General anesthetic considerations for the infant with shaken impact syndrome and pyloromyotomy: A case report

PATRICIA J. WADFORD, CRNA, MSN
Wake Forest, North Carolina

"Guard well your baby’s precious head,
Shake, jerk and slap it never,
Lest you bruise his brain and twist his mind.
Or whiplash him dead forever.”

—John Caffey

This is a case report of an infant with shaken impact syndrome who required general anesthesia for the repair of pyloric stenosis. This infant suffered neurologic impairment from shaking, which was not only the major etiologic factor but the mechanism of injury as well. Shaking an infant can have devastating consequences.

A thorough understanding of the mechanism of injury and the sequelae that result from this type of child abuse are paramount to prevent further damage if the infant requires general anesthesia. Anesthesia providers play a vital role in minimizing secondary injuries in infants with shaken impact syndrome.

Key words: Pyloric stenosis, shaken impact syndrome, whiplash-shake mechanism of injury.

Introduction

Each year approximately 1,100 children in the United States die from fatal shaking or blunt traumatic injuries inflicted by adults. Traumatic head injuries in infants can be caused by shaken impact syndrome (SIS), a serious form of child abuse. Shaken impact syndrome, also known as shaken baby syndrome, is defined as one or more episodes of violent shaking of an infant. Most shaken infants are less than 6 months old. This is the report of a 12-week-old male infant diagnosed with SIS, who required general anesthesia for the repair of pyloric stenosis.

Case summary

A 12-week-old, 5.23-kg white male, accompanied by his mother and grandmother, presented in the emergency room with a 3-week history of projectile vomiting and chronic bilateral subdural effusions secondary to SIS. An upper gastrointestinal series with barium indicated a gastric obstruction with no contrast in the duodenum after 30 minutes. Admission serum laboratory values showed sodium, 142 mEq/L; potassium, 3.2 mEq/L; chloride, 83 mEq/L; carbon dioxide content, 35.4 mEq/L; blood urea nitrogen, 12 mg/dL; creatinine, 0.7 mg/dL; and glucose, 72 mg/dL. Analysis indicated hypokalemic, hypochloremic metabolic alkalosis. The infant's current medication was phenobarbital 10 mg by mouth twice a day. Phenobarbital level was 10 µg/mL.

A diagnosis of pyloric stenosis was made and the infant was scheduled for a pyloromyotomy at 8:00 AM the following day. The infant was given nothing by mouth, and a nasogastric tube was inserted. A right femoral intravenous (IV) line was inserted to correct fluid and electrolyte imbalances.
An infusion of D_{5}1/4 normal saline with 20 mEq of potassium chloride per liter was started at a rate of 35 mL per hour.

The physical examination revealed positive bowel sounds, a visible peristaltic wave from left to right across the upper quadrants of the abdomen stopping at the midline, and a moderately firm 1 x 1 cm mass (olive) just above the umbilicus that was palpable following each peristaltic wave.

Preoperatively the infant was pale, thin, dehydrated, and lethargic. Review of patient history revealed an infant born with Apgar scores of 8 and 9 following a normal pregnancy and an uncomplicated course for the infant's first 9 weeks of life. At 9 weeks the infant was left by his mother in the care of his father. When the mother returned home, she found the infant to be lethargic with two black eyes. The father admitted to violently shaking the infant in an effort to make him stop crying, jarring a pacifier in the infant's mouth, swinging him upside down by one leg, and hitting the infant's head with his hands.

The mother took the infant to the emergency room where bilateral subdural effusions, fifth and sixth rib fractures on the left, a left leg fracture and a right arm fracture were diagnosed. The infant was admitted with injuries sustained from SIS.

Two days after admission, the infant began vomiting and having seizures. The seizures were treated successfully with phenobarbital, but the vomiting continued. The infant was given IV fluids and vomiting was treated with promethazine. The department of social services took legal custody of the infant, and he was discharged 12 days following admission to the care of his maternal grandmother, who was given physical custody.

