

Analysis of the role of serum IL-2R, IL-6, IL-8, IL-10, and TNF- α in the treatment of immune escape mechanisms in patients with minimal ma tumors

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Abstract: Tumor immune escape refers to the phenomenon that tumor cells evade the recognition and attack of the body's immune system through a series of mechanisms, thus surviving and multiplying in the body. These mechanisms include, but are not limited to, low immunity, recognition as autologous antigen, antigenic modulation, tumor-induced immunosuppressive effects, and areas of tumor-induced generation immunity. Tumor cells often have low immunogenicity, i. e. they lack sufficient epitopes to stimulate the body's immune system. In addition, tumor cells can also produce immune suppressive molecules or cells by interacting with host cells, thus inhibiting the body's immune response. In addition, tumor cells can also produce immune suppressive molecules or cells by interacting with host cells, thus inhibiting the body's immune response. In this paper, the role of serum IL-2R, IL-6, IL-8, IL-10 and 1 TNF- α in the treatment of immune escape mechanism in patients was analyzed for reference.

Keywords: serum IL-2R; IL-6; IL-8; IL-10; TNF- α ; patients with minimal ma tumors; immune escape mechanism

1. Role of serum IL-2R-based mechanisms in treating immune escape in patients with minimal ma tumors

Immune escape is a key process for tumor cells to successfully evade the immune system attack, in which the expression level of IL-2R is crucial for the activation and function of T cells. Further exploring the specific role of serum IL-2R in patients with minimal ma tumors will not only help to reveal how tumor cells evade the body's defense mechanism, but also can provide a theoretical basis for novel therapeutic strategies.

Interleukin-2 is a cytokine essential for T cell growth and activation. Normally, the high expression of IL-2R marks the active state of T cells, which helps the body to fight the invasion of tumor cells. However, the tumor cells have gradually learned to utilize various mechanisms to reduce the expression of IL-2R, thereby reducing the risk of being recognized by the immune system.

First, tumor cells are able to significantly inhibit the proliferation and activity of T cells by releasing specific immunosuppressive molecules, such as TGF- β or IL-10. These molecules act on T cells, resulting in a significant decrease in the expression of IL-2R, which in turn affects the normal recognition and attack of T cells on tumor cells. Secondly, tumor cells can also induce T cell apoptosis by expressing certain apoptosis-related molecules such as FasL. Apoptotic T cells no longer express IL-2R, implying that they have lost their ability to respond to IL-2. This process not only weakens the antitumor function of the immune system, but also creates favorable conditions for the further spread of tumor cells.

Moreover, tumor cells often upregulate certain immune checkpoint molecules, such as PD-L1, to inhibit T cell activity. After binding of these immune checkpoint molecules with the PD-1 receptor on T cells, they cause T cells to enter an incompetent state and no longer attack tumor cells. To break down this immune escape, researchers are working hard to develop new therapeutic strategies. Among them, the immune checkpoint inhibitors have become the research hotspots. By blocking key pathways such as PD-1 / PD-L1, these inhibitors are expected to restore the activity of T cells, thereby enhancing their ability to attack tumor cells. Moreover, the use of growth factors such as IL-2 to stimulate T cell proliferation and activity is also the focus of current research. Through these methods, we hope to

achieve a more personalized treatment plan in the future, bringing new hope for cancer patients[1-2].

2. The role of serum IL-6 in treating the mechanisms of immune escape in patients with minimal tumors

Interleukin 6 (IL-6) is a cytokine that plays an important role in immune responses and inflammatory responses. In the treatment of patients with tiny tumors, the mechanism of IL-6 mainly involves the following aspects:

Interleukin-6 (IL-6) is a 26 KD polypeptide synthesized and secreted by a variety of cells with a wide range of biological effects. Its main functions include regulating the growth and differentiation of B cells, enhancing CTL, the killing effect of NK cells, stimulating the proliferation and differentiation of hematopoietic stem cells, and promoting the synthesis of acute phase protein by hepatocytes. IL-6 plays an important role in maintaining the physiological balance of the body because IL-6 secreting cells in all systems. In addition, IL-6 has several other biological activities. For example, it can promote the expression of IL-2r on the surface of T cells and enhance the mitogenic effects of IL-1 and TNF on TH cells. In acute inflammatory responses caused by infection or trauma, IL-6 can induce the synthesis of acute phase reactive proteins, especially with the increase in amyloid A and c-reactive proteins. However, IL-6 does not stimulate the secretion of other cytokines by the corresponding cells, and its autocrine effect on immune cells is also relatively weak at physiological concentrations, suggesting that its main immunological function is to strengthen the effect of other cytokines. Notably, abnormalities in IL-6 in vivo have been implicated in multiple diseases. For example, IL-6 levels may be elevated in patients with multiple myeloma and psoriasis. Therefore, detecting the level of IL-6 is important for the diagnosis and treatment of some diseases. For IL-6 detection, ELISA (enzyme-linked immunosorbent test) or tetramethylazazole blue color method can be used. In order to ensure the accuracy of the test results, we need to pay attention to some matters, such as fasting for 12 hours before blood drawing, taking fresh blood for testing, relaxing the blood drawing, avoid the contraction of blood vessels caused by fear, increase the difficulty of blood collection, and the pressing time should be sufficient. Overall, interleukin-6 (IL-6) is a molecule with a wide range of biological activities, involved in various physiological and pathological processes of the body. Understanding the biological function of IL-6 and its expression in different diseases may facilitate a deeper understanding of the disease pathogenesis and provide ideas for the development of new therapeutic strategies.

