figures are rare and there is no necrosis or vascular invasion. All lesions show a population of cells with vacuolated cytoplasm resembling the physaliferous cells found in chordoma [4,5].

Conflict of interest statement

None.

References


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IRM d’une métastase d’un carcinome rénal dans un méningiome

A 61-year-old man had undergone resection of left renal cancer in 1999. In May 2005, he developed a continuous headache and MRI showed a left frontal convexity meningioma, 25 × 15 mm in size, with typical imaging features (Fig. 1). The mass was homogeneously isointense to the gray matter on T1- and T2-weighted images, with strong and homogeneous enhancement after gadolinium administration and a typical “dural tail sign”. There was no edema of the adjacent white matter and imaging findings were unchanged at follow-up MRI in March 2006. In November 2006, the patient developed progressive motor aphasia together with changes in behavior. MRI showed enlargement of the mass, whose size reached 30 × 41 mm, with marked peritumoral edema and a mild midline shift (Fig. 2). The signal intensity became inhomogeneous on T2-weighted images with appearance of intra- and peritumoral flow voids. T1-weighted images showed an irregular enhancement with a cystic-necrotic center. A malignant transformation of the meningioma was hypothesized and a left frontal craniotomy was performed. Pathologic examination demonstrated clear cell carcinoma with widespread necrosis occupying the majority of the lesion, indicating brain metastasis (Fig. 3). Meningothelial meningioma was recognizable only at the peripheral rim. Subsequent exami-

Figure 1  First MRI. (A) T2W axial section; (B) Postcontrast T1W coronal section. Left frontal meningioma (arrow), isointense to the cortex in T2-W images without perifocal edema, characterized by strong and homogeneous enhancement after gadolinium administration. On the basis of absent neurological signs and symptoms, surgery was not indicated.
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Figure 2  Follow-up MRI after 17 months, when the patient developed aphasia and behavioral changes. (A) T2W axial section; (B) Postcontrast T1W coronal section. The meningioma has enlarged, with appearance of a necrotic center, flow voids (arrowhead) and vasogenic edema (arrow).

Follow-up MRI after 17 months, when the patient developed aphasia and behavioral changes. (A) T2W axial section; (B) Postcontrast T1W coronal section. The meningioma has enlarged, with appearance of a necrotic center, flow voids (arrowhead) and vasogenic edema (arrow).

Metastasis of one tumor to another tumor is a rare pathological entity. Meningioma is the most frequent recipient tumor type and the majority of metastases arises from breast and lung cancers. Metastases from renal cancers to intracranial meningiomas are especially rare. Chambers et al. proposed the criteria for the diagnosis of a true tumor-to-tumor metastasis and for the exclusion of collision tumors [1]. Only five reported cases of metastasis from tumors of kidney, including ours, meet the criteria [2,3]. The preoperative diagnosis of metastasis to meningioma is challenging. Two authors described MR findings of markedly enhancing peripheral portions of the tumor with a necrotic core [2,3]. These patterns are, however, nonspecific and could be due to areas of calcification, foci of cystic degeneration or hemorrhage. One case had intra- and peritumoral flow voids like in our case [3]. Apart from ours, only one other case was reported with follow-up imaging, consisting of meningioma enlargement with development of peritumoral edema and inhomogeneous enhancement with irregular margins [4]. Malignant transformation of meningiomas, although rare, must be taken into account in the differential diagnosis [5]. Imaging findings can be quite similar and an accurate distinction from intrameningioma...

Figure 3  (A) Macrospecimen (H & E stain) and (B) microspecimen (H & E stain, 10 ×) of the tumor. Dura is seen in the upper part of the specimen with no infiltration (arrows). Meningothelial meningioma is identifiable at the peripheral rim of the specimen (arrowheads), where lobules and whorls of round-polygonal cells with small regular nuclei and eosinophilic cytoplasm are surrounding clear cell carcinoma (asterisk). Dilated vessels are spread around with thickened hyaline walls. Immunohistochemistry stainings for CD10 and EMA outlined the two different neoplastic components (not shown).
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metastasis is difficult to obtain. Multimodality MR imaging has become the preferred approach to characterize brain tumors. MR spectroscopy was so far applied to one case of intrameningioma metastasis, but the differential diagnosis with malignant transformation of meningioma could not be established [6]. In conclusion, atypical MRI features of meningiomas may suggest the possible occurrence of an intratumoral metastasis in patients affected by a systemic cancer, especially when significant changes are detectable on follow-up imaging.

Conflict of interest statement

None.

References


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Ictal hyperperfusion demonstrated by arterial spin-labeling MRI in status epilepticus

Hyperperfusion en IRM de perfusion par marquage des spins dans un cas d’épilepsie

Case report

39-year-old female patient who presented at first two days before with generalized seizures followed by partial seizures and cloni of the right leg on the day of admission. At first a CT scan was performed (Philips) that failed to reveal any underlying pathological finding. EEG showed a focus in the left frontal cortex. MR imaging was performed on a 3.0 T Magnetom Trio (Siemens; Erlangen, Germany). Arterial spin labeling (ASL) was performed with a PASL sequence, using a QUIPSII perfusion mode and the following parameters: 16 slices, voxel size: 3.4 × 3.4 × 6 mm, TA = 5:55 min, lambda = 0.9 mL/g, alpha = 95%, TE/TR/TI1/TI2/T1(blood3T)(ms) = 15/5000/700/1800/1496,19, Relative Cerebral Blood Flow (RelCBF) maps for ASL were calculated in-line by the MRI scanner, and off-line for CEPWI using the Syngo Perfusion (MR) software. Single-voxel MR spectroscopy was performed in both hemispheres, placing the voxel on the left in the frontal area affected on DWI. DWI with a 30 directions scan was acquired as well.

Diffusion imaging showed hyperintensity in the left frontal cortex (Fig. 1a) with decreased ADC values (Fig. 1b) and a decrease in fractional anisotropy (Fig. 1d). On MR spectroscopy there was slightly elevated lactate. This was compatible with status epilepticus; on ASL perfusion we had an important increase in perfusion on the CBF maps (Fig. 1g–h).

Magnetic resonance imaging plays a central role in the investigation of patients with epilepsy: not only in chronic but in acute symptomatic cases [1]. Perfusion techniques allow to investigate the epileptic brain and MR arterial spin-labeling perfusion uses no contrast [2,3]. ASL has been previously used to document patients with epileptic diseases [4–11] This case illustrates the capacity of MR perfusion with arterial spin-labeling to very sensibly demonstrate areas of hyperperfusion in epilepsy. This is nicely correlated in our case with clinical symptoms referable to the left parasagittal frontal cortex as well as to both electrophysiological findings and neuro-imaging with diffusion and spectroscopic findings. This again confirms ASL to be a more and more important part of the investigation of patient with epileptic syndromes.

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