Serological Surveyand Risk Factors Associated with Oncogenic Human Papilloma Virus among Women of Reproductive Age in Funtua Zone Katsina State, Nigeria

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Abstract: Papillomaviruses are ubiquitous and are members of a family Papillomaviridae of DNA viruses that infect humans and animals. Human papillomaviruses (HPVs) are small non-enveloped viruses that contain a double-stranded, closed circular DNA genome. Human papillomaviruses cause anogenital warts and are associated with anogenital malignancy including cervical, vaginal, vulvar, penile, and anal carcinoma. This study was carried out to determine the prevalence of HPV IgM among women of reproductive age and associated risk and demographic factors in Funtua zone, Katsina state. A cross-sectional serological survey enrolling 60 women of reproductive age attending ANC of General Hospital Funtua. Serum samples were obtained from randomly selected subjects. Samples were tested IgM HPV specific commercial enzyme-linked immunosorbent assay (ELISA) kit. The overall prevalence of was (36.6%) recorded. There was no statistical significant association between women of reproductive age with sociodemorgraphic and risk factors analysed in this studies ($P \le 0.05$). Women of reproductive age attending ANC should be screened for HPV infection and further studies with larger sample size is recommended. Since infection with HPV is life-long and has no known cure, primary prevention remains the mainstay of its control.

Keywords: HPV, DNA, prevalence, IgM, ELISA.

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I. Introduction

An HPV infection is caused by *human papillomavirus*, a DNA virus from the papillomavirus family, of which over 170 types are known (Bzhalava *et al.*, 2013). More than 40 types are transmitted through sexual contact and infect the anus and genitals (CDC, 2015). Risk factors for persistent HPV infections include early age of first sexual intercourse, multiple partners, smoking, and poor immune function (WHO, 2010). HPV is typically spread by sustained direct skin-to-skin contact with vaginal and anal sex being the most common methods. Occasionally, it can spread from a mother to her baby during pregnancy (CDC, 2015). It does not spread via common items like toilet seats (CDC, 2015). People can become infected with more than one type of HPV (CDC, 2015).

HPV is the most common sexually transmitted infection globally (Milner and Danny, 2015). Most people are infected at some point in their lives (CDC, 2015). In 2012, about 528,000 new cases and 266,000 deaths occurred from cervical cancer worldwide (WHO, 2014). Around 85% of these occurred in the developing world (WHO, 2010). In the United States, about 27,000 cases of cancer due to HPV occur each year. About 1% of sexually active adults have genital warts (CDC, 2015). While cases of warts have been described since the time of ancient Greece, their viral nature was not discovered until 1907 (Tyringet *et al.*, 2016).

Human papillomaviruses (HPVs) are a group of more than 200 related viruses. More than 40 HPV types can be easily spread through direct sexual contact, from the skin and mucous membranes of infected people to the skin and mucous membranes of their partners. They can be spread by vaginal, anal, and oral sex other HPV types are responsible for non-genital warts, which are not sexually transmitted (ACS, 2014).

Sexually transmitted HPV types fall into two categories: Low-risk HPVs, which do not cause cancer but can, cause skin warts (technically known as *Condylomata acuminata*) on or around the genitals and anus. For example, HPV types 6 and 11 cause 90% of all genital warts. HPV types 6 and 11 also cause recurrent respiratory papillomatosis, a less common disease in which benign tumors grow in the air passages leading from the nose and mouth into the lungs while High-risk HPVs, which can cause cancer. About a dozen high-risk HPV

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types have been identified. Two of these, HPV types 16 and 18, are responsible for most HPV-caused cancers (CDC, 2012).

HPV infections are the most common sexually transmitted infections in the United States. About 14 million new genital HPV infections occur each year (Satterwhite *et al.*, 2018). In fact, the Centers for Disease Control and Prevention (CDC) estimates that more than 90% and 80%, respectively, of sexually active men and women will be infected with at least one type of HPV at some point in their lives (Chesso *et al.*, 2014). Around one-half of these infections are with a high-risk HPV type (Hariri *et al.*, 2011).

