Abstract
A patient with multiple sclerosis is described who was treated for neurological symptoms thought to be a progression of his disease but subsequently found to be caused by lead poisoning secondary to the use of alternative medicine. His clinical signs improved with oral chelation therapy. Neurologists should consider asking about the use of complementary and alternative medicine before simply attributing symptoms and signs to exacerbation of multiple sclerosis.

Keywords: multiple sclerosis; lead; complementary medication; alternative medication

The use of complementary and alternative medicine is widespread among people with chronic illness1 and between one third and two thirds of patients with multiple sclerosis use alternative medicine, including homeopathic, herbal, chiropractic, and nutritional therapies.2–5 Multiple sclerosis is characterised by a progressive and relapsing time course and fluctuations in neurological status are easily ascribed to the natural course of the disease. Here we describe a patient with multiple sclerosis who was treated for neurological symptoms thought to be progression of his disease but subsequently found to be caused by lead poisoning secondary to using complementary and alternative medicine.

A 47 year old man was diagnosed with multiple sclerosis 9 years previously by two academic neurologists on the basis of slowly progressive ataxia, blurred vision, dizziness, horizontal nystagmus, positive Babinski’s responses, and urge incontinence. The diagnosis was confirmed by MRI. He had been treated with pulse methylprednisolone on six occasions for exacerbation of his symptoms. He also had depression treated with sertraline.

Five months before admission he noted worsening unsteadiness, tremor, impaired memory, irritability, and double vision. This was diagnosed as an exacerbation of multiple sclerosis and he received treatment for 5 days with pulse methylprednisolone. His symptoms continued to progress despite this therapy and he started using a walking stick.

Three months before admission he underwent neuropsychological assessment. This showed impairment of all phases of short term memory, but intact remote and immediate memory. The Wechsler memory scale revised memory quotient was 87, the verbal index was 89, the visual index was 88, delayed recall was 94, and attention/concentration was 127. Apart from attention, these indices were all at the lowest limits of normal and well below expectation. He had a compromised capacity to recognise new ideas. In the Wisconsin card sort trial the number of trials to recognise a shape was 26 (normal range 6–8), the number of trials to recognise numbers was 15 and fall (normal range 4–6), and the number of perseveration errors was 30 (normal range<8). The verbal fluency test was performed very poorly (mean words/letter/minute 5.3, normal 12–15) indicating that he had great difficulty in initiating and maintaining activity to demand. His score on the Rey figure test was below the 10th percentile (32/26 in 171 seconds) which is consistent with poor problem solving capacity. Assessment of mood and affect showed an Institute for Personality and Ability Testing (Champaign, IL, USA) IPAT depression inventory score of 34 out of 80 consistent with depression. The psychologist concluded that there was evidence of moderate neuropsychological impairment involving mainly the central and frontal regions of the brain that was consistent with subcortical demyelination with no localising cortical signs.

One month before admission he noted further worsening of tremor and ataxia with difficulty in bladder control. He also developed periumbilical colic, constipation, and anorexia. He was treated with ranitidine and omeprazole with no effect. His family noted that he became more confused with outbursts of violent behaviour and was occasionally incoherent.

One week before admission a full blood examination showed anaemia with haemoglobin (Hb) of 82 g/l and erythrocyte basophilic stippling. A measurement of blood lead concentration was recommended and was 4.4 µmol/l (92 µg/dl). This was performed by the Central Sydney Laboratory Service by induction coupled mass spectrometry and the recommended range of this laboratory is 0.00–0.48 µmol/l (0–10 µg/dl).

On admission, he was pale and underweight. There were no gingival lead lines. He had mild periumbilical tenderness but the general examination was otherwise normal. He was apa-
Fisher, Le Couteur

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the left upper limb (ulnaris and median) and

the right lower limb (sural) were within

normal limits. Brain MRI showed multiple

focal white matter hyperintensities mainly in

periventricular locations with marked involve-

ment of the inferior corpus callosum and

atrophy involving the cerebellar vermis (fig-

ure).

On subsequent questioning it was found

that 8 months before admission the patient

started a homemade homeopathic remedy
called plumbum metallicum for treatment of

symptoms of multiple sclerosis. The remedy

that he had taken was not available for analysis

but was prepared using lead. He continued

taking this remedy intermittently until admis-

sion. In addition, during this period he started

smoking marijuana for treatment of symptoms

of multiple sclerosis. He used a metallic silver

pipe made in Thailand that had metallic

inlays. A metallic lump the size of a match

head found inside the cone was tested by the

ACT Government Analytical Laboratories. It

contained 26%–39% lead and the laboratory

considered that this would give off large quan-
tities of lead with smoking. His family

members were also tested and not found to

have significant lead concentrations. This

probably excludes domestic or environmental

exposure to lead.

He was treated with oral chelation therapy

using 2,3-dimercaptosuccinic acid (DMSA or

succimer) in recommended doses (30 mg/kg/
day in three divided doses for 5 days and then

20 mg/kg/day in two divided doses for 14

additional days). The medication was toler-

ated well apart from mild metallic taste and

mercaptopurine odour. He responded with a rapid

decrease of lead concentrations from 4.4 to 1.9

µmol/l and improvement in Hb to 135 g/l. This

was accompanied by significant improvement

in his clinical signs. In particular he noted

marked improvement in lower limb power,
ataxia, tremor, cognition, and memory. The

abdominal colic, nystagmus, and diplopia

resolved fully. He was able to walk short

distances without a stick and stated that

he had never felt so good. He required

several courses of succimer because of mild

rebound increases in lead concentrations but

these were not associated with significant

symptoms.

This patient had many typical features of

lead toxicity including confusion, colic, anaem-

ia, and basophilic stippling. The increased

lead and porphyrin concentrations confirmed

the diagnosis. It is of interest that he had

marked cerebellar features and some cognitive

and behavioural problems. We think that these

features were secondary to lead toxicity and not

multiple sclerosis because they responded rap-

idly to chelation therapy and had not re-

sponded to previous methylprednisolone

therapy. In addition, there has been another

recent report of lead encephalopathy that pre-

sented with cerebellar encephalopathy and beha-

vioral disturbance.

Lead is not associated with the pathogenesis

of multiple sclerosis; however, lead poison-

ing can mimic many diseases. Our study indi-

cates that lead poisoning is a possible differen-
tial diagnosis for exacerbation of the

symptoms of multiple sclerosis, in particular

the cerebellar and cognitive features. The

rapid and dramatic response of many of his

symptoms and signs to oral chelation therapy

with succimer suggests that this is an impor-
tant diagnosis to consider.

The most likely sources of lead were a

homeopathic remedy and a pipe used to smoke

marijuana. He took both of these as alternative

medicine for his symptoms of multiple sclero-

sis but did not disclose this to his medical

practitioner. The use of complementary and

alternative medicine among patients with

multiple sclerosis is extensive and this case

report indicates that on some occasions they

may exacerbate symptoms. Therefore, neu-

rologists should consider asking about

such medicines before simply attributing

symptoms and signs to exacerbation of multi-

ple sclerosis.
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Lead poisoning from complementary and alternative medicine in multiple sclerosis

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