Neurology

Psychiatric disorders in neurology patients

John Moriarty

Psychiatric disorders

A high incidence of psychiatric disorders is found in patients attending neurologists. Among new outpatients, the prevalence of all disorders has been estimated at >50%.¹ Mood and anxiety disorders are present in 40% of patients, are associated with greater disability and are persistent.² Functional neurological symptoms or somatoform disorders may be present to some degree in up to one third of patients and these are the patients neurologists find more difficult to help.³ In the paper by Jeffries *et al*⁴ (see p 414), similarly high rates of psychiatric disorder are shown to occur among inpatients, with just over half of all patients having some form of disorder.

Often, these disorders are not formally recognised by neurologists and a minority of patients are referred for psychiatric intervention.⁵ Jeffries *et al* suggest a batch of relatively easy-to-administer questionnaires as a screening instrument with good sensitivity for the detection of psychiatric disorders.

Neurologists may find it implausible that they could be failing to diagnose psychiatric disorders in such a high proportion of their cases. Clinicians may be reluctant to give half of their patients an additional diagnosis when the implications for treatment are unclear to them. Patients may feel stigmatised. Caseness in psychiatry means exceeding a certain

threshold of symptomatology rather than implying any disease process, and some patients experience distress and fear of illness, which could be unhelpful to pathologise as disorder. The real challenge is to identify patients who have symptoms amenable to treatment and whose outcome is improved by psychiatric or psychological intervention. Psychosocial intervention such as explanation, education or problem solving may be what is required in many cases, whereas more definitive psychiatric or psychotherapeutic treatment may be needed in others.

At present, there are likely to be more than clinical factors at play in determining who is diagnosed and who is referred to psychiatry. Psychiatric disorder in neurology patients may be seen as beyond the expertise or remit of overburdened mental health services, with their focus on severe and enduring mental illness. Consultation-liaison services or specialist neuropsychiatry liaison services, if they exist, may be patchy and under-resourced. What services are available may differ from place to place as to whether they think it appropriate to take on the management of the whole range of psychiatric disorders, including dementia, mood disorder, psychosis, substance misuse as well as conversion or dissociative disorders.

There are implications from the increasing recognition of the prevalence and burden of psychiatric and psychological disorders among patients with neurological disease for training, service provision and research. Training of both neurologists and psychiatrists will need to include knowledge and skills in the recognition and treatment of common disorders. Service providers will need to look towards the provision of at least good enough psychological and psychiatric treatment services for this group of patients. Clinicians must aim to identify those patients or patient groups most likely to benefit from interventions and develop strategies for their cost-effective delivery. The gap between this aspiration and the current service is considerable.

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Narcolepsy and RBD

Towards a common pathway for narcolepsy and rapid eye movement sleep behaviour disorder

Rainer Dzierwas

Robust association between narcolepsy and RBD

Following the pioneering work of Schenk and Mahowald,¹ a robust association of idiopathic narcolepsy

and rapid eye movement (REM) sleep behaviour disorder (RBD) has recently been confirmed, suggesting that around

one third of patients with narcolepsy have concomitant RBD.² Although particularly severe cases with frequently occurring cataplexies and other associated symptoms of narcolepsy seem to be affected, the pathophysiological link between narcolepsy and RBD remains to be elucidated.

Contributing to this topic, in this issue, Mathis *et al*³ (see p 427) report on a HLA DR2-negative patient who developed both narcolepsy and RBD due to an isolated inflammatory lesion of the dorsomedial pontine tegmentum. Their finding is very interesting as it suggests a common pathway for narcolepsy and RBD at the level where REM muscle atonia is regulated. Apart from a conclusive neuroanatomical and pathophysiological survey,

the authors lent further support to their hypothesis by showing that the depicted anatomical region was also affected in published cases of either RBD or symptomatic narcolepsy due to circumscribed brain stem lesions.

