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COURSE OF NOCOSPITAL PNEUMONIA IN PATIENTS ON LONG-TERM ALV

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Annotation: *Hospital-acquired pneumonia (HP) accounts for 14 to 20% of the total structure of nosocomial infections, and ranks first in terms of mortality. The etiology of HP varies depending on the profile of the department and the microecological features of a particular hospital. The causative agents of HP are: P. aeruginosa, Enterobacter, E. coli, K. pneumoniae, Proteus spp., Serratia marcescens, H. influenzae, S. aureus, S. pneumoniae. In severe cases, highly resistant Gr (-) microorganisms (P. aeruginosa, Acinetobacter), methicillin-resistant S. aureus (MRSA), Candida spp., Aspergillus fumigatus, Pneumocysta carinii can be isolated. Legionella is responsible for less than 8% of HP cases. There are two variants of lung infection: the first is the endogenous route of infection (autoinfection); the second is an exogenous route of infection (from other patients or medical personnel), contaminated air, food, water, objects that come into contact with the patient's respiratory tract (bronchoscopes, catheters for sanitation of the tracheobronchial tree, etc.).*

Keywords: *Pneumonia, lung ventilation, patient, hospital, aspiration, bacterium.*

Community-acquired pneumonia (CAP) is an acute disease that occurs in a community-acquired setting, that is, outside a hospital, or is diagnosed within the first 48 hours of hospitalization, or develops in a patient who has not been in a nursing home/long-term care unit for ≥14 days, accompanied by symptoms of lower respiratory tract infection (fever, cough, sputum (possibly purulent), chest pain, shortness of breath) and radiological signs of "fresh" focal infiltrative changes in the lungs in the absence of an obvious diagnostic alternative.

Purpose of the study: to establish the etiological structure of ventilator-associated pneumonia (VAP) in the studied patients, to determine the pattern of antibacterial sensitivity of the main pathogens.

Nosocomial pneumonia - pneumonia that develops 48-72 hours after the patient's admission to the hospital and which did not exist and was not in the phase of the incubation period until the moment of admission.

Ventilator-associated nosocomial pneumonia - pneumonia that developed no earlier than 48 hours after intubation and the start of mechanical ventilation, in the absence of signs of a pulmonary infection at the time of intubation. However, in many cases, in

surgical patients, the manifestation of nosocomial pneumonia is possible even at an earlier date.

The frequency of hospital-acquired pneumonia reaches 20% of all hospital-acquired infections and is observed more often in patients after operations on the chest or abdominal cavity, in patients who are on mechanical ventilation and in patients with immunodeficiency.

The most common cause is microaspiration of bacteria that colonize the oropharynx and upper respiratory tract in critically ill patients. Lung contamination due to bacteremia or inhalation of infectious aerosols (i.e., airborne particles containing Legionella, Aspergillus, or influenza viruses) are less common causes. Ventilated endotracheal intubation poses the greatest risk; Mechanical ventilation accounts for > 85% of all cases, and pneumonia occurs in 9–27% of patients on mechanical ventilation. The highest risk of ventilator-associated pneumonia occurs during the first 10 days of intubation. Endotracheal intubation compromises airway protection, cough, and mucociliary clearance and facilitates microaspiration of bacterially contaminated secretions that accumulate above the inflated cuff of the endotracheal tube. In addition, bacteria form a biofilm on and in the endotracheal tube, which protects them from antibiotics and host immunity. In non-intubated patients, risk factors include prior antibiotic therapy, high gastric pH (due to stress ulcer prophylaxis or treatment with H2 blockers or proton pump inhibitors), concomitant cardiac, respiratory, hepatic, or renal failure. The main risk factors for postoperative pneumonia are age >70 years, abdominal or thoracic surgery, and functional exhaustion.

MATERIALS AND RESEARCH METHODS. The paper analyzes the treatment of 48 patients who were on mechanical ventilation in the intensive care units of the Regional Children's Hospital for the period 2018-2020. Depending on the presence of VAP, patients were divided into 2 groups. Group I or the main group consisted of 27 patients who developed VAP. Group II or control group consisted of 21 patients without the above complication. The criterion for inclusion of patients in the study was the duration of mechanical ventilation for at least 48 hours. Significantly greater sensitivity in determining the microorganisms responsible for the development of VAP was shown by cultures of tracheal aspirate from the lower respiratory tract. Sputum collection was performed using fibrobronchoscopy. Tracheal aspiration and bronchoalveolar lavage were also used. Culture of aspirate from the lower respiratory tract was more often positive in patients of the main group than in the control group: 74% (20) and 37% (10), respectively. In 59.2% (16) of cases in the main and 22.2% (6) in the control group, the flora was isolated in various associations. For the development of pneumonia in the main group, it significantly exceeded when seeding gram-negative bacteria: Pseudomonas spp. (6 cases), Acinetobacter spp. (3 cases), K1ebsiella pneum (2). The highest sensitivity in Pseudomonas spp was determined to ciprofloxacin (91.1% of isolates). Approximately equal activity against Pseudomonas spp was shown by sisomycin (77.8%), amikacin (80.0%) and

netilmicin (74.1%). The number of strains of Pseudomonas aeruginosa resistant to gentamicin reached 21.6%, and another 8.1% had moderate sensitivity. Only half of the Pseudomonas strains (54.5%) were susceptible to piperacillin. Of the III generation cephalosporins, sufficient activity was determined in ceftazidime: 71.4% of Pseudomonas isolates were sensitive to this ABP, while only 33.3% of strains were sensitive to cefotaxime (of which 25.0% had moderate sensitivity). Other gram-negative bacteria were sensitive to ciprofloxacin in 100.0% of cases: Klebsiella pneumoniae, E. coli, non-fermenting flora. However, 1/3 of Klebsiella strains (33.3%) had moderate sensitivity to ciprofloxacin. With respect to Klebsiella pneumoniae, amikacin was a rather active drug (42.8% - high and 28.6% - moderate sensitivity). The non-fermenting gram-negative flora, Klebsiella pneumoniae and E. coli were resistant to the first two generations of cephalosporins. 57.1% of isolated cultures of Klebsiella were sensitive to cefotaxime (of which 14.2% moderately).

Conclusions: Thus, the etiological structure of VAP was dominated by gram-negative. Pseudomonas spp., Acinetobacter spp., non-fermenting Gram-negative flora and Klebsiella pneumoniae were isolated most frequently. These microorganisms showed significant resistance to the main antibacterial drugs. The most active against gram-negative flora was ciprofloxacin. Sensitivity to other ABPs in different bacteria varied widely. Blood cultures were of limited value in determining the causative agent of VAP.

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