Factors that Influence the Incidence of Human Papilloma Virus in HIV Positive Women in Central Java, Semarang, 2020

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Abstract

Human Immunodeficiency Virus (HIV) infection is still a health problem in the world. HIV tends to attack cells that have CD4 surface antigens. A decrease in CD4 count at a certain amount can open up opportunities for other infections, one of which is the Human Papilloma Virus (HPV). The prevalence of HPV in women in Indonesia is quite high 11.4%, data obtained from research in Jakarta 13.9%, 11.5% in Bali, and 8.8% in Tasikmalaya. Several factors influence HIV and HPV, including: sociodemographic factors, sexual and other risk behaviors, sexual and reproductive health factors and HIV history factors.

Purpose: The purpose of this study was to determined the factors that influence the incidence of HPV in HIV positive women in Central Java, Semarang.

Methods: This study used a cross sectional analytic method. The research subjects were all HIV positive female patients in Central Java, Semarang. Data collection was done by interviewing respondents using a questionnaire. The sample in this study were 42 HIV positive women. Inclusion criteria: HIV-infected female patients who were willing to be respondents in this study and the exclusion criteria had a history of total hysterectomy. The relationship between variables was tested by using the chi-square test. Results were considered statistically significant if the p value was <0.05.

Results: The results of univariate analysis of 42 respondents, 22 respondents (52.4%) were positive for HPV. The results of the bivariate analysis of sexual behavior and other risky behaviors showed that there was a significant relationship between the number of partners so far ≥ 2 with the incidence of HPV in HIV positive women. The results of the Chi-square test of the variable number of partners so far were P = 0.019 or <0.05 with PR = 2.292 (95% confidence interval (Cl) = 0.64-6.076).

Conclusion: HIV positive women who have partners for ≥ 2 years have a 2.2 times greater risk of developing HPV than women who have sexual partners <2.

Keywords: HIV, sociodemographic factors, sexual behavior factors, other risky behaviors, sexual and reproductive health factors, HIV history factors, HPV.

Introduction

Human Immunodeficiency Virus (HIV) infection is still a health problem in the world. HIV infection is caused by the Human Immonodificiency Virus, an RNA virus that causes a decrease in the body's immune system. The virus enters the human body through blood, semen and vaginal fluids. Most of it is transmitted through sexual contact. HIV tends to attack cells that have CD4 surface antigens, which play an important role in regulating and maintaining the body's immune system.[1] A decrease in CD4 count at a certain amount can open up opportunities for other infections, one of which is the Human Papilloma Virus (HPV). The results of research conducted in Bali stated that of the 50 women with HIV, 60% were HPV positive.[2] The prevalence of HPV infection in the world reportedly increased in the last 35 years. [3] The cumulative incidence of HPV infection in general in young adults is 40% with a prevalence reaching 75-80%.[4] HPV is the most common viral infection in sexually transmitted diseases and is thought to play a role in cervical cancer. [5] It is the fourth most common cancer in women in the world, with an estimated 570,000 new cases in 2018,[6] about 90% of cervical cancer deaths occur in low-income countries. [6] Data in Indonesia in 2018 shows cervical cancer of 23.4 per 100,000 population with an average death rate of 13.9 per 100,000 population, while the prevalence of HPV in women in Indonesia is quite high at 11.4%, data obtained from research in Jakarta 13, 9%, in Bali 11.5%, and in Tasikmalaya 8.8%.[2,7] Several factors influence HIV and HPV, including: other factors that affect HIV and HPV apart from NK cells, IFN-γ levels, IL-12 levels, CD4 levels, and CD8 levels, among others: sociodemographics, sociodemographic factors, among others (age, marital status, education level, and occupation), sexual behavior and other risky behaviors. Sexual behavior and other risky behaviors include: (smoking behavior, age at first sexual intercourse, number of sexual partners to date, regular partners, and use of condoms), reproductive health and sexual reproductive health and sexual health factors, including: contraceptive use, previous cervical cytology, history of infectious diseases, number of parities, and history of abortion. HIV history included: time of first diagnosis, current use of ARVs, and history of using ARVs. The use of ARVs to increase CD4 cells may have an impact on HPV infection control, and decrease VL to a lesser extent.[8] Although there are many studies on HPV in both HIV-infected and uninfected women, there are few studies on the factors that influence the incidence of HPV in HIV-infected patients. The purpose of this study was to determine the factors that influence the incidence of HPV in HIV positive women in Central Java, Semarang.

