(10 mg/day). The motor score of the unified Parkinson's disease rating scale (UPDRS) in the off condition was 51, and in the on condition, 37. The dyskinesia score (six body parts, each scored 0–4, maximum score 24)[1] in the on condition was 15. The patient was operated on bilaterally in the STN according to the method of Limousin *et al*[1] with slight modifications, using neurophysiological recording (figure). Antiparkinsonian therapy was initially maintained. Three months after surgery, the motor score of the UPDRS in the off condition was 48, and in the on condition 36, when the stimulation was off; and improved to 37 and 22, respectively, when the stimulation was switched on. The patient had mild dyskinesias in the lower limbs for no more than 10% of the diurnal time. The dyskinesia score was assessed during the maximum motor response to a single morning dose of 50/200 mg benserazide/levodopa. Ten hours before this levodopa test, the stimulation was switched off, and the patient kept off levodopa. The dyskinesia score was 15 when the stimulation was off, and lessened immediately to 2 when the stimulation was switched on.

Unilateral STN stimulation induces hemiballism in healthy monkeys[3] and improves all parkinsonian symptoms,[1] including levodopa induced dyskinesias, in patients with Parkinson's disease.[2] Although the improvement of levodopa induced dyskinesias has been attributed by Krack *et al*[2] to the decrease of levodopa dosage, our patient showed a marked improvement after surgery despite the fact that the levodopa dose could not be decreased after optimising the antiparkinsonian therapy. The improvement of levodopa induced dyskinesias in our patient occurred both during activities of daily living and after a levodopa acute test. To minimise a possible maintained effect of the subthalamic STN stimulation, which hypothetically could have changed the dyskinesia threshold, the patient was in off drug and off stimulation conditions 10 hours before the levodopa acute test. Levodopa elicited a severe peak of dose dyskinesias that were relieved immediately when the STN stimulation was switched on. These data suggest that the effect of STN stimulation is different in healthy monkeys[3] compared with parkinsonian patients with levodopa induced dyskinesias, and suggest that the improvement of levodopa induced dyskinesias could be related directly to the effect of STN stimulation.

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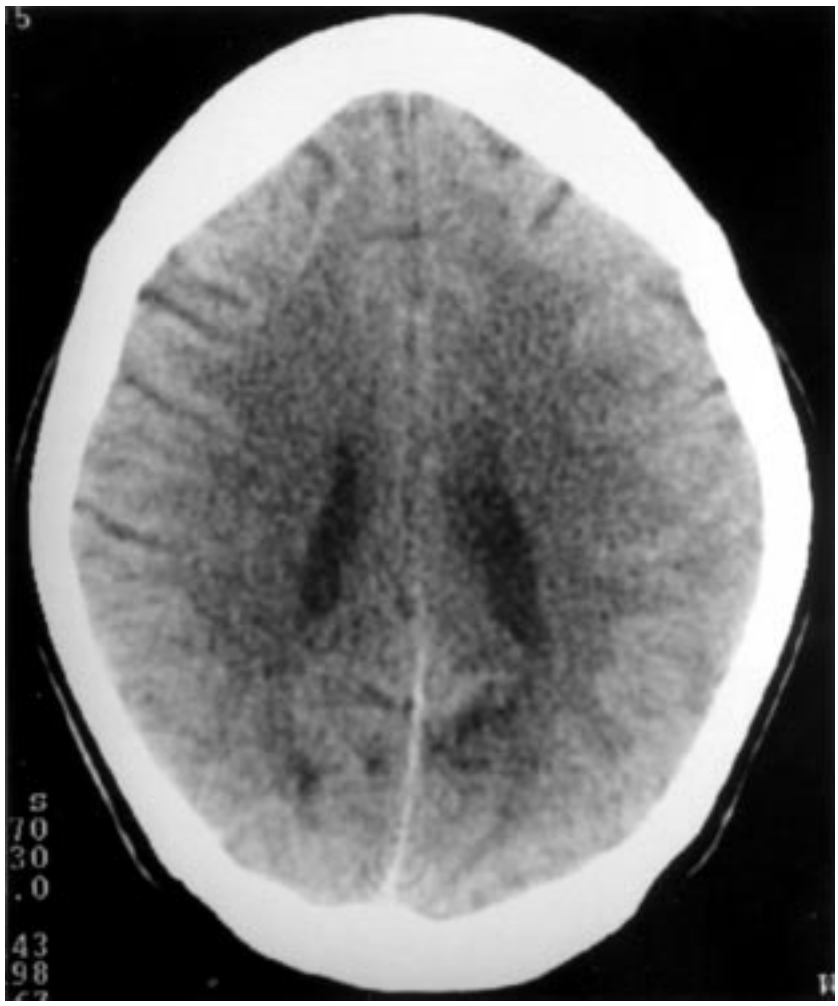
Cerebral infarction: a rare complication of wasp sting

It is stated that four people die in the United Kingdom every year from anaphylactic reactions to wasp and bee stings.[1] However, long term sequelae, including neurological complications, are rare. We report on a young woman who sustained a stroke after a wasp sting and review the literature with particular reference to possible underlying mechanisms of stroke.