Future research, of course, has to establish the presumed connection of narcolepsy and RBD in larger collectives of patients. Methodologically, such studies may apply advanced neuroimaging techniques, such as voxel-based morphometry and diffusion tensor imaging, which have recently been introduced for structural evaluation of the brain in neurological disorders. It might be interesting to compare groups of narcoleptics with and without RBD with regard to the integrity of fibre tracts involved in the generation of muscle atonia—that is, those originating from the pontine tegmentum and descending via the medial medulla to motor neurones.⁴

Looking at the topic in question from a different angle, one may choose to study patients with Parkinson's disease, multiple system atrophy and dementia with Lewy bodies. All these synucleinopathies are known to be associated with RBD, with one third of patients with Parkinson's disease and about 80% of patients with multiple system atrophy and dementia with Lewy bodies being

affected.^{5–7} Interestingly, apart from excessive daytime sleepiness, sleep episodes and hypnagogic hallucinations, sleep-onset REM periods on the Multiple Sleep Latency Test have repeatedly been described in the neurodegenerative disorders mentioned, which were especially prevalent in patients with concomitant RBD.⁸ The remarkable association of these features, especially the finding of multiple sleep-onset REM periods, is reminiscent of primary narcolepsy, and helped in coining the term “narcolepsy-like phenotype”.⁸ In the future, using voxel-based morphometry and diffusion tensor imaging, we could compare well-characterised groups of patients with and without RBD, with and without a narcolepsy-like phenotype, and with a combination of both. Although changes in different brain regions are to be expected because of the multifocal pathophysiology of the synucleinopathies, it may be speculated that fibre tracts and neurone populations located in the suggested “common pathway for narcolepsy and RBD” are exclusively affected in patients showing both syndromes.

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Limbic encephalitis

Limbic encephalitis: extension of the diagnostic armamentarium

Christian G Bien

Diagnostic spectrum of limbic encephalitis

The highly specific tests for anti-Hu, anti-Ma, anti-amphiphysin and anti-CV2/CRMP5 antibodies have been an important tool in the diagnosis of paraneoplastic limbic encephalitis (PLE).¹ The testing procedure for these antibodies is two tiered. Firstly, patients' sera are incubated with brain slices from a paraformaldehyde-perfused animal (usually rat or monkey). Specific binding patterns to brain cells allow the identification of the above-named “well-characterised” antibodies. After this immunohistochemical test, a confirmatory blotting test is performed on the brain homogenate (in comparison with a

positive control) or on the suspected antigen as recombinant protein. For both techniques, testing of serum is more sensitive than testing of cerebrospinal fluid (CSF). The antibodies detected in this way react with intracellular antigens. They portend a poor outcome. A relevant limitation, however, is that 40% of patients with PLE do not harbour any of these antibodies.¹

The diagnostic spectrum of limbic encephalitis was considerably broadened by the detection of antibodies against voltage-gated potassium channels in association with (usually) non-paraneoplastic limbic encephalitis (NPLE). These

antibodies are diagnosed in serum by radioimmunoprecipitation. On standard immunohistochemical analysis, strongly positive sera stain the molecular layer of rat cerebellum. The prognosis of affected patients is good with consequent immunotherapy.²

In the paper by Bataller *et al* (see p 381),³ the Philadelphia group summarises its most recent experience on autoantibody testing in cases with the clinical syndrome of limbic encephalitis. They report on several patients with PLE found to harbour in their sera and CSF novel antibodies reacting with rat brain neuropil in the molecular layers of the hippocampus and cerebellum on immunohistochemical analysis. Importantly, this reactivity could be picked up only if slices were prepared from a brain that had not been perfused but instead had been immersed in paraformaldehyde for several days. The staining pattern was more easily detected with CSF than with serum. The antigens recognised by these antibodies have not yet been determined, and no consistent banding pattern on western blotting has been identified. Most patients had ovarian teratomata or thymic tumours. They had a mostly