Method

The research design in this study used cross sectional[9,10]. This research was conducted at Obgyn polyclinic and Merpati polyclinic Dr. Kariadi Semarang. The study was conducted from 10 February to 30 April 2020. The sample of this study was taken from a population that met the inclusion criteria. The inclusion criteria were female patients who were infected with HIV and were willing to be respondents in this study and the exclusion criteria were having a history of total hysterectomy and were not willing to be respondents in this study. Data were collected by interview used a questionnaire.

Data analysis was carried out in a gradual manner, namely univariate analysis to describe the distribution and frequency of each variable, namely sociodemographic factors, sexual behavior factors and other risky behaviors, reproductive health factors and sexual

history factors of HIV. Bivariate analysis was carried out to test the hypothesis of the relationship between the independent variable and the dependent variable with the Chi Square (X^2) relationship test if P <0.05, there was a significant relationship between the independent variable and the dependent variable.

Results
Univariate Analysis
Overview of sociodemographic factors
Table 1.
Overview of sociodemographic factors with the incidence of HPV

Sociodemographic factors	Frequency (n)	Percentage (%)
Age		
>35 years	23	54,8
≤ 35 years	19	45,2
Marital Status		
Married	41	97.6
Not married	1	2.4
Education level		
Low (SD-SMP)	18	42,9
High (SMA and above)	24	57,1
Employment		
Work	18	42,7
Did not work	24	57,3

Based on table 1 shows that of the 42 respondents, 23 respondents (54.8%) were more than 35 years old, 25 respondents (59.5%) were married, as many as 41 respondents (97.6%) had higher education levels and as many as 24 respondents (57.3%) respondents did not work.

Overview of sexual behavior factors and other behaviors that are at risk of HPV incidence

Table 2.
Overview of sexual behavior factors and other behaviors that are at risk of HPV incidence

Sexual behavior factors and other risk behaviors	Frequency (n)	Percentage (%)
Smoking behavior		
Yes	5	11,9
No	37	88,1
Age at first sexual intercourse		
< 20 years	20	47.6
≥ 20 years	22	52.4
Use of condoms during sexual intercourse		
No	36	85,7

Yes	6	14,3
Number of current partners		
≥ 2	4	9,5
< 2	38	90,5
Number of couples so far		
≥ 2	32	76,2
< 2	10	23,8

Based on table 2, it shows that out of 42 respondents, 37 respondents (88.1%) did not smoke, for the first age at sexual intercourse <20 years, namely (52.4%), as many as 22 respondents (52.4%) did not use condoms when sexual relations, as many as 36 respondents (85.7%) had sexual partners currently <2, as many as 38 respondents (90.5%) had sexual partners so far ≥ 2.

Overview of sexual and reproductive health factors with the incidence of HPV
Table 3.
Overview of sexual and reproductive health factors with the incidence of HPV

Sexual behavior factors and other risk behaviors	Frequency (n)	Percentage (%)
Current contraceptive use		
Yes	18	42,9
No	24	57,1
Parity		
>2	6	14,3
≤ 2	36	85,7
Abortion		
Ever	13	31,0
Never	29	69,0
History of sexually transmitted diseases		
Ever	25	59,5
Never	17	40,5

Based on table 3, it shows that of the 42 respondents as many as 31 respondents (73.8%) did not use contraception at this time, as many as 32 respondents (76.2%) the number of parity ≤ 2, as many as 36 (85.7%) had never had an abortion, as many as 25 respondents (59.5%) had a history of sexually transmitted diseases.

History of HIV factors with the incidence of HPV
Tabel 4.
History of HIV factors with the incidence of HPV

HIV history factors	Frequency (n)	Percentage (%)
Time tested positive for HIV		
< 5 Years	14	33,3
≥ 5 Years	28	66,7

Years of use of ARVs		
< 5 years	15	35,7
≥ 5 years	27	64,3

Based on table 4, it shows data from 42 respondents as many as 33 respondents (78.6%) when they were declared HIV positive <5 years, as many as 33 respondents (78.6%) years of using ARVs.

HPV Incidence

Table 5.

