

be performed, at least in young patients, when no obvious cause is known. However, a more recent report shows a higher sensitivity of MRI in detecting this kind of malformation. In the presented case vertebral angiography was not considered clinically justified. The clinical contribution of MRI in those cases of anterior spinal infarction with an obvious traumatic event and in which a high quality cervical spine MRI has ruled out spinal vascular malformations and vascular dissection, may be limited and should not be considered mandatory.

MRI is a sensitive modality in the evaluation of the spinal cord for infarction. It rules out extradural compression, vascular malformations, and space occupying lesions. The differential diagnosis includes infectious or parainfectious myelitis, multiple sclerosis, and vasculitis. In inflammatory or demyelinating lesions, it is well known that T2 weighted images show lesions some time before the clinical onset, whereas with ischaemic lesions a normal or almost normal MRI is usually seen in the first few hours. The sudden onset of clinical symptoms after a rapid movement of the neck is very suggestive of infarction, but not of myelitis. In the acute phase after infarction the diameter of the spinal cord remains normal and diagnosis is based on signal abnormalities on the long TR sequences, which reflect the presence of cytotoxic oedema. However in the subacute phase, with the appearance of extensive vasogenic oedema, the high signal abnormalities are more evident and associated with cord swelling. In our case the anterior location and the temporal MRI changes, together with the sudden onset and rapid development of typical clinical symptoms, should help in differentiating spine infarction from other spinal cord lesions such as multiple sclerosis and neoplastic conditions.

We have not found any reported case of spinal cord infarction in which MRI was performed in the first four hours after the clinical event. In this acute phase only a subtle anterior linear hyperintensity was identified on the T2 weighted images. This abnormal intensity was not clearly seen on the fast spin echo sequence; however, we were able to identify it with a conventional spin echo sequence. Despite the fact that fast spin echo sequences have been accepted for routine use in the examination of spinal cord lesions, replacing conventional spin echo sequences, the second are more sensitive and should be used in selected cases, when obvious clinical lesions have not been clearly shown on the fast spin echo sequences. Fast spin echo imaging of the spine is in most ways similar to conventional spin echo imaging. However, there may be difficulties in detecting very small intramedullary lesions.⁶

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Myoclonus induced by tacrine

Several adverse effects of tetrahydroaminoacridine, or tacrine, have been reported since 1986, when Summers and colleagues described that its use could lead to an improvement in cognitive defects in patients with Alzheimer's disease. The most common of them are hepatic toxicity, which makes liver function monitoring necessary, and cholinergic effects. Convulsions have also been described.¹ We report on a patient with moderate Alzheimer's disease who presented with non-epileptic myoclonus during treatment with tacrine.

The patient was a 68 year old woman who had been diagnosed with dementia of probable Alzheimer's type four years previously. A mini mental test examination score was 12 points. She had no other disease and was treated with clometiazole. She had no history of epilepsy or myoclonus related to dementia. Four days before admission she had started treatment with 40 mg tacrine daily and 24 hours later she progressively developed generalised uncontrolled abnormal movements, affecting all her limbs and her mouth. These abnormal movements were non-rhythmic, with a spontaneous presentation, although they could be brought on by touch and sound. They were suggestive of myoclonus and were reduced after treatment with clonazepam. Blood analyses were normal. Brain MRI showed a moderate degree of cortical and subcortical atrophy and EEG showed mild and diffuse neuronal dysfunction with an absence of spikes. Myoclonus disappeared 24 hours after withdrawal of tacrine. A few months later, tacrine was restarted to confirm the causative relation. She developed generalised myoclonus during the next 48 hours.

To our knowledge, non-epileptic myoclonus may appear in 20%-30% of patients with very advanced Alzheimer's disease, but non-epileptic myoclonus in association with tacrine in humans has not been previously reported. Tacrine is a centrally active non-competitive reversible acetylcholinesterase inhibitor and its action results in a prolongation of cholinergic activity.² Svejdoва et al reported that non-epileptic myoclonus was induced in baboons (*Papio papio*) by 7-MEOTA (7-methoxytacrine, a tacrine derivative).³ They speculated about a possible anticholinergic effect of 7-MEOTA by an antagonistic action on the muscarinic acetylcholine receptors. This effect has also been suggested by other authors.

