Pituitary Apoplexy: A Pictorial Review.

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Learning objectives

Once pituitary apoplexy (PA) is suspected, CT and MRI are the imaging modalities of choice. Accurate interpretation of MRI and CT examinations requires an understanding of the pituitary' s anatomy, normal variants and common pathology.

The objetives of these review are to :

1. Know the clinical presentation of pituitary apoplexy (PA)

2. Understand the role of MRI and CT on its evaluation

3. Recognize its normal and pathologic imaging findings

Background

INTRODUCTION:

Pituitary apoplexy (PA) is a rare, life-threatening disorder caused by the expansion of a normal or neoplasic gland secondary to hemorrhage or infarction. It is twice more common in men than in women and the mean age of presentation is 57 y/o and it is uncommon in children.

Epidemiology:

Up to 65-90% of all PA occur in patietnts with pituitary macroadenomas. Anticoagulation, MEN I syndrome, dynamic pituitary functions test, radiation, bromocriptine therapy, trauma and great surgeries are also risk factors. The profile of the patients usually is a male with pituitary adenoma or a post-/peripartum female with hypovolemia and shock (Sheehan syndrome).

Signs and symptoms:

Its clinical presentation varies from benign to catastrophic, which may lead to permanent neurologic deficits or death. The pituitary sudden enlargement causes compression of the adjacent structures leading to a distinctive clinical setting characterized by sudden onset headache, visual impairment, ophtalmoplegia and autonomic/hormonal dysfunction. More serious manifestations are panhypopituitarism, acute adrenal crisis, shock and

disseminated intravascular coagulation. Long-term pituitary insufficiency is common in survivors.

Diagnosis and treatment:

PA requires a prompt diagnosis and treatment for diminishing morbidity and mortality rates. Imaging studies play an essential role since they can confirm the suspected clinical diagnosis, despict the etiology, evaluate the anatomy and assess the affected surrounding structures.

Accurate interpretation by MRI and CT of the pituitary gland requires an understanding of its anatomy, anatomic variants and common pituitary pathology. Technical factors of the MRI examination are also important.

Once made the diagnosis the treatment consists in surgical decompression, steroids and fluid/electrolyte replacement.

ANATOMY:

Normal anatomy:

Three components: Adenohypophysis (AH), Pars Intermedia (PI) and Neurohypophysis (NH).

-Adenohypophysis: 80% of gland. Wraps anterolaterally the NH. It is in charged of the secretion of STH, LTH, ACTH, TSH, FSH, ICSH, LH and MH. It has a portal circulation.

-Pars intermedia: < 5% of the pituitary. Located between AH/NH. Its function is to carry releasing hormones to the AD and NH.

-Neurohypophysis: 20% of pituitary gland. It is in charged of the storage and secretion of vasopressin and oxytocin. It has an arterial circulation and usually has a short T1, caused by vasopressin/oxytocin and presents a strong and uniform enhancement.

Normal variants:

Its size and configuration varies with age and between genders measuring # 6mm in children, 8 mm in males/post-menopausal females, 10 mm in young females and 12 mm in pregnant/lactating females.

The finding of an "Empty" sella is not unusual. It is caused by the protrusion of arachnoid CSF into the sella, which flattens and displaces posteroinferiorly the pituitary against the sellar floor.

Pituitary duplication is extremely rare with approximately 40 reported cases worldwide.

Up to 15-20% of normal patients have "filling defects" on T1 C+ MRI which correspond to cyst (Figure1) /nonfunctioning microadenomas.

DIFFERENTIAL DIAGNOSIS

Pituitary macroadenoma (nonhemorrhagic)

Craniopharyngioma

Rathke cleft cyst (Figure 1).

