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What is the new face of pertussis?

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I. Introduction:

Pertussis infection is known as a clinical description from ancient times, but to date it continues to be a challenge in terms of early detection, prevention of complications and the widespread prevalence of atypical clinical forms. Because of the non-specific initial symptoms, pertussis is important to be known and actively sought especially among the pediatric population.
Pertussis is an acute infectious disease - anthroponosis, which is presented with spastic coughand reprise, with or without vomiting.

The disease is described by Hippocrates, Celsius and Galen. A complete clinical description made Baillou in France in 1640. In 1679 Sydenham distinguished the disease and gave itsname – pertussis, meaning "strong cough".

The etiology of the diseases is clear -Bordetella pertussis is the microbiology agent, which was discovered in 1906 of Bordet and Gengoux.Bordetella pertussis, genus Bordetella is a Gram negative immobilecoccobacteria growing and cultivating in special cultures: potato-glycerin agar, agar of Bordet-Gengoux, chocolate agar. The bacteria isslightly resistant to external conditions and disinfectants. Its pathogenic effect is related to the toxins it produces:

More important toxins and their effects are:

• Philamenthaemagglutinin - leads to adhesion to the respiratory epithelium

• Adenylatecyclase toxin – leads tocAMP induces cellular damage

• Bordet-Gengouxthermo labiledermonecrotic toxin - leads to ischemic necrosis of the vascular endothelium

•Pertussiotoxin (PT) - activates lymphocyte subpopulations, increases histamine sensitivity

• Tracheal cytotoxin - suppresses DNA synthesis in respiratory epithelial cells

Epidemiology:

The infection keepers are sick people and healthy contaminants, the most contagious in the first week. The diseases is transmitted by air-drops, the incubation period is 1-3 weeks. The contagious index is 80%. In the pre-immunization period the disease was epidemic with a cycles of 2-5 years, with summer seasonality and a peak in August. The Immunity is a post-infectious but not lifelong –it lasts 3-5 years up to 12 years. The disease frequency: pre-immunization use to be 150 per100 000 and after vaccination: 2.7 per 100 000 (1993).

Pathogenesis:

Entrance - the lining mucosa of trachea and bronchi. The bacteria causes acute catarrhal bronchitis, due to the impact of PT on lymphocytes , the proliferation of mature lymphocytes, an outbreak of congestive excitation in the CNS cough center – initially reflexive, and subsequently "provoked" by various stressful stimuli. Immunogenesis of the disease is humoral with synthesis of complement-binding, viral neutralizing and haemagglutination inhibiting antibodies.

Clinical symptoms::

The incubation period is from 1 to 3 weeks. The development of the disease is in 6 weeks, and three stages of the disease are marked:

1. **Catharal period** - gradually increasing dry, predominantly nocturnal cough, no fever or presence of low grade fever, rhinorrhea, sneezing, conjunctivitis (2-7 days). This is the most contagious period.

2. **Convulsive period (Paroxysmal period**) –increasing frequency and strength of the cough. This period lasts 10-30 days.

Different stimuli such as eating, drinking, talking,laughing etc. can provoke coughing paroxysms. Typical are the series of coughs in the phase of expiration, followed by a specific inspiratory stridor sound - a reprise. Paroxysms may be accompanied by cyanosis and vomiting. The sick child's head is tense, red (cyanotic), with visible cervical veins, conjunctivalhaemorrhages, petechiae, abundant salivation,tongueand eyeballs protrusion. Paroxysm ends withspitting up of a clear sputum or vomiting.

Physical examination of the lungs is usually negative, but the presence of temperature combined with a worsening of respiratory status usually indicates secondary bacterial pneumonia.

3. **Convalescent period** - slow attenuation of paroxysms, residual chronic cough, which may last for months with intermittent worseningduring respiratory infections.

The disease forms are: mild (in immunized), medium andsevere form - with fever, severe condition, apnea and asphyxia during the attack, bradycardia. Possible Laryngospasm and death, severe encephalopathy, seizures and cerebral edema can be observed.

Complications:

- Mechanical - from the high pressure during attacks: nasal bleeding, frenulum rupture, hernias, rectal prolapse, etc.
- Subcutaneous / mediastinal emphysema in severe forms
- Pulmonary atelectasis and subsequent bronchiectasis
- From the central nervous system - encephalitis, deafness, blindness, paralysis as a result of hypoxia
- Seizures - in 1%, lethal outcome in 0.3% of the cases

Poor prognostic factors are leukocytosis over 50,000 leukocytes 10^9, concomitant pneumonia, ageunder one year.

