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Briefly Legal: Was Baby's Brain Damage Caused by Mismanagement of Preeclampsia?

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A 700-g, 27 1/7 weeks' gestation female infant is delivered by cesarean delivery to a 28-year-old, gravida 1, para 0 morbidly obese woman. To determine if the woman had gestational diabetes, her obstetrician ordered a glucose tolerance test, but because the woman vomited the glucola, the evaluation for diabetes was not performed. ***The plaintiff obstetrician was critical of the treating obstetrician for not making a greater effort to resolve the patient's glucose tolerance, including HbA1c and serial urine glucose testing. He stated it was standard to obtain a measure of glucose tolerance, and that could be done in several ways-beyond which he was required to tell the patient its importance, especially because of the patient's morbid obesity. The obstetrician retained by the defense explained that it was not standard to repeat the glucose tolerance testing or to order an HbA1c automatically. He further pointed out that it was the patient's fault that the test was not repeated.***

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Ten days prior to delivery, the woman called her obstetrician because she was experiencing severe right upper quadrant/epigastric pain. ***The woman stated on her deposition that her obstetrician told her to try to calm down and that it was her choice to go to the Emergency Department (ED) or not.*** **The plaintiff expert pointed out that this approach was negligent, given the risk of HELLP syndrome (Hemolysis, elevated liver enzymes, low platelets).** Because the pain was severe, she went to the ED. She rated her pain in the ED 8/10. Her blood pressure (BP) was 146/87 mm Hg, and her complete blood count (CBC) was normal except for a platelet count of 145/uL. Her urinalysis demonstrated +3 proteinuria. The documentation in the ED did not include anything regarding vision changes, hyperreflexia, headaches, or edema. An ultrasound was done to rule out gallstones. ***The plaintiff obstetrician pointed out that her elevated blood pressure, abdominal pain, and proteinuria should have concerned the ED physicians about preeclampsia, especially that leading to HELLP syndrome, which may present without very high blood pressures and even normal blood pressures. He also pointed out that a fetal ultrasound should have been performed to assess fetal growth and amniotic fluid level. He further pointed out that the ED physician should have sought an obstetrical consult and have ordered a fetal heart rate tracing. He considered that the failure to consult an obstetrician entering the ED was a gross deviation from the standard of care in a pregnant patient entering the ED.***

The abdominal ultrasound did not show gallstones. ***Plaintiff expert pointed out that even had it had, preeclampsia would not have been excluded.*** The ED physician diagnosed gastritis and told the woman to avoid fat in her food and eat small volumes of food. He then discharged her home without an obstetrical consult. ***The plaintiff obstetrician explained that right upper quadrant or epigastric pain was probably due to periportal and focal parenchymal necrosis, hepatic cell edema, Glisson's capsule distension, or a combination represented a potentially catastrophic situation. He also pointed out that the ED at that hospital had a policy that said pregnant patients over 20 weeks' gestation with findings consistent with, but not necessarily diagnostic of, preeclampsia must be transferred to Labor and Delivery.***

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Further, the plaintiff obstetrician pointed out that the diagnosis of preeclampsia, as recommended by ACOG, required a 2nd reading at least 4 hours later. However, he added that if the signs and symptoms are compelling enough, the diagnosis of preeclampsia must be made on a presumptive basis. The hospital policy stated that if a diagnosis of preeclampsia were made, then some or all of the following steps should be taken, depending on the obstetrician's evaluation: hospitalization, administration of at least 24 hours of intravenous magnesium sulfate and steroids, seizure prevention, serial platelet counts and liver function tests, close fetal surveillance, including frequent fetal heart monitoring and biophysical profiles, assessment of placenta sufficiency, management of maternal hypertension and potential non-emergent delivery depending on the evaluation. ***Plaintiff expert pointed out that, at a minimum, the patient should not have been discharged as it represented a significant violation of their own policies and procedures.***

Five days after the ED visit, the woman was evaluated by her obstetrician. She stated in her deposition that she shared with him the paperwork from the ED. She shared that her abdominal pain continued after the visit and was currently present despite dietary changes. She had gained 18 pounds in the past four weeks. The treating obstetrician did not comment if the weight gain was due

to excessive fluid retention. One blood pressure was performed in the obstetrician's office, and it was 126/64 mm Hg. ***The plaintiff obstetrician pointed out that the obstetrician needed to be concerned about preeclampsia and should have rechecked her urine and obtained multiple blood pressures 10 minutes apart. According to the mother, the treating obstetrician purportedly told the patient that there was nothing to worry about. The plaintiff obstetrician strongly disagreed with this assessment.***