HPV incidence

HPV status	Frequency (n)	Percentage (%)
Positive	22	52.4
Negative	20	47.6

Based on table 5 of 42 respondents, 22 respondents (52.4%) were positive HPV.

Table 6.

HPV High Risk

Kinds of HPV High Risk	Frequency (n)	Percentage (%)
HPV 51	1	4
HPV 52	3	13
HPV 66	3	13
HPV 68	2	9
HPV 56	2	9
HPV 18	3	13
HPV 39	1	4
HPV 53	1	4
HPV 58	2	9
HPV 59	2	9
HPV 16	3	13

Based on table 6, 3 respondents (13%) with HPV Type 52, 66, 18, 16 infection, 2 respondents (9%) with HPV type 68, 56, 58, 59 infection, 1 respondent (4%) with infection HPV types 51, 39, 53.

Table 7.

HPV Low Risk

Kinds of HPV Low Risk	Frequency (n)	Percentage (%)
HPV 54	1	4
HPV 55	1	4
HPV 42	2	8
HPV 43	4	15
HPV 44	4	15

HPV 6	4	15
HPV 86	1	4
HPV 26	1	4
HPV 70	1	4
HPV 81	3	11
HPV 53	1	4
HPV 40	1	4
HPV 61	1	4
HPV 72	1	4

Based on table 7, there are 4 respondents (15%) with HPV type 43,44,6 infection, 3 respondents (11%) with HPV type 81 infection, 2 respondents (8%) with HPV type 42 infection, and as many as 1 respondent (4%) with HPV infection type 54, 55, 86, 26, 70, 53, 40, 61, 72.

Bivariate Analysis

Table 8.

Relationship between Socioendographic Factors and the Incidence of HPV in HIV-infected women at RSUP Dr. Kariadi.

Socioendographic factors	X^2	p-value	PR	Cl 95%
Age	3,359	0,067	1,770	0,915-3,425
Marital Status	0,931	0,335	0,512	0,380-0,691
Education level	2,299	0,129	0,622	0,323-1,200
Work	0,795	0.372	0,762	0,411-1,413

Based on table 8, the Chi-square test results of the socioendographic factors obtained the value of the age variable P = 0.067 or <0.05, it can be concluded that the hypothesis of this study is that there is no relationship between age and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the variable of marital status, P = 0.512 or> 0.05, it can be concluded that the research hypothesis is rejected, namely there is no relationship between marital status and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the education level variable, the P value was obtained = 0.609 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between education level status and the incidence of HPV in HIV-infected women at Dr. Kariadi general Hospital. For the occupational variable, the P value was obtained = 0.762 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between educational status and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital.

Table 9.

The relationship between sexual behavior and other risky behaviors with the incidence of HPV in Dr. Kariadi General Hospital.

Sexual behavior factors and other risky behaviors	X^2	p-value	PR	Cl 95%

	X²	p-value	PR	CI 95%
Smoking bheavior	0,132	0,716	1,168	0,535-2,552
Age at first sexual intercourse	0,105	0,746	1,100	0,618-1,957
Use of condoms during sexual intercourse	0,016	0,900	1,056	0,488-2,489
Number of current partners	0,907	0,341	1,500	0,784-2,870
Number of couples so far	5,517	0,019	3,125	0,879-11,109

Based on table 9, the Chi-square test results of sexual behavior and other risky behaviors, the value of the smoking behavior variable is obtained P = 1.168 or> 0.05, it can be concluded that the research hypothesis is rejected, namely there is no relationship between smoking behavior and the incidence of HPV in HIV-infected woman at dr Kariadi General Hospital. For the variable age at first sexual intercourse, it was obtained that the value of P = 1.100 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between the age at first sexual intercourse and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the variable condom use during sexual intercourse, the P value is 1.083 or> 0.05, it can be concluded that the research hypothesis is rejected, namely that there is no relationship between condom use during sexual intercourse and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the variable number of partners currently, the value of P = 1.644 or > 0.05 can be concluded that the research hypothesis is rejected, namely that there is no relationship between the number of current sexual partners and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the variable number of partners so far P = 0.019 or <0.05, it can be concluded that the research hypothesis is accepted, namely that there is a relationship between the number of sexual partners so far and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital with PR = 2.292 (0.64-6.076) means that the factor of the number of sexual partners so far has a 2,292 times chance for the occurrence of HPV incidence in HIV-infected women.

