

Factitious Microcephaly and the Timing of Fetal Neurological Injury

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Case resume:

The patient is a 27-year-old primigravida. She is 5'1 ½ inches tall and weighs 190 pounds at the outset of pregnancy. The prenatal course is uneventful, during which the patient will gain 34 pounds; diabetes and GBS screening are negative. Periodic ultrasound examinations are consistent with normal growth, head size, and amniotic fluid volume. At 40.0 weeks gestation, she arrives in the early evening at the hospital complaining of contractions for two days. The fetal monitor reveals frequent fetal movements and a stable baseline rate with recurrent contractions. Vital signs are normal. A pelvic exam reveals the cervix to be 1.5 cm dilated, 100% effaced, with the head at a -1/-2 station with bulging membranes

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Four hours after admission, the cervix is 2.5 cm dilated. Artificial rupture of the membranes (AROM) reveals straw-colored fluid. The patient is given Demerol 125 mg. / Phenergan 50 mg. IM for pain, and the fetal monitor is removed overnight for 7 hours. The first dose of Ampicillin 2 gm. IV is given, to be repeated at 1 gm. IV q4 hours until delivery.

Shortly after the monitor is reapplied in the morning, the fetus suffers a sudden drop in heart rate to the 70-80s with a very protracted return to baseline over about 10 minutes. Within 30 minutes, the patient has a large emesis, and the fetus shows another prolonged deceleration. Shortly after the deceleration, the monitor is removed. Without obtaining the patient's BP or securing the recovery of the FHR pattern, the patient is allowed up to the bathroom. A gush of blood from her vagina pools on the floor when she stands up. She then walks toward the bathroom and faints in her husband's arms. She is returned to bed, and the monitor is replaced for about 12 minutes revealing a slow recovery of the FHR from a prolonged deceleration. The mother's heart rate (MHR) rate is 140, and she is diaphoretic. Following the episode, there are 7 to 8 contractions in 12 minutes, a frequency, along with the bleeding that required consideration of placental abruption. With the cervix still 4 cm. dilated, the patient is taken to the operating room in anticipation of a cesarean section. She is provided oxygen with a rebreather mask and maintained on her right side. The external fetal monitor is replaced with a fetal scalp electrode

(FSE), revealing an FHR of 110-118. Contractions are every 2 minutes, lasting 30-40 seconds, and mild-moderate intensity. The MHR is 124 and the BP 125/70. The FHR is stable at about 120, rising slowly to about 140 with obvious accelerations but absent variability and no decelerations.

In the inappropriate belief that the tracing had recovered, the cesarean section is abandoned to await progress in labor with plans to start oxytocin drip irrespective of the frequent contractions. Shortly thereafter, the patient is feeling the urge to push with contractions. Examination reveals the cervix has dilated rapidly to 9.5 cm with the head in the OP position. She is given analgesia to combat the strong urge to push. After 1.5 hours, the cervix is fully dilated. Believing the fetal tracing to be satisfactory apart from a “slight, uncomplicated fetal tachycardia,” the patient is encouraged to start pushing with each contraction despite very frequent contractions. The station of the presenting part is +1, and the position is OA. There is no mention of molding or caput. After 1.5 hours of pushing, 2nd stage, with the head in the OA position and still at +1 station, the physician applies mid-forceps. The FHR reveals a profound deceleration during the forceps application to about 110 bpm. The baby delivers 2 minutes later. The indication for the procedure is the concerning FHR changes and prolonged 2nd stage of labor.

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There is no pediatrician present at delivery. At birth, the male infant weighs 3085 gm. (25-50th percentile). Meconium is present. The Apgar scores are 2, 7,8, and 10 at 1,5,10, and 15 minutes. The initial heart rate is about 70 but greater than 100 by 1 minute of age. The infant is floppy. Resuscitation includes suction, stimulation, and positive pressure ventilation with bag-and mask x 5-6 min. He takes his first breath and cry at less than 1 min. He sustains respirations at about six minutes of age. The baby's head is found to be persistently turned to the right. The right corner of the mouth is also drooping, likely resulting from facial nerve palsy. There are prominent forceps marks and bruising across the right side of the face, including the eyelids and the cheek. The baby's length is 50 cm. The head circumference is 33 cm. The ponderal index is 2.47

Neither umbilical cord gases nor placental examination is obtained. The first pH at one hour reveals a pH of 7.24 with a base deficit of 5.4. At 8 hours of age, the baby is found to be jittery, secondary to hypoglycemia treated with 10% dextrose. Seizures begin at 15-16 hours and are treated with phenobarbital and Dilantin. CBC reveals a hematocrit of 42.4%, platelets of 250,000,

NRBCs 21. There are indicators of renal (creatinine) and hepatic (LDH) abnormalities and persistently elevated serum lactate. At 20 hours, the baby required intubation – attributed to respiratory depression secondary to the aggressive treatment with anticonvulsants; post-intubation arterial blood gases were normal with PaCO₂ of 35 mmHg. At 23 hours of age, he is transferred to a regional facility with seizures and facial asymmetry, including a right facial droop.

