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Pseudoatrophoderma colli: distinct entity or just a variant of confluent and reticular papilomatosis of Gougerot-Carteaud

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Abstract

Pseudoatrophoderma colli is a rare entity described in the same time period as confluent and reticular papillomatosis of Gougerot-Carteaud and the two conditions have certain similarities. Pseudoatrophoderma colli is clinically characterized by lesions with an atrophic and wrinkled appearance, which are mainly located on the trunk and neck (hence the name colli). Few references exist in the literature and most of them are very old. Histopathological findings are nonspecific, showing mild hyperkeratosis, thinning of the stratum granulosum, and acanthosis and papillomatosis in some areas. In the papillary dermis there is vascular dilatation with a sparse inflammatory lymphohistiocytic perivascular infiltrate. Fragmentation of elastic tissue has been described only in one case. There is no specific treatment, with variable responses to diverse therapies including ultraviolet light, vitamin A, lactic acid and minocycline. We describe in detail two patients with pseudoatrophoderma colli and show histology. The first patient was treated with minocycline 100mg per day for two months and the second patient was treated with lymecycline 600mg per day for three months and 300mg per day for another two months. Both patients demonstrated a good response within the first month of treatment.

Keywords: pseudoatrophoderma colli, confluent and reticular papillomatosis, lymecycline, minocycline

Introduction

Under the heading pseudoatrophoderma colli (PC), Becker and Muir in 1934 described the first case of this rare condition [1]. In 1957, nine further cases were described and all were females [2]. By the 1980s four new cases were added, two were males [3,4]. Familial case reports with possible autosomal dominant transmission have been described [3,5]. The validity of PC as a unique entity is not clear as it shares many similarities with confluent reticular papillomatosis of Gougerot-Carteaud. It has been proposed it could be a localized defect of keratinization [3].

Prior to the description by Becker and Muir, Gougerot described a similar process that he called brilliant parakeratosis, given its clinical features, in a patient who refused biopsy [6]; the author subsequently changed the name of the process to brilliant atrophy, in two patients in which the histopathologic findings showed epidermal atrophy, partial condensation of collagen, and elastic tissue destruction [7,8]. It is not clear if they represented PC.

Report of cases

Case 1

A 27-year-old woman presented with pruritic scaling brown plaques in the left posterior axillar line for one month (**Figure 1**). Afterwards new lesions appeared in the left anterior axillar line, right arm, chest (**Figure 2**), right lower quadrant, and popliteal fossa. Her medical history only revealed allergic rhinitis. A skin biopsy showed an epidermis with mild papillomatosis (**Figure 3**) and mild ortho- and parakeratotic hyperkeratosis; in the dermis a scarce superficial perivascular lymphoid infiltrate was found. Elastic fibers appeared normal (**Figure 4**).