Promote the growth and differentiation of B cells: the moderate expression of IL-6 in the tumor microenvironment can promote B cells to enter their germinal center, produce appropriate somatic high-frequency mutations, and then improve the affinity of their anti-tumor antibodies, and build a protective line for the immune system. B cells are not only the source of antibody production, but also the provider of tumor-associated antigens, ensuring that tumors are continuously monitored.

Enhancing the killing ability of CTL and NK cells: IL-6 significantly enhances the activity of cytotoxic T lymphocytes (CTL) and natural killer (NK) cells. These cells are the "scavengers" of the human body and are specifically responsible for the identification and clearance of infected or abnormal cells, especially those that develop genetic mutations. Under the regulation of IL-6, they can find and eliminate tumor cells more accurately and quickly, thus curbing the growth and spread of tumors.

Stimulate the differentiation of hematopoietic stem cells: IL-6 plays a critical role in regulating the proliferation and differentiation of hematopoietic stem cells. This not only ensures the formation and function of the various types of leukocytes in the immune system, but also is crucial for reconstructing the immune system damaged by the disease. Healthy hematopoietic stem cells provide fresh "soldiers" to the immune system, enabling the body to better fight disease.

In conclusion, the important role of interleukin 6 (IL-6) in the treatment of patients with minimal tumors cannot be ignored. The mechanism of action is complex and elaborate, involving multiple links of the immune response. However, we still need to investigate the specific mechanism of IL-6 in microtumor patients in great depth in order to provide more enlightenment for future therapeutic strategies[3-6].

3. The role of serum IL-8 in treating the mechanism of immune escape in patients with minimal tumors

Interleukin-8 (IL-8) is a cytokine that plays an important role in the tumor microenvironment. In the process of treating patients with tiny ma tumors, the mechanism of IL-8 is very complex and involves multiple interactions with the immune system.

First, IL-8 plays a key role in the immune escape of tumor cells. It is able to induce tumor cells expressing the Fas ligand, a molecule able to bind to the Fas receptor and trigger apoptosis. Through this mechanism, IL-8 helps tumor cells resist Fas-mediated cytotoxic effects and thus survive the immune challenge. Moreover, IL-8 also promotes the tumor cell expression of immune checkpoint molecules, such as PD-L1 and CTLA-4. These molecules are able to inhibit the activation and function of T cells, further reducing the body's immune response to tumors. However, the role of IL-8 is not entirely negative. Indeed, IL-8 also has some antitumor effects. It can stimulate the activation and aggregation of NK cells and T cells, which are important components of the body's anti-tumor immunity. By enhancing the activity of these cells, IL-8 contributes to the activation of the anti-tumor immune response. Moreover, IL-8 also induced the expression of Fas ligands by tumor cells, thus increasing the sensitivity of tumor cells to Fas-mediated cytotoxic effects. This implies that in some cases, IL-8 may help to direct tumor cells to apoptosis, thereby limiting their growth and spread.

In conclusion, the role of IL-8 in the treatment of immune escape mechanisms in patients with minimal ma tumors is a complex issue, involving multiple mechanisms and effects. The dual role of IL-8 in promoting immune escape and antitumor effects will need to be fully considered when deeply exploring and evaluating its potential use in therapy. Meanwhile, studies on how to effectively use IL-8 or other related molecules for tumor immunotherapy are still in progress to seek safer and more effective treatments.

4. The role of serum IL-10 in treating the mechanisms of immune escape in patients with minimal tumors

In exploring the role of serum IL-10 in the mechanism of immune escape in patients with minimal tumors, we cannot help digging into the complex mechanisms and potential therapeutic strategies involved in this field. IL-10, a seemingly ordinary immune regulator, actually has a huge influence in its subtle changes. It plays a key role not only in the inflammatory response, but also in the growth, spread and the game with the immune system.

In the tumor microenvironment, the role of IL-10 is particularly prominent. It is like a double-edged sword, which can not only promote the activation of immune cells, enhance the anti-tumor immune response, but also may be skillfully used by tumor cells to help them escape the attack of the immune system. This was mainly attributed to the inhibitory effect of IL-10 on dendritic cells and other antigen-presenting cells that play a crucial role in initiating T cell-mediated immune responses. When IL-10 gains the upper hand, T cell activation is limited, and tumor cells can quietly grow under the supervision of the immune system.

More complex, IL-10 not only affects the activity of immune cells, but also directly participates in the proliferation and survival of tumor cells. IL-10 was found to promote the malignant transformation of certain tumor cells and enhance their invasion and metastasis ability. This process involves a complex set of signal transduction pathways and the regulation of gene expression.

