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Briefly Legal: In Utero Manipulation Leads to Adverse Outcome

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The patient is a 26 y/o G5 P2 with a known sickle cell trait -with known Diamniotic, dichorionic (Di-Di) twins. She has a normal prenatal course with appropriate growth of both twins, who are about the same estimated weight. She is induced at 36+ wks EGA because of atypical preeclampsia.

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Upon admission to the hospital, a preliminary ultrasound exam reveals the twins to be in vertex / transverse lie presentations, and the decision is made to induce labor with the expectation of spontaneous delivery of Twin A and spontaneous conversion of Twin B to either vertex or breech after the delivery of Twin A.

She is observed on L&D for several hours before initiating Pitocin. With Pitocin, she makes rapid progress in cervical dilatation and receives epidural anesthesia within 3 hours. At fu1l dilatation of the cervix, she is transported to the delivery room in anticipation of vaginal delivery of Twin A. Appropriately, anesthesia is in readiness should problems develop with Twin B – and a double set up required. The fetal tracing of both twins remains reassuring despite very frequent uterine contractions. Within 15 minutes, Twin A, weighing 2438 grams, delivers spontaneously without incident.

Following the delivery of Twin A, with Pitocin still running, an ultrasound examination reveals that twin B is still a transverse lie. The EFM strip reveals no discernible tracing of Twin B after the delivery of Twin A, but it does reveal frequent uterine contractions coming every 2 minutes or less.

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The mother consented to cesarean delivery. The obstetrician then used an amnihook to rupture the membranes and pulled at what was thought to be the right leg (presumably the posterior leg) with the expectation of performing a total breech extraction. That extremity turned out to be the hand that was pushed back into the uterus. The obstetrician then identified the left leg (presumably the anterior leg) flexed, giving access to the foot, which he grabbed and pulled out the left leg. With sufficient descent, the right leg was flexed at the knee and also delivered. The baby, now a double-footling presentation, was then pulled to the scapula and rotated. One arm, splinted above the baby's head, was released by sweeping the arm across the chest. The obstetrician then delivered the shoulders and head without further difficulty.

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Twin B was born flaccid, pale with no cry, suck, or respiratory effort, and "decreased movement of his extremities." He was given PPV x 5 min and blow-by oxygen until 15 minutes of age. Apgar scores were 2,6,7 at 1,5,10 minutes, respectively. There was bruising and ecchymosis over the spine and foot. The head was molded with a decreased size of the fontanel. Cord blood gases were normal. The baby was sent to the normal newborn nursery.

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Within 20 minutes of life, retractions began. At 4 hours of life, the baby was transferred to the SCN. Within 5 hours, he could not latch and had a poor suck. He was found to have decreased activity, desaturation, and apneic episodes that became jerking rhythmic motions. A complete blood count and electrolytes were normal. Phenobarbital was started. A lumbar puncture revealed bloody cerebral spinal fluid (CSF) with 6,500 red blood cells per hpf, and 500 white blood cells per high power field, with a normal differential. Polymerase chain reaction (PCR) for herpes simplex virus (HSV) was negative. Blood and CSF cultures were negative. An electroencephalogram (EEG) was severely abnormal. Magnetic resonance imaging (MRI) showed diffuse cerebral, cerebellar, and brainstem infarctions within a watershed distribution. A magnetic resonance angiogram (MRA) and venogram (MRV) showed partial

left transverse sinus thrombosis. A coagulation evaluation was negative. He demonstrated cortical blindness, sensory hearing neural loss, speech, and developmental delays on follow-up.

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The lawsuit was brought against the physician. Pretrial discovery revealed the following allegations by the plaintiff:

- There was a failure to monitor Twin B immediately after delivery of Twin B properly.
- There was a failure to discontinue the Oxytocin in the face of excessive uterine activity and with the expectation of the need to manipulate the fetus.
- In choosing to manipulate the fetus, it was below the standard of care to fail to attempt to relax the uterus.
- With a back-down transverse lie, the prospects of a safe version were poor, and either a primary section for both twins or an elective cesarean section for Twin B was required.
- Given the immediate availability of the operating team and the initial difficulties in turning Twin B, efforts should have ceased and a cesarean section performed.

