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Nine patients with TSP who were born in the Caribbean were compared with an age and sex matched group of European white patients with clinically definite MS, all of whom had a progressive spastic paraparesis. Disability was scored using the Kurtzke Disability Status Scale. The patients with TSP were anti-HTLV1 positive and had HTLV-1 genome integrated into leukocyte DNA. Eight were female. The mean age was 53 years (range 13-65 years), the mean symptom duration was 12 years (range 1-5-23 years), and the mean Kurtzke disability score was seven (range five to eight). The mean age of the MS patients was 42 years (range 35 to 53 years), the mean symptom duration was 11 years (range seven to 17 years), and the mean Kurtzke disability score was five (range four to six). The spine was imaged by a Picker 0.5T superconducting machine with T1 weighted (SE5000, 5 mm contiguous parasagittal slices) using a surface coil. All MS patients and five TSP patients had additional T2-weighted sequences (SE2000, 5 mm contiguous parasagittal slices) to detect abnormal signal. Images were reported without knowledge of the individual diagnosis by one of the authors (EPGH du B).

Atrophy of the thoracic cord was seen in six of nine patients with TSP and five of nine patients with MS. Three of five patients with TSP who had T2-weighted images of thoracic cord had diffuse high signal and all three had atrophy (fig). Five of nine with MS had high signal return on T2 weighted images, one of whom did not have atrophy. The pattern of high signal was diffuse in two and focal or patchy in three (fig).

These results confirm the previous MRI finding of atrophy in the thoracic cord in a proportion of patients with TSP. However, a similar degree of atrophy is seen as frequently in patients with MS who had a progressive spastic paraparesis, a finding compatible with pathological studies where cord atrophy is present in 72% of patients with MS at necropsy. There was some difference in the pattern of high signal seen in the two groups with more diffuse and uniform high signal in TSP and focal or patchy high signal in MS. However, these differences in the MRI findings are slight and a reliable distinction between the two conditions cannot be made on these grounds.

Lewy bodies and subacute sclerosing panencephalitis

The occurrence of Lewy bodies in the nervous system is relatively specific to Parkinson's disease. Their association with other disorders may provide a clue to the aetiology of Parkinson's disease, especially when the cause of these disorders is known. We describe Lewy bodies in two patients with subacute sclerosing panencephalitis (SSPE). They are examples of long survival and the first has been reported for this reason.

A 14 year old boy presented with intellectual deterioration and absence attacks. When seen at the National Hospital he had generalised epileptic seizures, multifocal myoclonus, emotional lability, dysarthria, mild chorea and ataxia. Serum measles virus titre was elevated. An EEG showed periodic complexes and cerebrospinal fluid (CSF) showed a parietal Lange curve. His condition stabilized, but at the age of 21 years he deteriorated again. Six years later he was bed-bound, and died of bronchopneumonia.

The brain showed severe atrophy associated with widespread neuronal and myelin loss, gliosis, and occasional neurofibrillary tangles. Very few nerve cells remained in the substantia nigra with a couple of Lewy bodies present in each unilaterial section. Lewy bodies were also present in the locus minoris...
coeruleus, but not in the dorsal vagal nucleus, nucleus basalis, cerebral cortex, spinal cord, superior cervical sympathetic ganglia, and cardiac ganglia.

The second case was a nine year old boy presenting with intellectual deterioration and an epileptic seizure. A month later, when admitted to Hôpital Ste-Justine, Montreal, his parents noted that a rare encephalitis, his affect was inappropriate and there was mild orofacial chorea. The left planter was extensor and his gait ataxic. He developed generalised myoclonus with occasional opisthotonus, and an autonomic disturbance involving. Serum measles virus titre was 1024. CSF protein was 0.54 g/l, with no cells, and the measles complement fixation titre was 128. An EGG was dominated by low voltage slowing and periodic high voltage slow-wave complexes.

His condition stabilised, but he remained bed-bound and died at the age of 19 years. The brain showed generalised atrophy, with profound neuronal loss and gliosis in the cerebral cortex and hippocampus with a few microglial nodules and tangles. Some remaining neurons showed intranuclear inclusions. All central grey nuclei showed severe neuronal loss with glial and microglial reactions and several tangles. There was severe neuronal loss in the substantia nigra and locus coeruleus with tangles and Lewy bodies (fig). Lewy bodies were also found in the dorsal vagal nucleus, cerebral cortex, spinal cord or autonomic nervous system.