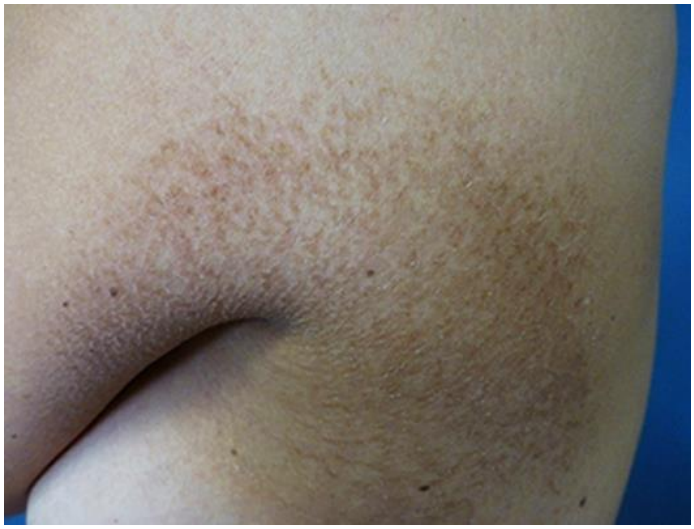

Figure 1. *Scaling brown plaques in the left posterior axillar line.*

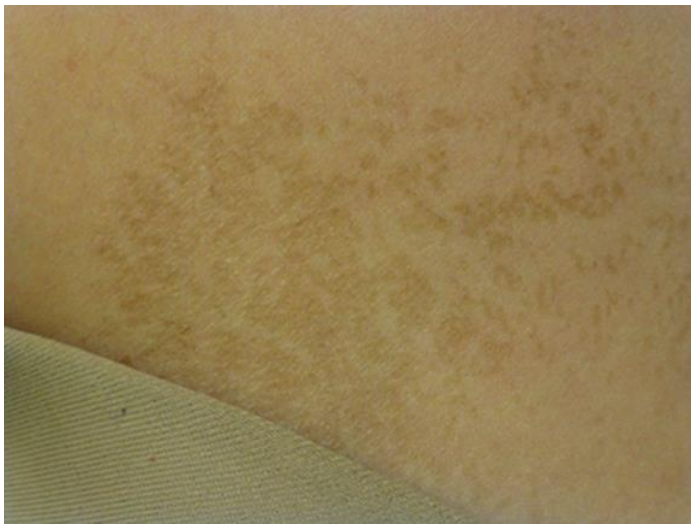

Figure 2. *Dirty-looking skin, with a brownish pigmentation over the breast.*

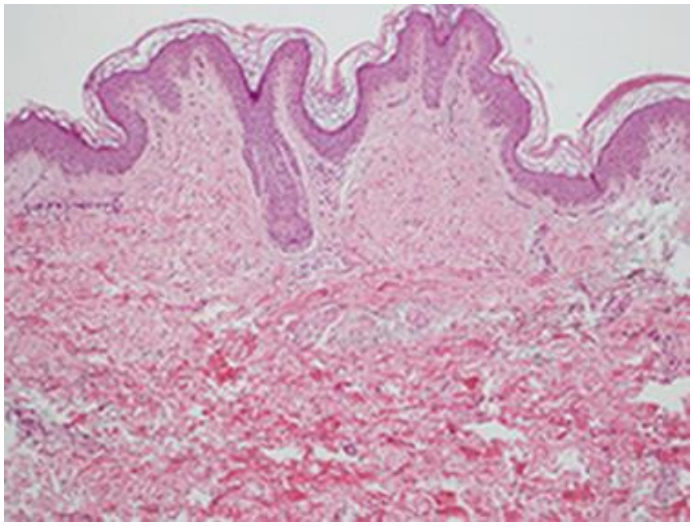

Figure 3. *Epidermis with mild papillomatosis. H&E, 100×.*

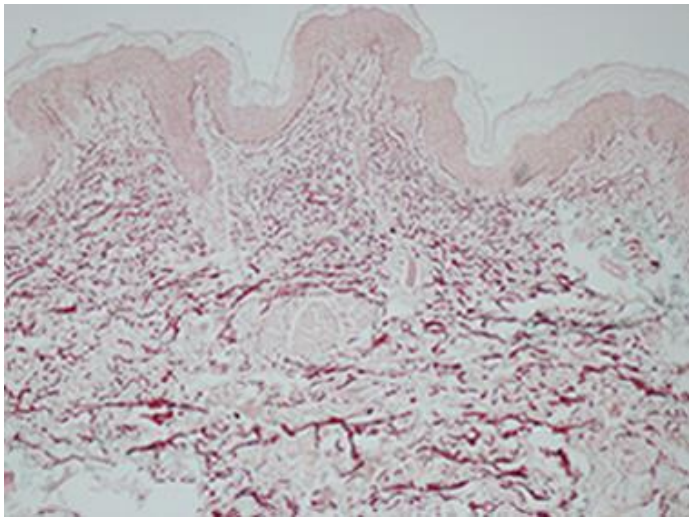

Figure 4. *Elastic fibers with normal appearance. Orcein, 100×.*

Periodic acid-Schiff staining was negative for mycotic elements. She was treated with minocycline 50mg twice a day for two months. Within the first month the patient noticed improvement, which was persistent during her two-month treatment.

Case 2

A 27-year-old healthy woman had a 10-year history of multiple hypopigmented, scaly lesions of atrophic appearance located in the abdomen and axilla (**Figure 5**). On the abdomen the lesions adopted a vertical orientation, whereas in the axillae they appeared with a reticular pattern (**Figure 6**). Repeated mycologic studies were negative. She was diagnosed with pityriasis versicolor and treated with oral itraconazole, topical imidazole, and shampoo with ketoconazole without improvement. A skin biopsy showed irregular acanthosis, loose stratum corneum (**Figure 7A)**, and irregular thickness and fragmentation of elastic fibers (**Figure 7B, C)**.

