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,1 although pizotifen and oxcarbazepine may be superior.2 Unfortunately, up to one third of patients cannot tolerate the drug in the doses required to alleviate the pain,3 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 neurons or in the gasserian ganglion.4 Recently, a novel antiepileptic drug lamotrigine has become available, and this is at least as potent as carbamazepine in inactivating Na+ currents,5 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 paroxysmal and 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 recursed) at 50 mg once a day by mouth, increased by 50 mg aliquots each day until a dosage level of 200 mg twice daily was reached after two weeks and then brought to a maintenance dose of 200–400 mg in divided daily doses.6 A similar regimen may be applied for idiopathic trigeminal neuralgia, although the need for faster dose adjustments may require an 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 antiguillatumergetic agent.1 Depression of excitatory transmission in the trigeminal caudal nucleus is believed to be part of the range of action of anti-idiopathic trigeminal neuralgia drugs,2 and thus lamotrigine relief of idiopathic trigeminal neuralgia may not necessarily be due to Na+ current inactivation.7

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. Preventing such a complication is, unfortunately, difficult. Animal experiments have shown that cerebral ischaemia is associated with raised extracellular glutamate concentrations, which can be measured by in vivo microdialysis. We report here monitoring 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 mid facial weakness lasting for three to four 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 revealed a normal opening pressure, and a CSF sample was normal. Intracerebral microdialysis was performed as previously described2 after stabilisation of the patient in the intensive care unit. The catheter was positioned at the M2 segment, 298-09 (19-9) μmol/l. This was “high” in comparison with the baseline normal in humans (20-25 μmol/l) as previously reported.2 An angio gram one day later showed severe focal vasospasm (no filling of the right anterior cerebral artery. Subsequent CT showed bilateral frontal hypodensities suggesting ischaemic changes (fig 1). The early severe focal vasospasm may have possibly complicated the development of the aneurysm clip. At the time of the initial study, the patient was very drowsy. The samples on day 2 disclosed a significant decrease in the glutamate concentration to 10 (9-86) μmol/l, which may have represented an improvement in the level of consciousness in the patient. On day 3 there was some increase in drowsiness, possibly related to generalised vasospasm and impending bifrontal ischaemia. The microdialysis fluid collections on this day also showed a significant increase in extracellular glutamate concentrations, to 242-53 (19-71) μmol/l. On day 4 clinical state improved with the patient being fully awake. The microdialysis recordings during this time showed a progressive decline in the glutamate concentration to 42-13 (3-77) μmol/l (see fig 2). This was supported by an improved clinical state, as judged by a committee on human experimentation at the University of Saskatchewan and informed consent was obtained before the study.


