RESEARCH PAPER

Mismatch Repair Status in Colorectal Carcinoma: A Retrospective Observational Study with Comparative Insights into MMR and MSI Testing

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ABSTRACT

Background: Mismatch repair (MMR) deficiency and microsatellite instability (MSI) are crucial biomarkers in colorectal carcinoma (CRC), with prognostic and therapeutic implications. This study aimed to evaluate the prevalence of MMR deficiency and its association with clinicopathological parameters in CRC.

Methods: A retrospective observational study was conducted over a period of two years (January 2024–December 2025) in a tertiary cancer centre. A total of 56 cases of CRC that underwent MMR testing were included. Histopathological parameters and clinical details were analysed. MMR by immunohistochemistry for MLH1, PMS2, MSH2, and MSH6 was performed, and cases were categorised as MMR-proficient (pMMR) or deficient (dMMR). Selective cases underwent further molecular analysis by NGS.

Results: Among the 55 evaluable cases, 13 (23.63%) demonstrated dMMR status, while 42 (76.36%) were pMMR. The most common deficiency pattern was MLH1/PMS2 loss (38.46%). dMMR was more frequently observed in patients ≤50 years and was associated with a higher number of tumour-infiltrating lymphocytes than pMMR cases. Molecular analysis in two of the cases identified pathogenic germline mutations in *MLH1* and *APC*, indicating an association with Lynch syndrome and Familial Adenomatous Polyposis, respectively.

Conclusion: MMR-deficient colorectal carcinoma is a distinct group with characteristic clinicopathological features. IHC remains a practical screening tool, while orthogonal molecular testing improves diagnostic precision. Integration of MMR/MSI testing in routine practice, through universal testing, is crucial for identifying hereditary syndromes and optimising targeted and immunotherapeutic management.

Keywords: *Mismatch repair (MMR), microsatellite instability (MSI), colorectal carcinoma, precision oncology*

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INTRODUCTION

Colorectal carcinoma (CRC) is the third most common cancer in men and the second most common in women worldwide. In India, CRC is the sixth most common cancer and accounts for over 4% of total cancer incidence and mortality in India.[1]CRC is a heterogeneous disease caused by the interaction of genetic and environmental factors, with significant differences between clinical presentation, prognosis, and individual treatment response.

The molecular pathogenesis of colorectal carcinoma is a multistep process, driven by cumulative genetic and epigenetic alterations.The chromosomal instability pathway involves sequential genetic events with early *APC* mutation, followed by *KRAS* activation and *TP53* loss, promoting the 'adenoma–carcinoma' progression.[2]The microsatellite instability pathway results from the defects in mismatch repair genes, leading to hypermutation. The CpG island methylation causes epigenetic silencing of tumour suppressor genes. These alterations dysregulate key signalling pathways, including Wnt/β-catenin, *EGFR*, and TGF-β, resulting in

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uncontrolled proliferation, impaired apoptosis, and malignant transformation.[3]

Mismatch Repair (MMR) proteins are a highly conserved family of nuclear enzymes that maintain genomic stability by correcting errors made by DNA polymerases. These errors during DNA replication could result from the addition of a mismatched base or from DNA slippage. From the large number of MMR proteins, four are important: the mutL homologue 1 (MLH1), the mutS homologue 2 (MSH2), the mutS homologue 6 (MSH6) and postmeiotic segregation increased 2 (PMS2) and are of clinical significance. MSH2/MSH6 and MSH2/MSH3, which occur as heterodimers, detect the errors that occur during replication. The MLH1/PMS2 complex initiates the repair process by creating a nick in the DNA.[4]On the other side, microsatellites are repeated sequences of nucleotides that are 1-6 bp. The length of repeated nucleotides can vary and thus can be classified accordingly as mono-, di-, tri-, tetra-, penta- and hexanucleotide repeats. These unique sequences cover 1-3% of the human genome and are pervasive all over the genome.[3] Even though they are abundantly seen in the non-coding regions, they have very prevalent functional roles.[5] Microsatellites are susceptible to replication errors made by the DNA polymerase enzyme, and a deficiency in MMR (Mismatch Repair) protein can lead to Microsatellite Instability (MSI).