The grandmother took the infant to the emergency room the day after discharge for continued vomiting. The infant's neurosurgeon ordered measurement of the phenobarbital level and a computed tomographic (CT) scan. The phenobarbital level was within normal limits, and the CT scan showed chronic bilateral subdural effusions with a small newer bleed in the left frontal region. The formula was changed to half strength, and the infant continued to vomit. The grandmother brought the infant back to the emergency room with complaints of "forceful vomiting that shoots across the room" and no wet diapers over the last 18 hours.

On the morning of surgery the infant was brought to the preoperative holding area where, despite the infant's state of rehydration, he remained lethargic. Cefoxitin 70 mg IV was given. The infant was then transported to the operating room and the following monitors applied: electrocardiograph, automated blood pressure cuff, and pulse oximeter. To maintain normothermia, a warming blanket was placed on the operating room table, the infant's head was covered with a 6-inch stockinette, and his arms and legs were wrapped in webril. The nasogastric tube was connected to suction, no secretions were aspirated, and it was placed to drain by gravity. Atropine 0.05 mg IV was given prior to induction to prevent bradycardia and the infant was oxygenated with 100% oxygen for 3 minutes. Just prior to induction the pacifier was removed, but the infant remained passive and lethargic, never crying or resisting.

Anesthesia was induced with thiopental 20 mg IV followed by succinylcholine 10 mg IV with the application of cricoid pressure. The trachea was intubatedatraumatically with a 3.5 mm uncuffed endotracheal tube. When bilateral breath sounds and end-tidal carbon dioxide were confirmed, the endotracheal tube was secured, cricoid pressure released, and a 12-gauge esophageal stethoscope was inserted.

Anesthesia was maintained with fentanyl 10 μg IV in divided doses, isoflurane, and oxygen. Atracurium 2 mg was given IV for skeletal muscle relaxation. Mechanical ventilation was instituted via the Bain circuit, and the infant was manually ventilated keeping the end-tidal carbon dioxide between 30 and 32 torr.

The surgery progressed uneventfully and at the conclusion, the neuromuscular block was reversed with edrophonium 3 mg and atropine 0.05 mg IV following return of a full train of four with a sustained tetanus for 5 seconds as noted using a peripheral nerve stimulator. Five minutes after the reversal was given, the infant was without spontaneous respirations. Narcotization was diagnosed and naloxone 0.05 mg IV was given. Two minutes after naloxone was given, the infant began spontaneous respirations and maintained his oxygen saturation, moved his arms and legs, and attempted to swallow.

After suctioning, the infant was extubated. Oxygen was given at 100% via mask. Deep spontaneous respirations were noted along with an oxy-
gen saturation of 98%, but the infant never cried. The infant was transported to the postanesthesia care unit (PACU) with oxygen. The PACU stay was uneventful and the infant was subsequently transferred to the pediatric unit. He had yet to cry.

Discussion

Infants, because of their unique anatomy, are especially vulnerable to head trauma when they are shaken. Table I lists anatomical areas of vulnerability. Trauma related to SIS is the result of a whiplash-shake mechanism of injury. Injuries sustained from SIS occur because of rapid acceleration-deceleration of the head attached to a fixed torso, Terson's syndrome, and Purtscher retinopathy.

Table I

<table>
<thead>
<tr>
<th>Infant anatomical areas of vulnerability</th>
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<tbody>
<tr>
<td>Large heavy head</td>
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<tr>
<td>Underdeveloped torso</td>
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<tr>
<td>Lax, weak neck muscles</td>
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<tr>
<td>Large subarachnoid spaces</td>
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<tr>
<td>Thin, soft, compliant skull wall</td>
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<tr>
<td>Lack of control and mobility of head and neck</td>
</tr>
<tr>
<td>Unmyelinated, soft, rapidly growing brain</td>
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<td>Stretchable fontanels</td>
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Injury is usually sustained while the infant is held around the thorax and shaken. Pressure applied around the chest by the shaker's hands inhibits venous return causing blood to pool in the larger vessels (Purtscher retinopathy). This results in cranial blood vessels containing an increased blood volume under increased pressure.