A 30 year old woman was seen in a casualty department, 45 minutes after a wasp sting on her left arm. She complained of immediate localised itch, followed by facial and arm swelling and widespread pruritis. She was noted to have a normal conscious level and widespread urticaria and her blood pressure at admission was 90/50. An intravenous infusion of gelofusine was started and she was given subcutaneous adrenaline (1 mg), intravenous hydrocortisone (100 mg), and intramuscular chlorpheniramine (10 mg). Her blood pressure responded and she had no further recorded hypotension. However, after infusion of gelofusine (3 l) over 2 hours she developed respiratory distress and hypoxia. Examination and a chest radiograph showed acute pulmonary oedema and she was intubated and ventilated for 36 hours. She received intravenous frusemide (150 mg in total over 8 hours), but did not require inotropic support. Chlorpheniramine (10 mg thrice daily) and hydrocortisone (100 mg thrice daily) were continued for 48 hours. After extubation she complained of difficulty seeing objects in her right upper visual field and a right homonymous quadrantinopia was demonstrated. Brain CT showed a left occipital infarct (figure).

She subsequently made a full recovery from the quadrantinopia. She was shown to have IgE antibodies to both wasp and bee venom and a positive skin test to wasp venom and underwent successful desensitisation to wasp venom.

Cerebral infarction in this woman occurred in the setting of anaphylaxis to a wasp sting. There was only a single recorded episode of hypotension which was rapidly corrected and was not thought to be sufficient to cause her stroke. The infarct was an occipital cortical lesion and not in a typical border zone distribution.



Computerised tomogram showing left occipital infarct.

Vascular complications of bee and wasp stings are rare. Cerebral infarction has only been reported in three other people.[2 3] In one three wasp stings were followed by collapse and a tonic-clonic seizure. Hypotension was not recorded. He was treated with adrenaline, barbiturates, and steroids. It is unclear whether the development of a hemiparesis preceded or followed this treatment. Brain CT confirmed cerebral infarction. Both other patients died after bee or wasp stings. At postmortem cerebral infarction was found in both.[3] The mechanism of cerebral infarction was not alluded to.

Acute myocardial infarction has been reported four times.[4] It has been suggested that this may be due to a combination of coronary vasoconstriction secondary to mediators released after wasp sting, aggravated by exogenous adrenaline given as part of the treatment and by platelet aggregation.[4 5] It is likely that the mechanism of cerebral infarction in this patient was similar. Wasp venom contains vasoactive, inflammatory, and thrombogenic peptides and amines, including histamine, leucotrienes, and thromboxane. The venom also contains allergenic proteins such as phospholipases which elicit an IgE response, resulting in mast cell activation.[6] Mast cell activation results in release of preformed substances such as histamine as well as de novo synthesis of other mediators. Constriction of coronary arteries has been shown to occur in response to histamine.[7] Both thromboxane and leucotrienes have been shown to be vasoconstrictors.[8] The adrenaline that the patient was given may also have been implicated in vasoconstriction, resulting in her cerebral infarct. Many of the factors released, including thromboxane and leucotrienes, cause platelet aggregation resulting in a prothrombotic state.

The other neurological complications of stings which have been reported are individual cases of ocular myasthenia gravis,[9] optic neuritis, limb numbness, and trigeminal neuralgia[10] and three cases of encephalopathy, one of which was fatal.[11] Postulated mechanisms include both a toxic effect of venom[9] and hypersensitivity to venom.[10 11]

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Cerebrospinal fluid manganese concentrations in patients with symmetric pallidal hyperintensities on T1 weighted MRI

Recently, there have been some reports that MRI shows characteristic brain lesions in patients with parenteral nutrition containing manganese (Mn), or hepatic failure, and that the serum or whole blood Mn concentration is often increased.[1–3] T1 weighted MRI in these patients has shown hyperintensity, always in the bilateral globus pallidus and sometimes in part of the brainstem, although no abnormalities have been found on T2 weighted MRI. The Mn concentrations of CSF in these patients, however, have not been previously measured, because values in control subjects were previously undetermined. The present study was designed to investigate the CSF Mn concentrations in control subjects, and to evaluate the concentrations in patients with symmetric pallidal hyperintensities on T1 weighted MRI.

