Aneurysms

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Intracranial aneurysms are focal outpouchings of the wall of the cerebral arteries. They mainly develop close to the circle of Willis, an anastomotic network linking the internal carotid arteries and the vertebrobasilar system, which surrounds the sellar region. Therefore, intracranial aneurysm may present as an intra-, supra-, or laterosellar mass lesion. In this area, unruptured aneurysms may exert mass effect on the pituitary gland or the stalk or the cranial nerves, or may be discovered incidentally on brain or sellar MRI. They might be misdiagnosed or missed on preoperative MRI, but it is important to be aware that intracranial aneurysms may coexist with a pituitary adenoma (Fig. 60.1). They may be fortuitously associated with any nonsecreting or secreting pituitary adenoma, but acromegaly carries an increased risk of harboring intracranial aneurysms, with a reported incidence ranging from 4 to 17 %. In this setting, development of intracranial aneurysm might be due to the arterial hypertension frequently observed in this disease . Aneurysms in acromegalic patients are said to be usually located on the anterior circulation, are small (mean diameter <7 mm), and seem not to be more prone to rupture than aneurysms observed in the general population. It is of critical importance to evoke the diagnosis of sellar aneurysm preoperatively, because unruptured aneurysms should benefit from dedicated care and also because cases of aneurysms accidentally discovered during transsphenoidal surgery have been reported, with variable outcomes. Management of intracranial unruptured aneurysms principally depends on their risk of spontaneous rupture, which is influenced by the size and location of the aneurysm. First, the larger the aneurysm, the higher the risk of rupture. Risk is considered minimal, <1 % per year, when aneurysm diameter is smaller than 7 mm, while it may reach 30 % at 5 years for giant aneurysms larger than 25 mm. Second, the location is crucial because it is fundamental in determining whether an intracranial aneurysm is intradural or extradural, especially in the sellar region. The origin of the ophthalmic artery divides the two subgroups. On one hand, extradural aneurysms rise before the origin of this artery. They are intracavernous and thus virtually never cause subarachnoid hemorrhage, but behave as would any cavernous lesion, compressing the adjacent structures or destroying the bony skull base. On the other hand, intradural aneurysms arise after the origin of the ophthalmic artery and may rupture in the subarachnoid space. In the sellar region, such aneurysms precisely arise from the superior hypophyseal artery and extend medially, from the supraclinoid segment of the internal carotid artery (so-called carotid-ophthalmic aneurysm) and usually develop upward, or grow downward from the anterior communicating artery. Note that intracranial aneurysms are multiple in about 20 % of cases and that carotid-ophthalmic aneurysms are often bilateral.

On MRI, intracranial aneurysms appear as well-delineated, round, hypointense lesions on

