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Sonographic Evaluation of Spinal Cord Birth Trauma with Pathologic Correlation

Paul S. Babyn¹
 Sylvester H. Chuang¹
 Alan Daneman¹
 George S. Davidson²

Birth trauma to the spinal cord is a serious potential complication of delivery. Determining the presence, severity, and extent of injury poses a difficult problem because of the often confusing clinical setting. Myelography has been recommended for assessing spinal cord birth trauma but is invasive and may not be helpful. The role of sonography in evaluating spinal cord birth trauma has not been previously described. We assessed the value of sonography in four patients, three of whom also had CT metrizamide myelography. Autopsy correlation was available in three patients. Sonography was able to easily demonstrate the cord configuration, allowing for multiple assessments over time. Internal cord echogenicity was helpful in a case of hematomyelia and in demonstrating the changes of myelomalacia.

Sonography is useful in evaluating neonates with severe spinal cord injury; it obviates the need for myelography and also may allow less severely injured patients to be assessed more frequently.

Birth trauma to the spinal cord is well recognized as a serious potential complication of delivery [1–8]. The presence, severity, and extent of injury are difficult to determine. Myelography, which is invasive, may not be contributory [6]. Although sonography has been used for neonatal disorders, its role in birth trauma has not been described. We therefore assessed the potential role of sonography in four patients with spinal cord birth injury, three of whom were also investigated with CT metrizamide myelography. Correlation with autopsy findings was available in three cases.

Materials and Methods

This study is based on four neonates who had findings of spinal cord birth injury. Three of the patients had CT metrizamide myelography using the established techniques of general anesthesia, lumbar puncture, and contrast injection. All CT scans were acquired on a GE 9800 scanner.

Sonographic evaluation of all four patients was done without sedation using either the Acuson 128 or ATL 450 portable units at the bedside. Two patients had follow-up examinations separated by intervals of 6 weeks. Both sector and linear array high-frequency transducers with near-field focus were used without a water bath. All infants were scanned from the posterior aspect of the neck after being turned into a lateral decubitus position. Continued stabilization of the neck and respiratory status was afforded as scanning of the upper spinal cord was performed.

Case Reports

Case 1

This baby boy was born at term after an uneventful pregnancy. Labor was spontaneous in onset with spontaneous rupture of membranes and cephalic presentation. Transverse arrest occurred at 7 cm and the infant was delivered vaginally following midforceps rotation.

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¹ Department of Radiology, The Hospital for Sick Children, 555 University Ave., Toronto, Ontario, Canada M5G 1X8. Address reprint requests to P. S. Babyn.

² Department of Pathology, The Hospital for Sick Children, Toronto, Ontario, Canada M5G 1X8.

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The birth weight was 3.1 kg. Apgar scores were 3 and 4 at 1 and 5 min, respectively. The baby required intubation and was transferred to our hospital. Evaluation showed a normally formed appropriately sized term, apneic infant. Facial movements were noted with flaccid paralysis of both upper limbs and minimal tone in the lower limbs. Withdrawal responses were seen only in the lower limbs, greater on the left than right. All deep tendon reflexes as well as anal tone were absent. A sensory level could not be determined. The infant also had signs consistent with hypoxic ischemic encephalopathy with CNS depression and presence of seizures. The EEG was grossly abnormal but visual evoked potentials and auditory brainstem responses were normal. The right clavicle was fractured and there was thought to be a concomitant right brachial plexus injury.

Initially, the infant was unstable, so CT metrizamide myelography was not performed until 2½ weeks of age. This showed a small high cervical cord (Fig. 1A). Sonography, which was performed at 1 month of age, also demonstrated the cord narrowing (Fig. 1B). The subsequent clinical course included continued apnea, spastic quadriplegia, seizure activity, and delayed cognitive development as well as recurrent respiratory and urinary tract infections. After several months without improvement in neurologic status, mechanical ventilation was discontinued. The infant died at the age of 9 months.

