T2 weighted brain MRI revealed bilateral symmetric high signal intensities in the SCC, the globus pallidus, the periaqueductal grey matter of the midbrain, the pontine tegmentum, the dentate nucleus, and the medulla oblongata (fig 1D–H). These lesions were hypointense on T1 weighted images (fig 1I). The shape of the SCC lesions was similar to that of case 1.

Discussion

Exposure to high concentrations of methyl bromide can result in gastrointestinal, neurological, and respiratory symptoms.[3 4] The neuropathological alterations associated with methyl bromide intoxication include small subarachnoid haemorrhage, capillary proliferation, demyelination, degeneration of neurons, and gliosis. The sites of involvement include the cerebral cortex, quadrigeminal bodies, red nuclei, dentate and olivary nuclei, and the superior cerebellar peduncles.[4] Ichikawa *et al* reported a case of methyl bromide intoxication involving bilateral symmetrical lesions on MRI in the putamen, the subthalamic nuclei, the dorsal medulla oblongata, the inferior colliculi, and the periaqueductal grey matter of the midbrain.[5]

Methyl bromide intoxication can be suspected on the basis of history of exposure, clinical findings, and results of laboratory studies. There are no reliable indicators of exposure to methyl bromide; due to its short half life, it rapidly becomes undetectable in human tissues, and serum bromide concentrations are considered to correlate poorly with clinical symptoms and outcome.[3]

To our knowledge, lesions confined to the SCC have not previously been reported in patients with methyl bromide poisoning. The imaging differential diagnosis of SCC abnormalities includes Marchiafava-Bignami disease, trauma, infectious diseases, acute disseminated encephalomyelitis, epilepsy, altitude sickness, neoplasia, radiation therapy, chemotherapy, hypoglycaemia, electrolyte abnormalities, renal failure, leukodystrophy, infarction, and hypertension.[1 2] In our cases, these diagnoses were excluded by both the clinical setting and the laboratory findings.

In conclusion, diffuse lesions in the SCC can be seen on MRI of patients with methyl bromide poisoning. If a patient with a splenial lesion is encountered, a detailed history regarding to their occupation and substance abuse should be obtained.

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Wasp sting induced autoimmune neuromyotonia

A 53 year old man was stung behind the knees by five wasps, subsequently identified as belonging to the species *Vespula germanica* (commonly known as ''yellow jackets''). Within a few minutes, he became dizzy, began to wheeze, and collapsed. He responded rapidly to intramuscular epinephrine administered by the attending paramedics, but showed a persistent sinus tachycardia during the subsequent hospital admission. Thus, when he was discharged the following day, he was put on sotalol for 1 week.

He had been stung by a wasp 1 year previously, suffering only extensive local swelling. There was no personal or family history of atopic disease or of reactions to ingested or topical allergens.

He remained free of symptoms for the next 5 weeks, but then developed rapid onset, severe, and painful muscular twitching throughout his limbs, profuse generalised sweating, and insomnia. For 3 weeks prior to his second hospital admission, he was treated with a combination of amitriptyline 10 mg nightly and gabapentin 300 mg thrice daily without effect.

On examination, the patient was afebrile but had hyperhidrosis and tachycardia. He appeared emotionally labile. There were continuous coarse fasciculations throughout all limbs, most markedly in the deltoid and quadriceps bilaterally. The limb tone was normal, with preserved power and normal sensation.

Serum creatine kinase (CK) level on admission was 10 756 IU/l. Serum urea and creatinine levels were normal and no urinary myoglobin was detected in laboratory analysis. The CK level returned to normal following 1 week of bed rest. Electromyography was characterised by spontaneous multiplet motor unit potential discharges with high intraburst frequency, typical of neuromyotonia. There was no evidence of polyneuropathy on nerve conduction study. Electroencephalography demonstrated poorly sustained 8 Hz alpha rhythm with posterior delta slow wave activity. Cerebrospinal fluid examination demonstrated 2 leucocytes/mm^3, protein 0.226 g/l and glucose 4.6 mmol/l (serum 6.3 mmol/l). No oligoclonal bands were detected. Magnetic resonance imaging of the brain and spinal cord, computed tomography scan of the chest and abdomen, and bone marrow aspiration were all normal.

