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Human papillomaviruses in anogenital warts in children: typing by in situ hybridisation

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Abstract

Objective—To identify the types of human papillomaviruses found in anogenital warts in children and to relate these to clinical and social information.

Design—In situ hybridisation using biotin labelled DNA probes to 11 types of human papillomavirus was performed on biopsy specimens from 17 children with anogenital warts.

Setting—Nuffield department of pathology and the department of dermatology, Oxford.

Patients—Children in one group were referred by general practitioners or paediatricians to the dermatology department, where biopsies were performed. The other children were seen in four different hospitals, and biopsy specimens were submitted to the laboratory at the physician's or pathologist's request.

Results—Of the 17 biopsy specimens, 10 contained cells positive with a probe to a genital human papillomavirus type (types 6 or 11), while six were positive with a skin virus type (types 2 or 3). One was negative. The virus type present bore no relation to the site or appearance of the warts. The virus type did, however, appear to correlate with groups of children. Skin types were commoner in older children (over 4 years), in those with a relative who had skin warts, and in children with warts elsewhere; there was no relation with the child's sex and no suspicion of sexual abuse in these children. These circumstances suggested non-sexual transmission, such as autoinoculation. In contrast, genital types were commoner in girls, in children under 3 years, in children with relatives with genital warts, and in those with no warts elsewhere. Nevertheless, there was suspicion or evidence of sexual abuse in only half these children, suggesting that other routes of transmission—for example, perinatal—might have been implicated.

Conclusion—Anogenital warts in children may contain either skin or genital wart virus type. Although the type of human papillomavirus present

may give some indication of the likely mode of transmission, this can be interpreted only in conjunction with all available clinical and social information. The type of virus does not provide proof of the presence or absence of sexual transmission.

Introduction

There has recently been a massive increase in the number of human papillomavirus types characterised by DNA technology, but some specific types are repeatedly found in certain sites and lesions. Thus in adults types 6 and 11 are found almost exclusively in anogenital warts,^{1,5} whereas types 1, 2, 3, and 4 are found in skin warts.^{6,7} This finding, in conjunction with epidemiological evidence, indicates that human papillomavirus types 6 and 11 are venereally—that is, sexually—transmitted. By extrapolation, it has been suggested that anogenital warts in a child may also be sexually transmitted and thus provide grounds for suspecting sexual abuse.

In the United States there is therefore a tendency to start social and criminal investigations for possible sexual abuse in all children with anogenital warts,^{8,10} although this has not been advocated in Britain.¹¹ Some have, however, proposed that the identification of a genital human papillomavirus type in anogenital warts in a child would support the possibility of sexual transmission.^{11,12} This assumes that the tissue distribution of human papillomavirus types in children is the same as in adults. Very little data exist to support this assumption and those data conflict. Three studies have reported a total of 23 patients under 13 years old.¹³⁻¹⁵ In 18 the anogenital warts contained one or more of the genital human papillomavirus types. In contrast, Fleming *et al* reported a boy of 5 years with human papillomavirus type 2 in both hand and perianal warts,¹⁶ and Androphy recently reported unpublished observations in which 25% of childhood anogenital warts contained type 2.¹⁷ Thus, the correlation of human papillomavirus type with anatomical site may

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not be as strong in children as in adults and anogenital warts in children may not necessarily be sexually transmitted. Further data are needed about the human papillomavirus types occurring in anogenital lesions and their relation to possible sexual abuse.

We studied biopsy specimens from 17 children with anogenital warts using in situ hybridisation with DNA probes complementary to both skin and genital human papillomavirus types to ascertain which types were present and at what frequency. We related this to clinical information to try to determine the routes of transmission that can occur.

Patients and methods

Patients—Twelve patients were seen in the dermatology department in Oxford and five in other hospitals. Material was submitted to us for in situ hybridisation at the physician's request. Of the 12 Oxford children, 10 were referred directly to the dermatology department because of the presence of genital warts. In nine of these a low index of suspicion of sexual abuse was based on the opinion of the general practitioner and health visitor, assessment by a paediatrician in five cases, and no isolation of venereal pathogens in vaginal, anal, and throat swabs. In one child sexual abuse was regarded as possible but could not be confirmed. One child was referred via the social services department and the paediatric department for treatment, having already been removed from her family because of encopresis and other behavioural disturbance and an admission of sexual abuse by the father. Another child was suspected of being abused, and his sister was in care because of previous sexual abuse. The other five children were seen in paediatric, dermatology, or gynaecology departments in other districts or regions. Sexual abuse was suspected in two but there was no evidence of abuse in the other three.

