Neonatal hypoglycemia

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The limit of hypoglycemia in newborns is not clearly defined, it depends on the gestational and postnatal age of the newborn, on concurrent pathological conditions and on the individual susceptibility of the individual to hypoglycemia. Glucose concentration varies depending on the sampling method – in plasma and serum, glycemia is 10-15% higher than in whole blood.

According to one of the definitions, hypoglycemia is defined as glycemia ≤ 2.2 mmol/l in the first 24 hours of life and **glycemia ≤ 2.5 mmol/l** after the 24th hour of life. In newborns, hypoglycemia is often transient, the lowest values usually reach 30 - 90 minutes after birth.

Etiology

Endocrine regulation of glycemia.

Causes of transient hypoglycemia

1. maternal metabolic complications:
   - excessive intrapartum glucose supply;
   - medications: terbutaline, ritodrine, propranolol, oral antidiabetics;
   - diabetes mellitus in pregnancy;
   - obesity, preeclampsia;
2. neonatal pathological conditions:
   - fetal growth restriction, hypotrophy; hypertrophy; prematurity;
   - postpartum hypoxia and asphyxia; sepsis; rival;
   - hypothermia/hyperthermia; hemolytic disease, polyglobulia;
   - transient neonatal hyperinsulinism;
   - insufficient glucose intake or sudden interruption of glucose supply.

Causes of persistent or recurrent hypoglycemia

- hypoglycemia lasts more than 7 days;

1. hyperinsulinism: pancreatic beta-cell hyperplasia, nesidioblastoma, adenoma, Beckwith-Wiedemann syndrome;
2. endocrine disease: pituitary insufficiency (growth hormone deficiency, septo-optic dysplasia), cortisol deficiency, congenital glucagon deficiency, adrenal medulla insufficiency;
3. inborn errors of metabolism: galactosemia, fluctosemia, glycogenosis type I, maple syrup disease, methylmalonic and propionic aciduria, tyrosinemia, carnitine defect, acetyl-CoA dehydrogenase and others.
4. neurohypoglycemia: disorders of glucose transport.

Clinical picture

- Hypoglycemia is often asymptomatic in newborns.
- Symptoms of hypoglycemia include:
  - sucking disorders, vomiting,
  - irritability, tremors, hyperreactive Moro reflex, central high-pitched crying, twitching, convulsions, hypotonia, coma,
  - tachycardia/bradycardia, tachypnea, grunting, cyanosis, periodic breathing, apnoeic pauses, pallor,
  - temperature instability.

Indications for postnatal hypoglycemia screening

Diagnostics and laboratory tests

Transient hypoglycemia

Persistent hypoglycemia

Treatment

Pathophysiology

Fetal metabolism

During pregnancy, the fetus receives the energy and nutrients needed for growth from the mother through the placental circulation and creates reserves necessary after birth. Glucose crosses the placenta using facilitated diffusion, but during maternal starvation or placental insufficiency, the fetus is capable of its own endogenous glucose production. Glucose metabolism provides 65% of the total energy production of the fetus, the rest is probably provided by lactate.

Glucose is not the only source of energy for the fetal brain. Studies show that the brain takes up ketones (products of fatty acid beta oxidation) more than glucose, and it is likely that ketones are used not only for incorporation into brain lipids but also as a source of energy for the brain. Lactate can also be metabolized.

The fetus is usually able to regulate its glucose circulation independently of maternal glycemia and placental transfer. This can be seen in some cases of placental insufficiency, when gluconeogenesis of the fetus is activated at the expense of its growth and storage, and in fetuses of mothers with suboptimal control of diabetes, when in response to a high supply of glucose through the placenta, the fetus increases insulin production, which leads to its increased growth and storage. Fetal metabolism differs from that of the adult in that the fetal insulin response to high glucose levels is attenuated and that insulin production is more sensitive to amino acid levels than to glucose. Insulin appears to play a more important role in fetal growth than in metabolic control. In extreme situations, there is a failure of glycemic control in the fetus, for example in severe prolonged placental insufficiency.

