EUROPEAN ACADEMIC RESEARCH
Vol. XIV, Issue 5/ August 2026

Impact Factor: 3.4546 (UIF)
DRJI Value: 5.9 (B+)

ISSN 2286-4822
www.euacademic.org

Expression and Diagnostic Performance of Cell-Cycle Biomarkers in HPV-Associated Cervical Lesions among Sudanese Women

THURAYA ABDELAH MOHAMMED ELHASSAN
College of Applied Medical Sciences, Department of Medical Laboratory Sciences
Shaqra University, Saudi Arabia
Dr. MUBARAK ELSAEED MUSTAFA ELKARSANY
Department of Pathology, Faculty of Medicine, Karary University, Khartoum, Sudan
Dr. MOHAMED ELFATIH ABDALLA ABDELLATIF ABDELWADOUD
Department of Histopathology and Cytology, Faculty of Medical Laboratory Sciences
Nile University, Khartoum, Sudan.
MUTAZ MOHAMED IBRAHIM ALI[1]
Independent Consultant and Jointly Appointed Assistant Professor
University of Medical Sciences and Technology (UMST), Khartoum, Sudan.

Abstract:

Background: *Histologic grading of cervical squamous lesions can be challenging when tissue is limited or obscured by inflammation, or when morphology is equivocal. Locally applicable evidence on immunohistochemical adjuncts remains scarce in Sudan. This study evaluated the expression and apparent diagnostic performance of p14, p15, p16, p53, and Ki-67 across HPV-associated cervical lesions in a referral-based Sudanese cohort.*

Methods: *This cross-sectional diagnostic-pathology study included 200 HPV-positive women. Consensus histology classified specimens as negative/benign (n = 20), low-grade squamous intraepithelial lesion (LSIL/CIN1; n = 30), high-grade squamous intraepithelial lesion (HSIL/CIN2-3; n = 100), or invasive squamous cell carcinoma (SCC; n = 50). Biomarker expression was assessed in formalin-fixed, paraffin-embedded tissue. Kruskal-Wallis tests, Spearman rank correlations, and receiver operating characteristic analyses assessed differences across lesion groups and discrimination of HSIL or worse (HSIL+) and invasive SCC.*

Results: *Expression increased with lesion severity for p53 (Spearman r = 0.822), Ki-67 (r = 0.789), p14 (r = 0.768), p15 (r = 0.736), and p16 (r = 0.697); all p < 0.001. For HSIL+, the apparent areas under the curve were 0.999 for p16 and Ki-67, 0.998 for p53, 0.992 for p14, and 0.978 for p15. For invasive SCC, p53 showed the strongest single-marker discrimination (AUC = 0.894), followed by Ki-67 (AUC = 0.854).*

Conclusions: *p16 and Ki-67 were the most clinically interpretable adjuncts for HSIL-oriented assessment, whereas p53 was more informative for the invasive phenotype. Because this referral-enriched cohort was also the derivation dataset, the reported performance estimates require prospective external validation before any marker is used as a stand-alone clinical threshold.*

Keywords: cervical cancer; human papillomavirus; immunohistochemistry; p16; Ki-67; p53; cervical intraepithelial neoplasia; Sudan

[1] Corresponding Author: Dr. Mutaz Mohamed Ibrahim Ali, Independent Research Consultant in Medical Laboratory Sciences, Egypt. Bank Street, Building 61, Flat 3, Wadi El-Natrun (Natrn Valley), Beheira Governorate, Egypt. Telephone: +20 45 355 2112; Email: moatasa@hotmail.com; ORCID: 0009-0000-4963-2955.

1. INTRODUCTION

Cervical cancer remains a major, yet largely preventable, cause of morbidity and mortality. The burden is concentrated in low- and middle-income countries, where HPV vaccination, organized screening, colposcopy, diagnostic pathology and treatment pathways are often fragmented [1-3]. In Sudan, disruption of health services has intensified the practical need for diagnostic strategies that use limited tissue and a small number of high-yield reagents.