Table 10.
The relationship between sexual and reproductive health factors and the incidence of HPV in Dr. Kariadi General Hospital

Reproductive and sexual health factors	X²	p-value	PR	CI 95%
Current contraceptive use	0,072	0,789	0,923	0,511-1,666
Parity	1,018	0,313	0,600	0,186-1,931
Abortion	0,673	0,426	1,275	0,720-2,257
History of sexually transmitted diseases	0,475	0,491	0,816	0,462-1,443

Based on table 10, the Chi-square test results of sexual and reproductive health factors, the value of the current contraceptive use variable is obtained P = 0.662 or> 0.05, it can be concluded that the research hypothesis is rejected, namely there is no relationship between current contraceptive use and the incidence of HPV. in HIV-infected women at RSUP Dr. Kariadi. For the Pap smear variable, the P value was obtained = 0.087 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between Pap smear and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For the abortion variable, it was obtained P value = 0.426 or> 0.05, it can be concluded that

the research hypothesis was rejected, namely that there was no relationship between abortion and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. For a history of sexually transmitted diseases, P = 0.491 or> 0.05, it can be concluded that the research hypothesis is rejected, namely that there is no relationship between a history of sexually transmitted diseases and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital.

Tabel 11.
The relationship between HIV history and the incidence of HPV in Dr. Kariadi General Hospital

HIV history factors	X^2	p-value	PR	Cl 95%
Time tested positive for HIV	2,339	0,126	0,588	0,274-1,262
Years of use of ARVs	1,434	0,231	0,675	0,337-1,351

Based on table 11, the Chi-square test results from the historical factor obtained that the value of the time variable was declared positive for HIV, it was obtained P = 0.071 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between the time when HIV was declared positive and the incidence of HPV in women who infected with HIV at Dr. Kariadi General Hospital. For the variable years of use of ARVs, the value of P = 0.071 or> 0.05 can be concluded that the research hypothesis was rejected, namely that there was no relationship between years of use of ARVs and the incidence of HPV in HIV-infected women at dr. Kariadi General Hospital.

Discussion

The relationship between sociodemographic factors and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. Based on table 8, the Chi-square test results of the socioendographic factors obtained a value for the variable age P = 0.067 or> 0.05, it can be concluded that the research hypothesis is rejected, it can be concluded that there is no relationship between age and the incidence of HPV in women infected with HIV in Dr. Kariadi General Hospital. This study is in line with Tropical Medicine and International Health (2018) that there is no relationship between age and the incidence of HPV with the results of the study P = 0.67 20, The British Journal in Kenya (2012) said that from a total number of studies of 498 women infected with HIV, the highest HPV occurred at an average age> 35 years but the results of the study were P = 0.136[13] and Yawo (2015) in Togo, with an average age of> 35 years but the results of the study were P = 0.972[11], but this research is not in line with research conducted by the World Health Organization (WHO). Comprehensive Cervical Cancer Control A guide to essential practice Second edition a, Switzerland: World Health, Organization; (2014), where it is said that at the age of 30 there is a high risk of HPV infection and is high at the age of> 35 years, Viroulogy Junral (2018) where it is said that Prevelations aged 36-40 have high levels of HPV following the increase in the age of the women[16].

The journal virology (2018) says that the incidence of HPV is higher in women 30-40 years with a peak age of 35 years, according to the Journal of Virus Eradication (2015), Cervical and anal HPV infection: Cylotogical and Histological abnormalities in HIV-infected women in Thaliand, where it is said that the age above> 35 years or people who are premenoupuse and post menoupouse are more prone to HPV because with a lower general

response, hormonal changes lead to weakness with a more fragile epithelium[14]. Although from the results of this study there was statistically no relationship between age and the incidence of HPV, the results of this study showed that the PR value was 1.770, meaning that women who were> 35 years old had 1.7 times the risk of HPV infection compared to those aged <35 years.

