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Injection of Polidocanol and mixed Digoxin and Furosemide in Treatment of Warts

Abeer A.Abd-Elhady, Amany I.Mostafa and Ghada M.AbdelKhalik
1 Dermatology, Venerology and Andrology Dept., Faculty of Medicine, Benha University, Benha, Egypt
E-Mail: abeereldishow2020@gmail.com

Abstract
Background: Many different therapies are available for patients with numerous warts. The majority of these treatments target the diseased tissue directly, but sometimes the amount of cellular damage is insufficient to release the cytokines that kill the latent virus. In such cases, patients seek for other treatments. In this article, we will have a look at the results of treating cutaneous warts with intralesional polidocanol and intralesional combination furosemide and digoxin. Final thoughts: Treatment of cutaneous warts with injections of Polidocanol and a combination of intralesional furosemide and digoxin seems to be a safe, highly successful, and adverse effect-minimizing option.

Keywords: cutaneous warts, Polidocanol, digoxin, furosemide.

1. Introduction

The human papillomavirus [HPV] causes cutaneous warts, which are harmless growths on the skin.Papillomaviruses are very contagious because they may inflict long-term infections that don't show any signs of systemic disease and almost never cause the host's death.[1] Warts are best treated with intralesional therapy rather than topical administration as it allows for larger medication concentrations throughout the lesion, which means fewer sessions and no need to treat each wart individually. This treatment option is recommended for patients with multiple resistant warts, as it has shown encouraging results in reducing or eliminating recurrence, improving the local immune response, and minimizing side effects. However, it is crucial for the patient to adhere to their drug regimen. [2] The effectiveness of administering digoxin and furosemide intralesionally to treat cutaneous warts is based on their ability to limit DNA replication via their interactions with cell membrane co-transporters and the influx of potassium. [3]Polidocanol induces coagulation at low concentrations by creating a negative charge on the outside of cell membranes; this leads to strong clots that are resistant to fibrinolysis. The final result is endovascular fibrosis and successful lumen occlusion due to thrombotic occlusion at the endothelial injury site [4].

Several dermatological off-label applications of polidocanol have already been detailed. Several skin conditions, including cutaneous focal mucinosis, hemangiomas, facial veins, digital mucous cysts, mixed skin ulcers, acne cysts, and intralesional polidocanol, have shown promising results in therapy. 5. Warts on the skin

When different types of the Human Papillomavirus [HPV] infect the skin and mucous membranes, the result is a benign growth called a cutaneous wart, scientifically known as Verruca Vulgaris. Transmission of infections may happen via direct skin-to-skin contact with sick people [which can happen even when no symptoms are present] or even inanimate objects. [6] Human HPV induces keratinocyte proliferation, which results in the distinctive growths. Because T-cell lymphocyte-mediated immunity is responsible for either eliminating the infection or suppressing it into latency, recurrence is frequent with or without therapy. [7] What causes warts?

The verruca-causing Human Papillomavirus is really a family of more than 150 distinct kinds of nonenveloped DNA viruses that share striking similarities in their genetic makeup [8]. An assortment of human papillomavirus subtypes, including 1, 2, 3, 4, 27, 29, -57, -60, -63, -65, -66, and -69, have been shown to be present in warts. Although most forms of human papillomavirus [HPV] are more often linked to cutaneous or mucosal/cutaneous lesions, in very rare instances [e.g., in people with impaired immune systems] a mucosal HPV may also produce cutaneous lesions [e.g., plantar warts]. It is via direct contact with infected human blood that the human papillomavirus [HPV] is transmitted from person to person. [9] In order to avoid detection by the immune system, the human papillomavirus [HPV] has adapted many tactics, one of which is to induce the overproduction of epithelial cells. In addition to affecting innate immune cells, HPV may hinder the activity of leukocytes produced from bone marrow. Furthermore, HPV has the ability to impede the antiviral immune response that is essential for clearing HPV. The evasion mechanisms of HPV enable it to cause chronic infections and have a role in the

development of many types of cancer. [10] Surfaces may harbor the human papillomavirus for months or even years. Both direct touch, as in a plantar wart, and indirect contact, via fomites, including floors, socks, shoes, towels, and sports equipment, are necessary for viral particle infection of a host. If you have HPV, the virus won't spread throughout your body or go through a viremic phase. The basal epithelial layer contains stem cells that are constantly proliferating. Once HPV comes into touch with a host, it obtains entrance to this layer. The first stage in integration is DNA damage, which may be caused by oxidative stress or the HPV protein. The processes that follow are dictated by the reactions to this damage. While HPV replicates, it inserts breaks in its DNA that does not be repaired. A sufficient amount of episomal HPV is produced, increasing the availability of additional HPV DNA for integration into the host DNA, and HPV takes use of this damage response pathway for its own reproduction. A driving force during viral replication, the DNA break-induced DNA damage response [DDR] initiates the buildup of replication components at the replication foci. [12] Warts seen in clinical settings:

The placement of the warts greatly determines the clinical appearance. Domed nodules with a papillomatous and keratotic ["verruciform"] surface may appear in places that do not experience mechanical pressure, such as the fingers, the dorsa of the hands and feet, the arms, the legs, or the trunk. The nodules can be skin-colored or yellowish-gray. Black spots, which match the histologically observed HPV-induced papillary necrosis, are also common observations. When the periungual zone is impacted, lesions tend to spread extensively around the nails. Another possibility is subungual growth, which leads to partial onycholysis. [13] Endophytic plantar warts [also known as mosaic warts] are a common clinical manifestation of plantar vulgaris. Like an iceberg, a solitary plantar wart only shows the top layer of the lesion to the naked eye. The rest of the wart is hidden under the surface. Callus often encases solitary plantar warts. Mosaic warts combine rather than appearing alone on the foot. sixteenth] Polydocanol

A nonionic detergent sclerosant, polidocanol binds to phospholipid membranes and causes structural disruptions. Polidocanol lyses blood cells, platelets, and the endothelium lining at high doses. Polidocanol induces coagulation at low concentrations by creating a negative charge on the outside of cell membranes; this leads to strong clots that are resistant to fibrinolysis. [15] Unapproved applications of polidocanol sclerotherapy in dermatology

The veins in the upper limbs

In a research including 76 out of 80 patients [95%] treated with 3% polidocanol [16], the dorsal hand veins were successfully eliminated.

2. Pyogenic Granuloma

Six out of seven patients treated with 0.5% polidocanol for pyogenic granulomas had the tumors completely removed, leaving hardly noticeable scars, according to the research. Nobody had any problems or recurrences after the treatment, and it was well-tolerated. [17]

III. Veins on the Face

Twenty individuals diagnosed with craniofacial venous malformation [CVFM] were the subjects of an investigation testing the efficacy of 3% polidocanol sclerotherapy. After sclerotherapy, patients were asked to rate their discomfort, functional impairment, cosmetic abnormalities, and impairment in daily life compared to before and after the procedure. [number one]

Quintessential Skin Ulcers

Two case reports involving patients treated with ultrasound-guided injection of polidocanol micro-foam for mixed-etiology lower leg ulcers were reviewed in the research. Vein incompetence was shown to be a significant cause of ulceration in both instances when ultrasonography indicated substantial saphenous vein incompetence. After the initial injection of polidocanol into their inadequate veins, both patients reported a significant improvement in their pain levels. At weeks 7 and 10, both patients had fully healed wounds. There were no reports of adverse effects. [19]

5] Mucinosis of the skin

Abnormal mucin deposition in the skin leads to cutaneous localized mucinosis. An example of cutaneous mucinosis on the knee that developed a year after joint replacement and was effectively treated with 1% polidocanol is shown in a case report. The patient showed improvement after just 2 sessions of therapy and did not have recurrence at the 2-year follow-up. [20] Scleromas

Intralesional foam sclerotherapy with 0.3% polidocanol [3% polidocanol diluted with normal saline] was administered to patients suffering from acne cysts. To make the foam, 1 milliliter of the diluted sclerosant was mixed with 4 milliliters of air. Within 48 hours, 8 patients had full resolution, and within one week, 2 patients had full resolution. At the conclusion of the 4-week follow-up, the therapy was unsuccessful for the other two

patients. In addition, just the treated area showed improvement in two individuals who had several acne cysts. At the 12-week follow-up, no recurrence had occurred in any of the patients who had shown improvement. The treated areas recovered quickly with little scarring, and no side effects were noted. Malformations of the Glomvae [21]

The cells that make up glomeruvenous malformations [GVMs] mirror the modified smooth muscle cells seen in the glomus body. These malformations manifest as many nodules or plaques that range in color from pink to blue. An instance of hereditary GVM in a 26-year-old male patient who responded well to 3% polidocanol sclerotherapy for the treatment of 10–15 nodules. After 6 sessions, the patient saw a 90% decrease in discomfort and a 60% flattening of the lesions. Pain during and soon after sclerotherapy was the most often reported adverse effect. No lesions resurfaced during the 6-month follow-up. [22]

Anti-Viral Ionic Treatment

Cardiac glycosides and loop diuretics are known to inhibit coupled cotransporters. Some medications that reduce K+ inflow include digoxin and furosemide, two loop diuretics. Digoxin inhibits Na-K-ATPase, whereas furosemide inhibits NKCC1. Adenovirus [AV], human herpesviruses varicella zoster [VZV], cytomegalovirus [HCMV], and herpes simplex virus [HSV] were among the DNA viruses that these medications were shown to inhibit, in addition to an RNA virus called encephalomyocarditis virus [EMCV]. [23]

The cellular ion cotransporters Na-K-ATPase and NKCC1 are the particular targets of digoxin and furosemide, respectively. Unlike other antiviral medications, this one targets the host cell. Inhibiting ATP-sensitive potassium channels reversibly halted cell proliferation by blocking the input of potassium ions, thus arresting cells in the G0/G1 phase of the cell cycle. the third

When used topically, furosemide and digoxin were shown to be effective in treating viral warts, according to many studies. Patients with common warts were the subjects of an investigation that assessed the systemic exposure, safety, and tolerability of Ionic Contra-Viral Therapy [ICVT] using a mix of topical furosemide and digoxin [24]. There was no indication of digoxin or furosemide systemic exposure, and ICVT was well-tolerated topically. The warts' diameter, height, and volume decreased at a quick and statistically significant rate. The effectiveness and safety of injecting digoxin and furosemide intralesionally into plantar warts was assessed in a research. If you have more than one plantar wart, the combination therapy is a safe and effective alternative. [23]

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