Pituitary abscess

Primary intrapituitary hemorrhage

Giant thrombosed intrasellar aneurysm

Images for this section:

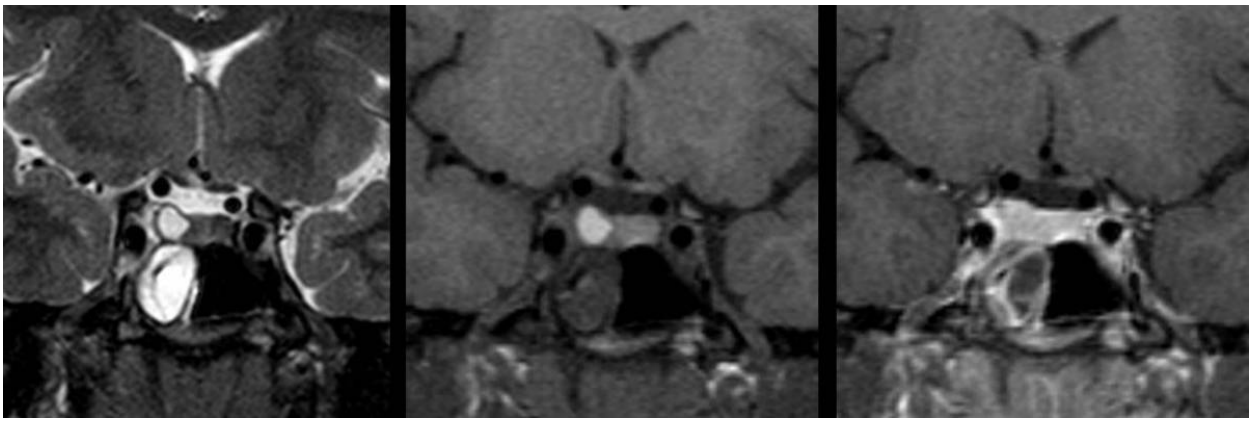

Fig. 1: 21 y/o female with Rathke cleft cyst. MRI images. From left to right T2, T1 and T1+G.

Findings and procedure details

MRI PROTOCOL:

In our center the standard protocol for studying the pituitary gland consists of:

-Sagittal and Coronal T1SE

-Coronal T2TSE

-Sagittal and Coronal T1SE after the administration of contrast. We use Gadobutrol 0.5 mmol/ml (Gadovist®) 0.1 cc/Kg at 2ml/s followed by 30cc of saline solution.

-In the case of lesions smaller of 10mm we perform a dynamic study.

IMAGING FINDINGS:

In the setting of an acute PA, images studies classically depict a > 10mm pituitary mass in an intra or combined intra-suprasellar location. It usually presents a "Snowman" or "figure of eight" morphology and may present hemorrhage (figure 2). Peripheral enhancement may also be present which is suggestive but not diagnostic of PA.

NETC: in acute stages (first 3 days) it is useful in the diagnosis of an hemorrhagic infarction. Hemorrhagic infarction is visualized as an enlarged sella with patchy or confluent spontaneous hyperdensities (Figure 3 and 4). TC is less useful in subacute (4 days to 1 month) and in chronic (> 1 month) hemorrhages since they may be confused with cystic degeneration, abscesses, and bland infarction, as all of these have lower absorption coefficients.

CECT: rim enhancement is suggestive but not diagnostic of pituitary apoplexy (Figure 3).

MRI: is more sensitive for detecting hematomas, especially in subacute and chronic stages, as well as identifying pituitaryadenomas. Findings by temporal evolution include:

<u>Acute:</u>

-Enlarged gland.

-T1WI iso-/hypointense with brain.

-T2WI hypointense (hemorrhagic) or hyperintense (nonhemorrhagic) pituitary. Acute compression of hypothalamus and optic chiasm may cause hyperintensity along optic tracts.

-Hyperintense in FLAIR (figure 4).

-DWI Restricted diffusion within adenoma may be an early sign of apoplexy with markedly decreased signal intensity in ADC map.

-T1WI C+ Rim enhancement is common (figure 6). Adjacent dural thickening and enhancement in 50% of cases.

-Thickening of sphenoid sinus mucosa in 80% of cases.

Subacute:

-Hyperintense in T1WI and T2WI.

Chronic:

-"Empty" sella (filled with CSF) hypointense in T1 and FLAIR, hyperintense in T2.

-Small pituitary remnant isointense in T1.

