EXPRESSION OF WNT/B-CATENIN PATHWAY MOLECULES IN SMALL BOWEL ADENOCARCINOMA

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Targeted sequencing of 30 small bowel adenocarcinomas reveals that Wnt pathway gene mutations (APC and CTNNB1) occur in 26.7% of cases, substantially lower than in colorectal cancer, yet aberrant β-catenin staining in 40% of tumors suggests alternative mechanisms may activate this pathway.

KEY POINTS

- TP53 was the most frequently mutated gene (56.7%), followed by KRAS (33.3%), APC (16.7%), PIK3CA (16.7%), CTNNB1 (10%), BRAF (6.7%), and CDKN2A (6.7%).
- APC mutations were significantly more common in mismatch repair deficient tumors (P=0.041), while CTNNB1 mutations occurred only in proficient MMR tumors; no cases had both APC and CTNNB1 mutations.
- Aberrant β-catenin immunostaining was observed in 40% of cases, but only 1 of 5 APC mutation cases showed this pattern, and 8 cases with aberrant staining had neither APC nor CTNNB1 mutations.
- Three of five APC-mutated cases were negative for cyclin D1 and c-myc and showed normal membranous β-catenin staining, suggesting the Wnt pathway may be less activated despite APC mutation.
- The authors suggest the Wnt/β-catenin pathway may be activated or deregulated by mechanisms beyond APC and CTNNB1 mutations in small bowel adenocarcinoma, relevant for researchers investigating SBA pathogenesis.

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