with compressed ventricles, confirmed the diagnosis. In this CT scan the basal cisterns were not compressed. Recently this sign was described as a predictor of outcome in severe head injury. The second CT scan when the patient recovered was normal. From the radiological point of view, the entity of acute water overload should be included among the differential diagnosis of small lateral brain ventricles.

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References


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Cortical vasoactive intestinal peptide in relation to dementia in Parkinson’s disease

Sir: Accumulating evidence has suggested analogies in the dementia of Parkinson’s and Alzheimer’s diseases. At least one third of the Parkinsonian population presents signs of intellectual deterioration. Histopathological stigmata associated with Alzheimer’s disease have been also observed at necropsy in brains from demented Parkinsonian patients. Biochemical investigations on post-mortem material have revealed a decrease in choline acetyl-transferase (CAT) activity as well as in somatostatin concentrations in the cerebral cortex of Alzheimer”” and demented Parkinsonian patients.”” The biochemical comparison between the two diseases was further extended to vasoactive intestinal peptide (VIP), a neuropeptide highly concentrated in the human cerebral cortex. Since cortical content of VIP was reported to be unaffected in brains of patients with Alzheimer’s disease,”” it appeared of interest to determine VIP levels in the frontal cortex of Parkinsonian subjects with and without dementia.

Brains from 24 control subjects with no evidence of neurological or psychiatric disease (mean age 73-2 ± 2-5 years, necropsy delay 12-3 ± 1-2 hours) and 23 Parkinsonian patients (mean age 72-6 ± 1-6 years, necropsy delay 14-8 ± 1-6 hours) were examined. Fourteen Parkinsonian patients were affected with intellectual deterioration diagnosed as previously reported.6 Tissue collection and dissection have been described elsewhere.7 The concentrations of VIP-like immunoreactivity (VIP-LI) were measured by means of a sensitive double-antibody radioimmunoassay method.11 The VIP antiserum used for this study recognized the region 15-28 of the VIP molecule. CAT activity was estimated by radioenzymatic assay as previously described.8

As expected,12 CAT activity was lower in the frontal cortex of Parkinsonian subjects when compared with controls (table). This increase probably reflected degeneration of the cholinergic pathway originating in the substantia innominata and projecting into frontal cortex. The deficit was greater in Parkinsonians with dementia suggesting that the lesion of the subcortico-cortical cholinergic system was implicated in the process of intellectual deterioration.7 Cortical VIP-LI concentrations were not significantly different from controls in Parkinsonian patients (table). No further significant difference was found when Parkinsonians were subdivided in nondemented or demented patients. These data suggest that neurons containing VIP in the frontal cortex are neither affected in Parkinsonian patients nor involved in dementing process in Parkinson’s disease, as previously reported for Alzheimer’s type dementia.10

These observations emphasize the biochemical similarities between Alzheimer’s and Parkinson’s types of dementia as far as cortical neurotransmitter systems are considered. Besides VIP other neuropeptides (CCK-8, Substance P, enkephalins) have been found in normal concentrations in brains from patients with both types of dementia.”” These results contrast with the damage of intra-cortical neurons, such as somatostatin containing neurons8,6 and subcortico-cortical (cholinergic, adrenergic) neuronal systems”” which are probably implicated in the dementing process for the two diseases.

Table VIP-LI and CAT activity in control and Parkinsonian frontal cortex (Brodmann area 9)

<table>
<thead>
<tr>
<th>CAT activity (nmol/h/mg protein)</th>
<th>VIP-LI</th>
<th>Control</th>
<th>Parkinson’s disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>all cases</td>
<td>non-demented</td>
<td>demented</td>
<td></td>
</tr>
<tr>
<td>5-98 ± 0.28</td>
<td>3-00 ± 0.28*</td>
<td>4-81 ± 0.39*</td>
<td></td>
</tr>
<tr>
<td>(12)</td>
<td>(20)</td>
<td>(7)</td>
<td></td>
</tr>
<tr>
<td>182-9 ± 9.94</td>
<td>176-5 ± 14.80</td>
<td>189-5 ± 29.99</td>
<td></td>
</tr>
<tr>
<td>(24)</td>
<td>(23)</td>
<td>(9)</td>
<td></td>
</tr>
</tbody>
</table>

Values are the means ± SEM; the number of brains is indicated in parentheses. *Significantly different from control (student’s t test; p ≤ 0.05).

