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Brain Death Determination

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INTRODUCTION

Patient MR was a seventeen year old female involved in a motor vehicle collision which resulted in a traumatic brain injury leading to declaration of brain death. The pathophysiology behind the traumatic brain injury, the concept and use of a brain tissue monitoring device, and the tests used in the declaration of brain death will be explored. When brain death is determined, the patient has the opportunity to become an organ donor. Thousands of people are waiting for organs to become available. This is a huge healthcare concern that may be decreased if the general public is more educated regarding the process of brain death determination. By having a better understanding of brain death, the hope is that there will be more people willing to make the decision to become an organ donor. In order to address the enormous need for organ donors, there has to be an understanding of brain death testing at the cellular level.

CASE PRESENTATION

Per EMS, Patient MR was unresponsive at the scene of the accident with a GCS of three, agonal breathing, and was intubated for airway protection. Upon completing x-rays and full body CT scans, it was discovered that MR had multiple facial fractures, multiple bilateral rib fractures with accompanying hemo-pneumothoraxes which required placement of bilateral chest tubes, severe bilateral pulmonary contusions, left tibia/fibula fracture, left femur and acetabular fractures, scattered small subarachnoid hemorrhages (SAH), global cerebral edema with possible signs of diffuse axonal injury (DAI). Upon arrival to ICU, there was no surgical intervention from a neurosurgical standpoint and the decision was made to place a Licox monitor which measures the brain tissue oxygenation, brain tissue temperature, and intracranial pressure within the white matter of the brain [2]. MR's neurological assessment consisted of size 4mm, sluggish pupils, a weak cough and gag, intermittent corneal reflex, assisting the ventilator, decerebrate posturing of the upper extremities to painful stimulus, and no response to pain in the lower extremities.

CASE PRESENTATION CONTINUED

The severe global edema of the brain occurred due to acceleration-deceleration at the time of the motor vehicle collision, and MR's inability to have adequate respirations to oxygenate the brain sufficiently. The brain accelerates at time of impact and quickly decelerates within the skull causing axonal shearing [8]. This global axonal shearing is seen within the sub-cortical white matter of the brain and is called DAI [7]. Shearing alone can be a mechanism for brain edema but MR also had inadequate breathing and blood loss due to her other injuries which also contributed to the edema. The lack of oxygen directly affects the production of ATP which is necessary for the brain to function normally. Without adequate ATP production the sodium/potassium pump is unable to function correctly and leads to an influx of intracellular sodium which results in edema [8]. Glucose is changed into pyruvate through the glycolytic pathway and then pyruvate enters the Krebs cycle and takes part in the synthesis of ATP molecules [8]. With lack of oxygen pyruvate is unable to move forward into the Krebs cycle and is converted into lactate [8]. Lactate only further stimulates sodium influx into the cell by stimulating the sodium/hydrogen exchange pump [8].

Once the edema occurs blood flow to the brain is inhibited. One mechanism to measure cerebral blood flow is to calculate cerebral perfusion pressure (CPP) [3]. CPP measures the pressure gradient of the mean arterial pressure (MAP) pushing blood through the cerebral circulation against the opposing intracranial pressure (ICP) [3]. For most traumatic brain injury patients, it is recommended to maintain the CPP between 50-70 mmHG [3]. CPP is maintained by increasing MAP and decreasing ICP. MAP is increased by use of vasopressors [6]. In the case of MR, the vasopressor used was norepinephrine. The MAP was also increased by use of 0.9% normal saline boluses within the first twelve hours in the ICU. Mannitol and 3% saline were used in the attempt to decrease ICP below the desired 20 mmHG. Both of these solutions work to move fluid from the intracellular compartment to the intravascular space which in turns decreases edema [8]. As stated earlier a Licox monitoring system was placed and measured brain tissue oxygenation (PbtO2). The Licox monitor itself is a thin, metallic electrode that is inserted into the white matter of brain through a burr hole in the skull as seen in figure one [6]. Treating PbtO2 numbers is often dependent upon institution but generally a PbtO2 maintained greater than or equal to twenty is the desired goal and numbers below twenty are strongly correlated to a poor prognosis or outcome [6]. Maintaining MR's ICP below 20mmHG, CPP at sixty or above, and PbtO2 above twenty was a very difficult challenge that became futile twenty-four hours after admission to the ICU.

Twenty four hours after admission, MR's ICP was ranging from 25-40mmHG and the higher her ICP then the PbtO2 would decrease below 10. Her ICP became less responsive to PRN Mannitol and would only stay below 20mmHG for approximately an hour following administration. During a routine hourly neurological assessment, the bedside nurse found that bilateral pupils were fixed and dilated. MR was still breathing above the set respirations on the ventilator and had a very weak cough. MR was taken to CT scan. The CT scan showed not only increased cerebral edema but also multiple, scattered areas of stroke. Upon returning from CT scan MR became very hypertensive with a systolic blood pressure (SBP) in the high 190's and tachycardia as high as 150's. These elevated vital signs lasted approximately thirty minutes and were quickly followed by bradycardia and hypotension. At this point MR's pupils were fixed and dilated, she had no cough, gag, or corneal reflexes, no response to painful stimulus, and was no longer assisting the ventilator. It was determined that MR met criteria for brain death testing.