Faced with such cunning enemies, we have to find smarter ways to respond. At present, there is some active exploration on how to use or regulate the level of IL-10 to improve tumor immunotherapy. For example, neutralization of IL-10 by antibodies or targeting of IL-10 using gene therapies in the hope of reshaping the antitumor capacity of the immune system. These frontier research not only bring new hope for patients, but also provide us with more deep understanding of the mechanisms of tumor immune escape.

However, many of the technical challenges and clinical trials are still needed to truly achieve the widespread use of these strategies. The efficacy and safety of each treatment regimen requires meticulous evaluation in various types of tumors and in patients with different conditions. Only then can we ensure that the most appropriate treatment regimen is tailored for each patient to maximize their survival and quality of life. We expect more breakthroughs and more victories in the protracted battle against tiny tumors [7-12].

5. TNF- α role in the treatment of immune escape mechanisms in patients with minimal ma tumors

TNF- α , this seemingly ordinary scientific term, actually contains the mystery and value of life science. TNF- α , or tumor necrosis factor- α , is a cytokine that plays a critical role in the human body. In the biomedical field, the value of TNF- α cannot be ignored. It is not only a biological marker, but also a bioactive substance with multiple values. First, TNF- α has significant antitumor effects. As we all know, tumor is one of the important diseases that threaten human life and health. However, TNF- α can effectively curb tumor growth and spread by inducing apoptosis of tumor cells. This discovery provides new ideas and direction for cancer treatment, making TNF- α a hot field for researchers to explore. After extensive experimental and clinical studies, the potential value of TNF- α in anti-tumor has been gradually recognized, which provides new possibilities and hope for tumor treatment. Besides this, TNF- α has important immunomodulatory functions. As a key factor in the immune system, TNF- α is able to activate immune cells such as macrophages, neutrophils, and natural killer cells, regulating their activity and function. This helps to enhance the body's resistance to various pathogens and prevent the occurrence of infection and disease. Through an in-depth study of the immunomodulatory mechanisms of TNF- α , one can better understand the operating principles of the immune system and provide theoretical support and practical guidance for the prevention and treatment of immune-related diseases. Meanwhile, TNF- α also has a significant anti-inflammatory effect. Inflammatory response is a natural response of the human body to injury, infection and other stimuli. However, excessive inflammatory responses can often lead to tissue damage and disease deterioration. TNF- α is able to promote the recruitment and activation of inflammatory cells, helping to eliminate inflammation and reduce tissue damage. This property makes TNF- α a powerful tool for the treatment of inflammation-related diseases, bringing new therapeutic options and hope for patients. In addition, TNF- α also has a role in promoting tissue repair. In the case of tissue damage, TNF- α is able to stimulate the growth and differentiation of tissue cells and promote the repair and regeneration of damaged tissues. This feature makes TNF- α with broad applications in tissue engineering, regenerative medicine, etc. By taking advantage of the tissue-tissue role of TNF- α , people can develop more effective regenerative medicine treatments to provide better medical services and rehabilitation support for patients. In conclusion, TNF- α has great value in several aspects, including antitumor, immunomodulation, anti-inflammatory, and tissue repair. With the continuous progress of science and technology and the deepening of research, people's understanding of TNF- α will be more comprehensive and deep. In the future, TNF- α is expected to play a broader role in the biomedical field and make a greater contribution to the development of human health. At the same time, we should also be aware that any kind of bioactive substance has two sides, and the rational use and control of the dosage is crucial. In exploring and utilizing TNF- α , we should maintain a scientifically rigorous attitude to ensure that it functions within a safe and effective range and make a positive contribution to the health and well-being of mankind[13-15].

The role of the immune escape mechanism of TNF- α , as a key inflammatory factor, in the treatment of patients with minimal ma tumors cannot be ignored. It is like a double-edged sword, both may become the right-hand man against tumors, and may become the accomplice of tumor cells.

First, TNF- α has a strong ability to promote tumor cell proliferation. It acts like a conductor, directing the tumor cells to expand wildly. In the tumor microenvironment, the increase in TNF- α leads to the accelerated growth of tumor cells, allowing the originally tiny tumors to grow rapidly. Secondly, TNF- α also plays a key role in suppressing immune cell activity. It is like a man behind the scenes, weakening the immune system against the tumor cells by suppressing the activity of the immune cells. As a result, the tumor cells can escape under the eyes of the immune system and continue to grow freely. Moreover, TNF- α has a role in promoting angiogenesis. Like an architect, it builds a rich network of blood vessels for tumors, delivering a steady stream of nutrients and oxygen. This undoubtedly provides a strong support for tumour growth and spread. Moreover, TNF- α also promotes tumor cell metastasis. It acts like a guide, directing tumor cells across tissue barriers to spread elsewhere. This process makes treatment of tumors more difficult because metastatic tumor cells can become new lesions even if the primary site is controlled.

6. Conclusion

Therefore, the immune escape mechanism of TNF- α in the treatment of tiny ma tumors. Only in this way can we give full play to the advantages of immunotherapy to bring better therapeutic effects to

patients.

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