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Intraventricular Air and Meconium-Stained Amniotic Fluid from Intrapartum Rupture of Myelomeningocele

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We report a case of intrapartum rupture of a myelomeningocele in which air and meconium-stained amniotic fluid were forced into the ventricular system.

Case Report

An infant girl was born to a 24-year-old primigravida after a 36-week pregnancy complicated by polyhydramnios. Presentation was cephalic, delivery spontaneous. The membranes ruptured during labor. The infant's weight was 2.4 kg, length 46 cm, head circumference 32.3 cm. The amniotic fluid was meconium-stained. A sacral myelomeningocele, 4.5 cm long, with a ruptured sac was apparent. Apgar scores were 3 and 6 at 1 and 5 min, respectively. Because respirations remained poor, the infant was transferred to the intensive care nursery at Children's Medical Center. On arrival at age 4 hr, her severe respiratory distress precluded immediate

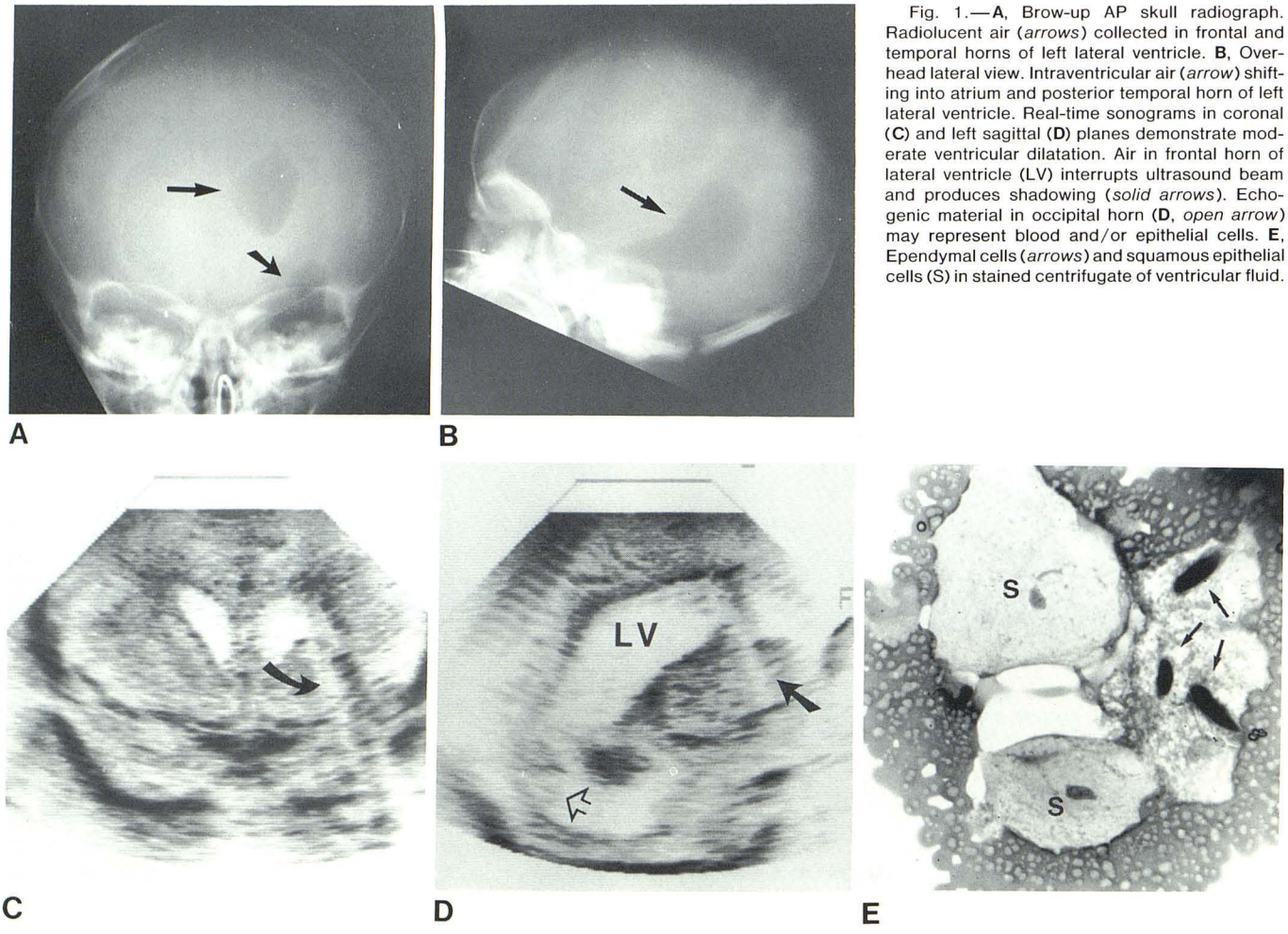

Fig. 1.—**A**, Brow-up AP skull radiograph. Radiolucent air (*arrows*) collected in frontal and temporal horns of left lateral ventricle. **B**, Overhead lateral view. Intraventricular air (*arrow*) shifting into atrium and posterior temporal horn of left lateral ventricle. Real-time sonograms in coronal (**C**) and left sagittal (**D**) planes demonstrate moderate ventricular dilatation. Air in frontal horn of lateral ventricle (LV) interrupts ultrasound beam and produces shadowing (*solid arrows*). Echogenic material in occipital horn (**D**, *open arrow*) may represent blood and/or epithelial cells. **E**, Ependymal cells (*arrows*) and squamous epithelial cells (S) in stained centrifugate of ventricular fluid.

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surgical closure of the spinal defect. She was very irritable and had almost continuous seizure activity. The anterior fontanelle was tense and bulging. Tendon reflexes were markedly hyperactive and no motor paralysis was detected in the lower extremities.

Skull radiographs obtained shortly after admission (figs. 1A and 1B) showed a considerable amount of air in the left ventricle. Sonograms (figs. 1C and 1D) demonstrated mildly enlarged ventricles with amorphous echogenic material in the occipital horn of the left lateral ventricle and shadowing from air in the frontal horn.

During the next few days the infant became less irritable, the fontanelle became soft and flat, and respirations improved. The myelomeningocele was closed surgically on the fourth day of life. Because of continuous leakage of spinal fluid from the back wound, a ventriculoperitoneal shunt was installed on day 15. Meanwhile her neurologic status worsened. She became less alert, seizures persisted despite anticonvulsive therapy, and apneic spells became more frequent, necessitating intubation and mechanical ventilation. A computed tomographic (CT) scan on day 17 showed enlarged lateral ventricles and a large porencephalic cyst in the occipital area. The infant's condition continued to deteriorate and she died on day 44. Permission for an autopsy was refused.

During the shunt placement ventricular fluid was aspirated for laboratory studies. Culture produced no growth. The cell count was 14/mm^3 with 60% polymorphonuclears and 40% lymphocytes. In addition to leukocytes, the stained centrifugate showed clumps of ependymal cells and stratified epithelial cells (fig. 1E).

Discussion

The presence of ventricular air on the initial skull film, of echogenic intraventricular material on sonography, and the finding of stratified epithelial cells in the ventricular fluid strongly suggest that during labor, air and meconium-stained amniotic fluid were forced through the ruptured myelomeningocele sac and entered the ventricular system.

It is well known that vaginal delivery is associated with increased risk to the infant with myelomeningocele [1–3]. Our case illustrates one of the mechanisms whereby perinatal neurologic injury may occur with this defect.

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