A 25-year-old man was admitted with sudden right-sided vision loss. Neuro-ophthalmological findings included a temporal field defect in the right eye and blurred vision. There was a trace of optic nerve pallor with an increase in cup-to-disc ratio compared with the normal left side. The patient spontaneously recovered visual function within a week. Ten days after onset, MRI of the anterior optic pathways demonstrated heterogeneous enlargement of the right half of the chiasm. The mass presented with central hypointensity on both T1- and T2-weighted images (WI) (Fig. 1A, B), without enhancement after gadolinium (Gd)-DTPA injection (not shown). This pattern suggested hemorrhage into an optic chiasm mass. The patient refused surgery despite being informed of the potential for recurrence and permanent vision loss.

Two years later, the patient again suffered an acute loss of eyesight. MRI (Fig. 1C–E) revealed the presence of an area of hyperintensity on T1-WI, with no enhancement after Gd-DTPA injection, and a peripheral hypointense rim on all sequences. Gradient-echo T2 (T2*) imaging was highly suggestive of a lesion containing hemosiderin with marked homogeneous signal loss. Surgical resection confirmed the diagnosis of cavernoma.

Whether sporadic or familial, cavernoma rarely occurs in the cranial nerve. Hemorrhage is a symptom in every case, and patients typically present with sudden-onset focal neurological deficit associated with the afflicted cranial nerve. Only 32 cases of optic tract cavernoma have been reported, including the present case [1–3]. In all of these cases, patients presented with either acute (18 cases) or subacute (14 cases) impairment of the visual field as a consequence of intratumoral hemorrhage. Along with the sudden visual loss and bitemporal hemianopsia, acute frontal or retro-orbital headache was present in 21 of the 32 cases. Fourteen patients had previous episodes of transient blurred vision, reflecting repeated episodes of bleeding. The differential diagnoses include optic chiasm apoplexy, and involvement or compression of the optic chiasm due to pituitary macroadenoma, meningioma, craniopharyngioma, glioma and metastases, or optic chiasm hematoma as a

Figure 1  Initial coronal T1- (A) and T2-weighted images (WI) (B) and, taken two years later, coronal T1-WI (C), T2-WI (D) and gradient-echo T2 or T2* (E) images. The initial MRI examination showed a heterogeneous mass in the right side of the optic chiasm, with central hypointensity on both T1- and T2-WI. Two years later, there was a slight enlargement of the mass, with a hyperintense spot superiorly on T1-WI and a central hyperintensity on T2-WI with a peripheral hypointense rim. On coronal T2*, the mass appeared to be very hypointense, a consequence of the presence of hemosiderin (E). The evolution of the signal and pattern on T2* are highly suggestive of a bleeding lesion — in particular, a cavernoma.
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).

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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