MMR testing by immunohistochemistry (IHC) detects the intactness/loss of MMR proteins (MLH1, MSH2, MSH6, PMS2), indicating the functionality of the respective genes. MSI testing by PCR-based panels detects length variations in microsatellites, reflecting genomic instability.[6] IHC is rapid, cost-effective, and indicates the mutated gene, whereas MSI testing is more sensitive and detects the change in length of microsatellites.

Mismatch repair (MMR) deficiency and microsatellite instability (MSI-high) are key biomarkers in precision oncology. MSI occurs in ~15% of sporadic CRC cases and 3% in Lynch syndrome, associated with hereditary nonpolyposis colorectal cancer (HNPCC) cases.[7] In colorectal carcinoma, they identify Lynch syndrome, guide prognosis (better stage-adjusted outcomes), and predict poor response to 5-FU therapy.[8] Critically, MSI-high tumours show strong responsiveness to immune checkpoint inhibitors such as pembrolizumab, making MMR/MSI testing essential for therapeutic decision-making.[9]

This study aims to determine the prevalence of mismatch repair deficiency in colorectal carcinoma and evaluate its association with clinicopathological parameters in a tertiary cancer centre.

MATERIALS AND METHODS

A retrospective, observational study of 2 years (January 2024 - December 2025) was conducted in the Department of Oncopathology, Yenepoya Medical College, Mangalore. All patients with a histological diagnosis of CRC and with subsequent MMR testing were included. The cases with relevant clinical details, as available in the test requisition form, were retrieved from the departmental archives. Additional clinical, radiological findings and treatment information were collected from the hospital medical records.

The surgical specimens (biopsy/resection) received in the oncopathology department were examined, and representative sections were submitted for histopathology evaluation. Haematoxylin and eosin (H&E) stained sections were assessedfor relevant histopathological parameters such as histological subtype, grade, tumour-infiltrating lymphocytes (TILs), lymphovascular invasion, perineural invasion and tumour budding.

Representative tumour sections of 4-micron thickness were cut from the selected blocks for IHC staining with four antibodies: The antibody panel was hMLH1 (clone GM011), hPMS2 (clone EP51), hMSH2 (clone RED2), and hMSH6 (clone EP49). The staining was performed on a manual platform, as per the institutional protocol.

Standard practice guidelines, such as the CAP protocol, were followed for reporting of the cases. The results were interpreted as **MMR proficient (pMMR)** when there was intact nuclear staining in at least 5% of the tumour cells or **MMR deficient (dMMR)** when there was loss of nuclear staining in one or more proteins. Cross verification of results on orthogonal platforms (PCR/NGS) was suggested when **equivocal/inconclusive IHC** results were obtained.

Two of the cases subsequently underwent molecular testing by NGS with hereditary cancer panels covering 137 genes, at a referral centre.

RESULTS

A total of 154 cases of colorectal carcinomas were reported during the study period, of which 56 underwent MMR testing and formed the study cohort. The age of our patients ranged from 26 to 85 years, with a median age of 51.5 years. The male-to-female ratio was 1.55:1 with slight male predominance. Left-sided CRCs were predominant (32, 68.08%) compared with right-sided tumours (15, 31.91%), with rectosigmoid carcinomas being the most common, constituting 49.1% of cases. Histopathologically, most cases were adenocarcinoma, NOS (48, 87.27%), followed by mucinous adenocarcinoma (6, 10.91%) and low-grade appendiceal mucinous adenocarcinoma (LAMN) (1, 1.82%). The majority of these cases were of histological grade 2 (34, 61.8%). TILs were high in 15 cases (27.3%), and most of these tumourswere right-sided tumours.

