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Contribution of Imaging in the Management of Pituitary Macroadenomas. About Three Observations in Casamance (South Senegal)

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ABSTRACT

Pituitary macroadenoma is a tumor lesion that is most often benign, non-encapsulated, non-secreting, larger than 1 cm and developed at the expense of the pituitary gland. The seriousness of this condition lies in the possibility of compression of the noble structures around the pituitary lodge, in particular the cavernous sinus, the optic pathways, the temporal lobe. The most feared complication is the occurrence of ischemic or hemorrhagic changes in the tumor called pituitary apoplexy. This complication can affect the patient's prognosis. This is especially true since the lesion can be discovered by chance, at an advanced age and most often in patients with multiple pathologies. Apart from these situations, the discovery can follow endocrine symptoms; which is rare. However, pituitary apoplexy is described in other lesions such as craniopharyngioma. These different presentations of the tumor must lead us to efficient management to avoid the occurrence of very serious complications. The aim of this article is to remind through 3 observations the clinical aspects, the imaging aspects, the complications and the management of this condition.

Keywords

Computed tomography scan, Pituitary macroadenoma, Craniopharyngioma, Apoplexy.

Introduction

The pituitary macro adenoma is a tumor lesion most often benign of more than 10 mm developed at the expense of the pituitary gland. It is discovered in the presence of endocrine symptoms or compressive phenomena on the visual pathways or neighboring anatomical structures. Or sometimes fortuitously during brain imaging performed for another independent reason. In our context, the endocrine assessment is difficult to obtain for technical or social reasons or simply due to a lack of specialized care. For a fortuitous discovery, the management must be the same because the risk of a complication such as pituitary apoplexy could be formidable. In this article, we illustrate the anatomo-radiological and prognostic aspects of 3 observations of pituitary macro adenomas and will address different epidemiological, clinical aspects, the management by imaging and the therapy of this pathology.

Observations

Observation 1

Mrs. SD, 67 years old, was referred to the imaging department for recent isolated amnesia and spatial disorientation without neurological deficit or impaired consciousness. She underwent a brain CT scan without and with contrast injection which revealed a sella turcica enlarged by a tumor process of 28 x 18 x 18 mm. The lesion is enhanced after injection and has a small lacuna within it. It exerts a mass effect on the left temporal lobe without visible edema. It was classified as grade 4 of the Knosp classification (Figure 1A).

She had undergone trans-sphenoidal sellar surgery. The post-surgical follow-up at 3 months had shown a para-sellar tumor residue of 17 x 12 x 9 mm. The previous symptoms had disappeared, in particular memory disorders (Figure 1B) and disorientation.

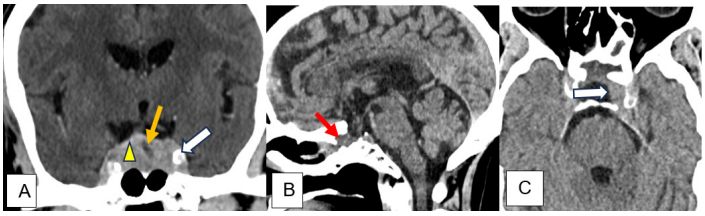

Figure 1: CT scan of the skull with injection. The coronal section shows in A a tissue process widening the sella turcica (yellow triangle) and invasion of the left cavernous sinus with a discrete mass effect on the temporal lobe opposite (white arrow in 1A). We also note a lacuna in the macroadenoma (thin arrow). The subsellar changes reflect the transsphenoidal pathway (arrow 1B) with a tumor residue visible on the left (arrow 1C).

Observation 2

Mr. BC, 84 years old, with a history of stroke, was referred to the imaging department for exploration of a febrile coma preceded by excruciating headache. The symptoms had been evolving for 3 days. Three months previously he had undergone a CT scan in another hospital structure and the rereading of which showed a tissue process of the macroadenoma type. The CT scan confirmed the sequelae of ischemia and showed a hemorrhagic macroadenoma increased in size compared to the previous examination and presenting spontaneously hyperdense hemorrhagic areas and associated with a meningeal and intraventricular hemorrhage and hydrocephalus. There was no aneurysm. He was admitted to intensive care and death occurred after 3 days (Figure 2).

