54 Lipoma

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Rather than true neoplasms or hamartomas, intracranial lipomas are thought to represent congenital malformations that result from an abnormal persistence and maldifferentiation of the meninx primitiva. This embryologic concept explains the high frequency of callosal and other brain malformations associated with intracranial lipomas. Typically occurring in the midline, the most common locations for these lesions are the corpus callosum and the quadrigeminal cistern. In about 10–15 % of cases, intracranial lipomas are encountered in the suprasellar/interpeduncular region. Although intracranial lipomas are usually asymptomatic and often discovered incidentally on brain CT or MRI, symptoms related to intracranial lipomas have been reported. However, it cannot be excluded that symptoms might be related to an underlying associated brain malformation rather than to this congenital fat pad itself. Thus, so-called osteolipomas of the tuber cinereum have been reported in young girls with precocious puberty, but similar lesions have been found incidentally at autopsy in previously asymptomatic men (Fig. 54.1). In addition, in the rare reported cases of excision of tuber cinereum lipoma, no effect of the surgery was described on the precocious puberty itself. Finally, complete removal of intracranial lipomas by surgery is illusive because such lesions are intimately adherent to the surface of the brain, as explained by their embryogenesis. Diagnosis of lipomas is straightforward on brain imaging. On CT, they exhibit low density typical for fat and may harbor superficial calcification (Fig. 54.1). On MRI, lipomas are well-demarcated homogeneous lesions, hyperintense on T1WI and T2WI, and do not enhance following administration of gadolinium (Figs. 54.1 and 54.3). Of note, dermoid cysts are usually heterogeneous and demonstrate gadolinium uptake of their wall. It is also noteworthy that fat appears with a low-T2* signal intensity and may thus be misdiagnosed as hemorrhage. Additional T1 fat-saturation sequence makes it possible to confirm the presence of fat in the lesion and, subsequently, the diagnosis of lipoma. Such sequence rules out hemorrhage, high-protein content lesion, or any other source of T1 hyperintensity. In the sellar region, suprasellar lipoma may also be mistaken for ectopic pituitary posterior lobe, as observed in dwarfism. In the latter case, this T1 ectopic bright spot is seen at the infundibulum; this T1-high signal intensity is not attenuated on T1 fat-saturated sequence: the sella, anterior pituitary lobe, and stalk are hypoplastic, and overall, normal signal hyperintensity in the posterior aspect of the sella turcica is lacking (Fig. 54.2). If intracranial lipomas are frequently observed in midline, laterosellar lipomas might be encountered along cranial nerves (Fig. 54.3).

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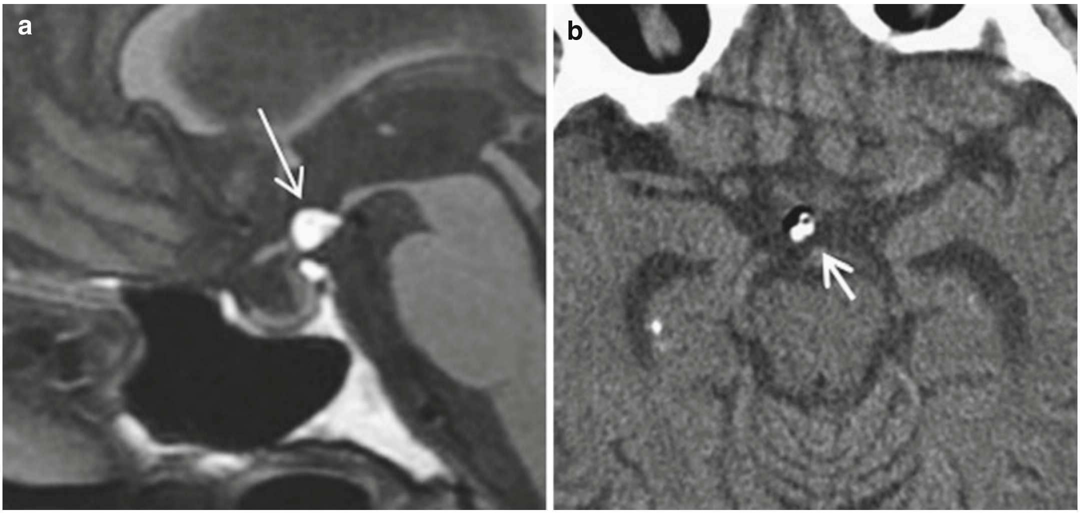

Fig. 54.1 Fortuitous osteolipoma in a 72-year-old man with gait disturbance. (**a**) Sagittal T1WI depicts a suprasellar round fat lesion of the tuber cinereum (*arrow*) with calcifications (*short arrow*), better demonstrated by (**b**) unenhanced CT scan