Pulmonary complications:

A) Early - bronchitis, laryngitis, middle ear inflammation, pulmonary atelectasis, activation of latent tuberculosis in pertussis, pneumothorax, pneumomediastinum. Pneumonia of secondary bacterial infection can be observed in 0.8-2% of all cases and 16-20% of the hospitalized patients, most often staphylococcal, streptococcal, haemophilus influenza.

B) Late onset complications: bronchial hyperreactivity, development of bronchiectasis.

There are no typical X-ray morphological changes for pertussis infection: paracardiacinfiltrates, hilaradenopathy, various areas of atelectasis can be identified, secondary focal pneumonia.

The clinical course of the disease does not correlate with the severity of X-ray changes.

Diagnostic methods:

• Clinical and epidemiological diagnosis: close contact with another family member with typical cough, coughing attacks with a typical clinical characteristic, non-immunized person.

• Complete blood count (hemogram) in the first period - lymphocytosis with leukocytosis of mature lymphocytes, which correlates with the severity of the disease, normal or accelerated ESR.

• Culturaleevaluation: Bordet-Gengoux culture of sputum or throat secretion (direct cough in petri dishes) - 7-12 days for a result.

• PCR test: rapid test, not affected by antimicrobial therapy, using secretions from upper respiratory airways with special Dacron swab.

• Serological diagnosis - establish at least 4-fold increase in the antibodies titer: ELISA (influenced by previous vaccination and previous infection).

• Direct Immunofluorescence microscopy - a difficult approach

Differential diagnosis:

1.In the catarrhal period: with acute respiratory diseases - influenza, Para influenza, rhinoviruses, adenoviruses, etc.

2. In the paroxysmal period: adenoviruses, cystic fibrosis, tuberculosis, foreign body, acute bronchiolitis, mycoplasma and chlamydial infections, RSV infections, etc.

Treatment:

• Etiological: macrolide antibiotics –they do not reduce the duration or severity of the disease, have only epidemiological significance:

- Erythromycin 40-50 mg / kg / d 14 days,
- Clarythromycin 15-20 mg / kg / d 10 days
- Azythromycin 10-12 mg / kg / day 7 days
- Biseptol 48mg / kg / day; Tetracyclines

Antibiotic therapy of concomitant bacterial pneumonia if needed.

• Pathogenetical:

Oxygen therapy, antihistamines, mucolytics and sedatives, steroids 1-2mg / kg.

Prevention:

In the Pre-War era, according to WHO data, 60 million children had been sick, of whom about one million died from complications. Historically, a killed pertussis vaccine - DTC (highly reactive) was first applied in the 50s, the efficacy of mass vaccination with whole cell vaccines was demonstrated in the following vaccination plan: 2-4-6m (or 2-3-4m), 15-18m, 4-6 years.

In the 1990s, a progressive increase in pertussis incidence with displacement of diagnosed cases in older age groups were reported in some of the developed countries with a long immunization period and high vaccine coverage (USA, Australia, Canada, the Netherlands)

Possible reasons include an improved laboratory diagnosis, limited duration of post-vaccination immunity, or both.

Two epidemiological phenomena have been observed: an increase in pertussis disease prevalence and "aging" of the infection, which necessitates a reassessment of the immunization schedules.

According to CDC - Atlanta, although primary immunization coverage in the US exceeds 90% since 1994, the number of reported cases is increasing from 1 010 for 1976 to 11 647 for 2003 with a peak in teenage age. The conducted studies show significantly increased morbidity at the age of 10-49 years.

Along with the significant control of the infection in the infant population which was immunized during the 1990s, there were epidemic outbreaks of the patients in higher age groups. As a result of the "aging" of the infection, young people are the main reservoir of the disease. Statistics in Bulgaria are no exception, is indicates a yearly trend Increase of cases. Patients over 8 years of age go up from 9.3% (1975-1981) to 15.8% (1990) and 20% (2003).

In response to the trend, immunization programs are being developed with the introduction of a universal and selective approach that aims to increase protection through re-immunization (booster doses of the pertussis vaccine) - Global Pertussis Initiative. Booster vaccines were introduced: Boostrix (GSK) and Adacel (Sanofi Pasteur)

In 2004, an analysis is being carried out in 30 European countries, 16 of which apply a second booster in pre-school or school age (France 11-13 years and Germany 9-17 years), Austria and Malta - third booster - 14-15 years. In most European countries "cell-free" vaccines are used for safety reasons and least side effects. Acellular vaccines that have conserved immunogenicity but have low reactogenicity and few observed side effects are: Infanrix (GSK) and Pentaxim (Aventis Pasteur).

Conclusion: Despite the modern approaches to vaccination and the profile of the pertussis, it remains a significant bacterial infection, particularly dangerous for breastfeeding children and early childhood, but with a main reservoir - adolescents and young people. Accumulating more knowledge of diagnostic methods and inventing new vaccines will enable the pertussis to be restricted and spread.

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