Persistent infection with a carcinogenic human papillomavirus (HPV) type is the central causal event in most cervical squamous carcinomas, but HPV positivity alone does not establish that an epithelial lesion has entered a transforming phase [4,5]. High-risk HPV E7 disrupts the retinoblastoma pathway and releases E2F-dependent cell-cycle activity, whereas E6 alters p53-mediated responses to cellular stress and DNA damage [6,7]. These mechanisms provide the biological rationale for tissue biomarkers that distinguish viral exposure from transformation.

p16 is the most established immunohistochemical surrogate of HPV-associated retinoblastoma-pathway disruption. Strong, diffuse block-type staining can support a diagnosis of transforming squamous disease when morphology is equivocal. Ki-67 identifies cells in active phases of the cell cycle and becomes abnormally distributed above the basal and parabasal layers as squamous lesions progress. The Lower Anogenital Squamous Terminology (LAST) recommendations support indication-based p16 use, particularly for an equivocal differential diagnosis or a lesion morphologically considered CIN2; they do not support indiscriminate replacement of morphology by immunohistochemistry [8,9].

p53, p14 and p15 provide complementary information but require more cautious interpretation. p53 immunoreactivity is not a direct surrogate for HPV infection: E6-mediated degradation, cellular stress, abnormal protein stabilization and mutation-like staining patterns can produce heterogeneous results. p14 and p15 are involved in cell-cycle restraint and senescence-associated pathways, but their diagnostic roles in routine cervical pathology are less established than those of p16 and Ki-67 [10,11]. Local evaluation is therefore important before proposing a practical biomarker hierarchy for a resource-constrained setting.

This study evaluated p14, p15, p16, p53 and Ki-67 expression across the histological spectrum of HPV-associated cervical lesions in Sudanese women. The primary objective was to quantify the association between each marker and ordered lesion severity. The secondary objective was to estimate the apparent discrimination of continuous marker expression for HSIL+ and for invasive SCC. We hypothesized that p16 and Ki-67 would be most informative for HSIL-oriented assessment, whereas p53 would contribute more strongly to characterization of invasive disease.

2. MATERIALS AND METHODS

2.1 Study design and setting

This hospital-based, cross-sectional diagnostic-pathology study included records and cervical specimens from 200 HPV-positive Sudanese women referred for diagnostic evaluation. Recruitment reflected service displacement: 80 participants (40.0%) were enrolled at Soba University Hospital, and 120 (60.0%) entered through decentralized

referral pathways encompassing River Nile State (including Shendi/River Nile RICC), Northern State, and cities in Eastern Sudan. Because the cohort was enriched for clinically significant lesions, it was analyzed as a referral series rather than a population-screening sample.

Eligible participants had documented high-risk HPV positivity, cervical cytology and/or colposcopic assessment, an adequate cervical biopsy or excisional specimen, and sufficient formalin-fixed, paraffin-embedded (FFPE) tissue for the core analysis. Specimens were excluded when fixation precluded interpretation, tissue was insufficient, prior cervical cancer treatment preceded sampling, a non-cervical primary tumor involved the cervix, or no reliable final histological diagnosis could be assigned.

2.2 Histopathology and study endpoints

Hematoxylin and eosin-stained sections were reviewed by two pathologists when dual review was available, and discordant interpretations were resolved by consensus. The reference-standard categories were negative/benign, LSIL/CIN1, HSIL/CIN2-3, and invasive SCC. The primary binary endpoint was HSIL+, defined as CIN2, CIN3, or invasive SCC versus negative/benign or CIN1. The secondary endpoint was invasive SCC versus all noninvasive categories. Interobserver agreement was evaluated using unweighted and quadratically weighted Cohen kappa statistics.

2.3 Immunohistochemistry

Immunohistochemistry was performed on 3-5 μm formalin-fixed, paraffin-embedded sections for p14, p15, p16, p53, and Ki-67 using the study laboratory's established staining workflow. Sections were deparaffinized and rehydrated, followed by antigen retrieval, primary-antibody incubation, and chromogenic detection. Positive and negative controls were included in each run; technically inadequate slides were classified as uninterpretable rather than biologically negative.

p16 assessment considered both nuclear/cytoplasmic distribution and the proportion of stained lesional cells, with block-type staining interpreted in conjunction with morphology. Ki-67, p53, p14 and p15 were recorded as percentages of positive lesional nuclei. Continuous percentage values were retained for severity correlations and receiver-operating-characteristic analyses. Biomarker interpretation was performed in the context of the final histological diagnosis; immunostaining was treated as an adjunct rather than a replacement for architecture and cytomorphology.