These patients with SSPE showed long survival of 13 and 10 years respectively. Tangles occur even in patients dying at a young age,2 but Lewy bodies have not been described in other cases. They were present in the substantia nigra and locus coeruleus, but not in other areas usually susceptible to Lewy bodies. We know of a third case of SSPE with onset at 21 years, and a nine year survival. In this case there were very few nerve cells in the substantia nigra, and some pale bodies, which are normally seen in Lewy body diseases.4

Apart from the Lewy body–Parkinson's disease spectrum Lewy bodies are confined to a small group of rare degenerative disorders.1 They may be by-products of a degenerative process, rather than of degeneration, thus explaining their occurrence in long-standing cases of SSPE showing periods of relative stability. SSPE is a destructive inflammatory process due to persistent measles infection, but the aberrant immunological mechanisms are not fully understood. Although there is no direct evidence for an infectious aetiology for Parkinson's disease, there are parallels with SSPE which results from an infectious agent acquired in early life, leading to a progressive disorder associated with disconnection in identical twins, and Lewy bodies pathologically.

**Combined neuroleptic malignant syndrome and the central anticholinergic syndrome**

The neuroleptic malignant syndrome (NMS) is an idiosyncratic reaction to neuroleptic drugs characterised by encephalopathy, rigidity, dysautonomia and hyperthermia.1 Dopamine receptor blockade appears central to both the parkinsonian rigidity,2 and possibly the encephalopathy.3 Because cholinergic receptor blockade can also cause encephalopathy, the differential diagnosis of NMS includes the central anticholinergic syndrome (CAS).5 Anticholinergic drugs, anticholinergic, which are used to treat Parkinsonism associated with neuroleptic use, are often used to treat the rigidity in NMS.6 However, anticholinergic could theoretically exacerbate the encephalopathy of NMS, possibly sparing the development of combined NMS and CAS. We report the first case of combined NMS and CAS.

After four months of lithium therapy, a 28 year old man developed a parkinsonian disorder was given 15 mg of daily haloperidol. One week later he was brought to the emergency room confused, agitated and hallucinated. Neurological examination disclosed a fever of 101°F, cogwheel rigidity, resting tremor, and dysmetria. Serum glutamic oxaloacetic transaminase 128 IU/I (normal 5–35), serum glutamic pyruvate transaminase 65 IU/I (normal 5–30), lactic dehydrogenase 373 IU/I (normal 90–220) were all increased in concentration. His creatinine phosphokinase was 7700 IU/I (normal 15–245). All other laboratory studies, including computed tomography, cranial and lumbar puncture were normal. The haloperidol and lithium were stopped.

Treatment included levodopa/carbidopa 25/200, and 4 mg of intramuscular benztrapine every two hours. Several hours after receiving the benztrapine, his mental status deteriorated from being agitated and confused to comatose, and his temperature increased from 101°F to 104°F. The rigidity was unchanged, but he developed urinary retention, decreased bowel sounds and large pupils, suggesting anticholinergic toxicity. Six mg of intravenous physostigmine was administered. Within minutes he was able to follow simple commands. His temperature decreased from 102°F to 99°F over the next hour.

The differential diagnosis of NMS includes CAS. In a recent review, Guze and Baxter state that a response to physostigmine is sufficient to differentiate between NMS and CAS.1 However, this case suggests that the two disorders can coexist in the same patient. Furthermore, since drugs that cause NMS have anticholinergic properties, cholinergic blockade by neuroleptic drugs may make patients more susceptible to CAS from even low doses of anticholinergic agents.

Two points are illustrated by this unusual case: First, anticholinergic agents, which do not have demonstrable efficacy in NMS are probably contraindicated in NMS. Second, patients with NMS should be carefully monitored for signs of anticholinergic toxicity.