Antibodies to the voltage gated potassium channel (VGKC) were raised, with a titre of 340 pmol/l on admission, in conjunction with a wasp venom specific immunoglobulin E (IgE) level of class 5/6 measured by radioallergosorbent test. Acetylcholine receptor and antineuronal antibodies were not detected.

Electrocardiography revealed paroxysmal atrial tachycardia requiring temporary treatment with oral amiodarone and digoxin. In an attempt to treat the neuromyotonia, the patient received oral carbamazepine up to a dose of 400 mg twice daily for the first 2 weeks after admission, without effect. He was then given a 5 day course of intravenous immunoglobulin at a dose of 0.4 g/kg daily, also without effect. Methylprednisolone 1 g was then administered intravenously every day for 5 days, but the neuromyotonia persisted. Oral mexiletine was then commenced at a dose of 200 mg thrice daily for the following 2 weeks, without significant symptomatic relief. Finally, the patient underwent plasma exchange therapy over 5 days, which was coincident with his symptoms beginning to subside. Nearly 4 months after onset, the neuromyotonia, sweating, and insomnia had completely resolved. Amiodarone and digoxin therapy was withdrawn with no recurrence of the cardiac arrhythmia.

Levels of both VGKC antibodies and total IgE fell in parallel with the patient's clinical recovery over the following weeks (fig 1).

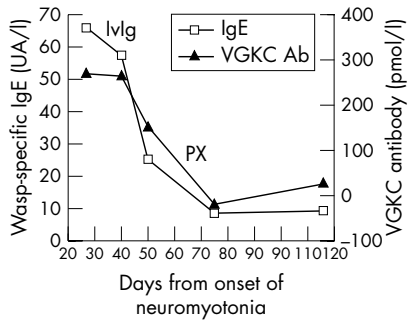

Figure 1 Graph showing the change in VGKC antibody and wasp venom specific IgE levels with time. The first measurements were taken approximately 3 weeks after the onset of fasciculations. There is a close relationship between the two indices, which matched the clinical recovery of the patient. The timing of immunoglobulin therapy and plasma exchange is also shown. VGKC, voltage gated potassium channel titre; IgE, immunoglobulin E titre; IvIg, intravenous immunoglobulin; PX, plasma exchange.

DISCUSSION

Our patient developed severe and refractory generalised neuromyotonia, with evidence of autonomic nervous system dysfunction, 5 weeks after apparent recovery from an anaphylactic reaction to multiple wasp stings. Both VGKC antibodies and wasp venom specific IgE levels were raised, and both fell in parallel with clinical recovery. We cannot be certain which of the treatment strategies, if any, were responsible for the eventual resolution of the condition in our patient; recovery may simply have reflected the natural course of a monophasic autoimmune process.

A delayed syndrome, consisting of central and peripheral nervous system demyelination with a relapsing and remitting course, was described in another patient also stung by a yellow jacket wasp (*V. pennsylvanica*).[1] Cerebral infarction, acute inflammatory polyradiculoneuropathy, encephalomyeloradiculopathy, optic neuropathy, and atrial arrhythmias have all been described as relatively acute sequelae of stings from creatures of the wider order Hymenoptera. Isaacs referred to his (later eponymous) syndrome as one of ''continuous muscle fibre activity'', though the term neuromyotonia has now become synonymous with spontaneous muscle fibre hyperactivity as a result of peripheral nerve hyperexcitability, frequently resulting in visible ''undulating'' myokymia such as that seen in our patient. The CK level is frequently found to be raised. There was no evidence for rhabdomyolysis in our patient.

Neuromyotonia is most frequently an acquired condition. It has been found associated with myasthenia gravis, and as a paraneoplastic entity associated with underlying thymoma and small cell lung carcinoma (11% and 6% of cases respectively in one series[2]), but there was no evidence for this in our patient. About 40% of cases are associated with antibodies to VGKCs that are present on peripheral nerves, with the hypothesis that the mechanism is one of failure to repolarise the distal motor nerve terminal, leading to hyperactivity.

Our patient also developed autonomic dysfunction with prominent hyperhidrosis, emotional lability, insomnia, and cardiac arrhythmia. The ''maladie de Morvan'' (or fibrillary chorea) has been used to describe neuromyotonia occurring with central nervous system features including insomnia, hallucinations, and hyperhidrosis.[3] Cerebral imaging has been reported as normal, as in the present case. Others have detected oligoclonal bands in the cerebrospinal fluid, not found in the present case. VGKC antibodies have been frequently demonstrated in reported cases of Morvan's syndrome, and in a form of limbic encephalitis,[4] suggesting that they may be present in a spectrum of neurological conditions.