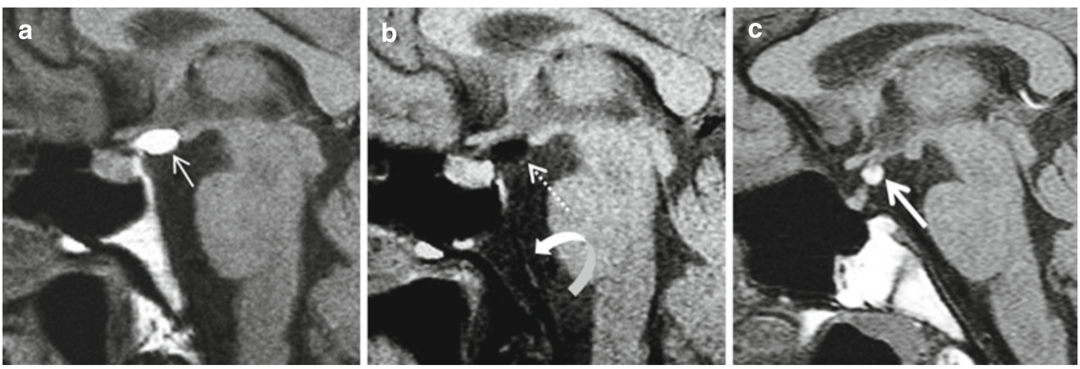

Fig. 54.2 Asymptomatic lipoma of the tuber cinereum in 49-year-old man with headaches. (**a**) Sagittal T1WI shows a typical suprasellar lipoma of floor of the third ventricle (*arrow*). The exclusive fatty nature of this lesion is confirmed on (**b**) T1WI with fat saturation, on which the hyperintense signal due to fat disappears (*dotted arrow*), as observed in the lesion and the fatty bone marrow of the clivus (*curved arrow*). This benign lipoma should not be misdiagnosed as an ectopic pituitary posterior lobe (*thick arrow*), as found in dwarfism and illustrated by (**c**) sagittal T1WI, which appears as a hyperintense bright spot at the infundibulum together with hypoplasia of the sella, anterior pituitary lobe and stalk, and absence of normal signal hyperintensity in the posterior aspect of the sella turcica

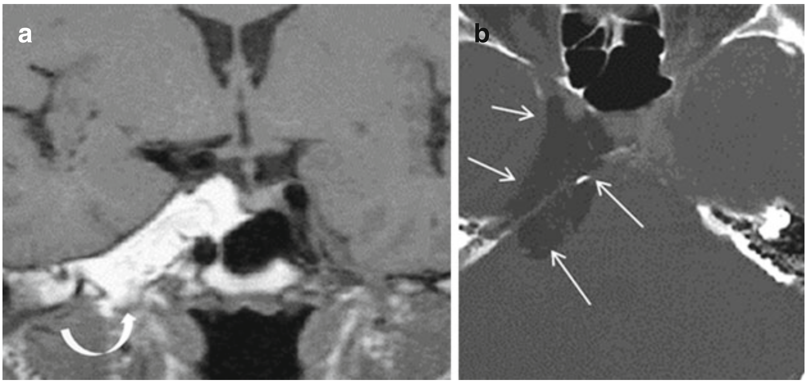

Fig. 54.3 Trigeminal lipoma in a 34-year-old woman with right-sided facial neuralgia. (**a**) Coronal T1WI depicts a right laterosellar hyperintense lesion that occupies the posterior aspect of the cavernous sinus and emerges through the foramen ovale (*curved arrow*). (**b**) Axial CT scan demonstrates the pure fatty nature of the lesion, which parallels the trigeminal nerve and its branches (*arrows*) (Reprinted with permission from Bonneville et al.)

Iatrogenic fatty substances may also be depicted in the sellar region. Iodinated lipiodol that has been used intrathecally may persist for decades and appear as fatty droplets with characteristic T1 hyperintensity (Fig. 54.4). After transsphenoidal resection of pituitary tumors, fatty materials may be used to fill the empty space after excised tissue at the bottom of sella turcica and prevent CSF leakage. In rare instances such fatty filling material may migrate within the subarachnoid space in the sellar region and be the source of subsequent infectious or vascular lesions (Fig. 54.5).