Concerning causation, the explanation of the depression and encephalopathy was attributed to umbilical cord compression caused by multiple manipulations or occult cord prolapse. Plaintiff experts maintained that the umbilical cord became compressed during various manipulations, ultimately leading to a total breech extraction. In addition, more probably than not, the problems of cord compression were abetted by the abruption of the placenta as the uterus, with frequent contractions, clamped down after the delivery of Twin A.

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The defense experts maintained that the standard of care was met and that the outcome was related to unpreventable and unavoidable complications of delivery. After pretrial discovery, the case was settled before trial.

Comment

From the outset of pregnancy, it is anticipated that twin gestations will have a greater risk of prematurity malpresentation, preeclampsia, and postpartum hemorrhage. In addition, the labor-management of twin gestation can be challenging. While di-amniotic, di-chorionic (di-di) twins may adopt any possible combination of presentations, in perhaps 70% of cases, they present with the first twin in cephalic presentation. For the second twin, other than those in breech presentation or back down transverse lie, their presentations are considered unstable with the potential to convert to cephalic or breech presentation after the delivery of Twin A. In most cases, when both twins are cephalic, vaginal delivery will be attempted. When the leading twin is non-cephalic, the twins will be delivered by elective cesarean section.

There is considerable variation in how cephalic / non-cephalic combinations are handled. However, even when the leading twin is cephalic, the majority will be ultimately delivered by cesarean section, including about a 5-10% risk of performing a cesarean for the second twin after the successful delivery of twin A. The indications for discordant delivery routes include mechanical issues where Twin B is larger than Twin A or is in a transverse lie (especially back down). On occasion, the cervix clamps down after the delivery of the first twin. Fetal distress in Twin B from cord prolapse cord and placental abruption with hemorrhage are complications that demand emergency intervention, and hence, the availability of expertise in anesthesiology not only for the rescue of the fetus but to assist in uterine relaxation should manipulation of the fetus into a deliverable position be required.

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It was initially thought that the longer the interval between deliveries, the greater the risk of adverse outcomes for the second twin. Indeed, as a generalization, second twins tend to have poorer outcomes than their preceding sibs; this seems to be true irrespective of the delivery route. (1) It has been shown, for example, that pH tends to fall during the interval between deliveries. This has led some investigators to recommend immediate internal podalic version and breech extraction of the aftercoming twin not presenting as cephalic or breech in the pelvis. This approach requires considerable experience and uterine relaxation for success. The procedure is known to be difficult with no guarantee of success (for which simultaneous preparation for cesarean sections must be made).

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Persistent attempt(s) at delivery under such circumstances are likely contraindicated and, if attempted, permit only a single effort by the most experienced obstetrician in the room. Persistent attempts at manipulation delay the resolution of any fetal compromise from asphyxia and increase the risk of fetal trauma and adverse outcome.

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Studies have shown that the FHR pattern of Twin B after the delivery of twin A is the better determinant of the time (and urgency) of intervention, whether by spontaneous or operative vaginal delivery or cesarean section. (2) The properly obtained fetal tracing will not fail to reveal problems of abruption or cord prolapse.

Statistics on breech deliveries are in flux; the likelihood of cesarean section for both twins is increasing while the use of induction and cesarean section for the second twin is diminishing. (3) In a retrospective study of the long-term trends in twin pregnancies and mode of delivery over 30 years, the authors found statistically significant increases in maternal age, and nulliparity, and in the number of twin deliveries, especially those conceived after IVF. (4) There was also a statistically significant reduction in term deliveries, mean birth weight and assisted vaginal deliveries – diverse characteristics that appear to conspire to increase the rate of cesarean section.