Acute focal myokymia has been reported in relation to the venom of the timber[5] and Mojave rattlesnakes. We postulate that the delayed onset syndrome in our patient was due to the development, during the 4 week period after the stings, of underlying VGKC antibodies—that is, an autoimmune, hypersensitivity-type response to an unidentified antigen of the many found in the venom of yellow jacket wasps,[6] possibly sharing epitopes with human VGKCs. The parallel decline in the IgE and VGKC antibody titres with clinical improvement provides limited evidence in support of this hypothesis.

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Left hemianomia of musical symbols caused by callosal infarction

Musical scores consist of two components, musical symbols and pitch notations. Musical symbols are elements of notation that do not denote pitch. Examples include time symbols (for example, ''4/4'', ''rit.'') and dynamic marks (''*f*'', ''*cresc.*'') as indicated by roman letters.[1] Pitch may be defined as the quality of a sound that fixes its position on a scale, indicated by ''notes'' written on, between, above, or below the five lines comprising the musical stave.[1] Case studies suggest that the left hemisphere is dominant in reading pitch notations,[2 3] but opinions are divided on the left hemisphere's superiority for naming musical symbols. In order to investigate this problem, we assessed hemispheric function in a patient with a callosal lesion.

In November 1997, a 69 year old, right handed businessman who suffered from hypertension and diabetes mellitus suddenly developed impairment of the movement of his left hand. When opening a desk drawer with his right hand, his left hand involuntarily caught in the drawer as the result of a cerebrovascular event. He was an amateur violinist and had been active in concerts as a soloist, or as a member in an ensemble. For example, at the opening ceremony of the concert hall of our city, he performed Beethoven's Romance in F major accompanied by the piano. He had been a member of a semiprofessional orchestra for 30 years. He met criteria for Grison's sixth level of musical culture.[4] After this cerebrovascular event, he was unable to play the violin as well as before. Even with familiar musical pieces, his left fingers could not move with their previous accuracy. Left fingers require finer movements than the right, as they press the violin strings. He could read musical scores, and movement of the bow with his right hand was unimpaired. Brain magnetic resonance imaging (MRI) showed an infarct of his corpus callosum (fig 1) which affected the whole of the body and the anterior half of the splenium.

In April 2001, we undertook neurological and neuropsychological examinations. For the statistical analysis, we used Fisher's exact test. A score of 24 points on a revision of Annett's hand preference questionnaire[5] confirmed that he was right handed. He was fully conscious and attained a normal score on the Mini-Mental State Examination (28/30) and Raven Coloured Progressive Matrices (27/36). He clearly remembered daily and social events. A Japanese version of the Rivermead Behavioural Memory Test yielded a normal score: a standard profile score of 18 (mean (SD), 19.73 (2.93)), and a screening score of 9 (mean (SD), 9.15 (1.78)). He had no weakness in any of his limbs, and no sensory disturbances, aphasia, dyscalculia, or visual discrimination difficulties were observed.

Examination of callosal functions revealed left unilateral ideomotor apraxia. To investigate somaesthetic transfer between cerebral hemispheres, the examiner touched a point on the patient's hand with a pen while the patient's eyes were closed, and then asked him to touch the corresponding place on the ipsilateral and contralateral hand using his thumb. When the examiner touched the patient's left hand, the patient correctly identified 16/16 on the left hand and 6/16 on the right hand ($p = 0.0002$); when his right hand was touched, he correctly identified 4/16 on the left hand and 16/16 on the right hand ($p = 0.00002$). Thus our patient

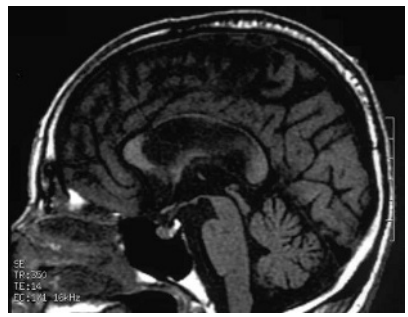

Figure 1 Brain magnetic resonance imaging showed an infarct of the corpus callosum affecting the whole of the body and the anterior half of the splenium.