Interleukin 1A polymorphism in AD

There are epidemiologic data suggesting that nonsteroidal anti-inflammatory drug use reduces the probability of developing AD. Interleukin (IL)—an inflammatory cytokine—is present in the neuritic plaques of AD. Du et al. (p. 480) found that the allele frequency of a specific polymorphism of IL1A slightly increased the risk of AD in heterozygotes and conferred a sevenfold increased risk in homozygotes. ♦ The accompanying editorial by Rogers (p. 464) links this observation to a number of possible avenues for treatment.

Hippocampal atrophy: AD vs mild cognitive impairment (MCI) vs normal aging

Jack et al. (p. 484) performed longitudinal MRI and cognitive testing in three groups of subjects followed up for a mean of 3 years. They document that cognitive decline correlates with hippocampal atrophy and that hippocampal atrophy progresses to an extent in AD > MCI > normal.

Intracranial atherosclerosis: Treatment and treatment failure

Thijs and Albers (p. 490) reviewed the charts and obtained follow-up interviews on 52 patients with TIA or stroke associated with evidence of intracranial atherosclerotic vessel disease. The majority of patients developed further cerebrovascular events—most within a brief period of time. They suggest that the high rate of recurrence argues for developing alternative treatment strategies (e.g., angioplasty).

♦ The accompanying editorial by Benesch and Chimowitz (p. 465) emphasizes that the anticoagulant treatment of the Thijs and Albers patients was not standard and that the end points for failure included TIAs (the majority of failures). Benesch and Chimowitz argue the overwhelming importance of determining whether adequate warfarin therapy is beneficial in such patients—a question currently under investigation in the WASID clinical trial.

Transplantation of neurons: A new treatment for stroke?

The expedited brief communication by Kondziolka et al. (p. 565) presents data from a pilot trial of cultured neuronal cells as a treatment for stroke. Twelve patients who were stable 6 months to 6 years post-completed stroke received cells. PET showed that blood flow increased following transplantation. Two patients had gains in motor function.

♦ The accompanying editorial by Zivin (p. 467) points out that the clinical observations are not necessarily significant and that this technique is likely to receive careful scrutiny by the Food and Drug Administration before it can be considered for clinical application.

CSF 14-3-3 protein for diagnosis of Creutzfeldt-Jakob disease (CJD)

Lemstra et al. (p. 514) studied the sensitivity and specificity of CSF 14-3-3 protein testing in 112 patients with CJD. Diagnosis was established by standard criteria. The test was highly sensitive (97%) and specific (87%), with the false positives being in patients with stroke and meningoencephalitis

(both unlikely to be clinically confused with CJD).

Is thyroid assessment needed during interferon beta (IFN β) treatment?

Thyroid dysfunction has been associated with IFN β treatment of MS. Monzani et al. (p. 549) followed 31 MS patients for 3 years and found that thyroid dysfunction was transient in four of the six patients with thyroid disease appearing in the first year. Thyroid disease did not appear after the initial year of treatment.

Mental retardation in Duchenne dystrophy (DD)

At least a third of DD patients are mentally retarded. Felisari et al. (p. 559) studied IQ in 66 boys with DD and characterized their dystrophin gene by PCR. Distal deletions, particularly of the DP140 region, were highly correlated with lower IQ.

CSF leak causing apparent pituitary tumor

Mokri and Atkinson (p. 573) report five patients with clinical and MRI features of CSF leak in whom the pituitary developed gadolinium-enhancing enlargement. Surgical repair of the leak resulted in the resolution of pituitary enlargement.

Effect of valproic acid (VPA) on lamotrigine (LTG) clearance

Kanner and Frey (p. 588) studied 28 patients on both VPA and LTE and found that VPA decreases LTG clearance and increases LTG levels independent of dose or drug level of VPA.

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