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Five days after this visit with her obstetrician, the patient sustained a grand mal seizure at home. Her partner heard a thump in the middle of the night, which awakened him. He observed that she had fallen off the bed and was having a seizure. Since the father of the baby was asleep, the duration of the seizure was not clear. He called the paramedics. Upon examination by the paramedics, the patient was postictal and had a BP of 178/144 mm Hg. She was 27 1/7 weeks gestation. ***The plaintiff experts pointed out that this situation could have been avoided if the standard of care had been met in evaluating the patient for preeclampsia. The plaintiff experts stated that eclamptic seizures were dangerous to both the woman and fetus. He noted that affected women might develop long-lasting cognitive issues. The literature on eclamptic seizures showed that during the seizures, the fetus sustains profound fetal bradycardia that may last up to 20 minutes as a measure of the severity of potentially injurious fetal hypoxia. In addition to acute events, studies on women with eclampsia have found increased white matter lesions demonstrated by magnetic imaging resonance (MRI) with lasting psychological and cognitive effects.***

The patient was then treated with midazolam by the paramedics. She arrived at an ED 35 minutes afterward and was given magnesium sulfate. The fetal heart rate was 208 beats per minute. The woman had a computerized brain tomography (CT) scan that showed brain swelling in the parietal-occipital region but no acute bleed. Over the next 2 hours, the baseline fetal heart rate decreased to 160 bpm. Most of the fetal heart rate tracing was not interpretable except for a 3 minute period that showed moderate variability. The mother's labs were notable for elevated liver function tests and a low platelet count; she was diagnosed with HELLP syndrome. Three hours after arriving in the ED, the baby was delivered by cesarean section. Immediately after delivery, the mother sustained another grand mal seizure followed by a cardiac arrest while the surgery was still underway. After being revived in the operating room, she was sent to the intensive care unit. After weeks of a stormy course in the intensive care unit, the mother eventually recovered except for the complication of bilateral retinal detachment.

The neonatal resuscitation team was present at the delivery and provided positive pressure ventilation for 15 seconds. The neonate had Apgar scores of 7[1] and 9[5]. She was appropriate for gestational age. Her weight was 700 grams, her head circumference was 23 cm, and her length was 31 cm. She had a normal physical examination, consistent with a gestational age of 27 1/7 weeks. The arterial cord gas had a pH of 7.18, a pC02 of 60 mm Hg, and a base excess of -7.5, and the venous cord gas had a pH of 7.15, a pC02 59, and a base excess of -9.7. ***Much discussion ensued among the experts about the cord gases. The defense said that the maternal seizure had no impact on the fetus because the cord gases were normal. The defense pointed out that the base deficits were inconsistent with a hypoxic-ischemic insult because the base excess was -7.5 and -9.7. The plaintiff expert pointed out that the seizure was 3 hours earlier than delivery and that no challenges, such as uterine contractions, were ongoing. The seizure was then, the cord gases long after recovery were now.***

Further, the plaintiffs stated that the difference in the cord pHs was too narrow (0.03), pointing out that the average difference between the cord venous and arterial pH is normally 0.07. Thus a difference <0.04 indicates that the gases were likely from the same vessel or the laboratory made a mistake. The plaintiff added that the venous cord deficit was worse than the arterial gas, which is physiologically not possible, another point in favor of the lack of validity of the cord gases. The plaintiff also noted that the cord gases should have been normal (7.35-7.45) because there was no period of labor or ongoing stress after the seizure, which with all reasonableness was accompanied by a significant amount of uterine activity, now dissipated. The plaintiff stated that the cord gases quoted in the literature refer to vaginal deliveries after labor and do not refer to infants born by cesarean section without a period of labor well after an obvious hypoxic-ischemic event. He noted that the pH was too low, the $pC0_2$ too high, and the base excess too low for a cesarean-sectioned neonate born without a period of labor.

