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The various MRI patterns of pituitary apoplexy

M. Piotin[1, 2], D. Tampieri[1], D. A. Rüfenacht[2], G. Mohr[3], M. Garant[1], R. Del Carpio[4], F. Robert[5], J. Delavelle[2], D. Melanson[1]

[1] Department of Radiology, Montreal Neurological Hospital and Institute, H3A 2B4 Montreal, Canada
[2] Department of Diagnostic and Interventional Radiology, Geneva University Hospital, CH-1211 Geneva 14, Switzerland
[3] Department of Neurosurgery, Sir Mortimer B. Davis Jewish General Hospital, H3T 1E2 Montreal, Canada
[4] Department of Radiology, Montreal General Hospital, H3G 1A4 Montreal, Canada
[5] Department of Pathology, Sir Mortimer B. Davis Jewish General Hospital, H3T 1E2 Montreal, Canada

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Abstract. The aim of this study was to describe the various MRI features, in correlation to surgical and pathological findings, in patients who presented with pituitary apoplexy (PA). Eleven patients presenting with PA, were evaluated with various MR protocols including spin-echo (SE) T1-weighted sequences in 9 of 11 patients, post gadolinium SE T1-weighted sequences in only 8 of 11 patients, and with T2-weighted SE sequences in 2 of 11 patients. All patients had transsphenoidal pituitary surgery after MR studies. The severity of presenting symptoms ranged from headaches to coma. Ten patients had pituitary macroadenoma; one had a non-hemorrhagic metastatic lesion into a non-adenomatous pituitary gland. Of the 11 patients, one was studied at the acute stage of PA (1 day after onset), 9 at the subacute period (3–15 days after onset), and one at the late stage (5 months after onset). Images compatible with intratumoral hemorrhage were found in all macroadenomas, whereas the metastatic pituitary lesion did not show evidence of bleeding. All gadolinium-enhanced studies showed partial tumoral enhancement. The SE T2-weighted studies demonstrated areas of low and high signal intensities in keeping with the presence of blood degradation contents. Pituitary apoplexy present with different MR features, including hemorrhagic and non-hemorrhagic characteristics on T1-weighted images. Gadolinium-enhanced images do not provide complementary diagnostic information when the presence of blood is assessed on plain images.

Key words: Pituitary apoplexy – Pituitary hemorrhage, – Pituitary tumors – MR imaging

Correspondence to: M. Piotin

Present address: 4 rue Euryale Dehaynin, F-75019 Paris, France

Introduction

Pituitary apoplexy (PA) is an acute clinical syndrome caused by degenerative changes of vascular origin, both necrotic and/or hemorrhagic, involving tumorous and non-tumorous pituitary glands [1]. Pituitary apoplexy is more often due to acute degenerative changes turning into pre-existing pituitary adenoma. The degree of hemorrhage and edema within the gland cause the mass effect, determining the presence and extent of the neurological signs and symptoms [2]. On CT an apoplectic pituitary tumor presents as a heterogeneous mass with areas of hyperdensity corresponding to the presence of hemorrhage. After contrast material injection, the infarcted part of the tumor remains unaltered, whereas the periphery exhibits ring-like enhancement, depending on the time after onset of PA [3, 4]. Pituitary macroadenomas and other large parasellar lesions are assessed better with MR because of the inherent contrast of signal intensity (SI) relative to surrounding structures. Furthermore, MR imaging has been proven to be superior to CT in identifying cavernous sinus invasion and optic chiasm compression [5, 6]. The usefulness of MRI in evaluating PA has been reported previously, and MRI has been shown to be superior to CT in detecting apoplectic changes, at least in the subacute period [3, 7–13]. The purpose of this article is to describe the different MRI features of PA at various stages after clinical onset, and to correlate the MRI patterns with surgical and pathological findings.

