Briefly Legal: Loss of Leg Secondary to an Umbilical Arterial Catheter

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A 27-year-old primigravida patient presented at 34 weeks gestation for routine prenatal check complaining of a decreased fetal movement. Her prenatal course had been unremarkable until 34 weeks, at which point ultrasound evaluation revealed mild fetal hydrops and an obvious atrial flutter with a ventricular rate above 200 bpm. The patient was sent immediately to the hospital, where a cesarean section was performed. At birth, the 3360-gram female infant presented with a regular heart rate of 80 bpm, decreased tone, and minimal breathing effort. The Apgar scores were 2 and 5 at 1 and 5 minutes, respectively. The baby was immediately intubated and given positive pressure ventilation. A venous cord gas showed a pH of 7.0, a pCO_2 of 82 mm Hg, pO_2 of 17 mm Hg, and a base deficit (BD) of 12.9. An arterial cord gas was not obtained.

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The baby was brought to the Newborn Intensive Care Unit (NICU). Her heart rate at this point was 120 bpm and regular, and the rest of the physical examination was unremarkable except for mild retractions. She required moderate ventilatory settings with inspired oxygen of 40%. By 30 minutes, umbilical lines were attempted. An umbilical venous line was successfully placed, but a right umbilical arterial catheter (UAC) could not be advanced beyond 7 cm. The calculation for a high-positioned UAC (see below) was 18 cm. Blood could be withdrawn only intermittently from this line, and it was left in place.

The first arterial blood gas showed a pH of 7.14, a PCO_2 of 61mmHg, a pO_2 of 51 mmHg, and a BD of 9. The inspired oxygen and ventilator settings were increased. About an hour after birth, the BP, taken indirectly, was found to be 34/20 with a mean of 28 mm Hg. In response, she was given 40 ml of normal saline over 1 hour. The chest radiograph showed bilateral pleural effusions. Bilateral thoracenteses were performed, producing several ml of clear yellow fluid, followed by the insertion of chest tubes. The ECG revealed normal sinus rhythm. Two and a half hours after birth, the initial (right) UAC was discontinued, and a left UAC was inserted without difficulty. There was no apparent evaluation of pulses and temperature over the lower extremities and gluteal areas.

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Despite the absent femoral pulses and the pale leg, the second UAC remained in place for several more hours. The leg gradually became mottled, dark, and edematous. Sixteen hours after birth, the neonatologist and the ***risk manager*** met with the mother to discuss the situation. After this discussion, perhaps stimulated by it, a decision was made to contact a higher-level NICU. The neonatologist at the referral center advised the application of topical nitroglycerin ointment, elevation of the right leg, and immediate transfer. ***Upon transfer to the referral hospital, evaluation of the infant's condition, and attempts to save the leg were undertaken. The leg was amputated. The postoperative course was unremarkable.***

The neonatologist and hospital were sued.

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Allegations

Plaintiff's experts were critical of failing to obtain an arterial cord gas at birth. This allegation seemed superfluous considering the severity of the venous pH

They were also critical that the second UAC was left in place since it could not be advanced. The treating physician and nurses countered that the baby was very sick and needed to have central arterial access. In response, Plaintiff's experts pointed out that a venous line was available and that the UAC

was not properly situated in the vessel's lumen and should have been immediately discontinued.

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The plaintiff experts pointed out that an evaluation of the lower extremities and gluteal area should have been performed after the insertion of the catheter, especially since the initial catheter could not be advanced. Further, the UAC should not have been manipulated and uncoiled but should have been discontinued. Arguably, the second catheter should not have been inserted, considering the disruption in the aorta from the first UAC. Again, the treating neonatologist renewed the assessment that the baby needed the UAC since she was critically ill. The plaintiff neonatologist said, at minimum, that the second UAC should not have been manipulated since the endothelium of the aorta is vulnerable.

The plaintiff neonatologist was appalled that a higher-level medical center was not immediately contacted when the perfusion of the leg was compromised and, further, that an individual from risk management was consulted before a specialized center was consulted.

The case was settled without going to court.

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Discussion.

In this case, the fetus was discovered at 34+ weeks' gestation to be in cardiac failure secondary to cardiac arrhythmia (atrial flutter) and presumably a persistently rapid heart rate. There was no structural cardiac abnormality in the fetus, and the rhythm became sinus immediately after birth.

