Journal of Human Growth and Development
2012; 22(1): 34-40

ORIGINAL RESEARCH

TRANSMISSION OF ANOGENITAL WARTS IN CHILDREN AND ASSOCIATION WITH SEXUAL ABUSE

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Abstract

Introduction: the incidence of anogenital condyloma acuminata in children shows remarkable increase in the last two decades, as well as by its association with sexual abuse. However, this relationship presents controversies regarding the etiology of infection, which makes answering challenging, particularly in legal-ethical aspects. **Objective:** review of the literature about HPV transmission in children and relationship with sexual abuse. **Methods:** synthesis of data from the query to Journal Citation Reports (JCR-ISI), Medical Literature Analysis and Retrieval System Online (Medline), Scientific Electronic Library Online (Scielo) and the Latin American and Caribbean Health Sciences (Lilacs). MeSH descriptors were used Terms with syntax (HPV [All Fields]) AND ("Child Sexual Abuse," [MeSH Terms]). Relevant articles between 1989 and 2009 were selected. Legal-ethical aspects were consulted in the criminal law and Brazilian Child and Adolescent Statute. **Results:** different forms of non-sexual transmission of HPV in children are documented, including the vertical transmission, self and hetero inoculation of cutaneous warts, and acquisition by instruments. Studies are conflicting as to the applicability of HPV-DNA to differentiate the sexual transmission and not sexual anogenital warts. **Conclusion:** evidence indicates that in children under two years non-sexual transmission of HPV should be strongly considered in the absence of genital lesions, another STD, or consistent history of abuse. The likelihood of association between HPV and sexual abuse increases directly with age, especially after five years.

Key words: papillomavirus infections; child sexual abuse; condylomata acuminata; infectious disease vertical transmission; domestic violence.

INTRODUCTION

Sexual abuse of children and adolescents is a worldwide phenomenon, both in developed and in developing countries[1]. Despite the high underreporting that this type of crime involves, 105 thousand cases of young women victims of sexual violence with non-fatal injury, with 27 000 cases between 10 and 14 years of age were conducted in the U.S. between 2004 and 2006[2].

It is estimated that 12% to 25% of girls and 8% to 10% of boys suffer some form of sexual abuse up to 18 years of age[3]. Girls are at greater risk of facing the problem, while boys are more likely to hide its occurrence[4]. The author of the sexual violence is generally known to the child and close family, sometimes responsible for its protection, often an expressive number of cases are performed by the biological father, grandfather or stepfather[5]. Less than 10% of children suffer sexual abuse by unknown[6].

Children who are sexually abused often have different health problems or changes in behavior, without necessarily reporting the violence. Bleeding or genital pain, vaginal discharge, early pregnancy, recurrent urinary tract infection, abdominal pain without apparent cause, recurrent headache, enuresis, encopresis and chronic constipation are some conditions which, although not specific, are associated with sexual abuse[3].However, in mostconfirmed cases of child sexual abuse, clinical examination findings are normal or nonspecific[1,7,8].

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Suggested citation: Jefferson D et al. Transmission of anogenital warts in children and association with sexual abuse. J. Hum. Growth Dev. 2012; 22(1): 34-40.
Manuscript submitted Apr 08 2011, accepted for publication Sep 10 2011.

The diagnosis of a sexually transmitted disease (STD) in a child agrees with the complaint of sexual abuse, and motivates a research when there is no such claim[9.10]. Some authors consider the diagnosis of a STD in childhood enough to requireevaluation to exclude sexual abuse[11]. Most studies agree, however, that the isolated presence of a STD is insufficient to ensure its occurrence[9].

The risk of STD acquisition depends on the type of molestation and is unlikely to occur when there is no direct physical contact with the author [5.12]. Its prevalence among children who suffer sexual violence has significant variability. Neisseria gonorrhoeae infection can range from 2.8% to 28%, the finding of Chlamydia trachomatis from 1.2% to 8%, Trichomonas vaginalis 1% to 6%, Treponema pallidum between 0.1% and 1.5 %, and herpesvirussimple 0.1% 0.5%[13.14]. About 60% of children with these STI have normal or nonspecific findings in anogenital examination[15].

