Pediatrics

Warts (Verruca Vulgaris) and Molluscum Contagiosum in Children

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Non-genital warts in children are very common and can be troublesome to the child when they are large and disfiguring. Warts are usually painless unless they are located in deep skin layers and in areas of weight-bearing, such as the soles of the feet. The causative viral agent of non-genital warts is known as the human papillomavirus. The diagnosis is based on the clinical examination of the non-genital wart and management can be classified into a wait-and-see approach, destructive approach, or immunomodulation.

Overview

Non-genital warts are defined as focal epithelial lesions that are characterized by hyperplasia and are caused by the human papillomavirus. Non-genital warts, as the name implies, are located on body surface areas that are not part of the genital tract. The lesions can be raised and circular, flat, deep, or present as plantar cysts.

Epidemiology of Non-Genital Warts in Children

The estimated annual incidence of non-genital warts is around 3 to 5 cases per 100
children aged between 5 and 14 years. The prevalence of non-genital warts in school-aged children is quite high and is estimated to be around 33%. Approximately, 10 to 21% of dermatology visits by children are related to warts’ disease.

The most common mode of transmission of non-genital warts is prolonged skin-to-skin contact with someone who has a wart. Additionally, the use of skin-care equipment can also transmit the disease between children. Swimming pools and communal showers have also been considered as potential places where a child might acquire the causative viral agent, human papillomavirus, and develop non-genital warts.

Non-genital warts are more commonly seen in white children compared to African-American or Asian children. On the other hand, focal epithelial hyperplasia, also known as Heck disease, is more commonly seen in American Indians. There is no sex predominance. Non-genital warts are rare during infancy and early childhood.

**Etiology and Pathophysiology of Non-Genital Warts**

Non-genital warts are caused by the human papillomavirus which is associated with other abnormal epithelial growth disorders including squamous cell carcinoma. There are different types of the human papillomavirus and each has been associated with a specific type of non-genital warts.

Common warts, also known as verruca vulgaris, are usually caused by the human papillomavirus types 1-4, types 26 and 27, and type 41. Plantar cysts are usually linked to the same types. Flat warts are commonly associated with the human papillomavirus types 3, 10, 38 and 49, but other types seen with common warts can also cause flat warts.

Another skin lesion that resembles common warts is caused by a completely different virus known as Molluscum Contagiosum (MC). Skin lesions that are caused by MC typically have a round configuration with a central umbilication. The condition is self-limited and not associated with squamous cell malignant transformation.

These viruses encode for a variety of signal molecules that are responsible for the induction of uncontrolled epithelial cell growth and proliferation. The increased epithelial proliferation is usually associated with nuclear dysplasia and loss of the apical configuration of normal epithelial cells.

Human papillomavirus types 6, 11, 16 and 18 have been linked to an increased risk of malignant transformation, especially in immunocompromised patients.

**Clinical Presentation Non-Genital Warts in Children**

Patients can develop new non-genital warts by either autinoculation or by acquiring the causative viral agent from skin-to-skin contact with an infected individual or the use of shared equipment with someone who has warts. The incubation period for the human papillomavirus is quite long and can range from 1 to 6 months which explains why most patients do not recall being in contact with someone who has had non-genital warts at the time of presentation.

Once warts develop, the clinical presentation and the characteristics of the lesions are different depending on the classification of the non-genital wart.

Common warts are characterized by hyperkeratosis; they appear as papules, are
rough and have irregular surfaces. Their size can range between 1 mm to 1 cm. They are most commonly seen on the hands and knees of children.

Deep palmoplantar warts begin as small shiny papules that eventually develop into deep lesions that are round and are sharply defined with a rough hyperkeratotic surface. Because of the location of these lesions and due to their deep location, they can cause pain. They are found on weight-bearing areas, such as heels and metatarsal heads. On hands, they are found in periungual and subungual regions.

Flat warts can be flat or slightly raised, are usually smooth and mildly hyperkeratotic. These lesions are usually smaller than common warts and occur on the face, hands, and shins. Size varies from 1-5 mm and number may be few to hundreds.

**Heck disease** is characterized by buccal, gingival or tongue involvement in children. The lesions form small plaques that are typically less than 5 mm in diameter and are most commonly seen in children of American Indian origins.

