International Medicine
www.theinternationalmedicine.org

Research Article

Section: Pathology

Evaluation of p53 and Ki-67 Expression in Cervical Intraepithelial Neoplasia (CIN) and Invasive Cervical Cancer: A Prospective Observational Study

Dr. Sunaina Singh[*1], Dr. Mahendra Singh[2], Dr. Chayanika Kala[3] & Dr. Lubna Khan[4]

[1,2,3,4]Department of Pathology, GSVM Medical College, Kanpur, India

ARTICLE INFO

Article History:

Received: 15-07-2025

Accepted: 19-08-2025

Keywords:

Cervical cancer

p53

Ki-67

Immunohistochemistry

CIN

***Corresponding author:**
Dr. Sunaina Singh
Department of Pathology, GSVM Medical College, Kanpur, India

ABSTRACT

Introduction: Cervical cancer is the fourth most common malignancy among women worldwide, predominantly affecting those in low- and middle-income countries due to inadequate screening and preventive measures. High-risk Human Papillomavirus (HPV) infection, particularly types 16 and 18, is a critical etiological factor in cervical carcinogenesis. Molecular markers like p53 and Ki-67 have emerged as valuable indicators of disease progression and prognosis, given their roles in cell cycle regulation and proliferation. **Aim and Objective:** To evaluate the expression of p53 and Ki-67 in cervical intraepithelial neoplasia (CIN) and invasive cervical carcinoma, and to correlate their expression with histopathological grades. **Materials and Methods:** This 15-month prospective observational study at GSVM Medical College, Kanpur, and J.K. Cancer Institute included histologically confirmed cases of CIN (I, II, III) and invasive cervical carcinoma in women aged 18 and above. Immunohistochemistry (IHC) assessed p53 and Ki-67 expression, with chi-square tests and p-values used to analyze associations between biomarker expression and histological grading. **Results:** The study included 100 participants with a mean age of 41.66 ± 12.00 years. Squamous cell carcinoma (SCC) was the most common (40%), followed by CIN3 (30%), CIN2 (20%), and CIN1 (10%). High Ki-67 expression was mainly seen in SCC and CIN3 ($p < 0.0001$). Aberrant p53 expression was rare (1.6% in SCC), while mild reduction was common in CIN3 (19.4%). **Conclusion:** The findings suggest that high Ki-67 expression and reduced p53 levels are associated with greater histological severity in cervical lesions. These markers hold significant prognostic value and could enhance early diagnosis and targeted therapeutic strategies in cervical cancer.

INTRODUCTION

Cervical cancer continues to pose a substantial threat to women's health worldwide, ranking as the fourth most frequently diagnosed cancer among women globally. Despite notable progress in prevention and early detection, it remains a major cause of death, particularly in low- and middle-income countries (LMICs), where healthcare infrastructure is limited. In 2020, the World Health Organization (WHO) estimated approximately 604,000 new cervical cancer cases and 342,000 related deaths globally [1]. The overwhelming majority of these deaths-nearly 90%-occurred in LMICs, attributed to inadequate access to screening, vaccination, and timely treatment services [2]. High-income countries have managed to significantly reduce cervical cancer incidence by implementing organized screening programs, such as the Papanicolaou (Pap) smear test, and adopting widespread human papillomavirus (HPV) vaccination. However, in countries like India and those in sub-Saharan Africa, where awareness is low and healthcare services are often inaccessible or underutilized, cervical cancer remains a critical public health issue [3]. Research has shown that over 80% of cervical cancers can be prevented through the early detection and treatment of precancerous lesions, underscoring the largely avoidable nature of this disease [4].

Cervical cancer typically evolves gradually, often over a span of several years, beginning with HPV infection and progressing through defined stages of cervical intraepithelial neoplasia (CIN). These precursor lesions are categorized histologically into CIN I (mild

dysplasia), CIN II (moderate dysplasia), and CIN III (severe dysplasia or carcinoma in situ), based on the extent of epithelial involvement. While not all precancerous changes progress to invasive cancer, identifying high-risk lesions using molecular markers such as p53 and Ḳi-67 can aid in clinical risk assessment and management [5]. In an effort to eliminate cervical cancer by 2030, WHO has outlined a strategic approach aiming for 90% coverage of HPV vaccination, 70% screening coverage, and 90% access to treatment for both precancerous and invasive cases. This strategy highlights the urgency of strengthening public health programs that focus on immunization and early detection, particularly in underserved and high-burden regions [6].