UACs are commonly used in critically ill neonates, can be placed relatively quickly and easily at the bedside, and be used for many critical functions, including infusion of intravenous fluid and medications, obtaining arterial blood samples, monitoring BP, doing exchange transfusions, and cardiac catheterization. UACs should be placed only by experienced individuals using strict aseptic techniques and only when required. A 5Fr catheter is used for babies >1500 grams, a 3.5 Fr catheter is for infants ≤1500 grams, and a 2.5 Fr catheter is for extremely low birth weight infants if a 3.5 Fr catheter is not possible.

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Once the umbilical arteries have been identified by their anatomic characteristics, one vessel is dilated with an iris forceps. Once inserted, lines cannot be advanced unless the field remains sterile. The artery is dilated, and the catheter is then **gently** advanced to a predetermined length that will place its tip at a high (T 6-9) or at a low (L3-L5) position to avoid false tracking (e.g., the catheter is advanced in the vessel wall outside the lumen),. The umbilical arterial line first travels inferiorly and posteriorly to its junction with the internal iliac artery. Following the artery's course, the catheter must turn superiorly to course through the common iliac artery and aorta. A high-positioned UAC (T6-T9) places the tip above the origin of the celiac axis and below the ductus arteriosus. A low positioned UAC (L3-L5places the tip just above the aortic bifurcation but below the major aortic branches. High-placed UACs have a lower incidence of vascular complications than those in low positions. UACs that are located in an intermediate position between the "high" and "low" positions should be pulled immediately to a "low" position or removed.

" Immediately after placement and suturing of the catheter, it is critical to inspect the infant's buttocks, back, and legs, looking for signs of decreased perfusion that would suggest that the catheter has been inadvertently placed in a gluteal or spinal artery or has created lower extremity ischemia. If these areas appear dusky or pale, immediate catheter removal is required."

A shorthand formula can be used to estimate the catheter length to be inserted. The umbilical arterial catheter length in centimeters

can be calculated from Shukla's formula: 3 x birth weight in kg + 9 for a UAC with the tip in the high position. Immediately after placement and suturing of the catheter, it is critical to inspect the infant's buttocks, back, and legs, looking for signs of decreased perfusion that would suggest that the catheter has been inadvertently placed in a gluteal or spinal artery or has created lower extremity ischemia. If these areas appear dusky or pale, immediate catheter removal is required.

"But for these negligent actions that fell below a reasonable standard of care, given appropriate care, the newborn would have survived had he not been dropped."

These evaluations must be performed even before obtaining radiographic evidence of line placement and tip location,

Radiologic confirmation of the UAC position is imperative. After the UAC is inserted and the proper tip location is confirmed, cm markings on the catheter should be noted and recorded. By documenting the length of a catheter inserted, any movement of the catheter inward or outward can be closely monitored with appropriate intervention. The insertion site must be kept clean and dry. Immediate attention must be placed to remedy a situation if sutures become loose to ensure proper maintenance of tip position. Vascular spasms may occur after catheter insertion or may be triggered by arterial sampling, usually secondary to temporary and reversible arterial constriction. Vascular spasm is characterized by transient cyanosis of the toes, but peripheral pulses are still palpable. A trial of reflex vasodilatation by warming the contralateral extremity generally resolves the spasm.

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Low doses of heparin (0.25-1.0u/ml) should be added to the fluid infused through the UAC to reduce the risk of thromboembolism (TE). Only catheters with end holes should be used since the risks of TE and infection are higher with catheters with side holes. UACs should not be replaced if signs of infection or vascular insufficiency in the lower extremities are found. Reinsertion of UACs is generally discouraged because of potential damage to the intimal lining of the arterial vessels with the potential for thrombotic complications. While any fluid, medication, or blood product may be infused through an umbilical venous catheter (UVC), inotropes, calcium boluses, and indomethacin should not be infused via the UAC. Blood and prostaglandin E1 may be infused through a UAC if there is no alternate venous access.

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Complications of UAC include:

1) ischemic and thromboembolic events were causing necrosis to the lower extremities and gluteal area. Arterial thromboembolism (TE) symptoms include pallor or coldness of the lower extremities and/or gluteal area and diminished or absent pulses.
2) vascular compromise to kidneys causing renal failure and hypertension and to the intestine causing necrotizing enterocolitis
3) hemorrhage from a dislodged catheter and extravasation from perforation of a vessel
4) hypoglycemia - if UAC tip streams glucose near celiac artery (located at T12 level)
5) air embolism
6) infection
7) direct peritoneal perforation or urachal/bladder injury
8) aneurysmal dilation with dissection of the abdominal aorta
9) spinal cord infarction
10) discrepancy in leg growth
11) acquired aortic coarctation,
12) mycotic aneurysms of the aorta, particularly in association with Staphylococcus aureus infection.
13) Miscellaneous - Unexplained thrombocytopenia, catheter-obstructed fluid delivery or increased in-line pressure, and concerns regarding lower body or extremity perfusion must be investigated for possible thromboembolism.