-T2* GRE: "Blooming" if blood products present

Images for this section:

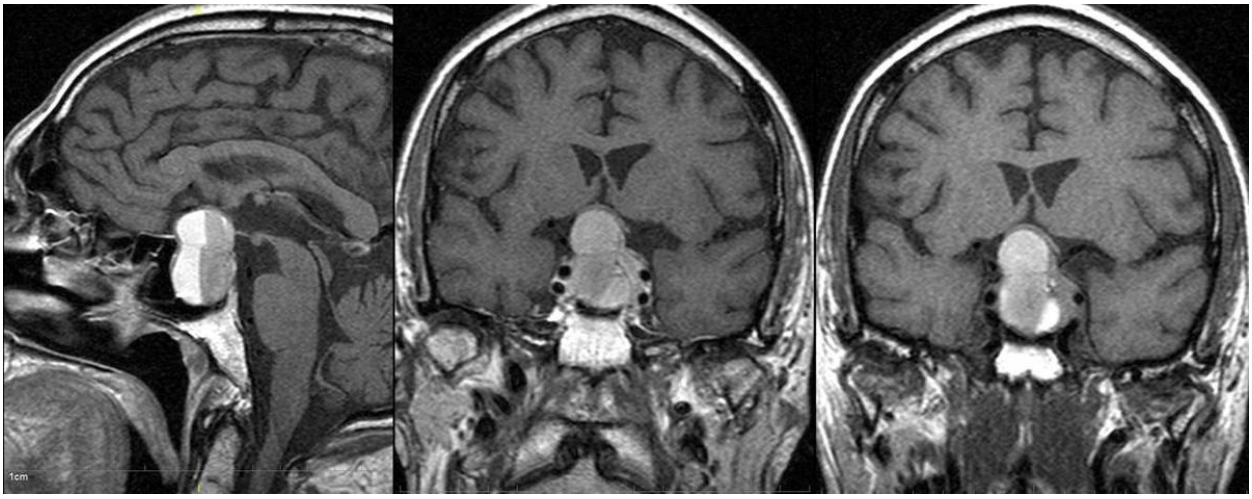

Fig. 2: 64 y/o with acute right eye visual impairment, headache and hyponatremia. Combined intra-suprasellar pituitary mass with a "Snowman" morphology compatible with hemorrhagic pituitary apoplexy. Left: sagittal T1. Center: T1. Right T1+G.

