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Molecular characteristics of two esophageal carcinosarcomas: A hint for the clonality of carcinomatic and sarcomatic tumor components

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Abstract

AIM: To study the clonality of the esophageal carcinosarcoma by using molecular approaches.

METHODS: Two esophageal carcinosarcomas were included in the study. Tumor area from dysplastic lesion, squamous cell carcinoma, basaloid cell carcinoma and spindle cell elements were microdissected separately. Each element was analyzed with 14 microsatellite markers and direct sequenced for *p53* gene and *ras* gene mutation.

RESULTS: Both tumors displayed a typical histologic feature of carcinosarcoma. Both cases showed the divergent differentiation by immunohistochemistry study. In case 1 the identical LOH at p53 and hMLH1 loci was detected. The heterogenous LOH was detected only in carcinosarcoma at RB1 and BRCA1 loci, while the LOH at ACTC locus was seen only in sarcoma. The same mutation of the splice site of exon 6-intron 6 displayed in the two tumor elements. In case 2, a coordinate LOH at RB locus was demonstrated in three types of tumor elements: squamous carcinoma, basaloid carcinoma and spindle cell element. A heterogenous LOH was seen only in spindle cells at TAP1 locus. No mutation in exon 5-8 of *p53* gene has been found in case 2. No mutation of K-ras gene was found.

CONCLUSION: Although the different differentiation, the two elements of esophageal carcinosarcoma may have a single clonality. The *p53* gene mutation occurred before the two differentiation directions switched. The distinct molecular genotype can be determined through molecular biological analysis. The microsatellite profiling can serve as an approach to find out which genetic alteration occurs before or after the differentiation is determines.

Key words: Esophageal neoplasms; Mutation; Genes, *p*53; Molecular biology; Heterozygosity loss

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