LETTERS TO THE EDITOR

Brachial plexopathy after Botulinum toxin administration for cervical dystonia.

The use of Botulinum toxin (BT) for the treatment of cervical dystonia is considered safe and effective.^{1,2} The existence of two preparations on the market justifies reporting not only the positive results but also the possible adverse reactions so that the best one can be selected for clinical practice.

Brachial plexopathy was once reported following cervical injection of BT.³ This occurred with the USA preparation (Oculinum) and was never mentioned with the UK preparation (Dysport).

A 32 year old housewife complained of involuntary movements of the neck for one month. There was no family history of involuntary movements. At age 29, dizziness and vertigo had been treated for some weeks with triethylperazine and Cinnarizine.

Right laterotorsion and retropulsion of the head was accompanied by pain and contracture of the posterior cervical muscles. Neurological examination was otherwise normal. The severity of this cervical dystonia was scored as 9 according to the Tsui rating scale (maximum 25).⁴

Laboratory investigations, including cervical NMR, were negative.

The patient was treated according to our current protocol with BT (Dysport). Two points in the right sternomastoid and 3 points in each side of the posterior cervical region (trapezius and splenius capitis) were each injected with 100 U, total amount 800 U.

There was no clinical benefit on the following days. On the sixth day, however, she complained of increasing neck pain irradiating to the left upper limb. There were also tongue sores and a certain degree of lip oedema, which were only evaluated retrospectively. A second administration of BT was then considered. Two weeks after the first treatment she was injected with a total dose of 400 U of BT distributed between 4 points (two in each side) on the posterior cervical region.

Fifteen days after this second administration of BT, she complained of weakness of the right upper limb. Pain was still present on the neck and on the left arm. Neurological examination, at this time, revealed decreased muscular strength (3/5) of right supraspinatus, deltoid and biceps; the right biceps reflex was absent and brachioradialis reflex inverted; on the left side there were no signs of neurological dysfunction except for the pain as mentioned above. Electromyography on the right deltoid and biceps showed fibrillations and positive sharp waves at rest, and reduced interference pattern on maximal contraction with pathological polyphasic (normal amplitude and duration) potentials. Right brachial plexus stimulation evoked normal motor responses on the deltoid. Median nerves F waves were normal. Somato-

sensory evoked potentials (SEP) were normal for median and ulnar nerves bilaterally. SEP of the radial nerves had normal plexus evoked responses, absent cervical (C2) waves and cortical waves were present on both sides with central conduction times at normal limits.

These electrophysiological studies were consistent with C5-C6 upper trunk plexopathy exclusively sensory on the left side and sensory-motor on the right side. The abundance of fibrillations on the right deltoid and biceps suggested motor end-plates disturbances.

This clinical picture remained unaltered for 6 weeks, on analgesics, and then rapidly improved. At that time, neurological examination became normal as did the electromyogram.

The clinical syndrome and the electrodiagnostic data were consistent with the diagnosis of partial bilateral brachial plexopathy. The sequence of events following the use of BT favours the interpretation of a causal relationship. The underlying mechanism might be due to a direct effect of BT on brachial plexus, causing pain on the left side by a chemical irritative mechanism on sensory fibres and paresis on the right side by motor-end plate blocking, due to transport of the BT by motor fibres. Alternatively, it might be due to an immune mediated mechanism similar to that found in serogenic peripheral neuropathy, which involves sensory fibres on the left side and both sensory and motor fibres on the right one.

The sores of the oral mucosa add support to the immunological hypothesis, since this is the mechanism proposed for this mucosal reactions to other drugs. In our experience of 102 treated patients, sores of the oral mucosa were found in four.

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Multifocal astrocytoma masquerading as possible progressive supranuclear palsy

There exists a small literature on Parkinsonism due to brain tumours as recently reviewed in this journal.¹ We would like to report a case of Parkinsonism whose evolution and clinical picture suggested pro-

Figure 1 The MRI scan (TR 2800, TE 80) at the level of the bodies of the lateral ventricles demonstrates an extensive area of high signal intensity in the right cerebral hemisphere involving the deep white matter. On the left side there is high signal intensity in the cortex and adjacent white matter of the posterior frontal and anterior parietal zones. Other sections demonstrated involvement of the right high convexity cortex and the mid portion of the corpus callosum.

gressive supranuclear palsy, but which was due to a multifocal cerebral astrocytoma.