The defense quoted the American College of Obstetrics and Gynecology (ACOG) "essential criteria" from the 2003 Manual, Neonatal Encephalopathy and Cerebral Palsy that must be met to define an acute intrapartum event sufficient to cause cerebral palsy. These "essential criteria" were: **1) evidence of metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit ≥ 12 mmol/L) 2. Early-onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation 3. Cerebral palsy of the spastic quadriplegic or dyskinetic type 4. Exclusion of other identifiable etiologies, such as trauma, coagulation disorders, infectious conditions, or genetic disorders.** ***The plaintiff experts pointed out that the "essential criteria" being quoted were not based on scientific literature but rather an attempt to protect obstetricians. He pointed out that specific evidence showed that the "essential criteria" of the 2003 ACOG Monograph was to impede a parents' understanding of the event(s) leading to their child developing a brain injury. The most obvious distortion of the scientific literature is***

on view in the section requiring a pH of ≤ 7.0 as one of the "essential criteria." The literature quoted in the Monograph does not support this position. The plaintiff experts also pointed out that the criteria for hypothermic intervention for babies born after an acute intrapartum hypoxic event severe enough to cause cerebral palsy were very different. Thus, the "essential criteria" do not pass scientific muster, a fact underscored by the fact that it was successfully defeated in legal challenges as not holding scientific credibility and therefore not allowed to be presented in a court of law. **(1-3)**

Ultimately the plaintiff expert pointed out the "essential criteria" in the 2003 edition were not included in the follow-up ACOG Monograph (Neonatal Encephalopathy and Neurologic Outcome) published in 2014. Additionally, these "essential criteria" that the defense quoted were criteria developed to assess term and near-term infants, not preterm neonates. The defense argued that the Apgar scores showed that the neonate did not suffer a hypoxic-ischemic insult. The plaintiff pointed out that the insult, the maternal seizure, occurred hours before the delivery, and the fetus had recovered from a cardiovascular standpoint, although neurologically injured. Furthermore, Apgar scores are not a valid measure of intrapartum hypoxic-ischemic insult.

The child's CBC revealed a white blood cell (WBC) of 5.5 u/L, a hematocrit of 38.4%, and a platelet count of 87 u/L. The neonate had several low blood glucose values that were timely treated with boluses of glucose. A bolus of normal saline was given immediately after birth. The first blood gas was drawn between 1.5 hours after birth and had a base excess of -6.6. The first lactate level was drawn at 4 hours, and it was 5 mm, and it remained elevated for the next 24 hours -- ***incurred during the maternal seizure, caused an elevation in lactic acid, and it gradually entered the circulation and then gradually dissipated through the circulation***. The neonate was briefly intubated for respiratory distress syndrome (RDS) and received surfactant. After several weeks of total parenteral nutrition, she eventually fed successfully. She developed cholestatic jaundice, apnea of prematurity, and a medically treated patent ductus arteriosus. She had several courses of antibiotics, but sepsis evaluations were negative. Her liver function tests and creatinine levels were all normal. ***The defense argued that these normal results ruled out a prior hypoxic-ischemic insult. The plaintiff expert disagreed and maintained that with an acute insult, lactic acid is more likely to be normal and inconsistently and unpredictably elevated even with a subacute or chronic insult.*** The neonate's brain ultrasound was negative; magnetic resonance imaging (MRI) was not performed during the admission.

The neonate was discharged at 75 days. On follow-up, she developed hypotonic cerebral palsy and severe developmental delays. MRIs were performed at 2 and 4 years of age. ***The plaintiff neuroradiologist read the MRIs as a combination of PVL types 2 (non-cystic with gliotic scarring), and 3 (diffuse with decreased myelination with associated injury of the axons) and mild ventriculomegaly. The plaintiff experts contended that the MRIs were consistent with a hypoxic and or ischemic event. However, because imaging was not done until after the neonatal period, the timing of the injury was not possible. The defense radiologist interpreted the MRIs as being normal.*** At age 5, the treating neurologist thought that the child had severe developmental delays and hypotonic cerebral palsy. ***The neurologist retained by the defense maintained that the child did not have cerebral palsy but was autistic. The plaintiff neurologist pointed out that neither he nor the treating neurologist thought she showed any evidence of autism.***