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Primary HIV-1 infection presenting with transient neurological deficit

An association between cerebrovascular disease (causing transient neurological deficit, transient ischaemic attacks, or cerebral infarction) and advanced HIV disease or AIDS is recognised.¹⁻³ There is also one report of ischaemic stroke as the first manifestation of HIV infection.⁴ We report a patient with primary HIV-1 infection who presented with a transient neurological deficit. A previously healthy 33 year old right handed male homosexual presented as an emergency with sudden onset of right sided weakness, dysarthria, and dysphasia. He had been unwell for 10 days with fever, pharyngitis, malaise, myalgia, transient non-pruritic macular rash on the upper chest, and transient paraesthesiae affecting the hands. On admission to hospital the neurological features were improving but mild weakness of the right lower limb and expressive dysphasia were present. The neurological deficit resolved completely within 24 hours. Fever (>39°C), a petechial enanthem on the hard palate, and cervical lymphadenopathy were also noted. There were no features of endocarditis or meningeal irritation. Atypical lymphocytes were present in a blood film with normal full blood count. The erythrocyte sedimentation rate was raised at 45 mm/h; serum C reactive protein concentration was 14 mg/l (normal <10 mg/l). A chest radiograph, echocardiogram, cerebral MRI, urinalysis, blood and urine bacterial cultures, and serological tests for syphilis were negative. An examination of CSF showed a lymphocytic pleocytosis (70 lymphocytes/mm³) and increased CSF protein (1.62 g/l; normal <0.45 g/l); HIV RNA gag and pol sequences were detected in the CSF by polymerase chain reaction. Antibody studies indicated recent HIV-1 seroconversion, with rising anti-HIV IgG (enzyme linked immunosorbent assay (ELISA)) and falling anti-HIV IgM (ELISA). Serology for Epstein-Barr virus, cytomegalovirus, and toxoplasma was negative. Serum anticardiolipin antibodies were detected at a low concentration (18 GPL U/ml, normal <10 U/ml); lupus anticoagulant was not detected. Fever and leg pains persisted for three weeks. He was treated with zidovudine, lamivudine, saquinavir, and low dose aspirin, and made a complete symptomatic recovery within one month. No explanation other than HIV infection was identified to account for the neurological features, which were assumed to have an ischaemic cause. Antiviral triple therapy was continued for six months. The most common neurological manifestations of primary HIV infection are lymphocytic meningitis, reversible acute encephalitis, and peripheral mononeuritis; these have previously been found to be associated with accelerated progression of HIV disease. Focal neurological events would seem to occur

rarely during primary HIV illness but they may be underreported; we were unable to identify other reports of transient neurological deficit in this setting. In patients with advanced HIV disease, transient neurological deficit, and cerebrovascular events including cerebral infarction may be associated with non-bacterial endocarditis, CNS infections (including *Cryptococcus* and *Toxoplasma*), and CNS tumours, but they may also occur in the absence of an identifiable underlying cause.¹⁻³ In these patients the mechanism for transient neurological deficit and cerebrovascular events in HIV disease is unknown; anticardiolipin antibodies, which may predispose to cerebrovascular disorders,⁵ were detected in 70% of patients with HIV-related transient neurological deficits in a controlled study.² In our patient, the cause of the transient neurological deficit is unknown; the possibilities include localised ischaemia caused by vascular obstruction or spasm, or a focal inflammatory lesion developing at the time of seroconversion. The preponderance of transient neurological deficit in late stage HIV disease and its occurrence during primary infection in our patient suggest a possible association with high HIV viral load. Primary HIV infection should be considered in at risk patients presenting with unexplained focal neurological symptoms.

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The pallidoreticular pattern of brain damage on MRI in a patient with carbon monoxide poisoning

Neuropathological changes of carbon monoxide (CO) poisoning include the pallidoreticular pattern of brain damage¹ consisting of bilateral necrosis of the globus pallidus and substantia nigra. We here report brain MRI of a CO poisoning victim that showed bilateral lesions of the globus pallidus and substantia nigra. This is the first neuroimaging demonstration of the pallidoreticular pattern of brain damage in a patient with CO poisoning.

