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Case Reports

Accidental Intracranial Hyperalimentation Infusion

ED BEAUMONT, M.D., SETH MALIN, M.D., AND ROBERT MENY, M.D.

From the Section of Newborn Services, the Department of Pediatrics and Communicable Diseases, The University of Michigan Hospitals, Ann Arbor, Michigan, and the Neonatal Intensive Care Unit, Hurley Hospital, Flint, Michigan

ABSTRACT. A case of infusion of hyperalimentation fluid into the subarachnoid space is presented. Massive loss of cerebral and cerebellar tissue and cerebrospinal fluid inflammation distinguish this case from a previous report.

One of the major improvements in neonatal intensive care over the past decade has been the introduction of total parenteral nutrition. Complications, however, are multiple and have been reviewed elsewhere.[1, 2] A fortunately rare complication is described in the following case report.

CASE REPORT

The patient was the 3240-g product of a term gestation born vaginally with Apgar scores of 8 and 9. An omphalocele with a base 10 cm in diameter was noted immediately. The defect was felt to be too large to close primarily, but was enclosed in a Silastic chimney and the contents were gradually returned into the abdomen. The baby was placed on peripheral hyperalimentation and was in room air by the 2nd postoperative day. Neurologic examination at this time was normal.

On the 12th day of life, definitive closure of the omphalocele was accomplished and the infant was weaned successfully from the ventilator. Peripheral hyperalimentation solution (dextrose, amino acid solution, and fat emulsion) was restarted via scalp vein several centimeters distant from the margin of the anterior fontanel using a plastic IV-overneedle catheter. On the 4th postoperative day, the child began to have generalized seizures shortly after the administration of ampicillin. Following reintubation and administration of anticonvulsants, a lumbar puncture was performed. The cerebrospinal fluid (CSF) was milky in color. Lab studies included: CSF glucose 777 mg% (serum 150 mg%), CSF protein 130 mg%, CSF triglyceride 69 mg% (serum 58 mg%), and microscopic exam revealed only 2 WBC/mm^3. Massive cerebral edema ensued and was treated with hyperventilation, osmotic diuretics, and steroids. Repeat lumbar puncture at 48 hr showed a CSF leukocytosis with 1120 WBC/mm^3, 93% of which were polymorphonuclear leukocytes (PMN). A third lumbar puncture the following day demonstrated an increase in the cell count to 6480 WBC/mm^3, although CSF protein was 397. Cultures of all specimens were negative.

Clinically, the child failed to initiate spontaneous respirations and remained ventilator-dependent until her death. She demonstrated minimal movements of her extremities, marked hypotonia and hyporeflexia, and was able to grimace, suck on her endotracheal tube and had wandering eye movements. Serial brain computerized tomography (CT) scans showed cerebral edema with progression to diffuse areas of infarction. Electroencephalogram (EEG) was severely abnormal with a nearly isoelectric recording. The child was transferred to our unit for reevaluation.

On arrival 6 weeks postinjury, the physical examination was unchanged. EEG revealed absent activity over the right hemisphere and only a few low amplitude theta-delta waves on the left. Visual evoked responses were absent. Brain stem auditory evoked responses were normal for age. Cranial ultrasound showed severe hydrocephalus. CT showed a generalized decrease in brain density with only brain stem being readily identifiable (Fig 1). The child died a short time after discontinuation of ventilator support.

AUTOPSY RESULTS

Gross Description

Examination of the skull and brain revealed bilateral subperiosteal hematomas and a right transverse sinus thrombosis. The meninges were markedly adherent to the overlying dura. The brain itself weighed only 110 g and collapsed when dissected from the dural surface. All normal landmarks were destroyed and all that remained was a thin rim of cortex surrounding a disorganized mass of white matter. The ventricular system was symmetrically enlarged. The pons and medulla appeared to be spared.

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Reprint requests to: Ed Beaumont, M.D., Section of Newborn Services. The University of Michigan Hospitals, Box 007, L3018 Women's Hospital, Ann Arbor, MI 48109.

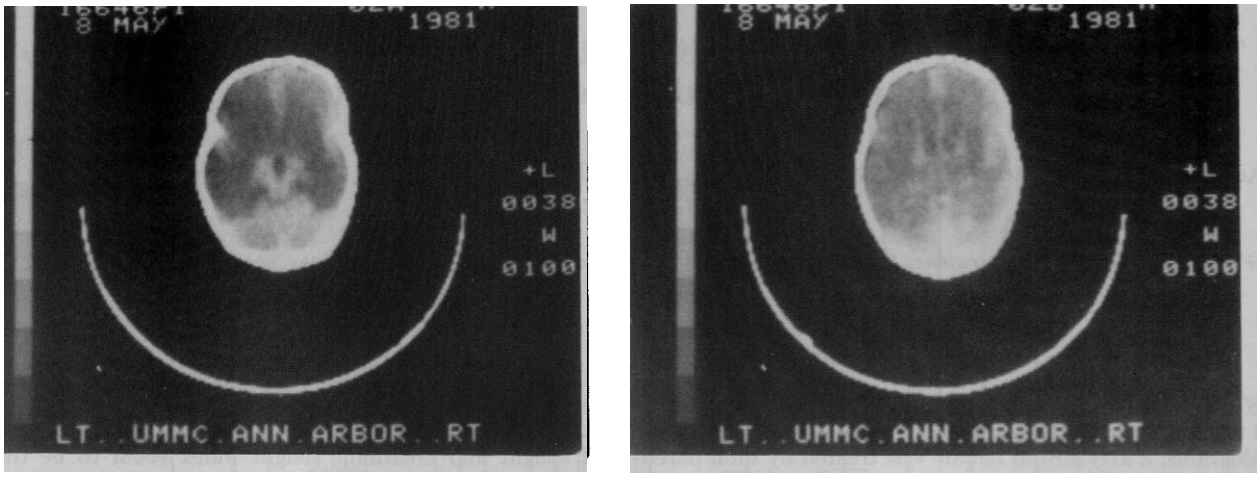

FIG. 1. CT scan showing loss of cerebral cortex and resultant ventriculomegaly.

Microscopic Description

Extensive adhesions existed between the surface of the brain and meninges. The subarachnoid space was hypercellular, with abundant foamy macrophages and focal accumulations of hemosiderin-laden macrophages. In addition, there were areas of granulomatous reaction with multinucleated foreign body giant cells containing lipid adjacent to areas of meningeal fibrosis.

There was dissolution of cortex with an abundance of macrophages and glial cells and only a few remaining viable neurons. Diffuse subependymal gliosis was present. Similar changes were present in the cerebellum. The brain stem architecture appeared preserved.

DISCUSSION

Although an entry wound was not able to be demonstrated, it is clear from the CSF laboratory findings that the hyperalimentation solution and lipid emulsion had reached the subarachnoid space. The scalp vein intravenous line had been inserted 14 hr prior to the onset of seizures. The IV rates were hyperalimentation fluid 20 cc/hr and lipid solution 5 cc/hr. In contrast to an earlier case report,[3] this infant mounted a significant inflammatory response as manifested by CSF pleocytosis, adhesions, and the histologic findings. Although individual variation in inflammatory response might play some role, it seems reasonable to implicate the ampicillin, hyperalimentation solution, and/or volume infused as being responsible for the difference in reaction. The presence of a highly acidic pH, hyperosmolar load, or sclerosing agents might account for the severe damage done to the brain. Extreme caution must be exercised when using scalp veins for IV infusions.

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