The Food and Drug Administration (FDA) has approved three vaccines to prevent HPV infection: Gardasil, Gardasil 9, and Cervix. These vaccines provide strong protection against new HPV infections, but they are not effective at treating established HPV infections or disease caused by HPV (Schiller *et al.*, 2012). Correct and consistent condom use is associated with reduced HPV transmission between sexual partners, but less frequent condom use is not (Winer *et al.*, 2006). However, because areas not covered by a condom can be infected by the virus (CDC, 2012), condoms are unlikely to provide complete protection against the infection.

I. Materials and Methods

Study Area

The study area is Funtua, which is among the three senatorial zones of Katsina State, Nigeria with a total area of $448 \, \mathrm{km}^2$ (173sq mi) and a population of 225,571 as per the 2006 census and 570,110 according to 2016 estimates. The inhabitants of the area are predominantly Hausa-Fulani and other tribes. The area comprises eleven local governments which include: Bakori, Danja, Dandume, Faskari, Sabuwa, Kankara, Malumfashi, Kafur, Musawa, Matazu, and Funtua.

Study design

The study is design base on cross sectional study to determine the prevalence and selected epidemiological features of cervical HPV infection.

Sample Size

The sample was be determine using the formula of (Sarmukaddam and Gerald, 2006) at 95% confidence level and a reported prevalence of 97.2% women presenting for Cervical Cancer Screening at the Federal Teaching Hospital Gombe (FTHG), North-eastern, Nigeria (Bashir *et al.*, 2017).

 $N=Z^2(P) Q/L^2$

Where;

N= Sample size

Z= Statistics for 95% = 1.96

P= Prevalence of previous study = 97.2%

L= Level of significance = 0.05

Q = 1-P(1-97.2%)

Therefore, the sample where rounded up to N = 60

Ethical clearance and consent

Ethical clearance was obtained from the Ministry of Health, Katsina state, and consent was obtained from both the hospital management and the patients to be subjected to the test, before the conductance of the test, and appropriate forms were designed and distributed to the hospital management before the inception of sample collection.

Inclusion and Exclusion Criteria

The inclusion criterion of the research is the woman being of reproductive age in the study area, while the exclusion included any other woman which the inclusion criteria do not involve, including those attending other hospitals within the study area.

Collection of blood sample

A total of 60 blood samples will be aseptically collected using syringe from the participants who gave their consent with assistance of the laboratory technologist. The whole blood was allowed to clot for 30 minutes and then centrifuged at 1000rmp for 10 minutes. The serum was carefully removed with a transfer pipette and transferred aseptically to a sterile, labeled, serum storage screw capped container and stored at -20°c in a freezer for analysis (Kabuga *et al.*, 2013).

Serological detection of HPV using IgM ELISA

The serum sample will be analyzed using Human papilloma virus Antibody IgM (HPV-IgM) ELISA kit manufactured by Melsin Medical Co Limited, with identification number CAT.NO:EKHU-0048 by strictly following the manufacturers' instructions.

II. Result

A total of 60 participants over 10yrs of age from pregnant woman attending ANC were recruited for the study with different characteristics (Table 1). Those included (100%:60/60) pregnant. Among the participants, (53.3:32/60%) were in the age group 31-40years. Distribution according to socio-economic status, (53.3:32/60%) were in medium socio-economic group and the participants participates in the study (35.0:21/60%) were in the secondary level education status. Among the participants, (95.0:57/60%) were house wife and (55.0:33/60%) were recorded as rural.

| Variable | Frequency Percentage (%) | • | • |
|-----------------------|--------------------------|------|---|
| Age group | | | |
| 10-20 | 04 | 6.66 | |
| 21-30 | 21 | 35.0 | |
| 31-40 | 32 | 53.3 | |
| 41-50 | 03 | 5.00 | |
| Socio economic status | | | |
| Low | 25 | 41.6 | |
| Medium | 32 | 53.3 | |
| High | 03 | 5.00 | |
| Education status | | | |
| Non-formal | 19 | 31.6 | |
| Primary | 19 | 31.6 | |
| Secondary | 21 | 35.0 | |
| Others | 01 | 1.66 | |
| Marital status | | | |
| Married | 56 | 93.3 | |
| Divorce | 04 | 6.66 | |

