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Esophageal squamous cell carcinoma (ESCC) is among the most common human cancers, with an overall v year survival rate of around 20%. To improve the diagnosis and prognosis of ESCC, we performed systema studies on the molecular alterations in the disease. Frequent gains of chromosomal bands 3q26, 8q24, 11q losses of 3p14 and 9p21, ampli cations of genes CCND1, EMS1 (CTTN), EGFR, PLK1, SKP2, PRKCI (PKCiot deletions of CDKN2A/B, FHIT and rearrangements of NTRK3, DTL and PTPRD were found. e mutation pro ling was characterized and potential therapeutic targets were identi ed. We further investigated intratumor heterogeneity (ITH) of the molecular alterations and constructed phylogenetic trees for genomic evolution, in whicl the mutations of ERBB4, FGFR2, BRCA2, ATM, TP53 and copy number changes of 11q13 and 9p21 were e events and those of PI3K/MTOR pathway, KIT, AURKA, CCND2 and 3q26 were late. By proteomic techniques an immunohistochemistry, multiple proteins were observed with high expression in tumor tissues but negative/low expression in morphologically normal operative margins. Especially, copy number alterations of ANO1, CDKN2A and high expression of p63 and ANO1 were also present in precancerous lesions (dysplasia). We further explo the mechanisms underlying the development and progression of ESCC and revealed that CRT, CTTN, PKCio SKP2 and PLK1 enhanced cell motility and resistance to apoptosis and promoted tumor growth and metastasis activating the PI3K-AKT pathway, inhibiting beta-catenin degradation and up-regulating the apoptosis suppresso Survivin. ese ndings extend our understanding of ESCC, providing theoretical foundation for elucidating the mechanisms underlying the tumorigenesis of the esophagus and progression of ESCC and for develop classi cation biomarkers and therapy targets for ESCC treatment.

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