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Subtypes of endocervical cancer: A retrospective, observational study at Charlotte Maxeke Johannesburg Academic Hospital

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Background. Cervical cancer is the second most common malignancy in women, particularly in developing countries. Endocervical adenocarcinoma (ECA) is less common than cervical squamous cell carcinoma (SCC), but its incidence is increasing globally, particularly in young women. The decline in the incidence of SCC is attributed to effective screening programmes.
Objectives. To assess the prevalence of ECA and its subtypes and to describe the clinicopathological characteristics of patients with these tumours at a tertiary South African institute between 2017 and 2019.
Methods. This was a cross-sectional, descriptive study of 156 ECA patients. Following ethical clearance, demographic data, clinical information and disease characteristics were obtained from departmental histopathological reports. Descriptive statistics were used to calculate the prevalence of ECA. We analysed the association between age, Papanicolaou (Pap) smear results, human papillomavirus (HPV) status, and HIV status with ECA.
Results. The prevalence of ECA was 6.8% and it was more commonly diagnosed in younger women. HPV-associated subtypes were the most common variants. The usual type of ECA accounted for 24.4% of cases. HIV status was documented in 64.0% of cases, of which 34.0% were positive. There were no statistically significant associations between ECA subtype and HIV status (p=0.81) or between ECA subtype and Pap smear results.
Conclusion. In South Africa, the prevalence of ECA is lower compared with Western countries, reflecting inadequacies in screening modalities of ECA at primary healthcare facilities. HPV prevails as a cause of endocervical carcinoma. HPV morphologic hallmarks serve as a practical guide in classifying ECAs according to their HPV status.

Keywords. endocervical adenocarcinoma; HPV; HIV; Pap smear; South Africa.

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Endocervical adenocarcinoma (ECA) is less common than squamous cell carcinoma (SCC) of the cervix. However, global estimates indicate that the rates of ECA, relative to those of SCC, are rising in developed countries, particularly in young women.[1] ECA accounts for 10 - 25% of all cervical cancers.[2] Persistent infection with high-risk human papillomavirus subtypes is the most important causative factor in preinvasive lesions and most SCCs and adenocarcinomas. Therefore, surveillance of precancerous lesions, which are at present more frequent than invasive cancers, is one of the indicators of cancer screening evaluation.[3] In developed countries the decline in cervical SCC is attributed to the early introduction of effective cytological screening programmes and implementation of HPV vaccination programmes.[1] Cytological screening effectively detects SCCs in the early stages, whereas adenocarcinomas have been reported to be less detectable by screening.[4] The changing prevalence of oncogenic types of HPV may also contribute to the increase in adenocarcinoma. Persistent viral infection with high-risk types of HPV is established as a necessary cause of both cervical cancer subtypes.[4]

To date, there have not been studies to assess if similar trends apply to sub-Saharan countries, including South Africa (SA). Owing to the enormous HIV burden in southern African countries, most studies have assessed cervical squamous cell carcinoma in HIV-positive women. Our retrospective study aimed to determine the prevalence of ECA and its varied subtypes and to describe the age distribution and HIV status of patients diagnosed at Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) in Johannesburg, SA, over a 3-year period (2017 - 2019).

Methods

A Systemised Nomenclature of Medicine (SNOMED) search yielded a cohort of 156 cases. Haematoxylin and eosin-stained slides of non-usual subtypes of ECA were retrieved and reviewed to confirm diagnoses. In addition, where HPV subtyping was not done, the authors used HPV histological hallmarks (apical mitoses and basal karyorrhexis) to subtype cases on microscopy.[5] Inclusion criteria encompassed all primary cases of ECA diagnosed between 2017 and 2019 and included mostly cervical biopsies rather than resection specimens. Tumour recurrences, metastases and endometrial biopsies were excluded from the data set. Histopathology report review included extraction of demographic data (age and primary hospital), clinical information (Pap smear result, HIV status, and CD4 cell count), as well as disease characteristics (type of biopsy, histological subtype and HPV association). Where HIV and CD4 cell count results were unavailable on the histopathology report, data were sought from the laboratory information system (TrakCare) (following approval from the Department of Microbiology and Infectious Diseases).
Descriptive statistics were performed. Data were abnormally distributed and age groups were defined as medians with interquartile ranges (IQRs). ECAs overall, and yearly prevalence was calculated and the results were expressed as percentages. Histological subtypes and HPV

associations were recorded and classified according to the World Health Organization (WHO) classification of cervical adenocarcinoma (5th edition).[5] Categorical data were summarised as frequencies and percentages. Associations of ECA subtype with HIV status and Pap smear result were assessed using the chi-square test, and statistically significant *p*-values were set at *p*-values ≤0.05.