She was treated with oral lymecycline 300mg twice a day for three months and afterwards with 300mg a day for another two months. After the first month of treatment she observed improvement of the lesions, which disappeared completely by 5 months.

Clinical presentation

The lesions of PC usually appear in healthy women between ages 15 and 36. They consist of brown macules with a depressed atrophic appearance or with a thin scaly and rough surface and the

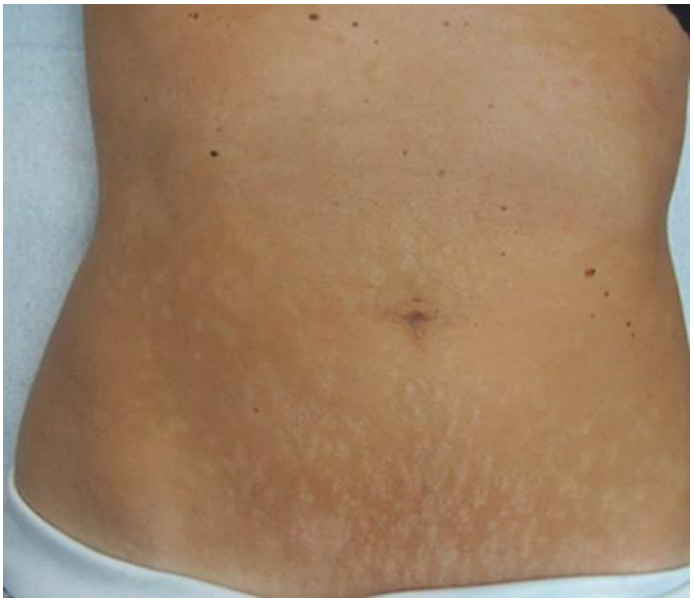

Figure 5. *Multiple hypopigmented lesions with atrophic appearance located on abdominal skin.*

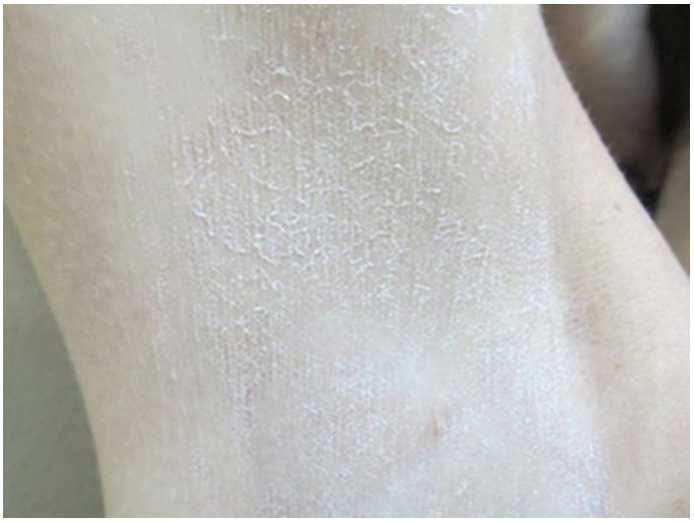

Figure 6. *Atrophic appearance of lesions in one arm, resembling cigarette paper.*

appearances have been compared to cigarette paper (**Figure 8**) or a thin layer of collodion (**Figure 9**), [2]. By stretching the skin, wrinkles are attenuated but do not disappear. Sometimes the condition resembles dirty-looking skin, with a grayish-brown pigmentation. According to Becker, in the discussion of the presentation of Obermayer [9] the stratum corneum loses its velvety appearance and is shiny in patches against reflected light. These lesions exhibit a variable size and occur singly or appear confluent, occasionally with a reticulated pattern, in well-defined areas such as the back, lateral neck, upper trunk, breast, axillary pillars, scapular regions, outer arms; sometimes several zones are involved [9-11]. They tend to be stable over time, showing spontaneous regression in one case after 8 years of evolution [2]. Itching may or may not be associated.