Patients and methods

Eleven patients (7 men and 4 women; age range 27–83 years, mean age 54 years) presenting with neurological symptoms suggestive of PA were studied by MRI at 1.5 T. Examinations were conducted at three medical centers and included spin-echo (SE) coronal and sagittal T1-weighted images with the following parameters: TR 370–650 ms; TE 15–30 ms; field of view 160–210 mm;

slice thickness 2.5–5 mm. Coronal SE T2-weighted images were obtained in 2 cases. Of the 11 patients, 8 had gadolinium-enhanced studies immediately after 0.05 or 0.1 mmol/kg IV administration of gadolinium-DTPA. The time elapsed between the clinical onset and the MR study ranged from 1 day to 5 months. All patients had microsurgical transsphenoidal tumor removal within 21 days after MRI. All surgical specimens were evaluated histologically, including immunohistochemical staining with antibodies to prolactin, growth hormone (GH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), thyroid-stimulating hormone (TSH), and adrenocorticotropic hormone (ACTH).

Results

Clinical presentation

The patients presented with various clinical symptoms with a severity ranging from severe headaches to coma (see Table 1). All patients had partial to total post-ictal hypopituitarism requiring hormonal substitution. No mortality occurred.

MRI findings

T1-weighted imaging. All patients had evidence of a pituitary lesion extending upward to the optic chiasm. All patients had bilateral cavernous sinus compression or sinus invasion, or both. In all but one case (case 6), the presence of intralesional areas of high SI was suggestive of the presence of blood. The high signal intensities were generally more pronounced in the periphery of the lesions (Fig. 1 a), except in 2 cases (cases 5 and 11) where the high SI was found in a more central position. In 1 patient (case 2) the entire lesion exhibited high SI (Fig. 3). In 2 patients (cases 9 and 10), a liquid-filled space with a fluid–fluid level inside the pituitary lesions was found; in both, the upper compartment appeared hyperintense while the lower compartment was isointense (Fig. 4).

T1-weighted images after IV gadolinium injection. After IV gadolinium application, different patterns of enhancement were observed. In four patients (cases 2, 4, 8, and 9), peripheral enhancement was seen (Fig. 1 b), whereas three other patients (cases 3, 5, and 7) showed global but inhomogeneous enhancement. A central and homogeneous enhancement was noted in one lesion (case 10; Fig. 2 b). The dura of the sphenoid planum enhanced in a case of macroprolactinoma (case 4; Fig. 1 b).

T2-weighted imaging. In the 2 cases (cases 5 and 10) where T2-weighted images were available, images showed areas of low and high SI. In 1 patient (case 5) a hypointense area on T1-weighted images corresponded to an area of marked low intensity on T2-weighted images suggestive of deoxyhemoglobin. In a second patient (case 10) the long TR/TE study showed intralesional areas of mixed high and low SI surrounded by a thin ring of marked low SI most likely representing hemosiderin deposition (Fig. 2 c).

Surgical findings

In all cases a transsphenoidal approach was used not allowing cavernous sinus exploration; thus, invasion of cavernous sinuses as seen on MRI could not be confirmed. Two lesions (cases 9 and 10) contained blood at surgery, a blood clot was found inside one tumor (case 9), and a brown "chocolate-like" liquid was aspirated from another pituitary lesion (case 10). The other nine pituitary lesions appeared as soft, friable tumors without macroscopic evidence of neither recent nor old hemorrhage.

Pathological findings

All but 1 patient (case 6) had pituitary macroadenomas. Hemorrhagic infarction was reported in all ten pituitary adenomas, whereas non-hemorrhagic infarction was noted within a metastatic epidermoid carcinoma into a non-adenomatous pituitary gland. One (case 8) of the ten adenomas showed extensive necrosis, compromising immunohistochemical staining, and subsequent determination of the phenotype.

MR correlation with histopathology

In 2 cases (cases 5 and 10) some isointense area on T1-weighted images corresponded to areas of marked T2 low intensity, with the rest of the tumor being hypo- or isointense on T1-weighted and hyperintense on T2-weighted images. Pathology disclosed for these lesions evidence of necrotic changes within prolactinomas with hemorrhagic infarction as well as presence of siderophages and fibroblasts indicating old and recent (probably a few months previously) hemorrhage (Fig. 2 a, c). The two pituitary tumors with fluid–fluid levels contained blood clots in one case (case 10), and a brown liquid in the second case (case 9), which both corresponded to old intratumoral hemorrhage. The upper fluid layer most likely represented free extracellular methemoglobin with the inferior layer corresponding to a sediment of red blood cell remnants (Fig. 4). Histopathological examination of the metastatic epidermoid carcinoma specimen (case 6) showed extensive non-hemorrhagic necrosis with infarction of the pituitary gland without evidence of bleeding, which confirmed the MRI findings wherein no evidence of blood was noted on MR images. Finally, gadolinium-enhanced studies were found not to contribute significantly when evidence of hemorrhage was already assessed on non-enhanced MR studies. Dural enhancement was noted in 1 case (case 4; Fig. 1 b).