Persistent infection with oncogenic HPV types is recognized as the primary cause of cervical cancer. HPV, a common sexually transmitted infection, is prevalent among sexually active individuals, with most infections resolving spontaneously due to immune clearance. However, persistent infection with high-risk types-especially HPV 16 and HPV 18-is responsible for more than 70% of cervical cancers worldwide [7]. Several behavioral and biological factors increase the likelihood of disease progression following HPV infection. These include having multiple sexual partners, early sexual debut, and immunosuppression, particularly among women living with HIV, who have a six-fold increased risk of developing cervical cancer due to impaired immune surveillance. Additional contributing factors include cigarette smoking, which introduces carcinogens that damage DNA and hinder immune response, and long-term use of oral contraceptives, which may influence the cervical epithelium through hormonal changes. Socioeconomic conditions-such as poverty, limited education, and restricted access to health services-further compound the risk and are linked with higher rates of both incidence and mortality. While HPV infection is necessary for carcinogenesis, it is not sufficient by itself. The progression to malignancy involves a complex interplay of viral persistence, host immune response, and other cofactors that influence epithelial transformation [8].

Preventive efforts therefore emphasize primary prevention through HPV vaccination and secondary prevention through routine screening. Vaccination campaigns are being expanded to include not only adolescent girls but also boys and adult women at higher risk, in order to strengthen herd immunity and reduce transmission. For low-resource settings, WHO advocates for cost-effective screening approaches, including HPV DNA testing and visual inspection with acetic acid (VIA), which offer practical alternatives to cytology-based screening [9].

At the molecular level, cervical cancer pathogenesis involves several steps beginning with infection of the cervical epithelium by HPV. If the immune system fails to clear the virus, it may integrate its genetic material into the host genome, a process that disrupts normal cell function [10]. The key viral oncoproteins E6 and E7 facilitate carcinogenesis by targeting tumor suppressor pathways. E6 accelerates degradation of p53, a protein essential for apoptosis and genomic stability, while E7 binds and inhibits the retinoblastoma protein (Rb), thereby disabling cell cycle regulation. This leads to sustained cellular proliferation, impaired DNA repair, and cumulative mutations. Subsequent changes, including abnormal DNA methylation and chromatin remodeling, further promote transformation to malignancy. The detection of biomarkers such as p16, Ki-67, and p53 has enhanced our ability to stratify lesions based on progression risk and improve diagnostic accuracy [11].

Histologically, cervical cancer evolves from CIN to invasive carcinoma, most commonly squamous cell carcinoma, which constitutes about 80–90% of cases. Adenocarcinoma is less common but tends to behave more aggressively and often evades early detection. Biomarker studies have played a pivotal role in refining the diagnosis and monitoring of cervical lesions. The tumor suppressor protein p53 is generally expressed at low levels in normal cervical tissue but may be either overexpressed or absent in high-grade dysplasia and invasive cancers due to E6-mediated degradation. Immunohistochemical (IHC) staining of p53 serves as a useful diagnostic tool in detecting neoplastic changes. Ki-67, a nuclear protein expressed during cell proliferation, also demonstrates a stepwise increase in expression from normal tissue to high-grade CIN and invasive cancer, correlating with disease severity. High Ki-67 expression is associated with aggressive disease and poorer prognosis. The dual staining of p16 and Ki-67 has further improved the sensitivity and specificity of screening protocols and is particularly useful in identifying lesions requiring immediate attention [12-14].

The study aims to evaluate the expression of p53 and Ki-67 in preinvasive and invasive cervical lesions. Objectives include histological grading of CIN and carcinoma cervix, assessing p53 and Ki-67 expression in CIN I-III and carcinoma, and analyzing correlations between lesion grades and biomarker expression to understand their diagnostic and prognostic significance in cervical neoplasia.

MATERIALS AND METHODS

This prospective observational non-randomized study was conducted over 15 months (July 2023 to October 2024) in the Department of Pathology, GSVM Medical College, Kanpur, in collaboration with the Department of Obstetrics & Gynecology and the J.K. Cancer Institute. It aimed to evaluate p53 and Ki-67 expression in histologically confirmed cases of CIN (I, II, III) and invasive cervical carcinoma in women aged 18 years or older who provided informed consent. Patients under 18, unwilling to consent, undergoing prior chemo/radiotherapy, or with recurrent cancer were excluded. Ethical approval was obtained, and the study complied with the Declaration of Helsinki.

RESULTS

The study participants had a mean age of 41.66 ± 12.00 years. Regarding parity, 12% were nulliparous (P0), 40% had one childbirth (P1), 33% had two (P2), and 15% had three (P3),indicating that the majority had at least one previous childbirth, with P1 being the most common parity group.

Table 1: Distribution of Histological Diagnoses

	Histological Diagnosis	Frequency [n]	Percentage [%]
0	SCC	40	40.0
1	CIN3	30	30.0
2	CIN2	20	20.0
3	CIN1	10	10.0
	P-Value (Uniform Distribution)		0.0002
	P-Value (Pre-Invasive VS Invasive)		0.0455

Among the histological diagnoses, SCC was most common (40%), followed by CIN3 (30%), CIN2 (20%), and CIN1 (10%). The distribution significantly differed from uniformity (p = 0.0002), and the difference between pre-invasive and invasive lesions was statistically significant (p = 0.0455), indicating a higher prevalence of invasive carcinoma.

