Research Article / *Araştırma Makalesi*

Ege Journal of Medicine / *Ege Tıp Dergisi* 2026; 65 (1): 33-39

Deficient mismatch repair in uterine adenocarcinoma: Frequency clinicopathological correlations and prognostic implications

Uterin adenokarsinomda MMR protein ekspresyon kaybı: Sıklığı klinikopatolojik korelasyonları ve prognostik etkileri

Ali Tümkaya[1] Kenan Serdar Dolapçıoğlu[1] Esin Doğan[2] Ahmet Beyazıt[1] Can Yazırlı[2]

[1]Hatay Mustafa Kemal University Department of Obstetrics and Gynecology Hatay/Türkiye

[2]Department of Pathology, Faculty of Medicine, Hatay Mustafa Kemal University, Hatay/Türkiye

ABSTRACT

Aim: Endometrial cancer is the most common gynecological malignancy worldwide. The Cancer Genome Atlas (TCGA) molecular classification system has identified four distinct molecular subtypes with mutation burden and copy number alterations. Deficient mismatch repair (dMMR) is an important molecular marker that provides superior prognostic information compared with traditional histopathological features and guides the treatment strategy. This study aimed is to determine the frequency of dMMR in patients with uterine adenocarcinoma and evaluate its relationship with clinicopathological and prognostic parameters.

Materials and Methods: This retrospective study analyzed 50 patients diagnosed with uterine adenocarcinoma/endometrioid carcinoma between January 2019 and November 2024. Immunohistochemical evaluation was performed using antibodies against MSH1, MSH6, PMS2, and MLH1 proteins. The patients were restaged according to the Gynecology and Obstetrics International Federation of FIGO 2023 staging system.

Results: Loss of mismatch repair (MMR) protein expression was observed in 20 patients (40%). The mean age of the participants was 59.1±8.5 years old. Immunohistochemical analysis revealed MSH2 loss in three patients (6%), MSH6 loss in four patients (8%), MLH1 loss in 13 patients (26%), and PMS2 loss in 17 patients (34%). Patients with dMMR were diagnosed at a younger age, had higher rates of lymphovascular invasion, and had a greater risk of metastasis (p:0,18; p:0,32; p:0,40 respectively).

Conclusion: dMMR provides valuable prognostic information for endometrial cancer. Molecular classification is expected to play a pivotal role in guiding adjuvant treatment approaches and developing personalized therapeutic strategies in the future.

Keywords: Endometrial cancer, microsatellite instability, Immunohistochemistry

ÖZ

Amaç: *Endometriyal kanser, dünya genelinde en sık görülen jinekolojik malignitedir. Cancer Genome Atlas (TGCA) moleküler sınıflandırması, mutasyon yükü ve kopya sayı değişikliklerine göre dört farklı moleküler alt tip tanımlamıştır. MMR protein ekspresyon kaybı (dMMR), geleneksel histopatolojik özelliklere kıyasla üstün prognostik bilgi sağlayan ve tedavi stratejilerini yönlendiren önemli moleküler belirteçlerden biridir. Bu çalışmanın amacı uterin adenokarsinomlu hastalarda dMMR sıklığını belirlemek ve bunun klinikopatolojik ve prognostik parametrelerle ilişkisini değerlendirmektir.*

Gereç ve Yöntem: *Bu retrospektif çalışmada, Ocak 2019 ile Kasım 2024 tarihleri arasında uterin adenokarsinom/endometrioid karsinom tanısı alan elli hasta analiz edildi. İmmünohistokimyasal değerlendirme MSH1, MSH6, PMS2 ve MLH1 antikorları kullanılarak yapıldı. Hastalar, FIGO 2023 evreleme sistemine göre yeniden evrelendirildi.*

Corresponding author: Ahmet Beyazıt
Hatay Mustafa Kemal University Department of Obstetrics and Gynecology Hatay/ Türkiye
E-mail: *beyazitahmet@hotmail.com*
Application date: 23.07.2025 Accepted: 21.10.2025