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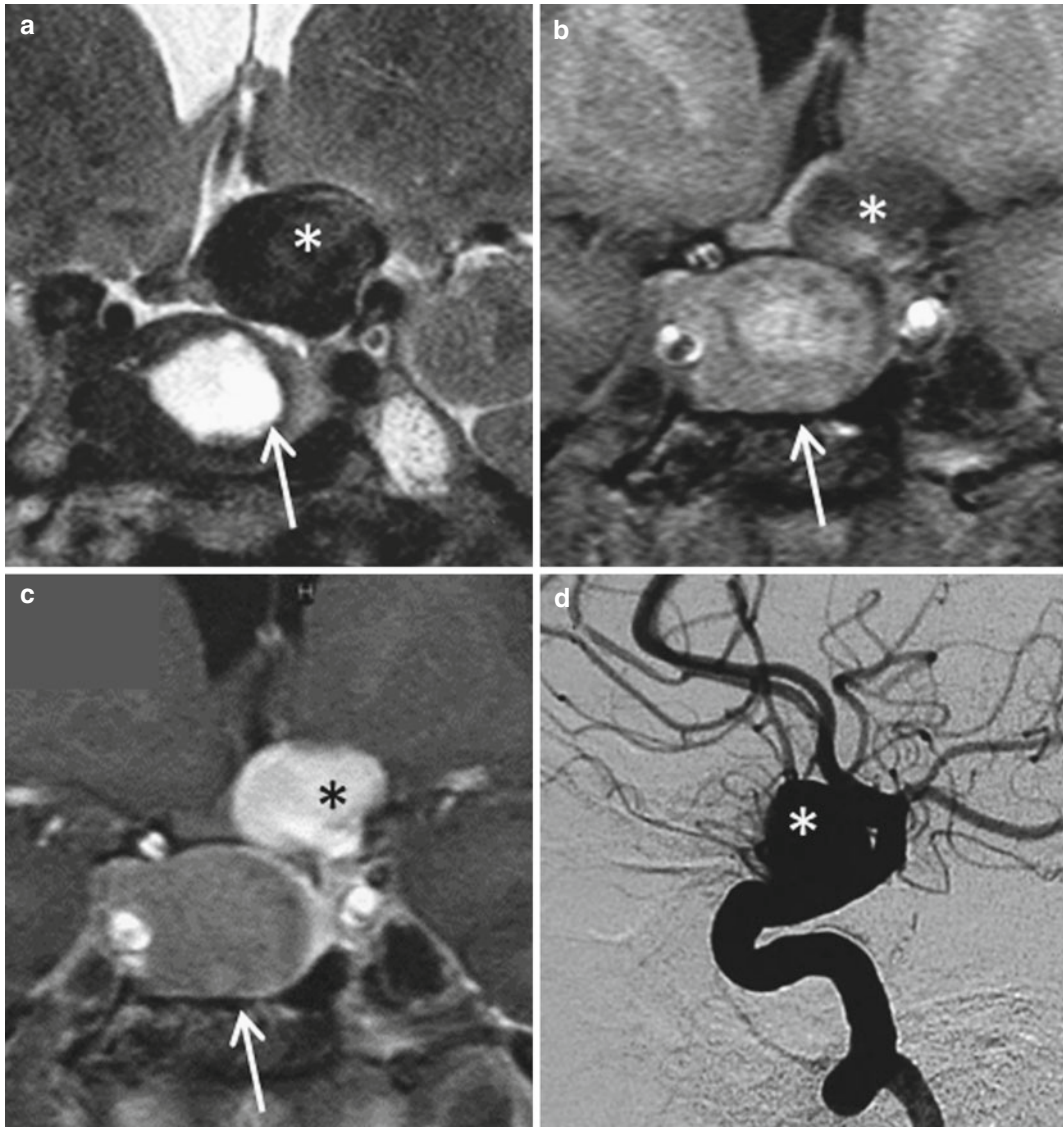

Fig. 60.1 Large supraclinoid left internal carotid artery aneurysm in a 60-year-old woman with acromegaly. (**a**) Coronal T2WI shows a large heterogeneous hyperintense pituitary adenoma (*arrow*) and a suprasellar aneurysm with characteristic flow void (*asterisk*) impinging on the left aspect of the optic chiasm. Coronal GE T1WIs (**b**) before and (**c**) after gadolinium injection clearly depict the adenoma invading laterally the right cavernous sinus, and a separate aneurysm that enhances after gadolinium administration, a feature usually observed in patent aneurysm on GE sequences. (**d**) Lateral view of left internal carotid artery angiogram confirms the nature and the location of this supraclinoid aneurysm

all spin-echo sequences. The signal intensity is so low that, in fact, they present with signal void or flow void (Fig. 60.2). This is obvious on T2WI, which is the best sequence to depict an aneurysm on MRI of the pituitary region. They usually lack contrast enhancement after gadolinium administration on spin-echo sequences, but may enhance on GE sequences (Fig. 60.2). In cases of small intrasellar aneurysm, this lack of enhancement may mimic pituitary microadenoma, particularly on contrast-enhanced T1WI (Fig. 60.3). Close attention should be paid to the other sequences, and especially MRA if needed. Similarly, midline suprasellar aneurysm may