Histopathology

Histopathology shows nonspecific findings, with a loose, thickened stratum corneum [5]. The spinous layer shows a variable thickness, with mild irregular acanthosis in some areas, thinner in others [11]. The granular layer appears thin and sometimes is reduced to a single layer or is nearly absent [5]. In areas of the thinned suprapapillary epidermis, the dermal papillae may reach to two to three rows of cells above the stratum granulosum [5]. In the dermis there is slight edema, dilated superficial blood vessels, and minimal to moderate lymphocytic infiltrate [5,11]. Staining for elastic tissue has been

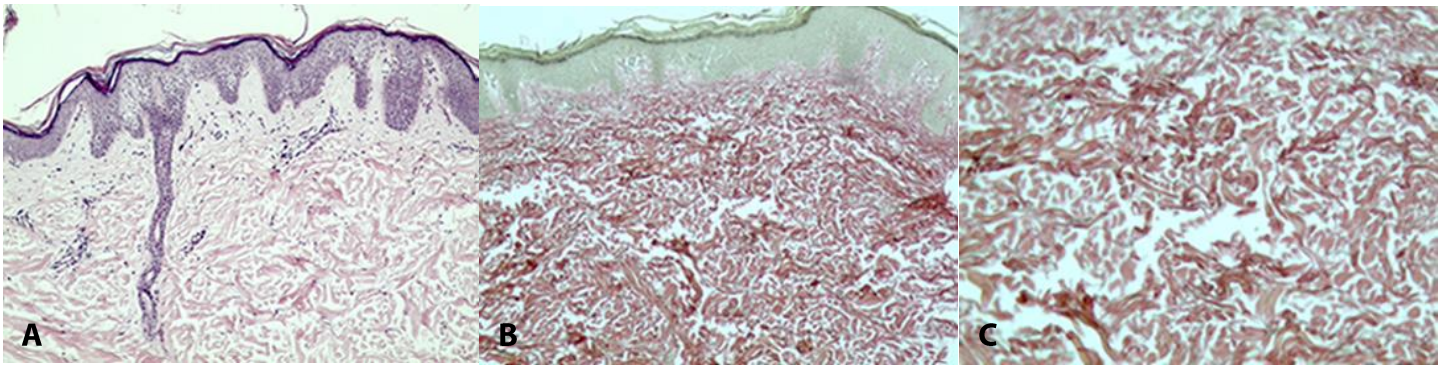

Figure 7. ***A)*** *Irregular acanthosis with a loose stratum corneum. H&E, 100×.* ***B)*** *Irregular thickness and fragmentation of elastic fibers. Van Giesson-orcein, 100×.* ***C)*** *Irregular thickness and fragmentation of elastic fibers, closer view. Van Giesson-orcein, 200×.*