Bulgular: *Yirmi hastada (%40) uyuşmayan onarım (MMR) proteini ekspresyonunda kayıp gözlendi. Çalışma grubunun ortalama yaşı 59,1±8,5 yıldı. İmmünohistokimyasal analiz, 3 hastada (%6) MSH2 kaybı, 4 hastada (%8) MSH6 kaybı, 13 hastada (%26) MLH1 kaybı ve 17 hastada (%34) PMS2 kaybı olduğunu gösterdi. MSI pozitifliği olan hastalar daha genç yaşta tanı aldı, daha yüksek lenfovasküler invazyon oranlarına sahipti ve lenf nodu metastazı açısından daha yüksek risk taşıdı (sırasıyla p:0,18; p:0,32; p:0,40).*

Sonuç: *dMMR değerlendirmesi, endometrial kanserde değerli prognostik bilgi sağlar. Moleküler sınıflandırmanın, gelecekte adjuvan tedavi yaklaşımlarını yönlendirmede ve kişiye özel tedavi stratejileri geliştirmede kilit bir rol oynaması beklenmektedir.*

Anahtar Sözcükler: *Endometriyal kanser, mikrosatellit instabilitesi, İmmünohistokimya*

INTRODUCTION

Endometrial cancer is the most common gynecological cancer worldwide, and its incidence and mortality rates have increased in recent years (1). Traditionally, endometrial cancer classification has been based on histopathological characteristics and clinical staging systems (2). However, the advent of molecular characterization has revolutionized our understanding of this heterogeneous disease.

In 2013, The Cancer Genome Atlas (TCGA) Research Network conducted a comprehensive molecular analysis of endometrial carcinomas, leading to the identification of four distinct molecular subtypes: POLE ultramutated, microsatellite instability hypermutated (MSI-H), low copy number, and high copy number(3). This molecular classification has demonstrated superior prognostic value compared to traditional histopathological grading and has significant implications for treatment decision making (2).

Microsatellite instability (MSI) occurs due to defects in the DNA mismatch repair (MMR) system, which is responsible for correcting errors that occur during DNA replication (4). The MMR system consists of several key proteins, including MLH1, MSH2, MSH6, and PMS2 (4). Loss of function of any of these proteins can lead to the accumulation of mutations in microsatellite sequences throughout the genome, resulting in MSI (5). When we assess protein loss using immunohistochemical techniques, we refer to this condition as deficient mismatch repair (dMMR)

The clinical significance of dMMR in endometrial cancer extends beyond its prognostic implications. Tumors with dMMR, which largely correspond to MSI-high tumors, have been shown to be particularly responsive to immune checkpoint inhibitors, such as pembrolizumab, which has received FDA approval for the treatment of MSI-high/dMMR solid tumors, regardless of tissue origin (6). This has made dMMR testing an essential component of the diagnostic workup for patients with endometrial cancer.

The current study aimed to evaluate the frequency of dMMR in patients with uterine adenocarcinoma treated at our institution and examine its relationship with various clinicopathological parameters. By utilizing the updated FIGO 2023 staging system and standardized immunohistochemical protocols, we aimed to contribute valuable data to the growing body of literature on the molecular characterization of endometrial cancer.

MATERIALS and METHODS

This retrospective cohort study was conducted at the Hatay Mustafa Kemal University Research Hospital in Turkey. The study protocol was approved by the institutional ethics committee (30.10.2024, decision number 26) and conducted in accordance with the principles of the Declaration of Helsinki.

Patient data were retrieved from the hospital database system for the period from January 1, 2019, to November 1, 2024. All patients who underwent hysterectomy during the study period were initially screened. Patients, with a pathological diagnosis of “uterine adenocarcinoma” or “endometrioid carcinoma” were included.

The inclusion criteria were as follows: (1) histopathologically confirmed diagnosis of uterine adenocarcinoma or endometrioid carcinoma; (2) availability of adequate paraffin-embedded tissue blocks in the pathology archive; (3) absence of significant necrosis, autolysis, or fixation artifacts in the tissue samples; and (4) complete clinical and pathological data.

