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Dexmedetomidine inhibits inflammation and angiogenesis and alleviates esophageal cancer progression through ITGA6/PI3K/AKT pathway

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Esophageal cancer (EC) is an aggressive malignancy with high mortality and poor prognosis worldwide. Dexmedetomidine (DEX) shows anticancer potential but its effects on this disease are unknown. This study aims to investigate the role and mechanism of DEX in esophageal cancer through the ITGA6/ PI3K/AKT pathway. *In vitro* results indicated that DEX dose-dependently inhibited the proliferation, migration, and invasion of EC cells, while promoting apoptosis. DEX significantly reduced the secretion and expression of pro-inflammatory cytokines and downregulated the expression of angiogenesis-related factors. Mechanistic studies revealed that DEX significantly downregulated the expression of ITGA6 in EC cells and inhibited the phosphorylation activation of the PI3K/AKT pathway. Overexpression of ITGA6 partially reversed the inhibitory effects of DEX on the malignant progression, inflammatory response, and angiogenesis of EC cells, while inhibition of ITGA6 enhanced the antitumor effects of DEX. *In vivo* results were highly consistent with the *in vitro* findings, further confirming the antitumor effects of DEX. DEX inhibits the ITGA6/PI3K/AKT pathway, thereby suppressing the inflammatory response and angiogenesis, ultimately alleviating the progression of EC.

Keywords: Esophageal cancer progression, Pro-inflammatory factors, Invasion, Migration, Apoptosis, Cellular angiogenesis