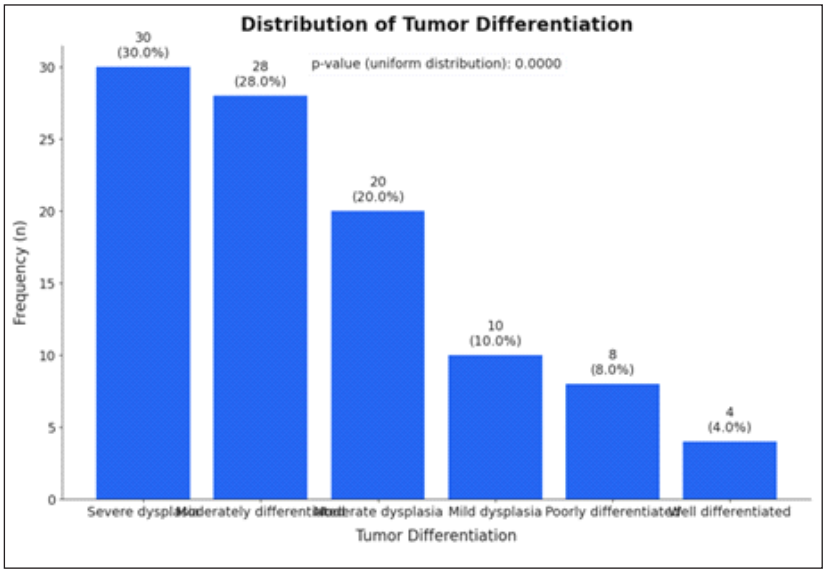

Figure 1: Distribution of Tumor Differentiation

Severe dysplasia (30%) and moderately differentiated SCC (28%) were the most frequent lesion types, followed by moderate (20%) and mild dysplasia (10%). Poorly and well-differentiated SCC accounted for 8% and 4%, respectively. The non-uniform distribution was statistically significant (p = 0.0000), indicating a skewed pattern of lesion differentiation.

Most cases showed high Ki-67 expression with mildly (22.6%) or moderately (17.7%) reduced p53. Severe reduction of p53 with high Ki-67 was seen in 9.7%, while 19.4% had normal p53 with mild Ki-67. Aberrant and normal p53 with high Ki-67 were rare (1.6% each), and some combinations showed no cases.

Table 2: Association between p53 Expression and Ki-67 Expression

	P53 Expression	Ki-67 Expression	Frequency [n]	Percentage [%]
0	Aberrant	High	1	1.6
1	Mildly Reduced	High	14	22.6
2	Moderately Reduced	High	11	17.7
3	Normal	High	1	1.6
4	Severely Reduced	High	6	9.7
5	Aberrant	Mild	0	0.0
6	Mildly Reduced	Mild	0	0.0
7	Moderately Reduced	Mild	0	0.0
8	Normal	Mild	12	19.4

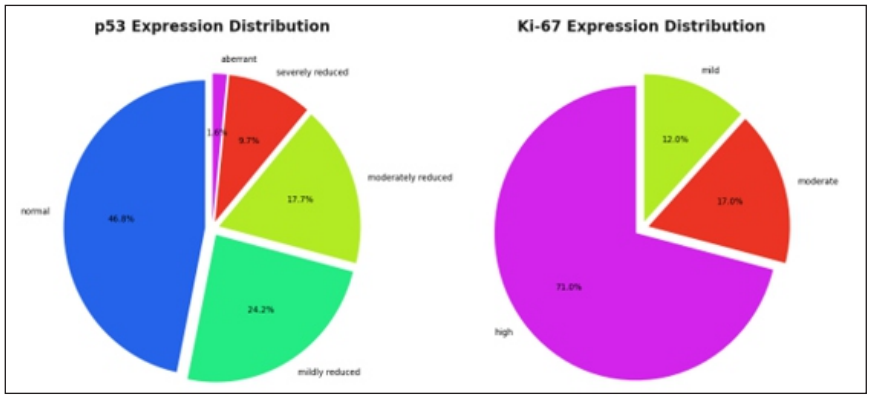

Figure 2: Distribution Patterns of p53 and Ki-67 Expression in Cervical Lesions

The pie charts show that most cases exhibited normal p53 expression (46.8%), followed by mildly (24.2%) and moderately (17.7%) reduced expression. High Ki-67 expression was predominant (71.0%), indicating high proliferative activity, while moderate and mild expressions accounted for 17.0% and 12.0% respectively, reflecting varied tumor proliferative potential.

