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

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Select the one correct answer.

1 Plasma exchange is an effective treatment for all of the following except:

A Myasthenia gravis exacerbation
B Chronic demyelinating neuropathy with monoclonal gammopathy
C Guillain-Barré-Strohl syndrome
D Multifocal motor neuropathy with conduction blocks
E Refsum's disease (phytanic acid accumulation)

2 Which of the following statements is wrong regarding the treatment of Guillain-Barré-Strohl syndrome?

A Plasma exchange alone is about as effective as intravenous immunoglobulines alone.
B Plasma exchange combined with immunoglobulines is more effective than one treatment alone.
C In mild and moderate forms the response to plasma exchange depends on the number of treatments.
D Plasma exchange is more effective if started within 7 days of disease onset.
E Immunoglobuline treatment does not have significantly more side effects than plasma exchange.

3 Which statement is wrong regarding epileptic seizures and syncope?

A Syncope can cause tongue biting and loss of urine.
B Seizures can be preceded by a sensation of vertigo.
C Disorientation during recovery from loss of consciousness is more frequent after seizures than after syncope.
D Syncope can be accompanied by seizure-like tonic-clonic movements.
E A syncope can reliably be distinguished from a seizure by an EEG performed after the event.

4 Which statement is true regarding the work-up of a patient with syncope?

A It is contraindicated to do a Schellong test in the emergency room for a patient who was admitted for a syncope 2 hours earlier and who has a normal general examination.
B It is contraindicated to perform carotid massage in the emergency room for an elderly patient who was admitted for a syncope 2 hours earlier and who has a normal general examination.
C The first step in the evaluation of a patient with a first syncope is an ECG.
D The majority of patients with abnormal results on laboratory blood tests have seizures rather than syncope.
E Tilt testing in patients with syncope due to ventricular and supraventricular tachyarrhythmias typically reproduces the arrhythmia.

5 The diagnosis in a patient with symptoms occurring in identical visual fields in both eyes before migraine headaches is

A Migraine with aura
B Retinal migraine
C Ophthalmic migraine
D Ophthalmoplegic migraine
E Basilar-type migraine

6 Which statement is true regarding the classification of headaches?

A Cluster headache is usually a symptomatic headache.
B The diagnosis of episodic migraine headaches is certain after a third migraine attack.
C SUNCT (short lasting unilateral neuralgiform headache with conjunctival injection and tearing) lasts usually longer than cluster headache.
D Chronic migraine or chronic tension-type headache is diagnosed if headache is present >90% of the time.
E Hemicrania continua typically responds to indomethacin.

(For correct answers, see page 98)

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Neuroradiology/Neuroanatomy

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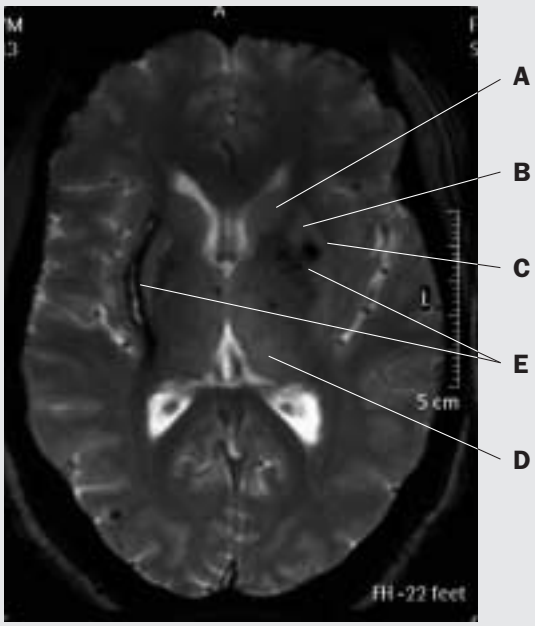

This 36-year-old African lady presented an acute left sensory-motor hemisyndrome 4 years prior to this MRI (FLAIR images). She takes 3 antihypertensive drugs and has no family history of cerebrovascular diseases.

Identify the anatomical structures "A–D" and make a diagnosis "E".

Picture kindly provided by the Département de Radiologie Diagnostique et Interventionnelle, CHUV.

(For correct answers, see page 98)

Read for you

■ **A. Rossetti**

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Seizures related to brain injury: incidence and the time to treat or not to treat

Seizures associated with a structural brain lesion are generally regarded as an indication to introduce a long-term anticonvulsant treatment (AED). However, the time of occurrence of the fits and the type of brain pathology may influence this decision.

A retrospective review of more than 5000 *multiple-sclerosis* patients [1] identified seizures not related to a previous history of epilepsy in *0.89%* (51 patients). Only 3 of them experienced seizures as initial neurologic event. Semiologically, the majority (35 patients) had generalised tonic-clonic seizures. Forty-five subjects received AED, only 5 of them showed subsequently intractable epilepsy.

