

MOLECULAR TRACING OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS WITH ASSOCIATED UNDIFFERENTIATED CARCINOMAS

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Poster

Pancreas

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Molecular tracing of two cases of undifferentiated pancreatic carcinoma arising from intraductal papillary mucinous neoplasms reveals increased KRAS burden through marked polyploidy, TP53 mutations, and MYC amplification as key alterations driving progression from pre-invasive to invasive disease.

KEY POINTS

- Multi-regional targeted sequencing, copy number variation analysis, and fluorescence in-situ hybridization were performed on two cases of undifferentiated carcinoma derived from IPMNs to trace molecular alterations from pre-invasive to invasive stages.
- Both cases showed identical somatic mutations in KRAS and TP53 across IPMN and invasive carcinoma components, with case 1 also harboring GNAS R201S and RNF43 S321* mutations.
- Undifferentiated carcinoma components demonstrated marked KRAS polyploidy (copy number average 5.1 and 6.1, maximum 26 and 15) and MYC amplification (copy number average 6.3 and 5.3), while IPMN components showed intermediate KRAS polyploidy (copy number average 2.2 and 4.3).
- Intermediate KRAS polyploidy and MYC amplification had already begun subsequent to TP53 mutation in intraductal components, suggesting these alterations precede invasion.
- This is the first report tracing molecular alterations associated with undifferentiated carcinoma tumorigenesis from pre-invasive intraductal neoplasm and may inform understanding of progression mechanisms in rare aggressive pancreatic cancer variants.

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