There are 2 peaks in the incidence of the disease, at paediatric age (5-15 years) and at 30 to 40 years.[1] In general, paediatric patients with MMD present ischaemic alterations; the most frequent are transient ischaemic attacks of the anterior territory, especially due to MCA involvement (posterior territory involvement is infrequent and associated with poorer prognosis), followed by cerebral ischaemic strokes. Symptoms are caused by the decreased arterial flow in the affected vessels; several trigger factors have been identified, including hyperventilation, stress, fatigue, and dehydration. In adult patients, however, MMD is usually associated with intracranial haemorrhages.[4] Most of these events are believed to be caused by rupture of the collateral vessels at the base of the cranium and arterial micro-aneurysms, resulting in intracranial haemorrhages, and less frequently subarachnoid haemorrhages.

Diagnosis of MMD is established by angiography findings confirming a steno-occlusive change in at least one ICA and/or its branches. In any case, the complications associated with this diagnostic test, its low sensitivity for detecting the formation of collateral vessels at the base of the cranium, and recent advances in MRI and CT mean that there is no need to perform invasive techniques to establish a definite diagnosis of MMD.

The only treatments with demonstrated positive results are direct and indirect revascularisation.[5] Direct revascularisation consists of the formation of an extra-intracranial bypass between the superficial temporal artery and the MCA (when symptoms affect the posterior territory, the bypass is performed between the occipital artery and the posterior cerebral artery). Indirect revascularisation consists of implanting vascularised tissue (normally temporal muscle) into the dura mater to take advantage of the disease's tendency towards angiogenesis to perfuse the ischaemic areas. These treatments have been associated with clinical and radiological improvements (unravelling the tangle of collateral vessels) and increased survival in several retrospective and prospective studies.[3,6] Such other treatments as antiplatelet drugs or watchful waiting showed no positive results.[3]

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Patient with parkinsonism and a history of methanol toxicity☆

Paciente con parkinsonismo y un antecedente de intoxicación por metanol

Dear Editor:

Methanol poisoning is a severe condition that leads to acute metabolic acidosis, neurological alterations, blindness, and even death. Methanol is not a toxic substance in itself; toxicity occurs when it is metabolised to formic acid, whose accumulation in the body causes the above-mentioned symptoms.[1]

We present the case of a 66-year-old Hispanic patient who visited the neurology clinic due to a 25-year history of postural instability, limited upper limb movement, and dysphonia with no dysphagia or tremor. Physical examination revealed vision loss with no changes in eye movements, bradykinesia, left laterocollis, and upper limb stiffness with no tremor. She also reported previous treatment with amantadine and levodopa/carbidopa, which did not improve her symptoms. A magnetic resonance imaging (MRI) scan showed bilateral lesions in the putamina (Fig. 1). In view of this finding, the patient was interviewed again and described an episode of alcohol intoxication at the age of 22, which caused her vision loss. The poisoning was caused by methanol, a common substance in adulterated alcoholic drinks.

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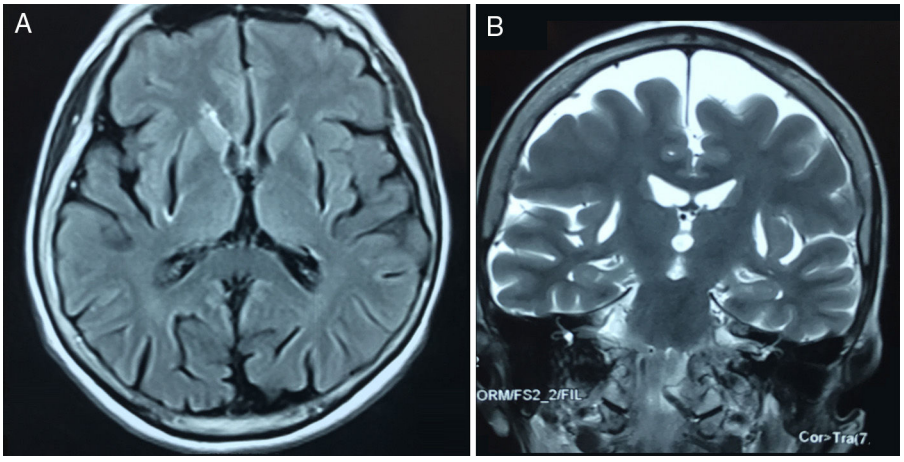

Figure 1 Axial FLAIR (A) and coronal T2-weighted (B) MRI sequences showing bilateral absence of the putamina.

The mechanisms by which methanol causes neurological damage are not fully understood, but formic acid, methanol's toxic metabolite, is believed to be responsible. Formic acid metabolism is very slow, causing it to accumulate in the body, generating a severe metabolic acidosis with an increased osmolar gap.[2]

The neuropathy and subsequent vision loss secondary to methanol intake are caused by the interruption of the mitochondrial function in cells of the optic nerve, leading to hyperaemia, oedema, and atrophy over a period of 12-24 hours after consumption. Damage affects the retrolaminar portion of the optic nerve; minimal or no damage to the retina is observed.[3]

The literature includes few reports of parkinsonism as a long-term consequence of methanol poisoning, due to infarction and subsequent necrosis of the putamen. Formic acid has classically been identified as a mitochondrial toxin which can alter cytochrome c oxidase function, leading to ATP depletion. Another theory suggests that formic acid causes damage by intervening in dopaminergic pathways and increasing dopamine-B-hydroxylase activity.[4] However, there is no clear explanation of why formic acid particularly affects the retina, the optic nerve, and the basal ganglia (putamen) but not other areas of the brain. It has been suggested that excessive accumulation of formic acid may be the cause of the damage; this may be explained by expression of aquaporins, especially aquaporin 1, 3, and 4, which are essential for osmotic and water balance of cells in the brain and the optic nerve.[5] These cells are responsible for transporting not only water, but also polar molecules such as glycerol or methanol to these tissues.

The patient gave written informed consent for the publication of the case and the corresponding images.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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