2.4 Statistical analysis

Categorical variables were summarized as counts and percentages, and continuous variables as means and standard deviations. Because biomarker percentages were bounded and were not assumed to follow a normal distribution, differences across the four histologic groups were assessed using Kruskal-Wallis tests. Spearman rank correlation quantified monotonic associations with ordered histologic severity.

Receiver operating characteristic curves were used to estimate the area under the curve (AUC) for continuous marker expression against the HSIL+ and invasive-SCC endpoints. The AUCs are apparent derivation-sample estimates because no independent validation set was available. Two-sided p values < 0.05 were considered statistically significant. Analyses followed transparent diagnostic-accuracy reporting principles, including explicit definition of the target condition and reference standard [12].

3. RESULTS

3.1 Cohort characteristics

The mean age was 39.77 ± 9.39 years. Final consensus histology identified 20 negative/benign specimens (10.0%), 30 LSIL/CIN1 lesions (15.0%), 100 HSIL/CIN2-3 lesions (50.0%), and 50 invasive SCCs (25.0%). Thus, 150 women (75.0%) met the HSIL+ endpoint. HPV16 or HPV18 was recorded in 143 participants (71.5%). Table 1 summarizes the cohort, and Figure 1 shows the distribution of final histologic diagnoses. Agreement between the two initial readers was almost perfect (unweighted kappa = 0.820; quadratically weighted kappa = 0.880). Disagreements were concentrated between adjacent grades rather than between benign and invasive categories.

Table 1. Selected characteristics of the HPV-positive referral cohort (N = 200).

Characteristic	Value
Age, years, mean ± SD	39.77 ± 9.39
Married	175 (87.5%)
Urban residence	97 (48.5%)
HPV16/18 genotype group	143 (71.5%)
Negative/benign histology	20 (10.0%)
LSIL/CIN1	30 (15.0%)
HSIL/CIN2-3	100 (50.0%)
Invasive SCC	50 (25.0%)
Soba University Hospital recruitment	80 (40.0%)
Decentralized referral recruitment	120 (60.0%)

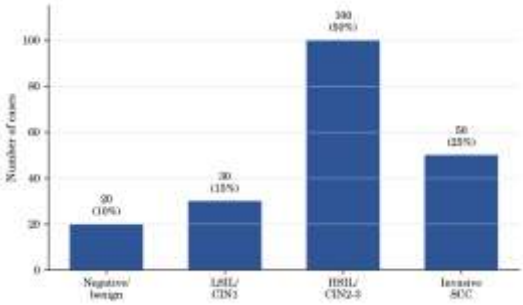

Figure 1. *Distribution of final consensus histology in the HPV-positive referral cohort.*

Age and clinical presentation differed between women with negative/benign or low-grade lesions and those with HSIL+. Women younger than 30 years accounted for 44.0% of the low/negative stratum but only 4.0% of the HSIL+ stratum; conversely, 63.3% of women with HSIL+ were aged 40 years or older. Symptomatic presentations, particularly abnormal bleeding, postcoital bleeding, and pelvic pain, were more frequent in the HSIL+ stratum (overall p = 0.003). HPV16/18 grouping did not differ significantly across the four

histologic categories (p = 0.543), underscoring the distinction between viral-risk classification and tissue-level evidence of transformation.

3.2 Biomarker expression across lesion severity

All five cell-cycle or proliferation-associated markers increased substantially across the histologic spectrum (Table 2; Figure 2). p53 showed the strongest monotonic association with severity (r = 0.822), followed by Ki-67 (r = 0.789), p14 (r = 0.768), p15 (r = 0.736), and p16 (r = 0.697); all p < 0.001. Mean p16 expression increased from 13.85% in negative/benign tissue to 87.88% in HSIL/CIN2-3 and 91.74% in invasive SCC. Mean Ki-67 expression increased from 15.30% to 81.93% and 91.44%, respectively.

