miR-495 are lower in clinical ESCC tissues than in adjacent non-tumor tissues. Moreover, the lower miR-495 expression correlated with increased lymph node metastasis (LNM), invasion and TNM stage. miR-495 overexpression predicted a favorable outcome in ESCC patients. miR-495 targeted a site in the 3'-UTR of Akt1, and miR-495 levels correlated inversely with Akt1 protein levels in ESCC tissue samples. Overexpression of miR-495 suppressed cell proliferation, blocked G1/S phase transition, and decreased migration and invasion by two ESCC cell lines in vitro and in vivo. Restoration of Akt1 protein levels in miR-495-overexpressing ESCC cells attenuated the inhibitory effects of miR-495. In addition, miR-495 suppressed cell cycle transition and the EMT signaling pathway through targeting Akt1, thereby inhibiting ESCC cell proliferation, migration, and invasion. Our results suggest that miR-495 may act as a tumor suppressor by targeting Akt1 in ESCC.