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The obstetricians, neonatologists, and neurologists whom the plaintiff's attorney retained were questioned about the cause of the child's adverse outcome. T***he plaintiff experts maintained that the adverse outcome was secondary to the hypoxic-ischemic event incurred during the maternal seizure. The blood was preferentially transferred to her vital organs (heart, brain, and adrenals) at the expense of other organs and tissues, including the uterus and placenta. The mother's brain showed cerebral edema, and her retinas were detached, underscoring the profound insult during the seizure. The fetal tachycardia underscored the stress that occurred during the seizure, an improbable development for anything other than recovery from a very severe hypoxic-ischemic insult. The defense opined that the neonate's adverse outcome was secondary to her prematurity and an "unknown genetic predisposition for developmental disorders and autism." The plaintiff experts strongly disagreed and called the defense's explanation of events "concocted" They pointed out that none of the treating physicians, those who directly cared for the child and who were unfettered by the need to protect the physician, opined or diagnosed a genetic issue or autism. Several metabolic and genetic tests, including microarray analysis, were performed after the lawsuit because the defense offered a metabolic or genetic basis as a probability. Further, they pointed out that all tests were normal. The defense further opined that around 80% of neonates born at this gestation were developmentally abnormal on follow-up examinations. The plaintiff disagreed and pointed out that unless anomalies, asphyxia, intrauterine growth restriction, multiple gestations, infection, or a defined problem after birth were involved, most 27 week infants did fine.***

The plaintiff experts pointed out that by not diagnosing preeclampsia, not only was the fetus compromised during the mother's seizures, but the mother needlessly suffered and developed long-term vision problems. They maintained that all adverse consequences would have been prevented by the timely diagnosis and management of even uncertain preeclampsia. Intervention may have never been necessary, and "rescue" was likely never to be necessary. The plaintiff experts pointed out that the fetus was deprived of antenatal steroids and magnesium sulfate by failing to diagnose

preeclampsia in a timely manner. Such timely and effective care would have avoided the need to rescue—delivery of the baby by emergency cesarean section at 27 1/7 weeks gestation. A course of antenatal steroids would have reduced the probability of RDS, PDA, and PVL; a 24-hour course of magnesium sulfate would have decreased the risk of cerebral palsy by 50%. Moreover, had preeclampsia been diagnosed, probably a significant prolongation of the pregnancy would have occurred. Also, had diabetes been diagnosed, it would have pointed to the need for close surveillance of blood pressures early on in the pregnancy because 30-40% of women with gestational diabetes develop preeclampsia.

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Discussion

General

Hypertensive disorders are the most common complications of pregnancy, with an incidence of about 10% of pregnancies worldwide. The incidence of preeclampsia has increased by 25% in the United States during the past two decades. Preeclampsia is a leading cause of maternal and perinatal morbidity and mortality. This disorder causes a significant increase in maternal, fetal, and neonatal mortality and morbidity. Hypertensive disorders of pregnancy are major contributors to prematurity. While prevention of hypertensive disorders of pregnancy has been an elusive target, early detection and treatment with safe and effective pharmacologic therapies effectively optimize the outcome for the pregnant woman and fetus.

The increased incidence of preeclampsia in the United States is likely related to the higher prevalence of predisposing disorders such as hypertension, diabetes, and obesity, the delay in child-bearing, and the use of assisted reproductive technologies with their associated increase in multifetal gestation. The global impact of preeclampsia is profound, with short- and long-term effects on both the mother and infant. Maternal pre-existing conditions for preeclampsia include nulliparity, multifetal gestations, preeclampsia in a previous pregnancy, chronic hypertension, pregestational diabetes, gestational diabetes, thrombophilia, systemic lupus erythematosus, prepregnancy body mass index greater than 30, antiphospholipid antibody syndrome, maternal age 35 years or older, kidney disease, assisted reproductive technology, and obstructive sleep apnea. Three to 7% of preeclamptic women have gestational diabetes. Preeclampsia affects multiple organ systems and may manifest with a wide variety of symptoms. The cause is unknown.

Classification

Preeclampsia is a disorder of pregnancy-associated with new-onset hypertension, which occurs most often after 20 weeks of gestation and frequently near term. Although often accompanied by new-onset proteinuria, hypertension and other signs or symptoms of preeclampsia may present in some women in the absence of proteinuria.

Gestational hypertension is defined as systolic BP of 140 mm Hg or more or a diastolic BP of 90 mm Hg or more, or both, on two occasions at least four hours apart after 20 weeks of gestation in a woman with a previously normal BP.