The exclusion criteria were as follows: (1) patients who received neoadjuvant therapy prior to surgical intervention, (2) inadequate tissue samples for immunohistochemical analysis, and (3) incomplete clinical or pathological data.

Histopathological Evaluation and Staging

All patients were re-evaluated and restaged according to the Gynecology and Obstetrics National Federation of the FIGO of 2023 staging system. Histological grading was performed according to standard criteria, with grade 1 and 2 endometrioid cancers classified as non-aggressive and grade 3 cancers classified as aggressive histological types.

Immunohistochemical Analysis

Formalin-fixed, paraffin-embedded tissue blocks were sectioned to a thickness of 3 µm mounted on poly L-lysine-coated slides. The sections were deparaffinized in xylene and rehydrated using a graded alcohol series.

Immunohistochemical staining was performed using a DAKO Autostainer Link 48 (Agilent Technologies, Santa Clara, CA, USA). Primary antibodies against MSH2 (Dako/Agilent, Clone: FE11, Cat No:IR00561, ready to use), MSH6 (Dako/Agilent, Clone: Ep49, Cat No:IR08661, ready to use), PMS2 (Dako/Agilent, Clone: Ep51, Cat No:IR00761, ready to use), and MLH1 (Dako/Agilent, Clone: Es05, Cat No:IR07961, ready to use) were used for immunost. Counterstaining was performed using hematoxylin.

Immunohistochemical evaluation was based on the nuclear staining patterns in the tumor cells (Figure-1,2). dMMR status was determined using a binary scoring system: “no nuclear expression loss (0)” and “nuclear expression loss present (1)”. Benign endometrial gland tissue was used as the internal positive control. All staining results were evaluated by researchers blinded to the clinical information of the patients.

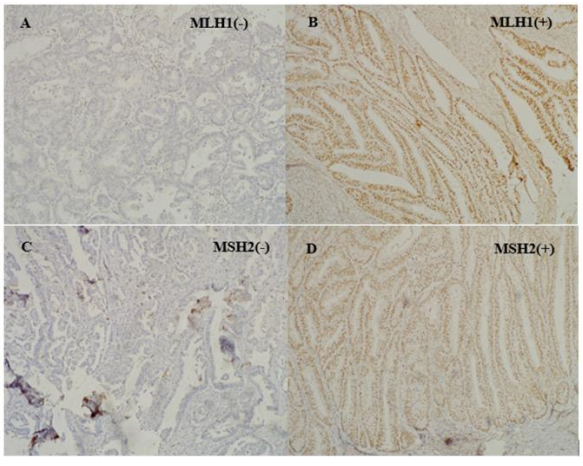

Figure-1. Immunohistochemical staining patterns of dMMR cases: A) MLH1 (-) B) MLH1 (+) C) MSH2(-) D) MSH2 (+)

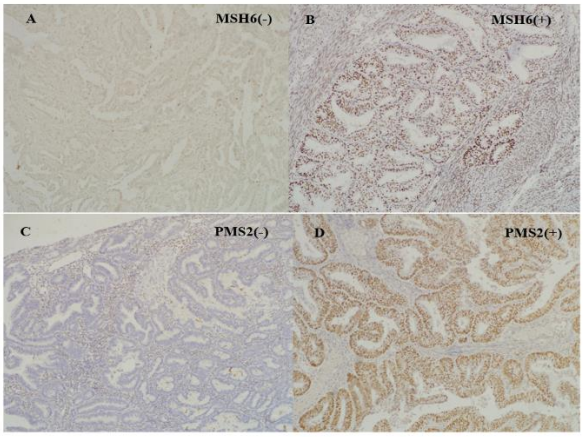

Figure-2. Immunohistochemical staining patterns of dMMR cases: A) MSH6 (-) B) MSH6 (+) C) PMS2 (-) D) PMS2 (+)

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27.0 (Armonk, NY, IBM Corp). Continuous variables are presented as mean± standard deviation if normally distributed, and as median (interquartile range) if not normally distributed. Categorical variables were summarized as numbers and percentages. Normality was assessed using the Shapiro-Wilk test.