Ethics

Ethical approval was obtained from the University of the Witwatersrand's Human Research Ethics Committee (ref. no. M191161) and the National Health Laboratory Service (ref. no. M150885).

Results

A total of 156 ECA cases were diagnosed during the study period. The youngest patient was 26 years old, and the oldest was 88. Biopsies were submitted from 15 hospitals and clinics serviced by the Department of Anatomical Pathology. The Department of Obstetrics and Gynaecology at CMJAH referred the bulk of the cases (19.0%; *n*=30).

Demographic and clinical characteristics are summarised in Table 1. The median (IQR) age of the patients whose tumours were assessed was 49 (41.25 - 62.00) years. The majority (30.13%; *n*=47) of our patients were between 40 and 50 years of age at presentation, while 28.6% (*n*=45) were older than 60 years. The most frequently examined specimens were cervical punch biopsies (88.5%; *n*=138) of cases. In contrast, cervical curettings and large loop excision of the transformation zone (LLETZ) biopsies comprised 1.9% (*n*=3) and 6.4% (*n*=10) of patient cases, respectively. Total abdominal hysterectomy specimen results were assessed in 3.2% (*n*=5) of cases, as previous cervical biopsies demonstrated adenocarcinoma *in situ* or their previous biopsy results were unavailable.

The overall prevalence of ECAs was 6.8%. Most cases (39.1%; *n*=61) were diagnosed in 2017, with a yearly prevalence of 2.7%. The least number of patients (26.3%; *n*=41) were diagnosed in 2019, and the prevalence rate in that year was only 1.8%.

An assessment of HPV subtyping was performed (at the time of initial histological examination) using *p*16 immunohistochemistry (IHC), a known surrogate marker for HPV infection. In 47.1% (*n*=73) of the cases, there was diffuse block-type positive staining (Fig. 1). HPV *in situ* hybridisation (ISH) was performed in 1 case of adenocarcinoma not otherwise specified (NOS), and HPV-ISH (HPV 16) was positive. In the remainder of cases (51%; *n*=80), where HPV status remained undetermined as neither *p*16 nor ISH was performed, HPV histological hallmarks (apical mitoses and basal karyorrhexis) were used to subtype cases microscopically. Using this WHO-recommended method, in the absence of ancillary studies, most (90.3%; *n*=141) ECAs were determined to be HPV-associated (Fig. 2).

HPV-associated adenocarcinoma NOS histology dominated overall (59.0%), compared with the usual-type (including villoglandular subtype) which accounted for 24.4% of cases. There were 11 (7.1%) cases of mucinous carcinoma, of which mucinous NOS was predominant in 6 (54.5%) cases. The remainder of mucinous carcinomas included 4 intestinal-type (36.4%) cases and 1 (0.6%) case of signet-ring cell type. Fifteen cases (7.7%) were classified as non-HPV-associated carcinoma, of which clear-cell carcinoma accounted for 8 (5.1%) cases, while HPV-independent gastric-type was seen in only 1 (0.6%) case (supplementary Fig. 3; http://coding.samedical.org/file/2350).

HIV results were documented for 100 (64.1%) cases. Of these, 34.0% were HIV-positive, and 30.1% were HIV-negative (Table 1).