MMR evaluation

A total of 113 carcinoma cases underwent MMR testing in our centre during the study period, of which the majority (71, 62.8%) were from GI sites, followed by the endometrium. Among the GI sites, carcinomas of colorectal sites were the most common, 56 (78.9%). In one

case, the test was deemed inadequate due to a suboptimal tumour in the subsequent serial sections. Among those cases with satisfactory results (55 cases), 42 (76.36%) showed intact nuclear staining in the tumour cells and were reported as MMR-proficient *(figure 1)*. In contrast, the remaining 13, or 23.63%, cases showed loss of nuclear expression of MMR proteins, i.e., MMR-deficient *(figure 2)*. Among the dMMR cases, MLH1/PMS2 loss was the most common pattern (5, 38.46%), followed by isolated MSH6 loss (2, 15.38%) &isolated PMS2 loss (2, 15.38%). Certain uncommon patterns were noted, such as loss of [MLH1/PMS2 & MSH6]; isolated MLH1 loss; MSH2 & MSH6 loss and isolated MSH2 loss, each seen in a single (7.69%) case. *(Figure 3).*

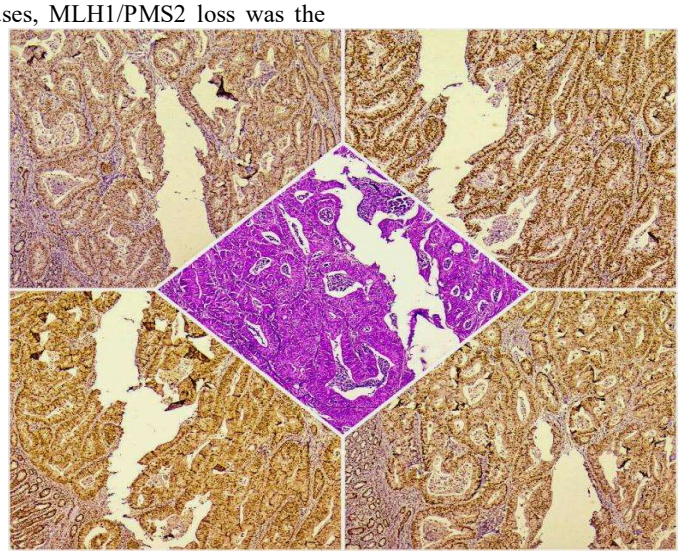

Figure 1: pMMR colorectal adenocarcinoma with intact nuclear expression of all MMR proteins.
H&E: 20X: Centre. IHC: 10X: Top left - MLH1; top right - PMS2; bottom left - MSH2; bottom right - MSH6.

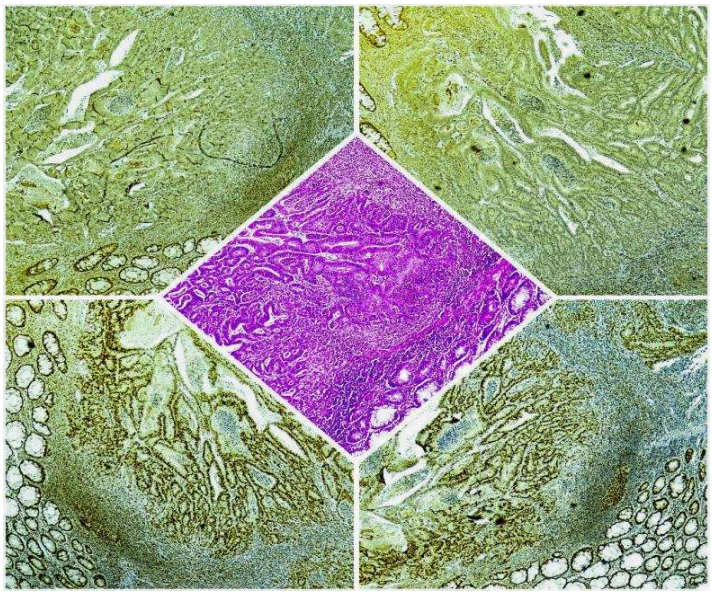

Figure 2: dMMR colorectal adenocarcinoma with loss of MLH1/PMS2
H&E: 10X: Centre. IHC: 10X: Top left - MLH1; top right - PMS2; bottom left - MSH2; bottom right - MSH6.