For categorical variables, associations between groups were examined using the chi-squared test (Pearsons’s chi-square, likelihood ratio chi-square, Fisher’s exact test, or Fisher-Freeman-Halton exact test, depending on expected cell counts). When insufficient data were available for analysis, only the restrictive measures were provided. A two-sided p-value of $p<0.05$ was considered statistically significant. No formal correction for multiple comparisons was applied.

RESULTS

Fifty patients with endometrioid carcinoma who underwent hysterectomy were included in this study. The mean age of the study population was 59.1± 8.5 years (range:30-78). Overall, dMMR was observed in 20 patients (40%), whereas 30 patients (60%) showed no MMR loss. The mean age of the patients with MMR loss was 57.1±8.53 years, compared to 60.37±8.33 years in the group with MMR loss.

According to FIGO 2023 staging system, the distribution of patients in stage 1 was (74% n = 37). Most patients were diagnosed at an early stage (Table-1).

Regarding myometrial invasion depth, half of the patients (n = 25) had invasion of less than ½ of the myometrium. Lymph node metastasis was detected in seven patients (14%); additionally, three patients had distant metastasis in the pelvic region (Table-1).

Immunohistochemical analysis revealed the following MMR protein losses: MSH2 loss in 3 patients (6%), MSH6 loss in 4 patients (8%), MLH1 loss in 13 patients (26%), and PMS2 loss in 17 patients (34%) (Table-1)

Tables-2 and 3 show the expression of MSI proteins and their clinicopathological correlations. Although the difference was not statistically significant, patients with dMMR were diagnosed at a younger age than those with proficient MMR (pMMR) (p: 0,185). Furthermore, the incidence of lymphovasculer invasion and the risk of lymph node metastasis were higher in dMMR cohort (p:0,32 and p:0,40 respectively)

Table-1. Clinicopathological Characteristics of Patients

Variables		**n (%)**
Stage	Stage 1	37 (74)
	Stage 2	4 (8)
	Stage 3	7 (14)
	Stage 4	2 (4)
MSH2	Present	47 (94)
	Loss	3 (6)
MSH6	Present	46 (92)
	Loss	4 (8)
MLH1	Present	37 (74)
	Loss	13 (26)
PMS2	Present	33 (66)
	Loss	17 (34)
Tumor size	<2.5 cm	9 (18)
	≥ 2.5 cm	41 (82)
Grade	Grade 1	13 (26)
	Grade 2	31 (62)
	Grade 3	6 (12)
Distant Metastasis	No	47 (94)
	Yes	3(6)
Myometrial Invasion	<1/2	27 (54)
	≥ 1/2	23 (46)
LVSI*	No	35 (70)
	Yes	15 (30)
Lymph node metastasis	No	43 (86)
	Yes	7 (14)
Aggressive tumor	No	44 (88)
	Yes	6 (12)

*Lymphovascular space invasion

Table-2. Expressions of MSH2 and MSH6 proteins and their clinicopathological correlations

