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The role of mitophagy in the pathogenesis of ischemic stroke

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Objective: More and more studies have shown that mitochondrial autophagy has a close relationship with the occurrence of cerebral ischemic stroke (CIS). Therefore, this paper reviews the mechanism of mitochondrial autophagy in the development of CIS. Methods: In this paper, we searched the databases of China Knowledge Network, Web of Science, PubMed, and other databases with the keywords "ischemic stroke" and "mitochondrial autophagy" by the method of literature search, and collected and analyzed the relevant literature in recent years. Results: The expression of NIX and BNIP3, as mitochondrial autophagy receptors in mammalian cells, is upregulated during the onset of ischemic stroke, which activates mitochondrial autophagy to ameliorate brain injury due to CIS. However, over-regulation of BNIP3 during ischemia-reperfusion leads to cell death. PINK1/Parkin, a classical pathway of mitochondrial autophagy, can play an anti-apoptotic, anti-inflammatory, and anti-oxidative stress role after the occurrence of CIS by regulating its expression level. Conclusions: Mitochondrial autophagy is activated during CIS and acts on other related pathological processes by removing damaged mitochondria, which are closely linked to biological processes such as apoptosis, oxidative stress, and inflammation in cells, thus affecting neuronal cell survival and death. Mitochondrial autophagy acts as an early defense mechanism after the onset of CIS to remove damaged mitochondria in a timely manner and reduce stimulation and damage to normal mitochondria, but when autophagy is excessive or blocked, it may exacerbate the damage. Therefore, as a double-edged sword, the mitochondrial autophagy process is expected to be a new target for the treatment of CIS.

Keywords: Mitophagy; Ischemic stroke; BNIP3

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The impact of astrocytes on the immune response of stroke

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Objective: After ischemic stroke (IS), a cascade of reactions is triggered, leading to inflammation and immune responses. Astrocytes (Ast), as the primary glial cells in the central nervous system, play a crucial role in maintaining brain homeostasis. Method: Activated Ast can increase the uptake of extracellular glutamate and sodium/potassium-ATP enzyme activity, improving the reconstruction of the blood-brain barrier during the acute phase of IS. Ast also secretes chemotactic and adhesion molecules, regulating the activity and function of immune cells, and influencing inflammation development. Ast can interact with neurons, phagocytizing and degrading apoptotic neurons and cell fragments, playing an important role in immune surveillance and clearance. Therefore, Ast is crucial in regulating brain immune and repair functions, controlling inflammation and immune responses, as well as promoting neuronal repair and regeneration. Additionally, Ast can undergo reactive astrocytosis after IS, isolating lesions and limiting neuroinflammation, but also impeding axonal regeneration. Reactive Ast can also interact with microglia to release pro-inflammatory substances, causing secondary neuronal damage. However, the removal of Ast will result in neuron survival. Results: Ast can take up extracellular glutamate, release neurotrophic factors, and stabilize extracellular fluid and ion homeostasis, displaying neuroprotective effects. Similar to microglia, Ast has a dual role in the immune response. Conclusion: Ast plays a neuroprotective role in IS.

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Delayed Treatment Resulted in A Better Outcome in Spontaneous Spinal Epidural Hematoma: Case Report

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Spontaneous Spinal Epidural Hematoma (SSEH) is a rare but potentially devastating medical emergency characterized by the accumulation of blood in the epidural space surrounding the spinal cord.

A 65-year-old male presented with sudden stabbing back pain and stiffness, which gradually progressed to complete paraplegia with bladder dysfunction four days before came to the hospital with no risk factors related. Magnetic Resonance Imaging (MRI) showed epidural hematoma. The patient underwent surgery to remove the hematoma and laminectomy for decompression. After five weeks in the hospital with appropriate and consistent rehabilitation, the patient`s symptoms improved significantly postoperatively, leading to discharge.

spinal epidural hematoma remains a rare but critical condition that requires prompt recognition and intervention to optimize patient outcomes.

Keywords: Spinal epidural hematoma, Rehabilitation, neurologic deficit

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Inherited neurological disorders in Consanguineous families in Palestine and Israeli Arabs.

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Consanguinity leads to a high prevalence of autosomal recessive disorders in inbred populations. Arab community in Israel and the Palestine are an example, but specialized medical care is limited in Palestine. Genetic diagnosis and genetic counseling with specific treatment are a must. In this study whole-exome sequencing as a first-line diagnostic tool in 83 Palestinian and Israeli Arab families with suspected neurogenetic disorders was performed and probable genetic diagnosis was established in 51% of the families (42 families). Pathogenic, likely pathogenic or highly suggestive candidate variants were found in the following genes extending and refining the mutational and phenotypic spectrum of these rare disorders: ACO2, ADAT3, ALS2, AMPD2, APTX, B4GALNT1, CAPN1, CLCN1, CNTNAP1, DNAJC6, GAMT, GPT2, KCNQ2, KIF11, LCA5, MCOLN1, MECP2, MFN2, MTMR2, NT5C2, NTRK1, PEX1, POLR3A, PRICKLE1, PRKN, PRX, SCAPER, SEPSECS, SGCG, SLC25A15, SPG11, SYNJ1, TMCO1, and TSEN54. Thus, this cohort has proven to be optimal for prioritization of new disease genes. Two separately published candidate genes (WWOX and PAX7) were identified in this study. Analyzing the runs of homozygosity (ROHs) derived from the Exome sequencing data as a marker for the rate of inbreeding, revealed significantly longer ROHs in the included families compared with a German control cohort. The total length of ROHs correlated with the detection rate of recessive disease-causing variants. Identification of the disease-causing gene led to new therapeutic options in four families. Finally, the results of the study carry consanguinity awareness for affected families and to health policy makers.