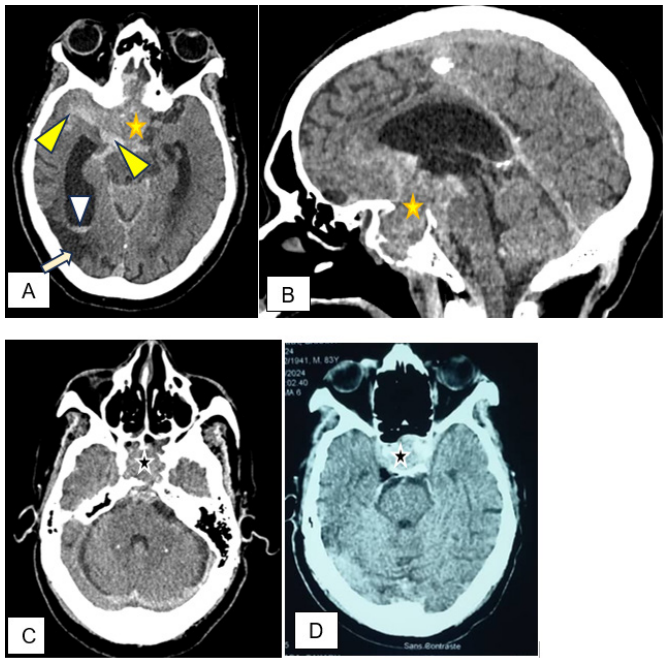

Figure 2: Contrast-enhanced CT scan showing a partially hemorrhagic pituitary tissue mass enlarging the sellar lodge (star in A, B, and C) and associated with meningeal hemorrhage (yellow arrowhead) and intraventricular hemorrhage (white arrowhead) and hydrocephalus. There is parenchymal hypodensity following ischemia opposite the right temporal horn of the ventricle (arrow). The CT scan performed one month earlier had shown a macroadenoma (star in D).

Observation 3

Child BA, 14 years old, was referred for imaging for assessment of impaired consciousness and recent blindness associated with intracranial hypertension and papillary edema. His CT examination with injection showed a normal sella turcica surmounted by a suprasellar mass with calcification, a cystic portion (20 HU) slightly denser than the cerebrospinal fluid and a spontaneously hyperdense portion (57 HU) not enhanced. Compression of V3 had caused active Tri ventricular hydrocephalus. V4 had a normal appearance (Figure 3). The diagnosis was that of apoplexy due to craniopharyngioma.

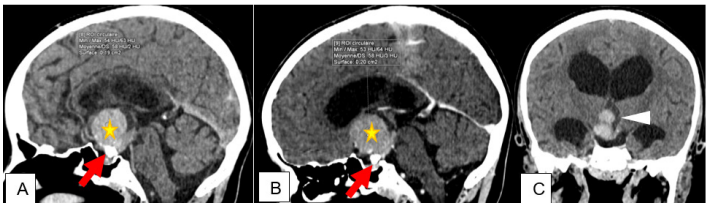

Figure 3: In this 14-year-old patient, CT scan showed a mixed suprasellar lesion, with calcification (arrow), hemorrhage (star) not enhanced after injection and cystic (arrowhead) in favor of a hemorrhagic craniopharyngioma. Compression of V3 is responsible for biventricular hydrocephalus.

Comments

Epidemiology and predisposing factors

Pituitary tumors rank second (16.8%) among central nervous system tumors after meningiomas (37.6%) according to the United States Central Nervous System Tumor Registry [1]. Most pituitary tumors arise from the anterior pituitary.

The size classification distinguishes microadenomas smaller than 1 cm and macroadenomas larger than 1 centimeter. The latter are twice as common as microadenomas. Giant adenomas are larger than 4 cm [1].

Pituitary macroadenoma is a benign, non-metastatic, non-encapsulated and most often non-secreting tumor proliferation [2]. 95% of pituitary adenomas are sporadic. However, genetic conditions can be a factor in the occurrence of this tumor, such as multiple endocrine neoplasia type 1, familial isolated pituitary adenoma. The adenoma can also be associated with the occurrence of pheochromocytoma and paraganglioma in the context of a mutation affecting succinate dehydrogenase, or with McCune Albright syndrome, or with X-linked Acro gigantism. These genetic factors are a source of tumor aggressiveness and recurrence. They are therefore important to look for in young people for better management [1].

Clinical aspects

Macroadenomas are most often hormonally silent. In addition, the

lesion must reach a significant volume to compress neighboring structures. This evolutionary profile allows the discovery of the macroadenoma fortuitously (incidentaloma) during imaging performed for another reason [1,3]. The macroadenoma can be discovered in the event of excessive development compressing adjacent anatomical structures; the optic pathways and the chiasm in particular. Blindness is thus explained in observation 3. The adjacent temporal lobe could also be compressed. The patient in case 1 presented with memory loss and recent disorientation attributed to compression of the left temporal lobe since the symptoms completely disappeared after partial excision of the macroadenoma.