Mechanism and incidence for thromboembolism with UACs

Insertion of arterial catheters may mechanically damage the vascular endothelium exposing subendothelial tissue and collagen to the circulating blood, causing adherence and aggregation of platelets, thereby releasing adenosine diphosphate and thromboxane A2 (a platelet stimulator and a very potent vasoconstrictor). These events can lead to vasospasm around the catheter, with complete vessel occlusion and tissue ischemia. A UAC should be placed only in babies who need to have them,

monitored diligently, and when no longer required, should be removed as soon as possible to minimize potential complications. Optimally, umbilical artery catheters should not be left in place for >5 days.

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The longer the catheter is in place, the risk of bloodstream infection and catheter-associated TE increase significantly. The reported incidence of arterial thrombosis in neonates with UACs ranges from 8 to 20 percent. Most UAC-associated thromboses are asymptomatic. In one study, two-dimensional abdominal sonography found that 1/3 of infants had abdominal aortic thrombi upon removal of the UACs. The authors found that the probability of developing aortic thrombosis in an infant with a UAC in situ for one day was approximately 16%, increasing progressively to 32% at seven days, 57% at 14 days, and 78% at 21 days. Although 50% of UAC-related thrombi disappear before discharge from the hospital, the long-term consequences of persistent thrombosis have not been studied systematically. UAC-associated TE has been linked to hypertension, renal function abnormalities, and leg length discrepancy in long-term follow-up studies.

Treatment for an ischemic and/or thromboembolic event – Principles and specifics:

- A blanched extremity is an indication for immediate removal of the catheter.
- Patients with TE should be cared for in a highly specialized NICU where radiographic studies, pediatric hematologists, and pediatric vascular surgeons are available should their services be necessary.
- Two-dimensional ultrasound and radionucleotide scanning are usually sufficient to confirm the diagnosis of TE. In some cases, the presentation of TE can mimic severe aortic coarctation.
- Treatment of TE is highly individualized and includes supportive care, generous intravenous fluids, the elevation of the involved extremity, topical application of 2% nitroglycerin ointment at a dose of 4 mm/kg body weight, applied as a thin film over the affected areas, and repeated after 8 hours. Anticoagulation, 4ibrinolytic therapy or surgical intervention may be necessary for specific instances.

Fetal arrhythmias

The cause of the baby's heart failure and compromise at the time of birth was tachyarrhythmia (atrial flutter). Whereas brief accelerations in fetal heart rate (FHR) near term greater than 15 bpm for up to 2 minutes are associated with fetal well-being, sustained elevations of fetal heart rate (tachycardia), irrespective of the underlying rhythm, may be associated with fetal cardiac failure in the form of hydrops fetalis or death. The first clinical clue to the presence of hydrops may be the complaint by the mother of decreased fetal movement, but more often, a rapid rate is detected by routine auscultation. Electronic fetal monitoring has contributed to the awareness of the possibilities of various heart rate and rhythm disturbances. While transabdominal recordings are increasingly available, they are sufficiently reliable to show fetal atrial P-wave morphology. Prenatally, delineating the rhythm disturbance requires evaluation by echocardiographic techniques to determine the function and rule out major structural defects of congenital heart disease and any signs of fetal heart failure. More recently, fetal magnetocardiography (fMCG) has become clinically available. Intrapartum, direct electrodes not only permits observation of the ECG waveform but also provide tachometric clues to arrhythmia that cannot be appreciated by analysis of ECG morphology. Except for complete heart block, arrhythmias associated with structural defects are rare.

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The conduction system of the fetal heart is functionally mature by 16 weeks of gestation, and the average heart rate between 18 and 22 weeks of gestation is 140 bpm. The automaticity of the sinus node is more rapid than the other potential pacemaker sites along the conduction system. An impulse will originate from the next fastest location of pacemaker cells, which may be in the atria, the AV node, or the Purkinje fibers resulting in the sinus rhythm as the fastest normal atrioventricular sequential stimulus. AV nodal or junctional rhythm at 70 to 100 bpm; and a ventricular rhythm at a rate of 45 to 70 bpm are often seen at the base of significant variable decelerations when the intense vagal stimulus has significantly suppressed the sinus node.

