HIDDEN PULMONARY RISK OF LONG-TERM AMIODARONE THERAPY: A CASE PRESENTATION

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Relevance. Amiodarone is an effective class III antiarrhythmic widely used for atrial fibrillation and ventricular arrhythmias. Due to its lipophilic nature and prolonged half-life, it accumulates in tissues and may cause systemic toxicity. Pulmonary toxicity is one of its most severe adverse effects, occurring in 4–17% of patients. Clinical manifestations are often nonspecific, including dyspnea and cough, frequently mimicking infectious pneumonia. Delayed recognition may lead to progressive interstitial lung disease and fibrosis [1]. Early diagnosis and prompt drug withdrawal are essential to prevent irreversible pulmonary damage.

Target. To present a case of amiodarone-associated pulmonary toxicity, emphasizing diagnostic challenges, radiological progression, therapeutic management, and the importance of multidisciplinary cardiopulmonary care.

Research methods. Amiodarone presented with exertional dyspnea, productive cough, weakness, and blood pressure variability. Initial chest radiography in October 2023 revealed bilateral irregular alveolar infiltrates on a background of emphysematous and fibrotic changes. The findings were interpreted as polysegmental bacterial pneumonia, and ceftriaxone therapy was initiated.

Follow-up radiographs demonstrated worsening infiltrative shadows and distortion of the pulmonary pattern. High-resolution CT revealed diffuse ground-glass opacities, reticular abnormalities, fibrotic changes, and a 44×48 mm consolidation in the left lower lobe with preserved bronchial lumens. Laboratory investigations, including inflammatory markers, autoimmune screening, and biochemical parameters, were within normal limits. Despite antibiotic therapy, subsequent CT after additional amiodarone exposure showed further progression of interstitial abnormalities.

Results and their discussion. Lack of response to antibiotics, radiological deterioration, and a clear temporal relationship with amiodarone administration suggested drug-induced interstitial lung disease. Amiodarone was discontinued and replaced with propranorm. Systemic corticosteroid therapy with methylprednisolone (12 mg daily) was initiated.

Follow-up CT in May 2024 demonstrated marked improvement, including resolution of consolidation and reduction of ground-glass opacities, leaving residual parenchymal compaction. Clinical symptoms significantly improved. The case highlights the difficulty in differentiating amiodarone toxicity from infectious pneumonia and underscores the importance of detailed medication history and high-resolution imaging. Early recognition and drug withdrawal were pivotal in preventing irreversible fibrosis [2].

Conclusions. Amiodarone-induced pulmonary toxicity should be considered in patients with persistent respiratory symptoms and progressive radiographic changes during antiarrhythmic therapy. Prompt discontinuation and corticosteroid treatment significantly improve outcomes. Close collaboration between cardiologists and pulmonologists is crucial for early diagnosis, individualized management, and prevention of long-term pulmonary sequelae.

LITERATURE

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2. Coward, W. R. The pathogenesis of idiopathic pulmonary fibrosis / W. R. Coward, G. Saini, G. Jenkins // Therapeutic Advances in Respiratory Disease. – 2010. – Vol. 4, № 6. – P. 367–388.

CRISPR: A MODERN TOOL FOR TREATING CANCER

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Relevance. Cancer is fundamentally a genetic disease characterized by the accumulation of somatic mutations, oncogenes activation, and tumour suppressor genes inactivation. Therefore, CRISPR technology is ideally suited for studying and treating cancer.

Target. To evaluate experimental strategies, therapeutic outcomes, safety considerations in preclinical and clinical studies with usage of CRISPR techniques.

Research methods. The study involved a comprehensive review and analysis of recent scientific articles focusing on CRISPR-based applications in cancer therapy.

Results and their discussion. Researchers use CRISPR to identify genes that promote tumor growth and to understand how cancer cells resist drugs, which has led to the discovery of many new potential drug targets [1].

CRISPR is being applied to modify immune cells for cancer immunotherapy. By editing T cells or natural killer cells, scientists can enhance their ability to recognize and destroy tumours. One of the most exciting applications of CRISPR is improving CART cell therapy. In which a patient's T cells are engineered to attack cancer cells. CRISPR can be used to remove inhibitory receptors such as PD1 or to insert new receptors that improve tumour targeting [2]. Researchers are testing CRISPR-modified CART cells in clinical trials for leukaemia, lymphoma, and solid tumours. These advances demonstrate how CRISPR can enhance existing therapies and create entirely new treatment strategies for cancer patients.

The primary therapeutic strategies is direct inactivation of oncogenes that drive tumour growth. CRISPR-based gene editing enables targeted disruption of mutated or amplified oncogenes responsible for uncontrolled proliferation [3]. Studies