Methods—Biopsy specimens were placed in formalin and routinely processed and embedded in paraffin wax. Sections were stained with haematoxylin and eosin or were submitted for in situ hybridisation. The method for in situ hybridisation was non-isotopic using biotinylated probes labelled by nick translation and an avidin-alkaline phosphatase detection system as previously described.^{18,20}

Probes—These were double stranded DNA probes received as gifts (see acknowledgements). Each probe was cut with appropriate restriction enzymes. In each case the restriction enzyme pattern on gel electrophoresis corresponded with the published pattern. A

negative control of pBR 322 was always used. A positive control was also always run with each test; this consisted either of a section of a skin wart known to contain human papillomavirus type 2 or the Y chromosome repeat sequence probe pHY 2.1 on male and female tissue sections.²¹

Results

All the biopsy specimens showed histological features of viral warts with papillomatosis, acanthosis, and intermittent parakeratosis. Changes suggestive of human papillomavirus infection (binucleation, nuclear enlargement, and irregularity and clearing of cytoplasm) were patchy in all the lesions but dysplasia was not present in any case. Keratinisation was present in all of the lesions except two (cases 10 and 16).

The results are summarised in the table. There were six boys and 11 girls. Human papillomavirus types 6 or 11 were present in the warts from 10 children, three boys and seven girls. Human papillomavirus types 2 or 3 were present in six, three boys and three girls. The vulval warts in one child were negative (case 1), and in another girl the perianal warts were negative but were regressing at the time of biopsy whereas similar warts on her buttocks and hands contained human papillomavirus type 2 (case 2).

Of the 10 patients in whom human papillomavirus types 6 or 11 were present, seven were 3 years old or under. In contrast, human papillomavirus types 2 or 3 were found only in children aged 4 years or more (although the age of onset of the warts was less in two cases).

Lesions described as dry common warts and moist fleshy condylomas were found to contain either genital or skin human papillomaviruses. Thus the appearance of the anogenital warts did not correlate with the human papillomavirus type and nor did the site. There was, however, some relation between the human papillomavirus type found in the child's anogenital warts and the presence and site of warts in relatives. Hand warts were the commonest warts present in relatives (six cases) and in four of these cases the child's anogenital warts contained a skin type (type 2 or 3). In three cases a relative currently had genital warts. In two of these abuse was thought to be possible and the child's genital warts contained human papillomavirus type 6. In the other case the warts contained human papillomavirus type 3, abuse was thought to be unlikely, and the child herself and other family members had plane warts elsewhere.

Clinical details of 17 children with anogenital warts, presence of warts in relatives, and type of human papillomavirus found

Case No	Sex	Age (years)		Anogenital warts		Site of warts		Relatives with warts		Suspicion of abuse	Type of virus in child
		Presentation	Onset	No	Appearance	Anogenital	Other	Relative	Site		
1	F	9	8.5	>20	Moist, fleshy	Vulval	Foot	None		No	Negative
2	F	7	4.5	>20	Moist and common	Perianal (buttock)	Hand, foot and buttock	Brother	Hand	No	Negative and 2
3	M	9	4	>20	Moist	Perianal	None	None		No	2
4	F	5	3	8	Common	Perianal	None	Mother	Hand	No	2
5	M	5	4	>20	Moist	Perianal	Hand and face	None		No	2
6	M	7	1.5	>20	Fleshy mass	Perianal	None	Mother	Hand	No	2
7	F	6.5	6	6	Plane	Vulval	Face and thigh	All family	Plane warts	No	3
								Father	Genital		
8	F	3	1	3	Dry, common	Natal cleft	None	Aunt	Genital	Possible	6
9	F	3	3	11	Moist	Perianal	None	Unknown		Confirmed	6
10	F	1	0.5	>20	Fleshy mass	Vulval and perineal	None	None		No	6
11	M	3	2.5	>20	Dry	Perianal and scrotal	None	None		Possible	6
12	F	8	8	12	Dry, fleshy	Vulval and intravaginal	None	None		No	6
13	F	7	7	3	Moist	Vulval	None	Mother (in past) and partner	Genital	Possible	6
14	M	4	3	10	Dry, common	Perianal and scrotal	None	Sister (in past)	Hand	Probable	6
15	M	3	3	>20	Dry	Perianal and perineal	None	None		No	6
16	F	2	2	3	Moist	Vulval	None	Mother (in past)	Genital	No	11
17	F	2	2	1	Dry	Perianal	None	Father	Hand	No	6 and 11

