demonstrated that inactivation of MYC oncogene reduced tumour growth in animal lymphoma models. These findings provide experimental evidence that CRISPR-mediated oncogene disruption can suppress tumour progression *in vivo* [3]. CRISPR technology has also been applied to correct inherited mutations linked to cancer. Studies reported the correction of BRCA1 mutations in human cells, demonstrating feasibility for hereditary breast and ovarian cancer therapy.

CRISPR based gene editing has thepotential to cause mutations in the genome by targeting unintended genes, this is particularly concerning in cancer therapy, where the goal is to target specific genetic mutations that drive tumor growth [3].

Conclusions. The prospective future of CRISPR in cancer treatment is exceptionally promising. With ongoing advancements in research surmounting existing barriers, there is tangible possibility for the creation of effective, individualized and minimally invasive treatments for cancer.

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A CLINICAL CASE OF SYSTEMIC SCLEROSIS ASSOCIATED WITH PRIMARY BILIARY CIRRHOSIS (REYNOLDS SYNDROME)

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Relevance. Reynolds syndrome is a rare overlap syndrome combining systemic scleroderma (SSc) and primary biliary cirrhosis (PBC). The polymorphism of the clinical presentation complicates diagnosis and therapeutic decision-making. This condition has been reported in only a limited number of cases in literature [1]. PBC within the syndrome can be asymptomatic or manifest as cholestasis, pruritus, jaundice, xanthomas [2].

Target. To describe the characteristics of a rare autoimmune disease – Reynolds syndrome.

Research methods. Literature review; clinical, laboratory and instrumental examinations (barium esophagram, CT, ultrasound, ECG, echocardiography, EGD).

Results and their discussion. A 74yearold female patient. Since 2003: Raynaud's syndrome, skin thickening of the hands, arthralgia, dysphagia, «pursestring

mouth». In 2006, SSc was diagnosed, methotrexate and cuprenil were prescribed (discontinued due to intolerance). Afterwards, the disease progressed: fingertip ulcers appeared and dysphagia worsened. Laboratory findings: positive ANA, anti-Scl-70, cryoglobulinemia. Radiographic findings: erosive arthritis. Glucocorticoid therapy has been initiated with a positive effect. In 2016, pruritus and abdominal syndrome joined. Class A PBC was diagnosed (AMA 1:5120), and the diagnosis was verified in favor of Reynolds syndrome. Treatment with ursodeoxycholic acid and budenofalk was administered. Complications: aseptic necrosis of the right femoral head, atherosclerosis of the vessels of the lower limbs with critical ischemia (amputation was avoided thanks to conservative treatment), compression fractures of the vertebrae (12 cm height loss). In February 2026, the patient was re-hospitalized. Complaints: polyarthralgia, firm swelling of the hands, severe dysphagia, pain in the hip joint and spine, paresthesia. Receives amlodipine, aspirin, ursodeoxycholic acid, atorvastatin, prednisolonum, and pentoxifylline. Denosumab, Ca^{2+} and vit. D. medications are planned. Additional examination: no evidence of pulmonary hypertension, 2x3 cm lesion in the lungs (stable compared to the previous CT), sclerodermic esophagitis. The patient's condition is satisfactory on current therapy.

Conclusions. This case illustrates the difficulties of diagnosing Reynolds syndrome. Knowledge of the syndrome is essential for the timely interpretation of the clinical presentation and the selection of the correct therapeutic tactics. The long-term evolution of symptoms and the late verification of PBC emphasize the need for screening of antimitochondrial antibodies in all patients with SSc. Confirmation of the syndrome determines the need for a multidisciplinary approach.

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