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Different modalities in the treatment of verruca

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Abstract
Verruca (wart) is a common proliferation of skin and mucosa duo to human papillomaviruses (HPV) infection. They might be as little as a few millimetres or as large as several centimetres. They might be skin-colored or brownish-gray in hue. There is no need for further histologic or virologic tests because the diagnosis has been made clinically.
There isn't a particular antiviral treatment for HPV available right now, however, some medications on the market disrupt the viral life cycle. There are several wart therapies accessible, however there can often be inadequate backing for their benefit, whether they are taken alone or in combination.
Damage to the infected epithelium that ends up in cell death, antigen exposure, presentation, and an immune response is the most conventional kind of therapy. The wart should thin out as a result of diminishing epidermal proliferation, or more particularly DNA replication, which additionally ought to prevent the propagation of new viruses.
Although there is currently no entirely appropriate course of treatment that would ensure complete cure, without relapses, wart treatment is still exceedingly difficult in contemporary dermatology. The majority of treatment methods involve numerous uncomfortable procedures rather than concentrating on eliminating the viral agent that is the cause of the illness. The main justification for the continued use of traditional medical practises and cures is this. The part doctors play in the healing process is being reevaluated. Any medical practise should be built on a partnership between the doctor and the patient, where accountability is shared and decisions are made together.

Keywords: Practises and cures, immune response

Introduction
The growth of skin and mucosa due to human papillomavirus (HPV) infection is known as a verruca (Wart) [1]. They might be as little as a few millimetres or as large as several centimetres. They might be skin-colored or brownish-gray in hue. There is no need for further histologic or virologic tests because the diagnosis has been made clinically [2].
Causative organism is Human Papilloma Virus (HPV) are A 55 nm-diameter class of tiny, non-enveloped double-stranded DNA viruses. Over 170 HPV genotypes have been identified. Their genome consists of circular [3].
Cryotherapy, electrosurgery, curettage, intralesional bleomycin, CO2 laser therapy, topical cytotoxic drugs (5-fluorouracil), infrared coagulation, pulsed dye laser, and surgical excision are among the therapeutic possibilities. However, many lesions return after a successful course of therapy, and other lesions are still resistant to it. Considered a viable therapeutic approach for the management of viral verrucae lesions, topical photodynamic therapy (PDT) [4].

Incidence and Prevalence
Frequency
The prevalence of warts is prevalent across the world. Warts are thought to affect 7–12% of the population, while the precise prevalence is unclear. 10–20% of school-age children have the condition [5].

Race
Despite the fact that anybody can get warts, white people seem to get them about twice as often than black people or Asian people do [6].

Age & sex

Any age can experience a wart. They are uncommon in infancy and early childhood, become more common in school-aged adolescents, and reach their peak between the ages of 12 and 16. The ratio of men to women is around 1:1 [7].

Etiology

Causative organism is Human Papilloma Virus (HPV) are A 55 nm-diameter class of tiny, non-enveloped double-stranded DNA viruses. Over 170 HPV genotypes have been identified. Their genome consists of circular [3].

DNA double strand containing approximately 8000 nucleotide base pairs, associated with histones forming a structure that has been compared to a "minichromosome" [8] (Fig 1).

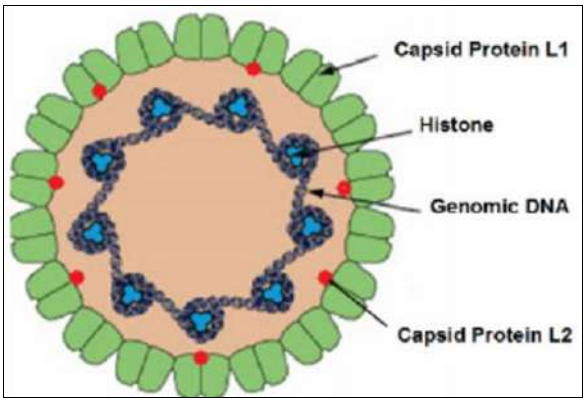

Fig 1: structure of human papilloma virus [9].

The cutaneous HPV types 1, 2, 3, 4, 7, 10, 27, and 57 are responsible for causing warts on the skin. HPV-6, 11, and sometimes HPV-16, 18, 31, 33, and 35 are responsible for anogenital warts. In children, exclusion of sexual abuse possibility is mandatory. Oral warts and HPV types 6 and 16 are related [10].