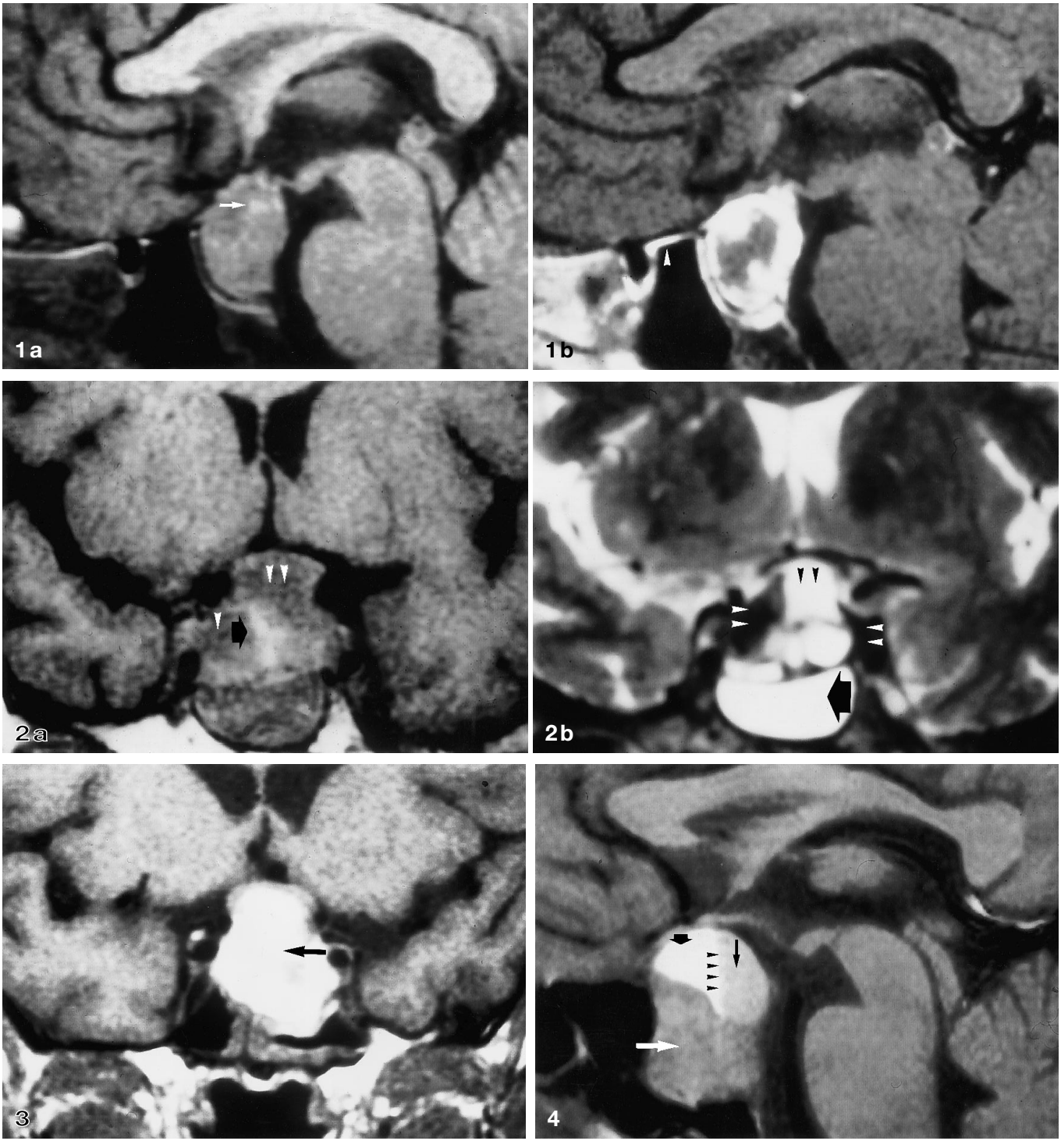

Fig. 1. **a** Case 4. Sagittal T1-weighted spin-echo (SE) image 13 days after onset of pituitary apoplexy (PA). There is a small spot of hyperintense signal which suggests the presence of blood *(arrow)*. Pathology reported hemorrhagic infarction within a prolactinoma. **b** Case 4. Sagittal post-gadolinium T1-weighted SE image showing rim enhancement, the non-enhancing central part of the macroadenoma most likely corresponding to area of infarction. The dura of the sphenoid planum is enhancing *(arrowhead)*

Fig. 2. **a** Case 5. Coronal T1-weighted SE image 4 days after onset of PA showing a central area of high signal intensity *(arrow)* relative to the white matter of the temporal lobe. The superior and the lateral parts are hypointense to the white matter of the temporal lobe *(arrowheads)*. The hyperintense signal may correspond to hemoglobin degradation content as extracellular methemoglobin. **b** Case 5. Coronal T2-weighted SE image. The lateral parts of the tumor are markedly hypointense *(white arrowheads)*. The medial part of the lesion *(black arrowheads)* is isointense to fluid accumulation into sphenoid sinus *(arrow)*. At surgery, there was no evidence of sphenoid sinus invasion by the tumor. Macroscopically the lesion consisted of dark-purple friable tissue without evidence of blood clot. Pathology reported acute hemorrhagic infarction in a prolactinoma as well as the presence of siderophages (containing hemosiderin) in keeping with a clinically silent previous hemorrhagic episode. The hypointense signal on T2-weighted image may represent deoxyhemoglobin at the acute phase of hemorrhagic PA but also hemosiderin (confirmed by the presence of siderophages at pathology) from a clinically silent previous bleeding

Table 1. Clinical, surgical, and pathological findings. *SS* sphenoid sinus; *SAH* subarachnoid hemorrhage; *LP* lumbar puncture; *CSF* cerebrospinal fluid

Case no./gender/ age (years)	Clinical symptoms	Onset to MR (days)	MR to surgery (days)	Surgical findings	Pathological findings