Table 3: Association between Lesion Differentiation and p53 Expression

	Lesion Differentiation	P53 Expression	Frequency [n]	Percentage [%]
0	Mild Dysplasia	Aberrant	0	0.0
1	Mild Dysplasia	Mildly Reduced	1	1.6
2	Mild Dysplasia	Moderately Reduced	0	0.0
3	Moderate Dysplasia	Aberrant	0	0.0
4	Moderate Dysplasia	Mildly Reduced	2	3.2
5	Severe Dysplasia	Aberrant	0	0.0
6	Severe Dysplasia	Mildly Reduced	12	19.4
7	Poorly Differentiated SCC	Mildly Reduced	0	0.0
8	Poorly Differentiated SCC	Aberrant	1	1.6

The table shows that mildly reduced p53 expression was most frequent in severe dysplasia (19.4%) and moderate dysplasia (3.2%), with minimal expression in mild dysplasia (1.6%). Aberrant p53 expression was rare, noted only in 1.6% of poorly differentiated SCC. No aberrant expression was observed in dysplastic lesions, indicating expression patterns vary by lesion severity.

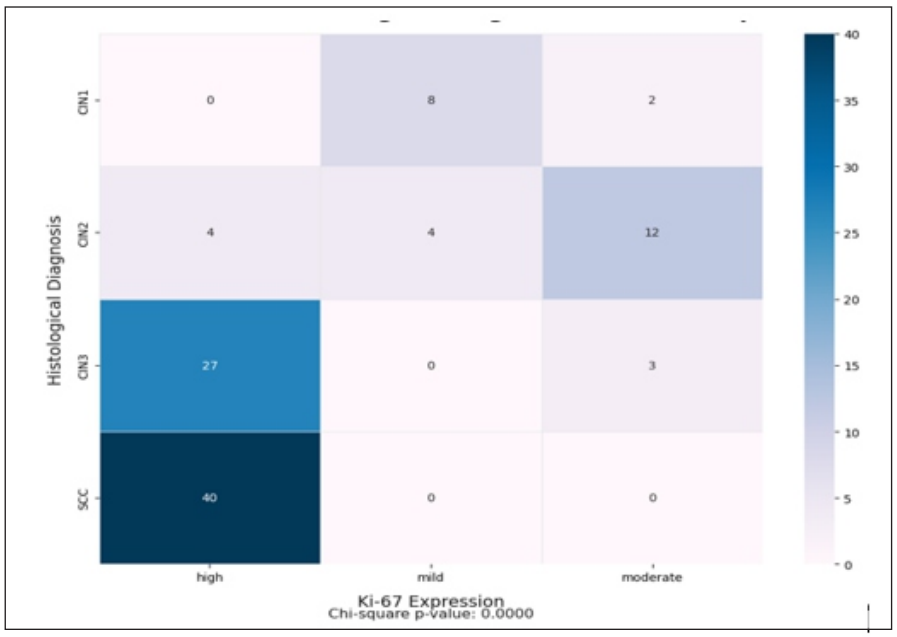

Figure 3: Association between Histological Diagnosis and Ki-67 Expression

High Ki-67 expression was predominant in SCC (40%) and CIN3 (27%), indicating increased proliferative activity in advanced lesions. Mild expression was mainly seen in CIN1 (8%) and CIN2 (4%). Moderate Ki-67 was rare, noted only in CIN1 (2%). No mild or moderate expression occurred in CIN3 or SCC, confirming Ki-67's association with lesion severity.

Table 4: Association between Histological Diagnosis and p53 Expression

	Histological Diagnosis	P53 Expression	Frequency [n]	Percentage [%]
0	Cin1	Aberrant	0	0.0
1	Cin2	Aberrant	0	0.0
2	Cin3	Aberrant	0	0.0
3	SCC	Aberrant	1	1.6
4	Cin1	Mildly Reduced	1	1.6
5	Cin2	Mildly Reduced	2	3.2
6	Cin3	Mildly Reduced	12	19.4
7	SCC	Mildly Reduced	0	0.0
8	Cin1	Moderately Reduced	0	0.0

The table shows that aberrant p53 expression was rare, seen only in 1.6% of SCC cases. Mildly reduced p53 was most frequent in CIN3 (19.4%), followed by CIN2 (3.2%) and CIN1 (1.6%). No p53 expression abnormalities were found in early CIN stages for aberrant or moderately reduced categories, indicating p53 alteration is more prominent in higher-grade lesions.

Table 5: Statistical Analyses

	Association	P-Value	Significance
0	p53 Expression Distribution	0.0008	Highly significant ($p < 0.001$)
1	Ki-67 Expression Distribution	0.0000	Highly significant ($p < 0.001$)
2	p53 Expression vs Ki-67 Expression	0.0000	Highly significant ($p < 0.001$)
3	Tumor Differentiation vs p53 Expression	0.0000	Highly significant ($p < 0.001$)
4	Histological Diagnosis vs Ki-67 Expression	0.0000	Highly significant ($p < 0.001$)
5	Histological Diagnosis vs p53 Expression	0.0000	Highly significant ($p < 0.001$)

The table reveals highly significant associations ($p < 0.001$) between various parameters. Both p53 and Ki-67 expression distributions were statistically significant. Strong associations were found between p53 and Ki-67 expression, tumor differentiation and p53, and between histological diagnosis with both p53 and Ki-67 expression, indicating their critical roles in characterizing cervical lesion behavior and progression.