Out of 60 participant included in this study, an overall prevalence of HPV IgM of (36.6%:22/60) are found to be positive (Figure 1). The result of prevalence of HPV in relation to educational status is shown in Table 4.2, those with higher prevalence recorded (42.8%:9/5) and while (26.3%:5/9) recorded in secondary and non-formal group. There is no statistical significant association between educational status and HPV infection $(X^2=2.044, df=3, P=0.5633)$. Analysis of result by occupational status showed that higher prevalence (50.0%:1/2) recorded in skilled while lowest in married (38.5:22/57). There is no statistical significant association between the occupation and HPV infection $(X^2=1.630, df=2, P=0.4426)$.

The data and result were analyses according to tribe of the participant and this is shown the higher prevalence of (100%:3/3) and the lowest as (35.8%:19/53) in Igbo tribe and Hausa tribes. There is no statistical significant association between tribe and HPV infection $(X^2=7.109, df=3, P=0.0685)$. Analysis of result by marital status shows the highest prevalence (50.0%:2/4) in divorce woman and lowest prevalence recorded (35.7%:20/56) in married woman. There is no statistical significant association between married and HPV infection $(X^2=0.3281, df=1, P=0.5668)$. There is no statistical significant association between socio economic status and HPV infection $(X^2=0.4642, df=2, P=0.7927)$.

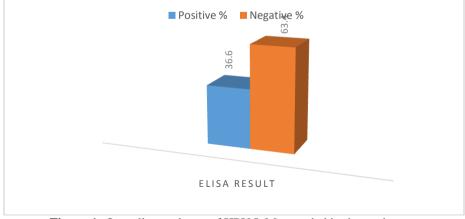


Figure 1: Overall prevalence of HPV IgM recorded in the study area

| Table 2: Prevalence of | HPV Infection i | n relation to | socio demos | raphic factors |
|-------------------------------|-----------------|---------------|-------------|----------------|

| Factors | Total | Positive (%) | Negative (%) | X^2 | P-value |
|------------------|-------|--------------|--------------|--------|---------|
| Educationstatus | | ` ' | | | |
| Non-formal | 19 | 05(26.3) | 14(73.6) | 2.044 | 0.5633 |
| Primary | 19 | 08(42.1) | 11(57.8) | | |
| Secondary | 21 | 09(42.8) | 12(57.1) | | |
| Others | 01 | 00(0.0) | 01(100) | | |
| Occupation | | | | | |
| House wife | 57 | 22(38.9) | 35(61.4) | 1.630 | 0.4426 |
| Skilled | 02 | 00(0.0) | 01(100) | | |
| Unskilled | 01 | 0(0.0) | 01(100) | | |
| Tribe | | | | | |
| Hausa | 53 | 19(35.8) | 34(64.1) | 7.109 | 0.0685 |
| Yoruba | 03 | 02(66.6) | 01(33.3) | | |
| Others | 04 | 04(100) | 00(0.0) | | |
| Maritalstatus | | | | | |
| Married | 56 | 20(35.7) | 36(64.2) | 0.3281 | 0.5668 |
| Divorce | 04 | 02(50.0) | 02(50.0) | | |
| Socio-economicst | atus | | | | |
| Low | 25 | 08(32.0) | 17(68.0) | 0.4642 | 0.7927 |
| Medium | 32 | 13(40.6) | 19(59.3) | | |
| High | 03 | 01(33.3) | 02(66.6) | | |

Analysis of data and result according to age group of the participant is shown in Table 3 were the higher prevalence was recorded (43.7%:14/32) among participants in 31-40yrs group and lower was recorded among age group 41-50yrs (33.3%:1/3). There is no statistical significant association between age group and HPV infection $(X^2=6.044, df=3, P=0.1095)$.

