Neurologist-in-training

The aim of this section is to prepare the neurologist-in-training for the FMH examination, to confront her or him with specific problems of everyday neurological practice and to give him or her updates on recent controversies in clinical neurology.

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Neurological MCQ

■ **P. Michel**

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Select the one correct answer.

1 Carpal tunnel syndrome is associated with each of the following, except:

A Amyloidosis
B Acromegaly
C Vasculitis
D Systemic use of corticosteroids
E Use of oestrogens

2 In carpal tunnel syndrome, which treatment has been shown in randomised controlled trials to be superior to placebo (for drug treatments) or superior to conservative treatment (for surgical interventions)?

A Nonsteroidal anti-inflammatory drugs
B Open surgical decompression
C Endoscopic decompression
D "Mini"-open surgical decompression
E Local corticosteroid injection

3 Which statement is wrong? Compared to the visual aura of migraine, occipital epilepsy

A more frequently has several attacks during the same day (clustering)
B manifests with circular visual patterns
C usually lasts <1 min
D can reliably be differentiated from visual migraine aura with interictal EEGs
E may be idiopathic

4 Headaches and epilepsy: which statement is wrong?

A Headaches are present in about half of patients after a generalised epileptic seizure.
B Headaches can be the only manifestation of partial seizures.
C Migraines triggered by seizures are called "migralepsy".
D Headaches are more frequent after generalised than after partial seizures.
E The following antiepileptics have been shown to decrease migraine headaches in randomised trials: valproate (Depakine®, Orfiril®), topiramate (Topamax®), and gabapentin (Neurontin®).

5 Which statement is correct regarding multiple sclerosis (MS)?

A A diagnosis of definitive MS cannot be made if there is no intrathecal synthesis of oligoclonal IgG.
B Visual evoked potential testing is not part of the current diagnostic criteria for diagnosing relapsing-remitting MS.
C Probable MS can be diagnosed on clinical grounds alone or on combined clinical and MRI information.
D Definite MS cannot be diagnosed in the presence of a normal MRI.
E MRI should not be used as a criterion for dissemination in time.

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6 Which statement is correct?

A Treatment with corticosteroids slows down the progression of the handicap in relapsing-remitting or secondary progressive MS.
B Beta-interferons are the only proven long-term treatment to prevent relapses.
C Patients with the primary progressive form of MS benefit from interferon-beta treatment.
D The EDSS (Expanded Disability Status Scale) should not be a factor in the decision to start immuno-modulatory medications.
E Intramuscular and subcutaneous preparations of beta-interferons are currently accepted by Swissmedic as preventive treatment for MS-relapses.

(For correct answers, see page 364)

References

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Neuroradiology/Neuroanatomy

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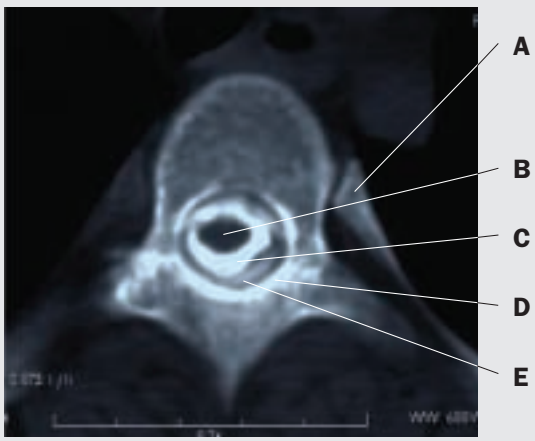

A 32-year-old lady with no past medical history complains about fluctuating diffuse headaches for the last 4 weeks, worse when upright. Her neurological exam is normal.

On this myelographic CT (spinal CT after injection of contrast into the thecal sac) identify the anatomical structures "A–D" and make a diagnosis "E".

Picture kindly provided by the Département de Radiologie Diagnostique et Interventionnelle, CHUV, and by Dr H. Russmann, Service de Neurologie, CHUV.

(For correct answers, see page 364)

Read for you

■ **A. Rossetti**

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Anoxic coma and neurobiochemical markers: do they really help?

Predicting whether a patient in anoxic coma will regain consciousness and awareness is a challenging task for the clinician. Until now, decision of continuing or withdrawing intensive treatment is mainly based upon clinical examination (GCS), EEG and somatosensory evoked potentials (SSEP). These elements, however, often are contradictory in the early setting or (especially the former two) may be influenced by drugs. Since many subjects in this condition will die or remain in a persistent vegetative state, early decision would have important ethical and practical implications. Two recent papers focus on biochemical markers in this setting.