Eclampsia is the convulsive manifestation of the hypertensive disorders of pregnancy.

Chronic hypertension is hypertension that predates pregnancy.

HELLP syndrome is an acronym for the presentation of **h**emolysis, **e**levated **l**iver enzymes, and **l**ow **p**latelet count that may develop in a severe form of preeclampsia.

Management

The cornerstones of management of a pregnant woman with preeclampsia are delivery or close antenatal observation, seizure prophylaxis with magnesium, and control of hypertension. Delivery always benefits the pregnant woman and not the fetus, except when severe uteroplacental insufficiency or intrauterine growth restriction IUGR) are present. The timing of delivery is determined by the severity of the disease and gestational age. Patients with mild preeclampsia may be observed and delivered, preferably vaginally, between 37-40 weeks' gestation. Of note, the 2019 ACOG Practice Bulletin 202 recommends expectant management of preeclamptic women unless they demonstrate "severe features," which the woman in this case report did not have. Thus, national guidelines would support the patient having been followed expectantly). However, severe preeclampsia mandates delivery at any gestational age. Prolongation of antenatal days may be considered to allow a course of antenatal corticosteroids to help with pulmonary maturity, reduction in severe intracranial hemorrhage, cystic periventricular leukomalacia, and a course of magnesium sulfate for neuroprotection.

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Consequences of eclamptic seizures

One of the severe complications of preeclampsia is maternal seizures during pregnancy. Prolonged generalized tonic-clonic seizure activity during pregnancy has been shown to

cause maternal acidosis, hypoxia, and brain trauma, including hemorrhage. Abruptio placenta is common after a prolonged seizure, occurring in 20–50% of affected women. Convulsive seizures are also dangerous for the fetus as the fetal heart rate slows during and for up to 20 minutes after a maternal convulsion, suggestive of fetal hypoxia. In addition to acute events, recent studies have found that women with a previous diagnosis of eclampsia have increased white matter lesions and lasting psychological and cognitive effects. Thus, maternal convulsions can acutely cause trauma to both the pregnant woman and the fetus and have long-lasting negative consequences.

Fetal consequences

Because of the accompanying impairment of uteroplacental blood flow, complications include fetal growth restriction, oligohydramnios, placental abruption, and non-reassuring fetal status.

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Short-term effects on the neonate

Neonates exposed to preeclampsia have an increased risk of respiratory morbidity, especially in preterm and late preterm infants. The pulmonary risks include RDS, need for respiratory assistance, and bronchopulmonary dysplasia. Additionally, feeding difficulties are more common for those infants born to mothers with preeclampsia. Magnesium sulfate, administered antenatally for prophylaxis of maternal seizures and neonatal neuroprotection, may cause decreased neonatal intestinal motility, necessitating a slower-than-normal introduction of enteral feedings. It is unclear if necrotizing enterocolitis is more common or not.

Hematologic abnormalities are often found in infants born to mothers with preeclampsia. Transient neutropenia and thrombocytopenia are often found, particularly in small for gestational age infants. Neutropenia may occur in up to 50% of infants born to mothers with preeclampsia. The severity is directly related to the severity of preeclampsia. A proposed mechanism for neutropenia is decreased neutrophil production secondary to a placental inhibitor factor. Neutropenia mainly affects the smaller and younger neonates. Thrombocytopenia occurs mainly in IUGR neonates, and it is generally found in the first 72 hours and resolves by ten days. The data on the mechanism of thrombocytopenia point to decreased platelet production from fetal hypoxia, microangiopathic sequestration, and destruction in the placental thrombi. Increased numbers of nucleated red blood cells and polycythemia have been found depending on the level of chronic fetal hypoxia. Maternally administered magnesium may cause respiratory depression at birth. Severe intraventricular hemorrhage and periventricular leukomalacia are less common in premature infants born to preeclamptic mothers than mothers without preeclampsia**.**

Long-term impact

The long-term impact on children exposed to preeclampsia with respect to growth, development, and health status is not established. Some authors suggest that exposure to a stressful intrauterine environment could accelerate organ maturation and improve the outcome of preterm infants, while others have not observed this. Follow-up studies for growth, development, and health status are needed to understand the potential repercussions of hypertension in pregnancy.

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