Table 2. Quantitative biomarker expression across final histology groups.

Marker	Negative /benign (n = 20)	LSIL/CIN1 (n = 30)	HSIL/CIN2-3 (n = 100)	Invasive SCC (n = 50)	Spearman r	p value
p14 (%)	15.65 ± 6.14	36.53 ± 8.69	68.44 ± 13.14	80.12 ± 10.04	0.768	<0.001
p15 (%)	18.90 ± 10.61	38.80 ± 14.90	73.93 ± 12.80	84.36 ± 10.50	0.736	<0.001
p16 (%)	13.85 ± 9.54	46.97 ± 10.24	87.88 ± 8.55	91.74 ± 5.62	0.697	<0.001
p53 (%)	2.75 ± 2.79	9.63 ± 7.39	53.95 ± 13.96	73.68 ± 14.00	0.822	<0.001
Ki-67 (%)	15.30 ± 7.46	35.13 ± 12.39	81.93 ± 9.77	91.44 ± 6.53	0.789	<0.001

Values are mean ± SD unless otherwise indicated. Spearman r represents correlation with the ordered four-level histological severity scale.

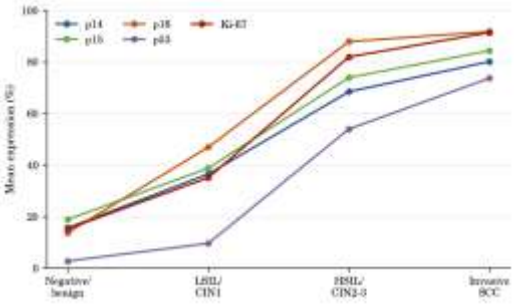

Figure 2. *Mean cell-cycle and proliferation biomarker expression across lesion severity.*

The steep rise in p16 and Ki-67 between LSIL/CIN1 and HSIL/CIN2-3 was clinically notable. p16 largely reached a plateau at the HSIL stage, whereas p53 and Ki-67 continued to rise in invasive SCC. This difference anticipated the endpoint-specific discrimination observed in the ROC analysis.

3.3 Apparent discrimination of HSIL+ and invasive SCC

For HSIL+, all five continuous markers showed very high apparent discrimination (Table 3; Figure 3). p16 and Ki-67 each yielded an AUC of 0.999; p53, p14, and p15 yielded AUCs of 0.998, 0.992, and 0.978, respectively. The ranking changed when invasive SCC was the target condition: p53 produced the largest AUC (0.894), followed by Ki-67 (0.854), p14

(0.836), p15 (0.815), and p16 (0.751). The lower invasive-SCC AUC for p16 reflects its high expression in both HSIL and invasive disease and, consequently, its limited ability to separate these two clinically advanced categories.

Table 3. Apparent AUCs of continuous biomarkers for two diagnostic endpoints.

Marker	AUC for HSIL+	AUC for invasive SCC
p14 (%)	0.992	0.836
p15 (%)	0.978	0.815
p16 (%)	0.999	0.751
p53 (%)	0.998	0.894
Ki-67 (%)	0.999	0.854

HSIL+ comprises CIN2, CIN3 and invasive SCC. AUC estimates were calculated in the same referral cohort used to develop the analysis and therefore represent apparent performance.

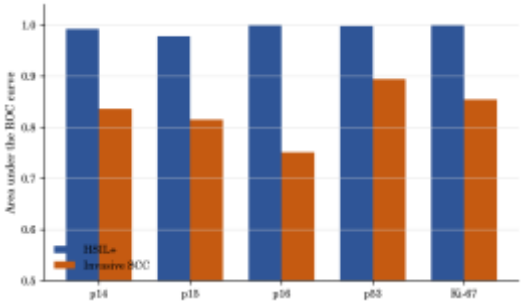

Figure 3. *Comparison of apparent biomarker AUCs for HSIL+ and invasive-SCC endpoints.*

4. DISCUSSION

This referral-based study identified a coherent tissue-biomarker gradient across HPV-associated cervical lesions. p16, Ki-67, p53, p14 and p15 all increased with histological severity, but their clinical interpretation was not identical. p16 and Ki-67 were most informative at the transition to HSIL, whereas p53 and Ki-67 were more informative when the target was established invasion. This distinction is biologically plausible and clinically useful: a marker can be highly effective for recognizing transforming disease without being equally effective for distinguishing HSIL from invasive SCC.