BRAIN DEATH TESTING

Brain death is defined as the, "irreversible cessation of all functions of the entire brain, including the brain stem" [9]. In order to determine brain death there are multiple tests that physicians perform. The brainstem reflexes which include oculocephalic, oculovestibular (cold calorics), corneal, cough, gag, and pupillary light response are usually tested first [1]. The oculocephalic reflex tests for CN III, IV, and VIII and also tests the functionality of the pons and midbrain [1]. The oculocephalic reflex test, also known as doll's eyes, is performed by turning the patient's head side to side and observing the movement of the eye [1]. The Oculovestibular reflex tests for CN III, IV, VI, and VIII and also tests the functionality of the pons and midbrain [1]. This test is completed by irrigating each ear canal with 50mL of ice water and observing for eye deviation [1]. The corneal reflex tests for CN III, V, VII and the functionality of the pons [1]. This test is completed by lightly brushing the cornea with a cotton swab and assessing for blinking of the eye [1]. The cough and gag reflex tests for CN IX, X, and functionality of the medulla [1]. The gag is tested by stimulating the deep posterior pharynx with oral suction, and the cough is tested by deep endotracheal suctioning [1]. The pupillary reflex tests for CN II, III, and the functionality of the midbrain [1]. This is completed by darkening the room and then shining a light into each eye and assessing for constriction of the pupil [1]. MR did not have any response to any of the above mentioned tests.

Another brain death test often performed is the apnea test. This tests involves obtaining a baseline arterial blood gas (ABG) and then removing the patient from the ventilator and placing a cannula into the endotracheal tube at 10 l/min [5]. The apnea tests usually lasts 8-10 minutes in duration and during this time the physician is assessing respiratory movement of the chest or abdomen [9]. After approximately 8 minutes another ABG is obtained and the practitioner is looking for either a Pco2 above 60 or an increase of the Pco2 by 20mmHG from the baseline ABG [9]. If there is not respiratory movement and an increase in the Pco2 then the apnea test is considered positive and this supports the clinical diagnosis of brain death [9]. The apnea tests shows that the brain is no longer functioning because the rapid increase of CO2 would cause the respiratory centers in the medulla to initiate breathing in order to clear the CO2 [5]. Unfortunately MR was not a candidate for apnea testing due to her extensive pulmonary injuries.

Not being able to perform the apnea test, the neurosurgery team decided to complete a nuclear flow study. This type of tests determines if there is blood flow to the brain. The patient is placed under a collimator with cameras that are anterior and parallel to the patient's face [4]. A radiopharmaceutical bolus is administered to the patient via an IV and pictures are taken to capture the blood flow up to the brain [4]. If there is blood flow to the brain, the bolus can be seen going up the carotids into the anterior and middle cerebral arteries [4]. When there is no blood flow to the brain, the bolus is seen through the carotids but stops at the base of the skull [4]. The bolus is not seen within the brain due to the increased intracranial pressure exceeding CPP which is supplied by the internal carotids [4]. The blood flow to the external carotids remains intact so the bolus is seen circulating to the face and scalp [4]. Often the flow to the face is increased which results in the "hot nose" sign as seen in figure two [4]. Upon completion of the nuclear flow study, it was determined that MR did not have blood flow to the brain. In combination with the brain stem function test and the results of the nuclear flow test, MR was declared brain dead and her family made the decision to proceed with the organ donation process.

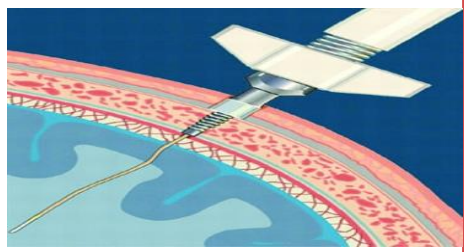

Figure 1: Illustration of Licox in place within brain parenchyma [6].

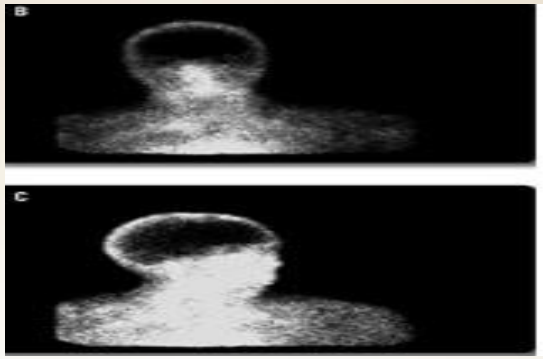

Figure 2: Nuclear flow study that illustrates no cerebral blood flow and the classic "hot nose" sign [1].

NURSING IMPLICATIONS

This case highlights the important role that nurses play in the care of a patient with a traumatic brain injury (TBI) that leads to brain death. It is imperative that nurses be aware of the mechanism of injury leading to the TBI and how this can affect the response of the brain. The bedside nurse needs to also be aware of what the goals of therapy are and how to measure these goals. Therefore, the nurse needs to be educated on not only the monitoring equipment but also the meaning of the numbers produced by that equipment. Nurses are integral in the early recognition of a patient that is progressing to brain death and this early recognition leads to more stability of the patient and increased organ function during organ procurement [1]. The brain is very complex and often the signs and symptoms can be confusing, and it is imperative that the bedside nurse works in close conjunction with the neurosurgery team to determine what the clinical signs mean for the patient's care. The nurse needs to pay close attention to even the little changes in a patient's exam because when it comes to the brain these changes may mean future complications with the patient's condition.

CONCLUSION

Patient MR was involved in a motor vehicle collision that resulted in a traumatic brain injury. By understanding the brain's response to an acceleration-deceleration injury the physicians and nurses were more cognizant of the complications that could occur. Due to the patient's exam and scans, a Licox monitoring system was placed to have a real time picture of the status of the brain at a cellular level. With this vital information, interventions were administered at the first indication that the cerebral edema was worsening. Unfortunately for this patient, the brain injury was too severe and she progressed to brain death. With the declaration of brain death, the patient was able to become an organ donor and save the lives of numerous individuals on the national donor list.

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ADDITONAL RESOURCE

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