Chondrodermatitis Nodularis Helicis Successfully Treated With Photodynamic Therapy

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Chondrodermatitis nodularis helicis (CDNH) is a painful inflammatory condition affecting the helix of the ear. We describe 5 patients with CDNH who were successfully treated with methyl aminolevulinate photodynamic therapy (PDT).

REPORT OF CASES

Five patients at our clinic, 3 women and 2 men, with a mean age of 77 years (age range, 58-88 years), were diagnosed as having CDNH. All cases presented with 1 or 2 small, painful erythematous nodules, 3 of which were centered by an ulcer, on the upper part of the helix (Figure 1). Two patients also had a painful erythematous lesion on the antihelix. Pressure-relieving devices, topical corticosteroids, and cryotherapy had been previously used with little or no improvement. In addition, in 1 case the lesion was a recurrence after surgical excision 2 years previously. The mean duration of the CDNH was 11.6 months (range, 6-24 months).

Conservative techniques, such as pressure-relieving devices and topical and intralesional corticosteroids, are considered to be the first-line option and attain a cure rate of 87% in 15 patients, albeit after a short follow-up period (mean duration of follow-up, 4.5 months). Perilesional collagen injection, laser excision, and cryosurgery have also been described with variable results. Surgical methods include curettage and cautery, as well as techniques for the removal of affected cartilage with skin and without skin. The mean cure rate of these surgical techniques is 84.2% (range, 69%-100%) with different cosmetic outcomes.

Figure 1. Chondrodermatitis nodularis helicis in a 76-year-old woman resistant to cryotherapy. A, Before. B, One month after 1 session of methyl aminolevulinate-photodynamic therapy (Metvix for 3 hours following by irradiation with light-emitting diode light [Aktilit; PhotoCure ASA, Oslo, Norway]) at a wavelength of 630 nm at 37 J/cm².
Lesions were treated with methylaminolevulinate-PDT. They were prepared by cleaning with alcohol and using a curette to scrape away crusts, the keratotic plug, or the superficial part of the more nodular lesions. Then methyl aminolevulinate, 16% (Metvix; Galderma, Sophia Antipolis, France), was applied on and 1 cm around the lesions and under the occlusive dressing (Tegaderm; 3M Healthcare, St Paul, Minnesota) and then covered with aluminum foil for 3 hours. Fluorescence was detected using visible ultraviolet light at 405 nm, which showed a diffuse soft red fluorescence in the lesional area (Figure 2). Subsequently, the treatment sites were irradiated with a light-emitting diode (LED) light source at a wavelength of 630 nm at 37 J/cm² (Aktilite lamp; PhotoCure ASA, Oslo, Norway). The treatment was well tolerated, and neither notable pain nor adverse effects were present during the irradiation or in the following days. All lesions resolved with an excellent cosmetic outcome (Figure 1), except for 1 patient whose disease did not completely respond after 3 treatments 1 month apart.

Chondrodermatitis nodularis helicis is a painful condition that is difficult to treat. Histologically, CDNH consists of a nodule of degenerate homogeneous collagen surrounded by vascular granulation tissue with an overlying acanthotic epidermis, and there may be a central ulcer through which the damaged collagen is extruded. In nearly all cases there is inflammation and fibrosis of the underlying perichondrium, and degenerative changes may be seen in the cartilage. Many authors view the condition as an example of transepidermal elimination of altered connective tissue. Pressure and a compromised local blood supply are believed to be key factors in the development and recurrence of CDNH. Various theories regarding its etiology have been published about the initial event: that it is the result of (1) cartilaginous changes, (2) hyperkeratosis leading to the perforation, (3) cold or poor circulation, and/or (4) traumatic injury to the collagen. As to the efficacy of PDT for CDNH, it could act on several pathogenic factors. Our hypothesis is based on (1) its anti-inflammatory and immunomodulatory action, (2) its effect on vascularization or on collagen, and (3) a possible chondroprotective effect. In skin areas treated with aminolevulinic acid (ALA)-PDT, inflammatory reactions seem to slow down because of the resident macrophages and mast cells death and the slow recovery to cytokine responsiveness of the surviving cell population. It is generally accepted that PDT causes acute inflammation, but it is also thought that the judicious application of acute inflammation can interrupt the process of chronic inflammation and stimulate healing. Topically applied methyl aminolevulinate sensitizes mainly keratinocytes; using protocols of irradiation, which may induce sublethal cellular effects, several cytokines are released by those cells, possibly resulting in a change of cytokine equilibrium in the inflammatory milieu and finally leading to a disruption of the chronic inflammation present in CDNH. In relation to the role of topical PDT on improving blood supply in CDNH, it has been proved that blood perfusion is increased immediately after irradiation and persists up to a week. Regarding the effect on collagen, no changes in synthesis of collagen type II, the major connective tissue component of ear cartilage, has been observed when chondrocytes were treated in vitro with ALA-PDT. Nevertheless, Park et al have demonstrated that after 1 month of treatment with ALA-PDT in human skin, the total collagen volume in the dermis significantly increased, with expression of type I and III procollagen; this could explain the excellent cosmetic results in our patients. Regarding the effect on cartilage, it has been shown that chondrocytes are not destroyed by ALA-PDT, whereas a decrease in the proportion of viable cells occurs in osteoblasts and synovial cells. In addition, PDT has been shown to modulate in vitro cartilage metabolism, activating a chondroprotective program in photosensitized cartilage in the context of osteoarthritis.

In our patients with CDNH, topical application of methyl aminolevulinate on the lesions resulted in a moderate fluorescence. This could be possible because it has been shown that inflammatory tissues emit some fluorescence when ALA or methyl aminolevulinate is applied. Perhaps it is due to the protoporphyrin IX (PpIX) accumulation into the inflammatory cells present on CDNH. However, the reason for the minimal adverse ef-
effects reported by our patients could be that noncancerous cells metabolize less methyl aminolevulinate into the photoactive PpIX than the cancerous ones, resulting in less fluorescence and also in fewer adverse effects.19,20

We propose methyl aminolevulinate–PDT as a novel method to treat patients with CDNH because it is non-invasive, free of adverse effects, can be reapplied if necessary, and has curative results comparable to those achieved by existing methods. More patients have to be treated in order to standardize PDT as the treatment of choice for CDNH.

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REFERENCES


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