While a well-publicized controlled trial of women with twins where the leading twin was cephalic showed no significant differences in outcome between mothers with planned vaginal delivery and elective cesarean section, this study and other initiatives to lower the cesarean section rate appear to have had little impact on the increasing trend to a cesarean section in cases of multiple gestations. (5)

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The potential misadventures with the second twin are well-illustrated in this case, where multiple attempts were made to effect vaginal delivery in a second twin that likely presented as a back-down transverse lie. The most obvious failure from the outset relates to the lack of monitoring of the fetal heart rate pattern to determine its well-being after the delivery of Twin A. Its FHR pattern was demonstrably normal immediately prior to the delivery of Twin A. Secondly, considerable uterine activity was shown on the monitor that required consideration of efforts to relax the uterus. Frequent contractions would also likely confound any attempt to modify the fetal presentation from transverse to either cephalic or breech. The multiple maneuvers attempted illustrate the pitfalls of turning a back-down transverse lie, of mistaking a hand for a foot, and the need to replace the extremity before the proper leg was identified. The anterior leg must receive traction lest the fetal buttock impinge on the symphysis and cause dystocia. In addition, placental abruption is a known risk factor for Twin B after the delivery of Twin A as the uterus diminishes in size, potentially reducing the size of the implantation of the placenta.

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As mentioned, the proposed mechanism of the fetal neurological injury was compression or spasm (perhaps an occult prolapse) of the umbilical cord – a known complication with twins or non-vertex presentations abetted by placental abruption. Several mechanisms may impair umbilical blood flow. Myocardial dysfunction secondary to hypoxia will create sluggish to absent blood flow secondary to poor heart contractility. Direct pressure or stretching of the cord, especially when the stretch is abrupt, can create vasospasm and narrowing in the umbilical vessels and impede blood flow. Stretching of the cord can occur as the fetus descends the birth canal, especially if the descent is rapid or the cord is short. The cords of monoamniotic twins can become entwined and restrict blood from flowing freely. The most plausible explanation involves the compression of the cord between the fetus and the uterine wall. This risk is enhanced in the presence of decreased amniotic fluid volume and when excessive uterine activity is present, manifested by either repetitive or strong contractions, as in this case. The turgor of the cord influences the presence or degree to which the cord is pressed. A large infant of a diabetic mother has a relatively thick cord that is less compressible than a thin cord which is typical of an intrauterine growth-restricted fetus.

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In order to explain the ubiquity of variable decelerations, especially in the 2nd stage of labor, it is believed that uterine contractions per se cause intermittent squeezing of the umbilical cord. These decelerations are probably related to head compression associated with maternal pushing. (6)

"Cord compressions typically result in variable decelerations, which appear as quick, irregular, often jagged drops in heart rate and reflect a fetal autonomic reflex response. Initially, compression of the umbilical cord occludes the thin-walled, compliant umbilical vein, decreasing fetal venous return and triggering a baroreceptor-mediated reflex rise in fetal heart rate (sometimes referred to as a "shoulder")."

Cord compressions typically result in variable decelerations, which appear as quick, irregular, often jagged drops in heart rate and reflect a fetal autonomic reflex response. Initially, compression of the umbilical cord occludes the thin-walled, compliant umbilical vein, decreasing fetal venous return and triggering a baroreceptor-mediated reflex rise in fetal heart rate (sometimes referred to as a "shoulder"). When intermittent and showing prompt return to the previously normal baseline rate and variability, variable decelerations are considered benign in the fetus with a good reserve. Further compression or vasospasm occludes the umbilical arteries, causing an abrupt increase in fetal peripheral resistance and blood pressure as the fetus again tries to protect itself. The abrupt rise in blood pressure triggers an increase in parasympathetic outflow and an abrupt decrease in heart rate. As the cord is decompressed, this sequence of events occurs in reverse. While variable decelerations represent anticipated physiologic reflexes to umbilical cord compression and do not represent periods of hypoxia or acidemia per se, severe and repetitive compression will eventually compromise oxygenation, especially and more so in a fetus with little reserve. Periods of tachycardia or prolonged recovery of the variable decelerations are secondary to a sympathetic response to hypoxemia. These spurts are referred to as "overshoots." There is no evidence that the FHR will fail to respond to cord compression and its pathophysiological consequences. In this case, no such surveillance was undertaken in haste to deliver Twin B vaginally.

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This paradigm helps to explain the normal umbilical blood gases in this asphyxiated infant. (7) A cord blood gas sample reflects the status before the cord is compressed. The average pH difference between the umbilical artery and the vein is 0.07. A pH difference of 0.11 or greater suggests umbilical vein occlusion. A wide difference between the two is the hallmark of occlusion of the umbilical cord.

Suggested Reading

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