Lamotrigine control of idiopathic trigeminal neuralgia

Carbamazepine is the drug of choice for idiopathic trigeminal neuralgia, being effective initially in 75% of patients; no other available drug is as effective, albeit pimozide and oxcarbazepine may be superior.1 Unfortunately, up to one third of patients cannot tolerate the drug in the doses required to alleviate the pain, and carbamazepine may cause aplastic anaemia, agranulocytosis, and hypersensitivity reactions.3 Carbamazepine may control idiopathic trigeminal neuralgia by suppressing Na+ currents either in the trigeminal causal nucleus or, selectively, in the gasserian ganglion.2 Recently, a novel antiepileptic drug lamotrigine has become available, and this is at least as potent as carbamazepine in inactivating Na+ currents, with fewer side effects. A search of the medical literature did not disclose previous studies of lamotrigine effects on idiopathic trigeminal neuralgia. Thus we obtained authorisation to prescribe lamotrigine in four patients with idiopathic trigeminal neuralgia, from whom informed consent was obtained.

Patient 1, a 55 year old man, developed typical idiopathic trigeminal neuralgia paroxysms in the right trigeminal branch. Oral carbamazepine (200 mg twice daily) almost completely controlled the paroxysms. In view of possible complicating side effects, the patient accepted the switch to lamotrigine. Carbamazepine was then stopped and replaced with lamotrigine on the following day (at which time the paroxysms had recurred) at 50 mg once a day by mouth, increased by 50 mg aliquots each day. At 100 mg per day, the paroxysms were controlled to a large degree, and relief grew to complete control at 100 mg three times a day. No adverse effects have been seen over six months.

Patient 2 was a 31 year old woman who developed typical idiopathic trigeminal neuralgia attacks involving the first three right branches. Carbamazepine at 200 mg twice a day almost completely controlled the paroxysms, with some attendant somnolence. At 600 mg daily, control was complete, but the patient was severely ataxic and could not drive. Discontinuation of treatment resulted in relapse. Lamotrigine produced complete relief at 400 mg in divided doses, without side effects, over six months.

Patient 3, a 75 year old woman, developed typical idiopathic trigeminal neuralgia in 1973 in the first and second left branches. Carbamazepine was effective only at 2000 mg (complete relief), with considerable side effects. Alcohol injection of the gasserian ganglion gave complete remission for three years. Subsequent recurrences were again treated with alcohol injection, but relief was always shorter. Glycerol injection was effective for four months. Idiopathic trigeminal neuralgia recurred. There were no sensory deficits or dysaesthesiae. Lamotrigine, begun as for patient 1, gave 90% relief at 150 mg three times a day by mouth. After two months, however, the economic burden on the patient led to Fogarty percutaneous compression of the gasserian ganglion, with analgesia at short term (2 weeks) 199-212.

Patient 4 was a 72 year old man who had frequent attacks of idiopathic trigeminal neuralgia in the left second and third branches for a few years. A sense of burning in the gums was reported lately. Notably, 50% of the attacks presented at night. Speaking and chewing triggered intolerable pain. Carbamazepine at 200 mg twice a day initially controlled the attacks, but a very soon produced cardiovascular intolerance. Carbamazepine was replaced with lamotrigine and attacks were completely controlled at 400 mg in divided doses over five months.

Lamotrigine may cause initial ataxia, diplopia, nausea, vomiting, and blurring of sight in 15-35% of the patients treated for epilepsy, but these disappear or are much reduced after dose adjustments.1 An allergic skin rash is seen in 3-17% of the patients. This can be reduced to no more than 10% if the drug is started at 50 mg once a day for two weeks, 50 mg twice daily for another two weeks, and then brought to a maintenance dose of 200-400 mg in divided daily doses.3 A similar regimen may be applied for idiopathic trigeminal neuralgia, although use of the faster dose increase schedule for rapid control. Doses of lamotrigine can be as high as 1300 mg daily. Carbamazepine and phenytoin speed up the elimination of the drug, whereas valproate slows it.

Lamotrigine is a potent antiglutamaterge.1 Depression of excitatory transmission in the trigeminal causal nucleus is believed to be part of the range of action of anti-idiopathic trigeminal neuralgia drugs, and thus lamotrigine relief of idiopathic trigeminal neuralgia may not necessarily be due to Na+ current inactivation.1 Our preliminary data require confirmation with a placebo controlled study.

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Clinical evaluation of vasospasm in subarachnoid haemorrhage by in vivo microdialysis

Patients in whom subarachnoid haemorrhage is complicated by vasospasm are at risk of developing cerebral infarction. Prophylactic use of such a combination, unfortunately, be difficult. Animal experiments have shown that cerebral ischaemia is associated with raised extracellular glutamate concentrations, which can be measured by in vivo microdialysis. We have used this technique with subarachnoid haemorrhage in whom extracellular glutamate was monitored, by in vivo microdialysis, for four hours every day for four days after operation.

A 38 year old farmer reported to the hospital with a five day history of severe frontal headaches, nausea, and vomiting. Before admission he had had an episode of right sided mild visual loss lasting for 24 to 36 hours. He also complained of photophobia and mild neck stiffness. Except for the neck stiffness no focal neurological findings were evident on examination. Lumbar puncture was immediately performed.

Intracranial Codman bolt was inserted over the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was complicated by intraoperative rupture of the superior projecting aneurysm during the application of the aneurysmal clip. An intracranial Codman bolt was inserted over the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT. A four vessel angiogram disclosed two large separate wide necked aneurysms on the right anterior communicating artery projecting into the right frontal lobe. Subarachnoid haemorrhage was confirmed on cranial CT.
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The use of microdialysis in patients with subarachnoid haemorrhage may be useful in the early detection of vasospasm and evolving cerebral ischaemia or infarction.

Figure 1 CT showing bilateral frontal hypodensities. (A) day 1; (B) day 3 (arrows).

Figure 2 Extracellular glutamate concentrations during the four days of recordings. Values are means (SEM).

Patients with subarachnoid haemorrhage or other conditions with a propensity to cerebral ischaemia.

In conclusion, we report on a patient with subarachnoid haemorrhage in whom the extracellular cerebral glutamate concentration was measured with in vivo microdialysis for four days after operation. There was a good correlation between the development of vasospasm, the clinical state, and the extracellular glutamate concentrations. In vivo microdialysis may be a useful predictive tool in labile cerebral ischaemic situations.

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