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Opinion Article

Platelet Mitochondrial Abnormalities and their Contribution to Delirium

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DESCRIPTION

Delirium is a neuropsychiatric syndrome characterized by acute cognitive disturbances, fluctuating attention and altered consciousness. It often affects individuals in critical care settings, post-surgical patients and those with underlying health conditions. The complexity of delirium arises from its multifactorial etiology, involving systemic inflammation, metabolic derangements and disruptions in cellular energy homeostasis. Recent research has suggested that alterations in platelet bioenergetics may play a role in the development and progression of delirium.

Platelets, also known as thrombocytes, are anucleate cells derived from megakaryocytes. While their primary function involves hemostasis and clot formation, platelets are also involved in immune modulation, inflammation and intercellular signaling. These activities rely heavily on energy metabolism, which is regulated by mitochondrial bioenergetics. Platelet bioenergetics, therefore, represents a critical aspect of their functionality, linking cellular energy metabolism with systemic physiological and pathological processes.

Mitochondria within platelets are responsible for adenosine triphosphate production through oxidative phosphorylation. This process provides the energy necessary for platelet activation, adhesion, aggregation and secretion of bioactive molecules. In addition to energy production, platelet mitochondria regulate redox balance, calcium homeostasis and apoptotic signaling. Dysregulation of mitochondrial function can impair platelet activity and contribute to systemic disturbances that may impact the brain and other organs.

The association between platelet bioenergetics and delirium lies in the shared involvement of metabolic and inflammatory pathways. Delirium is often accompanied by systemic inflammation, oxidative stress and metabolic disruptions. Platelets, as active participants in inflammatory responses, can amplify these processes through the release of pro-inflammatory cytokines, chemokines and reactive oxygen species. Dysfunctional platelet bioenergetics may exacerbate systemic inflammation, promoting the neuro-inflammatory state associated with delirium.

Oxidative stress is a characteristic of platelet bioenergetic dysfunction and is closely tied to the development of delirium. During oxidative phosphorylation, mitochondria produce reactive oxygen species as a byproduct. Under normal conditions, these species are neutralized by antioxidant systems. However, an imbalance between reactive oxygen species production and antioxidant capacity leads to oxidative damage. In platelets, oxidative stress can impair mitochondrial function, reduce ATP production and promote the release of inflammatory mediators.

Platelet bioenergetic dysfunction also impacts cellular signaling pathways involved in coagulation and vascular homeostasis. Increased platelet activation and aggregation can result in microvascular thrombosis and reduced blood flow to vital organs, including the brain. Cerebral hypoperfusion and ischemia are known contributors to delirium, as they disrupt neuronal metabolism and promote neuroinflammation. Platelets, through their bioenergetic alterations, may therefore influence the vascular contributions to cognitive dysfunction.

Another aspect of platelet bioenergetics relevant to delirium is the role of metabolic flexibility. Under physiological conditions, platelets can switch between oxidative phosphorylation and glycolysis to meet energy demands. This metabolic flexibility allows them to adapt to changes in oxygen availability and energy requirements. In pathological conditions, such as systemic inflammation or hypoxia, platelets may lose this adaptability, resulting in impaired energy metabolism and cellular dysfunction. Reduced metabolic flexibility in platelets has been observed in conditions associated with delirium, such as sepsis and major surgery.

The study of platelet bioenergetics in delirium involves the use of advanced analytical techniques to assess mitochondrial function and metabolic pathways. Methods such as extracellular flux analysis can measure oxygen consumption rate and extracellular acidification rate, providing insights into oxidative phosphorylation and glycolysis, respectively. These approaches

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have revealed that individuals with delirium exhibit alterations in platelet mitochondrial function, including reduced ATP production and increased oxidative stress markers.

One potential mechanism linking platelet bioenergetics to delirium is the release of mitochondria-derived danger signals. Damaged or dysfunctional mitochondria can release mitochondrial DNA, cardiolipin and other components into the extracellular space. These molecules act as damage-associated molecular patterns that activate immune cells and promote systemic inflammation. Platelet-derived mitochondrial danger signals may contribute to the neuroinflammatory environment observed in delirium, linking peripheral metabolic dysfunction with central nervous system pathology.

The clinical implications of platelet bioenergetic dysfunction in delirium are significant. Platelet bioenergetics may serve as a biomarker for identifying individuals at risk of developing delirium, particularly in critical care settings. Monitoring mitochondrial function and oxidative stress markers in platelets could provide early indications of metabolic and inflammatory disturbances. This information could guide interventions aimed at preventing or mitigating delirium.