LETTERS TO THE EDITOR

Occipital neuralgia: another benign cause of "thunderclap headache"

Headaches characterised by paroxysmal onset of severe, generalised pain ("thunderdap headache") are regarded as early signs of subarachnoid haemorrhage. It has even been suggested, that despite normal cranial CT and CSF studies, thunderclap headaches may be a sign of an unruptured intracranial aneurysm therefore heralding an increased risk for subarachnoid haemorrhage.[1] However, recent reports on the long term follow up of patients with thunderclap headaches show that with normal CT and CSF studies, thunderclap headaches are usually benign,[2] often vascular in origin.[3]

Over the past four years we have seen 12 patients (ages 27–54 years; 7 women, 5 men) with occipital neuralgia presenting with excruciating, generalied headaches of explosive onset. None had rheumatoid arthritis, recent trauma, or cervical spine abnormalities. The pain was shooting in 8 and throbbing in 4. All patients described nausea, vomiting and photophobia. Eleven experienced blurry vision, 7 vertigo or diziness, 5 neck stiffness, and 5 stuffiness of the nose. Pressure on the greater occipital nerve produced a positive Tinnel's sign in all cases, and 9 had hypoaesthesia in the sensory distribution of C2. Within 30 minutes of an occipital nerve block with 1–2 cc of 1% lidocaine, complete pain relief was achieved in 7 patients and significant improvement (residual pain less than 10% severity) in all the others. CT scans in 12/12 and CSF analysis in 5/5 were normal. Cervical spine x rays were normal in all patients.

Occipital neuralgia is caused by irritation or injury to the greater occipital nerve, and is generally characterised by uni- or bilateral throbbing pain that frequently radiates to the forehead and around the eye.[4] However, our patients illustrate a much more dramatic form of presentation mimicking subarachnoid haemorrhage. The greater occipital nerve is the continuation of the second cervical nerve root and receives branches from the superior cervical sympathetic ganglion, the trigeminal ganglion, the acoustic, and the vestibular nerves.[5] Occipital neuralgia is therefore frequently associated, as in the case of our patients, with autonomic dysfunction in the neck and face, vertigo, nose stuffiness, and visual disturbances.[4] The diagnostic clinical features for occipital neuralgia are the presence of a sharply circumscribed area of tenderness over the greater occipital nerve trunk as it crosses the superior nuchal line, sensory changes in the C2 distribution, and the response of the pain to infiltration of local anaesthetic near the tender area of the nerve trunk.[6] If the diagnosis of occipital neuralgia is established, the only potentially serious underlying conditions are craniovertebral anomalies of the cervical spine and cervical arthritis, which can be detected with cervical spine x rays.[6 7] Cervical spondylosis can present with thunderclap headaches and lead to chronic headaches associated with neck movements.[2]

Timely clinical recognition of the acute form of occipital neuralgia can help avoid unnecessary testing in patients with thunderclap headache and lead to the appropriate treatment with almost immediate pain relief.

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Pure sensory Guillain-Barré syndrome

The existence of a purely sensory form of Guillain-Barré syndrome is still subject to controversy, although the criteria for its diagnosis have been established. We report the case of a patient who had acute sensory neuropathy which, due to its clinical, cerebrospinal fluid and electrophysiological characteristics, may be considered a sensory form of Guillain-Barré syndrome.

Three days before admission, a 69 year old woman developed a sensory deficit, with a sensation of tightness and dysesthesias in her feet and hands, which increased in intensity and extension during the following days. Two days later she showed a markedly unsteady gait and clumsiness in handling her upper extremities. She was admitted to our centre the next day. She had been vaccinated against influenza a month before and had suffered from intense sore throat, specially on swallowing, without fever during the two weeks before admission.

On neurological examination, her mental status, cranial nerves, strength, light touch and pinprick sensation were normal, while there was a complete loss of vibration and arthrokinetic sense. There was dysmetria of the four extremities, the intensity of which rose considerably when she closed her eyes, hypotonia of the upper extremities, pseudoathetoid movements and total areflexia with cutaneous plantar reflexes in flexion. She showed marked truncal ataxia, a broad-based gait, and was unable to stand or walk without support. Romberg's sign was positive.

Routine laboratory tests (except for an ESR of 37 mm/h), levels of folic acid, vitamin B12, tumour markers (alfa-fetoprotein, CA-125, CEA), HIV and syphilis antibody tests, ANA, antiDNA, anti-Ro, anti-LA and antimitochondria antibodies, were normal. Serum IgG anti-Hu were negative. ECG, chest x ray, abdominal ecography and a thoracoabdominal CT scan were also normal. Two CSF examinations were carried out, on the

Table Nerve conduction values

	Day 6[1]	Day 18	Day 110	Lower limit of normal range
Motor				
Right median				
Amplitude (mV)	3·08	1·6	1·25	5·8
Distal latency (ms)	4·8	4·4	5·2	3·2
Conduction velocity (ms^{-1})	45·6	48	51·0	33·8
F wave latency (ms)	nd	nd	26·8	28·7
Left median				
Amplitude (mV)	2·96	nd	nd	5·8
Distal latency (ms)	3·8	nd	nd	3·2
F wave latency (ms)	23·6	nd	nd	28·7
Right ulnar				
Amplitude (mV)	6·44	nd	nd	5·3
Distal latency (ms)	2·8	nd	nd	2·8
Conduction velocity (ms^{-1})	50	nd	nd	47·4
Right peroneal				
Amplitude (mV)	2·28	nd	3·08	1·2
Distal latency (ms)	5·6	nd	5·6	4·2
Conduction velocity (ms^{-1})	42·3	nd	43	42·2
Sensory				
Right median (III-Wrist)				
Amplitude (μV)	ns	3·3	14·6	12·3
Distal latency (ms)	ns	3·7	3·1	2·2
Right ulnar (V-Wrist)				
Amplitude (μV)	ns	1·1	4·4	4·2
Distal latency (ms)	ns	3·1	1·9	1·8
Right sural				
Amplitude (μV)	ns	nd	2·6	6·4
Distal latency (ms)	ns	nd	3·7	3·3
Conduction velocity (ms^{-1})	ns	nd	37·8	41
Left sural				
Amplitude (μV)	nd	nd	2·9	6·4
Distal latency (ms)	nd	nd	3·9	3·3
Conduction velocity (ms^{-1})	nd	nd	35·8	41
Mixed				
Right median				
Amplitude (μV)	ns	1·2	1·8	5·5
Conduction velocity (ms^{-1})	ns	48·9	55·6	51·7
Left median				
Amplitude (μV)	ns	nd	nd	5·5
Conduction velocity (ms^{-1})	ns	nd	nd	51·7

LL = lower limit of normal range; ns = not seen; nd = not done.
[1] Days from the onset.