Peripheral neuropathy as a complication of neuroleptic malignant syndrome

Neuroleptic malignant syndrome (NMS), an idiosyncratic reaction to treatment with neuroleptic drugs, is characterised by diffusely increased muscle tone, altered consciousness, hyperthermia and instability of the autonomic nervous system. Only three cases of polyneuropathy in patients with NMS are known, one with unequivocal demyelination and two with no accurately classifiable neuropathy. We report a patient with NMS complicated by peripheral neuropathy. A 35-year-old woman presented with severe rigidity and a temperature of 42.5°C after four weeks of treatment for hypomania with haloperidol and procyclidine and later, because of disabling Parkinsonian symptoms, sulpiride. On admission to the intensive care unit she was alert and cooperative but unable to move, phonate, swallow saliva or cough. Her mental state was clear but after recovery there was a marked Parkinsonian tremor, reflexes were brisk and symmetrical. Apart from a well-demarcated decubital ulcer on the right heel examination revealed no obvious focus of infection (E. coli was grown from urine culture and staphylococcus aureus from the heel swab; the white cell count was elevated to 13.5 × 10^9, but blood cultures were negative). The creatinine kinase was elevated to 600 IU/l; other investigations including arterial blood gases, chest radiograph and routine biochemical screen were unremarkable.

Although treatment with broad spectrum antibiotics, bromocriptine and dantrolene led to some improvement in muscle tone, her pyrexia only resolved when she was paralysed and ventilated. Pneumonia five days after admission. When paralysis was reversed 48 hours later pyrexia and rigidity recurred, but then the patient displayed active resistance and catatonic posturing instead of Parkinsonian rigidity. This responded well to a series of three electroconvulsive shocks and two weeks after admission she was well enough to be transferred to a medical ward.

Her mental state and muscle tone were now normal, she was able to speak a few words and she soon managed to swallow liquids. All her muscles ached, she was unable to lift her limbs from the bed and repetitive movements were painful. There was generalised wasting of all muscle groups and tendon reflexes were difficult to elicit. The power in her limbs slowly improved, but four weeks after admission she was still too weak to stand. After six weeks she complained of pain in her right foot. Examination showed footdrop with loss of the ankle jerk and sensation to pinprick in a stocking like distribution and a few days later similar symptoms occurred on the left side. Her weakness gradually resolved, but the paraesthesiae in the right foot as well as the footdrop persisted. Fifteen weeks after admission she was independently mobile and discharged home.

Neurophysiological studies were first performed 14 weeks after admission. Muscle compound potentials from the small foot muscles were absent or very reduced in amplitude (0.4 mV). Motor conduction velocity was normal where measurable (tibial nerve 45 m/s). Sensory nerve action potentials were normal or of reduced amplitude (3 μv). Sensory nerve conduction velocity was within normal limits (sural nerve 36 m/s). Fibillations and positive sharp waves were present. The patient was critically ill, with pyrexia and septicaemia, she did not develop multiple organ failure, was only ventilated for three days and weaning was not required. Coincidental medical problems were excluded and there was no exposure to toxins or drugs known to produce neuropathy. As in the described cases of critical illness polyneuropathy, a toxic factor active during the acute phase of the illness is the most likely aetiological factor. In view of the differences discussed above, this may be specific to the neuroleptic syndrome itself, but other aetiological factors such as persistent pyrexia, septicaemia or an unidentified toxin cannot entirely be excluded. Further cases are needed to establish whether polyneuropathy is indeed a specific complication of NMS or just a non-specific manifestation of critical illness.

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References


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Epileptic attack, delirium, and periodic complexes in the EEG during mianserin treatment

Tricyclic and newer antidepressants have certain undesirable effects, including an increased susceptibility to delirium, myoclonic jerks, and epileptic convulsions. Two patients had an epileptic attack during mianserin treatment followed by delirium and EEG changes presenting as slow activity with periodic complexes similar to those seen in the Creutzfeldt-Jakob disease. A 61 year old male had suffered from paranoid schizophrenia since 1955. The patient was admitted to hospital due to increased psychotic symptoms in August 1987. On admission, medication previously used (promazine 200 mg and mianserin 60 mg in the evening) were continued and chlorpromazine 100 mg, three times a day, was introduced. He developed acute left-sided hemiplegia five days after admission. CT of the head showed central atrophy and a new right parietal infarction. Five days after the stroke the temporarily discontinued neuroleptic and antidepressant medications were reintroduced due to nocturnal delirium and a continuation of psychotic symptoms during the daytime. Two weeks later he had numerous grand mal attacks associated with periodic slow complexes similar to that seen in Creutzfeldt-Jakob disease (CJD) on EEG. Both symptoms subsided after the introduction of carbamazepine and discontinuation of mianserin. The patient recovered from the hemiplegia and delirium, and during a follow up period of 18 months, no progressive cognitive deterioration or new epileptic attacks were observed.