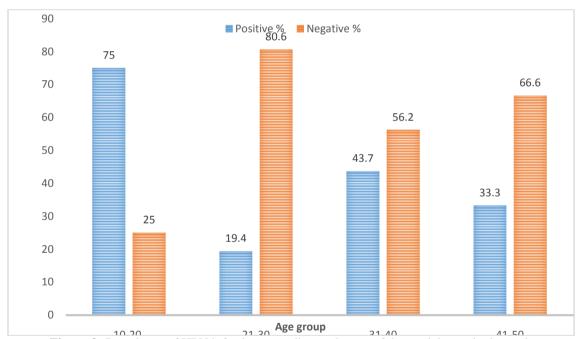


Figure 2: Prevalence of HPV infection according to the age of the participants in the study area.

The data and result were analyzed according risk factors as shown in Table 5. The higher prevalence was recorded among the participant with single sexual partner (41.6%) and those that have multiple sexual partners have lower prevalence (29.1%). There is no statistical significant association between sexual partner and HPV infection (X^2 =0.0071, df= 1, P=0.9327). Analysis according to parity is shows that those with primiparous have higher prevalence recorded (44.4%) compared to those with multiparous (34.7%). There is no statistical significant association between parity and HPV infection (X^2 =0.3287, df= 2, P=0.8485). Furthermore, those with HIV have higher prevalence (52.6%) and while those with no HIV infection had lower prevalence (29.26%). There is no statistical significant association between HPV infection and HIV (X^2 =2.129, df= 1, P=0.1446). The result based on age at sexual debut shows higher prevalence among participant with smallest age of sexual debuting 10-20yrs recorded (37.0%). There is no statistical significant association between age at sexual debut and HPV infection (X^2 =0.2512, df= 1, P=0.6162).

| Factors | Total | Positive | Negative | X^2 | P-value | |
|-----------------|-------|-----------|----------|-------|---------|--|
| Sexualpartner | | | | | | |
| Single | 36 | 15(41.6) | 21(58.3) | 0.007 | 0.932 | |
| Multiple | 24 | 07(29.1) | 17(70.8) | | | |
| Parity | | | | | | |
| Nulliparous | 05 | 02(40.0) | 03(60.0) | 0.328 | 0.848 | |
| Primiparous | 09 | 04(44.4) | 05(55.5) | | | |
| Multiparous | 46 | 16(34.7) | 30(65.2) | | | |
| HIVstatus | | | | | | |
| Positive | 19 | 10(52.6) | 09(47.3) | 2.129 | 0.144 | |
| Negative | 41 | 12(29.3) | 29(70.7) | | | |
| Ageatsexualdebi | ut | | | | | |
| 10-20 | 56 | 21(37.5) | 35(62.5) | 0.251 | 0.616 | |
| 21-30 | 04 | 01(25.0) | 03(75.0) | | | |
| STIstatus | | | | | | |
| Yes | 01 | 01(100.0) | 0(0.0) | 0.077 | 0.780 | |
| No | 59 | 21(35.5) | 38(64.4) | | | |

The prevalence of HPV IgM in relation to vaginal discharge which is associated with the clinical sign is shown in Table 4, were the higher prevalence was recorded (80.0%:4/5) among those with vaginal discharge. There is no statistical significant association between vaginal discharge and HPV infection ($X^2=2.610$, df= 1, P=0.1062). The higher prevalence was recorded (100%:1/1) among those with blisters than those without. There is no statistical significant association between blisters and HPV infection ($X^2=0.0778$, df= 1, P=0.7802). Those with vaginal bleeding havehigher prevalence of (50.0%) than those without vaginal bleeding (36.2%) and no statistical significant association between vaginal bleeding and HPV infection ($X^2=0.1584$, df= 1, P=0.6906). The result of prevalence of HPV in relation to burning sensation was shows with burning sensation has higher prevalence (75.0%) and while (33.9%) was recorded as the lower prevalence in those without burning sensation. There is no statistical significant association between burning sensation and HPV infection ($X^2=1.232$, df= 1, P=0.2671).