Incubation period

The incubation period might be anywhere from a few weeks and more than a year. Depending on the patient's immunological condition, they may grow in number and size or naturally diminish. Clinical warts typically take three weeks to eight months to develop, with average taking 2.9 months [11].

Mode & source of infection

A host must come into touch with virus particles through skin abrasions or macerations in order to get infected either by direct contact with the wart or indirectly through Floors, socks, shoes, towels, and sporting goods are examples of fomites. The HPV infection does not have a viremic phase or systemic spread [12].

HPV infection cycle & pathophysiology

Cutaneous warts can transmit from person to person directly or indirectly by contact with infected items or surfaces, with transmission being made easier by small tears in the epidermal barrier [1].

When the HPV virus makes contact with the host, it travels to the basal epithelial layer, which contains stem cells that are actively proliferating, and connects with cellular receptors. Viral DNA is formed inside the host cell without the host cell genome integrating during an incubation period of 1 to 20 months. Basal stem cells proliferate as a result of this process, and each one of them contains 20 to 100 copies of the viral DNA [13].

Basal cells move towards the epithelium's outer surface after differentiating into keratinocytes, hyper keratinization occurs and warty papule is formed. At the same time, the production of viral proteins is increased by triggering the viral genome promoter region. This leads to enhancement of viral genome amplification within the cell (Fig 2) [14]. On normal epithelium sloughing, virus particles are discharged and might potentially spread to other people [15].

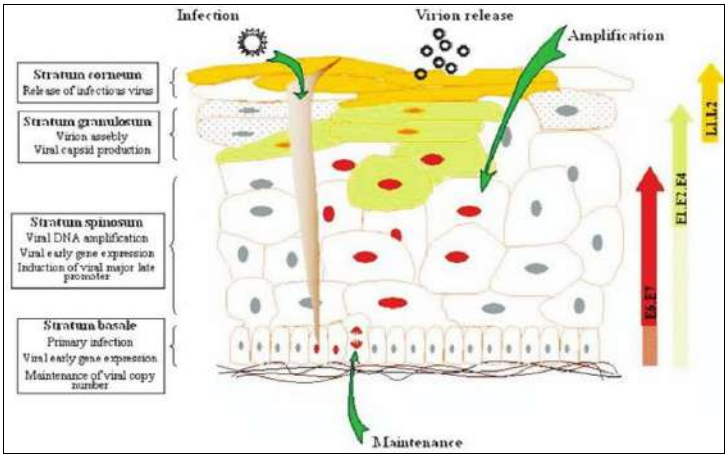

Fig 2: HPV infection cycle. Shown is the coordinated expression of the different viral protiens during the course of a reproductive infection [16].

Clinical Features

There are several different wart kinds that have been found, each with a unique form, place of involvement, and type of HPV involved. The following examples show that they are extracutaneous, anogenital, and cutaneous warts [17].

***Cutaneous manifestations* of HPV infections**

Common warts (Verruca vulgaris)

HPV 1, 2, and 57 are the causes of common warts. They are hyperkeratotic, exophytic and dome-shaped papules or nodules with irregular surface. May be single or grouped papules. They typically reside in areas that are vulnerable to trauma [18] (Fig 3).

Fig 3: verruca vulgaris.

Digit form warts

The HPV-1, 2 and 4 variety of common warts known as digit form warts are distinguished by their protrusions that resemble lengthy threads. It is most usually found on the face, where it shows a preference for the skin around the eyes, the alanasi, and the beard region [17].

Periungual warts

These warts, which collect around the fingernail or toenail, are warts. Around the nail plate, they appear as thicker, fissured cauliflower-like skin (Fig. 4). They frequently result in cuticle and paronychia loss. This type of wart frequently damages the nail by pulling it off the skin or by causing it to partially separate [19].

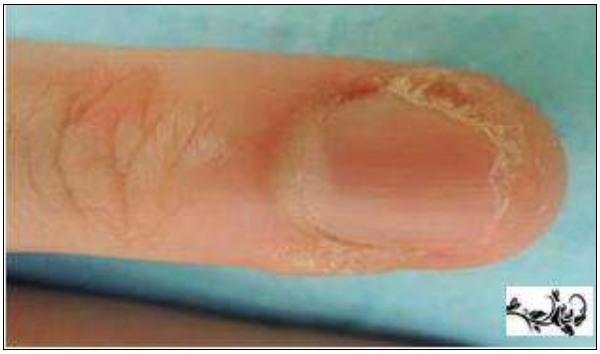

Fig 4: Periungual warts [19].