The p16 result accords with the established mechanism of high-risk HPV transformation. E7-driven retinoblastoma-pathway disruption produces compensatory p16 overexpression, making block-positive staining a practical surrogate of transforming HPV activity [6,13]. In this cohort, mean p16 expression rose sharply between CIN1 and CIN2-3 and changed relatively little thereafter. This pattern supports p16 as an adjunct for the low-grade/high-grade boundary, not as a reliable marker of invasion. It also reinforces the LAST principle that p16 should resolve a defined morphological question rather than be ordered routinely for every cervical biopsy [8,9].

Ki-67 supplied complementary evidence of abnormal proliferation. In non-neoplastic squamous epithelium, proliferating cells are normally confined to basal and

parabasal compartments. Expansion of Ki-67-positive nuclei through the epithelial thickness reflects loss of maturation-associated growth control. The marked increase observed in HSIL and invasive SCC is consistent with previous reports linking Ki-67 distribution to lesion grade and high-risk HPV-associated transformation [11,14]. Its continued rise in invasive SCC explains why its invasive-SCC AUC exceeded that of p16. p53 showed the strongest correlation with ordered severity and the highest single-marker AUC for invasive SCC. This finding should not be simplified to mean that p53 is an HPV surrogate or that high immunoreactivity necessarily proves TP53 mutation. High-risk HPV E6 can promote degradation of wild-type p53; conversely, invasive tumors may show altered protein stabilization, mutation-like staining patterns, DNA-damage responses or clonal heterogeneity. In practice, p53 is therefore better positioned as a selective marker for an invasive or biologically atypical phenotype than as a routine triage test for all HPV-positive lesions [10].

p14 and p15 also demonstrated strong gradients and high apparent AUCs for HSIL+. These findings are biologically consistent with increasing disruption of cell-cycle restraint and senescence pathways. Their incremental value beyond p16 and Ki-67, however, cannot be established from single-marker ROC curves. Correlated biomarkers can each show strong univariable performance without improving a parsimonious multivariable diagnostic model. In a setting where reagent availability is unstable, the practical question is not how many markers are statistically significant but which minimum panel changes diagnostic confidence and management.

The extremely high HSIL+ AUCs require guarded interpretation. First, the study population was referral-enriched: three quarters of participants had HSIL or invasive SCC, and many lesions were morphologically advanced. This creates spectrum effects that can separate diagnostic categories more sharply than would occur in a primary-screening population. Second, the AUCs were estimated in the same dataset used to define the analysis; there was no external cohort or resampling-based optimism correction. Third, semi-quantitative immunohistochemical percentages may be influenced by fixation, antigen retrieval, antibody performance and observer selection of lesional fields. Consequently, the reported values describe internal discriminatory signal rather than validated clinical performance.

The absence of a significant association between HPV16/18 grouping and histological category is also informative. Genotyping identifies viral risk, whereas p16 and Ki-67 identify tissue consequences of transformation. HPV16/18 testing has proven value in screening and risk stratification [15,16], but genotype alone cannot substitute for histological assessment in an HPV-positive referral population. The present findings therefore support a layered model: virological testing identifies risk; cytology and colposcopy direct assessment; histology establishes the lesion; and selected immunohistochemistry resolves defined morphological uncertainty.

For Sudanese referral pathology, a resource-conscious algorithm is preferable to universal panel staining. H&E morphology should remain the foundation. p16 is the first adjunct when CIN2 is being considered, when a lesion is equivocal, or when cytology, colposcopy and histology are discordant. Ki-67 can add evidence of abnormal proliferation when p16 and morphology do not provide a coherent answer. p53 may be reserved for suspected invasion or an atypical high-grade phenotype. p14 and p15 remain research-oriented until their incremental diagnostic value and reproducibility are demonstrated in independent cohorts. This morphology-first pathway is summarized in Figure 4.

