

Clinical Peculiarities of Odontogenic Purulent Inflammatory Diseases in Children

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Relevance of the study: Many issues of etiology, pathogenesis, prevention and treatment of purulent-inflammatory processes of the facial jaw-odontogenic purulent inflammatory diseases are focused on them by researchers. Also in children, acute inflammatory processes of the facial jaw often develop with a decrease in the immunological reactivity of the body, and the course of the disease and the possibility of complications are determined mainly by changes in the initial indicators of immunity.

Against the background of odontogenic inflammatory diseases and their decrease in immunity due to acute infectious diseases in children, their complications occupy a special place in the structure of this pathology.

In children with odontogenic inflammatory diseases, a sharp change in inflammatory processes is observed due to the relative maturity of organs and tissues, imperfection of immunity, incomplete formation of lymphatic tissues, the presence of anatomical and physiological features of the structure of teeth and jaws, susceptibility to injury and its complications.

The results of numerous epidemiological studies show that an average of 3-5 acute respiratory viral infectious diseases have been found to have a tendency to be diagnosed per child per year. The greatest prevalence of acute respiratory infections is seen in young children, preschool children and primary school children. Children in the first 3 years of life are diagnosed with infectious diseases 10-10 times more often than children aged 2-2.5 years and older in one year. Severe respiratory infections lead to a violation of the functional state of the body, lead to a violation of adaptation and the development of chronic pathology.

In children who are often diagnosed, the adverse factors that cause the inflammatory process in the face jaw can be conditionally divided into the following groups::

- Damage to the pulp as a result of odontogen - caries and its complications, inflammations in the periodont, inflammation damage the adrenal curtain;
- Formation of pathology by passing through the physiological pocket of gingival – parodont tissue;
- Mucostomatogenic – damage to the mucous membrane of the oral cavity (virus, fungus) through;
- Intraossal-purulent cysts, cystogranulema, periostitis, osteomyelitis.

The face-jaggerrokh Bernadsky yu. I. Odontogenic inflammations

Oral fluid saliva lesion;

Damage through the nose and upper respiratory tract;

Damage through the throat and oral cavity organs;

There is a correlation between clinical forms of inflammatory processes in the face –jaw. Under the toxic action of microorganisms in small quantities, their moderate concentration and the reaction of action through the body's immune forces, a limited purulent furnace – abscess develops.

In abscess, a complication of odontogenic purulent inflammatory diseases in children with frequent contractions, local complaints vary – from restriction of the opening of the mouth to painful swallowing and depend on the muscles involved in the process. The general condition of patients is characterized as satisfactory – body temperature can be subfebrile, general impurity is noted. The

normative amount of leukocytes in the blood or leukopenia is observed, ECHT has been found to increase imperceptibly within the norm or up to 11 – 20 mm/h.

Phlegmon, a complication of odontogenic purulent inflammatory diseases, is not limited, clinical manifestations differ in a high level of complaints of patients. In odontogenic phlegmon, local and general symptoms characteristic of abscess are detected, only slightly overexpressed.

The odontogenic phlegmon clinic varies depending on the degree of prevalence of the disease. In one or two areas, a hyperergic reaction is characteristic of phlegmon: body temperature ranges from subfebrile to 38.5 oC; in blood analysis, leukocyte levels increase (10-12x10⁹/l), ECHT is observed to increase to 40 mm/h.

Phlegmon from two or more areas is defined as scattered phlegmon. In the clinic of diffuse phlegmon, the reactive, toxic, terminal phases differ. A number of common signs of phlegmonous lesions of the desired location are identified.

Clinical manifestations of odontogenic purulent inflammatory diseases as a result of acute odontogenic phlegmon:

1. A significant state of intoxication of the body, that is, general weakness, tremors, pulse acceleration. In laboratory data: there is a significant leukocytosis, a shift of the leukocyte formula to the left, an increase in ECHT to 70 mm/h.
2. Violation of body temperature control – body temperature sometimes rises to 40 oC.
3. Local clinical signs are determined by the location of the lesion furnace – a tumor spreading to the phlegmon surfaces (under the skin) is characteristic, in this area there is skin hyperemia and pain in palpation; in this, the skin does not accumulate in the fold. Facial asymmetry and skin changes are not detected when the phlegmon is deeply embedded.
4. The following are characteristic of all diffuse phlegmon:
 1. Excitation of the lower jaw to the level of full output.
 2. Chewing when the chewing muscles are involved in the inflammatory process, the act of swallowing is sharply painful.
 3. Speech and breathing disorders
 4. Violation of salivary detachment.

Characteristic features of the development and complications of odontogenic phlegmon are observed in primary and secondary immunodeficiency and in individuals of an older age group. At the same time, the individual capabilities of protective reactions and life support systems differ in each type of one group. Phlegmon, which grows in old age and old age, is more common than in young and middle age groups, when a low-pain, slow-growing infiltrate is detected against the background of laboratory data confirming intensive inflammatory processes.

In 65% of the total number of patients, phlegmon with atypical course in this case, local symptoms against the background of general relief of the disease can be both underexpressed and overexpressed.

Odontogenic osteomyelitis-an infectious purulent-necrotic inflammatory process in the bone tissue of the jaws several scientists call the disease "panostitis", while other authors refer only to a purulent process associated with necrosis of the bone tissue. This form of osteomyelitis develops as a result of the fall of an odontogenic infection. The anatomical peculiarity of the lower jaws determines the special mechanism for the development of odontogenic osteomyelitis – prolonged exposure of microorganisms to the foci of injury inflammatory tissue, as well as the microbial agent, is directly and permanently associated.

Complication of odontogenic purulent inflammatory diseases acute stage of osteomyelitis is accompanied by intensive pain in the area of one, and later several teeth at first. The general condition

is often of medium weight. In restricted osteomyelites, body temperature can be subfebrile, reaching 40 °C in diffuse form, tremor and sweating are characteristic signs of intoxication. For 2-3 days, collateral edema of soft tissues appears, the feeling of pain in palpation increases, there is a significant thickening of the jaw.

In complications of purulent inflammatory diseases, hyperemia in the oral cavity and swelling of the alveolar esophagus both internally and externally, excitability of the teeth in the foci of injury and painful percussion are observed. If the process moves to the entire lower jaw, the purulent exudate spreads to the medial and lateral squamous muscle, creating an inflammatory contracture. Due to the anatomical peculiarities of the lower jaw, osteomyelitis is especially severe. Diffuse sclerosing osteomyelitis in the lower jaw can have symptoms of synovitis, pustulosis, and acne (or Sappho-syndrome).

Odontogenic testifies that the diagnosis of inflammatory processes is insufficient: from 20 to 50% of patients are directed to the stations with a misdiagnosis. In about 30-40% of them, the causal teeth are not removed; patients are not directed to the stationary in time, which aggravates the prognosis of the disease and ensures the transition of inflammation from one form to another.

Conclusion: The patient's body, diagnosed with odontogenic purulent inflammatory diseases, develops endogenous intoxication syndrome – a syndrome of incompatibility in the formation and release of bacterial EXO - and endotoxins, pathological exchange products and inflammatory mediators, as a result of the action of the causative agents of the disease. The expression of signs of intoxication in patients with acute purulent-inflammatory diseases depends on many exogenous and endogenous factors. From this, regular laboratory analyzes should be determined on the basis of their complete data for the analysis of the outcome of the disease.

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