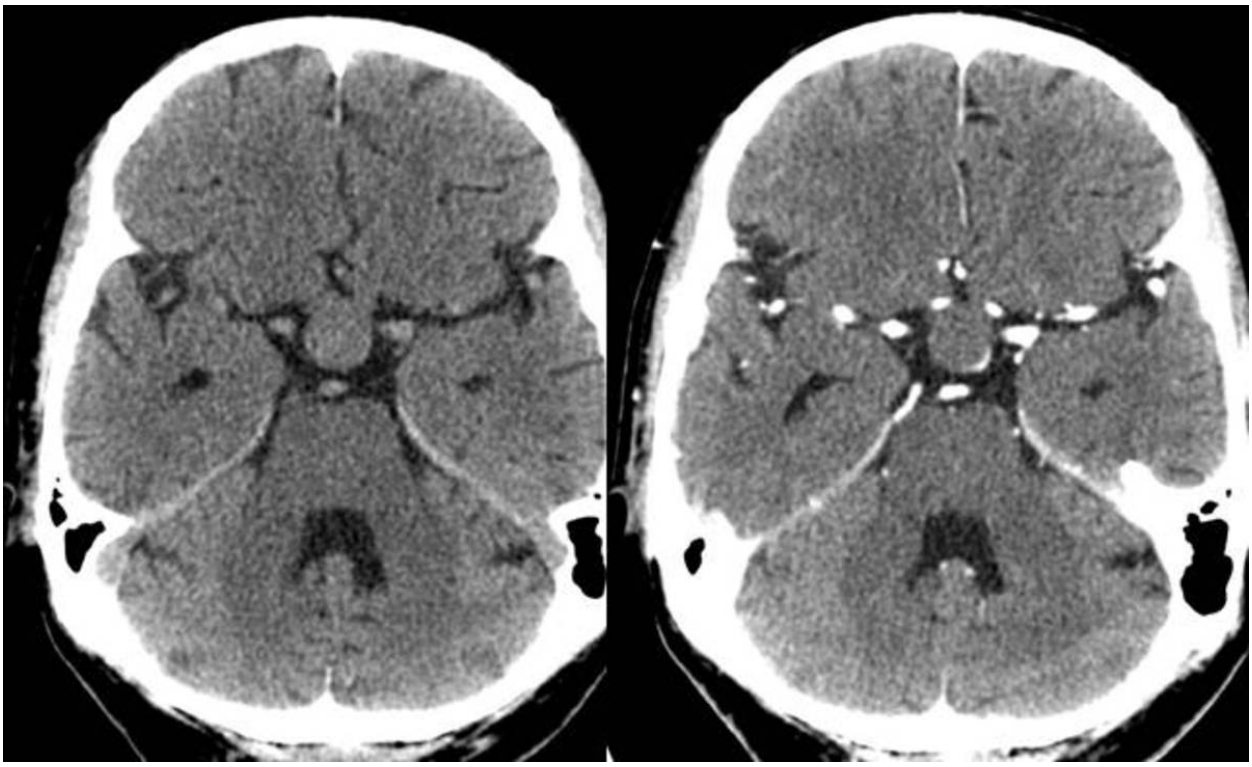

Fig. 3: 64 y/o male with sudden onset headache. Left NECT: spontaneously hyperdense pituitary gland. Right CECT: peripheral enhancement.

