

EANS/UEMS European examination in neurosurgery

Variants of questions with answers (compilation - Vyacheslav S. Botev, Department of Neurosurgery, M.Gorky Donetsk National Medical University)

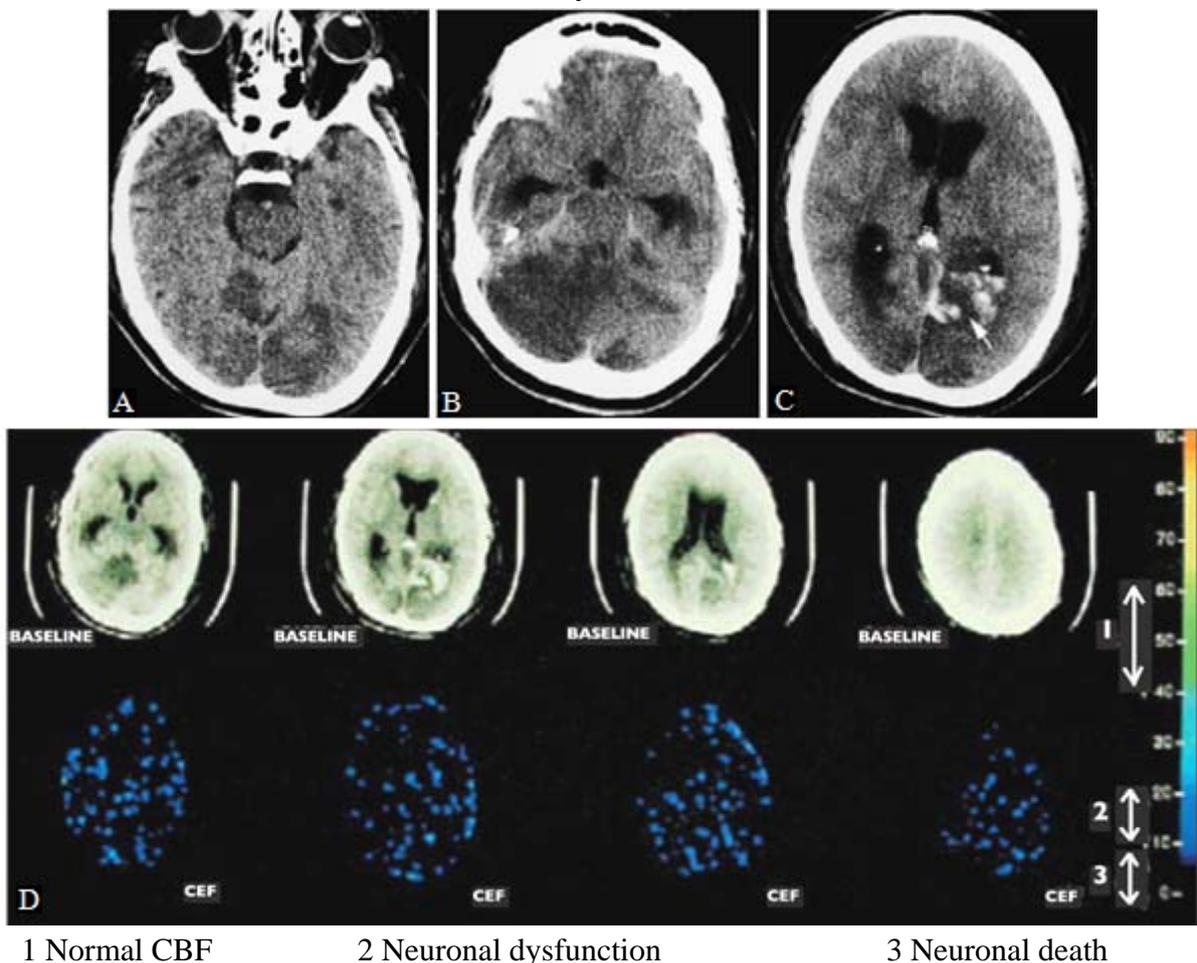
BRAIN DEATH SCENARIO

Just a year before, I had graduated from medical school in Kiev. I clearly remember my first night on call as a resident.

Around 2 AM, I was called to pronounce a patient dead. "Doctor, we need you in Neuro-ICU for a pronouncement" a nurse's voice said over the phone. "A pronouncement?" I asked. "Doctor?" she said again. It was the voice of a veteran, I could tell. "Oh, yeah, a death pronouncement. The patient just passed and we need a doctor to pronounce him as dead."

But so far it's been a teaching case.

A 44-year-old male was involved in a motor vehicle accident and had this non-enhanced CT (A). On the second hospital day, he remained comatose and a second CT (B, C) and xenon CBF study (D) were obtained.