The incidence of anogenital condyloma acuminata in children has increased markedly over the past two decades as the interest in its association with sexual abuse[14]. Anogenital warts induced by HPV can be transmitted by different forms of sexual abuse, either by oral-genital contact, genital-genital, genital-anal or digital manipulation of the child´s vagina and/or anus[16].

This relationship remains controversial and not always easily established, which makes the care of these cases challenging to the health professional, particularly with regard to ethical and legal consequences. Many health professionals do not feel safe to care for children with complaints of sexual abuse, or do not consider themselves capable of performing the clinical examination in these circumstances, particularly because of the limited knowledge about the dynamics of sexual abuse[9].

However, most studies argues that HPV infection does not necessarily result from sexual contact and that other forms of contamination should be considered[17]. Knowledge about the natural history of HPV infection and its mechanisms of transmission, as well as factors related to sexual abuse of children, are necessary for the proper handling of these cases. Thus, the objective is the review of related literature on mechanisms of HPV transmission in children and relation of infection with sexual abuse.

METHODS

Review of literature in the form of articles and data synthesis. Databases were consulted in the Journal Citation Reports (JCR-ISI), Medical Literature Analysis and Retrieval System Online (Medline), Scientific Electronic Library Online (SciELO) andthe Latin American and Caribbean Health Sciences (LILACS). The search strategyused DeCS / MeSH with the syntax (HPV [All Fields]) AND ("Child Abuse, Sexual"[MeSH Terms]). The search result found clinical trials on the mechanisms of HPV transmission in children and association with sexual abuse.Additional ethical and legal aspects were consulted in the Brazilian Penal Code and Child and Adolescent/Statute.

RESULTS

We identified and selected 21 articles indexed to the descriptors in the following databases from 1989 to 2009 that dealt with clinical trials of HPV transmission in children and association with sexual abuse. The summaries have been grouped into two tables, organized by the aspect of research: mechanisms of transmission of HPV (Table I) and the association between HPV and sexual abuse (Table II).

DISCUSSION

HPV can infect both skin and mucous membranes. Most infections regress spontaneously within two years in people with preserved immunity. More than 130 subtypes of HPV are identified so far, although only about a third of them can infect the anogenital mucosa, oral or laryngeal, prevailing subtypes 6, 11 and 16[38]. HPV infection is one of the most common STD in sexually active adults[39]. Approximately10% of the adult population has clinic genital lesions induced by the virus[10].

There is consistent evidence in literature associating HPV infection in cases involving sex crimes. In adult women who suffer sexual violence, its incidence ranges between 2% and 40%[40-42]. A longitudinal study, controlled and randomized, with 665 American young women found that those with a history of sexual abuse in the past 12 months were 4.5 times more likely to test positive for HPV 16 or 18 in the cervix, suggesting sexual abuse as a risk factor for infection by HPV[37].

Study with 42 prepuberal children with anogenital warts found that 28.6% acquired HPV by vertical transmission, 7.1% resulted from self inoculation of warts located on the hands, and only 4.8% were associated with sexual abuse. The mode of transmission of HPV was considered inconclusive in 59.5% of the studied children, being not possible to relate the infection to sexual abuse[20].

The maternal history of vulvar condyloma or cervical squamous intraepithelial lesion at time of delivery was observed in 50.3% of children under nine years who presented different clinical forms of the disease[30]. A similar finding was for HPV-induced lesions located in the oral cavity of children with frequent maternal history of vulva condyloma during pregnancy[25].