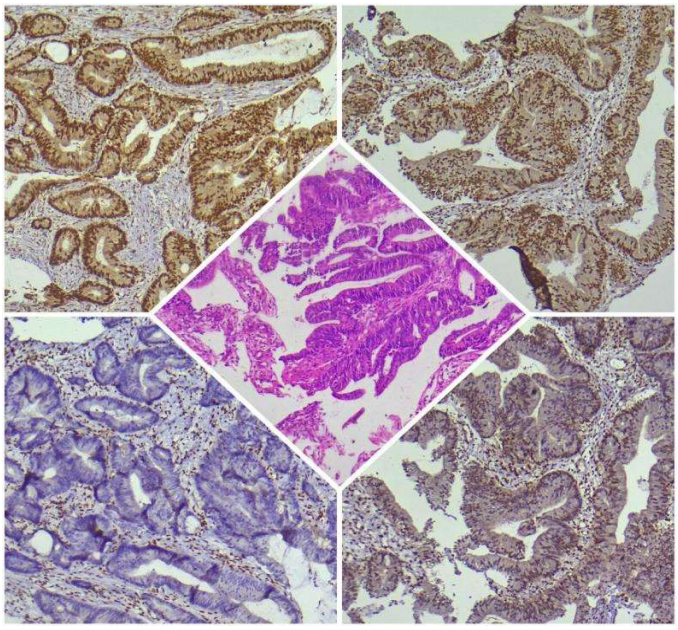

Figure 3: dMMR colorectal adenocarcinoma with isolated loss of MSH2
H&E: 10X: Centre. IHC: 10X: Top left - MLH1; top right - PMS2; bottom left - MSH2; bottom right - MSH6.

Clinical-Pathological Correlation:
Of the dMMR cases of CRC, most patients (9, 69.23%) were ≤50 years of age, compared to the remaining 4 (30.77%) patients who were >50 years of age. Among the dMMR cases, 7(53.9%) were right-sided tumours and the remaining 6 (46.1%) were left-sided. *(Figure4)*. On histopathological evaluation, 53.8% of these dMMR cases showed high TILs as compared to 24.6% of the pMMR cases with high TILs.

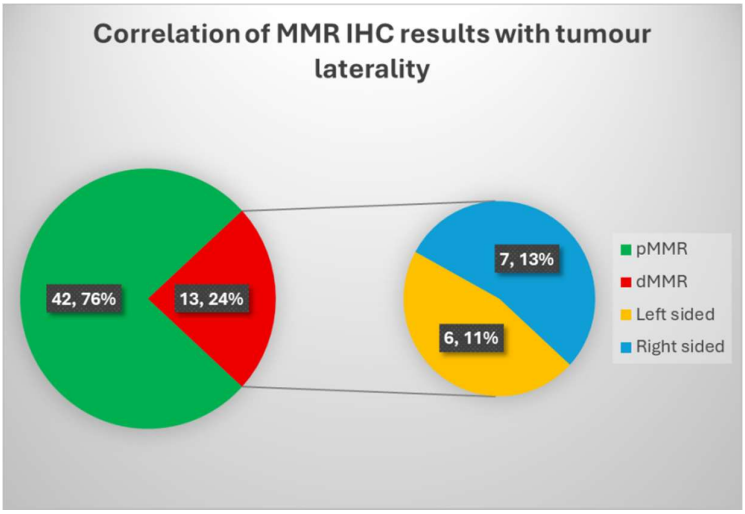

Figure 4: Pie of pie chart showing pMMR and dMMR CRCs with laterality distribution of dMMR CRCs.

Molecular analysis
Two of the CRC cases presented to us with a second malignancy, one with endometrial carcinoma and another with renal cell carcinoma, both within 18 months of initial presentation. The case of CRC with renal cell carcinoma and dMMR status subsequently underwent molecular testing by a hereditary cancer panel, which showed a pathogenic germline*MLH1* mutation in exon 19, i.e., c.2190del (p.Pro731LeufsTer52), suggestive of Lynch syndrome. Another case of pMMR with a strong family history also underwent hereditary testing and revealed a pathogenic germline mutation in the *APC* gene, i.e., c.637C>T (p.Arg213Ter), indicating a Familial Adenomatous Polyposis (FAP) syndrome.