The variable of marital status obtained a P value = 0.335 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between marital status and the incidence of HPV in HIV-infected women at dr Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there is no relationship between marital status and the incidence of HPV with the results of the study P = 0.08[12]. This study is in line with research conducted by Yawo (2015) in Togo which showed that there was no relationship between marital status and the incidence of HPV with the results of the study P = 0.748[11]. This study is not in line with research conducted by Edoardo (2017) in Italy showing that there is a relationship between marriage and the incidence of HPV with the results of the study P = 0.0002[17]. Although the results of this study statistically there is no relationship between marital status and the incidence of HPV, the results of this study show that the PR value = 0.512 means that married women have a risk of developing HPV by 0.5 times compared with unmarried women.

The educational level variable obtained a P value = 0.129 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between education level and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. This research is in line with research conducted by Monique et al (2018) in Amazonas showing that there is no relationship between educational status and the incidence of HPV with the results of the study P = 0.21[12]. This study is in line with research conducted by Yawo (2015) in Togo which shows that there is no relationship between education and the incidence of HPV with the results of the study P = 0.738[11]. This research is not in line with research conducted by Edoardo (2017) in Italy which shows that there is a relationship between education and the incidence of HPV with the results of the study P = 0.01[17]. Although the results of this study statistically there is no relationship between education level and the incidence of HPV, the results of this study show that the PR value = 0.662 means that women who have low education have a risk of developing HPV by 0.6 times compared to women with higher education.

The occupational variable obtained a P value = 0.372 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between work and the incidence of HPV in HIV-infected women at Dr. This research is in line with research conducted by Monique et al (2018) at Amazonas showing that there is no relationship between work and the incidence of HPV with research results P = 0.25[12]. This study is in line with research conducted by Yawo (2015) in Togo which shows that there is no relationship between education and the incidence of HPV with the results of the study P = 0.462[11]. Although the results of this study statistically there is no relationship between work status and the incidence of HPV, the results of this study show that the PR value = 0.762 means that working women have a risk of developing HPV by 0.7 times compared to women who do not work.

The relationship between sexual behavior and other risky behaviors with the incidence of HPV in Dr. Kariadi General Hospital. Based on table 9, the Chi-square test results of the factors of sexual behavior and other risky behaviors obtained a value for the variable number of sexual partners so far P = 0.019 or <0.05, it can be concluded that the research hypothesis is

accepted, namely that there is a relationship between the number of sexual partners during This is the incidence of HPV in HIV-infected women at dr Kariadi General Hospital with PR = 3.125 (0.879 - 11.109) meaning that the number of sexual partners > 2 has a chance of 3.125 times the occurrence of HPV in HIV-infected women and has a high level of correlation. This study is in line with research conducted by Hapsari (2019) in Mataram (NTB) which shows that there is a relationship between the number of sexual partners and the incidence of HPV with the results of the study P = 0.000[21]. An increase in the number of sexual partners is the most consistent risk factor for HPV infection and it is statistically increased for sexual partners who have sexual relations with commercial sex workers or work as commercial sex[22]. Kahn (2009) states that HPV infection can be acquired several months after sexual intercourse. The results of Franceschi's (2009) study show that changing partners $\geq$ 3 times has OR = 1.5[21]. This is because sexual behavior between partners will lead to an increase in the transmission of venereal diseases. This is because sexual behavior between partners will lead to an increase in the transmission of venereal diseases.

The smoking behavior variable obtained P = 0.716 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between smoking behavior and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there was no relationship between smoking behavior and the incidence of HPV with the results of the study P = 0.54 [12]. Although the results of this study statistically there is no relationship between smoking behavior and the incidence of HPV, the results of this study obtained a PR value = 1.168, meaning that women who have smoking behavior have a risk of developing HPV by 1.1 times compared to women who do not smoke.

The variable of age at first sexual intercourse obtained P value = 0.746 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between the age at first sexual intercourse with the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there is no relationship between educational status and the incidence of HPV with the results of the study P = 0.52 [12]. Although from the results of this study there was no statistical relationship between the age at first sexual intercourse with the incidence of HPV, the results of this study showed that the PR value = 1.100 means that women who have sexual intercourse for the first time at the age <20 years have a risk of HPV by 1,1 time compared to women who had their first sexual intercourse at the age of> 20 years.