1/M/64	Chronic fatigue, diabetes mellitus, headache, nausea, vomiting, right third and sixth and left sixth nerve palsies, bitemporal hemianopsia	5	0	Purplish tumor invading SS	Hemorrhagic infarction, ACTH-secreting adenoma
2/M/47	Headache, nausea, vomiting, left third nerve palsy	10	0	Hemorrhagic semisolid tumor	Hemorrhagic infarction, non-secreting adenoma
3/M/60	Headache, nausea, vomiting, nuchal rigidity, left third and sixth nerve palsies, bitemporal hemianopsia, left hemiparesis, loss of consciousness, evidence of SAH at LP	5	1	Necrotic purple tumor with hemorrhagic parts	Hemorrhagic infarction, non-secreting adenoma
4/F/43	Headache, vomiting, photophobia, bitemporal hemianopsia	13	1	Purplish friable tissue	Hemorrhagic infarction, prolactinoma
5/F/53	Diabetes mellitus, headache, right third, fourth, and sixth nerve palsies	4	3	Dark-purple pituitary tumor	Recent and old hemorrhage with fibrovascular proliferation, siderophages, prolactinoma
6/F/56	Headache, left third, fourth, and sixth nerve palsies, left trigeminal neuralgia	8	2	Soft grayish tumor	Extensive necrosis, epidermoid carcinoma
7/M/83	Headache, nausea, torpor, right third nerve palsy	6	10	Purplish friable tissue, SS invasion	Hemorrhagic infarction, non-secreting adenoma
8/M/53	Headache, nausea, torpor, and vomiting, bitemporal hemianopsia, xanthochromic CSF at LP	6	1	Purplish friable tissue	Totally necrotized tissue (unclassifiable)
9/M/42	Headache, left third and sixth nerve palsies, left trigeminal neuralgia (V_1), episode of nausea, vomiting, headache 5 months before admission	5 months	1	Capsulated lesion containing a brown liquid and some reddish tumoral tissue	Old hemorrhagic infarction (siderophages), prolactinoma
10/M/27	4-month history of fatigue, blurred vision, impotence, headache, torpor, bitemporal hemianopsia,	11	21	Friable, purplish tissue, clots	Recent and old hemorrhage (siderophages), prolactinoma
11/M/64	Coma, SAH at LP	1	1	Reddish friable tissue	Hemorrhagic infarction, non-secreting adenoma