An autopsy revealed nearly total disruption of the spinal cord at the C3–C4 level, with overgrowth of the surviving meningeal cells and proliferation of Schwann cells in the remaining cord tissue (schwannosis). The cord immediately adjacent to the site of rupture was grossly atrophic with irregular cystic spaces, severe loss of axons and myelin, gliosis, and a scattering of foamy macrophages seen microscopically. Destruction of the phrenic nucleus and its efferent fibers at the C3–C5 cord level led to Wallerian degeneration of the phrenic nerves and neurogenic atrophy of the diaphragm. Destruction of long tracts at the C3–C4 cord level caused Wallerian degeneration in descending fibers below C4, associated with a “dying-back” degeneration, in which the descending corticospinal tracts above the site of transection showed loss of axons and myelin up to the pons, and the ascending spinothalamic tracts and posterior columns below the lesion showed similar changes down to the lower thoracic levels. Also, a posttraumatic syrinx replaced much of the gray matter at the C5 level and continued down to the T9 cord level, resulting in neurogenic atrophy of the intercostal muscles.

Case 2

Baby girl G was born at term after an uneventful pregnancy. Labor was spontaneous with a left occipital posterior presentation. Midforceps were used. The birth weight was 3.65 kg, and Apgar scores were 1 at 1 min and 3 at 5 min. The child was intubated and ventilated because of severe asphyxia and transferred to our hospital. Evaluation showed the baby was well formed and of appropriate size for gestational age. Physical examination was unremarkable except for the neurologic findings. There was persistent apnea with hypotonia of the trunk and extremities. All deep tendon reflexes were absent with minimal response to painful stimuli without central appreciation. A large cephalohematoma was noted overlying the right occiput.

CT myelography was performed on the first day of life. Despite an atraumatic spinal puncture, the CSF remained bloody. There was occipital–C1 subluxation with a swollen cervical cord with irregular margin suggestive of possible laceration. Other early investigations included visual evoked potentials, which were normal, and somatosensory evoked potentials, which were absent on the left and abnormal on the right. Sonographic examinations performed at 6 weeks and 3 months of age demonstrated progressive marked narrowing of the anteroposterior cord diameter (Fig. 2A). The cord surface was irregular in the region of narrowing, with increased cord echogenicity and loss of the central canal echo. Repeat CT myelography at 3 months also demonstrated the altered cord dimensions with a ques-

tionable dural tear at the level of the dens (Fig. 2B). The caudal cervical cord assumed a normal configuration.

The child died at 3 months of age, and autopsy revealed total disruption of the cord at the C1 level. Microscopically, the cord adjacent to the brain showed vigorous gliosis, collections of foamy macrophages, and numerous swollen axons. Interruption of long tracts at the C1 level caused Wallerian degeneration in descending and ascending tracts. No posttraumatic syrinx was seen in the surviving spinal cord, and meningeal overgrowth, Schwann cell proliferation, and “dying-back” degeneration were absent.

Case 3

Baby girl N was born at 42 weeks gestation. The prenatal history was uneventful. Presentation was cephalic and labor was induced with prostaglandin, which stimulated rapid cervical dilatation. Despite attempts with both vacuum suction and forceps extraction, vaginal delivery was unsuccessful. The amniotic fluid was meconium stained at cesarean section. The birth weight was 3.55 kg, and Apgar scores were 1 at 1 min, 3 at 3 min, and 3 at 10 min. The child required immediate intubation and was transferred to our hospital. Neurologic examination showed persistent apnea, flaccid paralysis below the neck with absent reflexes, and lack of spontaneous movements to painful stimuli. No other birth injuries were noted. Some caput succedaneum was seen but physical examination was otherwise unremarkable. CT myelography performed the same day showed mild diffuse enlargement, probably the result of hematomyelia and swelling of the cervical cord. Sonography of the cervical region done at 6 and 12 weeks showed progressive decrease in cord size. Subsequent course (follow-up now in 3 months) has shown clinical improvement with return of all reflexes, spontaneous movement of the arms, and improved tone.