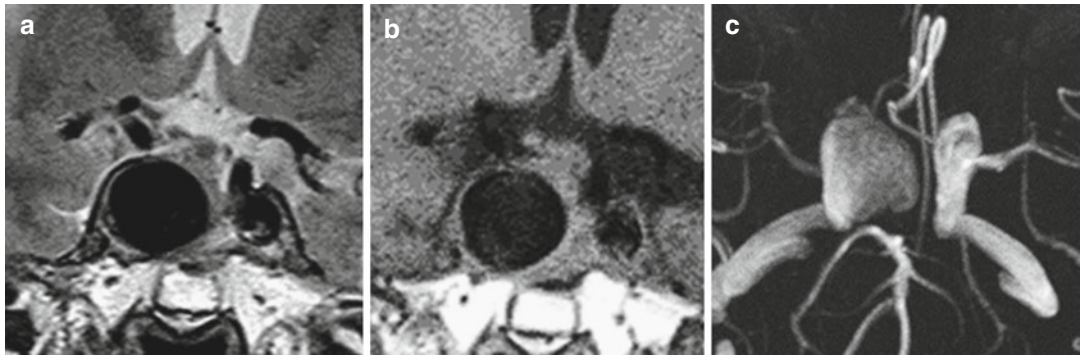

Fig. 60.2 Typical intracavernous internal carotid artery aneurysm in a 65-year-old woman with right ocular palsy. The aneurysm appears as a well-demarcated lesion within the right cavernous sinus and demonstrates marked hypointensity on (**a**) T1WI and (**b**) T2WI. (**c**) MRA confirms the arterial nature of this patent aneurysm

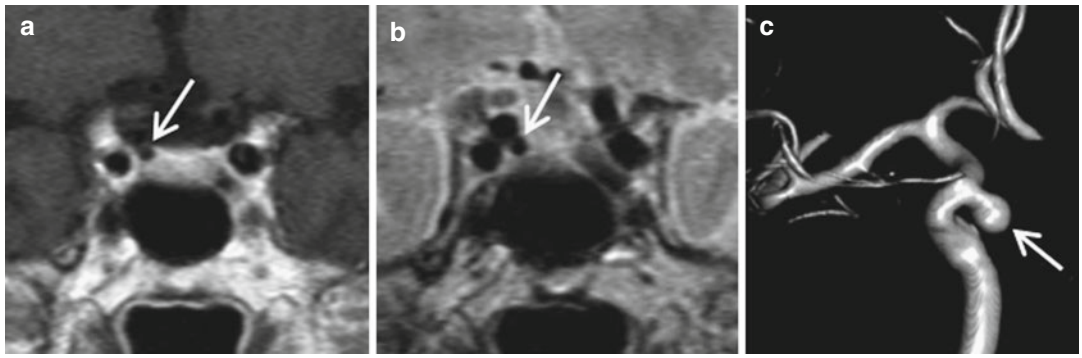

Fig. 60.3 Small intrasellar superior hypophyseal aneurysm in a patient with Cushing syndrome. (**a**) CE T1WI depicts a tiny nonenhancing lesion in the right part of the pituitary gland that may mimic a pituitary adenoma (*arrow*). The lesion appears very dark on (**b**) T2WI, a feature very suggestive of an aneurysm, as confirmed on (**c**) 3D volume rendering reformatted MRA. Note that no adenoma is demonstrated in this pituitary gland

