Update: Determining Brain Death in Adults

Case Presentation

Patient is a 37-year-old man who was an unrestrained passenger involved in a motor vehicle accident. He was found after several hours and immediately intubated at the scene. On arrival at the emergency room, he was comatose and intubated. Further workup showed no evidence of polytrauma affecting other areas of the body. CT scan of the brain showed diffuse edema and scattered hemorrhages throughout the brain suggestive of diffuse axonal injury. A fiberoptic intracranial pressure (ICP) monitor was placed, and his initial ICP was 30 mm Hg. He received IV propofol, fentanyl to facilitate mechanical ventilation, and a repeated dose of 1 gm/kg of mannitol to control ICP.

His examination—several hours after admission—showed he did not open his eyes to pain stimuli and had bilateral extensor posturing. Pupil size was normal, and corneal reflexes were intact. The oculocephalic reflexes were deferred, as the potential for cervical spine injury was unknown. Symmetric grimacing was noted after compression of the temporomandibular joints. He had a good cough reflex upon tracheal suctioning. He was triggering the ventilator. Tone in all extremities was increased, and reflexes were increased but subclonus. There were bilateral Babinski signs.

During the course of events, his ICPs measured between 10 and 40 mm Hg but responded well to intermittent doses of mannitol. Repeat CT scan showed evolving frontal and temporal lobe contusions without mass effect.

In the course of the next several days, the patient developed multiple increases in ICP that became refractory to osmotherapy despite target serum osmolality levels. Hypertonic saline (23% concentration) was subsequently administered, resulting in initially good response in his ICP management. However, surges in ICP continued despite repeated doses of hypertonic saline. His neurologic examination changed, and pupil responses disappeared, with pupils at 6 mm. Repeat CT scan showed a diffuse cerebral edema with effacement of cisterns and compression of the ventricular system. He suddenly developed hypotension and required intravenous phenylephrine. Urine production increased significantly, and diabetes insipidus was deemed likely with a specific gravity of 1001. He was treated with vasopressin infusion that not only stabilized polyuria but also resulted in better control of his arterial blood pressure. The nursing staff noted no triggering of the ventilator and no cough response, which prompted a full brain death examination.

The prerequisites for a brain death examination were carefully verified. Neuroimaging was considered fitting with the depth of coma. His current poor neurologic condition was considered irreversible despite multiple attempts in reducing ICP. At the time of brain death examination, ICP was consistently in the 60s. Patient had not received any barbiturates for ICP control nor paralytic agents or any other CNS-depressant drugs in the
last several days. The neuromuscular blocking agents were administered only at intubation, which was several days ago. Recent laboratory results were reviewed and showed no evidence of any metabolic or respiratory acidosis. Plasma serum sodium level was slightly elevated at 150 mmol/L but not considered a major confounder. Core temperature was 36°C. Systolic blood pressure was 110 mm Hg with continuous vasopressor infusion.

A detailed neurologic examination was then performed. Pupils were 6 mm in diameter and nonreactive to bright light. Corneal reflexes were absent. Oculocephalic reflexes were absent and confirmed by oculovestibular reflexes with no eye deviation toward the cold stimulus (50 cc of ice water was inserted in each ear with a 5-minute interval). No facial movement or grimacing was found to noxious stimuli. Gag reflex was absent, and no cough reflex with two passes of a suctioning catheter was detected. No motor response was noted in the arms. However, in the legs, there was a clear triple-flexion response.

Next, the conditions for an apnea test were evaluated. Patient was not hypotensive. Positive end-expiratory pressure (PEEP) was 5 cm of water, and he was not currently hyperventilated. A prior blood gas showed a pH of 7.45, partial pressure of oxygen (PO$_2$) of 100, and partial pressure of CO$_2$ (PCO$_2$) of 40. The chest x-ray showed diffuse infiltrates but no evidence of profound pulmonary edema. Patient was preoxygenated with 100% of oxygen for 10 minutes. Repeat baseline blood gas now showed a PO$_2$ of 250 mm Hg and PaCO$_2$ of 45 mm Hg. An insufflation catheter was inserted through the endotracheal tube, close to the level of the carina, and 6 liters of oxygen were administered. The ventilator was disconnected, and the patient was closely watched for 8 minutes. No respiratory excursions were noted, and a blood gas was redrawn at 8 minutes. While awaiting the results of the second blood gas, the patient was kept off the ventilator maintaining adequate apneic oxygen diffusion. Return blood gas showed a PaCO$_2$ of 62 mm Hg and a PaO$_2$ of 150 mm Hg. The patient was declared brain dead, and the time of the second blood gas was noted in the medical record and was considered the time of death. No ancillary test was needed. A comprehensive note summarizing the examination was dictated.