A 60 year old left handed man presented in March 1991, with a two month history of reduced dexterity of the left hand, associated with a tendency to drag the left foot, micrographia and softening of his voice. On examination he was cognitively preserved, softly spoken, with a positive glabella tap, mild tremor of the lightly closed eyelids, and cogwheeling rigidity of the left upper limb. There was no tremor of the limbs, and reflexes were generally brisk but symmetrical with downgoing plantars. Sensory examination was normal. Levodopa at 600 mg daily combined with carbidopa was ineffective and had to be reduced due to drowsiness. In view of the hyperreflexia and lack of response to levodopa preparations, a cranial CT scan was performed and was considered normal. When reviewed one month later, he was having frequent falls. On examination he had marked bradykinesia with nuchal rigidity. Ocular examination revealed normal downgaze but limitation of upgaze to 5-10° and the presence of excessive square wave jerks. There was bilateral cogwheel rigidity, with minimal left sided pyramidal weakness and asymmetrically brisk reflexes (left greater than right), with bilaterally downgoing plantars. A tentative diagnosis of possible progressive supranuclear palsy was made. In view of the rapid progression, however, a cranial MRI scan was performed (figs 1-3). This revealed a multifocal tumour (in retrospect this had been partially visible on the CT scan), which on stereotactic biopsy was shown to be a grade III multifocal cerebral astrocytoma. The patient died in June 1991, three months after initial presentation.

Progressive supranuclear palsy (PSP) is commonly misdiagnosed in the early stages, with 56% of cases in a recent series, having an incorrect initial diagnosis of Parkinson's disease.² Lack of response to levodopa preparations, and associated neuro-ophthal-

Figure 2

Figures 2 and 3 The MRI sections (TR 2,800, TE 80) at the level of the basal ganglia and cerebral peduncle respectively demonstrate high signal intensity in the right lentiform nucleus, the anterior limb of the internal capsule and some extension into the posterior limb of the internal capsule. High signal intensity is demonstrated in the right cerebral peduncle extending from the red nucleus anteriorly to the surface of the peduncle. The appearances are typical of an infiltrating multifocal tumour.

Figure 3

mological and pseudobulbar signs provide support for the clinical diagnosis of PSP. The presence of downgaze paresis is required for the diagnosis, but is not necessarily present in the early stages of the disease, so that at this stage other neurological findings such as hyperreflexia, extensor plantar responses and a more rapid clinical course may make one suspect PSP rather than idiopathic PD.³⁴ The limitation of upgaze was presumed to be due to bilateral involvement of the fronto-mesencephalic pathways for voluntary upgaze.⁵

This case showed evolution from a clinical picture compatible with PD through to one suggesting possible PSP, but with a suspiciously rapid clinical course. The MRI scan demonstrated a multifocal astrocytoma that masqueraded as PSP.

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Contralateral selective saccadic palsy after a small haematoma in the corona radiata adjacent to the genu of the internal capsule

Contralateral saccadic palsy with ipsilateral conjugate deviation of the eyes is usually attributed to lesions involving the frontal eye field (FEF) or connections with that area coursing through the internal capsule.¹² The lesions are usually so large that they cause obtundation and contralateral hemiparesis. Pathological confirmation of the lesions confined to the FEF or its connections has not been reported. We report a case of contralateral selective saccadic palsy with neither clouding of consciousness nor limb weakness after a very small haematoma in the corona radiata adjacent to the genu of the internal capsule.

A 50 year old woman developed acute dysarthria whilst in hospital for a broken leg. After defaecation, she had developed speech disturbance, immediately followed

by right facial drooling. Blood pressure was 160/100 mm Hg and pulse 90/minutes. On neurological examination, she was alert and cooperative. Her eyes deviated upwards and to the left but she was able to follow slowly moving targets in either horizontal direction. Optokinetic testing with targets moving to the patient's right evoked a normal nystagmus response but the eyes deviated tonically into an eccentric leftward position with the target moving to the left. She had minimal right lower facial weakness and mild paretic dysarthria. She complained of difficulty in swallowing liquids, and palatal and pharyngeal weakness was present on the right. There was no deviation of the tongue on protrusion. Facial and buccal sensation were normal. Motor, sensory and cerebellar functions were normal in the limbs except for the following signs of subtle corticospinal damage: when the fingers were stretched out voluntarily, abduction of the fifth finger (a *digiti quinti* sign of Alter³) and adduction and flexion of the first

CT scan shows a round hyperdensity in the left corona radiata adjacent to the genu of the internal capsule.

metacarpal (a hollow hand sign of Garcin⁴) were noted on the right side. Muscle strength of the legs was probably normal. Tendon reflexes were normal with flexor plantar responses.

A CT scan revealed a round hyperdensity, of approximately 7 mm diameter, located in the left corona radiata adjacent to the genu of the internal capsule, suggesting a small haematoma (fig).

Her oculomotor disorder disappeared within two weeks. Two months after the stroke, neurological examination was normal.

Our patient presented contralateral selective saccadic palsy and contralateral supranuclear facio-palato-pharyngeal paresis, but no weakness of the tongue and limbs.

According to recent anatomical studies in monkeys,⁵ the major pathway from the frontal eye field descends in or slightly anterior to the genu of the internal capsule near the caudate head. The very restricted lesion of our patient suggests that the descending pathway from the FEF in humans may pass through the genu of the internal capsule in parallel with the corticobulbar tract.

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