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FEATURES OF SPASMOPHILIA IN CHILDREN

ANNOTATION

Spasmophilia is a disease of young children, characterized by impaired mineral metabolism, increased neuromuscular excitability, and a tendency to spastic and convulsive states. The disease develops in 3.5-4% of children suffering from rickets. Clinically, hidden (latent) and overt spasmophilia are distinguished. Spasmophilia is diagnosed based on the clinical picture in the form of signs of increased neuromuscular excitability, periodically occurring spastic states of individual muscle groups. The diagnosis is also confirmed by the presence of hypocalcemia in combination with hyperphosphatemia, imbalance of blood electrolytes, and alkalosis. With timely treatment, the prognosis of the disease is favorable.

KEY WORDS: *spasmophilia, children, hypocalcemia, convulsions, neuromuscular excitability.*

Spasmophilia is a disease that occurs in young children due to the acute development of hypocalcemia against the background of disturbances in mineral metabolism and acid-base balance. This pathology is detected mainly at the age of 3 months to 2 years, i.e. in the same age range as rickets, with which spasmophilia has an etiological and pathogenetic connection [1-5]. Just a few years ago,

spasmophilia in pediatrics was quite common, but a decrease in the incidence of severe forms of rickets has led to a decrease in the number of cases of infantile tetany. Spasmophilia develops in 3.5 - 4% of children suffering from rickets; somewhat more often in boys [11-15].

The connection between spasmophilia and rickets has long been known, but was proven only in the 70s, when a low level of 25 hydroxycholecalciferol was discovered in the blood of children with spasmophilia.

ETIOLOGY AND PATHOGENESIS

The cause of spasmophilia is a decrease in the level of ionized calcium in the blood serum against the background of electrolyte imbalance, hyperphosphatemia and alkalosis. The mechanisms of development of hypocalcemia are important, which can be caused by dysfunction of the parathyroid glands, decreased absorption of calcium in the intestine, or increased excretion in the urine.

The development of spasmophilia is usually pathogenetically associated with the period of convalescence from moderate or severe rickets. Acute overproduction of the active form of vitamin D suppresses the function of the parathyroid glands, stimulates the absorption of calcium and phosphorus salts in the intestine and the reabsorption of them and amino acids in the renal tubules [6,9,12]. As a result, the alkaline reserve of the blood quickly increases and alkalosis develops.

Calcium begins to be intensively deposited in the bones, so its level in the blood quickly decreases to critical (below 1.7 mmol/l). At the same time, hyperkalemia occurs. Hypocalcemia, alkalosis and hyperkalemia determine the child’s convulsive readiness and increased excitability of his nervous and muscular systems [7,10,15].

An increase in the level of inorganic phosphorus in the blood can be promoted by feeding the child cow's milk with a high concentration of phosphorus and insufficient excretion of excess phosphorus by the kidneys; hypoparathyroidism. In addition to calcium-phosphorus metabolism,

hyponatremia, hypochloremia, hypomagnesemia and hyperkalemia are observed in spasmophilia [8].

Under these conditions, any external influence accompanied by fear, crying, high fever, vomiting, as well as the addition of an intercurrent disease can provoke spasms of certain muscle groups or clonic-tonic convulsions.

CLINICAL PICTURE

Clinically, hidden (latent) and overt spasmophilia are distinguished. Latent spasmophilia precedes overt spasmophilia. It can be suspected in a child in the presence of anxiety, shuddering, hyperesthesia, periodic tremor of the chin and limbs, twitching of individual muscle groups, dyspnea, which turns into carpopedal spasm, against the background of existing signs of rickets, often in the repair stage.

The most common symptoms of latent spasmophilia are:

- Khvostek's symptom - when tapping between the zygomatic arch and the corner of the mouth (in the area where the fibers of the facial nerve are located), contractions of the facial muscles (twitching) appear in the area of the mouth, nose, lower and upper eyelid;

- Erb's symptom - increased galvanic excitability of the nerves (muscle contraction when the cathode applied to the area of the median nerve is opened, with a current strength below 5 mA);

- Trousseau’s symptom – when the neurovascular bundle on the shoulder is compressed, a convulsive contraction of the fingers occurs in the form of an “obstetrician’s hand”;

- Maslov's symptom - with a slight prick of the skin of a child with spasmophilia, breathing stops at the height of inspiration (in a healthy child, such irritation causes increased and deepening of respiratory movements), this phenomenon is clearly revealed by pneumography;

- Lyust's symptom - rapid abduction of the foot outward with dorsal flexion when struck below the head of the fibula in the area n. fibularis superficialis.

Explicit spasmophilia, as a rule, manifests itself in the form of laryngospasm, carpopedal spasm and eclampsia (sometimes in combination with each other).

