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# Severe Hyperglycemia and Intracranial Hemorrhage in a Premature Infant with Fetal Malnutrition and Pulmonary Infection*

**Gülşen Erdem, M.D.** / Safiye Göğüş, M.D.*** / Müyesser Tuncer, M.D.*** / Sadık Demirsoy, M.D.******

Hyperglycemia in newborn infants is a metabolic problem as important as hypoglycemia. Hyperglycemia may contribute to the increased morbidity and mortality rate in neonates by causing hyperosmolality. Hyperosmolality in turn accounts not only for osmotic diuresis which results in greater water loss, but also for intracranial hemorrhage.[1]

In this article, we present a twelve day-old premature infant with fetal malnutrition who developed severe hyperglycemia following pulmonary infection, constant dehydration and abundant intracranial hemorrhage.

*Case Report*

**Case:** D. (HCH. 1054341). A 15 hour-old baby girl admitted to Hacettepe Children's Medical Center-Newborn Unit because of prematurity. She was born following the fourth gestation of a 23 year-old healthy mother. Her birth weight was 1580 gm. (below the tenth percentile of Lubchenco's intrauterine growth chart[2]) and gestational age was thirty five weeks. Her general condition was good, during the first days of life. Blood sugar levels were determined as 75-100 mg/dl by using the

* From Hacettepe Children's Medical Center, Hacettepe University, Ankara-Turkey.
** Associate Professor of Pediatrics.
*** Professor of Pediatrics and Pediatric Pathologist.
**** Resident in Pediatrics.

Somogyi-Nelson method.[3] The infant behaved well for the first 10 days. Then she became tachypneic and developed intercostal retraction and slight cyanosis. X-ray of the chest showed disseminated inflammatory changes in both lung fields. Therapy was started for pulmonary infection.

Soon after, the patient developed convulsions and was found to be dehydrated in spite of intravenous fluid (glucose 6 mg/kg/min.)for 2 days. Anticonvulsive drugs were administered. Blood sugar was measured as 400 mg/dl. Urine analysis showed specific gravity of 1028, 2 plus sugar which was confirmed chromotographycally to be glucose and no aceton was traced in the urine. Blood electrolytes and urea nitrogen levels were normal. However, the following data was recorded 24 hours later: Blood sugar was elevated to 3500 mg/dl; blood and urine osmolality were 405 mOs/kg. and 546 mOs/kg., respectively. Regular insulin was administered at a dose of 2 u/kg. subcutaneously, twice, at 1 hour intervals. Her condition deteriorated progressively. An extra insulin infusion of 0.1 u/kg./hr. was given.[4,5] Prolonged episodes of cyanosis and convulsions occured with no response to the treatment. Postmortem lumbar puncture was performed and disclosed bloody cerebrospinal fluid.

Postmortem examination revealed abundant subarachnoid hemorrhage on the left temporo-parietal area (Figure 1), intraventricular and intracerebral hemorrhages as well as bacterial bronchopneumonic infiltration. The pancreatic tissue was intact. No thrombo embolic phenomena could be detected in the central nervous system or elsewhere.