Table 1. Demographic and clinical data

Characteristic	*n* (%)*
Median (IQR) age at diagnosis, years	49 (41.25 - 62.00)
Age interval for ECA subtypes, years	
HPV-associated adenocarcinoma NOS	36 - 46
Usual type (including villoglandular)	
Mucinous carcinoma	
Serous carcinoma	58 - 68
HPV-independent gastric type	
HPV-independent adenocarcinoma NOS	
Clear-cell carcinoma	69 - 79
HIV status	
Positive	53 (34.0)
Negative	47 (30.1)
Unknown/no result	56 (35.9)
Pap smear	
Unknown/no result	111 (71.2)
Negative for intraepithelial lesion or malignancy	1 (0.6)
(LSIL)	4 (2.6)
(HSIL)	21 (13.5)
Atypical squamous cells of undetermined significance (ASCUS)	2 (1.3)
Atypical squamous cells cannot exclude HSIL (ASCH)	3 (1.9)
Atypical glandular cells (AGC)	2 (1.3)
Adenocarcinoma in situ	2 (1.3)
Adenocarcinoma in situ and HSIL	1 (0.6)
Adenocarcinoma	8 (5.1)
Carcinoma	1 (0.6)
*p*16 IHC stain	%
Positive	47.1
Negative	1.9
Not performed/no result	51.0
Associations	p-value
ECA subtype association with HIV status	0.88
ECA subtype association with Pap smear result	0.82
HPV association with HIV status	0.29

IQR = interquartile range; ECA = endocervical carcinoma; NOS = no special type; HPV = human papillomavírus; LSIL = low-grade squamous intraepithelial lesion; HSIL = high-grade squamous intraepithelial lesion; ASCUS = atypical squamous cell of undetermined significance; ASCH = atypical squamous cells cannot exclude high-grade squamous intraepithelial lesion; AGC = atypical aglandular lesion.
*Unless otherwise specified.

CD4 counts were available for 38% of the HIV-positive cases, with an average cell count of 507.68 (278.9) cells/μL (range 76 - 1 333 cells/μL).

Pap smear results were undocumented for 111 (71.2%) cases (Table 1). High-grade squamous intraepithelial neoplasia (HSIL) was recorded in 13.5% (*n*=21) of cases with a Pap smear result. Only 1.2% (*n*=2) of patients had adenocarcinoma *in situ* as the precursor lesion, with 0.6% (*n*=1) occurring in conjunction with HSIL. Malignancy was confirmed in 9 Pap smears.

There was no statistically significant association between ECA histopathological subtype and the Pap smear result (*p*=0.81). Similarly, there was no correlation between histological subtype and HIV status (*p*=0.80). There was no statistically significant association between HPV association and HIV status (*p*=0.16) (Table 1).

Discussion

Nearly one-third (30.1%) of our cohort were women aged 40 - 50 years, comparable with Pirog *et al*.'s study[6] in which the average age of patients

