group of ranolazine (38%, p<0.001), the dynamics was maintained on the 10th day (5,4%, p>0.5). Ranolazine (STEMI diagnosed patients) had a positive impact on the markers of electrical myocardial instability and its ischemia - as evidenced: decreasing of the heart rate, probability of cases of SDNN (standard deviation of NN intervals) decrease (decreasing of SDNN is a strong, independent and consistent risk factor for overall and cardiac mortality), QT dispersion (increased QT variability is a risk factor for sudden cardiac death) and maybe increase of the ratio of maximum velocity for differentiated deflection T.

This paper presents the possibility and expediency of using information systems in diagnostics – ECG digitalization and "Smart-ECG" which showed ranolazine efficiency for optimization of treatment of ST segment elevation syndrome, therefore, this study is relevant and needs further implementation.

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## PREVENTION OF POSTPNEUMONIC PULMONARY FIBROSIS IN PATIENTS WITH BILATERAL VIRAL-BACTERIAL PNEUMONIA

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Improving the effectiveness of viral-bacterial pneumonia treatment related to the COVID-19 pandemic has become particularly important, as it is this complication of viral infection that is associated with mortality or reducing quality of life. A common pathomorphological feature of viral pneumonia is the tendency to form thrombotic masses in the capillaries of the pulmonary circulation, in contrast to bacterial pneumonia, when pathological process dominates by intra-alveolar fibrinous exudation.

In both cases, with fibrinolysis insufficiency and low resorptive potential, there is a risk of developing postpneumonic pulmonary fibrosis (PPF) or in the form of lung carnification (intraalveolar fibrosis) or a process similar to fibrosing alveolitis. The development of postpneumonic pulmonary fibrosis is associated with the development of a restrictive type of chronic pulmonary insufficiency of varying severity depending on the area of the affected lung tissue.

In the infectious department of MNE "City Clinical Hospital № 3" we tested the method of a combined use of two drugs to prevent the development of PPF with different mechanisms of antifibrotic activity: peroral administration of seratthiopeptidase tablets of 20 mg twice a day and inhalation of budesonide 500 mcg twice a day.

Serratiopeptidase was chosen by us due to its inherent fibrinolytic, anti-inflammatory and anti-edematous activity. Serratiopeptidase, a serine protease with a molecular weight 60 kDa, has been significantly reported for its potent anti-inflammatory activity. The clinical use of enzyme was reported for many diseases like arthritis, sinusitis, inflammatory bowel disease and bronchitis etc. The current challenge toward developing serratiopeptidase into an effective broadspectrum anti-inflammatory drug is due to lack of precise molecular mechanism. This proteolytic enzyme was reported effective in many diseases precisely during surgical events for a long time, but there is a lack of research evidence and available literature (Tiwari M., 2017).

The drug also attracted our attention due to its hydrolytic inactivation of bradykinin, histamine and serotonin, it is able to directly reduce the dilation of capillaries and control their permeability. Serrathiopeptidase blocks plasmin inhibitors by stimulating its fibrinolytic activity. Due to a possible potentiation of anticoagulants, which are widely used in pneumonia caused by coronavirus, the use of the drug is not recommended in the presence of hemoptysis.

The local activity of the budesonide molecule is known to interact with specific receptors in the cytoplasm of the cell and form a complex that binds to DNA and stimulates the formation of mRNA after penetrating the cell. It leads to changes in the ribosomes of proteins corresponding to the properties of cells. The drug stimulates the synthesis of lipocortin, which inhibits the enzyme phospholipase A2, resulting in inhibition of the synthesis of prostaglandins and leukotrienes involved in the development of inflammatory reactions.

A combined activity of both drugs prevents the development of PPF in patients with a small area of the lung tissue (up to 10-15%) and significantly reduces the intensity of radiological manifestations of PPF in patients with moderate pneumonia (20-40%).It should be noted that the early indication of a combination of serratiopeptidase-budesonide can also significantly stimulate the effectiveness of antibacterial therapy in patients with viral-bacterial pneumonia, increasing the concentration of antibiotic in the lung tissue, promoting liquefaction of bronchial secretions, stimulating mucociliary clearance and reducing inflammatory swelling of the damaged tissues.

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## RECENT ACHIEVEMENTS AND TREATMENT STRATEGY IN OSTEOARTHRITIS (LITERATURE REVIEW)

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Osteoarthritis (OA) is the most common form of arthritis that widely afflicts population of elder age and causes disability frequently. Moreover, other less spread muscular-skeletal disorders, for instance, rheumatoid arthritis are far ahead in terms of drug development.

Main OA treatment strategies include pain relief and chondroprotection. As well as ten years ago, drugs for OA are divided into those improving pain and those which improve cartilage structure. Existing guidelines for the management of OA include the abovementioned pharmacological therapies in addition to education, weight loss, and exercising. Single new anaestetics appeared on the market for last decade, and oral and topical non-steroidal anti-inflammatory drugs and cyclooxygenase-II inhibitors, acetaminophen (paracetamol) are still prioritized. Opiates, intraarticular steroids are indicated when first-line agents fail. However, half of patients taking the drug report no relief even in case of administration of the best of these agents while the side effects and potential toxicities limit their use in a population who often has associated comorbidities.

Aim: toanalyze recent literature and distinguish progress in understanding nature and perspectives of OA treatment. The fact that OA is pleiotropic polyvariant disease with different molecular profiles and phenotype is one of recent significant discoveries. The following primary variants of OA are suggested to distinguish: chronic pain; inflammatory phenotype; metabolic syndrome phenotype; altered bone and cartilage metabolism phenotype; mechanical overloading phenotype and minimal joint disease phenotype. On the basis of etiology and provoking factor, OA may be different in secondary variants (crystal-driven, post-traumatic, autoimmune, occupation-associated), intra-and extraarticular types and one due to age-related association. These studies make us to conclude that classical inflammation does not uniquely drive OA. Hence, multiple molecular cascades may contain a key for successful treatment strategy.

Although osteoarthritis phenotype has been inconsistently reported in IL-6 knockout mice, therapeutic studies suggest that neutralization of IL-6 modifies OA in mice. A clinical trial using tocilizumab in hand osteoarthritis was completed in 2019 but has not yet been reported.

Targeting the proteases (disintegrin and metalloproteinase with thrombospondin motif (Adamts)-5) that degrade the cartilage extracellular matrix has long been regarded as an attractive approach to disease modification in osteoarthritis. A good safety profile and initial efficacy were demonstrated for a their small molecule inhibitor in phase 2 studies in knee OA. Other early phase trials investigate potential efficacy of Transforming growth factor α and epiregulin; Wnt pathway; CNS reuptake inhibitor; folate antagonist etc. Preclinical studies have shown that pharmacological inhibition of iNOS reduces OA progression and pain in a rodent model of OA as well. Bone Modulators – bisphosphonates seem to be perspective in number of OA cases. Their administration has been suggested to slow the bone remodeling process and could lead to chondroprotection. Strontium Ranelate is another antiosteoporotic drug capable of changing the balance between bone resorption and bone formation. It significantly attenuated cartilage matrix and chondrocyte loss, and decreased chondrocyte apoptosis in rats.