The study has several strengths. It used consensus histology as the reference standard, included sufficient numbers across four ordered lesion categories, retained continuous marker values and evaluated clinically meaningful endpoints. It also addressed a diagnostic question relevant to disrupted, resource-limited services rather than extrapolating uncritically from organized screening programs.

The limitations are equally important. The cross-sectional design cannot establish progression. Referral enrichment limits generalizability and makes predictive values prevalence dependent. Confidence intervals for the AUCs and external validation were unavailable. Glandular lesions and HPV-independent cervical tumors were not represented; therefore, the findings should not be extrapolated beyond HPV-associated squamous disease. Finally, an apparent diagnostic hierarchy does not demonstrate clinical utility; prospective studies must determine whether selective biomarker use improves diagnostic reproducibility, treatment decisions, or patient outcomes.

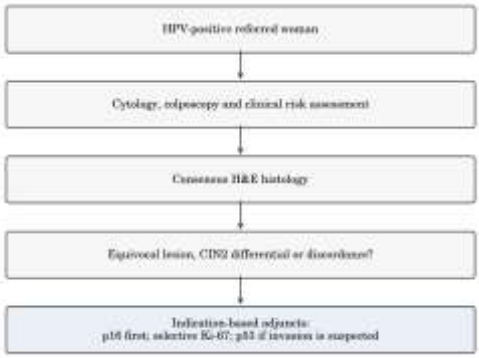

Figure 4. *Proposed morphology-first, indication-based biomarker pathway for referral cervical pathology. This pathway is interpretive and was not prospectively tested as a clinical algorithm.*

5. CONCLUSION

In this HPV-positive Sudanese referral cohort, p16 and Ki-67 most clearly identified the transition to HSIL, whereas p53 and Ki-67 were more informative for the invasive phenotype. A morphology-first, indication-based approach using p16 as the principal adjunct, selective Ki-67 for proliferative confirmation and p53 for suspected invasion is biologically coherent and potentially feasible in low-resource referral practice. The very high apparent AUCs should not be interpreted as externally validated accuracy; prospective multicenter confirmation with standardized staining, predefined scoring and independent validation is required.

DECLARATIONS

Consent for publication: Not applicable; no directly identifying participant information is presented.
Funding: This research received no external funding.
Competing interests: The authors declare no competing interests.

Data availability: Aggregate data supporting the findings are reported in the article. Access to de-identified individual-level data is subject to institutional approval and participant-confidentiality requirements.
Author contributions: Thuraya Abdelah Mohammed Elhassan: Conceptualization, investigation, data curation, methodology, formal analysis and writing - original draft. Mubarak Elsaeed Elkarsani: Conceptualization, methodology, supervision, validation and writing - review and editing. Mohamed Elfatih Abdalla Abdellatif Abdelwadoud: Methodology, co-supervision, pathology interpretation, validation and writing - review and editing. Mutaz Mohamed Ibrahim Ali: Manuscript development, scientific interpretation, writing - review and editing, project administration and correspondence.
Acknowledgements: The authors acknowledge the participating women and the clinical, cytology, histopathology and laboratory teams who supported specimen collection, processing and review.