Vertical transmission commonly occurs during vaginal delivery in women with HPV, even if there are no clinical signs of the disease. Less commonly, the

Table 1: Summary of articles on the transmission of HPV in children

Author	Year	Summary
Rogo KOet al.[18]	1989	HPV transmission in cesarean section with intact membrane, suggestingtransplacentaltransmission.
Obalek S et al.[19]	1990	High frequency of cutaneous HPV subtypes in children with anogenital wartssuggests self inoculation.
Handley J et al.[20]	1993	HPV 6 and 11 and skin viruses 1 and 2 in anogenital lesions of children suggest non-vertical and non-sexual transmission.
Cason J et al.[21]	1995	Disagreement of 31% between maternal and newborn HPV DNA indicates another source of postnatal contact.
Smith EM et al.[22]	1995	Tested positive for HPV DNA in oral swab samples of newborns in 4% of the samples.
Puranen M et al.[23]	1997	Pairing of HPV DNA between mother and newborn with 43 % of disagreementpoints non-vertical postnatal contact .
Frazier L[24]	1998	Latency period of HPV in children up to 24 months. Identification of HPV 1, 2 or 3 inanogenital lesions.
Kui LL et al.[25]	2003	HPV 16 or 18 in 55% of oral lesions in children, not necessarily in agreement with maternal subtype.
Myhre AK et al.[26]	2003	Positive PCR for HPV in 3.0% of genital samples from healthy girls without history of sexual abuse.
Cheung PC et al.[27]	2004	Colposcopic findings can not differentiate form of transmission of anogenital warts in children.
Simmons KJ et al.[28]	2005	Latency period of 3 to 5 years in children with laryngeal papillomatosis induced by HPV.
Marcoux D et al.[29]	2006	HPV DNA did not define type of transmission of anogenital warts in children. Low frequency of HPV 7 and 57.
Jones V et al.[30]	2007	HPV vulvar or maternal cervical intraepithelial neoplasia in 50.3% of births of children with anogenital lesions.

Table 2: Summary of the articles about association of HPV with children sexual abuse

Author	Year	Summary
Derksen DJ[31]	1992	About 70% of cases of anogenital warts in children is associated with sexual abuse.
Slaughter L et al.[32]	1992	Colposcopy increases 10 times the diagnostic sensitivity of minimum genital trauma resulting from sexual abuse.
Handley J et al.[20]	1993	Sexual abuse found in 4.8% of children with anogenital warts. Inconclusive transmission in 59.5%.
Gutman LT et al.[33]	1994	Presence of HPV 6, 11 or 16 in 33% of sexually abused girls.
Siegfried E et al.[10]	1998	Identification of HPV 16 in 5% of girls between 1 and 16 years with history of sexual abuse.
Stevens-Simon et al.[34]	2000	Prevalence of 16% of HPV DNA in girls from 5 to 12 years with normal physical examination and sexual abuse confirmed.
0Jesus LE et al.[35]	2001	Confirmation of sexual abuse in 71% of children with anogenital warts through interdisciplinary assessment.
Kui LL et al.[25]	2003	Sexual abuse occurred in 22% of children with genital HPV lesions submitted to medical and social evaluation.
Sinclair KA et al.[36]	2005	Anogenital and laryngeal lesions have predictive value of 36% for 4-8 year-old sexual abused children and 70% over 8 years
Jones Vet al.[30]	2007	Prosecutions confirm the sexual abuse of 2.3% of children with HPV-induced lesions.
Wingood GM et al.[37]	2009	4.5 times more likely of HPV 16 or 18 in cervical cancer when there is sexual abuse in the last 12 months.

contamination of the newborn in women undergoing cesarean section, seems independent of the membrane integrity, possibly via ascending transplacental[18]. The possibility of HPV transmission to the fetus remains poorly understood, although there are inferences that it can occur via hematogenous, via the semen during fertilization, or maternal ascending, as it occurs in other infections during pregnancy[16].