Metabolic changes during childbirth

During childbirth, the continuous supply of nutrients from the placenta is suddenly interrupted and metabolic changes occur immediately in order to maintain the supply of energy and nutrients for the function of vital organs.

The oxygen supply is temporarily interrupted, therefore anaerobic metabolism must be started, which, however, requires a higher availability of substrates than aerobic metabolism. In addition, the newborn must adapt to the starvation-feeding cycle and also to the fact that instead of glucose from the placenta, the main source of energy is now fat from the stored adipose tissue and from the milk diet. After delivery, the plasma level of insulin decreases and there is a rapid washout of catecholamines and pancreatic glucagon. These endocrinological changes lead to the release of enzymes key to glycogenolysis (release of glucose from stored glycogen in the liver, heart muscle and brain) and gluconeogenesis (formation of glucose in the liver), lipolysis (release of fatty acids from stored adipose tissue) and ketogenesis (beta oxidation of fatty acids in the liver). In the immediate postpartum period, the main source of energy for most organs is glucose. However, it turns out that lactate appears to be preferred over glucose and ketones in the brain.

Newborn Metabolism

Metabolic changes similar to those during childbirth, however, on a significantly smaller scale, occur in the newborn during fasting-feeding cycles. Immediately after feeding, energy sources (fatty acids and to a lesser extent carbohydrates) from milk are available. Some organs, such as the kidneys, use only glucose as an energy source, while others, such as the brain, are able to use ketones as well. Excess glucose from the diet is stored as glycogen in the liver or converted to fat and stored in adipose tissue, as are excess fatty acids absorbed from the diet. With a certain distance after each feeding, there is a decrease in blood glucose and the activation of glycogenolysis and gluconeogenesis. After glycogen is depleted, gluconeogenesis is the main source of glucose. A newborn produces glucose at a rate of about 4-6 mg/kg/min. An alternative source of energy for some organs (e.g. the brain) is lipolysis and ketogenesis. Ketogenesis is simultaneously a source of energy and cofactors needed for gluconeogenesis.

The metabolism of the newborn depends on the formation of key enzymes (glycogenolysis - liver phosphorylase; gluconeogenesis - phosphoenolpyruvate carboxykinase; ketogenesis - carnitine acyltransferase) and the triggering of enzymatic activity by hormonal changes. The main hormone regulating glucose metabolism in the newborn is glucagon. Its concentration rises when blood glucose levels drop. Glucagon induces the activity of glycogenolysis, gluconeogenesis and ketogenesis enzymes in the liver. Neither insulin nor catecholamines, cortisol and thyroid hormones nor growth hormone have a very significant role in glucose metabolism in newborns, but in extreme cases they affect it (hyperinsulinism leads to hypoglycemia, as well as rare cases of hypopituitarism and cortisol deficiency). The adaptation of the digestive tract also significantly affects the metabolism of the newborn. The initiation of enteral nutrition triggers the production of gastrointestinal regulatory peptides and hormones involved in intestinal adaptation (intestinal growth, mucosal differentiation, initiation of motor activity and development of digestion and absorption).

Differences between the energy metabolism of a newborn and an adult

In newborns fed with milk, during the normal cycle of starvation-feeding, the formation and use of ketones occurs to an extent that is usual in adults only during prolonged starvation. In addition to glucose and ketones, the source of energy for newborns is also lactate. Insulin plays a less significant role in glucose metabolism in neonates than in adults, and a lower sensitivity of target organs to insulin is also likely. Disorders of glucose metabolism in newborns cannot be compared with adults, but it is necessary to relate them to the metabolism of healthy newborns. Neonatal hypoglycemia should be viewed more as a disturbed adaptation of its metabolism than as a strictly pathological condition.[1]

References

External links

- On-line newborn percentile calculator (Intergrowth) (http://intergrowth21.ndog.ox.ac.uk/en/ManualEntry/Compute)

Related Articles

- Hypoglycemia
- Glycemia

Reference

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