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DISCUSSION

Mismatch repair (MMR) is a key biomarker in colorectal carcinoma, and ~15% of these cases show deficient mismatch repair (dMMR). The vast majority of these cases are sporadic, accounting for 12-17%, while the inherited cases account for ~3%, largely associated with Lynch syndrome.[10]In the current era of precision oncology, assessing dMMR or its phenotypic consequence, microsatellite instability (MSI-H), is essential for determining prognosis, guiding immunotherapy, and directing patient-specific adjuvant chemotherapy.[11]In early-stage CRC, dMMR/MSI-H is associated with better stage-adjusted survival and lower rates of lymph-node and distant metastases compared with pMMR/MSS tumours.[12] For metastatic CRCs without immunotherapy, the prognostic benefit of dMMR/MSI-H is less clear and sometimes neutral or even adverse.[13]

Colorectal carcinoma is primarily a disease of older adults, and these results were similar in our study, where the median age of our patients was 51.5 years. The majority of patients were males with a male-to-female ratio of 1.55:1, which is similar to that reported by ***RajatPandey et al.*** *and****Anurag Mehta et al.***[14, 15] Left-sided tumours were the most common, with the rectosigmoid being the most common site; these findings are similar to those reported by ***TijanaDenčićet al.***[16]

The dMMR cases constituted 23.63% of all the cases that underwent MMR testing in CRC, which is similar to the existing literature worldwide, as shown in Table 1.***Pangarkar M et al.***and***Kale et al.*** reported a frequency of 16.67% and 13.85%, whereas **Sharma et al.,** in their study on a limited sample size, reported a higher frequency of 32.72%.[17]***Yassen, N.N. et al.***reported a frequency of 28% of MSI-H cases, which is slightly higher.[18]These findings suggest a geographic and ethnic variation in the frequency of MMR deficiency.

The most common deficient pattern observed in our study was loss of MLH1/PMS2, noted in 38.46% of the cases. ***Tran NQ et al.*** reported this pattern in 38.45% of their cases, whereas ***RajatPandey et al.***reported it in 54.5% of cases.[14,19]***Scarpa M et a*l.** reported loss of MLH1/PMS2 in 32.14% of cases, followed by isolated MLH1 loss in 21.43% of cases.[20]

Table 1: Comparison of clinicopathological and MMR data from various studies across the globe.

	Our Study	RajatPandey*et al.* (14)	Anurag Mehta *et al.* (15)	Tran NQ et al. (19)	Scarpa M et al. (20)
Median Age (Yrs)	51.5	52.5	52	NA	69
Gender (M:F)	1.55:1	2.08:1	1.9:1	1.31:1	1.08:1
Site (L:R)	2.13:1	1.84:1	1:1.4	1.37:1	1:1.34
MMR (dMMR)	23.21 %	14.3%	39.01 %	16 %	24.78%
MLH1/PMS2	38.46 %	54.5%	-	38.45%	32.14%
Isolated PMS2	15.38%	9.1 %	-	30.77%	7.14%
Isolated MSH6	15.38%	-	-	0	3.6%
TILs (overall)	27.3%	-	-	-	37%

Uncommon MMR patterns

In the present study, 4 (30.8%) cases showed uncommon patterns, with one case of MLH1/PMS2 loss showing additional MSH6 loss. ***Scarpa M et al.*** reported certain uncommon patterns of MMR IHC, including triple loss of MLH1, PMS2, and MSH2 in 14.29% of cases and isolated MSH6 loss in 3.57% of cases.

Isolated losses of MSH6 & PMS2 were noted in 2 cases each, constituting 30.76% of cases overall, which is significantly higher than in the existing literature.[21]This variation could be due to the limited sample size and further necessitates correlation with larger regional studies.