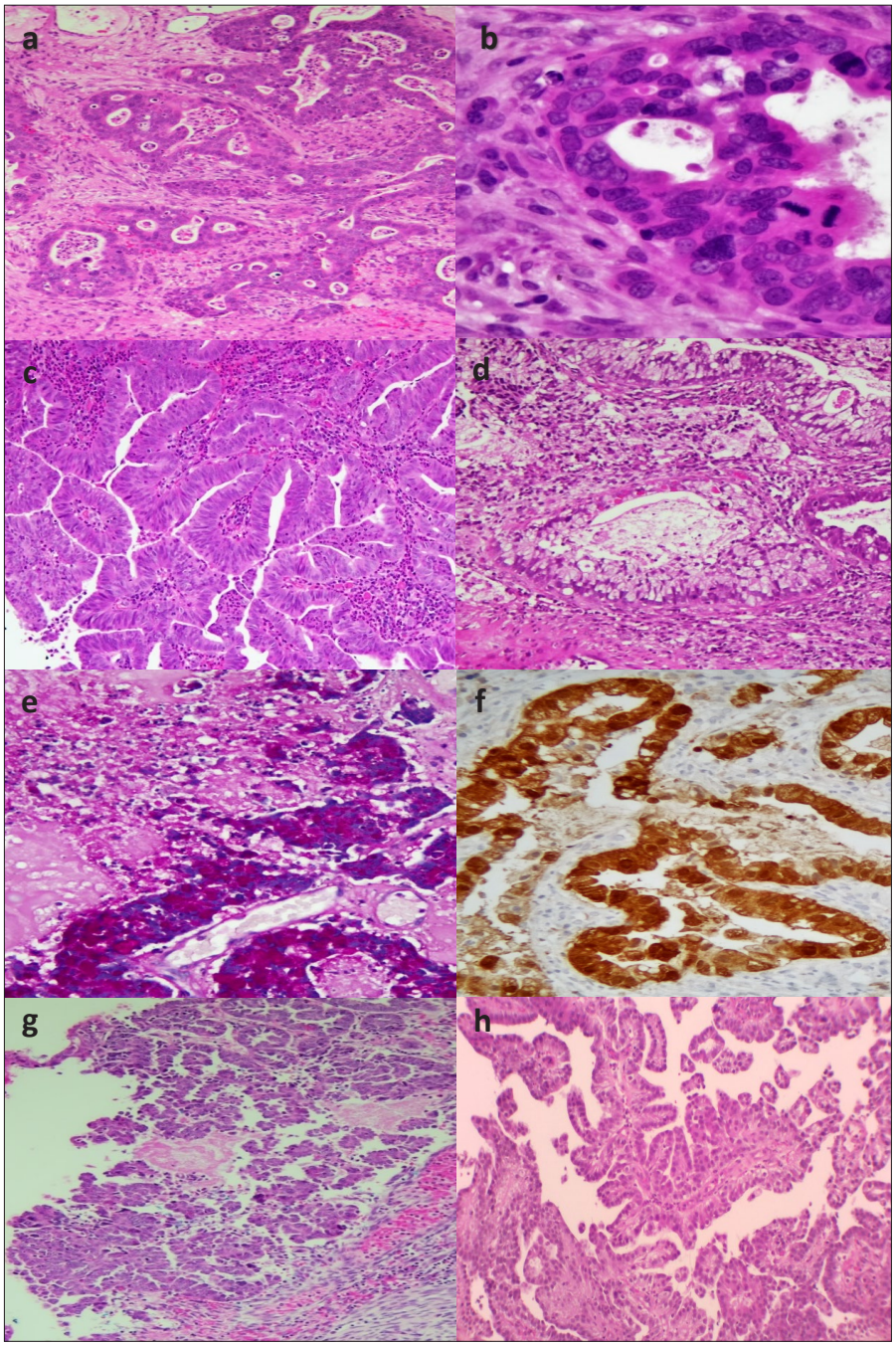

Fig. 1. ECA histological subtypes. HPV-related ECA (A - F): usual-type ECA (A and B); villoglandular (C), mucinous carcinoma NOS (D), periodic acid-Schiff with diastase (D-PAS) stain in mucinous carcinoma(E); and p16 IHC stain showing diffuse block-like positivity in tumour cells (F). HPV-unrelated carcinomas (G - H): serous carcinoma (G); and clear-cell carcinoma (H). Original magnification: 100× (A, C, D, G and H); 200× (E and F); and 400× magnification (B). (ECA = endocervical carcinoma; HPV = human papillomavirus; NOS = no special type; IHC = immunohistochemical.)

with ECA was 45 years. The study was a review into the pathogenesis and diagnostic criteria of both HPV positive and HPV negative cervical adenocarcinoma. In contrast to studies conducted in developed countries, our study showed ~4% lower prevalence of ECA.[2,7] The basis for this may be the reduced detection of ECA on Pap smears, given their high endocervical origin, which influences the number of patients referred for colposcopy-directed biopsies.[3,8,9] Of the recorded Pap smear results, the cytological detection of ECA was low at 5.1%, raising the possibility of similar challenges in our setting in diagnosing ECA. Furthermore, women in developing countries generally lack access to screening programmes, unlike their counterparts in developed countries.[8] In addition, low literacy levels and lack of funding for screening programmes create further hindrances in diagnosing and combating ECA.[10,11] In our study, atypical squamous cells of undetermined significance (ASCUS) was found in 1.3% of screened women, LSIL in 2.6%, and HSIL in 13.5%. Our LSIL and ASCUS results are comparable with those documented by Sachan *et al*.,[12] who reported ASCUS in 2.9%, LSIL in 5.1%, and HSIL in 0.5% of women screened during their study. The higher number of HSIL lesions in the present study may be attributed to the low turnout for voluntary screening. Therefore, most women present later with high-grade dysplasia.[12] The implementation of the 2001 National Cervical Screening Programme in SA by the National Department of Health has been challenging, as evidenced by the incidence and mortality rates associated with cervix carcinoma. Similar challenges are encountered in most other sub-Saharan African countries.[13,14]