mimic proteinaceous Rathke cleft cyst (RCC), which sometimes appears with marked homogeneous T2-low signal intensity (Fig. 60.4). Of note, flow turbulences are often observed within large aneurysms, and appear as signal heterogeneities on spin-echo sequences, giving a marble-like pattern. Similarly, repetition artifacts resulting from intrasaccular arterial pulsations may sometimes be depicted beside large aneurysms along the phase axis. When an aneurysm is suspected, it should be confirmed by adjunctive MRA. Aneurysms may also be partially thrombosed, mainly when large, thus giving different signal intensities according to the age of the clotting. Such thrombosed aneurysms appear with characteristic heterogeneous iso- to hypersignal intensities on both T1 and T2 WIs (Fig. 60.5). While the noncirculating clotted parts of the aneurysm sac do not enhance after contrast media administration, the aneurysm walls do enhance on GE 3D T1WI. Differential diagnosis may be difficult with other nonenhancing hemorrhagic lesions such as pituitary apoplexy. Because a giant aneurysm with partial internal clotting may mimic a solid destructive tumor of the skull base, MRA or conventional angiography should definitely be performed in doubtful cases before biopsy or surgery is considered. The finding of a residual patent lumen on images helps confirm the diagnosis of aneurysm. Other sellar lesions returning T1 hyperintensity may also be mistaken for partially thrombosed aneurysm. Signal heterogeneity, especially on T2WI, is a key feature of thrombosed aneurysm, a sign usually not observed in a protein-rich content lesion such as, for example, RCC.