REFERENCES

1. Arbyn M, Weiderpass E, Bruni L, de Sanjose S, Saraiya M, Ferlay J, et al. Estimates of incidence and mortality of cervical cancer in 2018: a worldwide analysis. Lancet Glob Health. 2020;8(2):e191-e203.
2. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, Bray F. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2021;71(3):209-249.
3. World Health Organization. Global strategy to accelerate the elimination of cervical cancer as a public health problem. Geneva: World Health Organization; 2020.
4. Schiffman M, Castle PE, Jeronimo J, Rodriguez AC, Wacholder S. Human papillomavirus and cervical cancer. Lancet. 2007;370(9590):890-907.
5. Doorbar J, Egawa N, Griffin H, Kranjec C, Murakami I. Human papillomavirus molecular biology and disease association. Rev Med Virol. 2015;25(Suppl 1):2-23.
6. Moody CA, Laimins LA. Human papillomavirus oncoproteins: pathways to transformation. Nat Rev Cancer. 2010;10(8):550-560.
7. Yeo-Teh NSL, Ito Y, Jha S. High-risk human papillomaviral oncogenes E6 and E7 target key cellular pathways to achieve oncogenesis. Int J Mol Sci. 2018;19(6):1706.
8. Darragh TM, Colgan TJ, Cox JT, Heller DS, Henry MR, Luff RD, et al. The Lower Anogenital Squamous Terminology Standardization Project for HPV-associated lesions: background and consensus recommendations. Arch Pathol Lab Med. 2012;136(10):1266-1297.
9. Clinton LK, Miyazaki K, Ayabe A, Davis J, Tauchi-Nishi P, Shimizu D. The LAST guidelines in clinical practice: implementing recommendations for p16 use. Am J Clin Pathol. 2015;144(6):844-849.
10. Murphy N, Ring M, Heffron CC, King B, Killalea AG, Hughes C, et al. p16INK4A, CDC6, and MCM5: predictive biomarkers in cervical preinvasive neoplasia and cervical cancer. J Clin Pathol. 2005;58(5):525-534.
11. Nam EJ, Kim JW, Hong JW, Jang HS, Lee SY, Jang SY, et al. Expression of p16 and Ki-67 in relation to the grade of cervical intraepithelial neoplasia and high-risk human papillomavirus infection. J Gynecol Oncol. 2008;19(3):162-168.
12. Bossuyt PM, Reitsma JB, Bruns DE, Gatsonis CA, Glasziou PP, Irwig L, et al. STARD 2015: an updated list of essential items for reporting diagnostic accuracy studies. BMJ. 2015;351:h5527.
13. Wentzensen N, von Knebel Doeberitz M. Biomarkers in cervical cancer screening. Dis Markers. 2007;23(4):315-330.
14. Ouh YT, Kim HY, Yi KW, Lee NW, Kim HJ, Min KJ. Enhancing cervical cancer screening: review of p16/Ki-67 dual staining as a promising triage strategy. Diagnostics (Basel). 2024;14(4):451.
15. Castle PE, Stoler MH, Wright TC Jr, Sharma A, Wright TL, Behrens CM. Performance of carcinogenic human papillomavirus testing and HPV16 or HPV18 genotyping for cervical cancer screening of women aged 25 years and older: a subanalysis of the ATHENA study. Lancet Oncol. 2011;12(9):880-890.

16. Dillner J, Rebolj M, Birembaut P, Petry KU, Szarewski A, Munk C, et al. Long-term predictive values of cytology and human papillomavirus testing in cervical cancer screening: joint European cohort study. BMJ. 2008;337:a1754.

AUTHOR BIOGRAPHICAL NOTES

Thuraya Abdelah Mohammed Elhassan *(Student researcher and first author)*
Thuraya Abdelah Mohammed Elhassan is a doctoral researcher in histopathology and cytology at Karary University. She earned a BSc in Histopathology and Cytology from Shendi University in 2000 and an MSc in Histopathology and Cytology from Sudan University of Science and Technology in 2008. Her doctoral research focuses on HPV-associated cervical lesions, cell-cycle biomarkers, apoptosis, and tissue microarray methods among Sudanese women.
Mubarak Elsaeed Elkarsani *(Supervisor and second author)*
Mubarak Elsaeed Elkarsani is Professor of Pathology and Microbiology at Karary University and the principal supervisor of the doctoral study. He provided senior conceptual, methodological, and supervisory guidance.
Mohamed Elfatih Abdalla Abdellatif Abdelwadoud *(Co-supervisor and third author)*
Mohamed Elfatih Abdalla Abdellatif Abdelwadoud is Assistant Professor of Histopathology and Cytology and served as co-supervisor of the doctoral study. He contributed expertise in pathology methods, laboratory interpretation, and scientific supervision.
Mutaz Mohamed Ibrahim Ali *(Corresponding author)*
Mutaz Mohamed Ibrahim Ali is an Assistant Professor and consultant in histopathology and cytology. He contributed to manuscript development, scientific interpretation, and critical English-language revision and serves as the corresponding author. Correspondence: moatasa@hotmail.com.