Case 4

Baby boy H was born at 41 weeks gestation to a 23-year-old known carrier of hemophilia A. Spontaneous onset of labor occurred with spontaneous rupture of the membranes some 20 hr prior to delivery. The labor was augmented with oxytocin, and epidural anesthesia was administered. Vaginal delivery failed twice, including an attempt with forceps extraction secondary to cephalopelvic disproportion. Late decelerations with poor beat-to-beat variability were noted and a cesarean section was performed. Thick meconium was noted at delivery. The Apgar score at 1 min was 1 with no spontaneous respirations. Immediate intubation and resuscitation were instituted. The baby was then transferred to our hospital. The child was well formed with a birth weight of 3.34 kg. A large right cephalohematoma was noted. He was apneic and bradycardic whenever disconnected from the respirator. Normal facial movements as well as brainstem reflexes (grasp, suck, and corneal) were present. The baby was areflexic with spontaneous movements of the left upper extremity only. Weak withdrawal to pain was seen in all extremities.

Pertinent laboratory investigations demonstrated a hemoglobin of 18.2 g/dl (182 g/l), WBC 26,000 with normal platelet count, the PT was 15.9 sec, while the PTT was greater than 150 sec. Factor VIII assay was less than 1%. Cranial sonography showed diffuse bilateral increased periventricular echogenicity with slitlike ventricles compatible with hypoxic ischemic encephalopathy. A head CT scan was compatible with severe hypoxic ischemic encephalopathy and moderate subarachnoid hemorrhage. Sonography of the cervical cord showed widening of the cord at the cervicomedullary junction and marked increased spinal cord echogenicity suggestive of bleeding into the cord as well as extradural hemorrhage (Fig. 3).

A factor VIII infusion was able to raise the level only to 42%. Generalized seizures developed and the infant deteriorated rapidly over the subsequent 48 hr. He died at 3 days.

Fig. 1.—Case 1.

A, Transverse CT myelogram with flattened upper cervical cord.

B, Sagittal sonogram showing small brainstem caused by cord degeneration. Note widened CSF space (arrow).

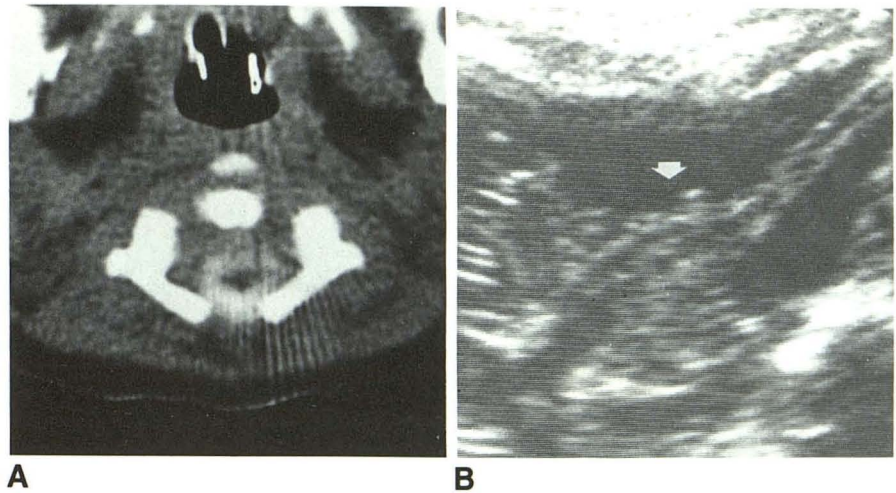
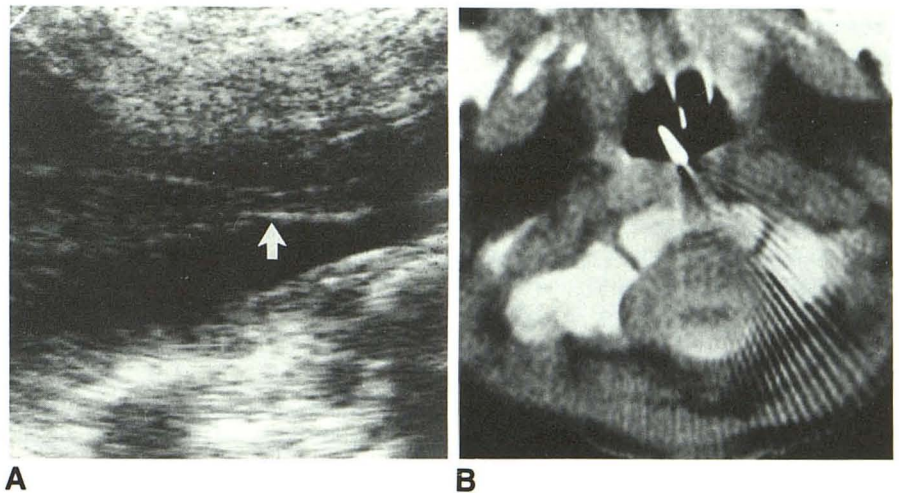


Fig. 2.—Case 2.