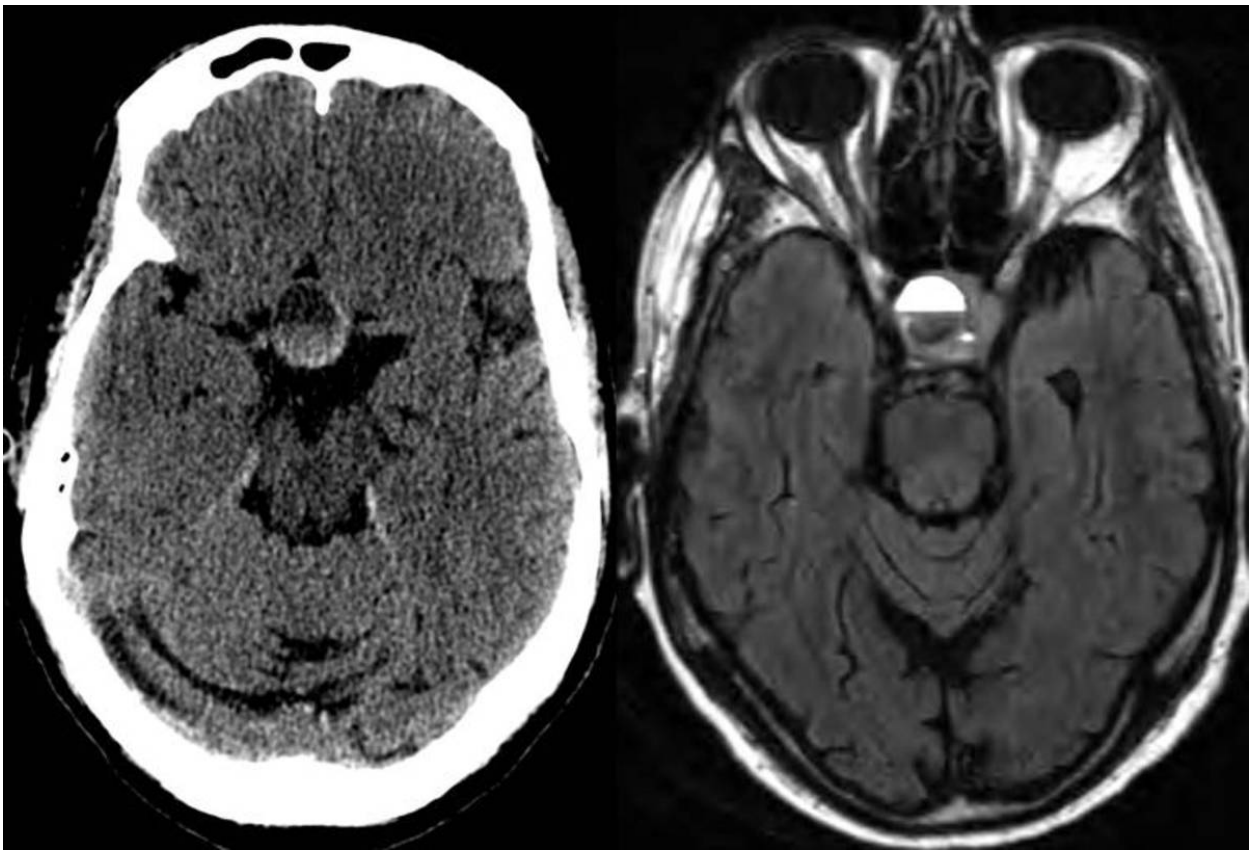

Fig. 4: 64 y/o with acute right eye visual impairment, headache and hyponatremia. Left: NECT shows liquid-liquid level suggestive with acute hemorrhage. Right: T2

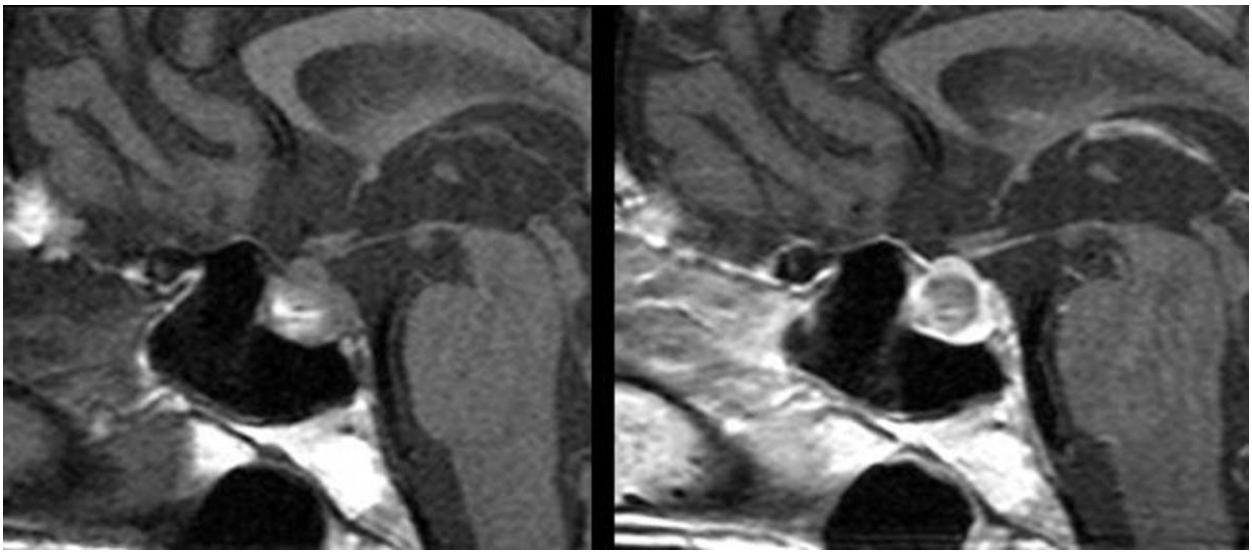

Fig. 5: 66 y/o male with MEN I syndrome, Cushing disease and panhypopituitarism. Left: T1 spontaneous hyperintense images suggestive of hemorrhage. Right: T1+G shows peripheral enhancement.

Conclusion

A high clinical suspicion and knowledge of the imaging appearances of PA are cardinal in the diagnosis, therapeutic management and prognosis of these patients. For these reasons, the radiologist must be aware of the imaging characteristics of this disease.

Personal information

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