Pathological Images

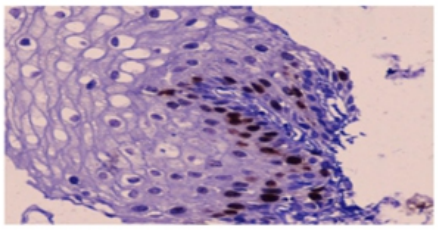

Fig 1. Expression of ki67 in preinvasive lesions of cervix (high power view, 40x)

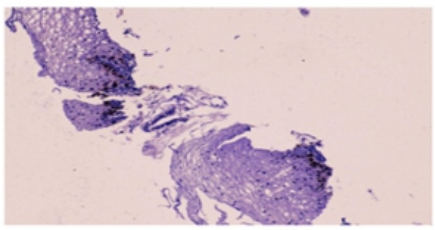

Fig 2. Expression of ki67 in preinvasive lesions of cervix (lowpower view, 10x)

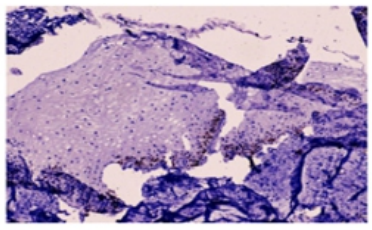

Fig 3. Expression of ki67 in preinvasive lesions of cervix (high power view, 40x)

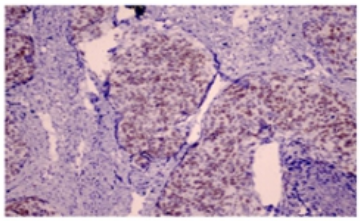

Fig 4. Expression of ki67 in carcinoma cervix (high power view, 40x)

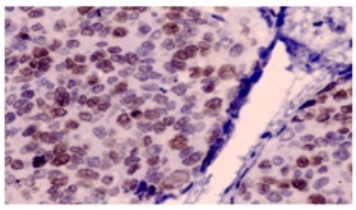

Fig 5. Expression of ki67 in carcinoma cervix (high power view, 40x)

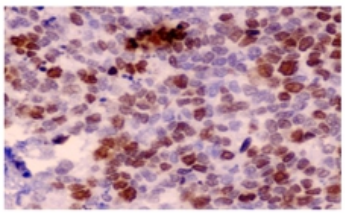

Fig 7.Expression of ki67 in carcinoma cervix (high power view, 40x)

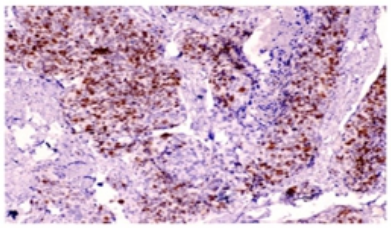

Fig 6. Expression of ki67 in carcinoma cervix (low power view, 10x)

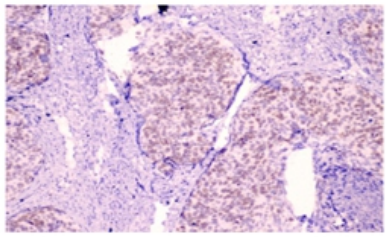

Fig 8. Expression of ki67 in carcinoma cervix (low power view, 10x)

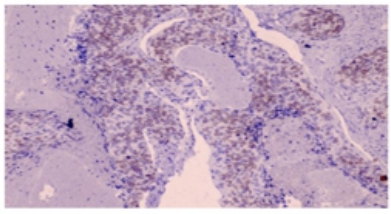

Fig 9. Expression of ki67 in carcinoma cervix (low power view, 10

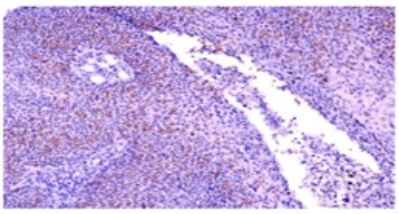

Fig 11. Expression of ki67 in carcinoma cervix (low power view, 10x)

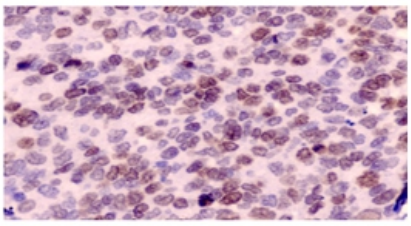

Fig 10.Expression of ki67 in carcinoma cervix (high power view, 4

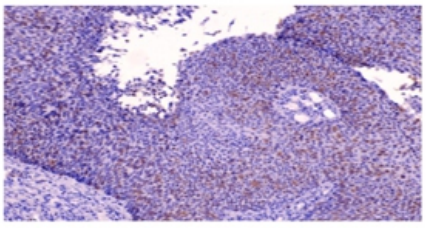

Fig 12.Expression of ki67 in carcinoma cervix (low power view, 10x)

