Primary healthcare clinicians and Dermatologists and should maintain vigilance for PCT in younger patients with compatible cutaneous phenotypes, pursue a comprehensive diagnostic workup.

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NEUROBLASTOMA GENETIC AND MOLECULAR ASPECTS

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Relevance. Neural crest cells are found in areas like the adrenal glands, chest, and abdomen, which are common sites for neuroblastoma. The maturation, movement, and development of these cells are controlled by various genes, with MYCN playing a major role. Problems with these genes, like overexpression, can lead to tumors. From a genetic perspective, neuroblastoma is linked to the overexpression of the MYCN gene, mutations in genes like ALK and PHOX2B, and changes in the ATRX gene and TERT gene rearrangements, which are more common in high-risk cases [1]. In 85% of cases, mutations in the ALK gene are found at positions F1174, F1245, and R1275. These mutations occur in both types of neuroblastoma – those that happen by chance and those that are inherited. Studies suggest that sometimes these ALK mutations are connected with changes in the MYCN gene [2].

Molecular mechanisms

The MYCN gene helps neuroblastoma cells get energy through a process called aerobic glycolysis. This gene also controls many processes that are important for normal cell development and the formation of cancer. MYCN increases the use of glucose and glutamine, which helps the tumor grow in cancer like neuroblastoma. The RAS-MAPK pathway is active in the primary form of neuroblastoma. This pathway is linked to a worse outlook and poorer results. Mutations in genes such as ALK are connected with the activation of this pathway [2].

Target. To understand the genetic and molecular changes that cause neuroblastoma. This includes looking at variations in the MYCN gene, its overexpression, and changes in other genes like ALK (anaplastic lymphoma kinase), PHOX2b, and others.

Research methods. The information was gathered from existing research and studies.

Results and their discussion. Small molecules that block the MYCN gene, like inhibitors of PI3K and GSK3, have been found to reduce the survival of cells that have amplified MYCN genes. These GSK3 inhibitors affect many neuroblastoma cells and can lead to their death. Treatment for low-risk neuroblastoma usually involves surgery to remove the tumor and standard chemotherapy. For high-risk neuroblastoma, treatment is divided into three phases: induction, consolidation, and maintenance. During the induction phase, chemotherapy drugs like etoposide, vincristin, doxorubicin, and cyclophosphamide are used.

Conclusions. In summary, there is a lot of variation in the MYCN gene, and this variation often leads to high-risk neuroblastoma. The MYCN gene is also linked to various other genes and molecular pathways.

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CHRONIC MESENTERIC ISCHEMIA

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Relevance. Chronic mesenteric ischemia (CMI) is a condition characterized by inadequate blood flow to the small intestine, primarily caused by stenosis or occlusion of the mesenteric arteries [1-2]. This condition often presents with postprandial abdominal pain, weight loss, and risk of progression to bowel necrosis. The diagnosis and management of CMI are complex, involving various imaging and surgical techniques. Understanding the clinical and anatomical features of CMI is essential for timely intervention and improved patient outcomes [3].

Target. To analyze the clinical features, diagnostic approaches, and surgical outcomes of 16 patients diagnosed with CMI at the Grodno University Clinic.

Research methods. Patients underwent physical examinations, duplex ultrasound, multidetector computed tomography (CT), magnetic resonance imaging (MRI), and angiography. The severity of the disease was classified based on clinical symptoms and anatomical findings. Treatment strategies included conservative management, endovascular revascularization, and open surgical procedures, with follow-up assessments to evaluate efficacy.

Results and their discussion. Patients ranged from 45 to 60 years of age, with a predominance of males (11 out of 16). The majority of patients exhibited postprandial