mention of early neurological impairment during infancy or any systemic features. Thus, we can hypothesize that the adult form presents as a much more indolent and benign disease.

CT and MRI both offer clues to the diagnosis by demonstrating the unique pathognomonic triad that includes cysts, leukoencephalopathy and asymmetrical calcifications [5]. On histology, the key feature of LCC is a proliferative small-vessel angiopathy with Rosenthal fibers [1]. In our case, only non-specific hemorrhagic changes were seen, as the cyst was damaged during the sampling procedure.

To our knowledge, our patient is the oldest of all cases reported so far to present with the onset of LCC. Furthermore, this patient had no neurological dysfunction and was professionally active until her retirement. Thus, this case supports the possibility of a long-lasting asymptomatic form of LCC. As evidenced by the CT and MRI scans of our patient taken 14 year earlier, the disease may remain latent for years. The cysts developed slowly, over decades, and the diagnosis was made at the time of intracystic hemorrhage, a highly unpredictable event. This may explain the wide interindividual variability among patients with LCC. Diagnostic imaging and follow-up are essential, while brain biopsy can be avoided as the neuroimaging triad is highly characteristic.

Conflict of interest statement

The authors report no conflicts of interest.

References


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Primary dural lymphoma with vault involvement mimicking meningioma

Lymphome dural primitif avec envahissement de la voute simulant un méningiome

A 25-year-old immunocompetent man presented with frontal headaches, vomiting and left hemiparesis.
resonance imaging (MRI) studies revealed a 3-cm parietal extra-axial tumor. The mass was lobulated and slightly hypointense on T1-weighted imaging (WI) in relation to gray matter and on T2-WI, with heterogeneous enhancement after administration of gadolinium. A superior sagittal sinus (SSS) extension associated with the ‘dural tail sign’ (Fig. 1) suggested aggressive meningioma. There were no abnormalities in the adjacent vault.

The patient refused surgery but, 5 months later, was admitted with generalized seizures and left hemiplegia. MRI showed an increase in the size of the mass, with adjacent vault extension and moderate scalp involvement (Fig. 2). Preoperative angiography revealed complete occlusion of the SSS related to tumor invasion, although no blush was noted on the tumor (Fig. 3). During surgery, the SSS was ligated both anterior and posterior to the occluded portion to allow removal of the dural mass. Histological examination of the mass demonstrated large round cells with nuclear polymorphisms, numerous mitoses and geographical necrosis (Fig. 4), which led to a diagnosis of anaplastic meningioma.

Three months later, the patient presented with a fast-growing scalp vault mass on the left parietal side. Computed tomography (CT) and MRI scans showed an extensive and intensely enhancing scalp mass with vault involvement, but no local recurrence of the dural tumor (Fig. 5). Pathological and immunohistochemical examinations of the resected mass led to the diagnosis of anaplastic large B-cell lymphoma. CD30 and anaplastic lymphoma kinase (ALK) markers were positive (Fig. 6). The patient was transferred to the oncology unit for chemotherapy and radiotherapy. A 22-month follow-up revealed no systemic dissemination of the malignancy and no neurological deficits.

Primary dural lymphoma (PDL) is extremely rare and accounts for around 7% of all primary central nervous system (CNS) lymphomas [1,2]. PDL usually involves sites that are rich in meningothelial cells, and results in a localized mass or plaque-like thickening of the dura that radiologically resembles a number of other diseases, such as meningioma or subdural hematomas (SDH) [2,3]. Because of its high cellularity and nucleus-to-cytoplasm ratio, lymphoma usually appears hyperdense on non-contrast CT scans, isointense to hypointense relative to gray matter on T1-WI and isointense to hypointense on T2-WI [3,4]. It is typically avascular on angiography, whereas a blush pattern or vascular
Preoperative angiography shows complete occlusion of the anterior third of the superior sagittal sinus.

Histopathology shows large round cells with nuclear polymorphisms, mitoses and large vessels (H&E stain, ×200).

Cell immunopositivity for CD30 and anaplastic lymphoma kinase (IHC stain, ×200).

Encasement is apparently rare [5]. The high cellularity of lymphoma decreases the extracellular space and restricts the normal random, or Brownian, movement of water molecules. This results in tumor hyperintensity on diffusion-weighted imaging (DWI) and hypointensity on apparent diffusion coefficient maps [3].

