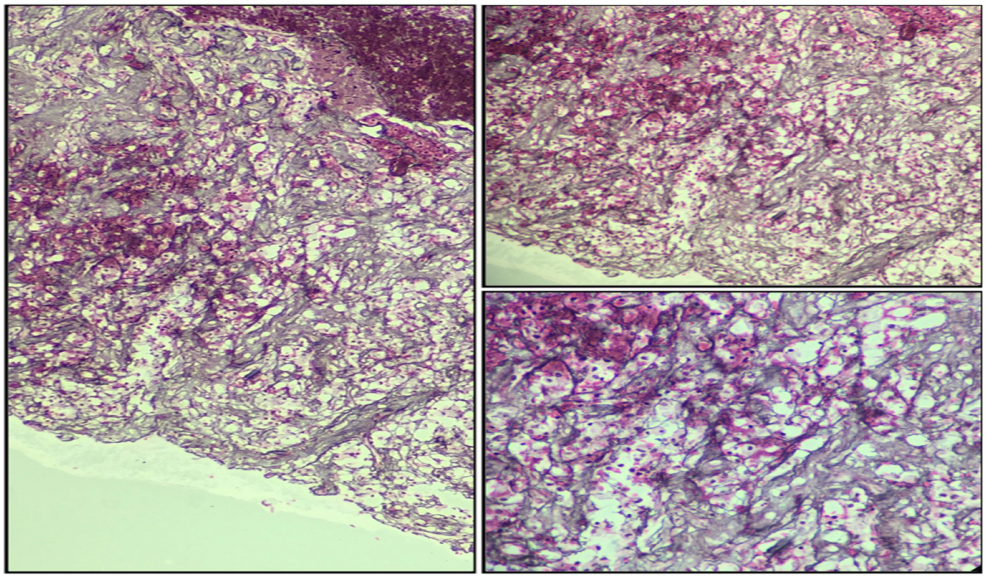

**Figure 4.** Pathology specimen. Reticulin Stain Infarcted pituitary adenoma with extensive ischemic necrosis

**Table 1.** Biochemical test while on admission

| Test | Value | Normal Value |
|---|---|---|
| Sodium | 137 | 136.0-145.0 mmol/L |
| Potassium | 4.4 | 3.6-5.0 mmol/L |
| Chloride | 97 | 98-111 mmol/L |
| Bicarbonate | 23 | 22.0-31.0 mmol/L |
| BUN | 18 | 7.0 -21.0 mg/dl |
| Creatinine | 1.1 | 0.52-1.04 mg/dl |
| TSH | 3.44 | 0.4-5.6 IU/ml |
| Total T4 | 11.07 | 6.09-12.23 µg/dl |
| Prolactin | 9.26 | 5.18-26.53 ng/ml |
| Diluted Prolactin | 23.6 | 5.18-26.53 ng/ml |
| ACTH | 41.3 | 0.00-46.00 pg/ml |
| Aldosterone | 3 | < 28 ng/ml |
| Cortisol AM | 17.8 | 6.2-29 MCG |
| IGF-1 | 108 | 109-284 ng/ml |
| FSH | 2.3 | 3.03-8.08 MU/ml |
| A1C | 11.5 | 4.2%-5.8% |

## 3. Discussion

Pituitary apoplexy was first described by Bailey in 1898.[3,6] However, it was Brougham who in 1950 evaluated known reported cases and established the syndrome now known as apoplexy.[7] This entity usually manifests with ocular palsy, visual field and visual acuity disturbances, nausea, vomiting, photophobia and hypertension.[1–9] Apoplexy may be encountered in patients with a rapidly growing pituitary adenoma, not identified until the development of symptoms related to the former.[9] A wide range of risk factors for the development of pituitary apoplexy have been reported in the literature, with poorly controlled arterial hypertension often identified as the culprit.[1,4,5,8] Other potential precipitating factors include pituitary irradiation, major surgery, coagulopathies, head trauma, pregnancy, dynamic pituitary gland studies, and hormone replacement therapy, among others. Hypertension induced degenerative microvasculature alterations are known to precipitate apoplectic episodes.[1–9]

The clinical features are quite variable.[7] Literature suggest the most commonly identified early manifestation is retro-orbital headache, as a result of stretching and irritation of the dura mater overlying the sella turcica.[4,5,7] Visual deficits, including cranial nerve palsy, are also common, secondary to lateral and optic chiasm compression (75%). Extravasation of blood or necrotic tissue into the subarachnoid space can cause meningeal signs such as fever, photophobia and altered mental status.[5,7] Our patient presented with intractable headache, uncontrolled arterial hypertension, unilateral opthalmoplegia, ptosis and decreased visual acuity.

A vast majority of patients suffering from pituitary apoplexy (nearly 80%) will have deficiency of at least one anterior pituitary hormone.[4] Panhypopituitarism, adrenal insufficiency, hypothyroidism, hypogonadism, growth hormone deficiency and diabetes insipidus may all occur as a result of pituitary apoplexy.[2,8] Fortunately, our patient did not develop any hormonal deficiency during the acute event.