According to LaPlace's Law, wall tension in a cylindrical structure increases as the product of pressure and radius. Abnormal wall tension is responsible for rupturing of the blood vessels in the head. These ruptured blood vessels can result in intracranial hemorrhage and cerebral edema, which in turn causes Terson's syndrome or increases in intracranial pressure.

Once the shaking has stopped, perfusion of the damaged vessels results in vascular leakage. This leakage causes acute retinal edema and hemorrhage.

The hallmarks of SIS are subdural hematomas and bilateral retinal hemorrhages. Table II lists the signs and symptoms of SIS. The shaken infant, however, often does not present with the classical signs and symptoms of child abuse but can show minimal or no physical evidence. Any infant presenting with unexplained seizures, vomiting associated with drowsiness or lethargy, subdural hematoma, failure to thrive, or signs of physical abuse should be evaluated for a diagnosis of SIS.

Sudden deceleration forces applied to the head of an infant cause a shearing of the cerebral bridging veins spanning the surface of the brain to the dura. The disruption of cerebral bridging veins results in subdural hematomas, deep white matter injuries, brain lacerations, and global hypoperfusion with cortical necrosis. The most common intracranial manifestation of SIS is posterior fossa subdural hematoma.

Injury to the calvaria can produce complex, depressed, and bilateral fractures. Skull fractures typically seen in SIS are occipital or parietal and cross sutures.

Brain damage in infants results from diffuse axonal injuries occurring in two phases. The initial injury begins with the application of biomechanical forces (shaking) to the cranium, neural tissue and vasculature, causing lacerations which result in the formation of hematomas. Secondary injury results from the pressure of the hematoma(s) on the brain causing hypoperfusion, hypoxemia, edema, and intracranial hypertension. SIS can result in death, but the shaken infant who survives is prone to epilepsy, mental retardation, visual loss including blindness, spasticity, quadriaparesis, hearing loss, hydrocephalus, and microcephaly.

During acceleration, the most dependent body part of the applied pressure is the face. As the pressure increases, fluid from engorged venous channels rushes into the eye causing subretinal, retinal, and preretinal hemorrhage. Acceleration...
also causes the vitreous to put stress on the retinal photoreceptors. Bilateral retinal hemorrhage has been associated with SIS. When intracranial pressure increases, the pressure in the central retinal vein also increases, causing retinal hemorrhages. Retinal hemorrhage is significant in SIS because in some cases, retinal hemorrhage has been detected before subdural hematoma. Increases in intracranial pressure are also transmitted by optic nerve sheaths, reducing venous outflow from the eye. Optic nerve sheath distension occurs as a result of this increase in pressure and may be an explanation for optic nerve sheath hemorrhages seen in SIS.

Violent shaking of an infant produces forceful waves that are transmitted through the facial bones and orbit causing an impact on the globe. The retina is injured as it hits against the sclera.

The shaker's hands encircling the infant's chest during shaking can cause rib fractures and chest bruising. Spiral fractures of the long ones, metaphysical lesions, corner or bucket handle fractures, and epiphysseal separations can occur if an infant is shaken by the extremities.

Pyloric stenosis

Pyloric stenosis is the result of hypertrophy of the pyloric smooth muscle, with edema of the pyloric mucosa and submucosa. Pyloric stenosis is more common in males and develops over a period of days to weeks, usually during the second to sixth week of life.

The development of pyloric stenosis causes progressive obstruction of the pyloric valve, which leads to projectile vomiting of feedings without bile. This vomiting results in varying degrees of fluid and electrolyte imbalance, the extent of which depends on the length of time involved.