Additionally, molecular testing was performed in two of the cases, one of which showed a germline*MLH1* mutation, confirming the association with Lynch syndrome. ***Sür Y et al***., in their study, found an association of 26.4% cases of CRC associated with Lynch syndrome.[22]

Challenges and pitfalls of MMR IHC testing

In routine diagnostic practice, pre-analytical / analytical challenges and interpretative pitfalls are occasionally encountered, making an otherwise straightforward diagnosis difficult.Suboptimal fixation, a key pre-analytical issue, could result in weak or absent nuclear staining, leading to false-negative results. In such a scenario, it's vital to evaluate well-fixed areas and look for internal positive controls to confirm the validity of test results.Dot-like or granular, punctate nuclear staining for MLH1 is a well-documented artefact in dMMRtumours, specifically associated with M1 (Ventana/Roche) and G168-15 (BD Biosciences) clones. Although it can be mistakenly interpreted as an intact expression, it is considered an aberrant pattern indicating loss of protein

function.[23]Heterogeneous or "patchy" staining in MMR IHC is a well-documented pitfall, particularly in MSH6 and PMS2. Tumours may display mixed retained and lost expression within the same tissue section and are often characterised by sharp, abrupt, or subclonal loss of staining, which can be misidentified as pMMR if the slide is not meticulously scanned.[24] This pattern reflects genuine intratumoral molecular diversity rather than a staining artefact; therefore, reporting heterogeneous loss (e.g.,"subclonal MSH6 loss in approximately 10% of tumour cells") helps drive appropriate follow-up. Patients with heterogeneous MMR protein loss have approximately a 50% chance of being MSI-H and eligible for ICI therapy.[25] Weak or suboptimal nuclear staining (significantly fainter staining than internal controls) should not be interpreted as "intact" or "positive" (proficient). Non-nuclear staining patterns such as cytoplasmic, perinuclear, or fine-punctate ("dot-like") staining are not

true nuclear expression and should be considered abnormal or indeterminate.[26]**Prior chemo/radiation therapy** can induce MMR protein expression loss, particularly MSH6, creating a dMMR phenotype that may not reflect the patient's tumour baseline status. [26]

Orthogonal testing platforms: (MSI testing by PCR and NGS)

The inconclusive MMR results need to be cross-verified by MSI testing on orthogonal platforms such as PCR or NGS. PCR-based assays detect length variations/stutter in microsatellites using fluorescent multiplex PCR and capillary electrophoresis. A variety of panels exist, including the traditional Bethesda panel (two mononucleotide (BAT-25, BAT-26) and three dinucleotide markers). (D2S123, D5S346, and D17S250) and a more sensitive Pentaplex Panel [mononucleotide markers (BAT-25, BAT-26, NR-21, NR-24, and NR-27)]. Some common commercial platforms in routine practice are Promega MSI Analysis System, Idylla™ MSITest (Biocartis), and newer ones like TrueMark™ MSIAssay (Thermo Fisher). The results of MMR immunohistochemistry are found concordant with PCR-based MSI testing in colorectal cancer; however, a lower concordance has been observed in endometrial cancer.[27] Limitations of PCR-based MSI testing include longer turnaround times, higher tumour volume requirements and varying sensitivity across different tumour types.

NGS-based MSI testing analyses multiple loci and offers high sensitivity and specificity, with the added advantage of simultaneous detection of pathogenic variants and estimation of tumour mutational burden (TMB) when part of comprehensive panels. The performance of MSI testing on NGS depends on panel design and validation; however, recent data quote non-inferiority to IHC and strong concordance across various tumour types.[28]Adequate tissue-tumour, longer turnaround time, higher running cost and requirement of resources/bioinformatics are the familiar limitations of NGS testing.

These methods are complementary and assess different aspects of the mismatch repair system. Generally they are highly concordant (>90%), however, the combined use improves diagnostic accuracy (99%), thus guiding appropriate treatment decisions, particularly immunotherapy.

CONCLUSION

MMR/MSI testing in colorectal carcinoma is integral to precision oncology. Immunohistochemistry, a commonly available ancillary tool, serves as a reliable surrogate for microsatellite instability (MSI) testing in routine clinical practice. The clinical implications of MMR/MSI testing include prognostication, identification of Lynch syndrome, and selection of patients for immunotherapy. Integrating PCR and next-generation sequencing-based molecular techniques for cross-verification of results can further enhance diagnostic accuracy and facilitate more precise, personalised treatment strategies in colorectal cancer.

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