The result and prevalence of HPV in relation to trimester of pregnancy is shown in Figure 4, were the higher prevalence was recorded (100%:1/1) among those in first trimester group and while (35.0%:7/20) recorded as the lower prevalence among those with second trimester of pregnancy. There is no statistical significance association between trimester of pregnancy and HPV infection ($X^2 = 1.703$, df= 2, P=0.4268).

Table 4: Prevalence of HPV infection in relation to clinical symptoms observed in the study area

| Clinical Sign | Total | Positive (%) | Negative (%) | X^2 | P-value | |
|------------------|-------|--------------|--------------|--------|---------|--|
| Vaginaldischar | ·ge | | | | | |
| Yes | 05 | 04(80.0) | 01(20.0) | 2.610 | 0.1062 | |
| No | 55 | 18(32.7) | 37(67.2) | | | |
| Blisters | | | | | | |
| Yes | 01 | 01(100) | 00(0.0) | 0.0778 | 0.7802 | |
| No | 59 | 21(35.5) | 38(64.4) | | | |
| Vaginalbleeding | | | | | | |
| Yes | 02 | 01(50.0) | 01(50.0) | 0.1584 | 0.6906 | |
| No | 58 | 21(36.2) | 37(63.7) | | | |
| Burningsensation | | | | | | |
| Yes | 04 | 03(75.0) | 01(25.0) | 2.232 | 0.2671 | |
| No | 56 | 19(33.9) | 37(66.0) | | | |



Figure 4: Prevalence of HPV infection in relation to trimester of pregnancy in the study area

Analysis of result in relation to screening program is shown in Table 6, and higherprevalence (37.2%) was recorded among unscreened group and while (35.2%) was slightly recorded as the lower prevalence among screened participants. There is no statistical significance association between screening program and HPV infection (X^2 =0.0192, df= 1, P= 0.8897).

| Table 6: Prevalence of HPV | infection in relation | to screening among the | participant in the study area |
|-----------------------------------|-----------------------|------------------------|-------------------------------|
| Tuble of the function of the f | mirection in relation | to sercening among the | participant in the stady area |

| Factors | Total | Positive | Negative | X^2 | P-value |
|---------------------|-------|----------|----------|--------|---------|
| Screening | | | | | |
| Yes | 17 | 06(35.2) | 11(64.7) | 0.0192 | 0.8897 |
| No | 43 | 16(37.2) | 27(62.2) | | |
| Reasons for uptake | | | | | |
| Doctors request | 04 | 02(50.0) | 02(50.0) | 1.431 | 0.4890 |
| Self-interest | 04 | 02(50.0) | 02(50.0) | | |
| Screening program | 09 | 02(22.2) | 07(77.8) | | |
| Reason for Non-upta | ake | | | | |
| Painful | 15 | 09(60.0) | 06(40.0) | 5.161 | 0.0757 |
| Expensive | 11 | 03(27.2) | 08(72.7) | | |
| I am hey | 17 | 04(23.5) | 13(76.4) | | |

III. Discussion

The majority of this study's participant are within the 31-40 age demographic, a finding indicating that those women of middle, child bearing age are the highest in terms of attending the clinic, and probably more prone to HPV. Overwhelmingly, the participants included in the study are of medium socio-economic status. This corroborated the finding of CDC (2013), and Nejo *etal.*, (2018) who stated unemployment and low income earnings as significantly correlated with HPV prevalence.

More than one third of the participants in this study were educated up to secondary school level, a finding in conformity with findings of Nejo *et al.*, (2018) who reported that their study participants largely have secondary education. The finding also in concordance with submissions from Thomas *et al.*, (2004) and Kennedy *et al.*, (2016). Esere, (2008) suggests that lack of education may predispose individuals to sexual properties that mat trigger acquisition of HPV.