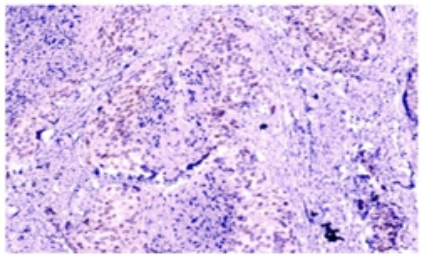

Fig 13. Expression of p53 in carcinoma cervix (low power view, 10x)

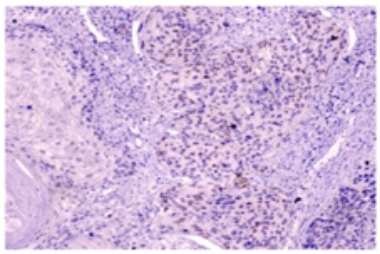

Fig 15. expression of p53 in carcinoma cervix (low power view, 10x)

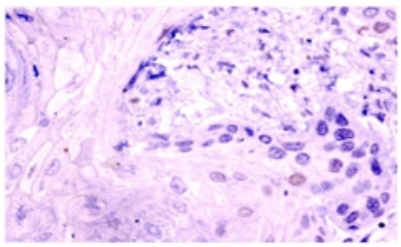

Fig 14. Expression of p53 in carcinoma cervix (low power view, 10x)

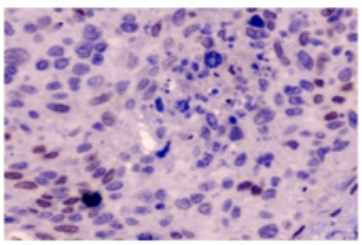

Fig 16. Expression of p53 in carcinoma cervix (high power view, 40x)

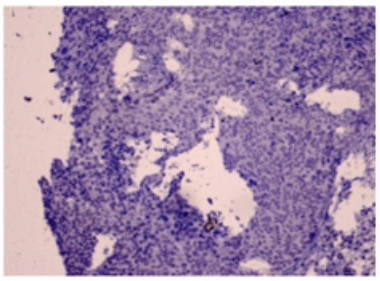

Fig 17. Expression of p53 in carcinoma cervix (low power view, 10x)

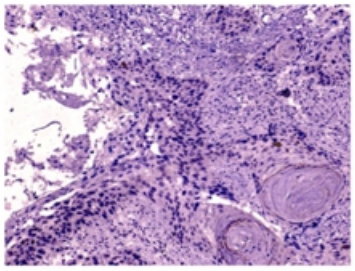

Fig 19. Expression of p53 in carcinoma cervix (low power view, 10x)

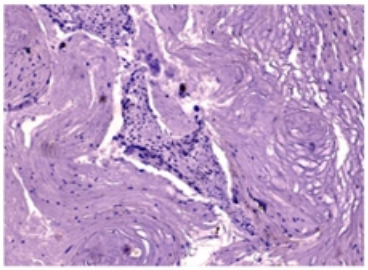

Fig 18. Expression of p53 in carcinoma cervix (low power view, 10x)

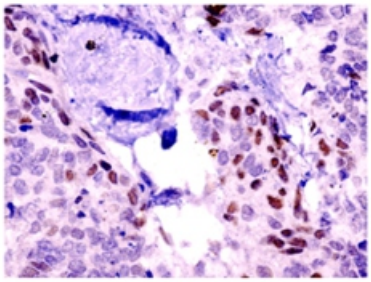

Fig 20. Expression of p53 in preinvasive lesions (high power view, 40x)

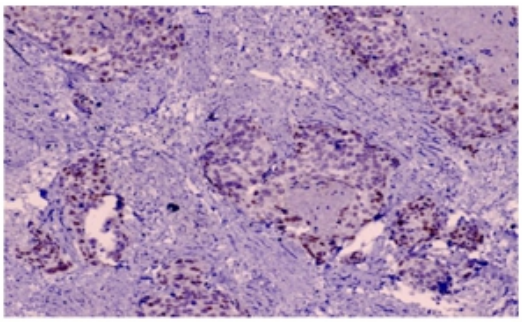

Fig 21. Expression of p53 in carcinoma cervix (low power view, 10x)

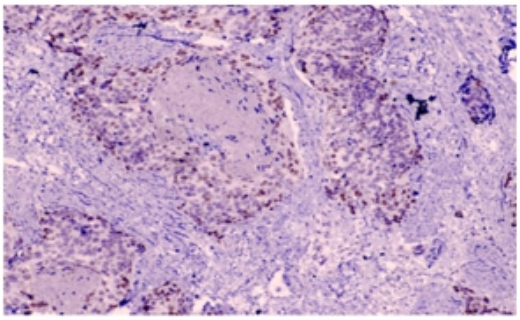

Fig 22. Expression of p53 in carcinoma cervix (low power view, 10x)

