LETTERS TO THE EDITOR

Transient partial verbal amnesia

Transient partial verbal amnesia (TPVA) is transient amnesia characterised by selective impairment of verbal memory. TPVA is uncommon,1–3 and its existence is not generally accepted. We describe a patient with TPVA, on whom we performed detailed neuropsychological examinations during the attack. The aetiology is of interest, as the attack mimics the usual cause of permanent verbal amnesia.4,5

The patient was a 58-year-old right-handed man who had been a cook. Soon after he left home for work, he reported that his memory was somewhat abnormal. He could cook as usual, but repeatedly asked questions such as, “What did the customer order?” His colleagues sent him to our hospital. On examination, general physical and neurological examinations were unremarkable. He did not abuse alcohol or drugs. He showed no evidence of cardiovascular or cerebrovascular disorders. During the interview he repeated “I can remember your (doctor’s) face, but not your name.” He remembered that the radio was on in his restaurant, but had no memory of what the radio announcer said. His digit span was normal (six digits), but after a few minutes, he forgot the fact that he had repeated the digits. He showed slight retrograde amnesia, being unable to remember some of the things that had happened since the evening of the day before. His speech was fluent, Naming, writing, and reading were normal. No evidence of aphasia, acalculia, or apraxia was observed.

Routine laboratory studies including EEG were unremarkable. Brain CT and 123I-IMP single photon emission computed tomography (SPECT) revealed no abnormalities. All these studies were conducted during the attack. We observed him continuously after admission, and found that his amnesia disappeared approximately 10 hours later, when he then could remember verbal material. Brain MRI (T1 and T2 weighted images) conducted three weeks after the attack was unremarkable.

We conducted Wechsler memory scale—revised (WMS-R) and Raven’s standard progressive matrices (RSPM) tests during and two weeks after the attack (table). WMS-R and RSPM showed that his IQ was within the normal range during and after the attack. There was no clear discrepancy between verbal IQ and performance IQ. The results of WMS-R showed that the attention index was within normal limits during and after the attack. The general memory composite included verbal subtests and visual subtests. There was a definite discrepancy between verbal and visual memory index during the attack. After the attack, his verbal memory index increased remarkably; his visual memory index also showed a minor increase, which was probably caused by the fact that the tests were performed twice. The subtests contributing to delayed recall index can be separated into verbal and visual material (table). Although his delayed visual recall was normal during and after the attack, his delayed verbal recall was severely impaired during the attack and it normalised after the attack. Thus the results of this analysis revealed that his anotograde amnesia was limited to verbal material, and that delayed recall was severely impaired, whereas immediate recall was intact.

To prove the existence of TPVA, we believe that it is necessary to satisfy two kinds of criteria. One of them is the criterion of transient amnesia,4 and the other is that of verbal selectivity. We chose the following eight points as criteria for TPVA. (a) The attack onset should be witnessed. (b) Information should be available from a capable observer who was present for most of the attack. (c) There must be clear cut, anterograde amnesia during the attack. (d) There should be no other major neurological signs or symptoms except amnesia. (e) The memory loss should be transient, usually lasting hours or up to one day. (f) Epileptic features must be absent. (g) Patients with recent head injury or active epilepsy must be excluded. (h) Amnesia must be limited to verbal materials.

Three papers have reported TPVA.1–3 Two patients described by Okada et al had some degree of visual memory preservation in the course of transient global amnesia.1 One patient did not satisfy criterion (h), and the second did not satisfy criteria (a), (b), and (f). Damasio et al also reported a patient with transient impairment of verbal memory but relative preservation of orientation to place, familiarity with previously known persons, and partial insight into her actions during the episode.2 Their patient satisfied only criteria (c), (e), and (g). Matias-Guiu et al reported four patients with transient amnesia affecting verbal material.6 We were unable to ascertain whether their patients did not satisfy all the criteria, however, because they failed to give detailed information. On this basis, therefore, none of the patients described in previous reports provide adequate proof of the existence of TPVA.

