Gamma vinyl GABA in the treatment of Levodopa-induced dyskinesias in Parkinson’s disease

In non-human primates blockade of the GABAergic inhibitory striato-pallidal pathways to the lateral segment of the globus pallidus causes chorea, whereas stimulation causes a Parkinsonian syndrome. This has led to renewed interest in the potential value of gabapentinergic agents in the treatment of Parkinson’s disease and the complications of levodopa therapy.

We have investigated the irreversible inhibitor of GABA transaminase, gamma vinyl GABA (GVG) in the treatment of disabling levodopa-induced chorea in 5 patients with Parkinson’s disease. The patients had a mean age of 54 (41–74) years, a mean duration of disease of 11 (7–21) years, a mean duration of levodopa therapy 9 (6–18) years and a mean levodopa dose of 760 (450–1400) mg/day. All the patients were also receiving subcutaneous apomorphine, 4 selegiline and 1 bromocriptine.

The patients’ dyskinesias were assessed over a one week baseline period on optimum anti-Parkinsonian therapy. They were then given GVG 3 Gm/day of GVG for one week and then 3 Gm GVG for a second week. Assessment of dyskinesia severity was carried out using a 4-point scale after a standard therapeutic dose of sc apomorphine.

The patients began self-scoring diaries for three days of each week to assess the number of hours “on” with and without dyskinesias and the number of hours “off”. Baseline assessments showed that dyskinesias were more severe later in the day in all patients. On GVG no change in dyskinesia severity occurred as judged by either the apomorphine challenges or the self-scoring diaries, but there was a mean increase in off hours from 3 to 4 hours.

Four patients were unable to tolerate more than 2 Gm GVG due to increased severity of Parkinsonian symptoms. The other patient also noticed worsening of Parkinsonism on 3 Gm GVG.

Contradicting results with probabide, a gabamnergic agonist, have been reported in levodopa induced dyskinesias in Parkinson’s disease.3 GVG was reported to aggravate Parkinsonism without improving tardive dyskinesias in psychotic patients on sustained neuroleptic therapy. GABA mimetic drugs therefore appear to have complex and contradictory actions in patients with movement disorders. This study is of interest in that aggravation of Parkinsonism occurred without significant reduction in dyskinesias suggesting that these two phenomena may not be inextricable.

Correspondence to: Dr Jalil, Avda Salvador 2194, Santiago, Chile


Hypochromia iridis in acquired Horner’s syndrome

The uncommon condition of congenital Horner’s syndrome consists of ptosis, miosis, facial anhidrosis and hypochromia of the affected iris. This condition commonly results from injury to the brachial plexus at birth. The mechanism of the hypochromia iridis is generally thought to be that of failure of pigment development rather than loss of pigment that has already formed. Hypochromia of the iris following acquired Horner’s syndrome has been reported but is rare. We report a case of this rare but interesting manifestation of damage to the sympathetic nervous system to the eye.

A 17 year old man was involved in a motorcycle accident and suffered brachial plexus trauma, with loss of power and sensation in the right arm followed by pain. Examination 23 years later revealed partial C7 and complete C8 and T1 paraparesis with corresponding sensory loss. He had post-traumatic brachial plexopathy pain for which he was seeking advice. Examination also revealed a right Horner’s syndrome with loss of pupil in the right eye, his left being coloured grey/green.

Several mechanisms by which alteration in sympathetic activity may influence iris pigmentation have been proposed.1 There may be failure of delivery of noradrenaline or other melanin precursors to the melanocytes in the iris, perhaps mediated via cyclic adenosine monophosphate. There may be loss of activation of prostaglandins, or their precursors, or some melanotropic moiety, that are involved in melanin synthesis. Several cases of depigmentation of the iris2 or heterochromia3 of the same eye may occur. These are thought to be caused by congenital or traumatic factors,4 and have been reported following injury to the sympathetic nervous system, but this condition in the acquired state appears to be rare, although it may often be unrecognised.

Correspondence to: Mr Paul Byrne

2 Ogie JH. On the influence of the cervical portion of the sympathetic nerve and spinal cord upon the eye and it’s appendages, illustrated by clinical cases, with observations. Medico-chirurgie Provincialis 1850;41:397–440.

Shoulder pain from glomus tumour

Localised pain in the shoulder often suggests a brachial plexus neuropathy or cervical radiculopathy. Pain limited to a small area with sensory loss suggests a focal nerve lesion such as a neuroma. Glomus tumours rarely cause arm pain distally; they rarely occur proximally.

A 41 year old woman presented with pain in the right shoulder over a period of months. The pain was localized to the lateral suprascapular area. The tender area, which had been present for several years, was less than 1 cm in diameter and located lateral to the spine of the right scapula. A friendly ‘touche’ on the shoulder would cause an