The VENOPORT study from Portugal [2], including prospectively 91 patients with *sinus thrombosis,* found early seizures (within 2 weeks) in *34%* (31 patients). Five of them experienced late recurrence, and 3 more patients had late seizures without early convulsions, thus 9.5% of the cohort had late seizures. Haemorrhage was a strong predictor of seizures. The authors conclude that AED for one year may be indicated in subjects with early seizures and/or haemorrhage on the initial neuroradiological study. However, only 1/8 patients with early seizures who did not receive AED, but 4/18 with AED, developed recurrent seizures (12.5 vs 22%).

A prospective analysis of 247 patients surviving a *subarachnoid haemorrhage* [3] showed that epilepsy (i.e. recurrent seizures) developed in *7%*. Cerebral infarction and subdural haematoma were risk factors. In-hospital ("acute") seizures, although associated with a poor outcome, did not predict occurrence of epilepsy. Thus, the authors state that prophylactic AED is not routinely warranted, but should be restricted to patients with focal brain injury (i.e. cerebral infarction or subdural haematoma).

A systematic review of reports and trials regarding prophylactic AED in patients after *severe traumatic brain injury* [4] highlights that about *12%* of them develop seizures and that there is a level A evidence that prophylactic treatment with phenytoin in the first 7 days reduces the likelihood of having early post-traumatic convulsions. However, prophylactic AED is not indicated beyond the 7-day period (level B). In case of mild traumatic brain injury, powerful studies are still lacking, at present prophylactic AED is not warranted. In conclusion, the authors recall that early seizures (occurring in the first 7 days) do not appear to predict the occurrence of late seizures. Thus, prophylaxis is thus only directed against early seizures.

The PFO-ASA study group reviewed the cohort of 581 young (mean age 42 years) patients with a *cryptogenic stroke* enrolled in the original prospective study [5]. Early seizures (in the first 7 days) occurred in 2.4% (14 patients). Twenty patients (*3.4%* of the whole population) experienced late seizures, 6 of them had early seizures before. Interestingly, late seizure occurrence did not differ between patients who were treated or not after early convulsions. Independent predictors of late seizures were early seizures, large infarcts and cortical lesions.

Considering patients with *brain tumours,* a systematic review mentioned a risk of *20–40%* of experiencing seizures by the time of diagnosis [6]. At least 20–40% more would develop seizures afterwards. However, the meta-analysis did not show any benefit of a prophylactic AED treatment (level A), and side effects severity appeared to be higher in oncologic patients.

In summary, owing to the fact that phenytoin has been shown to impair recovery after brain trauma and shows proepileptogenic properties in animal models, caution is warranted in prescribing prophylactic AED after traumatic brain injury. Current evidence only shows AED efficacy in preventing early seizures (but not recurrent ones) in case of severe traumatic injuries. Long-term AED prescription before seizures is not routinely indicated in venous thrombosis, stroke, subarachnoid haemorrhage, brain tumour or multiple sclerosis.

References

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Neurologist-in-training

Answers to MCQ

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

Answers to Neuroradiology/Neuroanatomy

A Head of the caudate nucleus
B Putamen
C External capsule
D Thalamus
E Chronic hypertensive intracerebral haemorrhage (right putamen) and multiple asymptomatic microhaemorrhages (left putamen, thalami, left internal capsule)

Comment

This lady's hypertensive intracerebral haemorrhage and multiple asymptomatic microhaemorrhages (or "microbleeds") are likely to be related to her severe hypertension. Whereas cerebral amyloid angiopathy is unlikely in this young lady with deep microbleeds, (familial) cavernomatosis cannot be ruled out; the severe hypertension, absence of epilepsy, of a family history and of significant superficial lesions are arguments in favour of a hypertensive origin of these bleeds.

Hypertension is the major risk factor for intracerebral haemorrhages, especially when it occurs in deep and brainstem structures. Hypertension may also increase the risk for a variety of other types of cerebral lesions, such as lacunar and non-lacunar ischaemic strokes, leucoaraiosis and associated cognitive impairment. Microbleeds have now been recognised as being a marker for cerebral end-organ damage by hypertension, especially if poorly controlled [1]. These microbleeds are usually silent, and sometimes appear as lacunes on non-contrast CT. It remains unclear whether they represent primary bleeds or secondary blood extravasation after ischaemic lacunar infarcts.

MRI, especially gradient echo (or T_2* or susceptibility weighted) sequences are a highly sensitive method to diagnose acute or chronic intracerebral haemorrhages. It has now been shown that MRI including these sequences is equivalent to non-contrast CT in the acute setting when it comes to differentiating ischaemic from acute haemorrhagic strokes [2, 3].

References

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