The following day a portable cranial ultrasound reveals effacement of the cerebral ventricles and an overall mild diffuse increase in parenchymal echogenicity consistent with cerebral edema, which is likely based on hypoxic-ischemic encephalopathy - follow up is recommended within 72 hours. There is no mention of microcephaly.

Laboratory values reveal abnormalities in the liver (LDH) and renal (creatinine) function.

On DOL 3, an EEG is markedly abnormal consistent with severe cerebral/cortical injury. A spinal tap reveals xanthochromic (blood-tinged) fluid. At DOL 5, a CAT scan of the head reveals effacement of the lateral ventricles with diffusely decreased attenuation throughout the cerebrum with increased attenuation differences in the cerebral hemispheres (a prolonged partial injury) and the brain stem cerebellum and inferior thalamus (an acute injury). These results give rise to one of two clinical scenarios: a) An acute sentinel event of total asphyxia with severe cerebrum, brainstem, cerebellum, and hypothalamus hypoxia associated with residual subacute, prolonged, partial cerebral hypoxia, or b) An acute sentinel event of total asphyxia with severe cerebrum, brainstem, cerebellum, and hypothalamus hypoxia followed by another episode of subacute, prolonged, partial cerebral hypoxia. The fetal monitor tracing is more compatible with the second sequence.

The child is microcephalic, suffers seizures, and is diagnosed with CP (spastic quadriplegia), severe developmental delay, and cortical visual impairment on follow-up. He dies at 14 years of age. Follow-up reveals the baby has seizures, cerebral palsy, spastic quadriplegia, microcephaly, and severe developmental delay. The baby also has a cortical visual impairment, all attributed to an anoxic/ischemic event at birth.

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Causation:

The initial tracing is reactive with normal variability, cyclical activity, and absent decelerations. This tracing bespeaks normal neurological responsiveness and absent hypoxia in the fetus. Despite the significant uterine activity, monitoring is intermittent. When re-attached after a 7-hour hiatus, the fetus shows decelerations. In getting up to go to the bathroom, the patient experiences faintness and vaginal bleeding. Upon return to bed, it is evident that the fetus has suffered another prolonged deceleration in association with a dramatic increase in uterine activity and a marked abnormal recovery consistent with an acute neurological injury. During the 2nd stage, pushing is associated with indicators of additional injury. With the application of mid-forceps, there is a profound fetal deceleration from a baseline of 180 to about 110 bpm just prior to delivery. The newborn shows trauma from the forceps, including bruising and facial nerve paresis. To these developments, the neonatal course, including hypoglycemia and seizures with evidence of acute injury to the liver, the kidneys, and the brain, points with singular specificity to hypoxic-ischemic and potentially traumatic injuries arising during the intrapartum period.

This is consistent with multi-organ injury with dysfunction (failure) in severe global fetal hypoxia. Finally, there is no obstetrical, neonatal, neuroradiological, or clinical evidence that the baby had an abnormal brain or had sustained any neurological injury prior to admission to labor and delivery.

Allegations:

The injury to the fetus derives from significant deviations from the standard of care, including the early rupture of membranes, likely at the high station, the failure to appreciate the poor feasibility of safe vaginal delivery, the failure to properly maintain the fetal monitor tracing in the face of abnormalities of both heart rate pattern and uterine contractions, the failure to properly recognize, monitor and respond to excessive uterine contractions and abnormalities in the fetal heart rate pattern, and ultimately the failure to recognize proper indications and contraindications for operative delivery.

Proper care would have timely recognized the excessive uterine activity and maintained continuous observation providing early detection of decelerations in the heart rate. The frequent decelerations during that time and the numerous risk factors reducing the feasibility of safe vaginal delivery in the foreseeable future required a timely, atraumatic cesarean section.

Indeed, there is no comment in the medical record about excessive uterine activity and no action to minimize the frequency of contractions. Despite the bleeding and excessive uterine activity unrelated to oxytocin, no provider considered the possibility of placental abruption.

