How good are you?

Questions

1. Are these statements true or false?
   (a) 90% of large cortical infarcts are visible on CT within 48 h of symptom onset.
   (b) 40% of lacunar or small cortical infarcts are visible within 48 h after symptom onset.
   (c) Cerebral infarcts are most easily seen at 2–3 weeks on brain imaging.
   (d) The infarct is of CSF density by 3 months.
   (e) Fast spin echo and proton density sequences are the sequences of choice for identifying haemorrhage.

2. Please read the following passage.
   A 58-year-old publican awoke one morning with vertigo. He was also troubled by a worsening headache. The following day he became aware of intermittent horizontal diplopia. He booked an appointment with his general practitioner the next morning, but unfortunately was admitted to hospital that night with a decreasing conscious level.
   
   On examination he was febrile at 38.2°C. General examination was otherwise normal. There was no neck stiffness. GCS was 7 (E2, M3, V2). Fundoscopy was normal. Pupils were mid-sized and sluggishly reactive to light. He had a convergent squint. Reflexes were generally brisk with upgoing plantars.
   
   He underwent an urgent CT scan, followed by a limited MRI scan with T1, T2 and FLAIR sequences that were all normal. You are telephoned at 9 pm for advice.
   
   (a) Which of the following strategies would you recommend?
   
   (i) Urgent lumbar puncture and decide on further management depending on the results.
   (ii) Urgent MR angiogram with fat-suppressed T2 sequences the following morning.
   (iii) Observe overnight and re-examine the following day.

Paul Goldsmith and Graham Lennox
Department of Neurology, Addenbrooke’s Hospital, Cambridge, UK. Email: pg255@hermes.cam.ac.uk; drslennox@aol.com
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A 74-year-old lady was found collapsed in her bedroom at her residential home. Her past medical history was unremarkable except for bad osteoarthritis of her knees and hips. She smoked 10 cigarettes per day and enjoyed her sherry. She took ibuprofen for her pain. On examination she had a right homonymous hemianopia and a right sided facial weakness. She had an expressive dysphasia. The remainder of the cranial nerve examination was normal. In her limbs she had grade 2/5 weakness on the right in a pyramidal distribution with corresponding hyperreflexia and an upgoing right plantar. Her blood pressure was 170/95 and a left carotid bruit was audible. The remainder of the examination was normal.

Hb 12.5 g/dL, white cell count 12.4 × 10⁹/L (neutrophils 70%, lymphocytes 29%), platelets 330 × 10⁹/L, clotting studies: normal, Na 138 mmol/L, K 5.4 mmol/L, urea 28.5 mmol/L, creatinine 550 mmol/L, albumin 38 mmol/L, Ca 1.7 mmol/L, PO₄ 2.2 mmol/L, urate 480 µmol/L (N: 150–390 µmol/L), CXR & ECG: normal.

(a) What is the likely cause of the renal failure?
(i) Rhabdomyolysis
(ii) Hypertension
(iii) Non-steroidal anti-inflammatory drug induced damage
(iv) Hypoparathyroidism
(v) Cholesterol embolization syndrome

(b) Give one investigation to confirm the cause of the renal failure.
(i) Renal ultrasound
(ii) Renal angiogram
(iii) Urine dipstick plus microscopy
(iv) Urine microscopy & eosinophil count
(v) Parathormone level
(vi) Renal biopsy
(vii) Vitamin D level

(c) Give one specific management for this patient’s renal failure.
(i) Alkalinization of urine
(ii) Stop NSAID
(iii) Fluid restrict
(iv) ACE inhibition
(v) i.v. calcium gluconate
Answers

1. (a) T  
(b) T  
(c) F  
(d) T  
(e) F

Infarcts may become invisible from day 10–21 as they become isodense with the surrounding brain. This problem, known as ‘fogging’, affects CT as well as MRI. MRI with diffusion weighted imaging is probably the method of choice for detecting infarcts of all sizes early on.

Whereas CT will pick up almost all acute cerebral haemorrhages, routine MRI sequences are relatively insensitive. In contrast, in the later phase, MRI can be used to detect haemorrhage indefinitely on account of the low signal from haemosiderin (gradient echo sequences are the most sensitive for identifying haemorrhage). Fast spin echo & proton density sequences are relatively insensitive. (See Wardlaw 2001.)

2. (a) (i)  
(b) (iii) and (v)

Most neurologists would want to exclude infection by CSF examination once a posterior fossa mass lesion had been excluded by brain imaging. Even though the presence of fever doesn’t rule out the possibility of a brainstem stroke, there is no evidence that immediate heparin therapy is of benefit in basilar thrombosis. The answer to (b) is open to wide debate with many clinicians having their own personal approaches to the management of acute, but undiagnosed, focal syndromes. (See Williams & Nadel 2001.)

3. (a) 
Ganglia are defined as collections of nerve cell bodies located outside the central nervous system. There are two types: the autonomic ganglia & the craniospinal ganglia. The latter include the dorsal root ganglia and the ganglia of cranial nerves V, VII, VIII, IX & X. They are concerned with sensory relay. (See Fig. 1; image courtesy of Dr Howard Freedman).

4. (b) 
(See Fig. 2; from www.anatomy.dal.ca) The epidural space is the potential space between the skull and the dura mater and is the space into which epidural arterial haemorrhage occurs, usually from traumatic rupture of the middle meningeal artery (the classical ‘extradural haemorrhage’).

5. (a) (i)  
(b) (iii)  
(c) (i)

Rhabdomyolysis is a common cause of acute renal failure, particularly on intensive care units. It is often picked up late because there is no muscle pain, or any history of muscle injury. Thus the diagnosis needs to be considered in any patient whose urine is dipstick positive for blood. An absence of red blood cells on microscopy then confirms the diagnosis. This is much quicker and cheaper to do than a specific myoglobin assay. Typical biochemistry is a high phosphate and potassium with a low calcium. The muscle enzymes CK, aldolase, AST & LDH may be very high.

The myoglobin is less likely to crystallise in the presence of alkaline urine so many units give regular small aliquots of alkali until the urine pH is raised, provided a good urine output is maintained. Stopping the NSAID is appropriate in any acute renal failure but is not a specific management in this case.

Neurological associations of rhabdomyolysis
- Epilepsy
- Any cause of coma or immobility (stroke in this case)
- Drug addicts
- Malignant hyperpyrexia
- Prolonged exercise
- Myositis
- Infections (particularly viral)
- McArdle’s syndrome & other metabolic myopathies
- Alcohol
- Carbon monoxide poisoning
- Overdoses, e.g. monoamine oxidase inhibitors
- Fibrates and statins
- Glue sniffing

See Warren et al. 2002

References