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Intraoperative aneurysms rupture during the pre-dissection stage

Much has been written about intracranial aneurysms which rupture as they are being dissected, probably because of the frequency of this event (24%, in one well known series).3 Less has been written about rupture of aneurysms during the pre-rupture phase, after the craniotomy is started and before the aneurysm is exposed, the pre-dissection stage, discussed by Batjer and Samson. Fortunately, this is uncommon. (Yasargil 1964; Charles G Drake)

Batjer and Samson encountered it four times in a series of 307 consecutive aneurysm operations and questioned whether the operations should have been aborted. I report an experience of rupture of an aneurysm before dissection.

A 59 year old right handed white woman experienced the acute onset of severe headache 48 hours before admission. She had no neurological symptoms and the only abnormal finding was neck stiffness. Computed tomography (CT) of the head was normal but lumbar puncture disclosed grossly bloody fluid under an opening pressure of 210 cm H2O. Left carotid angiogram showed an anterior communicating artery aneurysm 3 mm in diameter pointing forward and down. During opening the patient was given 50 cc of 20% mannitol, but spinal fluid was not drained. A slow drip of nitroprusside was started. As the chiasmatic cistern was opened, and before the aneurysm was displayed, the brain herniated massively into the craniotomy opening. A hand held brain retractor was left in place to allow escape of blood. At the time of rupture the patient's blood pressure was 100/80 mm Hg. Fingertips to 40/60 mm Hg. Immediately after rupture the patient was given 40 mg of nitroprusside and 20 mg of dexamethasone intravenously. Five minutes after rupture nitroprusside was stopped and a nitroglycerin drip (40 mg in 50 cc of 5% dextrose and water) was started. Within two minutes, that is seven minutes after rupture, the brain suddenly returned to its pre-rupture position and the BP fell to 60/40 mm Hg. Frontal lobe amputation, which had removed only 5 cc of brain, was terminated and the BP was allowed to rise slowly over the next 45 minutes. No further attempt to...
thiamazole 30 mg daily improved all the symptoms, including meralgia paresthetica.

A year later, the left lateral cutaneous nerve of the thigh sensory conduction velocity had improved to 58.8 m/s with normal amplitude (3.6 µV).

To discover the frequency of localised sensory disturbances in hyperthyroidism, we studied clinically 20 patients with Graves' disease, including these two cases (101, 102-stry thyroid patients, 10 euthyroid, after treatment). Symptoms consistent with mono-neuropathy such as localised sensory disturbance or muscle weakness, were present in nine (45%). These symptoms were dysesthesia, paraesthesia or hypoaesthesia on fingers in five patients, hypoaesthesia or dysesthesia on the lateral aspects of the thigh in seven patients, and bilateral foot drop in one patient (case 1). Tinel's sign of median nerve was present in seven of 10 hyperthyroid patients and five of 10 euthyroid patients. A positive Tinel's sign was found in 60% of thyrotoxic patients but also in 14.5% of 282 normal controls (Chi-square = 23.6, p < 0.0001). Our two cases demonstrated a combination of mononeuropathy and thyrotoxicosis. These mononeuropathies were confirmed by nerve conduction studies and improved following treatment for thyrotoxicosis. In addition, the denervation findings and low amplitude evoked responses without conduction block of the peroneal nerve in case 1 suggest mono-axonopathy associated with thyrotoxicosis. The dissociation between complete foot drop and no conduction block of the peroneal nerve supports this possibility.

In euthyroid patients, the frequency of both the symptoms and Tinel's sign diminished with the duration of treatment. In a detailed electrophysiological study of patients with thyrotoxicosis, loss of functioning motor units with normal conduction velocities demonstrated motor neuron dysfunction and the remarkable capacity of motor neurons to resume normal function. Individual nerves are more sensitive to mechanical damage if a generalised peripheral neuropathy is present. It seems likely to us that the fragility of nerve axons associated with hyperthyroidism predisposes to mononeuropathies.

Correction:
This letter was printed in the August issue with only one MRI image.

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Figure The MRI on the left shows diffuse high signal with atrophy of the thoracic cord in TSP. The right shows the patchy high signal typically seen in MS. (SE2000, 5 mm sagittal slices).
entrapment of blood vessels around the circle of Willis. The histopathological findings in these vessels usually include fibrosis of the media and endothelial hyperplasia and occasionally complete occlusion.