A, Sagittal sonogram showing spinal cord narrowing at cervicomedullary junction with widened CSF space and slight increased echogenicity at age 3 months (arrow).

B, Axial CT myelogram showing marked flattening of upper cervical cord.



An autopsy revealed a large subgaleal hematoma, related to forceps manipulation, and tearing of the dura and most of the spinal cord at the cervicomedullary junction. There was edema of the lower brainstem and severe hematomyelia with blood filling the dura at the site of cord laceration and extending down in the central gray matter to the mid-thoracic level. The hematomyelia caused destruction of the phrenic nuclei and the thoracic motor neurons supplying the intercostal muscles, resulting in respiratory arrest after birth and hypoxic-ischemic encephalopathy with necrosis of neurons throughout the brain.

Discussion

Severe spinal cord injury resulting from birth trauma is infrequent, generally occurring after a difficult breech extraction or, more rarely, secondary to cephalic delivery. The incidence of milder degrees of injury is not known but autopsy estimates of significant neonatal spinal cord and brainstem injury derived from infants dying in the first few weeks of life are about 10% [1]. The mechanism of injury is excessive longitudinal traction on the spinal cord especially when combined with flexion, hyperextension, or torsion. Prematurity, intrauterine malposition, asphyxia, dystocia, and precipitate delivery are lesser factors [2, 5, 8]. Although involvement of any part of the spinal cord may be seen, the site of injury generally depends on the presentation. With breech deliveries the lower cervical and/or upper thoracic spinal cord is most commonly injured, while in cephalic presentation it is the cervical spinal cord cephalad to the brachial plexuses [2-4].

Towbin [1] has described three main pathologic patterns of injury that may occur alone or in combination: (1) meningeal damage with epidural hemorrhage, the most common manifestation of spinal injury in the newborn; (2) laceration and avulsion of spinal nerve roots; and (3) laceration and distortion of the cord ranging from edema, focal hemorrhage, and necrosis to complete cord transection.

Clinical evaluation of spinal cord birth injury is difficult, as the typical physical findings of truncal and extremity hypotonia, absent deep-tendon reflexes, and paradoxical breathing patterns can be seen in other pathologic processes [6, 7]. A sensory level may not be evident [6, 7]. Associated cerebral lesions often from asphyxia and brachial plexus injury can combine to make differentiation impossible. Clinical suspicion is often triggered by a history of a difficult breech extraction; however, similar injuries following easy cephalic delivery or cesarean section have been reported [4]. The diagnosis is often missed in the neonatal period as the condition may be confused with spinal muscular atrophy, respiratory distress syndrome, or cerebral palsy [6, 7].

Radiologic investigation plays a key role in the management of these patients, often suggesting the diagnosis or excluding other conditions [9]. Plain films of the cervical spine are usually normal but should be obtained because fractures or dislocations may be seen, as in case 2. The chest radiograph may show a bell-shaped thorax. Myelography with CT has been the single most important method of radiologic investigation

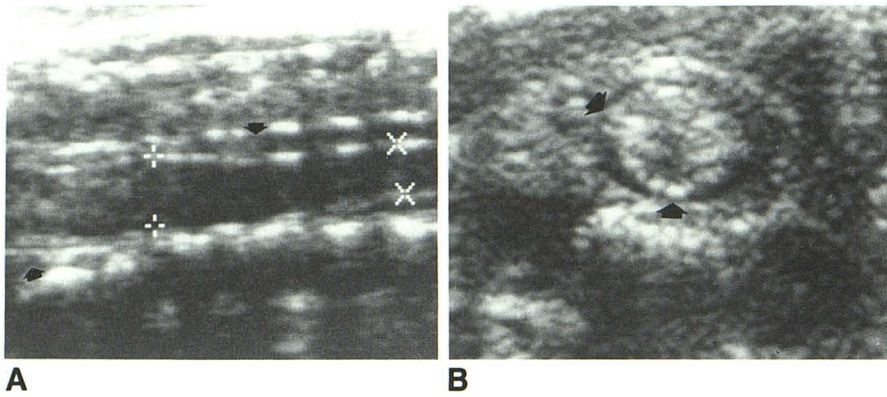


Fig. 3.—Case 4.