A premature beat is defined as a depolarization of the atrium, AV node, or ventricle that occurs earlier than the automatic rate of the sinus node or site of origin of the intrinsic rhythm. Therefore, a premature beat comes earlier than the expected beat-to-beat interval, while an escape beat comes later or at the normal (interpolated) rate for the site of origin. A compensatory pause generally follows conducted atrial premature beats and ventricular extrasystoles before the return of normal atrioventricular rhythms. Premature atrial contractions (PACs) are considered benign and much more common than ventricular premature contractions (PVCs). Approximately 90% of studies in which fetal arrhythmias are detected involve isolated extrasystoles or bigeminal rhythm. Follow-up on extrasystoles involves weekly auscultation to detect tachycardia and monthly routine ultrasound evaluation to develop hydrops. The mother should be assessed for hyperthyroidism and avoid caffeine and other stimulants during the pregnancy.

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Supraventricular tachycardias (SVT) with rates above 200 bpm, SVT occurs in 1—3% of fetuses with isolated atrial ectopic beats. The arrhythmia can be classified as automatic, from an ectopic focus, or reentrant with or without an accessory bypass tract. Reentrant mechanisms are more likely to have sudden paroxysmal onset following a premature depolarization and be transient in duration. Ectopic tachycardias occur due to enhanced or altered automaticity and may occur continuously. Even when brief, SVT causes a significant flow reversal in the ductus venosus, back toward the umbilical vein. Persistent SVT may lead to hydrops fetalis and fetal demise due to decreased cardiac output. Of interest, the abnormal rhythm may occasionally be terminated by the intentional or inadvertent compression of the umbilical cord between the uterine wall and fetus or around the fetal neck. With atrial flutter, the atrial rate is >400 bpm with a 2:1 AV block, with ventricular rates generally between 200—300 bpm. With atrial fibrillation, the atrial rate is over 400 bpm with variable ventricular rates.

Preterm fetuses, even those with hydrops, may be treated by maternal (transplacental) therapy with medications beginning with digoxin, which may be augmented with procainamide, amiodarone, or propranolol. Those with hydrops usually required multiple drugs. In addition, direct, in utero, transabdominal intramuscular injections into the fetus of digoxin may be used to achieve therapeutic levels of digoxin. Most of these fetuses convert under in-utero treatment, while the remainder do so after birth. About one-third will eventually relapse – especially those with WPW.

"In this case, tachyarrhythmia causes fetal heart failure. The heart failure was mild at the time of birth, and the atrial flutter spontaneously resolved. Nevertheless, the child was sufficiently compromised at birth with low Apgar scores, pH, BD, and problems of adaptation that the placement of a UAC was deemed necessary and defensible."

Summary of case

In this case, tachyarrhythmia causes fetal heart failure. The heart failure was mild at the time of birth, and the atrial flutter spontaneously resolved. Nevertheless, the child was sufficiently compromised at birth with low Apgar scores, pH, BD, and problems of adaptation that the placement of a UAC was deemed necessary and defensible. The management of its placement and subsequent events led to allegations of negligence. It seems probable that the insertion of the first UAC was false tracking, as suggested by blood being aspirated intermittently at the tip of the UAC. When the UAC is appropriately placed in the lumen of the aorta, blood should flow freely. As a result of the improper placement, the inner lining of the lower aorta was damaged and became a nidus for thrombus formation. This catheter should have been immediately removed as soon as blood flow was found to be compromised and when attempts to withdraw blood were unsuccessful.

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Evaluation of the legs and gluteal area should have been done. The left UAC at this point was inserted and was found to be coiled in the mid-aortic region. A check for femoral pulses and leg perfusion absolutely should have been performed, and perhaps another catheter should not have been attempted, even if leg perfusion and pulses were normal. A few manipulations that were tried probably resulted in additional intimal damage and the perfect storm for TE. When the sterile field was removed, the leg was noted to be white, the pulses could not be palpated bilaterally, and the groin was edematous. These findings mandated the immediate removal of the UAC. Irrespective, the catheter remained in situ for hours while compromised perfusion and color changes were observed while the health care team entertained hopes of a spontaneous reversal. During this time, the lack of circulation to the baby's leg would lead to its amputation.

The physician and nurses did not reach out to another center, try any intervention or transfer the baby to higher level care until 17 hours after the event. Incomprehensibly, the hospital risk management was called in before medical resources were consulted about intervention and transfer.

Suggested reading

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