THE USE OF INTERLEUKIN INHIBITORS IN CHRONIC HEADACHES

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Abstract
Migraine is a chronic disease that accounts for 15% worldwide. It is one of the most important causes of disability among women under 50 years of age. Migraines cause difficulties in socio-economic and everyday life. Chronic migraine causes a sharp decrease in the quality of life of the patient, leading to an increase in the cost of the disease, both directly and indirectly. In order to prevent migraine-related disability, today there are ways to prevent disease attacks and prevent seizures. Chronic migraines usually occur after episodic migraines. This has been confirmed in a large number of studies. Immunological mechanisms in migraines are studied as far as hanuz

Keywords: migraine, tension headache, chronic headache, comorbid disorder.

Introduction

As a risk factor, chronic migraine can be attributed to socio-economic disorder, female gender, obesity, various life problems, asthma, secondary headaches, head and neck injuries, snoring and nocturnal insomnia, incorrectly selected treatment measures. Various metataxllyls have shown that depression and over-the-Counter Drug Administration have also been implicated in chronic migraines. One of the risk factors is hypertension, and there is an inextricable connection between migraines and vascular diseases. Chronic migraine is a complex disease that occurs in 1-2% of the world's population, usually starting in the form of a progressive headache and has a tendency to develop from low-frequency attacks to high-frequency attacks. While chronic migraine pathogenesis has not been fully studied, pathways down the ogriq modulation, especially-PAG, three-horned nerve hypersecurity, lead to central sensitization, leading to a decrease in nociceptive nausea, increased cerebral cortex excitability, hematoencephalic obstruction is impaired, and chronic neurogenic inflammation occurs. Interleukin - 6 is a pleiotropic cytokin and is involved in immune management, inflammation, blood dressing, and oncogenesis. This tsitokin was researched by Japanese researchers in 1986. To date, significant progress has been made in understanding the mechanism of this tsitokin. Il-6 exerts its effects by two components according to its receptor: ligand-binding chain with a weight of 80 kda and non-binding ligand glycoprotein 130 (gp130), in a number of cases they are treated as a single receptor (il-6R). It is with this in mind that IL-6 receptor inhibitors have been developed (tosilizumab, sarilumab, levilimab).

Il-6R stimulation induces phosphorylation of tyrosine to activate the JAK-STAT-signaling pathway, initiating transcription of specific genes. Thus, il-6 operates on the" il-6 → il-6R → JAK-STAT " signaling pathway. Tumor necrosis factor, on the other hand, works on" FNO-α → NF-kB". For this reason, il-6 blockade is effective when fno-α inhibitors are not working. Interleukin - 6 affects the immune system to V - I T-lymphocytes, hepatocytes, blood-forming cells, vascular endothelial cells, etc. It is involved in the implementation of an immune reaction,

blood dressing and inflammation. It affects cell proliferation, differentiation, cell survival and apaptosis. Il - 6 activates the release of proteins, including S-reactive protein and serum amyloid A, in hepatocytes during the inflammatory process. Production of il-6 increases in Ra, with serum levels correlating with disease activity indicators. Increased Il-6 activity leads to systemic inflammatory reactions as well as osteoclast activation and bone tissue destruction . In RA pathogenesis, the assessment of cytokinin makes it possible to find a therapeutic target. In addition to Il-6 and il-6R inhibitors, fno-α inhibitors are also widely used. The first use of Il-6R inhibitors was tosilizumab, sarilumab. Ra da il-6R inhibitors can be used in monotherapy and are also listed in international medical recommendations. Reception of il-6R as a target is explained by the fact that the receptor concentration is relatively low compared to the il-6 concentration, therefore, it is easy to choose a dose and draw up a treatment scheme. Il-6R, as well as the complexity of Gp130 receptor structure, cause signals from il– 6 to pass through the tuli pathways-classical and trans-signaling pathways. Glycoprotein 130 responds to the passage of the signal not only from il-6, but also through other interleukins, such as il-27. Therefore, there is no way to predict the outcome of the il-6R blockade in advance. IL-6R antogonists (tosilizumab, sarilumab) prevent the Binding of il-6 to the receptor via classical and Transsignal pathways. Il-6 antibodies bind to olokizumab, IIIA il-6 and completely block the activity of the receptor complex. Olokizumab (okz) is a monoclonal antitanine directed to g4k immunoglobulin. It has a positive effect on il-6. Okz induces conformational changes to il-6. The" Il-6R – il-6 – gp130 " complex is exposed to okz. The amino acid GLU42-asn47 binds to the Gp130 receptor, indicating high thinning activity. Today, only as an inhibitor of il-6, ra da olokizumab has confirmed its effectiveness, and the rest of its preparations are being proven effective. The comparative analysis of Okz and tosilizumab shows that there are no differences between their safety and effectiveness. Olokizumab has been proven effective in large clinical studies. A study in volunteers confirmed that the drug in question had no serious side effects. Studies have shown that ra is safe when administered in multiple doses. For this reason, it also allows you to choose the optimal amount of medication. CREDO studies have shown a 20% positive result on 5 of 7 criteria compared to the drug placebo. Comparative control between Adalimumab and okz also showed no statistical differences.

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