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Understanding the Association of KRAS G12C Status with Age at Onset of Metastatic Colorectal Cancer

Chloe Reble*

Department of Pediatrics, University of Toronto, Toronto, Canada

Introduction

Colorectal Cancer (CRC) remains a significant health burden globally, with metastatic disease posing a considerable challenge in management and prognosis. The KRAS gene mutation, particularly the G12C mutation, has garnered attention for its role in driving CRC progression. This article explores the association between KRAS G12C status and the age at onset of metastatic CRC. Understanding this association is crucial for personalized treatment strategies and improving outcomes in CRC patients, Colorectal Cancer (CRC) is one of the most prevalent malignancies worldwide, with a substantial impact on morbidity and mortality. While early detection and treatment have improved outcomes, metastatic CRC presents a formidable challenge due to its advanced stage at diagnosis and limited treatment options. KRAS mutations, particularly the G12C mutation, have emerged as significant drivers of CRC progression, influencing disease aggressiveness and therapeutic response. This article aims to examine the relationship between KRAS G12C status and the age at onset of metastatic CRC, shedding light on its implications for clinical management [1,2].

Description

KRAS is a proto-oncogene that encodes a small GTP ase protein involved in cell signalling pathways regulating proliferation and survival. Mutations in KRAS are among the most prevalent genetic alterations in CRC, occurring in approximately 30-40% of cases. The G12C mutation, characterized by a substitution of Glycine (G) with Cysteine (C) at codon 12, is one of the most common KRAS mutations in CRC. It leads to constitutive activation of downstream signalling pathways, promoting tumour growth, invasion, and metastasis. The age at diagnosis of CRC varies widely, with most cases diagnosed after the age of 50. However, a subset of patients presents with metastatic disease at an earlier age, posing challenges in management and prognosis. Several factors influence the age at onset of metastatic CRC, including genetic predisposition, environmental exposures, lifestyle factors, and molecular alterations. Understanding these factors is crucial for risk stratification and early detection strategies.

Understanding the association of KRAS G12C status with age at onset of metastatic colorectal cancer elucidates critical insights into disease progression. KRAS mutations, particularly G12C, are prevalent in colorectal cancer, influencing treatment outcomes. Analyzing age-related patterns sheds light on disease dynamics, potentially aiding in personalized treatment strategies. Early-onset cases may indicate aggressive disease behavior, necessitating tailored therapeutic approaches. Conversely, late-onset metastasis might implicate distinct molecular mechanisms or environmental factors. Investigating this association enhances our understanding of colorectal cancer heterogeneity and informs clinical decision-making for improved patient outcomes [3-5].

Recent studies have investigated the association between KRAS G12C status and the age at onset of metastatic CRC. While the literature is still evolving, emerging evidence suggests a potential correlation between KRAS G12C mutation and earlier age of metastasis. Several retrospective analyses and cohort studies have reported a higher prevalence of KRAS G12C mutation in younger CRC patients with metastatic disease compared to older counterparts. Furthermore, preclinical models have demonstrated enhanced tumorigenicity and metastatic potential associated with KRAS G12C mutation, supporting its role as a driver of aggressive disease phenotype, The underlying mechanisms linking KRAS G12C mutation to early onset of metastatic CRC are multifaceted. Constitutive activation of KRAS signaling pathways promotes tumor initiation, progression, and metastasis through dysregulation of cellular processes, including proliferation, survival, angiogenesis, and Epithelial-Mesenchymal Transition (EMT). Moreover, KRAS G12C mutation confers resistance to targeted therapies, such as anti-EGFR monoclonal antibodies, limiting treatment options in metastatic CRC patients.

Conclusion

The association between KRAS G12C status and age at onset of metastatic CRC has significant clinical implications for risk assessment, prognosis, and treatment selection. Younger CRC patients with KRAS G12C mutation may benefit from intensified surveillance strategies for early detection of metastatic disease and tailored therapeutic interventions. Additionally, the development of novel targeted agents and combination therapies targeting KRAS signalling pathways holds promise for improving outcomes in this subset of patients, the association between KRAS G12C status and age at onset of metastatic colorectal cancer represents a promising avenue for understanding disease pathogenesis and guiding clinical management. Targeting KRAS-driven signalling pathways holds potential for improving outcomes in younger CRC patients with aggressive disease phenotype. A multidisciplinary approach integrating genomic profiling, clinical risk stratification, and therapeutic innovations is essential for optimizing care and maximizing survival in metastatic CRC patients.

References

1. Brooks, Matthew L., Carla M. D'antonio, David M. Richardson and James B. Grace, et al. "Effects of invasive alien plants on fire regimes." *Biosci* 54 (2004): 677-688.
2. Brochet, Anne-Laure, Matthieu Guillemain, Hervé Fritz and Michel Gauthier-Clerc, et al. "The role of migratory ducks in the long-distance dispersal of native plants and the spread of exotic plants in Europe." *Ecography* 32 (2009): 919-928.
3. Nagendran, Monica and Daniel Riordan. "Automated cell-type classification in intact tissues by single-cell molecular profiling." *Elife* 7 (2018).
4. Braithwaite, Richard W., W. M. Lonsdale and J. A. Estbergs. "Alien vegetation and native biota in tropical Australia: The impact of mimosa pigra." *Biol Conserv* 48 (1989): 189-210.
5. Breen, A.J, Bonneaud C.C and Healy S.D, et al. "Social learning about construction behaviour *via* an artefact." Animal Cognition 22 (2019): 305-315.

***Address for Correspondence:** Chloe Reble, Department of Pediatrics, University of Toronto, Toronto, Canada, E-mail: chloereble@utoronto.ca*

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