Response of Confluent and Reticulate Papillomatosis of Gougerot and Carteaud to Topical Tretinoin

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Confluent and reticulate papillomatosis (CRP) of Gougerot and Carteaud is a rare cutaneous disorder characterized by persistent, usually asymptomatic, dark papules and plaques centrally located on the back, intermammary, and epigastric areas. The eruption spreads out peripherally into a fading reticulated pattern. The pathogenesis is poorly understood, but there are several theories. Many different treatments, with varying success rates, have been attempted. We present 3 patients with CRP who had excellent results in the areas treated with topical tretinoin. The only difficulty with therapy is applying the tretinoin to the back, which sometimes necessitates a second person. However, if this situation can be overcome, topical tretinoin provides an effective, safe alternative to systemic therapies. Response to tretinoin provides support that CRP is a disorder of keratinization. Finally, the fact that 2 of the patients were brothers may support the idea that CRP has a hereditary influence.

Confluent and reticulate papillomatosis (CRP) of Gougerot and Carteaud is a rare skin disorder originally described in 1927.[1] We discuss the treatment of 3 patients and a review of CRP.

Case Reports

Patient 1—A 17-year-old Haitian man had a pruritic skin eruption on his face, back, shoulders, and chest for 5 months. Family history was significant for a brother with CRP (Patient 2), father with diabetes, and mother with eczema. Examination revealed hyperpigmented, reticulated papules and plaques. A potassium hydroxide preparation from the back was negative for fungal organisms. No fluorescence was seen on Wood's light examination. A biopsy revealed papillomatosis, hyperkeratosis, and a sparse perivascular infiltrate. A periodic acid-Schiff stain was negative for fungal organisms. A diagnosis of CRP was made. It was unresponsive to topical ammonium lactate, ketoconazole, corticosteroids, and salicylic acid sulfur soap. Treatment with topical tretinoin 0.01% gel to all affected areas once daily was begun with a moisturizing cream to use as necessary. In 6 weeks, there was a marked improvement with flattening and fading of the papules and plaques. Within 10 weeks, the condition was almost completely cleared.

Patient 2—A 17-year-old Haitian man was seen with nonpruritic, hyperpigmented, reticulated papules and plaques noted on his face, trunk, posterior neck, and shoulders. Prior treatment with sodium hyposulfite was unsuccessful. Topical corticosteroids produced minimal improvement. A potassium hydroxide preparation from the back was negative for fungal organisms. No fluorescence was seen on Wood's light examination. His brother (Patient 1) has CRP. A clinical diagnosis of CRP was made, and he was given topical tretinoin 0.025% gel once daily and a moisturizing cream to use as necessary. No biopsy was taken because this patient never returned for subsequent follow-up because of insurance difficulties. However, he continued sporadic self-treatment with the tretinoin. The patient was seen 8 weeks later when he accompanied his brother (Patient 1) on a follow-up visit. His face was completely clear, with much improvement of his neck, shoulders, chest, and back.

Patient 3—A 22-year-old Jamaican man presented with a pruritic eruption on his face, posterior neck, back, shoulders, and chest, which he has had "since his early teens." He was unsuccessfully treated with

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salicylic acid soap. His family history is significant for a sister with asthma and mother with eczema. Examination revealed hyperpigmented reticulated papules and plaques. A potassium hydroxide preparation from a shoulder was negative for fungal organisms. No fluorescence was seen on Wood's light examination. Biopsy revealed slight epidermal hyperplasia with hyperkeratosis, a papillomatous surface, and a somewhat decreased granular layer. A periodic acid-Schiff stain was negative for fungal organisms. A diagnosis of CRP was made. The patient was given topical tretinoin 0.025% gel once daily and a moisturizing cream to use as necessary. By 7 weeks, the patient's skin was much smoother, with flattening and fading of most of the papules and plaques. The patient was not seen for 6 months, but continued to apply the tretinoin to his face and chest only. His face was completely clear and his chest was moderately improved. His back, which was not treated with tretinoin, was still severe. The patient was instructed to use the tretinoin on his back, in addition to his face and chest, and a moderate improvement was noted on his back after 4 weeks.