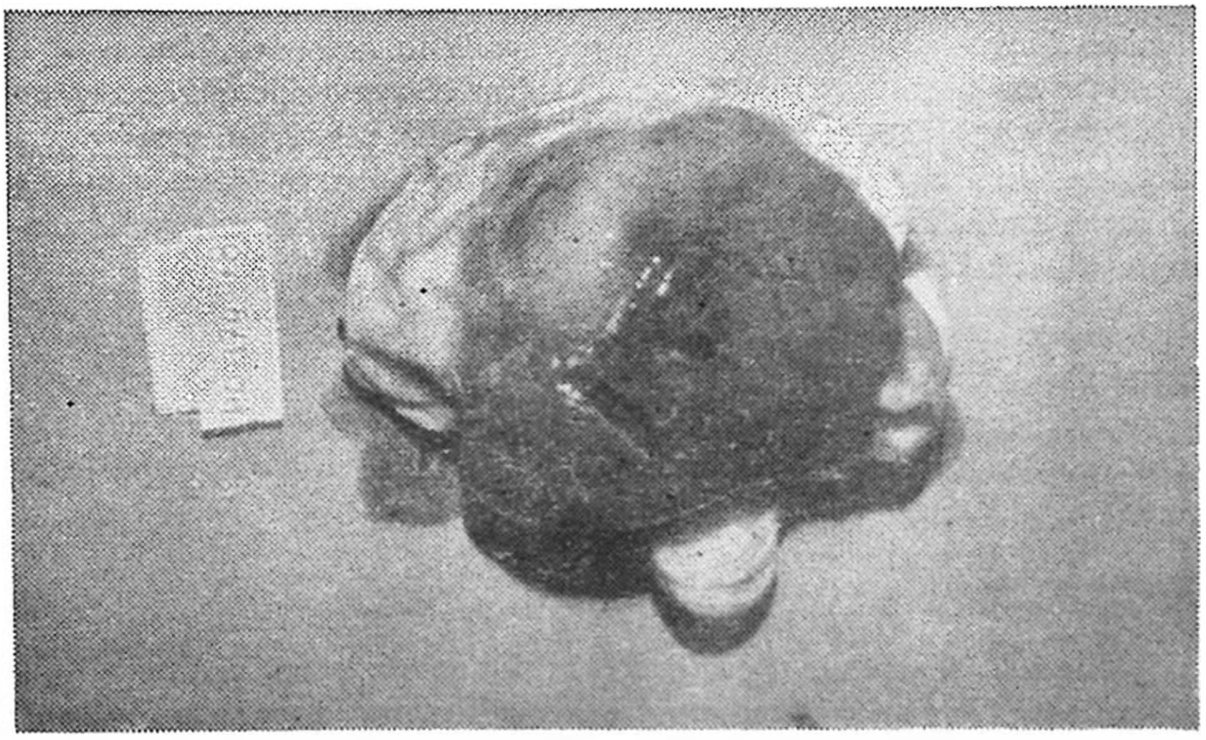

**Figure 1**

## *Discussion*

Hyperglycemia is a common problem in small premature infants who receive glucose infusions during the first 48 hours of life.[1,6-7] In spite of many experimental and clinical studies, the pathogenesis of early hyperglycemia has not been explained.[8,9] However, the present case should be considered separately since hyperglycemia developed not before the 10th day of life.

Transient diabetes mellitus in early infancy is another discrete clinical entity that has been described in small-for gestational age-full term infants during the first six weeks of life.[10-11] In the pathogenesis of this clinical entity, infection accounts for 28 % of the cases.[10]

It is known that such stressful situations as infection, burn, trauma and surgical procedures can cause impaired carbohydrate tolerance by increasing the tendency towards hyperglycemia in adults.[12] It has been shown that the endogenous glucose production; glucogenolysis, gluconeogenesis and lipolysis increases during sepsis.[13,14] In addition, insulin antagonist hormons, glucagon, glucocorticoid and cathecolamine also increase during infections.[14-17] In cases of juvenile diabetes, carbohydrate intolerance and requirement for insulin increase during infection and other stress.[18]

In our case, the presence of pulmonary infection may be considered as a trigger mechanism impairing the carbohydrate metabolism thus resulting in hyperglycemia. There have been no reported studies on the mechanism of this type of hyperglycemia in neonates. However, it could be explained by the same mechanism of hyperglycemia observed in adults with infection. Precise knowledge on late neonatal hyperglycemia secondary to infection, can be obtained in future by measuring the levels of insulin, insulin receptors, glucagon, glycocorticoid and cathecolamine of such babies.

In our case, it is felt that hyperglycemia is primary. Intracranial hemorrhage may be secondary to a rapid dehydratation as well as to an altered cerebrospinal fluid pressure in response to hyperglycemia. The latter mechanism was also postulated by a recent study in puppies by Arant et al.[19]

## *Summary*

The role of pulmonary infection in the development of hyperglycemia in a twelve day-old premature infant with severe hyperglycemia and intracranial hemorrhage is discovered.

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