The investigation of the presence of HPV DNA in oral swab samples of newborns has shown conflicting results, ranging from 4% to 87%[16,22]. In addition, the HPV detected in the newborn may be different from that identified in the mother between 31% and 43% of the cases[22, 23]. This pairing divergence allows us to assign to the postnatal infection other source not associated with sexual abuse[16].

The appearance of anogenital warts in the first years after birth can be justified by the long latency period of HPV in cases of vertical transmission. Similar condition can be observed for perinatal infection by Chlamydia trachomatis, which may last two to three years after birth[43] . Most of the studies limits the period within 24 months ofbirth, although fewer investigations suggest longer periods of three to five years,based on incubation of laryngeal papillomatosis [24.28]. This hypothesis, however, conflicts with the results that the average age in laryngeal HPV infections were lower than that found in anogenital lesions[36].

In addition to the incubation period, the child's age at the time of appearance of lesions has important relationship with a diagnosis of sexual abuse. Study of 124 children with anogenital warts, oral or laryngeal, found an average age of 6.5 ± 3.8 years for children victims of sexual abuse, compared with 3.6 ± 2.3 years in cases where the abuse was dismissed[36].

The predictive value of HPV-induced lesions in cases of sexual abuse was 36% for children between four and eight years old, and 70% in over eight years. The results suggest that the probability of HPV infection may be due to the sexual abuse increases significantly with the child´s age. At the same time, anogenital warts in the first two years of life are associated with other methods of transmission, other than sexual contact [36].

The relationship between tests to identify the subtype of HPV DNA and sexual abuse of children shows mixed results. Among 40 young women raped with age between one and 16 years, was found 5% of positive PCR tests for HPV 16[10]. In another study, HPV DNA was detected in the genital tract of 16% of abused girls aged between five and 12 years, with normal physical examination, while no cases were observed in those in which sexual violence was not proved[34]. In another group of confirmed sexual abused girls, HPV subtypes 6, 11 or 16 were observed in 33% of the cases[33]. On the other hand, HPV DNA was detected in genital swabs for 3.0% of healthy girls and no history of sexual abuse[26].

Some authors argue that the identification of HPV viral subtype can add information about the transmission of anogenital warts in children[17]. However, the diagnosis of HPV DNA are insufficient to differentiate safely vertically transmitted injuries of those resulting from sexual exposure[24]. Although DNA- HPV 16 or 18 was found in 55% of warts located in the oral cavity of children, pairing between mother and son do not necessarily show consistent, avoiding to associate these cases with the vertical transmission[25]. Study with 42 children detecting HPV DNA was associated both with the possibility of vertical transmission, such as through sexual contact[20]. In another study of 72 children under 12, patients with anogenital warts, was not possible to establish the mode of warts transmission based only on the identified HPV subtype[29].

Anogenital warts in children can also be caused by HPV 1, 2 or 3, usually found in skin warts, indicating the possibility of self inoculation or hetero inoculation practiced without intent for parents and/or other caretakers[19, 20.24]. In addition, anogenital warts in children may be associated with both HPV 6 and 11 as the cutaneous HPV 1 and 2, indicating the likelihood of transmition different from vertical and sexual[44]. HPV7 or 57 infections in chidren show similar difficulty of interpretation, although affect significantly fewer cases[29.]

Evaluation of colposcopic anogenital warts does not allow to differentiate those arising from sexual abuse from the ones transmitted by other mechanisms. However, the test proves effective for diagnosing and documenting minor injuries to the anogenitalmucosa resulting from sexual abuse, difficult to identify during the clinical examination[27.45]. In such cases, the use of colposcopy increases by almost ten times the minimum identification of traumatic injuries, assisting in documentation and in the process of evidence of sexual abuse[32].

