result of arteriovenous malformation, venous angioma or capillary telangiectasia.

Repeated MRI reveals the hallmark features of cavernoma: a heterogeneous mass in the optic chiasm containing foci of blood of different "freshness", with a central focus of methemoglobin and a peripheral rim of hemosiderin. As with brain exploration, T2* is the most sensitive technique for demonstrating hemosiderin-laden macrophages around lesions in the optic pathways.

Because of a tendency for recurrent hemorrhage within two years, surgical resection is usually recommended [4]. Evacuation of the hematoma or reduction of the lesion mass improves the visual impairment in 20 out of 30 surgically treated patients. In conclusion, we here report on a rare case of optic chiasm cavernoma evoking a T2* pattern.

References


O. Naggara *
E. Meary
R. Marsico
C. Oppenheim
J.-F. Meder

Department of Morphological and Functional Imaging, centre hospitalier Sainte-Anne, Descartes University, Paris-V, Paris, France

F. Nataf

Department of Neurosurgery, centre hospitalier Sainte-Anne, Paris, France

* Corresponding author. Neuroradiology, Sainte-Anne Hospital, 1, rue Cabanis, 75014 Paris, France
E-mail address: o.naggara@ch-sainte-anne.fr (O. Naggara).


Coexistence of reversible cerebral neurotoxicity and irreversible cerebellar atrophy following an intrathecal methotrexate chemotherapy: Two case reports

Coexistence d’une neurotoxicité cérébrale réversible et d’une atrophie cérébelleuse irréversible dans les suites d’une chimiothérapie intrathécale par méthotrexate : à propos de deux cas

Our first case was a 14-year-old boy with acute B-cell leukemia who was treated with intrathecal methotrexate and Ara-C (cytarabine), but no radiation therapy. Five days after the first treatment, he fell into a coma, which was resolved, although severe ataxia and dysarthria persisted. Fifteen days after symptom onset, MRI findings showed T2-weighted hyperintensities in the supratentorial subcortical white matter (Fig. 1a), cerebellar cortex, deep white matter and thalamus. There was also enhancement of the cortex in the occipital lobes (Fig. 1b and d). Diffusion imaging was normal. On follow-up MRI 24 months later, the high signal intensities were reduced, but severe cerebellar atrophy was evident (Fig. 1e and f). The cerebellar atrophy continued to increase over the next 18 months.

Our second case involved a seven-year-old boy with lymphoma, undergoing intrathecal methotrexate treatment and Ara-C, again without radiation therapy. At 24 hours after his second treatment, he presented with ascending myelitis. Early MRI showed thickening and contrast enhancement of the cauda equina consistent with arachnoiditis (Fig. 2a). Lumbar puncture excluded a central nervous system (CNS) lymphoma. Initially, MRI of the brain was normal but, following the second treatment, there were cerebellar hyperintensities on Fluid Attenuation Inversion Recovery (FLAIR) without contrast enhancement. Follow-up MRI two years later revealed global progressive brain atrophy, but no focal lesions (Fig. 2b–e).

Methotrexate is prescribed for childhood leukemia and lymphoma. Its CNS toxicity is well documented [1]. Acute neurotoxicity is observed in 5.8–18.4% of patients and is usually reversible. Chronic neurotoxicity is rare, but may develop into necrotizing leukoencephalopathy. MRI plays a central role in the assessment of such cases [2,3]. The early MRI changes seen after intrathecal methotrexate treatment may have several origins, such as foci of demyelination or microinfarction. They may also be linked to metabolic or vascular effects, but these are not yet completely understood. Recognition of these changes is important because of the potential confusion with metastatic disease, paraneoplastic syndromes or comorbid neurological disorders that do not require dose reduction or drug discontinuation.