In the children themselves warts were present in other than the anogenital area in four cases. The anogenital warts in two of these were negative for human papillomaviruses (cases 1 and 2), but in both of the others (cases 5 and 7) contained skin human papillomavirus types. One of the children (case 5) had the same virus type in his hand warts as in his anogenital warts, but none of the other lesions were tested. In case 2 the hand and buttock warts contained human papillomavirus type 2, while the perianal wart was negative (but regressing at the time of biopsy). None of the children with genital human papillomavirus types had warts elsewhere.

The signal was strong in sections of all the biopsy specimens despite variation in the time of formalin fixation between two days and several weeks. Cross reactions were seen at low stringency ($T_m-30^\circ\text{C}$) between probes for human papillomavirus 2 and 3 and between probes for human papillomavirus 6 and 11 and at medium stringency ($T_m-17^\circ\text{C}$) between those for human papillomaviruses 6 and 11. In paraffin sections using DNA probes cross hybridisation is thought to be likely at higher stringency than under other conditions especially between types 6 and 11.²² At very high stringency ($T_m-7^\circ\text{C}$) one human papillomavirus type probe alone remained positive, staining with the other human papillomavirus type probes being eliminated in all but one case.

Discussion

Three recent studies have reported genital human papillomavirus types (6, 11, 16, and 18) in anogenital warts in children,¹³⁻¹⁵ which, by extrapolation from adults, supports the assumption that most of these warts are sexually transmitted. By contrast our study showed that such warts contained a human papillomavirus type usually associated with skin warts in six out of 17 cases, suggesting that non-venereal transmission may be common.

One explanation of the difference might be the range of probes that we used, since previous studies using *in situ* hybridisation to type human papillomaviruses in anogenital warts have tested only for the genital types.¹³ Indeed there have been few studies of human papillomavirus types in anogenital warts which have used both skin and genital type probes.^{3,15}

Another explanation may be that the technique we used was incapable of accurately distinguishing between human papillomavirus types. The probes we used all conformed, however, to the published data on restriction enzyme patterns for the particular human papillomavirus type. Furthermore, positive and negative controls worked appropriately with each batch of staining, and hybridisation did not occur with both skin and genital human papillomavirus probes in the same specimen. We therefore think it unlikely that our technique failed to discriminate clearly between human papillomavirus types. This being so, it is clear that anogenital warts in children do not necessarily contain genital human papillomavirus types.

Our data suggest that clinical examination is poor at predicting the human papillomavirus type present. Thus while the plane warts in one case contained human papillomavirus type 3 (as expected), in all the other cases no correlation was found between clinical appearance and human papillomavirus type. Both moist and dry anogenital warts contained either skin or genital human papillomavirus types. We therefore suggest that it is unwise for a doctor seeing anogenital warts in a child to predict the human papillomavirus type on the basis of the site or appearance of the warts alone.

From our data we cannot deduce whether anogenital warts containing genital human papillomavirus types

are sexually transmitted while those containing skin types are not. The number of cases in this study was small, but the results suggest the presence of two groups of children, one with anogenital warts containing skin human papillomavirus types and one with anogenital warts containing genital papillomavirus types. Warts containing skin types occurred in older children and equally in boys and girls and were associated with warts elsewhere on the child and with hand warts in relatives. There was no evidence or suspicion of sexual abuse. Thus skin human papillomavirus types occurred in circumstances favouring non-venereal transmission. Autoinoculation and normal close family contact are both possible routes of transmission. In contrast, children with anogenital warts containing genital human papillomavirus types were more likely to be girls, to be aged 3 years or under, and to have a relative with genital warts. Suspicion of sexual abuse was present in half. In none of these children were there warts elsewhere on the body. Thus some of the cases of anogenital warts containing genital human papillomaviruses occurred in circumstances favouring sexual transmission. Others, however, occurred in the absence of such evidence, and in view of the young age of many of these children perinatal transmission must be considered likely. It is widely accepted that laryngeal papillomas, which are caused by human papillomavirus type 11, are transmitted during passage through the mother's infected birth canal. Laryngeal papillomas most commonly occur in children up to the age of 5 years.²³⁻²⁵ Thus, although the finding of a genital human papillomavirus type in anogenital warts in a child may support sexual transmission, it does not provide proof. Equally, the presence of a skin human papillomavirus type does not disprove sexual transmission since these have occasionally been reported in anogenital warts in adults,²⁶ and transmission of skin human papillomaviruses could also occur from the hand of an adult during sexual abuse.

