EDITORIAL

Molecular Profiling in Colon Cancer: Integrating Research with Clinical Practice

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Colon cancer ranks third among malignancies worldwide and second among causes of cancer-related death [1]. In developing countries like Pakistan, infrastructure and therapeutic options remain limited, and the majority of our patients present with advanced metastatic disease. Moreover, the median age at presentation in our part of the world is under 47 years, roughly a decade earlier than that of the typical Western cohort [2]. It is now clinically necessary to comprehend the biology of this illness in our community.

Colon cancer acts like a collection of distinct diseases, each with a unique clinical trajectory and susceptibility to treatment. Microsatellite instability testing now identifies the patients who benefit from immune checkpoint inhibition [3]. While Rat Sarcoma viral oncogene homolog (RAS) and B-Raf proto-oncogene, serine/threonine kinase (BRAF) profiling guide targeted therapies and flag tumors likely to behave aggressively. Circulating tumor DNA adds a further layer, providing a real-time readout of disease progression and is most helpful in identifying minute residual disease following resection [4]. These markers increasingly inform multidisciplinary tumor board discussions, allowing treatment to be personalized rather than allocated solely on the basis of histopathology or tumor stage.

However, Western populations, whose tumor biology may differ from our own, continue to provide the vast majority of the therapeutic procedures used here. In Pakistani cancer-hospital series, around 72% of colorectal cancer patients had left-sided tumors, a distribution that deserves study on its own terms rather than being assumed to mirror Western data [5, 6]. Developing local, evidence-based guidelines is essential to providing our patients with the personalized treatment they deserve; it is a clinical necessity, not a luxury.

This space is what the accompanying paper begins to address. Different autosomal signatures across metabolic and growth-signaling pathways are described in the paper on age- and sex-stratified transcriptomic profiling of colon adenocarcinoma. This suggests that an older man and a young woman with histologically identical tumors may have very different underlying biology. These results support considering age, gender, and molecular subtype in addition to histology and stage when choosing a course of treatment.

Building local molecular registries, confirming these markers in our community, and setting up profiling systems that our hospitals can realistically sustain are among the practical difficulties that lie ahead. Every local study contributes to a thin body of information, and public-private collaborations and cost-effective testing techniques will likely be required to make profiling practical in resource-constrained settings. Artificial intelligence is also evolving as a tool to support this type of precision oncology by helping integrate genetic, pathological, radiological, and clinical data.

We hope this work will stimulate more regionally based research grounded in the molecular features of our own patients. It is a welcome contribution to those endeavors.

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