THE PERIPHERAL IMMUNE RESPONSE OF POST-STROKE EPILEPSY

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Abstract: The development of post-stroke epilepsy requires the adjustment of treatment, taking into account the potential for drug interactions. An essential component of recovery following a stroke is a range of rehabilitation measures, the level of success of which is influenced, among other factors, by the patient's rehabilitation potential, which in turn is influenced by their emotional state, cognitive function, and stress tolerance and most important pars is immune disorders. The exact mechanisms whereby the injured brain sends out these first DAMP signals to trigger acute systemic inflammation still remain unclear.

The aim of the research was determine the impact of immune response on the early phase following a stroke and the restoration of impaired functions among patients.

The peripheral immune response to stroke is initiated within minutes after stroke onset. DAMP are originated from dying or stressed cells within the ischemic brain or actively secreted by immune cells upon activation. Circulating DAMPs activate peripheral immune cells and provoke a massive expression and release of pro-inflammatory cytokines into the bloodstream. Within the acute phase, stroke also induces the mobilization of more leukocytes from the spleen and the bone marrow as well as the activation of neurogenic pathways. In the subacute phase, within hours to days after stroke onset, a state of immunosuppression is triggered. The prolonged overactivation of neurogenic pathways as well as DAMP and other pro-inflammatory mediators acutely released after stroke gradually induce lymphopenia due to massive cell death and the pronounced bias towards the monocyte differentiation pathway in bone marrow hematopoiesis.Research into biomarkers of post-stroke epilepsy is crucial for enhancing the early detection and management of this condition. By identifying specific genetic factors, proteins, and neuroimaging findings associated with pse, healthcare providers can develop more targeted and effective treatment strategies. For example, certain genetic markers may indicate a higher predisposition to developing epilepsy after a stroke, allowing for preventive measures to be put in place. Additionally, monitoring changes in biomarker levels over time can help healthcare providers assess the progression and adjust treatment plans accordingly. Furthermore, the use of biomarkers in the diagnosis of post-stroke epilepsy can also aid in predicting outcomes and prognosis for patients. By correlating certain biomarkers with seizure recurrence, severity, and response to treatment, healthcare providers can better anticipate the course of the disease and make informed decisions about patient care. This personalized approach to managing pse can lead to improved quality of life and reduced healthcare costs in the long run.

Conclusion. In addition to their diagnostic and prognostic utility, biomarkers of post-stroke epilepsy also offer valuable insights into the underlying pathophysiology of the condition. By

studying the molecular and cellular changes associated with pse, researchers can uncover new potential targets for therapy and develop innovative treatment approaches. Overall, the continued investigation and validation of biomarkers in the context of post-stroke epilepsy hold great promise for advancing our understanding of the disease and improving outcomes for patients.