Management of pituitary apoplexy is tailored by the patient's hemodynamic stability and on the presence of electrolytes disturbances.[8] According to current United Kingdom guidelines for the management of pituitary apoplexy, patients with hemodynamic instability, altered visual fields, altered men-

tation, and/or decreased visual acuity should be started on intravenous glucocorticoid therapy.[8] Furthermore, even if the patient is hemodynamically stable, but has serum cortisol lower than 550 mmol/L, intravenous glucocorticoids should also be considered.[8] Given that our patient remained hemodynamically stable during the admission, infusion of glucocorticoids was not required. There are controversies regarding conservative versus surgical management of pituitary apoplexy, with no definitive trial or case series providing evidence to support one over the other.[5] Decision should be reached by a multidisciplinary team while taking into consideration the patient's profile and presentation. Our patient was managed surgically due to the concerns over rapidly progressive visual changes.

Pituitary apoplexy is frequently misdiagnosed due to signs and symptoms that may mimic other, more common intracranial pathologies such as subarachnoid hemorrhage, meningitis, meningoencephalitis, migraine, stroke and cavernous sinus thrombosis.[3] High suspicious index is required for prompt diagnosis, in order to decrease morbidity and mortality.

## 4. Conclusion

Pituitary apoplexy is an endocrinology emergency that can often be misdiagnosed. It usually presents in middle aged men, with higher prevalence seen in non-functioning tumors. Prompt diagnosis has demonstrated to significantly decrease morbidity and mortality, such as in our patient, who had complete resolution of symptoms after tumor removal. The most likely precipitant factor of pituitary apoplexy in this patient was uncontrolled arterial hypertension. This case had an atypical presentation of a nonfunctional adenoma causing pituitary apoplexy, with a quite fortunate outcome. Our case serves to illustrate an interesting variation of the usual clinical presentation of pituitary apoplexy, as well as illustrating a potential mimicker of said diagnosis. Furthermore, it also demonstrates a quite rare cause of pituitary apoplexy, as non-functional tumors are rarely associated with the aforementioned clinical entity. This case report will advance the overall awareness of pituitary apoplexy as an uncommon, though complicated and potentially fatal diagnosis, if not recognized and treated properly.

## Conflicts of Interest Disclosure

The authors have declared no conflicts of interest.

## References


[1] Biousse V, Newman NJ, Oyesiku NM. Precipitating factors in pituitary apoplexy. J Neurol Neurosurg Psychiatry. 2001; 71: 542-545. PMid:11561045 https://doi.org/10.1136/jnnp.71.4.542

[2] Johnston PC, Hamrahian AH, Weil RJ, et al. Pituitary tumor apoplexy. Journal of Clinical Neuroscience. 2015; 22(6): 939-944. PMid:25800143 https://doi.org/10.1016/j.jocn.2014.11.023

[3] Kim JP, Park JP, Kim SB, et al. Pituitary apoplexy due to pituitary adenoma infarction. Journal Korean Neurosurgery Society. 2008; 43: 246-249. PMid:19096606 https://doi.org/10.3340/jkns.2008.43.5.246

[4] Ranabir S, Baruah MP. Pituitary Apoplexy. Indian Journal of Endocrinology and Metabolism. 2011; 15(Supplement 3): s188-s194. PMid:22029023 https://doi.org/10.4103/2230-8210.84862

[5] Rajasekaran S, Vanderpump M, Baldeweg S, et al. UK guidelines for the management of pituitary apoplexy. Clin Endocrinol. 2011; 74(1): 9-20. PMid:21044119 https://doi.org/10.1111/j.1365-2265.2010.03913.x

[6] Pal A, Capatina C, Tenreiro AP, et al. Pituitary apoplexy in nonfunctioning pituitary adenomas: long term follow up is important because of significant numbers of tumor recurrence. Clin. Endocrinol. 2011; 75(4): 501-504. PMid:21521336 https://doi.org/10.1111/j.1365-2265.2011.04068.x

[7] Sadek AR, Gregory S, Jaiganesh T. Pituitary apoplexy can mimic acute meningoencephalitis or subarachnoid hemorrhage. International Journal of Emergency Medicine. 2011; 4: 63. PMid:21975129 https://doi.org/10.1186/1865-1380-4-63

[8] Vargas G, Gonzalez B, Guinto G, et al. Pituitary apoplexy in nonfunctioning adenomas: a case control study. Endocrine Practice. 2014; 20(12): 1274-1279. PMid:25100377

[9] Yelwatkar SJ, Jain VV, Banote P. Apoplexy in pituitary macroadenoma presenting with total ophtalmophlegia and preserved vision: A rare entity. Journal of Mahatma Gandhi Institute of Medical Sciences. 2013; 18(2): 140-143. https://doi.org/10.4103/0971-9903.117797