The role and mechanism of collagen type VI alpha 6 (COL6A6) on tumor growth and metastasis in - pituitary neuroendocrine tumor (PA) was determined. COL6A6 was downregulated in PA tissues and cell lines, which was negatively associated with the expression of prolyl-4-hydroxylase alpha polypeptide III (P4HA3) in the progression of PA. Overexpression of COL6A6 significantly suppressed tumor growth and metastasis capacity in PA. In addition, P4HA3 worked as the upstream of the PI3K-Akt pathway to alleviate the antitumor activity of COL6A6 on the growth and metastasis of both AtT-20 and HP75 cells. Furthermore, the inhibitory effect of COL6A6 on cell proliferation, migration and invasion, and epithelial-mesenchymal transition (EMT) was reversed by P4HA3 overexpression or activation of the PI3K-Akt pathway induced by IGF-1 addition, which provided a new biomarker for clinical PA treatment [1)].

1)
Long R, Liu Z, Li J, Yu H. COL6A6 interacted with P4HA3 to suppress the growth and metastasis of pituitary neuroendocrine tumor via blocking PI3K-Akt pathway. Aging (Albany NY). 2019 Oct 17;11. doi: 10.18632/aging.102300. [Epub ahead of print] PubMed PMID: 31627190.

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