Plane warts (Verruca plana)

They are primarily brought on by HPV 3 and less frequently by strains 10 and 27. They frequently affect the face, hands, and arms and are skin-colored flat-topped papules with minor scaling that measure between 2 and 5 mm in diameter [20] (Fig 5).

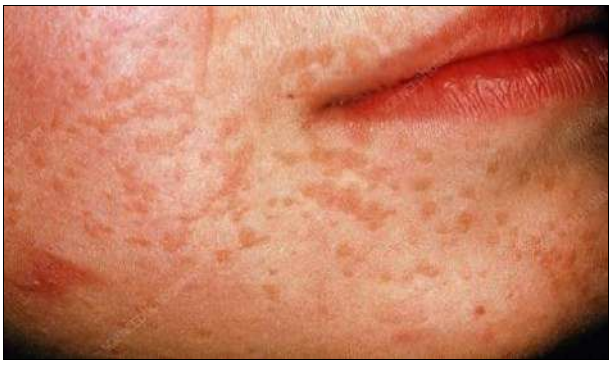

Fig 5: Verruca plana [20].

Verruca plantaris, or plantar warts

They arise out of HPV 1, 2, 4, and 57. They might start as papules that resemble sago grains but later pick up a more characteristic kerato-tic surface with a collar of thicker keratin. They are endophytic/exophytic. They have a punctuate pattern of multiple pinpoint blood vessels [21] (fig 6-3). They could combine to form enormous plaques known as mosaic warts [21] (Fig 6-4).

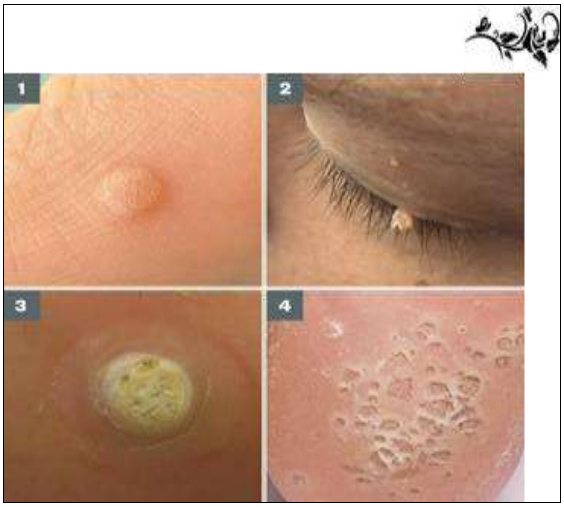

Fig 6: Different types of warts.

1) Common Wart. 2) Digit form Wart.
3) Plantar Wart. 4) Mosaic Wart. [22]

Anogenital manifestations of HPV infections

Condylomata acuminata (Anogenital wart)

They are brought on by HPV strains 6 and 11. Small, verrucous papules, discrete, sessile, smooth-topped papules or nodules, or even asymptomatic, enormous exophytic, cauliflower-like masses, are all possible manifestations of these warts. The lesions can impact the perineum, external genitalia, crural folds, anus, rectum, and urethra in both men and women, and in women they can even extend to the vagina and cervix [23].

Buschke-Lowenstein tumor (Giant condyloma)

Large exophytic lesions with a verrucous, fungating surface are common on the penis or in the perianal region, and they are typically linked to HPV types 6 and 11. Local invasion and its peculiar histological appearance, in which koilocytes and cellular atypia are virtually absent, are its defining characteristics [24].

***Extracutaneous manifestations* of HPV infection**

Oral condylomata

After oral intercourse with an infected partner, condyloma acuminata has been linked to HPV types 6 and 11 and has been observed to affect the mucosa of the gingiva, cheeks, lips, and hard palate. Oral warts may be separated from verrucous carcinoma, localised epithelial hyperplasia, and squamous papillomas based on their histologic appearance [25].

Recurrent respiratory papillomatosis (The condition known as la with Papilloma)

The throat's HPV infection is what contributes to the malignancies. They frequently appear in the larynx, especially near the vocal folds and in the area above the

vocal folds termed as the ventricles, where they exhibit as many or, rarely, solitary, lumpy, white growths that mimic cauliflower [26].