A 17 year old previously healthy man was found unconscious in a bathroom. He was transferred to an emergency room and diagnosed as having CO poisoning due to faulty ventilation. Hyperbaric oxygen therapy improved his consciousness from coma to being fully alert. However, he had parkinsonism. Severe akinesia was noted although there was no tremor or rigidity of the limbs. Additional hyperbaric oxygen therapy for six months did not improve his neurological deficit. Brain MRI, a year after CO exposure, disclosed bilateral pallidal lesions: the globus pallidus appeared streaked with a decreased signal intensity on T1 weighted images and an increased signal intensity on T2 weighted images (figure A). Also noted was an increased signal intensity of the substantia nigra on T2 weighted images (figure B). There was otherwise no alteration on brain MRI. A CSF examination showed a decrease in homovanillic acid concentration to 5.9 ng/ml (normal range 28.83-54.28 ng/ml) with normal values of CSF protein and sugar concentrations and normal cell counts.

Auer and Benveniste¹ described the pallidoreticular pattern of brain damage in necropsied cases of CO poisoning. They speculated that the iron rich regions could be affected by binding of CO to iron molecules. In our patient, bilateral pallidal lesions on MRI were consistent with necrosis because of MR signal features. A decreased concentration of homovanillic acid in the CSF² and an increased signal intensity of the substantia nigra on T2 weighted images seem to be due to neuronal loss and gliosis in the substantia nigra.

The pallidoreticular pattern of brain damage¹ to our knowledge has not been reported on MRI in patients with CO poisoning. We emphasise the possible involvement of the substantia nigra in some

victims of CO poisoning. In those patients, parkinsonism would be explained partly by neuronal loss in the substantia nigra in addition to the bilateral pallidal necrosis.³

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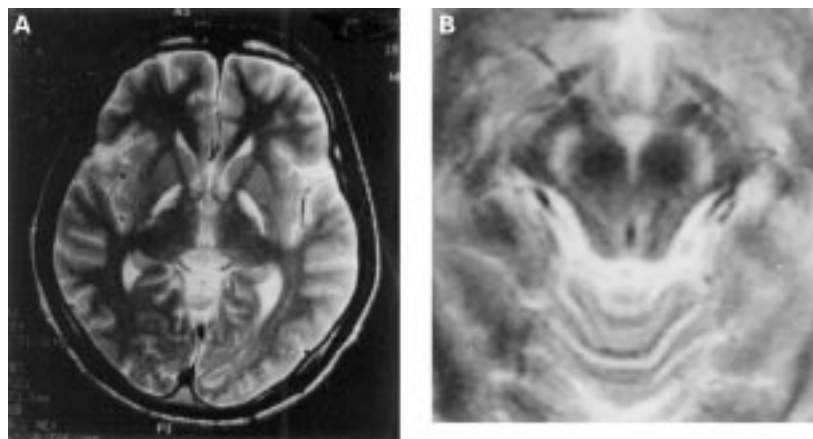
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Information searching for multiple sclerosis

The acquisition of information is an important part of scientific work. In recent years, available information sources have multiplied exponentially, and as a result, we have come to find ourselves immersed in an "information overload". Although we feel committed to keeping up to date with the advances in neurology by using our information seeking skills, paradoxically the new, widely expanded information technologies have made it more and more difficult to gain knowledge about relevant literature sources and to retrieve information. Under the pressure of this situation, many of us have resorted to the passiveness of searching only the most commonly used databases such as Medline and Current Contents to acquire the information we need.

In the frequent practice of retrieving information needed for patient assistance, it is accepted that the use of Medline gives good results for European and North American countries. However, in searches directed towards investigation, we suspected that other, perhaps less accessible databases, could provide valuable additional information. To determine to what extent this might be true, we performed a literature search in the field of multiple sclerosis.

The analysis began by connecting to the 179 Science and Technology databases included in the host Dialog (Knight-Ridder Inc), using the commands for multiple database searching. To avoid classification problems due to variations in the internal structure of the databases, we requested that the search term "multiple sclerosis" appear in any part of the document. As the databases cover different periods and have different indexing policies, we restricted the search to the complete year of 1995 and, at the beginning, to the title field. However, we realised that using only the title would lead to poor results (for example, 613 titles in Biosis instead of the 1100 we used later), so we used the entire reference fields instead. Finally, we excluded databases devoted to medical Industries and ones that are contained in more general information sources (for exam-



(A) Axial T2 weighted MRI at the level of the basal ganglia. The globus pallidus shows an increased signal intensity. (B) Axial T2 weighted MRI of the midbrain. The substantia nigra shows an abnormally high signal intensity.