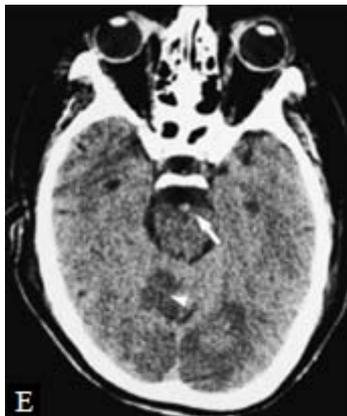


Questions

1. What does the initial CT show?
2. What is the patient's prognosis based on the second CT examination and xenon CBF study?
3. At what blood flow rate does electrical activity of the cerebral cortex fail?
4. Do movements of the limbs in patients who have met all brain death criteria rule out the diagnosis of brain death?
5. What are the cardiac manifestations of brain death?
6. Is it possible to predict which comatose patients will progress to brain death?

BRAIN DEATH SCENARIO

Answer



1. The admission nonenhanced CT (E) shows increased density in the basilar artery (arrow), suggesting thrombotic occlusion. In addition, decreased density in the vascular territory of both posterior cerebral arteries and the right superior cerebellar artery (arrowhead) indicates occlusion of the basilar artery, or embolization from the basilar or vertebral arteries. In the setting of trauma, vertebral artery dissection is the probable cause.
2. Nonenhanced CT examinations (B, C) obtained 24 hours after the injury show bilateral cerebellar infarcts, hemorrhagic transformation of the left PCA territory infarct (arrow), cerebral swelling, and acute hydrocephalus. These findings confirm evolution of infarcts in territories supplied by the vertebrobasilar circulation, as well as complicating edema and hydrocephalus due to compression at the level of the fourth ventricle or cerebral aqueduct.

The xenon CBF study is a recently developed CT technique for quantitatively

measuring CBF (D). Four levels of the brain are repeatedly scanned while the patient breathes xenon gas. The rate of increase in CT density of the brain reflects the local CBF, which can be measured and displayed as a color map. Normal CBF is 50 ml/100 g/min. Brain tissue becomes ischemic and neuronal function is impaired at flow <20 ml/100 g/min. Cellular death occurs when CBF is lower than 10 ml/100 g/min. In this case, CBF is 0–10 ml/100 g/min, indicating absent cerebral perfusion and brain death. The xenon CT can be used to assess regional perfusion in patients with cerebrovascular disease, vasospasm, and to adjust ventilation parameters in trauma patients with impaired cerebral autoregulation.

3. About 20 mL/100 g/min.

4. No. Two types of movements have been described in brain-dead patients:

1. Brain death–associated reflexes:

These have been found in approximately 75% of brain-dead patients, and current guidelines recognize the persistence of these reflexes. They include muscle stretch reflexes, plantar flexor and withdrawal responses, and abdominal reflexes.

2. Brain death–associated automatism:

These movements include undulating toe flexion movements, eyelid opening, respiratory-like movements, head turning, eyelid and tongue myoclonus movements of the upper and lower face, and the “Lazarus sign.” The Lazarus sign refers to complex movements of the upper extremities and may include extension and pronation of the arms, a drawing up of the arms toward the chest in a “praying” posture, or other complex movements. It is thought to be localized to the cervical spinal cord and represents hypoxia to neurons in these areas that have been functionally isolated from the brain.

5. Despite aggressive cardiovascular support, patients determined to be brain dead progress to cardiovascular collapse within 1 week. In fact, most die within 2 days after the diagnosis of brain death. Heart rate variability is lost (loss of vagal function) as well as heart rate response to atropine-like drugs. The electrocardiographic changes associated with the initial stage of brain death include widening of the QRS complex (i.e., Osborn waves), prolongation of the QT segment, and nonspecific ST-segment changes. More advanced stages of brain death are marked by bradycardia, followed by conduction abnormalities, including atrioventricular block and interventricular conduction delays. Atrial fibrillation is relatively common in the terminal stages of brain death, with atrial

activity often continuing after the cessation of ventricular complexes.

6. It is not possible to determine with confidence which comatose patients will progress to brain death, even though several studies have identified clinical parameters associated with poor neurologic outcome. The best predictors of neurologic outcome are the Glasgow Coma Scale score at presentation and the level of brain stem function observed within the first 24 hours after presentation. The lack of pupillary response to light or corneal reflexes on initial examination is a very poor prognostic sign. By 72 hours after cerebral insult, the motor response to pain increases in predictive value; absent or posturing response to pain is associated with poor neurologic recovery.

References

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