Variables		MSH2			MSH6		
		Present (n=47)	Loss (n=3)	p	Present (n=46)	Loss (n=4)	p
Age	Median (IQR)	60 (55-65)	55 (52-58)	!	60 (55-65)	55 (52-58)	!
Tumor Size	<2.5	7 (14.9)	2 (66.7)	0.08†	6 (13)	3 (75)	0.016†
	≥2.5	40 (85.1)	1 (33.3)		40 (87)	1 (25)	
Myometrial invasion	<1/2	25 (53.2)	2 (66.7)	1.00†	24 (52.2)	3 (75)	0.614†
	≥1/2	22 (46.8)	1 (33.3)		22 (47.8)	1 (25)	
Lymph Node Metastasis	No	41 (87.2)	2 (66.7)	0.37	40 (87)	3 (75)	0.414†
	Yes	6 (12.8)	1 (33.3)		6 (13)	1 (25)	
LVSİ*	No	33 (70.2)	2 (66.7)	1.00†	32 (69.6)	3 (75)	1.00†
	Yes	14 (29.8)	1 (33.3)		14 (30.4)	1 (25)	
Grade	Grade 1	12 (25.5)	1 (33.3)	0.323‡	12 (26.1)	1 (25)	0.752‡
	Grade 2	30 (63.8)	1 (33.3)		29 (63)	2 (50)	
	Grade 3	5 (10.6)	1 (33.3)		5 (10.9)	1 (25)	
Metastasis	No	45 (95.7)	2 (66.7)	0.173†	44 (95.7)	3 (75)	0.226†
	Yes	2 (4.3)	1 (33.3)		2 (4.3)	1 (25)	
Aggressive Tumor	No	42 (89.4)	2 (66.7)	0.324†	41 (89.1)	3 (75)	0.411†
	Yes	5 (10.6)	1 (33.3)		5 (10.9)	1 (25)	

*Lymphovascular space invasion; !: not computable value; †: Fisher's exact; ‡: Fisher-Freeman-Halton Exact test

Table-3. Expressions of MLH1 and PMS2 proteins and their clinicopathological correlations

Variables		MLH1			PMS2		
		Present (n=47)	Loss (n=3)	p	Present (n=46)	Loss (n=4)	p
Age	Median (IQR)	60 (55-65)	58 (56-63)	0,90	60 (55-65)	58 (56-63)	0,87
Tumor Size	<2.5	8 (21.6)	1 (7.7)	0.41†	7 (21.2)	2 (11.8)	0.69†
	≥2.5	29 (78.4)	12 (92.3)		26 (78.8)	15 (88.2)	
Myometrial invasion	<1/2	20 (54.1)	7 (53.8)	1.00‡	17 (51.5)	10 (58.8)	0.84‡
	≥1/2	17 (45.9)	6 (46.2)		16 (48.5)	7 (41.2)	
Lymph Node Metastasis	No	33 (89.2)	10 (76.9)	0.35†	29 (87.9)	14 (82.4)	0.67†
	Yes	4 (10.8)	3 (23.1)		4 (12.1)	3 (17.6)	
LVSİ*	No	26 (70.3)	9 (69.2)	1.00†	22 (66.7)	13 (76.5)	0.69‡
	Yes	11 (29.7)	4 (30.8)		11 (33.3)	4 (23.5)	
Grade	Grade 1	11 (29.7)	2 (15.4)	0.56§	9 (27.3)	4 (23.5)	0.95§
	Grade 2	22 (59.5)	9 (69.2)		20 (60.6)	11 (64.7)	
	Grade 3	4 (10.8)	2 (15.4)		4 (12.1)	2 (11.8)	
Metastasis	No	35 (94.6)	12 (92.3)	1.00†	31 (93.9)	16 (94.1)	1.00†
	Yes	2 (5.4)	1 (7.7)		2 (6.1)	1 (5.9)	
Aggressive Tumor	No	33 (89.2)	11 (84.6)	0.64†	29 (87.9)	15 (88.2)	1.00†
	Yes	4 (10.8)	2 (15.4)		4 (12.1)	2 (11.8)	

*Lymphovascular space invasion; !: not computable value; †: Fisher's exact; ‡:Yates Correction, §:Likelihood ratio

DISCUSSION

This study provides valuable insights into the frequency and clinical significance of dMMR in patients with uterine adenocarcinoma. Our findings demonstrate that MMR protein loss occurs in 40% of endometrioid carcinomas, which is consistent with previous reports showing dMMR frequencies ranging from 25% to 35% in endometrial cancers (7, 8).

Molecular classification of endometrial cancer has emerged as a powerful tool for risk stratification and treatment planning in recent years. The TCGA classification system has identified MSI-high/dMMR tumors as distinct molecular subtypes with unique biological characteristics and clinical behavior. These tumors typically exhibit a high mutation burden, immune infiltration, and favorable response to immunotherapy (3).