Finally, patients who develop cystic warts present with a single nodule on the sole of their feet. The lesions may or may not be hyperkeratotic. These lesions are cystic and, if incised, a cheesy material can be expressed from the lesion.

**Diagnostic Workup for Non-Genital Warts in Children**

The diagnosis of non-genital warts is a clinical one and not dependent on laboratory findings. For research purposes, one can detect the human papillomavirus structural proteins from a biopsy taken from a lesion by immunohistochemical staining. HPV can be detected in younger lesions and cannot be found in old standing lesions. Viral DNA identification is also possible with polymerase chain reaction assays or Southern blot hybridization techniques.

The detection of the human papillomavirus and the sub-classification of the type of the virus can be helpful in the prediction of the risk of malignant transformation, especially in immunocompromised children.

Paring warts are useful in the confirmation of the diagnosis of common warts as it can reveal small black dots within a pale background, a sign that is suggestive of thrombosed capillaries.

If a biopsy is taken, which is rarely performed nowadays, the different histological patterns that can be seen are dependent on the type of the wart.

Common warts usually show digitated epidermal hyperplasia, acanthosis, and papillomatosis. Deep plantar warts appear like common warts, but are usually located deeper in the skin. Flat warts are identical to common warts histopathologically with the exception of more basophilic nuclei within the granular cell layer.

The main difference between common warts and Heck disease on histopathological examination is the presence of a thinner layer of keratin and intracellular edema in the latter.

The wall of cystic warts is composed of basal, squamous, and granular cells that are dysplastic and have large nuclei with eosinophilic cytoplasmic inclusion bodies.
Treatment of Non-Genital Warts in Children

Management options for non-genital warts include destructive methods, a wait-and-see approach, immunomodulation, antimitotic drugs against the increased epithelial cellular proliferation, and surgery. Treatment should be started with the least expensive, least painful and least time-consuming methods. Invasive procedures and expensive treatments should be preserved for refractory and large warts.

<table>
<thead>
<tr>
<th>Asymptomatic</th>
<th>Painful or cosmetically troubling</th>
<th>Plantar warts</th>
<th>Molluscum Bodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous resolution</td>
<td>• Salicylic acid</td>
<td>• Some advocate for debridement prior to topical therapy</td>
<td>• Disappear spontaneously after 6-12 months or sometimes longer</td>
</tr>
<tr>
<td></td>
<td>• Cryotherapy with liquid nitrogen</td>
<td>• Others apply topical salicylic acid only</td>
<td>• Topical antibacterial for ruptured lesions or scratched lesions to prevent secondary infection</td>
</tr>
<tr>
<td></td>
<td>• Duct tape</td>
<td></td>
<td>• Cryotherapy for older children</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• Avoid sharing towels, sheets</td>
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A wait-and-see approach is a reasonable option for children with common warts because, approximately, two-thirds of the cases will resolve spontaneously. However, when the warts are large and very disfiguring, treatment can be sought by the child or her or his caregivers.

The most common destructive therapy for non-genital warts in children is salicylic acid. Cryotherapy can be also used in children with non-genital warts with excellent results. Surgery should be reserved for difficult cases and where rapid resolution is required. Complete clearance of warts has been reported to be highest with curettage and electrodesiccation, followed by cryotherapy, and least successful with podophyllin. Laser therapy should be avoided in children because it is painful and can cause scarring.

Immunotherapy can be also used to treat non-genital warts in children. The most commonly used agents are cimetidine and imiquimod. Cimetidine should be used systemically, while imiquimod can be applied topically to the lesion.

Antimitotic drugs such as 5-fluorouracil and bleomycin should be avoided in children because they were not tested in the pediatrics’ population, and are associated with extensive skin damage, ulcer formation, and scarring. The antiviral cidofovir was used in a single study that involved only 7 children. While the antiviral resulted in complete resolution of the multiple hand and feet warts in four children within 12 weeks, generalization among other groups of children should not be attempted.

**Prognosis**

65% of lesions resolve spontaneously within 2 years. Recurrence is common in immunosuppressed patients and they are refractive to treatment.

Malignant changes are rare but are reported and the condition is known as verrucous carcinoma. It is a slow-growing locally destructive squamous cell carcinoma and can rarely metastasize.
References


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