DISCUSSION

The study's demographic findings reveal a mean age of 41.66 ± 12.00 years, aligning with existing epidemiological data that show peak incidence of cervical intraepithelial neoplasia (CIN) and squamous cell carcinoma (SCC) in women in their late reproductive to perimenopausal years A comparable mean age of 41.7 years was also observed in a Portuguese multicenter study on CIN2/3 and invasive cervical cancer. Multiparity, especially P1 and P2, was common and reinforces its role as a significant risk factor for persistent HPV infection and cervical carcinogenesis. SCC predominance (40%) highlights delayed diagnoses, indicating the need for improved early screening strategies [15, 16].

The lesion distribution in this study-SCC (40%), CIN3 (30%), CIN2 (20%), and CIN1 (10%)-reflects the established model of cervical carcinogenesis, where progressive dysplasia culminates in invasive cancer. A Korean study similarly found that CIN2 and CIN3 commonly preceded SCC, supporting this continuum. The statistically significant non-uniform distribution ($p = 0.0002$) further validates the stepwise transformation of cervical neoplasia. International literature from China and Portugal also reports SCC as the predominant histological finding in biopsy-confirmed cases. Additionally, the 60:40

division between pre-invasive and invasive lesions is comparable to a retrospective Indonesian study, which noted that over 70% of women were diagnosed at an advanced stage, largely due to inadequate early screening practices. These findings underscore the urgent need for widespread cervical cancer screening programs [6, 15, 17].

Tumor differentiation is a critical prognostic marker in cervical cancer. In the present study, severe dysplasia (30%) and moderately differentiated tumors (28%) were most prevalent, with a statistically significant chi-square value ($p < 0.0001$), reflecting trends seen in global datasets that indicate poorly differentiated tumors, though less frequent, are associated with poorer outcomes. A cohort study demonstrated that p53 and Ki-67 expression levels were significantly correlated with tumor grade and recurrence, highlighting how histological differentiation mirrors underlying molecular changes. Similarly, a Korean study by Choi et al. found that high-grade dysplasia was strongly associated with increased risk of progression to invasive carcinoma, often accompanied by molecular disruptions such as p53 degradation and elevated Ki-67 proliferation indices. These observations underscore the clinical relevance of tumor differentiation and biomarker profiling in cervical pathology [18, 19].

This study observed null p53 expression in 38% of cases and aberrant expression in only 1%, suggesting that p53 inactivation likely results from HPV E6-mediated degradation rather than TP53 mutation. A statistically significant non-uniform distribution ($p = 0.0008$) affirms its prognostic relevance. Stoenescu et al. reported that reduced p53 expression correlates with higher tumor grades and advanced stages. High Ki-67 expression was detected in 71% of cases, with a significant p-value (< 0.0001), highlighting its role as a proliferation marker. Verma et al. and Choi et al. found that Ki-67 expression rises with tumor grade and dysplasia severity, respectively. A significant correlation between p53 loss and Ki-67 elevation ($p < 0.0001$) suggests enhanced proliferation due to impaired tumor suppression, consistent with cervical and other squamous carcinomas [19-22].

Multivariate models demonstrated that reduced p53 expression and elevated Ki-67 levels independently predict lesion severity and clinical prognosis. Comparable findings in cervical and prostate cancer studies highlight that the combined use of these biomarkers improves risk stratification and enhances prognostic accuracy, aiding in personalized patient management and therapeutic decision-making [23].

CONCLUSION

This study offers notable strengths, including a well-characterized patient cohort, comprehensive histopathological evaluation, and rigorous statistical analysis, providing valuable data on p53 and Ki-67 expression in cervical cancer and supporting their utility as diagnostic and prognostic biomarkers. However, limitations such as the absence of follow-up data, lack of HPV genotyping, potential selection bias, and its single-center nature may restrict generalizability. Future studies should incorporate prospective multi-center designs, HPV genotyping, and functional validation of biomarkers. Despite these constraints, the study significantly contributes to cervical cancer research and underscores the potential for biomarker-based stratification and personalized treatment approaches.