Our analysis revealed that PMS2 was the most frequently lost MMR protein (34%), followed by MLH1 (26%), MSH6 (8%), and MSH2 (6%). This pattern is consistent with the known biology of MMR protein function, where MLH1 and PMS2 form a heterodimer, and the loss of one protein typically results in the loss of its partner. Similarly, MSH2 and MSH6 form functional heterodimers (9).

The clinical significance of our findings extends beyond the mere frequency determination. We observed that patients with dMMR were diagnosed at a younger age, had higher rates of lymphovascular invasion, and posed a greater risk for metastatic disease. These findings have important implications for treatment planning and patient counseling.

A younger age at diagnosis in dMMR patients may be related to an underlying genetic predisposition, particularly in cases associated with Lynch syndrome (7). Lynch syndrome is a hereditary cancer predisposition syndrome caused by germline mutations in MMR genes and accounts for approximately 2-5% of all endometrial cancers. Patients with Lynch Syndrome typically develop cancer at a younger age and may benefit from enhanced surveillance protocols (9).

The association between dMMR and lymphovascular invasion is particularly noteworthy because LVSI is a well-established risk factor for lymph node metastasis and recurrence in endometrial cancer (10). In a study by Keverline et al., it was demonstrated that patients with endomerial cancer exhibiting deficient mismatch repair (dMMR) have an elevated risk of lymphovascular space invsion, with a relative risk of 1.63, compared to those with proficient mismatch repair (pMMR)(11). FIGO 2023 staging system demonstrated that LVSI is a significant prognostic factor, and our findings suggest that dMMR may provide additional information in this context.

The implications of dMMR testing extend beyond prognostic stratification to therapeutic decision-making. The FDA approval of pembrolizumab for MSI-high/dMMR solid tumors has made dMMR testing a critical component of the diagnostic work-up for patients with endometrial (12). MSI-high/dMMR tumors have shown remarkable response rates to immune checkpoint inhibitors, with objective response rates exceeding 50% in some studies (13).

Furthermore, molecular classification has implications for adjuvant therapeutic decisions. The ESMO-ESGO-ESTRO consensus guidelines now recommend molecular classification for all endometrial cancers, and treatment recommendations are increasingly tailored based on molecular subtypes (12). MSI-high/dMMR tumors may benefit from different adjuvant therapy approaches than with other molecular subtypes (14).

The standardized immunohistochemical approach used in this study provides a practical and cost-efficient method for dMMR testing. Although next-generation sequencing and PCR-based methods are available for MSI testing, immunohistochemistry remains the most widely used technique in clinical practices (15). The binary scoring system used in our study provides clear and reproducible results that can be easily implemented in routine pathology practice (16).

Our study had several limitations. The retrospective design and single-center nature of this study may have limited our ability to detect statistically significant associations between some variables. Additionally, long-term follow-up data were not available to assess the effect of dMMR status on the survival outcomes.

Future studies should focus on larger, multicenter cohorts with longer follow-up periods to better understand the prognostic implications of dMMR status in endometrial cancer. Additionally, integrating molecular classification with other emerging biomarkers, such as tumor-infiltrating lymphocytes and PD-L1 expression, may provide more refined prognostic and predictive information.

CONCLUSION

This study demonstrated that microsatellite instability is present in 40% of uterine adenocarcinomas and is associated with distinct

clinicopathological features. Patients with dMMR tumors were diagnosed at a younger age, had higher rates of lymphovascular invasion, and were at greater risk for metastatic disease. These findings support the clinical utility of dMMR testing in endometrial cancer and highlight the importance of molecular classification for treatment planning.

Our findings contribute to the growing body of evidence supporting the clinical utility of molecular classification of endometrial cancer. As we move toward an era of precision oncology, molecular characterization will become increasingly important for optimizing treatment strategies and improving patient outcomes in this field.

Acknowledgement: This study was supported by the Mustafa Kemal University Scientific Research Projects Unit under Project Number 25.TU.001

Conflict of Interest: Authors declared no conflict of interest

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