In contrast, our patient showed a transient amnestic attack confined mainly to verbal material, which lasted for about 10 hours. We believe that our case fulfils all our criteria. The administration of detailed memory tests during the attack is important for the qualification and quantification of TPVA. The result of the neuropsychological examinations during the attack revealed that verbal memory impairment was severe, although the visual memory index was within the normal range. We believe that this is the first report of clear-cut TPVA.

Memory impairment in patients with bilateral temporal lobectomy is consistent with that of patients during an attack of transient global amnesia. In previous reports, SPECT showed a significant hypoperfusion in the bilateral temporal lobes or the left thalamus during this sort of attack.5,6 On the other hand, there are clinical similarities between the transient amnesia of our patient and the chronic verbal amnesia of patients with dominant temporal lobectomy,7 although no abnormality was detected by SPECT during the attack in our patient. Although this study can make no definite conclusions concerning the aetiology of TPVA, the present results are consistent with the idea that temporary dysfunction of the medial part of the dominant (usually left) temporal lobe causes TPVA.

Table Neuropsychological tests in transient partial verbal amnesia

<table>
<thead>
<tr>
<th></th>
<th>During the attack</th>
<th>Two weeks after the attack</th>
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<tbody>
<tr>
<td>RSPM</td>
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<td></td>
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<tr>
<td>IQ</td>
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<td>WAIS</td>
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<tr>
<td>VIQ</td>
<td>99</td>
<td>99</td>
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<tr>
<td>PIQ</td>
<td>115</td>
<td>113</td>
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<tr>
<td>F IQ</td>
<td>107</td>
<td>106</td>
</tr>
<tr>
<td>WMS-R</td>
<td>Attention index</td>
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<td>103</td>
<td>123</td>
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<td>General memory index</td>
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<tr>
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<tr>
<td>Visual memory index</td>
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<td>123</td>
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<tr>
<td>Delayed recall index</td>
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<td>123</td>
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<tr>
<td>Recall</td>
<td>Immediate</td>
<td>Delayed</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Verbal memory</td>
<td>8/50</td>
<td>5/00</td>
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<tr>
<td>Logic memory*</td>
<td>7/8</td>
<td>0/8</td>
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<tr>
<td>Visual memory</td>
<td>39/41</td>
<td>39/41</td>
</tr>
<tr>
<td>Visual reproduction*</td>
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<td>6/6</td>
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<tr>
<td>Visual PA†</td>
<td>6/6</td>
<td>4/6</td>
</tr>
</tbody>
</table>

*Correct score total score. †Correct responses/total responses for the last trial.

RSPM = Raven’s standard progressive matrices test; WAIS = Wechsler adult intelligence scale; WMS-R = Wechsler memory scale—revised; VIQ = verbal IQ; PIQ = performance IQ; F IQ = full IQ; PA = paired associates.


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Systemic effect of local botulinum toxin injections unmasks subclinical Lambert-Eaton myasthenic syndrome

In recent years botulinum toxin type A has been introduced as a new symptomatic therapy for focal dystonias. Generalised muscle weakness is reported after overdoses of botulinum toxin, but is a rare clinical phenomenon. However, botulinum toxin spreads to muscles distant from those injected, even in low doses, as shown by single fibre electromyography. There have been no reports of botulinum toxin in patients with a pre-existing alteration of neuromuscular transmission. In one of our patients suffering from blepharospasm local botulinum toxin injection uncovered a Lambert-Eaton myasthenic syndrome (LEMS).

