LETTERS TO THE EDITOR

Guillain-Barré syndrome following jellyfish stings (*Pelagia noctiluca*)

Stings from jellyfish (*Cnidaria: Scyphomedusae*) are common world-wide and pose a significant public health problem in some coastal areas. They usually cause a mild local dermatitis, but rarely serious or fatal systemic reactions occur.¹ The neuromuscular manifestations reported include localised neuropathy and mononeuritis multiplex,²⁻⁴ but no case of Guillain-Barré syndrome following jellyfish stings has been described.

A 39 year old English man sustained multiple jellyfish stings over both legs in July off the north coast of Majorca. There was immediate local discomfort and a few punctate lesions developed over a few minutes. He caught the jellyfish and described it as palm sized, translucent and red. One week later, he complained of tingling in both heels. During the ensuing week the tingling spread to the hands and he developed mild proximal weakness of the limbs. He was unsteady and unable to work as a van driver. He had been well and had had no recent respiratory or gastrointestinal illness.

The patient was referred to us two months from the onset by which time his symptoms had generally improved. He had minimal proximal muscle weakness in the legs, mild diminished sensation to light touch and pinprick in a glove-and-stocking

distribution. He also had an unsteady gait. All the tendon reflexes were absent except for a barely elicitable left knee jerk. Position and vibration sense were intact. Nerve conduction studies showed a prominent demyelinating neuropathy with conduction block (table).

One month later, there was no weakness or sensory disturbance, but there was still mild ataxia of gait. The knee jerks were just present bilaterally. Repeat nerve conduction studies showed some improvement. He was seen again three months later and clinical examination including reflexes were normal. The nerve conduction studies were further improved (table).

Jellyfish are stinging aquatic invertebrates that belong to the phylum Cnidaria. Most jellyfish are harmless to humans but a few can cause serious problems. They have stinging organelles (nematocysts) which can penetrate the upper dermis and discharge venom which diffuses into the systemic circulation. The venoms are polypeptides and enzyme compounds which may be both toxic and antigenic to humans.¹ The principal clinical reactions appear to result from a direct toxic effect, although allergic reactions may play a significant role in the pathophysiology of jellyfish stings.⁵

The most common adverse reactions are mild local dermatitis; rarely serious or fatal systemic reactions occur. The neurological manifestations reported include delirium, stupor, central respiratory failure and muscular weakness.¹ Two cases of localised neuropathy following jellyfish stings^{2,3}; and a case of mononeuritis multiplex after suspected man-of-war (*Physalia physalis*) sting⁴ have been reported.

Our patient developed Guillain-Barré syndrome, presumably on the basis of an

aberrant immune response to the jellyfish venom. This was also thought to be the underlying mechanism in the case of mononeuritis multiplex described, in which an acute radial neuropathy developed in the arm stung, followed by involvement of the radial, ulnar and axillary nerves in the contralateral arm one week later.⁴

P. noctiluca is present world-wide and is common in the Mediterranean sea. Jellyfish stings in the Mediterranean sea coincide with the swimming period which begins in May and extends to September, with peaks in the months of July and August. Our patient's description of the jellyfish fits that of *P. noctiluca*.

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Table 1 Serial nerve conduction studies.

Date	Motor										Sensory					
	Right peroneal					Right tibial					Right median					Right radial (antidromic) Amp (µV)
	DML (ms)	Amp(mv) dis	prox (knee)	F (ms)	CV (m/s)	DML (ms)	Amp(mv) dis	prox (knee)	F (ms)	CV (m/s)	DML (ms)	Amp(mv) dis	prox (elbow) (ms)	F (ms)	CV (m/s)	
6.10.92	8-8	1-5	1-0	85-2	19	7-6	0-6	0-2	83-2	23	5-6	2-3	2-5	37-2	46	12
9.11.92	8-0	2-1	0-8	Absent	33	6-0	0-5	0-2	74-8	24	5-6	6-0	6-0	39-8	50	27
1.02.93	7-6	1-0	1-0	62-8	36	7-3	1-0	0-3	63-3	36	5-4	11-0	10-0	32-2	50	30

DML—distal motor latency
Amp—amplitude
CV—conduction velocity

Recurrent Guillain-Barré syndrome following acute filariasis

Filariasis is one of the commonest parasitic disease, affecting over 150 million people. It is widespread in tropical and subtropical zones, and is a markedly disabling condition because of lymphatic, ocular, and skin complications.¹ Among filariasis, a lymphatic form (*wuchereria*), two cutaneous forms (*loiasis* and *dracunculiasis*) and a serosal form (*Dipetalonema perstans*) are capable of causing neurological disease. Neurological manifestations associated with or attributed to infection with various species of filaria, however, are reported to be rare.¹⁻³

Among the parasites, vivax malaria complicated by Guillain-Barré syndrome has been reported by Padmini and Maheswari⁶ but the association of filariasis with the syndrome is hitherto unreported. We report the first patient with features of recurrent Guillain-Barré syndrome in whom each episode was preceded by a severe attack of acute filariasis.

The patient, a 43 year old man known to have filariasis with lymphoedema of the

right leg, presented with three episodes of Guillain-Barré syndrome, each of which was preceded by a severe attack of acute filariasis.

In September 1987, he presented with a severe attack of acute filariasis, manifested by high grade fever with chills and rigors, tender inguinal lymphadenopathy and lymphoedema of the right leg. Blood analysis showed leukocytosis (Tc-14 000/mm³), eosinophilia (18%), a raised erythrocyte sedimentation rate (37-mm/h), and numerous microfilariae later identified as *Wuchereria bancrofti*. The serum immunoglobulin E level was raised. Other routine urine, haematological, and biochemical tests were normal. He was given diethylcarbamazine citrate 300 mg/day in three divided doses, with additional analgesics and antipyretics. Within one week, he became afebrile and the painful swellings in the right inguinal region and his lower leg also regressed.

Three weeks later, he developed glove and stocking paraesthesiae. This was followed by the development of flaccid, areflexic tetraparesis (MRC grade 2/5 proximal

and 1/5 distal group of muscles) with severe, bilateral, lower motor neuron facial weakness. The neurological deficit reached a peak after three weeks. The plantar responses were flexor and there was distal loss of all sensory modalities in all four limbs. The peripheral nerves were not thickened. Examination of CSF on the 10th day showed an acellular response with an elevated protein content of 500 mg/dl. A stained specimen (Giemsa) of CSF did not reveal any microfilariae. Nerve conduction studies were suggestive of demyelinating radiculoneuropathy (table). Thus in view of the clinical picture and evidence of demyelinating radiculoneuropathy, a diagnosis of Guillain-Barré syndrome preceded by acute filariasis was made.

The patient was put on supportive treatment. Over the next four weeks he recovered sufficient limb function, so that he could feed himself and stand with support. Repeat electrophysiological testing at this stage indicated a mild improvement in motor and sensory nerve conduction. He continued to improve with physiotherapy and was discharged after six weeks in