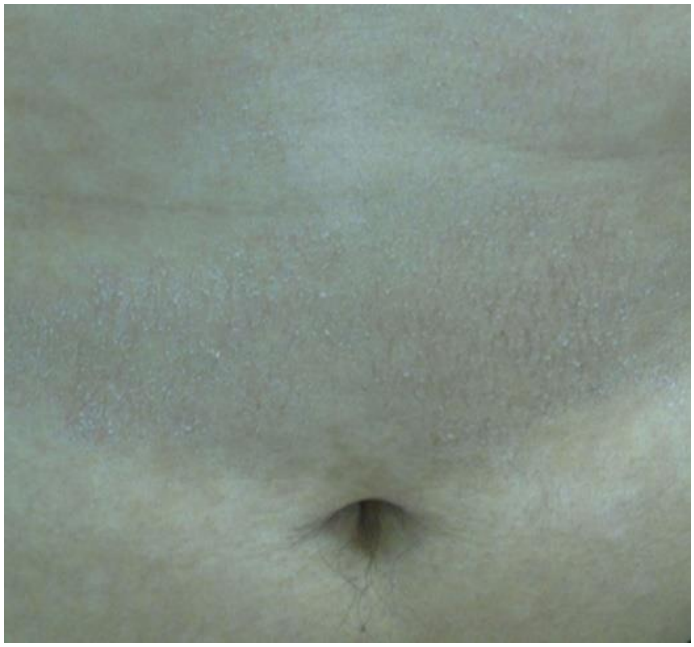

Figure 8. *Scaly and rough surface, which has been compared to cigarette paper.*

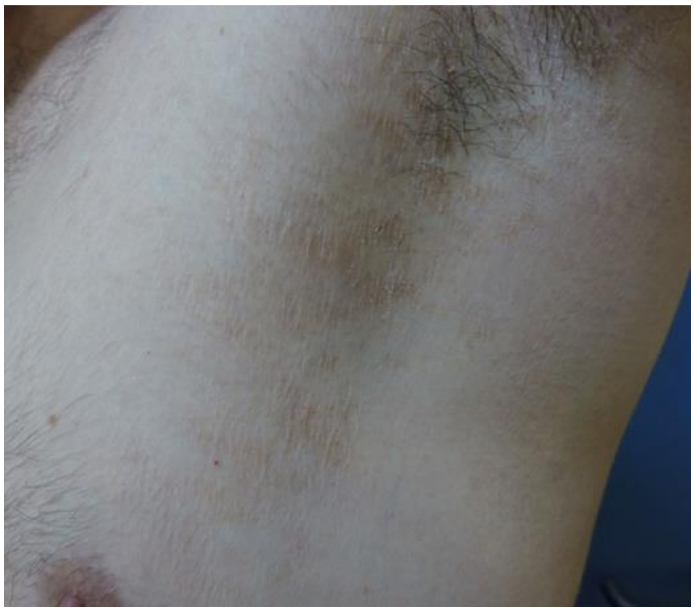

Figure 9. *Rough surface of lesions like a thin layer of collodion.*

normal in most cases, with fiber fragmentation in only one case [2]. Clinical and histopathological findings of all the cases reported up to date as PC are summarized in **Table 1**, including those of Gougerot [6-8]. Cases in which elastic fibers were altered are summarized in **Table 2**.

Differential Diagnosis

Some conditions such as eczema craquele (EC), tinea versicolor (TV), acanthosis nigricans (AN), and confluent reticular papillomatosis of Gougerot-Carteaud (CRP) usually do not represent diagnostic difficulties but can look alike. Certain similarities do really exist with EC, but this process appears during winter, is mostly in older people, and affects the trunk or extremities with a monomorphous presentation characterized by dry crisscrossing

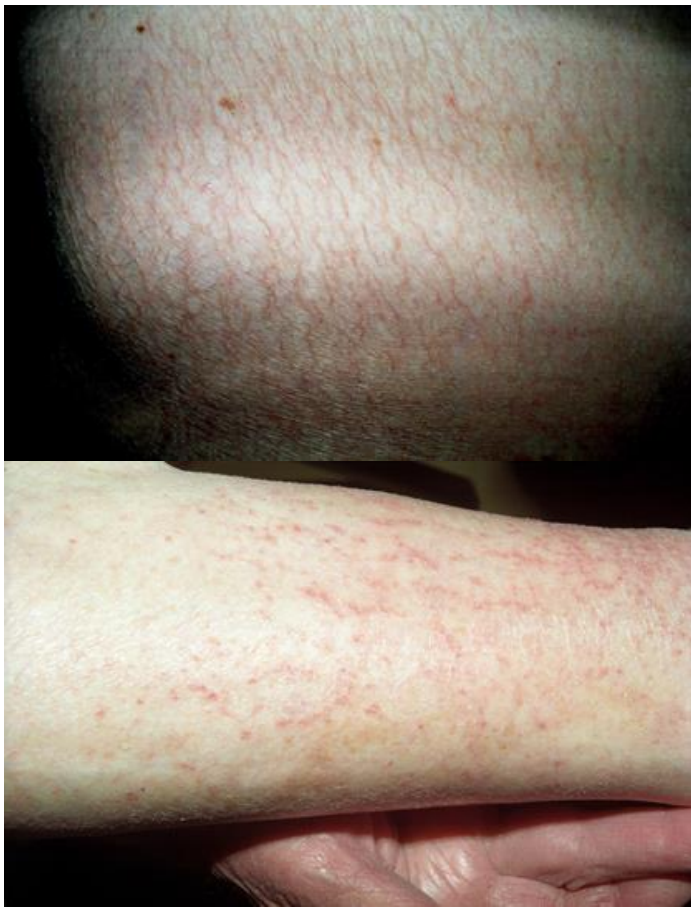

Figure 10. *Eczema craquele: dry crisscrossing scales similar to fine cracks of porcelain, affecting trunk or extremities.*

scales, similar to the fine cracks of porcelain (**Figure 10**).