Persistent vomiting results in the excessive loss of gastric hydrochloric acid and hydrogen ions. As hydrogen ions are lost, the kidneys excrete potassium in exchange for hydrogen ions, an attempt to maintain a normal arterial pH. Continued vomiting results in sodium depletion, and the kidneys begin excreting potassium and hydrogen ions due to the lack of sodium ions. This results in a dehydrated infant with hyponatremic, hypokalemic, hypocloremic metabolic alkalosis. Pyloric stenosis is a medical emergency, not a surgical emergency. It is imperative to correct fluid and electrolyte imbalances prior to surgical intervention. Because 80% of an infant's body weight is water, infants with intravascular volume depletion are prone to circulatory insufficiency.

Infants with pyloric stenosis are always considered to have a full stomach (many have had an upper gastrointestinal series with barium). Anesthetic considerations are outlined in Table III. The infant should not be extubated until fully awake.

<table>
<thead>
<tr>
<th>Table III</th>
<th>Anesthetic considerations in infants with pyloric stenosis</th>
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<tr>
<td>Preoperative assessment should include attention to volume status, serum electrolytes, and urinary output</td>
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<tr>
<td>Infants are always considered to have a full stomach</td>
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<tr>
<td>Insert nasogastric or orogastric tube prior to induction</td>
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<td>Induction technique: Rapid sequence or awake intubation</td>
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<tr>
<td>Avoid inhalation inductions</td>
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Maintenance of anesthesia can be accomplished by a variety of techniques. Nitrous oxide, narcotics, volatiles, and muscle relaxants have all been used successfully in various anesthetic combinations. Muscle relaxation may be needed during the delivery of the pylorus prior to repair and for replacing the pyloris in the abdomen following the repair.

Today, pyloric stenosis is usually detected by pediatricians before infants reach a state of severe dehydration. This, however, is not always true as illustrated by this case report. The infant's head trauma overshadowed the pyloric stenosis.

Another unique characteristic of this case was the infant's lethargy, noted preoperatively and postoperatively, that could have been caused by the sedative effects of phenobarbital, dehydration, or SIS. It may very well have been a combination of all three.

Conclusion

The most important consideration in the anesthetic management of an infant with SIS is to minimize further brain injury. Keeping in mind that brain injury in infants occurs in two phases, anesthetists play a vital role in minimizing the secondary injury phase by utilizing anesthetic agents that decrease intracranial pressure, thereby de-
creasing cerebral blood flow and cerebral metabolic oxygen requirements. It is also important to prevent hypotension, hypoxemia, and edema. Since infants with SIS are often treated with antiseizure medications, these infants may be resistant to nondepolarizing neuromuscular blocking drugs.

Anesthetizing an abused infant is an emotionally exhausting experience. These infants can present with a host of problems unrelated to the surgery they require. The infant with SIS who requires general anesthesia presents a challenge to the most experienced anesthetist. A thorough preoperative evaluation, vigilance in intraoperative monitoring, and an understanding of injuries involved in SIS are critical for a successful outcome.

Shaken infant syndrome is a preventable form of infant neurologic trauma that carries a high rate of morbidity and mortality. Anesthetists can play an important role in identifying these infants, minimizing secondary injuries, and promoting public awareness. Anesthesia providers must join the medical profession's fight against this deadly but preventable form of infant abuse. A united health care team must teach the public how very dangerous it is to "shake the baby."

REFERENCES


AUTHOR

Patricia J. Wadford, CRNA, MSN, received her nursing education at Watts Hospital School of Nursing, Durham, North Carolina, in 1980; her bachelor of science in Nursing from the University of Central Florida, Orlando, Florida, in 1992; her anesthesia certificate from the Raleigh School of Nurse Anesthesia, Raleigh, North Carolina, in 1994; and her master of science in Nursing from the University of North Carolina, Greensboro, North Carolina, in 1994. She is currently employed as a nurse anesthetist by Raleigh Anesthesia Associates at Rex Hospital in Raleigh.

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