Pathological studies have shown that the acute neurotoxicity seen with methotrexate appears to be due to a direct effect of the drug [3] whereas the late effects are apparently caused by vascular damage induced by the treatment. In our two cases, the early extracerebellar signs were reversible, whereas the cerebellar atrophy appeared later. A reduction of cortical volume in the cerebellar vermis has been found in younger children [4]. Its postnatal development renders the cerebellum particularly vulnerable [5]. In our cases, the early extracerebellar changes regressed, compatible with the available literature, while the cerebellar changes occurred later and were persistent. This supports, on the one hand, different tissue susceptibilities to neurotoxic agents as well as differences in their chronological occurrence. Early subcortical white-matter diffusion and FLAIR abnormalities are asymptomatic and usually reversible [6], and are consistent with leukoencephalopathy. Late atrophy is not related to early MRI abnormalities and is unpredictable. In contrast, acute neurotoxicity is revealed by acute neurological deficits, as seen in both our cases, as reversible imaging features [1]. As for the leptomeningeal enhancement observed in our second
Correspondences 113

Figure 1  First cranial MRI shows cortical T2-weighted hyperintensities (A) and enhancement in the occipital lobes (B, D). Follow-up MRI three months later shows disappearance of the T2 hyperintensities (C), but persistence of enhancement (D). The beginnings of cerebellar atrophy are also evident (C). Follow-up MRI performed 19 months later shows global cerebellar atrophy on T2-weighted images (E, F).

IRM cérébrale initiale montrant des hyperintensités T2 (A) au niveau du cortex ainsi qu’une prise de contraste au niveau des lobes occipitaux (B, D). IRM réalisée trois mois plus tard montrant la disparition des hyperintensités T2 (C) mais la persistance d’un rehaussement après injection de produit de contraste (D). On note une atrophie cérébelleuse débutante (C). IRM réalisée 19 mois plus tard montrant une atrophie cérébelleuse globale sur les images pondérées T2 (E, F).

Figure 2  Sagittal contrast-enhanced T1-weighted image of the spine shows thickened and enhanced nerves (A). Coronal FLAIR (B) and T2-weighting (C) reveal no significant lesions. Follow-up MRI shows global atrophy (D, E), visible along with hyperintensity of the superior cerebellar hemispheres (D).

IRM médullaire dans le plan sagittal pondéré T1 après injection de produit de contraste montrant un épaississement et une prise de contraste des racines nerveuses (A). Les images coronales FLAIR (B) et T2 (C) ne montrent pas de lesions. Une IRM de contrôle montre une atrophie globale (D, E) ainsi qu’une hyperintensité hémisphérique cérébelleuse supérieure (D).

case, chemical arachnoiditis was probably the cause, especially as lumbar puncture excluded CNS involvement of the lymphoma.

References

Schwannome mélanocytique cervical

Il s’agit d’un patient de 24 ans, qui a consulté pour une impotence fonctionnelle de l’hémicorps droit évoluant progressivement depuis six mois. L’examen clinique a retrouvé une hémiparésie droite, un syndrome pyramidal et des téguments normaux. L’IRM a mis en évidence un processus cervical intradural, extramédullaire extériorisé en sablier par le foramen de conjugaison C5—C6 droit qui est élargi. La moelle est refoulée vers la gauche (Fig. 1 et 2). Le processus est hyperintense en T1 (Fig. 3), hypointense en T2 avec une cavité syringomyélique étendue de C6 à D3 (Fig. 4). Ce processus est rehaussé discrètement après injection de gadolinium. Le diagnostic d’une tumeur pigmentée mélanomateuse a été soulevé devant le signal en IRM. La topographie était compatible avec un schwannome. L’étude histologique après exérèse chirurgicale complète a conclu à un schwannome mélanocytique.

Le schwannome mélanocytique représente 1% des tumeurs des tissus mous et 10% des schwannomes intraduraux. Seuls 80 cas ont été publiés [1,2,3]. Il existe deux variantes, l’une non psammomateuse et l’autre psammomateuse faisant partie du syndrome de Carney qui associe des myxomes, des tâches pigmentaires et des anomalies endocriniennes. Notre patient n’avait pas de telles anomalies. Le schwannome mélanocytique atteint le plus souvent les racines nerveuses mais peut toucher les nerfs crâniens et le système nerveux central ou autonome. Des localisations extraneurales sont décrites (estomac, os, tissus mous…) [1,4,5].