against herpes simplex virus. Antibodies against HIV and CSF cultures were negative. Three weeks later, she developed polydipsia and polyuria with a daily urinary output of up to 7 l. Anterior pituitary hormone function was normal. A water deprivation test by the method of Miller and colleagues was performed and the table showed a progressive increment in serum osmolality of 277, 289, 302, 308, and 315 mmol/kg. A diagnosis of partial central diabetes insipidus was made and she was given a course of subcutaneous 1-dimino-8-d-arginine vasopressin (DDAVP). Brain MRI showed the absence of the posterior pituitary “bright spot” in sagittal T1 weighted images. Six months later there was still considerable polyuria, which improved with nasal DDAVP treatment.

Hypothalamic-pituitary dysfunction after acute meningoencephalitis is very rare. A case of central diabetes insipidus was described as a complication of herpes simplex encephalitis in a patient with AIDS. We report here the first case of herpes simplex encephalitis associated with diabetes insipidus in a previously healthy subject. Polyuria and polydipsia can also result from nephrogenic diabetes insipidus with insensitivity to the antidiuretic actions of vasopressin, which can be caused by several drugs such as aminoglycosides. Although our patient had been treated with amikacin, the persistence of the clinical manifestations after drug withdrawal makes this an unlikely cause. Furthermore, both the water deprivation and hypertonic saline infusion tests clearly indicated a partial central diabetes insipidus. The absence of the posterior pituitary “bright spot” on T1 weighted MRI images, although reported in neurogenic diabetes insipidus, occurs in a large proportion of healthy people.

ENDOCRINOLOGY


Although Charcot described amyotrophic lateral sclerosis almost 130 years ago, we have known little about its etiology until very recently. This book, which accompanied the 1994 Marseille neuromuscular conference, exudes the optimism which followed the discovery of superoxide dismutase mutations in familial ALS, and the results of the Riluzole study.

The book opens with 16 chapters addressing pathogenesis, with a stress on excitotoxic mechanisms, in particular the role of glutamate. SOD1 defects have only been detected in 20% of familial ALS cases, suggesting that glutamate may only play a part in a cascade of processes leading to cell death. The evidence for glutamate transporters, post synaptic receptor changes, and autoimmune against calcium channels or gangliosides is discussed, together with the evidence from animal models implicating abnormalities of neurofilament genes, and neurotoxins.

The issues of causation having been set out, the niceties of differential diagnosis and natural history are discussed in relation to clinical trial design. In the final section, various therapeutic modalities are discussed, including neurotrophins, glutamate receptor antagonists, and mechanisms of inhibiting excitotoxins. Inevitably, many of the management approaches are based on animal models of diseases which do differ from ALS/MND, and which are reminiscent of the logic by which selegeline was considered a possible neuroprotective agent in idiopathic Parkinson’s disease. Nevertheless, the biological principles which are being uncovered may have a role in neurodegenerative diseases other than ALS.

The many short chapters (34 in 277 pages) offer a “bite sized” approach to the subject matter, allowing an easy acquaintance with many of the issues. Whereas some of the chapters are extremely informative, the overall effect of the text is reminiscent of a bowl of Shreddies placed before a Shredded Wheat eater—arguably everything appears to be present, yet something is still missing.

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