For the variable condom use during sexual intercourse, the P value is obtained = 0.900 or> 0.05, it can be concluded that the research hypothesis is rejected, namely that there is no relationship between condom use during sexual intercourse with the incidence of HPV in HIV-infected women at Dr. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there is no relationship between condom use status during sexual intercourse with the incidence of HPV with the results of the study P = 0.97 [12]. Although the results of this study statistically there is no relationship between the use of condoms during sexual intercourse with the incidence of HPV, the results of this study show that the PR value = 1.056 means that not using condoms during sexual intercourse has a risk of HPV by 1 times compared to those who use condoms.

The variable number of partners currently has a value of P = 0.341 or> 0.05, it can be concluded that the research hypothesis is rejected, namely that there is no relationship between the number of current sexual partners and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there was no relationship between the number of current sexual partners and the incidence of HPV with the results of the study P = 0.87 [12]. Although from the results of this study there was no statistical relationship between the number of current sexual partners and the incidence of HPV, the results of this study showed that the PR value = 1.500 means that women who have the number of sexual partners> or equal to 2 have a risk of developing HPV by 1.5. times compared with women whose number of sexual partners was <2.

The relationship between sexual and reproductive health factors and the incidence of HPV in Dr. Kariadi General Hospital. Based on table 10, the results of the Chi-square test of sexual behavior and other risky behaviors, the value of the current contraceptive use variable is obtained P = 0.789 or> 0.05, it can be concluded that the research hypothesis is rejected, namely there is no relationship between current contraceptive use. this is with the incidence of HPV in HIV-infected women at RSUP Dr. Kariadi. This study is in line with research conducted by Monique et al (2018) in Amazonas showing that there is no relationship between current contraceptive use and the incidence of HPV with the results of the study P = 0.50[12]. Although the results of this study statistically there is no relationship between contraceptive use and the incidence of HPV, the results of this study show that the PR value = 0.923, meaning that women who use contraception have a risk of developing HPV by 0.9 times compared to women who do not use contraception.

The parity variable obtained a value of P = 0.313 or > 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between parity and the incidence of HPV in HIV-infected women at dr Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) at Amazonas showing that there is no relationship between parity and the incidence of HPV with the results of the study P = 0.09[12]. This study is in line with research conducted by Yawo (2015) in Togo which shows that there is no relationship between parity and the incidence of HPV with the results of the study P = 0.74[11]. Although the results of this study statistically there is no relationship between parity and the incidence of HPV, the results of this study show that the PR value = 0.600 means that women who have parity> 2 have a risk of developing HPV by 0.6 times compared with women who have parity ≤ 2.

The abortion variable obtained a value of P = 0.426 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between abortion and the incidence of HPV in HIV-infected women at dr Kariadi General Hospital. This study is in line with research conducted by Monique et al (2018) in Amazonas which shows that there is no relationship between abortion and the incidence of HPV with the results of the study P = 0.30[12] Even though the results of this study are statistically there is no relationship between abortion and the incidence of HP, However, the results of this study showed that the PR value = 1.275 means that women who have had an abortion have a risk of developing HPV by 1.2 times compared to women who have never had an abortion.

History of sexually transmitted diseases P = 0.491 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between a history of sexually transmitted diseases and the incidence of HPV in HIV-infected women at dr Kariadi General Hospital. This study is in line with research conducted by Tristan (2017) in Thailand which shows that there is no relationship between a history of sexually transmitted diseases and the incidence of HPV with the results of the study P = 0.22[15]. Although the results of this study were statistically no relationship between a history of sexually transmitted diseases and the incidence of HPV, the results of this study showed that the PR value = 0.816 means that women who have a history of sexually transmitted diseases have a risk of developing HPV by 0.8 times compared with women who do not have a history of sexually transmitted diseases. The relationship between sexual and reproductive health factors and the incidence of HPV in Dr. Kariadi General Hospital. Based on table 11, the results of the Chi-square test of sexual and reproductive health factors showed that the time variable was declared positive for HIV, it was obtained P = 0.126 or> 0.05, it can be concluded that the research hypothesis was rejected, namely there was no relationship between the time when HIV was declared positive and the incidence of HPV in HIV-infected women at RSUP Dr. Kariadi. This research is in line with research conducted by Edoardo (2017) in Italy which shows that there is a relationship between the time when HIV positive and the incidence of HPV[17] and the results of the study P= 0.62. Although the results of this study were statistically no relationship between the time HIV was positive and the incidence of HPV, the results of this study obtained a PR value= 0.588, meaning that women who were first diagnosed with HIV <5 years had a risk of developing HPV by 0.5 times compared with women who first time HIV diagnosis ≥ 5 years.

