Necroptosis is a programmed death pathway involving in pituitary adenoma tumorgenesis

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Introduction: Pituitary adenomas impose burden of morbidity due to hormone hyper secretion and related effects on patients. Molecular mechanism underlying its incidence, development and progression have yet to be elucidated which can provide insights into new and more efficient therapeutic approaches. The involvement of necroptosis as an appealing way of cell death in pathogenesis of pituitary adenomas is perused in the current study.

Methods: The expression level of necroptosis crucial mediators (RIP1K, RIP3K, and MLKL) was assessed via Real-Time PCR in tumor tissues of prevalent functional and non-functional pituitary adenoma and normal Pituitary tissues. The effect of Shikonin on the cell viability and induction of apoptosis or necrosis in the presence and absence of necroptosis inhibitor (Necrostatin-1) were evaluated in pituitary adenoma cell line (GH3).

Results: Our results revealed that RIP1K expression level was increased in tumor tissues of different types of pituitary adenomas which was associated with significant decrease in the expression level of RIP3K and MLKL in tumor tissues comparing to normal pituitary. Shikonin reduced the percentage of GH3 viable cells in a dose dependent manner which was associated with the induction of apoptosis and necrosis. The Shikonin-induced cell death was diminished in response to suppression of necroptosis.

Conclusion: Necroptosis pathway is involved in the regulation of pituitary tumor cell proliferation. Suppression of necroptosis resulted in an accelerated cell proliferation which can cause pituitary tumor formation. Therefore, necroptosis biomarkers can be perused as hallmark mediators and the necroptosis pathway activation can be targeted as a therapeutic solution in management of pituitary tumors.

Speaker Biography

Alireza Sheikhi is the manager of the BioChem lab and senior scientist in Armenia (clinical lab and cancer research).

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