Pituitary apoplexy is a serious complication that can be life-threatening. The first observations date back to 1898 [3-5] and were then defined as a clinical syndrome by Brougham et al in 1950. Pituitary apoplexy is the most often sudden occurrence of a hemorrhage, a hemorrhagic infarction or an infarction within a macroadenoma, whether secreting or not. It typically manifests itself with clinical signs such as intense headaches, sometimes "thunderclap" headaches, vomiting, cranial nerve damage, panhypopituitarism and impaired consciousness. A clinical picture that justifies emergency brain imaging [3,6,7]. Opinions are divided on the frequency of occurrence of infarction as a cause of apoplexy. Most authors agree on the rarity while Semple reported about 50% of causes on a series of 55 cases of macroadenoma [8]. The genesis of the infarction could be multifactorial; circulatory collapse, a reduction in tumor perfusion, vascular compression by tumor expansion or vasculopathy.

Although many factors remain unknown in the genesis of pituitary apoplexy, some are considered to be contributory, such as pregnancy, endocrine stimulation tests, pituitary irradiation, anticoagulant treatment or treatment for erectile dysfunction, minor head trauma, diabetes mellitus or ketoacidosis, cerebral angiography, hemodialysis, surgery (appendectomy, thyroidectomy, laminectomy). Some of them may therefore require pre-therapeutic imaging to eliminate a possible clinically silent macroadenoma [6,8,9]. Does the secretory aspect of the adenoma play a role in the genesis of the infarction? In the cases reported by Kim and Alarifi, ACTH hormonal secretion was found [6,10].

Apoplexy can occur on other sellar or perisellar lesions such as a pituitary cyst, hypophysitis, microadenoma or craniopharyngioma. Case 3 shows a hemorrhagic and calcified suprasellar lesion with a sella turcica of normal size suggesting apoplexy on craniopharyngioma (Figure 3).

Sheehan's syndrome or hemorrhagic or ischemic phenomena discovered in histopathological studies or surgically are not considered pituitary apoplexy [3,9].
However, the differential diagnoses for pituitary macroadenomas are essentially suprasellar tumor lesions, particularly craniopharyngioma, Rathke's cyst, and primary or secondary bone tumor lesions of the clivus. An important negative sign for all these lesions is the absence of widening of the sella turcica. Semiological analysis of the lesions provides important diagnostic guidance.

Imaging

The role of imaging in a pituitary macroadenoma is to perform a comprehensive loco-regional assessment. To do this, a CT scan of the skull without and with injection may be sufficient. MRI, if available, should be performed for its best resolution. It is not necessary to make millimeter slices on the sella turcica, the lesion being large enough to be significantly visualized in the three planes of space both on CT and MRI.

Computed tomography is usually the first option for examination in the clinical presentation of pituitary apoplexy because of its availability and speed in an emergency situation. The diagnosis of macroadenoma is easy in the presence of an enlarged sella turcica containing a tissue process (Figures 1 and 2). Computed tomography detects hemorrhage in the form of spontaneous hyperdensity in the macroadenoma in 20 to 40% of cases as illustrated in Figure 2. Its efficiency in detecting spontaneous hyperdensity is good in the first 6 hours (acute phase). Beyond this, the hemorrhage tends to become isodense then hypodense [7]. In the acute phase, because the hemorrhage appears isointense, MRI can miss it. Beyond this phase, MRI is the technique of choice for detecting hemorrhage. Its performance is approximately 89%. The performance of MRI is due to its ability to detect different hemoglobin metabolites on different T1, T2 sequences [7,11]. What imaging is preferred? In our practice only CT is available more in our region. But if MRI is available, it should be performed.

Meningeal hemorrhage associated with apoplexy is a rare situation that makes observation 2 special. Kim also reported a similar case. In case of association with meningeal hemorrhage, imaging must formally eliminate an underlying aneurysm rupture [6].

The imaging assessment specifies:

- The relationship with neighboring organs (chiasma, V3, temporal lobe)
- The extension into the cavernous sinuses by the Knosp classification [12]

The latter makes it possible to establish the prognosis better than the Hardy-Wilson classification. The SIPPAP classification (S: superior, I: inferior, P: right parasellar, P: left parasellar, A: anterior, P: posterior), which takes into account the extension into the spaces adjacent to the sellar lodge, makes it possible to combine the different classifications into one. It was proposed by Edal et al. in 1997. The measurements of the lesion must be specified in the 3 planes [13,14].

Imaging also plays a role in post-surgical monitoring or in the event of therapeutic abstention. In the latter case, systematic research into risk factors must be carried out in order to avoid the occurrence of apoplexy. For imaging, this involves the search for cerebral vascular lesions

Support

Patient management is generally guided by clinical symptoms. However, the incidental discovery of lesions is often problematic due to the absence of symptoms associated with the lesion, which nevertheless, presents the same evolutionary potential compared to a symptomatic lesion.