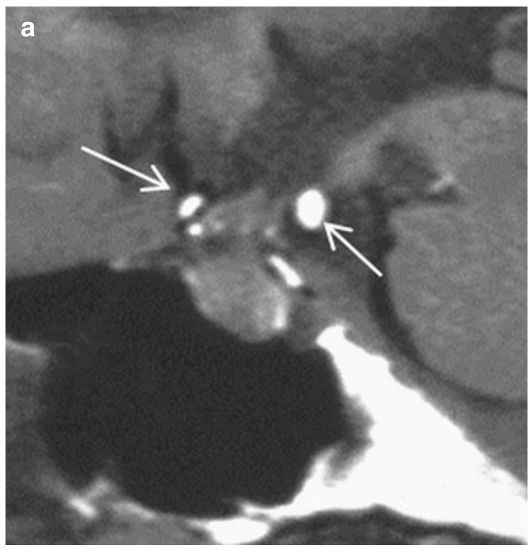

Fig. 54.4 Subarachnoid remnants of contrast material in a 79-year-old woman with a history of intrathecal injection of an iodinated lipid-containing contrast agent. (**a**) Sagittal T1WI shows multiple hyperintense nodules trapped in the chiasmatic cistern (*arrowheads*) (Reprinted with permission from Bonneville et al.)

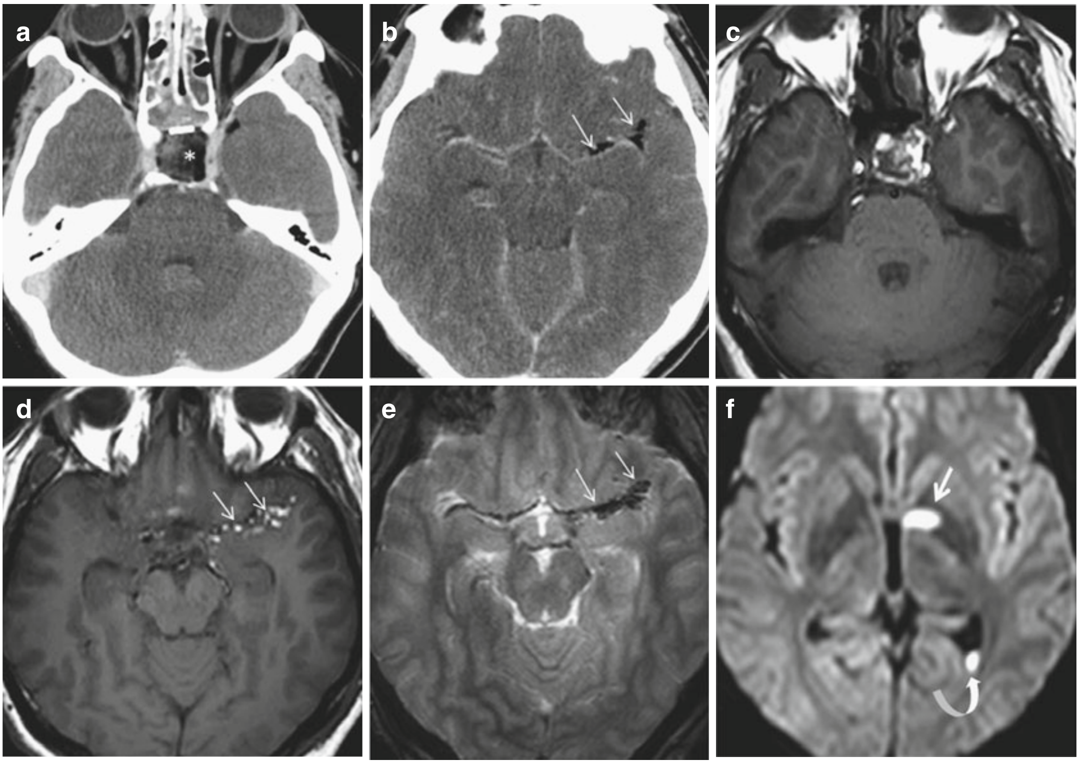

Fig. 54.5 Brain imaging of a 39-year-old man operated on for a macroadenoma via transsphenoidal approach 3 months previously and now suffering from febrile encephalomeningitis with right hemiparesis. (**a**, **b**) CE CT scan shows fat packing of the sellar surgical site (*asterisk*) with partial migration in the left sylvian fissure along the middle cerebral artery (*arrows*). (**c**, **d**) Axial unenhanced T1WIs confirm that the hypodense abnormalities observed on CT were not postoperative air, but true droplets of characteristic hyperintense fat (*arrows*). (**e**) GE T2WI illustrates how fat appears hypointense and can mimic blood on this sequence. (**f**) DWI depicts capsulolenticular ischemia (*thick arrow*). This lesion might be due to arteritis secondary to potential irritation of the left middle cerebral artery encased in the subarachnoid fat or the meningitis. Intracranial infection is established, with visualization of cellular debris and sedimentation in the left occipital horn (*curved arrow*)

Further Reading

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