ophthalmoplegia in our patient might be the result of an immune reaction located in the wall of the cavernous sinus. Tests for lupus should be systematically done in every patient affected by painful ophthalmoplegia.

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References

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Triphasic EEG discharges in metrizamide encephalopathy

Sir: The water-soluble contrast agent metrizamide has proved safe and effective in various neuroradiologic procedures. The adverse effects of this agent differ in some respects from those of other contrast media, and have been well described. Seizures and transient cerebral symptoms have been seen occasionally, but increasing attention has been focused on a prolonged toxic encephalopathy with stuttering, obtundation, asterexis, and myoclonic jerks, which on at least one occasion has relapsed after recovery from the initial episode. Various EEG changes have been described after metrizamide injection: triphasic waves associated with metabolic encephalopathy have recently been considered. We encountered a patient with toxic encephalopathy after metrizamide myelography whose EEG showed triphasic waves on several occasions. Such patients may provide insight into the mechanisms of triphasic EEG discharges in metabolic encephalopathies.

A 65-year-old female had been healthy except for hypertension treated with salt restriction, but sufferer left leg and foot pain since an automobile accident in 1968. She was referred for implantation of a transcutaneous nerve stimulator after failure of analgesics and antidepressants. Strength was normal except for 4/5 proximal and 3/5 distal weakness in the left leg. Pain and touch sensation were decreased over the lateral and posterior aspects of the left leg, thigh, and buttocks, and the medial aspect of the left foot was hyperesthetic. Deep tendon reflexes were brisk and symmetric in the arms and hypoactive in the legs, without plantar response. Laboratory studies were normal. Metrizamide myelography of the thoracolumbar region was carried out, and on the following morning the patient abruptly became confused and agitated. All medication given before or during the myelogram had been discontinued. Neurologic examination was unchanged except for disorientation, monosynaptic responses to stimulation, and asterexis. Complete blood count, blood glucose, serum electrolytes and calcium, liver function tests, arterial blood gases, and cerebrospinal fluid were normal. An electroencephalogram showed continuous triphasic sharp waves of 100–200 μV amplitude and 2–4 Hz frequency, which were maximal frontocentrally and decreased in amplitude as they extended posteriorly. Occasional theta activity was present in the frontal, parietal, and occipital areas, but alpha rhythm was absent and background activity was unchanged by stimulation or eye opening. The patient's mental state improved over the next several days, no further studies were undertaken, and she underwent implantation of a dorsal column stimulator. Subsequent EEGs have shown diffuse theta slowing, without triphasic waves of symmetry. This patient developed an acute encephalopathy with asterexis and triphasic EEG discharges after metrizamide myelography. No underlying or precipitating medical or neurologic disorder was found, but the subsequent persistence of EEG slowing raises the possibility of a pre-existing mild encephalopathy placing her at risk for this complication. The persistent EEG slowing despite return of the mental state to normal could also reflect a mild but persistent encephalopathy afterward, analogous to the persistent head bobbing reported in two patients by Davis and coworkers.

In these patients, the movement disorder was present for several months after myelography despite normal mentation, and the EEG was diffusely slow in one patient. The mechanism and generator sites of triphasic waves are unknown, but subcortical or thalamic generation has been suggested, and could be supported by evidence of subcortical origin for other rhythmic EEG patterns, and of the role of thalamic and brain stem centers in the generation of normal EEG rhythms.

Kaada studied 79 patients undergoing lumbar myelography, none of whom developed encephalopathy, and observed transient and diffuse slowing in 13 and epileptiform discharges in none. Ropper, Chiappa, and Young studied prospectively the effects of metrizamide myelography or cisternography in 61 patients, without encephalopathy. Six percent of patients had either paroxysmal discharges or slow activity with some sharp features. This suggested the possibility of diffuse cerebral uptake of metrizamide after lumbar injection, with both irritative and depressive effects.

Vincent and Zimmerman briefly reported a patient who developed confusion, agitation, tremor, and asterexis after lumbar myelography. The EEG demonstrated generalised slowing and bifrontal triphasic delta transients, and clinical and EEG findings resolved within 48 hours. The authors ascertained the previous occurrence of at least two similar cases, one patient showing triphasic EEG discharges.

Two patients with transient confusion and asterexis after lumbar myelography were reported by Rubin, Horowitz, and Katz. The EEG showed bilateral and independent paroxysmal sharp transients and slow waves in the posterior temporal, parietal, and occipital areas. Bertoni et al. reported two patients with similar features, one of whom had no EEG and the other a normal tracing when the encephalopathy had virtually resolved. A CT scan of the brain in the former patient demonstrated widespread cerebral uptake of metrizamide.

Two mechanisms for encephalopathic complications of metrizamide myelography were proposed by Bertoni et al.: interference with the sodium-potassium ATPase pump and competitive inhibition of glucose metabolism as a 2-deoxyglucose analogue. No in vitro inhibition of a partial reaction of the cation pump was found, but metrizamide in concentrations analogous to those after lumbar injection competitively inhibited microbial hexokinase. This finding suggests that the encephalopathy might be due to impaired glucose metabolism.

The clinical and EEG similarities between several recent cases of metrizamide encephalopathy and metabolic derangements typified by hepatic encephalopathy...
might suggest a common mechanism for the generation of triphasic EEG discharges, possibly the impairment of glucose metabolism in subcortical nuclei. Investigation of cerebral glucose metabolism in those patients with metrizamide encephalopathy who have triphasic EEG activity, and study of the effects on the EEG of experimental disturbance of cortical and subcortical glucose metabolism, may clarify the pathogenesis of triphasic waves.

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References

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Evoked Potentials for the Assessment of Central Nervous Disorders: an international symposium and a workshop will be held 27-29 June 1984 at the University Hospital of Lausanne, Switzerland. Information may be obtained from: PA Despland MD, EEG-EMG and Neurophysiological Unit Dept of Neurology, University Hospital – CHUV, 1011 Lausanne, Switzerland.

Notices

Behavioral Neurology Society. The Third Annual meeting will be held in Boston on 10 April 1984. Further information may be obtained from: Dr Francois Boller, MD, Medical Center, University Drive C, Pittsburgh, Pennsylvania 15240, USA.

International Congress of Physical Medicine and Rehabilitation. The 9th Congress will be held 13-18 May 1984 in Jerusalem, Israel. Information may be obtained from: J. Chaco, MD, PO Box 983, Jerusalem, 91009, Israel.

Neuro-ophthalmology. Joint world meeting. A joint meeting of the International Society of Neuro-ophthalmology and the Congress of the Study Group of Neuro-ophthalmology and Neuro-genetics of the World Federation of Neurology will be held in Antwerp, 14-18 May 1984. Information may be obtained from: Professor A Neetens, Academic Hospital, University of Antwerp, Wilrijkstraat 10, 2520 Edegem, Belgium.

Eighth International Symposium on Parkinson's Disease

Under the auspices of the World Federation of Neurology, Research Committee on Extrapyramidal Disease, this Symposium will be held in New York 9-12 June, 1985. Information may be obtained from Kenneth J Bergmann, MD, Department of Neurology, Mount Sinai School of Medicine, City University of New York, New York, NY 10029, USA.