As in the series of Anzalone and Landi, a past history of arterial hypertension was uncommon in our series.

The lacunar syndromes in the present series included sensorimotor syndrome in five patients, pure motor hemiparesis in four and in three patients ataxic-hemiparesis. Two cases had cysticerci in brain parenchyma, suprasellar in five, one interpeduncular, one insular, one meningeal and diffuse arachnoiditis in two cases.

Cerebral angiography was performed in five patients, and evidence of vasculitis was found in three patients.

Cystercerosis is not usually considered in the differential diagnosis of lacunar syndromes, this becomes important in areas of the world where cystercerosis is endemic.

We agree with Anzalone and Landi with the concept that early CT scanning must be performed in patients with lacunar syndrome, particularly if the patient is young, normotensive or resident in countries where neurocystercerosis is a frequent health problem.

In the latter we would strongly recommend the routine examination of cerebrospinal fluid including immune reactions for cystercciosis.

FERNANDO BARRAGARREMENTERIA

MATTERS ARISING

Non-vascular aetiology of lacunar syndromes

We have read with interest the paper by Anzalone and Landi on non-isaemic causes of lacunar syndromes. From 31 August 1985 to 31 October 1989, we studied prospectively our patients from the stroke clinic that included several cases of lacunar infarctions and lacunar syndromes. We have found several cases of lacunar syndromes due to non-vascular aetiology. The main cause of lacunar syndrome in our patients was neurocysterciosis.

Some of these cases have been reported elsewhere. Our series include 12 patients aged 18 to 57 years, mean 34 5 years. During the last four years 733 patients attended our stroke clinic and from these we found 114 cases with lacunar syndrome. The twelve cases due to neurocysterciosis are 8 3% of lacunar syndromes.

In two patients the lacunar syndrome (atatic hemiparesis in both) were produced by parenchymal brain cysterciosis, in the remaining patients the lacunar syndrome was produced by cerebral infarction (lacunar infarction). In most cases it was located in the capsular area and in only two patients was the lesion located in the subcortical area. These infarctions were associated with subarachnoid cysterci. It has been well recognised that subarachnoid cysterci usually induced subacute, chronic or recurrent menigitis with abnormal thickening of leptomeninges at the base of the skull and inflammatory

As prosopoeia. This suggests dysprosopoeia, altering the equally simple adjectic dys- prosopoe. Where Sigmund Freud has trod, we surely dare to go.

PETER EAMES

BOOK REVIEWS

Therapy of Parkinson's Disease. Neurologic Disease and Therapy Series 5). Edited by WC KOLLER and G PAULSON (PP 583 Illustrated; Price $125.00 (US and Canada), $130.00 (All other countries). New York: Marcel Dekker Inc. 1990. ISBN 0-8247-2189-4

Having enjoyed reading and commending Koller's Handbook of Parkinson's Disease last year, I looked forward to learning more about therapy from this new book edited jointly with Paulson. Their preface reminds us, perhaps a little unkindly, that few of the contributing authors are old enough to remember patients with end-stage Parkinson's disease "who lay in sawdust"...or, those given levodopa in whom we "witnessed dramatic changes as patients in Stages III or IV became completely functional". Very true, but I am not sure we would all agree with Koller's introductory remark that "the types of therapy have dramatically increased..."...due in large part to the discovery of MPTP model of parkinsonism." The contents show 69 contributors in serried ranks, amongst whom are included almost all of the USA doyens as well as a selection of Europeans.

The five main sections cover Assessment and measurement of symptoms and signs, Pharmacologic agents, Surgical approaches, Diet in therapy, and Other therapeutic approaches.

The result is a very useful and comprehensive survey of treatment which is up-to-date, well referenced and authoritative. Modern techniques range from dopaminergic, a re- birth of stereotactic thalamotomies, levodopa infusions and, to be right up to the minute the DATATOP trial of selegiline and tocopherol; all are reviewed in detail.

There are however faults. In a fact laden text, good writing and thereby easy reading are just as important in a more discursive series of essays. And here the book dis-appoints, despite the publisher's attractive printing format illuminated by clear illustrations and diagrams. Many contributors have careened the edifice of rational, reflective prose in favour of an almost obsessive devo-