Meningiomas usually demonstrate a distinct pattern of contrast enhancement, and may have associated hyperostosis or calcifications. The dural tail sign noted on enhanced MRI is highly suggestive of, but not specific to, meningioma. It is infrequently observed in other cranial masses, such as primary cerebral lymphoma, metastases, glioblastoma and hemangiopericytoma [5]. Chronic SDH are often iso- or hypointense on T1-WI and hypointense on T2-WI, and the capsule typically enhances with contrast administration [2]. Surgery is the appropriate treatment for meningioma and SDH [4].

Although there is as yet no optimal treatment for PDL, surgical excision followed by radiotherapy and chemotherapy are recommended [2,6]. Pathological studies can distinguish lymphoma from other dural diseases.

Magnetic resonance imaging (MRI) axial T1-weighted sagittal (a) and axial images after gadolinium (b) 2 months after surgery. There is a huge posterior scalp mass with adjacent bone involvement away from the surgical site, which shows a cystic residual pouch.
Conflict of interest statement

None.

References


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Spinal cervical metastasis from a glioblastoma multiforme treated by percutaneous vertebroplasty: A case report

Un cas de métastase vertébrale cervicale de glioblastome multiforme traité par vertébroplastie percutanée

A 61-year-old woman was attended in our institution for the treatment of a pathologically proven right temporal glioblastoma multiforme (GBM) revealed by partial seizures. The patient first underwent a surgical resection of this lesion. Then, adjuvant local radiotherapy (60 Gy) and chemotherapy (temozolomide) were administered to the patient. One year later, an asymptomatic C7 vertebra lesion was depicted on MR images. Spine MRI showed a hyperintense signal of the C7 vertebral body on Short Tau Inversion Recovery (STIR)-weighted images (WI) and that enhanced after gadolinium injection. Slight anterior extension was also seen (Fig. 1A). Cervical computed tomography (CT) revealed an osteolytic lesion without disruption of the posterior wall (Fig. 1B and C). It is noteworthy that FDG (18F) PET-CT and bone scintigraphy were both negative. On iterative spinal imaging, progression of the lesion of the C7 vertebra was concomitant with the appearance of a cervical pain. A percutaneous biopsy was performed via an anterolateral approach, under fluoroscopic guidance, that revealed a metastasis from GBM.

A percutaneous vertebroplasty (PV) was proposed as a palliative treatment, and performed in order to obtain pain relief and consolidation of the collapsed vertebra. The patient was placed in supine position and underwent conscious sedation. After local anesthesia with lidocaine, an 11-G, 10-cm-length needle (Thiebaud Biomedical Devices, Margencel, France) was inserted in the C7 vertebral body via a right anterolateral approach under bi-plan fluoroscopic guidance (Fig. 2). Bone cement (Polymethyl Methacrylate [PMMA], Osteopal V, Biomet, Valence, France) was then injected in the vertebral body. Tungsten (5 mg) was added to the cement in order to increase its radiopacity. No complication occurred during the procedure except an asymptomatic mild cement anterior leakage. The patient was discharged 24 hours after the procedure. Cervical pain was highly improved by the PV at 1-month follow-up. No adjuvant cervical radiotherapy was performed.

GBM is an aggressive primitive cerebral neoplasm originating from brain glial cells. In rare cases such as in this case report, extracranial metastases may occur [1, 2].

Extracranial metastases from GBM seem to be more frequent in multifocal GBM, in GBM located in supratentorial space and in periventricular regions. The incidence of these extracranial metastases is higher in patients treated by surgical resection [1]. In decreasing order, metastatic sites from GBM are: lung and pleura (60%), local lymph nodes (51%), bone (31%) and liver (22%) [3].

Vertebral metastases are exceptional and have only been described in few case reports [2, 4]. Imaging aspects of the bone metastases from GBM in the previously published cases were, as in our case, osteolytic on CT-scan and presented a hyperintense signal on T2-WI, with enhancement on T1-WI after gadolinium injection [2].

PV is more and more used since it has been first described for the treatment of aggressive haemangioma in 1987 by Galibert et al. [5]. Moreover, PV has then demonstrated its efficiency to reduce pain related to bone metastases [6].

To our knowledge, no case of PV in a GBM spinal metastasis has previously been reported. We proposed a PV for the palliative treatment of this lesion as an alternative to open surgery and radiation therapy because it was both painful and located within a bearing zone. Open surgery would have been a too invasive procedure for a patient in palliative care. Radiation therapy, even if non-invasive, would not have prevented from the risk of spinal fracture. PV has the advantage of being a minimally invasive technique that provides pain relief and prevents from fracture risk. Compressive symptoms such as radiculopathy or spinal cord compression were absent in this case, but would have contraindicated the PV if present.