In the present study, the overall prevalence recorded (36.6%) is lower than the 76% prevalence by Auwal *et al.*, (2013) and higher than the 26.3% reported by Thomas *et al.*, (2004). This can be due to the variances in the occurrence of risk factors and the extent of spread of the etiologic agent across the study areas. Also, the figures reported by Auwal *et al.*, (2013) were from sexually transmitted disease clinic, whereas this study was from the ANC unit of general hospital Funtua local government.

Some literatures like Devuyst *et al.*, (2003) reported prevalence as high as 66.1% from Africa and a prevalence of 40% was reported by Castellsauge *et al.*, (2001). In Hanoi, prevalence is very low (20%), in Colombia it is about 14.8%. In Burkina Faso, it is about 66.1% Didelot-Rosser *et al.*, (2006); Molano *et al.*, (2002); and ANH *et al.*, (2013). Globally, 75% acquire it in their lifetime (Aral and Holmes, 2008). However, the estimated global prevalence is 11.7%, and 24% for sub-sahara Africa (Bruni *et al.*, 2010). If taken amongst the content of those previous researches, this study seems to be at the midpoint, with prevalence comparable to the adjusted average from Saharan Africa.

Married woman were less prone to be positive than divorce ones in the study, single sexual partners also predispose the participants to being positive, although those are not statistically significant. HIV status greatly influenced the positivity which agree with the findings of Neijo *et al.*, (2018), Auwal *et al.*, (2013), Idso *et al.*, (2009) amongst others. These are explainable by the fact that HIV positive weakens the immune system, subjecting it to HPV attack.

The divorced participants possess a higher prevalence which is in conformity with the finding of Nejo et al., (2018) and this may not be unconnected with changes in sexual orientation and behavior. The fact that single sexual partner's women possess a higher prevalence than those with multiple partners can be attributed to higher promissory on men

IV. Conclusion

An overall higher prevalence of 36.6% was obtained from this study. Of all the sociodemographic analysed in this study none of them was found to be statistically significant associated with HPV infection. Similarly, none of the risk factor studied were associated with HPV infection.

V. Recommendation

Even though no statistically significant association was found between the risk factors and the prevalence, there is the need to reevaluate attitudes of women, increase awareness, and initiate measures to

drastically reduced, and ultimately eliminate the HPV menace. In the future, additional research endeavors should be targeted towards the conductance of further studies with similar design in other to get a better outlook on the nature of the prevalence in other community. Vaccination with HPV should be stated in earnest, to control the exacerbation of the menace.

