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CASE REPORT

A Case Report of Maternal Cerebral Hemorrhage in Preterm Pregnancy

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ABSTRACT

Intracerebral hemorrhage is a rare event during pregnancy that can result in serious morbidity and mortality for the mother and infant. In this article, we describe a case involving a multiparous woman at 34 weeks gestation who presented with a 5-cm intracerebral bleed. Within 2 hours of presenting, she underwent an emergency cesarean and craniotomy to remove the hematoma. The woman's altered mental status fully abated, and she was discharged with minimal neurologic deficits. The newborn was born healthy.

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Intracerebral hemorrhage (ICH) is a rare event during pregnancy that can result in serious morbidity and mortality for the mother and infant. In the Nationwide Inpatient Sample for survey years 1993 through 2002, pregnancy-related ICH occurred in 6.1 women per 100,000 births (Bateman et al., 2006). At the time of birth, ICH occurred in only 0.002% of women in the general population, and postpartum ICH incidence was greater than the antepartum rate (Bateman et al., 2006). Salonen Ros, Lichtenstein, Bellocco, Petersson, and Chattingius (2001) suggested that the risk of circulatory disease events increased within days surrounding birth, including the time before birth. In two retrospective analyses of ICH and pregnancy, Takahashi et al. (2014) and Yoshimatsu et al. (2014) showed a strong correlation between cerebrovascular disease (CVD) and ICH, indicating the need to identify symptomology and diagnosis in a timely manner.

For the nonpregnant population, six major risk factors for ICH have been identified. These include advanced age, male sex, race, hypertension, high alcohol intake, and low serum cholesterol (Zazulia & Diringer, 2011). Rates of incidence are almost doubled for African American and Hispanic patients comparatively. Asian patients have a significantly greater incidence, almost double that of African American and Hispanic patients, and chronic hypertension is the leading cause in over half of all ICH cases (Zazulia & Diringer, 2011).

Factors during pregnancy that significantly increase the risk for intracerebral hemorrhage include hypertension, coagulopathy, advanced maternal age, race, tobacco and cocaine use, and preeclampsia and eclampsia (Bateman et al., 2006). There are other causes of pregnancy-related ICH including arteriovenous malformation, aneurysm, Moyamoya disease, and other CVDs (Takahashi et al., 2014). In women affected by pregnancy-related ICH, cerebrovascular malformation was noted in 7.1%. The demand of pregnancy on the body changes the fine balance between hemodynamics, blood volume, and clotting factors (Salonen Ros et al., 2001). The stress of these changes can cause significant cerebrovascular events without regard Q1 to underlying CVD.

The pathophysiology of ICH is complex regardless of origin. After an ICH is sustained, there are myriad consequences for brain tissue. Hematoma expansion is a significant factor in continuing tissue destruction. This results in mechanical injury due to compression and tissue shifts in the intracranial cavity (Zazulia & Diringer, 2011). Hematoma volume and expansion are significant predictors in 30-day functional

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outcomes and mortality. ICH also causes obstructive hydrocephalus, another important predictor in 30-day functional outcomes. Hydrocephalus is caused by two mechanisms, initially by extravasation of blood into the ventricular system, which impairs the flow of cerebral spinal fluid, creating obstructive hydrocephalus. Later, the blood and debris block the arachnoid villi and impair reabsorption of the cerebral spinal fluid (Testai & Aiyagari, 2008). Hydrocephalus causes increases in intracranial pressure (ICP), creating neurologic deficits.

Significant brain tissue injury occurs with increased ICP from cerebral edema. Cerebral edema occurs in three phases after ICH is sustained. The first phase is within hours of the hemorrhage. Edema volume increases by 75% in the first phase of cerebral edema after ICH. In this phase, the clot retracts and causes extrusion of osmotically active proteins into the surrounding white matter. The second phase occurs within the first 2 days after an ICH. In this phase, a significant inflammatory response occurs that causes a disruption in the blood–brain barrier, significantly worsening the edema. The third phase occurs after the first 2 days and is significant because of the hemolysis that leads to hemoglobin-induced neuronal toxicity. This can lead to apoptosis and necrosis in the adjacent brain tissue, causing additional damage. Additional damage is caused by ischemia in the area around the clot due to increased ICP that can compress the microvasculature. There are several other factors involved in secondary brain damage after ICH. These include biochemical, inflammatory, cellular, and molecular mediators of brain damage. These factors are important to consider in management of brain damage after ICH (Testai & Aiyagari, 2008).

The symptomology of cerebellar ICH includes headache, altered mental status, nausea, vomiting, and vision changes, including double vision (Nyquist, 2010). It can include vertigo and the inability to walk without associated hemiparesis (Aghi, Ogilvy, & Carter, 2012). After initial presentation, patients experience a significant and rapid decline in level of consciousness as the intracerebral pressure increases because of compression of the brain stem. This can lead to coma, herniation, and death (Nyquist, 2010). This rapid decline must be triaged and managed in a timely and appropriate manner to prevent significant morbidity and mortality.

Case Study

The focus of this case study was a 28-year-old Hispanic woman, gravida two, with one term birth, no preterm births or abortions, and one living child, who was pregnant with a single fetus at $34\frac{1}{7}$ weeks gestation. Her first pregnancy was uncomplicated, and she gave birth by cesarean. Her current pregnancy was medically and obstetrically uncomplicated except for a false positive quadruple screening test result and referral to maternal–fetal medicine for Level 2 ultrasonography, the results of which were normal. The woman had no significant medical history and no history of head trauma the day of presentation or in the preceding days. She did not smoke and reported no illicit drug use.

On the day of admission, while shopping with her husband, the woman complained of headache, nausea, and dizziness. Because of the sudden onset of symptoms, her husband proceeded to the immediate care center associated with the hospital. On arrival at the immediate care center, the woman was unable to ambulate. A rescue squad was called, and she was transferred to the hospital, a Level 1 trauma center. This hospital is the only such facility in the county and is a Level 2 obstetric urban care facility, caring for neonates 30 weeks gestation and older. The obstetrics unit has an average of 2,000 births annually.

The woman presented to the labor and delivery unit complaining of a severe headache, nausea, and vomiting. Her mental status was clearly altered upon initial assessment. Although she was able to answer simple questions and perform simple commands, she was unable to answer questions about events that happened immediately before her admission, but she was aware of who she was and where she was. She kept reiterating how terribly her head hurt and complained of significant sensitivity to light.

She was placed on continuous electronic fetal monitoring (EFM) and noted to have a Category 1 fetal heart rate tracing with a baseline of 120 beats per minute, moderate variability, accelerations present, decelerations absent. She was having

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