Discussion

CRP usually begins as asymptomatic, discrete, low, dome-shaped or flat-topped reddish papules 1 to 2 mm in diameter. They soon become gray or brown and enlarge to a size of 4 to 5 mm, with the surface resembling a flat wart. Papules increase in number and size by peripheral extension. Those centrally situated coalesce into a diffuse plaque, while those peripherally situated spread out into a pigmented reticulated pattern that gradually fades as it approaches the surrounding unaffected skin. The lesions begin to appear in the intermammary and epigastric regions and later over the spine between the scapulae. They assume a rhomboid pattern, with the long axis running cephalocaudally. CRP spreads upward and outward on the breasts and the superior portions of the shoulders and downward along the midline toward the pubis and sacrum. The axillae and neck may also be involved. It is interesting to note that all 3 of our patients had classic lesions involving the face (which is not considered typical for this condition). The progression of the disease is usually slow, with occasional spontaneous resolution.

The pathology of CRP is similar to acanthosis nigricans. The epidermal pathology demonstrates a prominent serrated pattern, slight acanthosis, and irregular thinning over the papillary tips. The granular layer is thin. Elongated papillae may contain dilated capillaries surrounded by a mild lymphocytic infiltrate.

CRP tends to occur more often in female and in black patients and usually appears around the age of 20.[2] At least 2 familial cases have been reported.[3] The etiology is unknown, but there are several different theories. Some suggest that the disease may represent an abnormal response to colonization with *Pityrosporum orbiculare* because CRP clinically resembles tinea versicolor. *P orbiculare* has been demonstrated in the lesions of some patients, and treatment with topical selenium sulfide, topical miconazole, or oral ketoconazole may produce improvement in some cases. Another possibility is an abnormal response to colonization by follicular bacteria.[4] A keratinization disorder has been postulated as an etiology[5] that could be genetic or acquired, perhaps secondary to colonization with *P orbiculare* or *Staphylococcus*-derived toxins.[6] Reports of disease improvement using vitamin A derivatives for treatment support the view that CRP is a keratinization disorder.[5,7] An endocrine etiology has been suggested because occasional cases are associated with thyroid dysfunction, diabetes, Cushing's syndrome, or hypopituitarism. An association with atopy has also been suggested.[8]

Many different treatments, with different success rates, have been used for CRP. Treatments such as keratolytics, urea, corticosteroids, 5-fluorouracil, ultraviolet light, and x-ray therapy have generally proved disappointing. Topical selenium sulfide has proved beneficial in several cases,[3,9,10] but has not been effective in others. Selenium sulfide has both keratolytic and antifungal properties.[10,11] The effect of selenium sulfide may be due to its keratolytic effect rather than its antifungal properties because *P orbiculare* is not discovered in most patients after potassium hydroxide, Wood's light, and histologic examinations. Several studies support the use of minocycline and show high rates of success,[6,12-14] whereas other studies do not show improvement with minocycline treatment. Minocycline's effectiveness may be due to antiproliferative, anti-inflammatory, or antibiotic properties.[12-14] One study suggests that minocycline may be a safer alternative to ketoconazole and oral retinoids with regard to potential hepatotoxicity and teratogenicity in women of childbearing age.[12] Another study suggests that it be used in adults and nonpregnant patients with CRP.[14] Tetracycline,[9] erythromycin,[9] and doxycycline[1] have also been effective in treating some patients.

Topical (tretinoin)[8] and oral (isotretinoin and etretinate)[5,7,15] vitamin A derivatives have also been used with success. It has been suggested to treat patients with urea and tretinoin first. If there is treatment failure, oral vitamin A derivatives or minocycline can be used with caution. However, other studies do not show benefit with topical tretinoin treatment.[7,9,15]

Our patients responded very well to topical tretinoin (0.01% or 0.025% gel once daily), but only in the areas in which it was applied. We must continue to treat these patients for a longer period to see whether

it is possible to taper the medication without causing a relapse of the CRP. The major drawback of topical tretinoin is that it is difficult to apply to the back without the help of a second person. However, if a patient is able to apply the medication to his or her back, topical tretinoin may provide a safer alternative to systemic therapies. Potential side effects (skin redness, peeling, or discomfort) were not noted, possibly because all patients were instructed to use a moisturizing cream as necessary. The effectiveness of tretinoin in these patients supports the hypothesis that CRP is a disorder of keratinization.

Patients 1 and 2 provide another example of CRP in relatives. These patients may have been genetically predisposed to develop the disease. Because few familial cases have been reported, it is possible that there are multiple etiologies of this disease. Perhaps some cases have strong environmental influences while others have strong genetic influences. All 3 patients reported that their mothers had eczema. It is interesting to speculate that this "eczema" may actually have been CRP as well. Finally, the fact that diabetes and asthma were part of the family history in these patients may be purely coincidental or may provide support for an association with CRP.

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