◀ **Fig. 3.** Case 2. Coronal T1-weighted SE image showing total high signal intensity *(arrow)* in keeping with hemorrhagic infarction into a non-secreting PA

Fig. 4. Case 9. Sagittal T1-weighted image. Macroadenoma *(white arrow)* exhibiting a fluid–fluid level pattern *(arrowheads)* 5 months after onset of pituitary apoplexy. At surgery, a brown liquid was aspirated. The tumor consisted of reddish tissue. Pathology reported a prolactinoma with old hemorrhage. Fluid–fluid level may represent sedimentation of blood cell remnants slightly hyperintense to the pons *(thick black arrow)* surmounted by extracellular methemoglobin appearing hyperintense *(thin black arrow)*

MR correlation with clinical presentation

One patient (case 3) who presented with sudden onset of left hemiparesis had evidence of significant bilateral stretching of the carotid siphons at MRI. No correlation to the severity of clinical symptoms could be established with regard to tumor volume, mass effect, and tumor phenotype. Cranial nerve palsies were present in keeping with cavernous sinus compression or invasion (Table 1).

Discussion

Pituitary apoplexy is a clinical syndrome characterized by sudden onset of headache, vomiting, visual impairment, diplopia, disturbance of consciousness, and autonomic or hormonal dysfunction [4]. It is a rare but life-threatening and sometimes fatal disorder [2]; therefore, PA requires prompt recognition and appropriate medical intervention to avoid unfavorable outcome. Infarction of a pituitary tumor with secondary hemorrhage or edema causes rapid expansion of the lesion with ensuing acute compression of adjacent structures, compromising neurological function [2]. Usually, the concomitant destruction of hypophyseal tissue leads to multiple endocrine deficiencies [4]. Since the description by Brougham in 1950, PA has become an increasingly recognized clinical entity, with an incidence ranging from 0.6 to 9 % of all pituitary tumors [1, 4, 12–16]. As previously stated, there is no evidence of a preferential histological tumor type associated with PA [17].

Table 2. MR findings. *SI* signal intensity; *n.a.* T2-weighted sequence not performed/no post-gadolinium study

Case no./gender/age (years)	T1-weighted	Gadolinium enhancement	T2-weighted
1/M/64	Isointense, peripheral high SI	n. a.	n. a.
2/M/47	Global high SI	Rim	n. a.
3/M/60	Mixed areas of iso- and high SI	Heterogeneous	n. a.
4/F/43	Isointense, spot of high SI	Rim dural enhancement	n. a.
5/F/53	Low and isointense, central foci of high SI	Heterogeneous	Low and high SI
6/F/56	Isointense	n. a.	n. a.
7/M/83	High SI on the right	Heterogeneous	n. a.
8/M/53	Peripheral high SI	Rim	n. a.
9/M/42	Peripheral high SI, liquid–liquid level	Rim	n. a.
10/M/27	Peripheral high SI, liquid–liquid level	Central	Low and high (mixed), ring of low SI
11/M/64	Central high SI	n. a.	n. a.

MR features of intratumoral hemorrhage

Another advantage of MRI is that it affords an estimation of the age of the hemorrhage. Two macroprolactinomas (cases 5 and 10) with histopathological evidence of both old (siderophages) and recent hemorrhages demonstrated areas of low SI on T2-weighted images which was attributed to hemosiderin deposits from a previous bleeding. It was also somewhat unexpected since pituitary tumors lack a blood–brain barrier, and the accumulation of hemosiderin-laden macrophages (siderophages) does not usually occur [18]. In case 5 the medical history did not mention a previous episode of PA, thus suggesting the possibility of clinically silent intratumoral hemorrhage. Contrary to intracerebral hematomas, in which the earliest changes appear in the periphery, high SI on T1-weighted images may appear more centrally in hemorrhagic pituitary lesion as in the case of two of our patients (cases 5 and 11) [19]. Because our retrospective study encompassed three different imaging centers using various MRI protocols, only 2 patients had SE T2-weighted sequences.