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Reference

- [1]. American Cancer Society (2014). Cancer facts and Figures 2014 exit Disclaimer. Atlanta: American Cancer Society.
- [2]. Auwal I.K, Amin M, Atanda, Tukur J, and Sarkinfada F, (2013). Prevalence and risk factors of high risk Human Papilomavirus infection among women attending gynecology clinics in kano, Northen Nigeria. *Bayero journal of pure and applied sciences*, 6(1): 67-71
- [3]. Bashir A, Abdulmalik YB, Musa SK, Mukhtar GL, Muhammad HR, Ummusalma AS, Bahauddeen SD, Sani A and Saddig AB, (2017). Academic project write-up and data analysis: A workshop manual prepared and organized by department of microbiology faculty of natural and applied sciences umaru musa yar'adua university, katsina, (14-16).
- [4]. Bruni, L., Barrionuevo-Rosas, L., Albero, G., Serrano, B., Mena, M., Gomez, Z., Muñoz, J., Bosch, FX., de, SS. Human Papillomavirus and related diseases report. ICO Information Centre on HPV and Cancer (HPV Information Centre); 2016.
- [5]. Centers for Disease Control and Prevention (2012). HPV-Associated Cancers Diagnosis by Age.
- [6]. Center for disease control (CDC), (2015). The link between Human papilloma virus (HPV) and cancer.
- [7]. Centers for Disease Control and Prevention. STD curruculum for clinical educators. Genital Human papillomavirus. 2013:1–37.
- [8]. Chesson HW, Dunne EF, Hariri S, Markowitz LE, (2014). The estimated lifetime probability of acquiring human papillomavirus in the United States. Sexually Transmitted Diseases; 41(11):660-664.
- [9]. De Vuyst H, Steyaert S, Van Renterghem L, Claeys P, Muchiri L, Sitati S, Vansteelandt S, Quint W, Kleter B, Van Marck E, Temmerman M, (2003). Distribution of human papillomavirus in a family planning population in nairobi, kenya. Sex Transm Dis.; 30:137–142.
- [10]. Einstein MH, Martens MG, Garcia FA, Ferris DG, Mitchell AL, Day SP, (2002). Clinical validation of the Cervista HPV HR and 16/18 genotyping tests for use in women with ASC-US cytology. Gynecology Oncology, 118:116–22.
- [11]. Eklund C, Zhou T, Dillner J, (2010). Global proficiency study of human papillomavirus genotyping. Journal of Clinical Microbiology, 48.
- [12]. Ermel A, Qadadri B, Morishita A, Miyagawa I, Yamazaki G, Weaver B, (2010). Human papillomavirus detection and typing in thin prep cervical cytologic specimens comparing the Digene Hybrid Capture II Assay, the Roche Linear Array HPV Genotyping Assay, and the Kurabo Gene Square Microarray Assay. *Journal of Virology Methods*, 169:154–61.
- [13]. Esere MO, (2008). Effect of sex education programme on at-risk sexual behavior of school-going adolescents in Ilorin Nigeria. African Health Science; 8(2):120–125.
- [14]. Esere A, Qadadri B, Morishita A, Miyagawa I, Yamazaki G, Weaver B, (2008). Human papillomavirus detection and typing in thin prep cervical cytologic specimens comparing the Digene Hybrid Capture II Assay, the Roche Linear Array HPV Genotyping Assay, and the Kurabo GeneSquare Microarray Assay. *Journal of Virology Methods*, 169:154–61.
- [15]. Hariri S, Unger ER, Sternberg M, (2011). Prevalence of genital human papillomavirus among females in the United States, the National Health and Nutrition Examination Survey, 2003–2006. *Journal of Infectious Diseases*; 204(4):566–573.
- [16]. Idso C, (2009). Sexually transmitted infection prevention in newly single older women: a forgotten health promotion need. *Journal* for Nurse Practitioners; 5(6):440–6.
- [17]. Kennedy NT, Ikechukwu D, Goddy B, (2016). Risk factors and distribution of oncogenic strains of human papilloma virus in women presenting for cervical cancer screening in Port Harcourt, Nigeria. The Pan African medical journal; 23:8510.11604.
- [18]. Nejo, Y.T, Olaye, D.O, and Odaibo, G.N, (2018). Prevalence and risk factors for genital Human Papilomavirus infection among woman in southwest Nigeria. 6(1): 105-112.
- [19]. Satterwhite CL, Torrone E, and Meites E. (2018). Sexually transmitted infections among US women and men: Prevalence and incidence estimates. Sexually Transmitted Diseases, 40(3):187-193.
- [20]. Schiller JT, Castellsague X, Garland SM, (2012). A review of clinical trials of human papillomavirus prophylactic vaccines. Vaccine; 30 Supplement 5:F123-138.
- [21]. Thomas JO, Herrero R, Omigbodun AA, Ojemakinde K, Ajayi IO, Fawole A, Oladepo O, Smith JS, Arslan A, Munoz N, Snijders PJ, Meijer CJ, Franceschi S, (2004). Prevalence of papillomavirus infection in women in Ibadan, Nigeria: a population-based study. British Journal Cancer: 90:638–645.
- [22]. Winer RL, Lee SK, Hughes JP, Adam DE, Kiviat NB, Koutsky LA, (2006). Genital human papillomavirus infection: incidence and risk factors in a cohort of female university students. *American Journal of Epidemiology*, 157(3):218–226.

Abdulkadir B.. "B Scan of Orbit with Its Clinico-Surgical Correlation." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 11, 2019, pp 27-33.