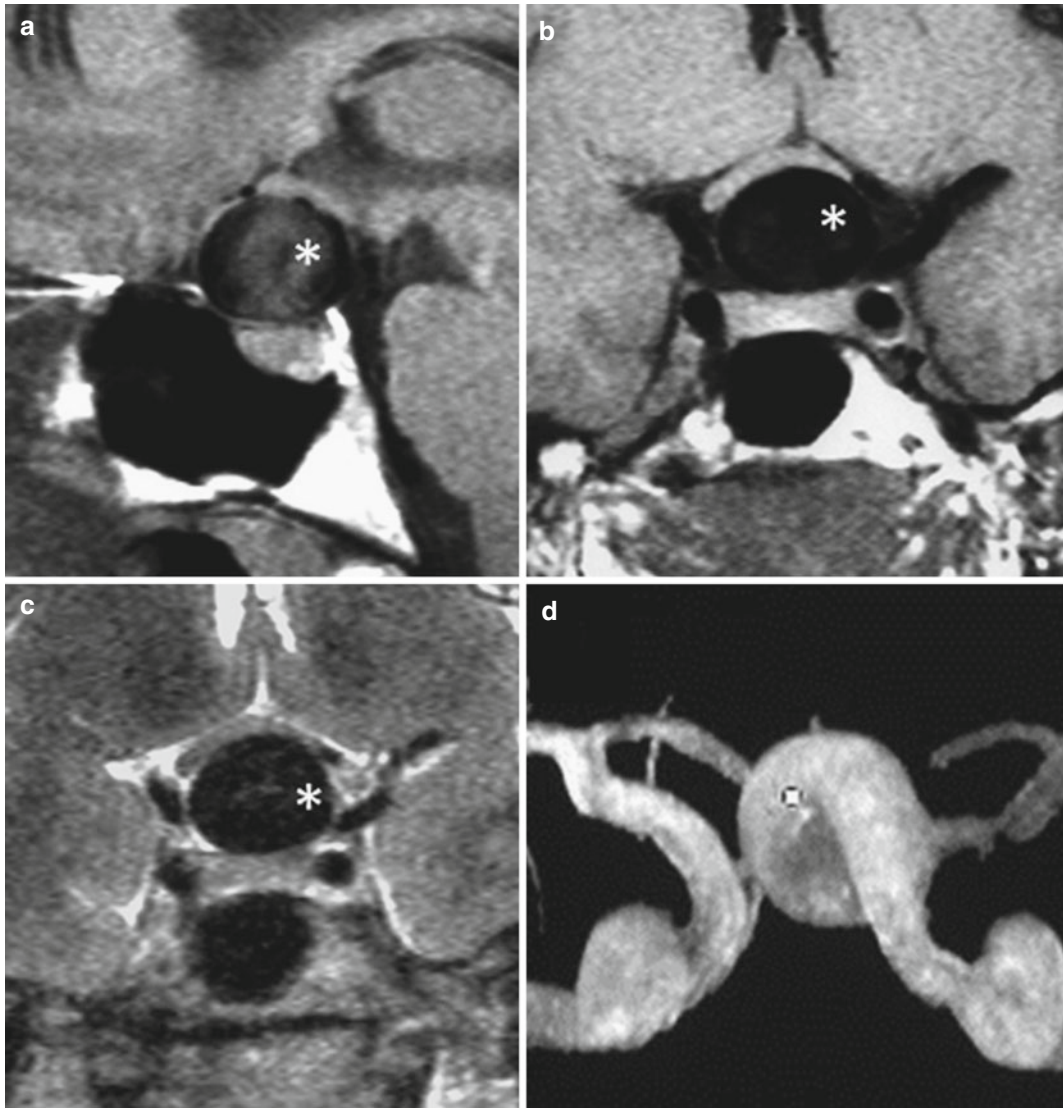

Fig. 60.4 Midline suprasellar aneurysm. (**a**, **b**) Sagittal and coronal T1WIs show a large, well-demarcated suprasellar hypointense lesion impinging on the optic chiasm that appears with a homogeneous hypointense signal on (**c**) T2WI, thus mimicking a suprasellar mucoid RCC. (**d**) MRA rules out this hypothesis by confirming the flow in this aneurysm

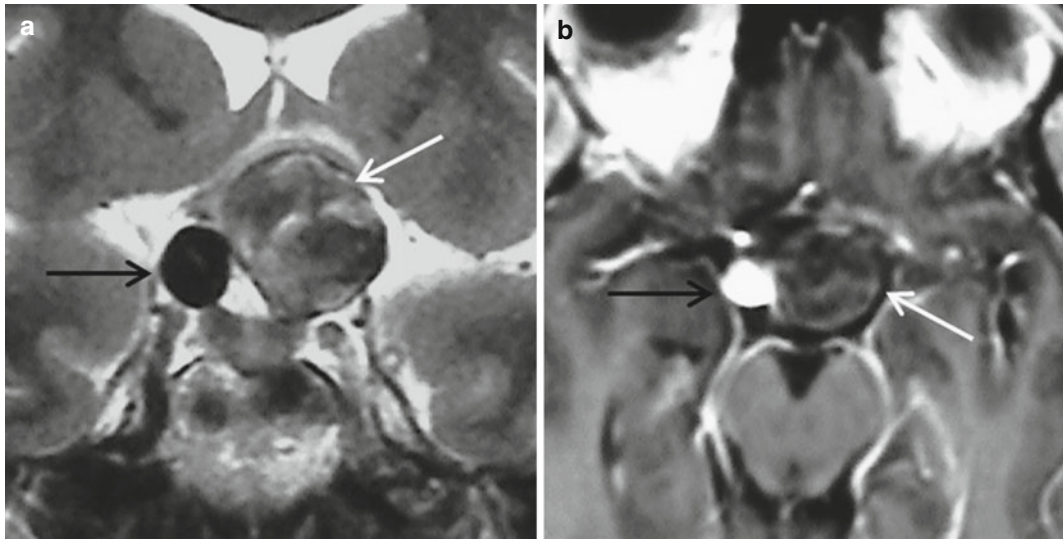

Fig. 60.5 Bilateral carotid-ophthalmic aneurysms in a 58-year-old woman with vertigo. (**a**) Coronal T2WI and (**b**) axial GE CE T1WI illustrate two different aneurysms in the same patient: a small patent aneurysm (*black arrow*) on the right side, appearing as a flow void on T2WI and enhancing on GE CE T1-WI, and a large thrombosed aneurysm on the left side (*white arrow*), impinging the optic chiasm, demonstrating heterogeneous isointense signal on T2WI and not enhancing on (**b**) (Reprinted with permission from Bonneville et al.)

Further Reading

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