References


Acute myelopathy in a drug abuser following an attempted neck vein injection

Sir: Numerous neurologic complications have been described in association with intravenous drug abuse.1 Myelopathy was initially described in four patients by Richter and Rosenberg in 1968.2 Since then, several more cases have been reported.3–6 Our patient is unique in that the complication ensued within minutes after the injection, from a site quite close to the affected segments. Also, most cases of myelopathy reported in literature have occurred in association with heroin abuse and we are not aware of similar complications reported in association with methylenphenidate (Ritalin) or pentazocine (Talwin) abuse.

A 37-year-old black male who was known to have been a drug abuser of Ritalin and Talwin for nearly ten years was attempting to shoot up a crude tap water suspension of crushed tablets into one of his left sided neck veins when the needle inadvertently struck a spot and caused an acute severe tingling and numbness along his left side. In the next twenty minutes he developed a severe left sided weakness ascending from the toes to involve the whole arm. He could neither walk nor stand, and also experienced a strange feeling on the right side. The needle was withdrawn only after the contents were injected. There was no history to indicate that the patient had stopped using drugs prior to this episode. The next morning, 12 hours after the onset of symptoms he was brought to the hospital. He was lucid, afebrile, normotensive and without distress though confined to bed. Generalised scars of the skin along veins, confirmed a long term intravenous drug abuse. The group of lymph nodes in the posterior triangle of the neck were slightly enlarged and moderately tender on both sides, suggesting chronic inflammation, but there did not seem to be an acute suppurrative process. The rest of the general physical examination was unremarkable.

On neurologic evaluation, mental status was normal though conforming to a personality disorder. All cranial nerve functions were intact. Hypotonia was noted on the left side, where extremities could be barely lifted against gravity. On the right side, the arm and leg could only be moved against minimal resistance. A definite upper sensory level was obtained bilaterally for pain and temperature at C4-C5 segments but vibration, position and touch sensations were preserved. Tendon reflexes were bilaterally sluggish without asymetry and plantar responses were absent. The sphincter tone was flaccid. Signs of meningeal irritation and deformity or tenderness of the spine were absent.

A myelogram obtained soon after admission showed no spinal blockage or cord compression although there was a suggestion of swelling of the cord and nerve roots bilaterally at C4-C5 segmental levels. Spinal radiographs did not reveal osteomyelitis or other abnormality. Spinal fluid was colourless with a cell count of 100 WBC per ml (74% polymorphs, 26% lymphs) and 400 RBC per ml, protein was 37 mg/dl, glucose 57 mg/dl and Gram stain showed no organism. Blood, urine and spinal fluid cultures showed no growth. Serum and spinal fluid VDRL was non-reactive. Other investigations that included chest film, ECG, blood counts, electrolytes, liver function tests and urinalysis were normal. The patient, however, refused to have further investigations for possible vasculitis.

Therapy included a three day course of broad spectrum antibiotic, and a short five day course of corticosteroid and physical therapy. A significant improvement was noted from second day onwards. At the end of 2 weeks, he was able to walk unaided, dragging his left leg. Muscles innervated by C3-C7 segments, however, continued to be weak, bilaterally. Shoulder abduction and external rotation could not be performed against moderate resistance and elbow flexion was weak against full resistance. In the lower extremities, only a minimal weakness of left foot dorsiflexors was noted. There was mild increase in tone of all extremities and tendon jerks were bilaterally hyperactive associated with pathological features such as finger flexion in the upper extremities and crossed adductor response in the lower extremities. Plantar responses were equivocal on both sides while the sensory findings remained unchanged.

In most reported cases, the causal relationship of myelopathy to intravenous drug abuse has only been speculative. The proposed mechanisms of pathogenesis are multiple and may differ in individual cases. Infection including those of rare Gram negative organisms5 is a frequent complication of intravenous drug abuse. The incidence of spinal osteomyelitis and epidural abscess is much higher in this population and if untreated may result in myelopathy.6,7 Another known complication in this group is necrotising vasculitis8 which can affect arteries of multiple systems. At least in one patient myelopathy resulted from spinal vasculitis and was proved by biopsy.9 Since in some patients myelopathy occurred after resuming the drug abuse following a period of abstinence, a possible immunologic or hypersensitivity reaction affecting the spinal cord has been suggested, although not proved.10 Ischaemic myelopathy was suggested in some patients who had hypotension11 and developed myelopathy at segmental levels most vulnerable to ischaemia. Other proposed mechanisms include particulate

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