A Dutch group considered prospectively 110 patients, including 67 subjects who were comatose after 48 hours (12 died and 31 regained consciousness within two days), assessing serially serum neuron specific enolase (NSE) and short latency SSEP until death or discharge [1]. The peak NSE values were registered 48 hours after admission. No patient with NSE >25 µg/l or with absent SSPE regained consciousness. On the other side, of 22 patients with preserved SSEP, one had a good outcome; of 29 subjects with NSE <25 µg/l, 3 regained consciousness. In a multivariate analysis NSE and GCS were stated clearly superior to SSEP in forecasting good neurological outcome. However, the predicting value of good outcome for NSE alone (<25 µg/l) was only 10%, SSEP alone (preserved) 4% and the combination of the two 18%. Of course, the specificity and predicting value for bad outcome was 100% for all tests.

In Germany 27 subjects were prospectively enrolled, with repetitive assessment of short latency SSEP, serum NSE as a neuronal and S-100B as a glial marker [2]. NSE peaked after 72 hours, S-100B after 24 hours. Although NSE at a cut-off of 43 µg/l had a sensitivity of 91% and a specificity of 100% in predicting poor outcome (similar values for S-100B and the combination of the two), the published data are insufficient to calculate the predicting value of good outcome. Absent SSEP predicted in 100% a poor outcome, preserved SSEP were slightly more often recorded in good outcome subjects.

Since all tests in this particular context should have a predicting value and a specificity of 100% for a bad outcome, the predicting value for good outcome remains dismal. Although neurobiochemical markers may help finding some more patients with poor prognosis than with "classical" methods, the aim of identifying most of them (or, considering the problem in the inverse manner, identifying most of those who will regain consciousness) is far from being reached. And, by the way, different cut-off values probably reflect different laboratory assessments, and in most hospitals (including university clinics) results of such markers are only available with a delay of several days. Waiting for the revolution, the combination of all clinical and paraclinical elements remains the best way (although very far from perfection) in the assessment of patients in anoxic coma.

References

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Neurologist-in-training

Answers to MCQ

1 C **2** E **3** D **4** C **5** B **6** E

Answers to Neuroradiology/Neuroanatomy

A Rib
B Spinal cord (dorsal level)
C Intrathecal contrast mixed with cerebrospinal fluid
D Epidural cavity (filled with venous plexus and fat)
E (correct diagnosis): Extradural spinal fluid collection due to a spontaneous spinal fluid leak.

Comment

This lady's headaches were worse in the upright position and nearly disappeared when lying down ("orthostatic headaches"). There was no recent history of head or neck trauma. At lumbar puncture, opening pressure was very low (0 cm H_2O), confirming the suspicion of intracranial hypotension. There were no cells and total proteins were normal. The search for a spinal leak by MRI showed diffuse contrast-enhancement of cerebral meninges and a possible leak on a mid-dorsal level of the spine. Lumbar CT-myelography demonstrated extravasation of contrast into the peridural sac with entry and exit points at D8 and D2 respectively. A peridural blood patch at mid-dorsal level relieved her symptoms completely.

Except for headaches, other neurological symptoms of intracranial hypotension include dizziness, nausea, vomiting, tinnitus, neck and interscapular pain, arm radiculopathy, horizontal diplopia, blurry vision and (rarely) facial numbness or weakness. Most of the times, physical examination is normal. However, there may be mild neck stiffness, a slow pulse rate ("vagus pulse"), unilateral or bilateral sixth nerve palsy and superior binasal visual field defects [1]. A history of minor trauma is often elicited and an underlying connective tissue disorder is present in some patients with spontaneous dural tears [2].

The frequent Gadolinium-enhancement of meninges on MRI is probably due to secondary thickening of the pachymeninges with an increase in fibroblasts and thin-walled small blood vessels [3]. Spinal MRI, radioisotope cisternography or myelography may show leakage sites, mostly in the thoracic or cervical spine [1].

In orthostatic headaches due to lumbar puncture, a single epidural blood patch gives relief in the large majority of patients. In patients like the one presented here, with spontaneous spinal leaks, only about one third of patients respond well to this treatment, and another 50% will respond after several epidural blood patches [4].

References

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