Most cases in our study lacked characteristic morphological features to group them into a specific subtype such as usual type, mucinous, or many others under the current WHO classification of ECA. As a result, most cases (59%; *n*=92) were grouped into adenocarcinoma NOS, comprising 59.0% of the study population. Usual-type adenocarcinoma was the next most common subtype, but was much lower than that quoted in previous research which showed that the usual-type comprised up to 80% of cases of ECA.[1,7] This disparity may be seen because of the varied histological patterns of ECA.[7] This creates diagnostic difficulties, especially in small biopsies such as cervical punch biopsies, which were the most frequently examined specimens (88.5%) in the present study. Hesitancy of the pathologist to commit to a final diagnosis or a specific histological subtype on cervical biopsies may explain this outcome. In many cases, the definite classification was deferred to the excision specimen. However, considering the WHO's recently recommended HPV histological hallmarks (apical mitoses and basal karyorrhexis), it is hoped that pathologists may be able to commit to a definitive diagnosis on biopsy specimens which will help guide further patient management.

In the present study, the least reported subtypes were HPV-independent tumours. These included clear-cell carcinoma (5.1%) and serous-cell carcinoma (1.3%), which are rarely diagnosed in the literature.[15] In the current WHO classification of ECA, primary serous carcinoma of the cervix

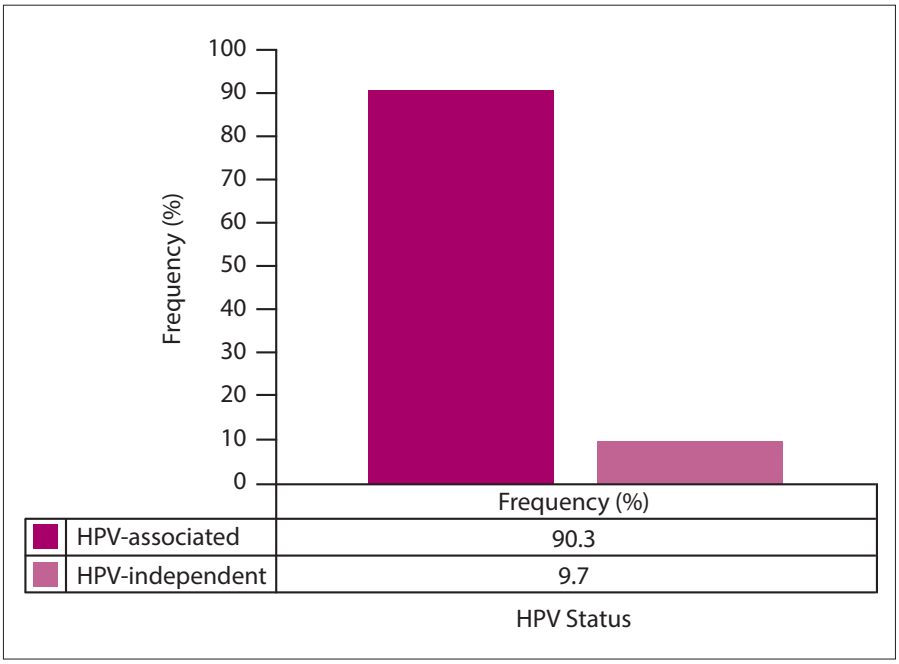

Fig. 2. Column chart illustrating the classification of ECA according to HPV status. (ECA = endocervical carcinoma; HPV = human papillomavirus.)

is a diagnosis made based on an aberrant *p*53 immunohistochemical staining pattern without HPV infection, after excluding extension from the uterus and ovary.[5]

Overall, HPV-associated ECA subtypes were the most commonly diagnosed subtype (90.3%) in our study, and their prognosis is known to be better than HPV-independent adenocarcinomas.[16] HPV in-situ hybridisation was rarely performed, due to resource limitations and a lack of *in-situ* hybridisation facilities. HPV subtyping using *p*16 IHC demonstrated diffuse block-type positivity in 49.0% of the cases. HPV histologic hallmarks (apical mitoses and basal karyorrhexis), as referenced in the WHO classification of cervical adenocarcinoma were used to subtype the remainder of 51.0% cases on microscopy. Despite HPV status playing a role in prognosis, it is of no importance in treatment. There is no specific treatment strategy based on histologic type or relation to HPV, as all ECA tumour subtypes are treated similarly.[1,2,17,18]