A 68 year old female with mild depressive symptoms treated with a low doxipine dose (35 mg/day) developed a major depressive episode during the summer of 1989 and was admitted to a psychiatric hospital. At admission she was extremely depressed (Hamilton depression rating scale score 31), but showed no cognitive deterioration in the Mini-Mental State Examination (MMSE score 24), and her EEG was normal. Doxipine treatment was discontinued and mianserin was introduced successfully, reaching 90 mg on the evening, resulting in a therapeutic mianserin concentration of 35 nanomoles/litre (therapeutic level 200–450 nanomoles/litre). Two weeks after admission she had an epileptic attack, after which she showed cognitive impairment (MMSE score 8), had myoclonic jerks, and met the DSM-III-R criteria for delirium during the next six days. Mianserin treatment was discontinued. Since her second EEG showed generalised slowing with periodic complexes similar to those seen in CJD, she was transferred to the Department of Neurology where laboratory results including CSF and CT of the head were normal. After the delirious episode EEG was normal, cognitive functioning restored (MMSE 25), and the myoclonic jerks disappeared. DWI was normal. The study was approved by the ethics committee of the hospital.

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BOOK REVIEWS


This multi-author text, with 68 contributors, covers the entire spectrum of clinical MRI. Like many other radiological texts it is really too large and heavy. Our copy is already showing signs of sagging. At least we have been allowed to publish the first 12 chapters as a separate volume.

The initial sections cover the physical basis and technical aspects of magnetic resonance imaging. These are based on the excellent monthly course organised by John Hesselink and Robert Mattray at the M.R. Institute in San Diego. They are readable, extremely well illustrated and, above all, understandable. Included is a chapter on clinical spectroscopy, which might better have been called "Spectroscopy for the Uninitiated" and which left us with a better understanding of its potential future clinical role.

Images in the brain and spine sections are excellent. Some illustrations in the body section are rather disappointing and are not "state of the art". This is a problem with all yearbooks as authors are always given date before publication. The editors have recognised this and include future development sections in many chapters. The musculo-skeletal chapters are superbly illustrated and written.

In some areas the clinical emphasis will seem strange to a British readership. For example, two pages are devoted to spinal cord tumours, 27 to examination of the testes and a single number to tempo-mandibular joint dysfunction.

As a neuro-MR reference work this adds little to the much smaller "MRI of the CNS" by M. Brant-Zawadski and D. Norman. The chapter on normal neuro-anatomy cannot compare with "Cranial and Spinal MRI" by Daniels, Haughton and Naidich. The compact "Clinical MRI" by V. M. Runge is more popular in the UK. Nevertheless, this authoritative general reference work on clinical MRI is excellent value at £118.

R.J. BARTLETT
A.A. NICHOLSON


This book is the proceedings of one of the meetings to be held regularly in the UCLA Forum in Medical Sciences. The subject of the meeting and the title of the book reflect increasing interest in the morphological and physiological substrates of higher cognitive functions. The availability of modern imaging techniques, including MRI, SPECT and PET together with more precise quantitative morphological measurements, neuropsychological assessments and molecular biology have considerably contributed to the understanding of the relationship between cerebral structures and higher cognitive function. The application of these new methods to the brain has yielded rich dividends in an area of biomedical research which hitherto escaped scientific scrutiny.

This book gives a good, if not comprehensive, account of recent developments in the investigations of higher cognitive functions. The 14 chapters and the concluding overview are of consistently high standard. Several papers explore the morphological correlates of higher cognitive functions, like Siegel's investigations of the dendritic arborisation or Diamond's excellent analysis of cortical changes brought about by learning and experience. Other chapters review physiological correlates of cerebral functions as afforded by modern imaging techniques. Even more traditional approaches are illuminated: Pandya and Yeterian give an overview of the architectonic, neuronal circuitry and connections of the cerebral cortex against the background of brain evolution and function.

The weakest parts of the book are the discussions which conclude each chapter. These might have been interesting for the participants at the meeting in the heat of the argument, but fall flat for the reader removed from the immediacy of the symposium. This
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