In conclusion, we agree with Oriel that the presence of anogenital warts in a child is by itself not a marker for sexual abuse.¹¹ In contrast to previous published studies in children our results indicate that anogenital warts may contain either skin or genital human papillomavirus types. Typing of human papillomaviruses in anogenital warts in children may thus be helpful as part of the investigation, but the results must be interpreted only in conjunction with all available clinical and social information. The type of papillomavirus should not be regarded as providing definite evidence of either the presence or absence of sexual transmission.

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Control of defecation in patients with spinal injuries by stimulation of sacral anterior nerve roots

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Abstract

Objective—To observe the effects of stimulation of the sacral anterior roots on anorectal and low colonic pressures and to programme implanted stimulators to produce defecation.

Design—Prospective study of 12 consecutive patients.

Setting—Spinal injuries unit and university gastrointestinal physiology department.

Patients—12 Patients with complete supraconal spinal cord lesions. Their injuries had been sustained at least two years before the study.

Interventions—A Brindley-Finotech intradural sacral anterior root stimulator was implanted in all patients. Three months postoperatively the stimulator settings were adjusted after measurement of simultaneous anorectal and low colonic pressures.

Main outcome measures—Full defecation.

Results—Six patients achieved complete rectal evacuation of faeces using the implant and subsequently did not require manual help for defecation. For all but one of the patients the total time taken to complete defecation was reduced, and all were free from constipation, the most prevalent gastrointestinal symptom in patients with spinal injuries.

Conclusions—Sacral anterior root stimulators can be programmed to achieve complete unassisted defecation and can considerably improve the quality of life of patients with spinal injuries.

Introduction

Spinal cord injuries often occur in fairly young people, who have the prospect of an almost normal life expectancy but a considerably impaired quality of life. These patients not only experience severe dysfunction of voluntary movement but also have impaired bladder, bowel, and sexual function.

Urinary problems in patients with spinal cord injuries have been extensively studied, and with the advent of intermittent self catheterisation,¹ electrical stimulation

of the bladder,² and advances in diagnostic techniques considerable improvements have been made in managing lower urinary tract and renal function. In contrast, the management of bowel disorders and, in particular, the intractable constipation that is so common in these patients has remained essentially unchanged over the past two decades.

The first sacral anterior root stimulator was implanted in a patient with a spinal injury in 1976.^{3,4} Since then roughly 300 patients throughout Europe have had such devices implanted, with much success in managing the neurogenic bladder. The stimulator was initially developed to improve bladder emptying,^{2,5} but as the parasympathetic and somatic nerves that supply the distal colon, anorectum, and anal sphincter are all derived from the same sacral spinal roots that are used for electromicturition it seemed likely that the stimulator could also be used to induce defecation in paraplegic patients. Little has been reported regarding the action of the stimulator on colorectal motility,⁶ and there are few reports of full evacuation with this technique.^{7,8}

We report clinical and manometric data on 12 consecutive patients with spinal injuries who had sacral anterior root stimulators implanted.

Patients and methods

PATIENTS

We studied 12 consecutive patients (three women and nine men aged 21-49 (mean age 33)). All had received traumatic spinal injuries that had resulted in complete supraconal cord lesions (10 thoracic, two cervical). Sacral anterior root stimulators were not implanted in patients until at least two years after their injuries occurred to allow for any neurological recovery. The time since implantation ranged from three months to six years (mean time 26.3 months), and all patients included in the study were using the stimulator effectively to empty the bladder.

After surgery for implantation we assessed evidence

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