Differential Diagnosis

Cutaneous warts

Skin warts may resemble lichen nitidus, molluscum contagiosum, localised hyperkeratosis (callus), comedones, nevi, acrochordons, acrokeratosis verruciformis [27].

Anogenital warts

Angiokeratoma of Fordyce, Condyloma lata, molluscum contagiosum, pearly penile papules or vestibular papillomatosis, lymphangiomas, lichen nitidus, and epidermoid mass all resemble anogenital warts [27].

Laboratory Studies

The existence of a virus is confirmed by immunohistochemical detection of HPV structural proteins, however this method has low sensitivity. The detection of viral DNA by Southern blot hybridization is more accurate and specific for identifying the HPV type. For testing, the polymerase chain reaction (PCR) amplifies viral DNA. Cervical pap smear to detect HPV in cancer cervix is an important screening test [28].

Management of cutaneous warts

Though there isn't an exclusive antiviral remedy for HPV as of yet, a number of the ones that are available interfere with the viral life cycle. There are multiple wart therapies, but the evidence confirming their efficacy, whether taken alone or in combination, tends to be inadequate [29].

Disruption to the pathogenic epithelium, leading to in cell death, antigen exposure, presentation, and an immune response, is the most conventional form of therapy. The wart ought to grow less thick and the generation of fresh viruses should be slowed down by hindering epidermal proliferation, or more precisely DNA replication [29].

Epidermal injury can be attributed to chemical, physical, and other mechanisms as listed below [30].

Chemical

Salicylic Acid (SA) is hypothesised to operate by encouraging the exfoliation of epidermal cells. SA exists in SA paints in quantities that vary from 10% to 26% when combined with lactic acid (10%–20%). Also readily available are plasters with a 40% SA content and creams with a 50% SA content [30].

Silver nitrate: Children and adults with hand and foot warts received therapy with a 10% solution of silver nitrate, and after six weeks, 63% of patients had cleared up [31].

Pyruvic acid: Used as a peeling agent, pyruvic acid is a potent keratolytic agent. Multiple plantar warts are treated with one drop of pyruvic acid solution 70%, made by dissolving in a water/ethanol solution, twice daily for two weeks [32].

An 80–90% solution of trichloroacetic acid (TCA) and monochloroacetic acid (MCA): TCA is a caustic substance. When used properly, a superficial ulcer develops and cures without leaving any scars. For a maximum of 8–10 weeks, it is administered directly to the wart surface once every week. Every two weeks, the common and plantar wart group receives MCA applications at a concentration of 76% until all warts have been totally treated [33].

Physical

a) Cryotherapy

The most popular approach for treating warts uses liquid nitrogen given via cryo spray, which is sustained for 5 to 30 seconds depending on the location and size of the wart. You can use a cotton bud by dipping it into the cryogen and pressing it firmly on the lesion until an ice halo develops around the bud. Until the warts are gone, it is repeated every two to three weeks for a total of no more than six treatments [34].

b) CO_2 laser

Owing to the clearance rates provided by multiple investigations, 75% of those who obtain CO_2 light therapy experience full recuperation [35].

c) Electrocautery

In order to burn and remove warty lesions, electrosurgery uses high frequency electrical currents in the form of thermal coagulation or electrocautery [35].

d) Pulsed dye Laser

The most used laser is a pulse dye laser (PDL) (585 nm), which works by damaging wart vasculature by absorbing haemoglobin with a peak between 585 and 595 nm. It is challenging to assess effectiveness since treatment regimens differ between research. No significant difference in result was reported in one randomised controlled trial (RCT) between groups receiving PDL, cryotherapy, or cantharidin [36].

e) Photodynamic therapy

In certain secondary care facilities, photo-dynamic therapy (PDT), a damaging treatment, is offered. A particular wavelength of light activates aminolevulenic acid (ALA), causing chemical harm and tissue loss in wart cells that have ingested the substance. Success rates are greater for warts that are thin or planar, however there have been reports of 80–90% clearance rates [37].

f) Surgical removal

Because the healing scar may be unpleasant and surgery may not always completely remove all virus-infected tissue, it is advisable to avoid surgical excision of warts at pressure points like the soles [38].