Laryngospasm occurs most often when the child is crying or frightened. Moderately severe laryngospasm is accompanied by pallor and difficult hoarse or sonorous inhalation followed by noisy breathing. When the glottis is completely closed, the child is frightened, gasps for air, becomes covered in cold sweat, the skin becomes cyanotic in color, and loss of consciousness is possible for a short time. After a few seconds, a noisy inhalation is heard, breathing is gradually restored and the child, having calmed down, falls asleep. An attack of laryngospasm usually proceeds favorably, but can recur, especially with inadequate treatment. It is extremely rare that an attack of laryngeal stenosis is prolonged and can be fatal. In the most severe cases, sudden cardiac arrest (cardiac tetany) is possible.

Carpopedal spasm is a tonic contraction of the muscles of the limbs, which take a characteristic position (the hand is in the “obstetrician’s hand” position, the foot is in the “pes equines” (“horse foot”) position, with the big toes in a state of plantar flexion). This condition of the hands and feet can be short-term, but can persist for several hours and even days (in the latter case, reactive swelling of the back of the hand and foot appears). Carpopedal spasm can be observed when changing a child's clothes. It is possible to develop spasms in other muscles: ocular (with the development of transient strabismus), masticatory (with trismus and rigidity of the occipital muscles), m. orbicularis oris (lips - in the form of a “fish mouth”), smooth muscles (with urination and defecation disorders), etc.

Eclampsia is a rare and most severe form of spasmophilia. It manifests itself in the form of an attack of clonic-tonic convulsions, which cover all voluntary and involuntary muscles, lasting from a few seconds to 20-30 minutes. The attack begins with twitching of the facial muscles, then convulsions of the limbs, stiffness of the neck muscles occur, laryngospasm and respiratory distress occur, and general cyanosis appears. Consciousness is lost, foam appears at the mouth,

spontaneous bowel movements and urination are observed. Fever may occur. With a prolonged attack of eclampsia, breathing and heartbeat may stop. Children in the first year of life often develop tonic convulsions and laryngospasm, while older children develop clonic convulsions and carpopedal spasm. A very prolonged eclamptic state can negatively affect the central nervous system and cause delayed motor development in the future.

DIAGNOSIS OF SPASMOPHILIA

Spasmophilia is diagnosed on the basis of a typical clinical picture in the form of signs of increased neuromuscular excitability, periodically occurring spastic states of individual muscle groups (facial, laryngeal, respiratory, skeletal). In addition, the child's age, time of year, as well as clinical and radiological symptoms indicating a period of convalescence of rickets are taken into account. If a young child, in the presence of clinical, biochemical and radiological signs of rickets, manifests the above-described picture of increased convulsive readiness and neuromuscular excitability, laryngospasm occurs - the diagnosis is beyond doubt.

The diagnosis is confirmed by the presence of hypocalcemia in combination with hyperphosphatemia, imbalance of blood electrolytes, and alkalosis. Laboratory data reveal a decrease in total calcium less than 1.75 mmol/l, ionized calcium below 0.85 mmol/l, and respiratory alkalosis. The ECG shows prolongation of the QT interval and other signs of hypocalcemia.

TREATMENT OF SPASMOPHILIA

Children with an obvious form of the disease are subject to hospitalization; treatment of the latent form is possible at home. With laryngospasm, it is necessary to provide access to fresh air and create a dominant focus of excitation in the brain by irritating the nasal mucosa (blow into the nose, tickle, bring ammonia), skin (injection, patting and pouring cold water on the face), vestibular apparatus (shaking the child), changes in body position.

In treatment, limit unpleasant procedures (injections, examination of the pharynx) as much as possible, perform them carefully so as not to provoke an attack of laryngospasm, eclampsia.

For convulsions, administration of diazepam is indicated. Diazepam has an immediate but short-term (30 minutes) effect. In cases where the etiology of seizures is not entirely clear, the administration of diazepam gives time to conduct additional examination and verify the cause of tetany. Diazepam is administered at the rate of 0.5% - 0.1 ml/kg body weight intravenously or intramuscularly, but not more than 2 ml once; with a short-term effect or incomplete relief of seizures, re-introduce diazepam at a dose of 2/3 of the initial dose, after 15-20 minutes, the total dose of diazepam should not exceed 4 ml;

At the same time, intravenous administration of calcium-based drugs is carried out. It should be remembered that rapid intravenous administration of calcium can cause bradycardia and even cardiac arrest. The use of parenteral calcium-based drugs is continued until the symptoms are relieved; after the attack is relieved, they switch to oral administration.

For hypocalcemic convulsions, a 10% solution of calcium gluconate is slowly administered intravenously at a dose of 0.2 ml/kg (20 mg/kg) (after preliminary dilution with 20% dextrose solution by 2 times). After 3-4 days, against the background of intensive therapy with calcium solutions, vitamin D is prescribed for the treatment of rickets.

Hypocalcemia is often accompanied by hypomagnesemia - magnesium-based drugs are used in combination with vitamin B6 (magne-B6, etc.). Stopping breathing and heartbeat requires emergency hospitalization and pulmonary-cardiac resuscitation, oxygen therapy.

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