A 63-year-old woman presented with a three year history of severe blepharospasm and mild oromandibular dystonia. A periorbital injection with a total dose of 12 nanograms of botulinum-toxin-A-complex (Porton Down, UK) had been performed in another hospital. After three days the blepharospasm had improved but a mild ptosis on the left eye, nausea, fatigue, and generalised muscle weakness had occurred, all of which resolved within eight weeks. Twelve weeks after the first injection, when we saw the patient for the first time, SFEMG of the right EDC-muscle showed no abnormality: fibre density 1-6, blockings 0%, MCD 22-2 μs (upper limits of our laboratory: fibre density 1-9, mean MCD 38 μs). There was no possible relation between the muscle weakness and the botulinum toxin injection. The weakness remained unclear; the low dose would not be expected to cause generalised muscle weakness.

The blepharospasm recurred 20 weeks after the first treatment with botulinum toxin and the patient requested a further injection. We used a smaller total dose of 8 ng botulinum toxin. After four days the muscles of the pelvic and shoulder girdles became weak and fatigued. The patient had difficulties in rising from a chair and climbing stairs. We found normal motor conduction velocities in the peroneal and tibial nerves and normal sensory conduction velocity in the sural nerve. EMG of the right EDC muscle showed no abnormalities. Laboratory findings of blood and CSF, including immunological parameters, were normal. Anti-AChR antibodies were absent. SFEMG from the right EDC muscle showed a slightly increased fibre density of 1-6, blockings 1% and an increased jitter (MCD 46-2 μs) (fig b). EMG from the thenar muscles after repetitive median nerve stimulation revealed a slight increment of 118% after 3/sec-stimulation and an increment of 229% after 50 stimuli with a frequency of 20/sec (fig a).

These findings were not compatible with a systemic effect of a low dose of botulinum toxin, but suggested an additional presynaptic disturbance of the neuromuscular junction, particularly Lambert-Eaton myasthenic syndrome (LEMS). An intensive search for tumour formation was performed. X-ray of the chest was normal, but cytology of the bronchoscopic lavage showed malignant cells. CT of the lung showed a circular lesion in the upper-lobe of the left lung. Thoracotomy was performed and the left upper lobe was removed. The histopathological examination demonstrated an adenocarcinoma of the upper lobe of the left lung (UICC-Stage 1; pT1, pN1, M0). The weakness had disappeared completely three months after removal of the tumour.

Botulinum toxin inhibits the release of acetylcholine from the presynaptic site of the motor nerve terminal. After overdoses of botulinum toxin, development of generalised muscular weakness was reported. In the majority of studies and in our own series of more than 2000 local injections, no patient experienced weakness of remote muscles. However, SFEMG can detect botulinum toxin induced disturbances of neuromuscular transmission in muscles distant from those injected before weakness is clinically evident. In our case both low-dose botulinum toxin injections coincided with a period of transient pelvic-girdle muscle weakness. If the weakness had been induced by the systemic botulinum toxin effect only, we would expect an involvement of more distal muscles. SFEMG findings of an increased jitter were not specific, but consistent with a botulinum toxin induced disturbance of neuromuscular transmission. However, electromyographic findings of a marked incremental response to high-rate repetitive stimulation could not be explained as an effect of the small botulinum toxin dose and it indicated an additional disturbance of neuromuscular transmission. Incremental responses are well known in systemic botulism, but they are not reported in cases who received only therapeutic doses of botulinum toxin. Lambert-Eaton myasthenic syndrome (LEMS) was therefore suspected and a malignant lung tumour was found.

LEMS is an acquired autoimmune disorder of neuromuscular transmission. Secondary to the IgG-induced blockade of voltage dependent calcium channels at the presynaptic site, quantal release of acetylcholine is reduced. Electromyographic findings confirm the clinical diagnosis by an increased compound muscle action potential amplitude during high-frequency repetitive nerve stimulation or following brief exercise. In single-fibre studies, increased jitter and blocking are found, which improve with high rates of stimulation and are worse after rest. In our case the LEMS had been asymptomatic before the botulinum toxin injections. The additional and reversible effect of botulinum toxin on the neuromuscular transmission was found.

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**Figure** EMG from the thenar after repetitive median nerve stimulation; 50 stimuli, f = 20/sec. (a) and SFEMG from the right EDC muscle (b).
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