Molecular cytogenetics of ovarian granulosa cell tumors by comparative genomic hybridization

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摘要

Abstract

OBJECTIVE: Patients with stage I granulosa cell tumors (GCTs) may occasionally develop metastasis, which is hard to predict using pathologic criteria. It is interesting to elucidate whether certain chromosomal imbalances (CIs), detected by comparative genomic hybridization (CGH), could be useful prognostic markers. METHODS: CGH was used to identify CI(s) in 37 adult-type GCTs from 36 women. Nonrandom CIs were compared with clinical and pathological features to evaluate their significance as a prognostic marker. RESULTS: Twenty-two (61%) of the 36 primary tumors had CIs. One woman's tumor showed identical CIs to another tumor that occurred in contralateral ovary 2 years later, supporting a metastatic nature. The nonrandom CIs included losses of 22q (31%), 1p33-p36 (6%), 16p13.1 (6%), and 16q (6%) and gains of 14 (25%), 12 (14%), and 7p15-p21 (6%). No tumor exhibited high-level amplification. The associations between each CI and pathological features, including the growth pattern, tumor size, and mitotic activity, were not evident. The only CI repeatedly detected in tumors with metastasis was monosomy 22, which presented in 2 of the 4 cases with metastasis but also in 2 of the 5 cases without recurrence for more than 5 years. CONCLUSIONS: Monosomy 22 was the most common CI in GCTs, which often coexisted with trisomy 14 (in 55% cases). Deletion of 22q seems to be, albeit not very specific, associated with the risk of early metastases of stage I disease. The role of loss-of-function mutation(s) of certain putative tumor suppressor gene(s) on 22q is worthy of further investigations