Our unfavorable socioeconomic context is an obstacle to in-depth exploration and adequate management. In view of these observations presented above, we believe that it is necessary to manage a macroadenoma discovered incidentally as if it were symptomatic.

Patients should undergo a clinical assessment, particularly looking for damage to the cranial nerves and visual pathways, and an endocrine biological assessment. The search for risk factors for apoplexy is essential. Hormonal treatment can significantly regress the lesion without resorting to surgery for prolactin adenomas. Spontaneous regression is also possible [15].

All these elements will allow to judge a surgery in order to prevent apoplexy whose prognosis is generally poor. The trans-sphenoidal route offers in view of the observation 2 an interesting minimally invasive surgical possibility. In the context of apoplexy, surgery by trans-sphenoidal route and a replacement treatment by corticoid gives good results [16].

Conclusion

The diagnosis of macroadenoma is relatively simple and must be considered in the presence of any tissue process widening the sella turcica. Unless there is a complication, the lesion is most often isodense. Apoplexy is a dramatic situation reflecting a hemorrhagic or ischemic transformation or sudden hemorrhagic ischemia, the prognosis of which is quite poor. This therefore requires that risk factors and symptoms be taken into account in the management, especially in the event of an incidental discovery. Transsphenoidal surgery is a less invasive therapeutic option with good results.

References

1. Shih RY, Schroeder JW, Koeller KK, et al. Primary Tumors of the Pituitary Gland:Radiologic-Pathologic Correlation. Radiographics. 2021; 41: 2029-2046.
2. Dekkers O. M, A M Pereira, J A Romijn, et al. Nonfunctioning pituitary macroadenomas: treatment and long-term follow-up. J Clin Endocrinol Metab. 2008; 93: 3717-3726.
3. Boellis A, Di Napoli A, Romano A, et al. Pituitary Apolplexy: an update on clinical and imaging features. Insights Imaging. 2014; 5: 753-762.
4. Bailey P. Pathological report of a case of acromegaly with special reference to the lesions in hypophysis cerebri and in the thyroid gland and a case of hemorrhage into the pituitary. Phila Med J. 1898; 1: 789-792.
5. Brougham M, Heusner AP, Adams RD, et al. Acute degenerative changes in adenomas of the pituitary body with special reference topituiary apoplexy. J Neurosurg. 1950; 7: 421-439.
6. Joo PK, Bong JP, Sung BK, et al. Pituitary Apoplexy due to Pituitary Adenoma Infarction. J Korean Neurosurg Soc. 2008; 43: 246-249.
7. Donegan D, Erickson D. Revisiting Pituitary Apoplexia. J Endocr Soc. 2022; 6: 1-10.
8. Semple PL, Webb MK, De Villiers JC, et al. Pituitary apoplexy. Neurosurgery. 2005; 56: 65-73.
9. Del Valle MM, De Jesus O. Pituitary Apoplexy. https://www.ncbi.nlm.nih.gov/books/NBK559222/
10. Alarifi A, Alzahrani AS, Salam SA, et al. Repeated remissions of cushing disease due to recurrent infarctions of an ACTH producing pituitary macroadenoma. Pituitary. 2005; 8: 81-87.
11. Goyal P, Utz M, Gupta N, et al. Clinical and Imaging Features of pituitary apoplexia and role of imaging in differenciation of clinical mimics. Quant Imag Med Surg. 2018; 8: 219-231.
12. Araujo-Castro M, Cancella AA, Vior C, et al. Radiological Knosp, Revised Knosp and Hardy-Wilson Classifications for the Prediction of Surgical Outcome in the Endoscopic Endonasal Surgery of Pituitary Adenomas: Study of 228 cases. Front Oncol. 2022; 11: 807040.
13. Sirat S, Anwar M, Hilal K, et al. Magnetic resonance imaging grading of pituitary macroadenoma – SIPAP classification revisited. Eur J Radiol Open. 2023; 10: 100486.
14. Edal A L, Skjödt K, Nepper-Rasmussen HJ. Et al. SIPAP--a new MR classification for pituitary adenomas. Suprasellar, infrasellar, parasellar, anterior and posterior. Acta Radiol. 1997; 38: 30-36.
15. Santos ABA, França MM, Hirosawa RM, et al. Conservative management of pituitary tumor apoplexy. Conservative management of pituitary tumor apoplexy. Arq Bras Endocrinol Metab. 2011; 55: 345-348.
16. Lee JH, Kim JH, Moon KS, et al. Pituitary Apoplexy: Surgical Experience with 16 Patients. J Korean Neurosurg Soc. 2007; 42: 83-88.