The family was gathered in a quiet room and told that the patient had passed away. The meeting with the family involved the charge nurse and a pastor and was largely supportive. The family spontaneously expressed a strong desire for organ donation (the patient’s driving license was reviewed, and a consent for donation was noted). The organ procurement organization (OPO) was contacted for further consultation and explanation. Organ donation was granted. Donation was successful and consisted of kidneys, heart, lungs, pancreas, and liver. The patient’s parents received a thank-you letter explaining their son helped—and, frankly, saved—five patients, two of whom had been on the transplant waiting list for more than 6 years.

Questions

1. What are the major confounders in brain death examination?
A. Single day of propofol infusion for ICP treatment, but stopped 3 hours previous.
B. Pentobarbital infusion for ICP treatment, but stopped 5 days previous
C. Hypernatremia (155 mmol/L)
D. Bladder temperature of 35°C.

The correct answer is B. Barbiturate infusion is a major confounder due to considerably long half-lives (pentobarbital t ½ for up to 48 hours). Brain death examination can proceed only if barbiturate plasma levels are below therapeutic range. Propofol is a quickly eliminated drug and, in absence of severe hypothermia, should not be a major confounder. Mild hypernatremia is expected in many patients who have diabetes insipidus and is not considered a confounder. A plasma sodium level over 160 mmol/L could theoretically impact neurologic examination. In general, core body temperature over 35°C may allow a brain death examination.

2. What conditions would predict aborting an apnea test?
A. Neurogenic pulmonary edema on chest x-ray
B. PEEP of 10 cm of water
C. Prior pulmonary disease
D. Use of vasopressors

The correct answer is B. PEEP of 5 cm may allow apneic diffusion oxygenation, but higher levels before disconnection could cause rapid deoxygenation. Unstable blood pressures, despite the use of vasopressors, may complicate an apnea test and could lead to its discontinuation.

Diagnosis Coding
The underlying problem in this case is traumatic brain injury without mention of skull fracture, which in ICD-9-CM² is coded in the Concussion section. Other variables affecting the code are contusion, no mention of open wound, and more than 24 hours of unconsciousness without ever regaining consciousness prior to death. The code for this condition would be:

851.05 Concussion with cortex (cerebral) contusion without mention of open intracranial wound, with loss of consciousness greater than 24 hours without regaining consciousness.

Brain death should also be coded. Currently ICD-9-CM² indexes Brain death to:

348.89 Other conditions of brain

This is a very nonspecific code, which might cause problems if used alone, and it should be listed as the second code in this case. A separate code for brain death has been requested and, it is hoped, will be approved for use beginning October 1, 2011.

The final diagnosis code list is:

851.05 Concussion with cortex (cerebral) contusion without mention of open intracranial wound, with loss of consciousness greater than 24 hours without regaining consciousness.
348.89 Other conditions of brain

E&M Coding
The patient is seen in consultation by a neurologist for the performance of the brain death determination. The best way to code for this encounter would be to use the total time spent with the patient and family and using the counseling and coordination of care method of coding. For an inpatient who has Medicare, one would need to spend 70 minutes total with the patient and family performing the testing and more than half of that time must be expended discussing the results of the brain death determination and consequences with the family, showing them the imaging studies, and outlining the procedure of organ procurement. The proper code would be 99223 for a Medicare patient, which is the same code used for level 3 initial hospital care. Remember that as of January 1, 2010, consultation codes no longer can be used for Medicare inpatients or outpatients. For a patient who has 3rd party insurance, one would use the code 99255 if 110 minutes total was spent with the patient and family and more than 50% was spent counseling and coordinating care. Code 99254 would be used if 80 total minutes was expended and more than 50% was spent counseling and coordinating care.

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