A, Longitudinal sonogram of upper cervical cord shows widening of cord with increased echogenicity at cervicomedullary junction just to left of + signs. Extradural hemorrhage along outside of cord (arrows). Normal cord outlined by Xs.

B, Transverse sonogram at level of + signs in Fig. 3A shows widening cervicomedullary junction with replacement of normal cord hypoechoogenicity with markedly increased hyperechogenicity of cord (arrows). Central canal not seen.

in cases in which the possibility of spinal cord trauma have been raised [9]. Recently, MR has been of benefit in evaluating both acute and chronic spinal cord trauma [10, 11]; however, present availability of MR is still limited and most MR suites are not well equipped with the necessary complex life-support and monitoring systems needed for neonates.

The CT myelographic appearance seems dependent on the time interval following trauma. Initially, there is cord edema, hematomyelia, and extradural hemorrhage giving rise to widening of the cord often coupled with complete block. As the cord swelling resolves, the cord diameter may appear relatively normal on CT, obscuring the diagnosis; later there is progression to a small cervical cord. Overlying fibrosis may lead to extradural block as well.

Sonography has gained an increasing role in the evaluation of neonatal spinal lesions including congenital syringomyelia, tumors, and spinal dysraphism [12]. Because of the incomplete ossification of the posterior elements, scanning of the neonate is easily performed with high-frequency near-focus transducers with or without water bath. The appearance of the normal cord and craniovertebral junction is well known [12, 13]. Sonography has been shown of benefit in intraoperative evaluation of spinal cord trauma, demonstrating vertebral fractures, malalignment, epidural hematomas, dural-archnoid adhesions, myelomalacia, and posttraumatic cysts [14]. Neonatal sonographic evaluation in spinal cord birth injury has not been previously published to our knowledge. Our cases demonstrate several advantages of sonographic evaluation compared with CT myelography, including the ability to easily perform initial and follow-up examinations without sedation at the bedside. The study is nontraumatic to the patient and is easily reproducible. Although our study is limited by the small number of patients, we believe that initial cord edema, hematomyelia, and hemorrhage outside the cord can be assessed. The surface irregularity of the cord and subsequent cord caliber changes from axonal degeneration are easily seen on sagittal scans. Altered increased echogenicity of the cord with loss of central canal differentiation allows the diagnosis of myelomalacia, especially when combined with progressive decrease in cord caliber [14].

Pathologic correlation shows that damage to the spinal cord resulting from birth trauma may be much more extensive than is at first apparent. Early on, the associated hemorrhage both into and surrounding the spinal cord masks the disruption of the neural elements. Further damage to the nervous system follows the initial injury. In cases 1 and 2 there was massive Wallerian degeneration of axons distal to their point

of transection. Patient 1, who survived the longest, also showed a "dying-back" degeneration of nerve fibers proximal to their point of transection; for example, the corticospinal tracts were dying back up to the level of the pons, and the spinothalamic tracts were dying back to the lower thoracic cord level. Case 1 also showed extensive proliferation of meningeal tissue and Schwann cells in the surviving cord near the site of the tear. This was not seen in patients 2 and 4, who survived only 3 months and 3 days, respectively. Post-traumatic syrinx can occur, as was seen in patient 1.

Sonography is of benefit in evaluating neonates with severe spinal cord injury. It should also be helpful in assessing lesser degrees of injury, including nerve root avulsions and focal hemorrhages. It is hoped that sonography will allow clinicians to pursue their clinical suspicions more frequently and that it will be used to demonstrate milder degrees of injury resulting from traumatic delivery or to evaluate other types of birth injury, such as clavicular fracture or cephalohematoma.

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