The variable year of use of ARVs obtained a value of P = 0.231 or> 0.05, it can be concluded that the research hypothesis was rejected, namely that there was no relationship between years of use of ARVs and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital. This study is in line with research conducted by Edoardo (2017) in Italy showing that there is a relationship between the years of use of ARVs and the incidence of HPV with the results of the study P = 0.81[17]. Although from the results of this study there was no statistical relationship between years of use of ARVs and the incidence of HPV, the results of this study showed that the PR value = 0.675 means that women who have received ARV therapy <5 years have a risk of developing HPV by 0.6 times compared to women. who received ARV therapy for ≥5 years.

Types of HPV, Another study in Pune, India, obtained CIN data from colposcopic biopsy materials in HIV patients, where high risk HPV DNA was found in 52% CIN 1, 50% CIN 2, 67% CIN 3[18] and invasive cervical cancer. HPV prevalence studies conducted in 3 regions in Indonesia (Jakarta, Tasikmalaya, Bali) studied 2686 women, and obtained data on the presence of HPV infection in 305 (11.4%), of which 13.9% were in Jakarta, 8.8% in Tasikmalaya, 11.5% in Bali. The study found the most 3 types of high risk HPV from positive HPV samples, namely types 52, 16 and 18, with different prevalences in each region. In Jakarta, it was found that the prevalence of HPV types 52, 16 and 18 was 14.8%, 10.7% and 6.6% respectively, in Tasikmalaya 16.0%, 12.3% and 17.3%, in Bali 18, 0%, 15.0% and 12.0% [18]. There were also multiple HPV infections in 63 (20.7%) samples of HPV positive cases. The three types of HPV that are most often found in malignant cells of cervical cancer, namely

types 16, 18, 52[19]. This shows that HPV infection in cervical cancer patients, especially in Dr. Kariadi's RSUP, is more dominated by HPV type 18 followed by HPV type 16 and 52[18,19].

The results obtained from this study showed that 53.4% of HIV women were infected with HPV, compared to non-HIV patients (47.6%). Of the 22 HIV cases with HPV positive, it was found that type 16 was 3 people (13%), type 18 was 3 people (13%). A total of 4 people (18%) could not determine the type, and whether it was a single or mixed infection.

When compared with the prevalence of HPV type 16/18 infection in Indian women with HIV in the Pune region, there seems to be a similarity in that there are more HPV types 16 and 18 infections[18]. In this study, the age of patients with HPV type 16 infection showed a prevalence of 0% for ages <30 years, and 13.6% for ages ≥ 30 years. Meanwhile, for HPV type 18, 4.5% were <30 years old, and 9% for those aged ≥ 30 years. The Pune study found a higher prevalence of HPV type 16/18 infection for ages ≥ 30 years, of which 28.3% in women under 30 years and 40.5% in women 30 years and over[18]. Why the results obtained in this study are different from those in Pune, it is still necessary to do more in-depth research.

Conclusion

There is a relationship between the number of sexual partners so far and the incidence of HPV in HIV-infected women at Dr. Kariadi General Hospital.

Conflict of Interest

There is no conflict of interest.

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References

1. Clark B, Chetty R. Postmodern cancer : the role of human immunodeficiency virus in uterine cervical cancer. Mol Biol 2002; 55(1) : 19-24.

2. *Putu et al.* Prevalensi infeksi *Human Papilloma Virus* tipe 16 dan 18 serta lesi prakanker dari pemeriksaan *Pap smear* berbasis cairan pada pasien dengan infeksi *Human Immunodeficiency Virus.* Fakultas Kedokteran Universitas Udayana/RSUP Sanglah, Denpasar. Majalah Patologi VOL. 23 No.2 2014.

3. Shew ML, Fortenberry JD. HPV infection in adolescents: natural hist complications, and indicators for viral typing. Semin Pediatr Infect 2005;16(3):168-74.

4. Patel H, Wagner M, Singhal P, Kothari S. Systematic review of incidence and prevalence of genital warts. BMC Infect Dis. 2013;13:1-14.