The importance of recognition and prevention of excessive uterine activity, also known as tachysystole or uterine hyperstimulation, has been well-recognized for many decades. It does not reliably make labor go faster. (1) It is associated with neonatal HIE, while prolonged pushing increases the risk of fetal hypoxia and seizures. (2) (3)

There is no plausible benefit to permitting excessive uterine activity to the point of fetal distress. When excessive uterine activity is found, it must be dealt with as a preventive measure, irrespective of abnormal fetal response. When it is spontaneous, there must be

consideration of the potential for placental abruption, especially in the presence of bleeding, fainting, and vomiting. In the presence of decelerations and spontaneous hyperstimulation, intervention is required.

“Early rupture of the membranes increases the risk of decelerations during labor and molding of the fetal head, and when performed with an unengaged presenting part, it adds the risk of prolapse of the umbilical cord without commensurate benefit in terms of enhancing the progress of labor. (4, 5)”

Early rupture of the membranes increases the risk of decelerations during labor and molding of the fetal head, and when performed with an unengaged presenting part, it adds the risk of prolapse of the umbilical cord without commensurate benefit in terms of enhancing the progress of labor. (4, 5) Not rupturing the membranes at the high station as required by the standard of care would have diminished the likelihood of decelerations, which would have enhanced fetal reserve and potentially avoided injury.

We believe that in many cases, the tracing permits the affirmative diagnosis of fetal injury, sometimes, as in this case, many hours prior to delivery. (6, 7), Unfortunately, this insight into the timing of the injury is often not considered in the evaluation of the newborn for therapeutic hypothermia. (8) Therapeutic hypothermia requires initiation within 6 hours of birth on the premise that the injury occurred around the time of delivery.

Defense allegations:

These entirely consistent opinions from the plaintiff's obstetrical, neonatology, and pediatric neuroradiology experts were met with disparate rebuttals from 2 defense experts.

One expert acknowledged the vasovagal episode while using the bathroom with resulting fetal bradycardia as the cause of injury but believed that the injury was not preventable. The other expert believes that the injury occurred antepartum.

Neither commented on the excessive uterine activity and intermittent decelerations. Nor did they opine on the appropriateness of sending the patient to the bathroom under the circumstances. Neither believed the tracing required intervention despite the abnormal features. They opined that the conduct and presumably the timing of the delivery was appropriate. One conceded that “A case could be made for assessing the patient at least a half-hour earlier concerning the delivery

They both opined that the meconium staining of the 40-week fetus was likely to present well before delivery, reflecting an earlier insult. At term, the passage of meconium is more likely related to gestational age than to some prenatal insult. As mentioned, there is no basis for an antenatal injury.

Both accept that the fetal head is microcephalic at birth based

on the single value of 33 cm. at birth. That head circumference falls between the 8%ile and the 12%ile. Neither opinion took into account the impact of labor and operative vaginal delivery on the single assessment of the fetal head circumference. The newborn is “obviously not IUGR.” but suggests a significant change between the 38-week ultrasound and the delivery measurements. It would be reasonable to assume that some other process affects the head or brain development, with the insult occurring well before labor, most probably days to weeks before.” This allegation in an otherwise asymptomatic mother and an acute neurological injury in her fetus seems quite improbable: there is no complaint of decreased fetal movement, and the fetal heart rate pattern on admission is quite reassuring. An arrest of fetal head size at 38 weeks with the head in the 50th percentile cannot possibly result in microcephaly two weeks later. The growth curve of the fetal biparietal diameter at that point is almost flat.

It should be noted that ultrasound examinations were performed: at 18, 24, and 38.2 weeks gestation. In each case, the HC measurements are consistent with gestational age. While the medical record provides no comments on molding and caput, there are obvious forceps marks and bruising along the face's right side, including the eyelids and the cheek.

The use of a single HC measurement suggests a diagnosis of congenital microcephaly that cannot and does not reasonably justify such a diagnosis, given the numerous observations strongly inviting the inference that the baby's head circumference has been artificially reduced by the number of days of uterine activity, the excessive uterine activity, the prolonged labor, and ruptured membranes, the non-productive pushing and the forceps delivery.

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In 1983, Sorbe and Dahlgren studied the molding of the fetal head of 319 vaginal deliveries using a photographic method. (9) They documented the size and form of these infants' heads both immediately after delivery and three days later (a measurement lacking in this case), during which certain diameters of the head returned to a larger size. They found that infants born to primiparous women showed significantly higher degrees of head molding than those born by multiparous women. Oxytocin stimulation during labor prolonged labor or at least prolonged contractions and instrumental deliveries resulted in increased fetal head molding.

The importance of fetal presentation at birth (not stated here), the duration of labor, the mother's age, and the infant's birth weight were also analyzed concerning the molding of the fetal skull during labor. Similarly, studies show that the dimensions of the lateral ventricles are quite diminished immediately after vaginal delivery. (10)

The case was adjudicated.

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