Impaired glucose tolerance and small fiber neuropathy

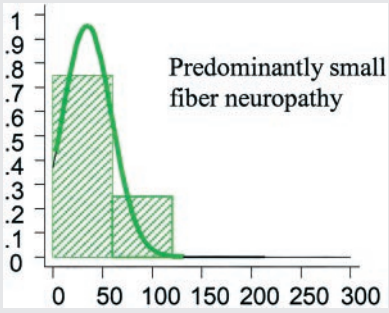
Sumner et al. found that impaired glucose tolerance (IGT) has an unexpectedly high prevalence among patients with clinically confirmed sensory neuropathy of unknown cause. IGT-associated neuropathy is less severe than DM-associated neuropathy. Small caliber sensory nerves may be the earliest detectable sign of neuropathy in glucose dysmetabolism.

see page 108

Painful sensory neuropathy from abnormal glucose metabolism?

Commentary by William R. Kennedy, MD

Several investigators have recently shown that impaired glucose tolerance (IGT) is common among patients with idiopathic sensory neuropathy.[1,2] The Sumner et al. study assessed oral glucose tolerance in patients with neuropathy of unknown cause and found that neuropathy was less severe in patients with impaired glucose tolerance than in diabetics. Indeed, most patients with impaired glucose tolerance had normal nerve conduction studies, and the mean distal leg epidermal nerve fiber density in this group was at the lower limits of the normal range.[3] They infer that neuropathy with IGT and diabetic neuropathy may have a common mechanism, with the severity of neuropathy reflecting the degree of abnormal glucose metabolism.



The relationship between IGT and neuropathy is intriguing and certainly requires further study. The frequency of IGT in neuropathy needs to be compared with a control group studied concurrently. Longitudinal prospective study of patients with IGT and neuropathy is needed to determine the course of the neuropathy and the likelihood of progression to diabetes. This study adds to the growing literature on this important subject.

References

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2. Singleton JR, Smith AG, Bromberg MB. Painful sensory neuropathy associated with impaired glucose tolerance. Muscle Nerve 2001;24:1225–1228.
3. McArthur JC, Stocks EA, Hauer P, Cornblath DR, Griffin JW. Epidermal nerve fiber density: normative reference range and diagnostic efficiency. Arch Neurol 1998;55:1513–1520.

Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury

Chang and Lowenstein review evidence on the prophylactic use of antiepileptic drugs (AEDs) after severe traumatic brain injury. Phenytoin decreases the rate of seizures occurring within 1 week of injury, while AEDs do not appear effective in preventing later seizures.

see page 10

continued on page 2

Morbidity of Guillain-Barré syndrome in the intensive care unit

Henderson et al. reviewed 117 patients with GBS requiring intensive care. Pulmonary morbidity occurred in half of the patients, mostly linked to mechanical ventilation. Hyponatremia (<130 mmol/L) occurred in 15%. Other major complications were uncommon: pulmonary embolism (3%); GI bleeding (5%); perforated ulcer (2%); and hyperglycemia (4%). Reducing ICU morbidity in GBS will require better management strategies for mechanical ventilation.

see page 17

New gene for demyelinating peripheral neuropathies

Street et al. describe mutations in the *LITAF/SIMPLE* gene on chromosome 16 as the molecular basis of Charcot-Marie-Tooth disease type 1C. This suggests that *LITAF/SIMPLE* may play a role in the degradation of proteins critical to peripheral nerve function.

see page 22

Interferon β-1A in primary progressive MS

Patients with PPMS have not been included in most therapeutic trials of IFNβ. In an exploratory study, Leary et al. investigated exclusively PPMS and demonstrated both the feasibility of trials in PPMS and that a 30-μg dose of IFNβ-1a was well tolerated but without benefit.

see page 44

A distinctive new cerebroretinal vasculopathy

Vahedi et al. report a kindred with an autosomal dominant disorder characterized by infantile hemiparesis, migraine, MRI-documented leukoencephalopathy with dilated perivascular spaces and microbleeds, and retinal arteriolar tortuosity with retinal hemorrhage. The condition is distinguishable from other hereditary conditions affecting both retinal and cerebral vessels.

see page 57

The accompanying editorial by Martin Dichgans tabulates the hereditary cerebroretinal vasculopathies, including CADASIL, HERNS, HVR, CARASIL, and Fabry disease. He notes the frequency of migraine and cerebral microbleeds in these diseases and considers the various diagnostic strategies indicated in patients suspected of having such disorders.

see page 8

Vascular risk factor control

Ruland et al. assessed rates of awareness, treatment, and control of risk factors after stroke. Glucose >200 mg/dL was found in 33% of diabetics, only 30% of treated hypertensives had a blood pressure <140/90 mm Hg, and only 43% of patients with hypercholesterolemia were on lipid-lowering medication.

see page 64

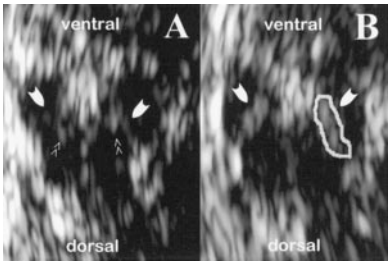
continued on page 3

GDNF for treatment of PD

Nutt et al. conducted a double-blind trial of the neurotrophin, GDNF, administered intraventricularly in 50 PD subjects. GDNF produced numerous adverse effects, particularly sensory symptoms, weight loss, and hyponatremia. Moreover, GDNF did not improve parkinsonism, possibly because it did not penetrate brain parenchyma.

see page 69

Brain parenchyma sonography discriminates PD and atypical parkinsonian syndromes



Walter et al. studied 50 patients: 25 with idiopathic PD and 25 with APS. Sonography discriminated idiopathic PD from atypical parkinsonian syndromes with a 96% specificity and a 91% sensitivity. Substantia nigra hyperechogenicity was the most useful finding to indicate idiopathic PD, whereas substantia nigra echogenicity was normal in atypical parkinsonian syndromes.

see page 74

Regional specialization of neural networks for reading in young children

Gaillard et al. used fMRI to identify the language networks implicated in reading in 5- to 7-year-old children. They found regionally specific and lateralized activation in the left inferior temporal lobe, the superior temporal sulcus, and midfrontal cortex. Reading networks in early readers are similar to adults.

see page 94

Alterations of brain chemistry in autism

Children age 3 to 4 years with autism spectrum disorder (ASD) have been found to have cerebral enlargement when compared to children with normal or delayed development. Here, Friedman et al. tested hypotheses of neuronal overproliferation in ASD using proton echoplanar spectroscopic imaging to characterize regional brain chemistry. Alterations in cellular composition were demonstrated in children with ASD but the direction of effects implicated mechanisms other than neuronal overproliferation.

see page 100

January 14 Highlights

Neurology 2003;60;1-3
DOI 10.1212/WNL.60.1.1