The relationship between sexual abuse and HPV infection has variable outcome in literature, concluding with an occurrence between 0.1% and 70% of children with genital warts[30,31,46]. Different methods of investigation of the circumstances of sexual abuse and child assessment influence the results. A retrospective study of children between six months and nine years of age found 2.3% of sexual abuse when subjected to evaluation by the judicial system[30]. In other experiments, confirmation of sexual abuse has reached 22% when associated medical and social assessment, and 71% when applied interdisciplinary research[25.35].

Most authors consider that the diagnosis of sexual abuse depends crucially on the quality of history, the experience of professionals involved in assessment and examination of the child[47]. However, there are reports that another etiology of genital warts can be confused with those caused by HPV. Considering the possible legal ramifications, it is appropriate to confirm infection with biopsy of typical or suspicious lesions, or to use another diagnosis method valid for HPV[36.48].

The American Academy of Pediatrics considers sexual abuse as physical genital contact, oral or anal sex with the child, including actions that do not involve direct relationship with the abuser, such as exhibitionism, voyeurism and pornography[12].Most sex crimes against children, however, does not involve acts of vaginal or anal penetration, which makes the probative material evidences uncommon[7].

In Brazil, criminal law typifies the crimes of rape and rape of vulnerable in Articles 213 and 217-A, respectively. The law criminalizes any sexual act done without the victim's consent with the use of force or psychological intimidation, or sexual acts committed against children under 14 years old, sick people, mentally ill or any condition that prevents valid consent or offering resistance[49].

Articles 13 and 245 of the Estatuto da Criança e do Adolescente(ECA), Law nº 8069, 1990, establish a legal duty of health professionals to communicate the Council of Guardianship suspected or confirmed cases of abuse against children and adolescents under 18 years, including sexual abuse[50]. This action is fundamental to the protection of children and adolescents in a vulnerable position, interrupting the cycle of sexual violence, often within the family[5].

However, the Law does not establish which situations are considered suspected or confirmed of sexual abuse, no criteria or procedures that must be adopted by health professionals for this diagnosis[50]. Thus, it is for health professionals carefully evaluate whether there are consistent grounds to proceed with the communication to the protection agency.

This evaluation should not be precipitated by external factors and, where possible, should incorporate interdisciplinary actions involving physicians, psychologists and social assistants[5]. Health professionals should know the possible ways of transmission and incubation periods of HPV-induced anogenital and oral warts, to ensure adequate assistance to the child and consider the need to guide and enlighten the parents or legal guardians[47].

Although there is no consensus on how to proceed in those cases, the high prevalence of non-sexual transmission of HPV in children under two years old makes sexual abuse unlikely, though can not be decisively away. It is debatable whether the isolated presence of oral oranogenital warts in children under two years should necessarily be communicated to the government services ofchildren protection with suspicion of sexual abuse. It is exceptwhen associated with abnormalities on physical examination, presence of other STD,change in the child´s behavior, or if sexual abuse is suspected or confirmedby parents or other caretaker or guardian[51].

After the child is two or three years old, the communication to the competent authority and the sexual abuse investigation areconsidered necessary measures against anogenital and oral lesions induced by HPV. The conduct is based on the greater likelihood of association with sexual abuse found in different studies, even when there is no complaint by the parents or in the child's narrative[51].

The association between oral and anogenital warts induced by HPV and sexual abuse of children reserve contradictory aspects. Different forms of non-sexual transmission of HPV in children are documented. Evidence suggests that in children under two or three years, non-sexual transmission of HPV should be strongly considered, since there is no other concomitant STD, history of sexual abuse, evidence of genital trauma or ruptured hymen.

In children above that age the likelihood of sexual abuse becomes greater. However, in any age of child sexual abuse can be definitely ruled out. Studies are conflicting regarding the applicability of methods for identification of HPV subtype to differentiate sexual and nonsexual transmission forms of anogenital and oral warts in children. The genitoscopia and anoscopy also do not differentiate the type of transmission by theappearance of the lesions. However, these methods have shown better sensitivityand higher specificity for identifying genital and perianal minor injuries resulting from sexual abuse.

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