We examined five patients with the appropriate hyperintensity on T1 weighted MRI, aged from 31 to 72 years (mean 55.8 (SD 16.9) years); two with parenteral nutrition containing Mn (patients 1 and 2), two with Child's grade B cirrhosis (patients 3 and 5), and one without any specific factors relating to Mn or hepatic failure (patient 4, who had parkinsonism). In addition, we investigated 10 age matched control subjects without hyperintensity, aged from 28 to 78 years (mean 54.2 (SD 15.9) years) (table). The MRI was performed on a 1.5 Tesla magnet. In all five patients, T1 weighted MRI in the patients showed hyperintensity in the bilateral globus pallidus and in the region of the substantia nigra or the quadrigeminal plate, although T2 weighted MRI and brain CT showed no abnormalities. Ten control subjects from the neurology and psychiatry service with no history of parenteral nutrition containing Mn, or hepatic failure, showed no abnormal findings on T1 weighted MRI. We obtained blood and CSF samples from the five patients and 10 control subjects with informed consent. The serum, whole blood, and CSF Mn concentrations were measured by a standard method using graphite furnace atomic absorption spectrometry (Model VARIAN SPECTRA A-40) within 1 month after recognition of the symmetric pallidal hyperintensities. The CSF Mn concentrations were measured by diluting the sample with 0.5% (v/v) nitric acid to yield absorbance values within the linear range and injecting 200 µl into the furnace. The mean serum, whole blood and CSF Mn concentrations were calculated for the patients and control subjects. The non-parametric Mann-Whitney *U* test was used to assess the significance of differences between the two groups.

The serum, whole blood and CSF Mn concentrations of the patients and control subjects are listed in the table. All the serum and whole blood concentrations of the control group were within the normal range, and their CSF concentrations were mean 0.47 (SD 0.25)µg/l, a relatively narrow range. The CSF Mn concentration (2.1 µg/l) of patient 4, which was the lowest in the patient group, was much higher than 2 SD above the mean of the control group, but the serum Mn concentration of patient 4 and the whole blood Mn concentrations of patients 1, 3, and 4 were all within the normal range. The serum and CSF Mn concentrations of the patient group were significantly higher (p=0.023 and p=0.002 respectively) than

Serum, whole blood, and CSF Mn concentrations in five patients with hyperintensity on T1 weighted MRI and 10 control subjects without hyperintensity

	Age (y)	*Sex*	*Primary disease (parenteral Mn dose (mg/day)/duration (days))*	*Serum Mn (µg/l)*	*Whole blood Mn (µg/dl)*	*CSF Mn (µg/l)*
Patients:						
1	31	F	Pylonephritis (1.1/20)	1.7	1.6	6.7
2	46	F	Wernicke's encephalopathy (1.1/51)	2.1	4.4	3.8
2	64	M	Child's grade B cirrhosis	2.2	2.4	3.0
4	66	M	Parkinsonian syndrome	1.2	1.6	2.1
5	72	F	Child's grade B cirrhosis	2.3	4.2	3.1
Mean (SD)	55.8 (16.9)			1.90 (0.45)	2.84 (1.37)	3.74 (1.76)
Control subjects:						
1	28	F	Acute disseminated encephalomyelitis	1.5	1.7	0.7
2	40	M	Chorea-acanthocytosis	1.5	2.0	0.9
3	43	M	Multiple sclerosis	1.3	1.7	0.2
4	45	F	Neurosis	0.9	1.0	0.6
5	49	M	Guillain-Barré syndrome	1.9	2.0	0.4
6	57	F	Parkinsonian syndrome	1.0	1.3	0.6
7	61	F	Malignant syndrome	0.8	1.4	0.2
8	70	M	Progressive supranuclear palsy	0.9	1.1	0.3
9	71	M	Progressive supranuclear palsy	1.2	1.5	0.6
10	78	M	Parkinson's disease	1.3	2.8	0.2
Mean (SD)	54.2 (15.9)			1.23 (0.34)	1.64 (0.53)	0.47 (0.25)

Normal ranges: serum Mn 0.2-1.6µg/l; whole blood Mn 1.3-3.1µg/dl; CSF Mn concentrations have not been determined.