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BIOCHEMICAL BASIS OF THE DEVELOPMENT OF GOUT

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Abstract

This article discusses the development of gout and the processes that occur at the cellular level. It provides information about the etiology of the disease, its causes, biochemical changes and symptoms, as well as measures to combat such symptoms. The article also presents data on which substance metabolisms are disrupted in the development of this disease.

Keywords

Uric acid, nucleotides, proteins, cofactor, treatment, disease of the rich.

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Annotatsiya

Ushbu maqolada podagra kasalligining kelib chiqishi, hujayra darajasida ro`y beradigan jarayonlar haqida so`z boradi. Bu kasallik etiologiyasi, kelib chiqish sabablari, biokimyoviy o`zgarishlar va belgilari, shu turdagi belgilalariga qarshi kurash choralari haqida ma`lumotlar beriladi. Bu kasallik kelib chiqishida qaysi moddalar metabolizmi buzilishi haqida ham ma`lumotlar beriladi.

Kalit so`zlar

Siydik kislota, nukleotidlar, oqsillar, kofaktor, davolash, boylar kasalligi.

БИОХИМИЧЕСКИЕ ОСНОВЫ ВОЗНИКНОВЕНИЯ ПОДАГРЫ

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Аннотация

В данной статье рассматриваются причины возникновения подагры и процессы, происходящие на клеточном уровне. Представлена информация об этиологии заболевания, причинах его возникновения, биохимических изменениях и симптомах, а также о мерах борьбы с такими проявлениями. Также приводятся данные о нарушениях обмена веществ, связанных с развитием данного заболевания.

Ключевые слова

Мочевая кислота, нуклеотиды, белки, кофактор, лечение, болезнь богатых.

Gout is a disease characterized by an increased level of uric acid in the blood serum. This disease was first identified in the 18th century. Later, in the 19th century, the causes of the disease began to be studied. According to the studies conducted during that time, it was mainly wealthy individuals who suffered from this condition, which is why it became known as the “disease of the rich.” The main reason for this naming was believed to be the high consumption of wine and red meat during that period. Subsequent studies on cell structure and its chemical composition revealed that there are multiple factors contributing to the development of this disease. The main cause of gout is either increased synthesis of uric acid or difficulty in its excretion from the body. Now, let us take a closer look at this issue.

Uric acid is considered the final product of nucleotide degradation. In addition, increased denaturation of amino acids can also contribute to its accumulation, although this factor is rarely observed. The disruption of nucleic acid degradation occurs in two main forms:

1. Hereditary (genetic) factors:

This type is caused by mutations in the genes responsible for the activity of enzymes such as PRPP, ADA, and HGPRT, which are essential for the breakdown of nucleic acids. It can also result from a deficiency of amino acids involved in their

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synthesis. PRPP (phosphoribosyl pyrophosphate) is an enzyme that synthesizes purine bases from ribose-5-phosphate. A deficiency of PRPP leads to insufficient purine synthesis. As a result, the body compensates by intensifying the breakdown of cellular nucleic acids or nitrogen-containing compounds, which increases uric acid levels. HGPRT (hypoxanthine-guanine phosphoribosyltransferase) is an enzyme that converts the intermediate product xanthine into uric acid during nucleic acid degradation. A deficiency of HGPRT causes the accumulation of xanthine, which lowers blood pH levels. This disrupts the body's acid-base balance and reduces uric acid transport, potentially leading to various pathologies. ADA (adenosine deaminase) does not convert adenine directly into xanthine. Instead, it first converts adenine into inosine, which is then transformed into xanthine. A deficiency in ADA impairs the breakdown of adenine nucleotides. When the cell requires adenine, other nucleotide metabolic pathways are upregulated to compensate, which in turn increases uric acid production. All of the above-mentioned factors are classified as hereditary causes of gout.

2. Acquired causes:

In gout caused by acquired factors, the level of uric acid in the blood may be within the normal range, nucleic acid metabolism functions properly, and enzyme activity is not impaired. However, the excretion of uric acid through the kidneys is disrupted. Normally, uric acid is filtered through the renal glomeruli and enters the primary urine. In certain conditions, problems with the reabsorption of primary urine can occur, causing uric acid to be reabsorbed back into the bloodstream instead of being excreted. This situation is commonly seen in conditions such as pyelonephritis, interstitial nephritis, and renal failure.

This type of disruption is acquired during the course of a person's life and is not inherited. The main symptoms of gout include the accumulation of uric acid in the joints, especially in the knee and heel areas. This leads to joint inflammation, joint degradation, and joint pain. One of the most characteristic symptoms of this disease is pain in the big toe, often accompanied by a burning sensation in that area. Additionally, uric acid can accumulate in the cartilage behind the ears and in the finger joints, leading to the formation of tophi. Due to impaired kidney filtration, uric acid may combine with metals like calcium and potassium, potentially leading to the formation of kidney stones.

The treatment of the disease should be carried out in several stages and continuously. The main goal is to activate the excretion of uric acid through the kidneys. Medications that accelerate this process are prescribed. Additionally, the intake of products such as raw meat and red wine should be limited.

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