Virucidal agents

a) Formaldehyde: In concentrations between 3 and 10%, formaldehyde soaks have been used to treat verrucas and have been shown to have an 80% cure rate in youngsters. A 0–75% gel version of formaldehyde is also offered [29].

b) Glutaraldehyde: It has been claimed that a 10% glutaraldehyde paint is equivalent to SA paint in eradicating plantar warts. Deep necrosis reports show the danger of repeated application, and glutaraldehyde usage should be cautious, especially at concentrations that exceed 10 percent [39].

c) Cidofovir: Intralesional cidofovir can be used as a first-

line therapy for all individuals with cutaneous viral warts and seems to give an effective therapeutic alternative choice for warts resistant to traditional techniques [40].
Recalcitrant warts that do not react to the routine medication can also be treated with topical cidofovir, which is a secure and effective option [41].

Antiproliferative agents

a) Vitamin D: Vitamin D regulates immune function and regulates cell division and proliferation. The vitamin D receptor (VDR), which is found in keratinocytes, melanocytes, fibroblasts, and immune system cells of the skin, mediates its actions. Previously, 64 patients with persistent warts received intraregional vitamin D3, with results demonstrating that 90% of patients had full clearance and 6.66% had a partial response [42].

b) Podophyllin and podophyllotoxin: Both normal skin and warts can be harmed by podophyllotoxin, which can prevent cell division by interfering with the mitotic spindle. Therefore, its usage is prohibited during pregnancy and in high quantities. It is a common therapy for anogenital warts, although research on its effectiveness for cutaneous warts is lacking [5].

c) 5-Fluorouracil (5-FU): This drug prevents DNA synthesis and harms basal layer cells that are proliferating. Topical 5-FU has been successfully used to both common and plane warts on the hands and feet. It causes irritation and occasionally erosions when applied topically or intravenously [29].

d) Bleomycin: For 40 years, warts have been treated topically with bleomycin, a cytotoxic drug used in systemic chemotherapy. Warts can either be injected with bleomycin solution or have it applied to the surface then stabbed into the wart [43].

e) Retinoids: Retinoids modify the quality and quantity of the stratum corneum and impact epidermal proliferation and differentiation, which reduces the size of warts. Acutretin 0.5-1 mg/kg/day for up to 3 months generally reduces the majority of lesions, however there is a significant chance of recurrence after stopping treatment [44].

Immunological therapy

a) Imiquimod

e) Retinoids: Retinoids modify the quality and quantity of the stratum corneum and impact epidermal proliferation and differentiation, which reduces the size of warts. Acutretin 0.5-1 mg/kg/day for up to 3 months typically reduces the majority of lesions, nevertheless there is an elevated likelihood of recurrence following quitting therapy [45].

b) Contact immunotherapy

A local delayed hypersensitivity reaction is triggered at the location of the wart by diphenylcyclopropenone/diphencyprone (DPC) or squaric acid dibutyl ester (SADBE), which additionally triggers a local immunological response. Eight years after receiving DPC therapy, those with palmoplantar warts had an 88% full wart eradication percentage. A 2-year follow-up period indicated no instances of recur after a median course of therapy of 5 months [46].

c) Intralesional immunotherapy: Antigenic activation of the host-cell-mediated immune system has been employed to promote wart elimination using intralesional Candida, mumps, and tuberculin antigens. Although reported clearance rates for this sort of intralesional immunotherapy range from 47% to 87%, there isn't enough solid data to warrant its usage [47].

d) Zinc oxide and zinc sulfate

Antigenic activation of the host-cell-mediated immune system has been employed to promote wart elimination using intralesional Candida, mumps, and tuberculin antigens. Although reported clearance rates for this sort of intralesional immunotherapy range from 47% to 87%, there isn't enough solid data to warrant its usage [48].
Warts can be effectively treated with topical or oral zinc without suffering any serious side effects. It has been discovered that intralesional 2% zinc sulphate causes wart removal. The injection location experiences a noticeable influx of inflammatory cells as a result [49].

Conclusion

Although there is currently no entirely appropriate course of treatment that would ensure complete cure, without relapses, wart treatment is still exceedingly difficult in contemporary dermatology. The majority of treatment methods involve numerous uncomfortable procedures rather than concentrating on eliminating the viral agent that is the cause of the illness. The main justification for the continued use of traditional medical practises and cures is this. The part doctors play in the healing process is being reevaluated. Any medical practise should be built on a partnership between the doctor and the patient, where accountability is shared and decisions are made together.

Conflict of Interest
Not available

Financial Support
Not available

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