MR features of the mass effect

One patient of our series (case 3) who presented with left hemiparesis and subarachnoid hemorrhage had evidence of right carotid siphon stretching by the tumor. The study of the relationship of the intracavernous tumor extension with the carotid siphon was facilitated by the signal void produced by high flow within the arterial lumen [20]. In PA hemispheric dysfunction, such as hemiplegia, can be explained by the sudden expansion of the pituitary tumor due to hemorrhage or edema. This may produce mechanical compression of the carotid siphon [2]; otherwise, any rapid increase in intracranial pressure resulting from subarachnoid hemorrhage may also lead to these neurological deficits. Cavernous sinus invasion and optic chiasm compression were not surgically proved due to difficulty in visualizing these structures via a transsphenoidal approach. However, MR offers substantial information in identifying tumor in the cavernous sinuses, which are not usually explored at surgery and would be treated with radiation therapy. Tumor extension into the sphenoid sinus was not as easily identified by MR due to its resemblance to benign inflammatory conditions as stated in one of our cases, where sphenoid sinus invasion was suspected on MRI and not found at surgery [21].

Differential diagnosis

Metastatic carcinomas turning into pituitary adenomas have been reported previously [22]. One of our cases (case 6) was very unusual in that a metastatic epidermoid carcinoma was the cause of the non-hemorrhagic PA. The patient medical history and the specimen's histopathological examination did not suggest the existence of a previous PA. Craniopharyngiomas may also be hyperintense on both T1- and T2-weighted images because of the protein content within the cysts, thus making it difficult to distinguish it from pituitary tumors with hemorrhage [23]. Moreover, they may also present with intratumoral hemorrhage. Clinically, craniopharyngiomas usually present with slowly progressive neurological deficits. This is in contrast to PA, in which patients present with acute neurological manifestations. Thus, the documentation of a sellar mass with increased SI on short TR/TE sequences in patients with sudden onset of headache, and visual deterioration from isolated oculomotor nerve palsy leading to ophthalmoplegia, is of diagnostic importance as this most likely denotes PA [10]. Infarction of non-tumorous anterior pituitary glands with subsequent PA has been reported [17]. No such case was seen in our limited series. An increased frequency of intratumoral hemorrhage was noted in prolactinomas and macroadenomas in patients undergoing bromocriptine therapy. Yousem et al. [18] noted intratumoral hemorrhage in 22 % of non-secreting adenomas and in 33 % of prolactinomas. None of the patients in our study received this treatment prior to onset of PA. Post-gadolinium dural enhancement was noted in one macroprolactinoma. This non-specific finding has been reported previously in non-complicated macroadenomas [24].

Why is there hemorrhage into apoplectic pituitary macroadenomas?

The hypothalamo-hypophyseal portal vessels are the main blood supply to the anterior pituitary lobe; however, the anterior lobe has a double and variable system of vascularization, portal and arterial [25]. Increasing intrasellar pressure due to progressive tumoral growth might preclude blood supply from the low-pressure portal venous system leading to ischemia and subsequent infarction, without compromising high-pressure arterial pituitary input. Moreover, some pituitary adenomas are known to receive direct arterial blood supply [26, 27]. These two potential arterial blood supplies might lead to hemorrhage into the infarcted pituitary tumor.

Conclusion

Apoplectic pituitary tumors with hemorrhagic infarction may harbor different SI patterns (iso- to totally hyperintense, peripheral or central high SI) on T1-weighted images. Fluid–fluid level formation inside a pituitary tumor is most likely in keeping with iterative intratumoral bleeding as is attested by the presence of old and recent bleeding at pathology. Gadolinium-enhanced images do not provide complementary diagnostic information.

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