REFERENCES

1. Kim SI, Kim J-WJJoGO. Book Review: IARC handbooks of cancer prevention, volume 18: Cervical cancer screening. 2022;33(4):e65.
2. Thomsen LT, Kjær SKJBm. Human papillomavirus (HPV) testing for cervical cancer screening in a middle-income country: comment on a large real-world implementation study in China. 2021;19:1-3.
3. Vu M, Yu J, Awolude OA, Chuang LJCpic. Cervical cancer worldwide. 2018;42(5):457-65.
4. Simms KT, Steinberg J, Caruana M, Smith MA, Lew J-B, Soerjomataram I, et al. Impact of scaled up human papillomavirus vaccination and cervical screening and the potential for global elimination of cervical cancer in 181 countries, 2020–99: a modelling study. 2019;20(3):394-407.
5. Sun C, Mustafe AA, Liu B, Ma YJZdxxbYxbJoZUMs. Global disease burden of cervical cancer and the association of screening coverage with quality of disease management.1-7.
6. Agustiansyah P, Sanif R, Nurmaini SJBMJoB, Research T. WHO global strategy in eradication of cervical cancer. 2021;5(9):818-24.
7. Castellsagué XJGo. Natural history and epidemiology of HPV infection and cervical cancer. 2008;110(3):S4-S7.
8. Gadducci A, Barsotti C, Cosio S, Domenici L, Riccardo Genazzani AJGE. Smoking habit, immune suppression, oral contraceptive use, and hormone replacement therapy use and cervical carcinogenesis: a review of the literature. 2011;27(8):597-604.
9. Toliman P, Kaldor J, Tabrizi S, Vallely AJC. Innovative approaches to cervical cancer screening in low-and middle-income countries. 2018;21(3):235-8.
10. Ibeanu OAJCb, therapy. Molecular pathogenesis of cervical cancer. 2011;11(3):295-306.
11. van Bogaert L-JJJoGO. Cervical preneoplasia biomarkers: a conundrum for the community based gynecologic surgical pathologist. 2014;25(1):3-5.
12. Gustinucci D, Rossi PG, Cesarini E, Broccolini M, Bulletti S, Carlani A, et al. Use of cytology, E6/E7 mRNA, and p16INK4a–Ki-67 to define the management of human papillomavirus (HPV)–positive women in cervical cancer screening. 2016;145(1):35-45.
13. Ouh Y-T, Kim HY, Yi KW, Lee N-W, Kim H-J, Min K-JJD. Enhancing cervical cancer screening: review of

p16/Ki-67 dual staining as a promising triage strategy. 2024;14(4):451.

14. Yang X, Lu LJDr. Expression of HPV-16 E6 protein and p53 inactivation increases the uterine cervical cancer invasion. 2015;65(02):70-3.
15. Amaro-Filho SM, Golub JE, Nuovo GJ, Cunha CB, Levi JE, Villa LL, et al. A comparative analysis of clinical and molecular factors with the stage of cervical cancer in a Brazilian cohort. 2013;8(3):e57810.
16. Baskaran K, Karunanithi S, Sivakamasundari I, Sundresh N, Thamaraiselvi B, Swaruparani SJIJRPS. Overexpression of p53 and its role as early biomarker in carcinoma of uterine cervix. 2013;4:198-202.
17. Munhoz NG, Rodrigues DA, Pedregosa JF, Rodrigues JO, Junqueira MSG, Yonamine PTK, et al. The use of molecular markers (p16, Ki-67 and E-Cadherin) in uterine cervical biopsies. 2009;3(1):10-7.
18. Menezes HLd, Jucá MJ, Gomes EGdA, Nunes BL, Costa HO, Matos DJAdg. Analysis of the immunohistochemical expressions of p53, bcl-2 and Ki-67 in colorectal adenocarcinoma and their correlations with the prognostic factors. 2010;47:141-7.
19. Choi S-K, Kim TJ, Hong SB, Lee HTJKJoCLS. Expression of p53 and Ki-67 in cervical dysplasia with human papilloma virus infection or non-infection. 2004;36(2):178-84.
20. Stoenescu T, Ivan LD, Stoenescu N, Azoicăi DJRM-CaSdMsNDI. Assessment tumor markers by immunohistochemistry (Ki67, p53 and Bcl-2) on a cohort of patients with cervical cancer in various stages of evolution. 2011;115(2):485-92.
21. Verma R, Gupta V, Singh J, Verma M, Gupta G, Gupta S, et al. Significance of p53 and ki-67 expression in prostate cancer. 2015;7(4):488-93.
22. Gorban N, Ten V, Pankratov VJVO. Ki-67 and p53 expression as prognosticators of clinical course aggressiveness of squamous cell cancer of the larynx. 2007;53(6):668-73.
23. Moul JW, Bettencourt M-C, Sesterhenn IA, Mostofi F, McLeod DG, Srivastava S, et al. Protein expression of p53, bcl-2, and KI-67 (MIB-1) as prognostic biomarkers in patients with surgically treated, clinically localized prostate cancer. 1996;120(2):159-67.

How to cite: Sunaina Singh, Mahendra Singh, Chayanika Kala, Lubna Khan. Evaluation of p53 and Ki-67 Expression in Cervical Intraepithelial Neoplasia (CIN) and Invasive Cervical Cancer: A Prospective Observational Study. *International Medicine, 2025;11 (1):1-10.*