HIV infection remains rampant in SA, increasing the risk for cervical carcinoma six-fold.[10] This positive association between HIV and cervical carcinoma has been reported in Western counties such as Italy, France and Spain.[19] Similar findings were noted in studies from Uganda and Tanzania.[20] A slightly higher number of our ECA cases were co-infected with HIV, suggesting that a synergistic relationship may exist between HPV infection and HIV. However, given the limited data set on HIV and CD4 cell count results, these findings were not statistically significant. Despite some studies reporting positive associations, studies in Africa, notably Tanzania and Uganda, performed at the beginning of the AIDS pandemic, and in 2011, showed no association between HIV and cervical carcinoma.[21] This negative association is attributed to the fact that the progression of dysplasia to malignancy is multifactorial and not only dependent on immune status.[21,22]

Silverberg *et al.*[23] found that cervical neoplasia risk and thus, cancer risk is increased in women with <500 cells/µL. The average CD4 count recorded in HIV-positive cases in our study was 507 cells/µL. Hence, no significant associations were apparent in our cohort. It is important to note that the results may not be a true reflection of our cohort as only a limited number of CD4 count results were available. Similar results regarding low CD4 count and its associated increased risk of HPV infection were found in a study by Chakravarty.[24]

Study limitations

While most patient results were anticipated to be available on the NHLS LabTrack system, it is acknowledged that a subset of patients may have had their HIV and CD4 cell count tests conducted in private laboratories, to which we lack access.

Upon reviewing clinical request forms and searching the NHLS data system, only 64% of HIV results could be documented. Among those with positive HIV results, only 38% had an accompanying recorded CD4 count. Data collection was compromised by several factors:

Incomplete clinical data: The inadequacy and partiality with which clinical data and results are filled out by clinical colleagues (on patient request forms) contributed to missing information.

Data entry errors: Upon receipt and registration of specimens, multiple variables require entry, e.g. patient name, hospital number and date of birth among other identifiers – errors in this process can compromise data retrieval from the NHLS TrakCare system.

System migration issues: CMJAH's migration to a new patient identification and registration system resulted in changes to patient identification numbers. In many cases, the previous numbers were not adequately registered, complicating the retrieval of historical data and thus limiting our access to patient results.

Our ethical clearance did not permit us to directly contact patients whose HIV results were not found on the NHLS LabTrack system.

The limitations highlighted above significantly impacted our ability to obtain HIV results and CD4 counts for each of our patients. Our assessment and statistical analyses for HIV results and CD4 counts are thus only reflective of the cases in which these results were obtained and we cannot extrapolate these results to the entire cohort in our study.

The Pap smear results were not documented in many cases (71.2%). Therefore, ECA's low detection rates on Pap smear results should be interpreted cautiously. Limited data may have influenced this result, and a larger cohort of cases with available Pap smear results may give more accurate results with implications in the interpretation thereof.

Confirmatory HPV subtyping using genotyping, *p*16 IHC or HPV-ISH was not assessed in this study. Further, HPV subtyping and a more complete data set on HIV and CD4 count can be used in future studies to investigate the association between ECA, HPV subtypes and HIV status, as well as CD4 count. The correlation between biopsy results and the final diagnosis made on excision specimens can be assessed in future studies.

Conclusion

The overall prevalence of ECA in SA is lower compared to Western countries, reflecting the inadequacies in the screening modalities of

ECA in our primary healthcare facilities. HPV still prevails as a cause of endocervical carcinoma. However, our study demonstrates that the morphological classification of ECA, according to the current WHO guidelines, should be strictly adhered to. In the absence of ancillary studies, HPV histological hallmarks serve as a practical guide in classifying ECAs according to their HPV status.

Further research is needed to investigate the impact of HPV vaccination and screening programmes on the incidence of ECA in southern Africa.

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