5. *Setiawati D. Human Papilloma Virus* dan kanker serviks . Fakultas Ilmu Kesehatan UIN Allaudin, Makassar. Public Health Science Journal.VOL. VI No.2, Juli-Desember 2014.

6. Arbyn Marc , Weiderpass Elisabete, et all. Estimates of incidence and mortality of cervical cancer in 2018: a worldwide analysis. Lancet Glob Health (2019); https://doi.org/10.1016/ S2214-109X(19)30482-6

7. Vet JNI, Boer MA, Akker BEWM, et all. Prevalence of human papillomavirus in Indonesia: a population-based study in three regions. British Journal of Cancer. 2008; 99: p.214-218.

8. Djoerban Z.Djauzi S. HIV/AIDS di Indonesia Buku Ajar Ilmu Penyakit Dalam Edisi VI Jilid I. Jakarta:Balai Penerbit FKUI; 2015

9. Notoatmojo S. Metodologi penelitian kesehatan: Rineka Cipta; 2010. hal. 115-30.

10. Sabri L. Statistik kesehatan .Jakarta: Rajawali pres; 2008. hal. 181-2.

11. NyasenuI Tufa Yawo , Gbeasor-KomlanviI Adjidossi Fifonsi, et all. Prevalence and distribution of Human Papillomavirus (HPV) genotypes among HIV infected women in Lome,Togo. PLOSS ONE 2019; 14(2): e0212516. https://doi.org/10.1371/journal. pone.0212516

12. Teixeira Figueiredo Monique, Sabidó Meritxell Sabidó, et all. High risk human papillomavirus prevalence and genotype distribution among women infected with HIV in Manaus, Amazonas. Virology Journal (2018) 15:36 https://doi.org/10.1186/s12985-018-0942-6.

13. Vuyst De H, Mugo NR, et all. Prevalence and determinants of human papillomavirus infection and cervical lesions in HIV-positive women in Kenya. British Journal of Cancer (2012) 107, 1624–1630

14. Ramautarsing A Reshmie , Phanuphak3 Nittaya, et all. Cervical and anal HPV infection: cytological and histological abnormalities in HIV-infected women in Thailand, Thailand. *Journal of Virus Eradication* 2015; 1: 96–102

15. Delory Tristan , Ngo-Giang-Huong Nicole, et all. Human Papillomavirus infection and cervical lesions in HIV infected women on antiretroviral treatment in Thailand, Thailand. Journal of Infection (2017) xx, 1e11. http://dx.doi.org/10.1016/j.jinf.2017.02.007

16. World Health Organization(WHO). *Comprehensive Cervical Cancer Control A guide to essential practice Second edition a, Switzerland: World Health, Organization*; (2014)

17. Tartaglia Edoardo , Falasca Katia, et all. Prevalence of HPV infection among HIV-positive and HIV-negative women in Central/Eastern Italy: Strategies of prevention. ONCOLOGY LETTERS (2017) ;14: 7629-7635.

18. Joshi SN, Gopalkrishna V, Kumar K, et all. Cervical squamous intra-epithelial changes and human papillomavirus infection in women infected with human immunodeficiency virus in Pune, India. Wiley-Liss, Inc : J.Med.Virol. 2005; 76: p.470-475

19. Maker Luh Putu Iin Indrayani, Moestikaningsih, et all. Prevalensi Infeksi *Human Papilloma Virus* tipe 16 dan 18 serta Lesi Prakanker dari Pemeriksaan Pap Smear Berbasis Cairan pada Pasien dengan Infeksi *Human Immunodefisiensi Virus* di Salah Satu Klinik Infeksi Menular Seksual di Denpasar, Bali.2014

20. Carmago Milena, R_10-Ospina Del Luisa Del, et all. Association of HIV status with infection by multiple HPV types. Tropical Medicine and International Health (2018), volume 23 no 11 pp 1259–1268

21. Hapsari Y. Prevalensi dan faktor resiko highrisk HPV pada inspeksi visual asam asetat positif di Mataram. Jurnal Kedokteran 2019,8(2):12-16

22. Sangwa-Lugomo G, Ramankumar AV, Mahmud S. Liaras J,Kayembe PK, Tozin RR, Et al.Prevalence and determinants of high-risk human papillomavirus infection in women from a sub-saharan African community. Sexually transmitted disease. 2011;38(4);308-315