Pathophysiological roles of calcium channels and transporters in multiple myeloma

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Video Byte

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Abstract

Multiple myeloma (MM) is a common type of plasma cell cancer that remains aggressive and incurable despite the development of several treatments. Approximately 70–80% of patients with MM have myeloma bone disease, which involves bone fractures and high blood calcium (Ca^{2+}) and affects MM prognosis. Various calcium channels and transporters help balance calcium levels, so they may be closely related to MM prognosis. For example, plasma membrane calcium channels allow calcium ions to enter cells, while proteins involved in store-operated calcium entry (SOCE) mediate calcium release from sites in the endoplasmic reticulum (ER)/sarcoplasmic reticulum (SR). Mitochondrial calcium channels regulate calcium uptake into mitochondria, which contributes to SOCE, and calcium-ATPases pump calcium ions from the cytoplasm back into the ER/SR or extracellular space. These molecules have been reported to be altered in the context of MM, but the specific mechanism by which their dysfunction leads to MM remains unclear. Further research on these molecules will help clarify the details of MM pathogenesis and enable the development of new biomarkers and treatment strategies for this devastating disease.