**Gastrointestinal tumours**

**Clinicopathological correlation with mutation profiling of colorectal cancer for KRAS, BRAF, NRAS and PIK3CA genes in Indian patient cohort**


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**Aim/Background:** In India, colorectal cancer continues to be one of the leading causes of cancer-related mortality with annual 25,000 deaths recorded by Globocan 2012. In this population-based study, we examined the prevalence of KRAS, BRAF, NRAS and PIK3CA mutations in 150 colorectal cancer patients from different regions of India and their correlation with geographic distribution and clinicopathological characteristics such as age, sex, diagnosis, tumour pathology, histology, immunohistochemical, therapy and blood profiling.

**Methods:** Formalin-fixed paraffin-embedded tissues (N = 150) were prospectively collected from Indian CRC patients during the period from January 2013 to July 2015. Genomic DNA was isolated from tissue sections and screened for mutations in KRAS (exon 2, exon 3), BRAF (exon 15), NRAS (exon 2, exon 3) and PIK3CA (exon 9, exon 20) genes using automated DNA sequencing.

**Results:** Overall, the KRAS tumour mutation rate was 23% (35/150 positive cases). There was significant association (p < 0.05) between KRAS mutations, age and tumour differentiation. Statistical analysis revealed a higher prevalence of colorectal cancer with mutated KRAS gene in northern regions of the country. No significant association was observed between KRAS mutations and gender (p > 0.05). About 8% (12/150) of cases showed mutations in the BRAF gene. All were of the V600E type, which were frequent in patients who were more than 50 years old. BRAF mutations were more frequent in well-differentiated tumours. NRAS mutations were observed in 2% (3/150) of the cases. About 5% (8/150) of cases had PIK3CA mutations. No significant association of PIK3CA mutation with age, tumour differentiation, location, and other parameters was noted.

**Conclusions:** Our study indicates that KRAS, BRAF, NRAS and PIK3CA mutations in Indian colorectal cancer patients occur at lower levels compared to those of the population in Western developed nations. Our findings are consistent with the hypothesis that differences in patients’ origins and related genetic backgrounds may contribute to and even determine the incidence rate of somatic mutations in candidate cancer genes.

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