Confluent reticular papillomatosis of Gougerot-Carteaud may masquerade as tinea versicolor [13] or be associated with it [14]. It has also been debated whether acanthosis and CRP are similar entities but most believe they are distinct [15]. As with AN, CRP may also appear in obese and hyperinsulinemic patients, but differs in location, usually in axillae and neck in AN; AN also generally has a darker appeareance. Confluent reticular papillomatosis of

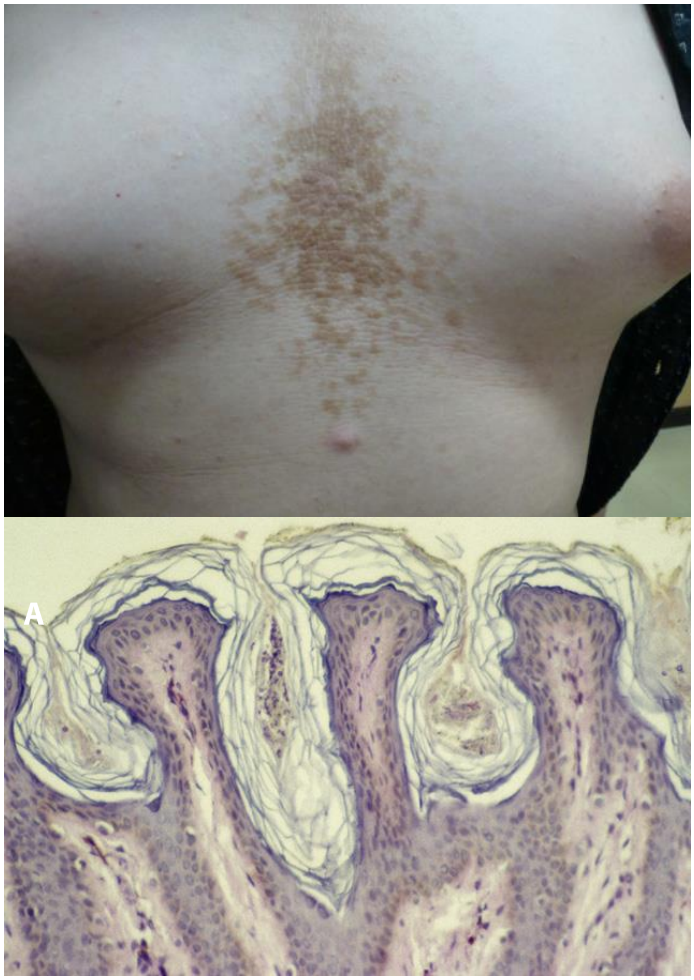

Figure 11. ***A)*** *Confluent and reticulated papillomatosis: papular brownish lesions attenuated towards healthy skin, in the midline of anterior trunk.* ***B)*** *Confluent and reticulated papillomatosis: acanthosis, papillomatosis with finger-like projections into the stratum corneum; compact hyperkeratosis; pigmentation of the basal layer; numerous yeasts in the stratum corneum. H&E, 200×.*

Gougerot-Carteaud appears with coalescent papules in the mid chest, arranged with a reticulated pattern peripherally [16]; histopathological findings show more acanthosis, papillomatosis, and pigmentation in AN than in CRP [17].

The main condition in the differential diagnosis of PC is CRP, with which it shares several similarities. As clinical differentiating features the latter has a mostly papular appearance, with more hyperpigmented or brownish lesions, which appear confluent and reticulated and are often attenuated towards areas of normal skin. Confluent reticular papillomatosis of Gougerot-Carteaud tends to be distributed over the anterior and posterior midline of the trunk, axillae, neck, and submammary region (**Figure 11A)**. Their main differences along with features of AN are summarized in **Table 3**. Histopathological findings of CRP show acanthosis, variable papillomatosis, and fingerlike projections into the stratum corneum, which shows compact hyperkeratosis; in the basal layer increased melanin deposits can be found (**Figure 11B)**.

Although it has been proposed that negative stain for fungus is one of the diagnostic criteria for CRP [18], the finding of numerous yeasts without filamentous forms has been reported in some cases of CRP [19,20], up to 25% of the cases in the series of Davis. The role of other organisms such as the bacteria *Dietzia spp*, as suggested by Scheinfeld et al. has yet to be established [15].

Some authors believe that PC is a separate entity from CRP and could represent a nevoid and hereditary defect of keratinization [3]. However, for some authors, given their similarities, PC may be considered a variant or transitional form of CRP [2,21]. Nevertheless, the second of our reported cases presented with long standing lesions over 10 years and never evolved to CRP, a similar experience as has been seen in many other cases with no transitional forms [2,5,9,10-12] and lesions lasting from 2 to 15 years.

In a recent study of 20 cases of CRP with mixed ethnicity from Bogotá (Colombia), [22], the authors described the presence of thin shiny plaques with wrinkled cigarette paper-like appearance that gave the impression of linear pseudo-striae. According to these authors, this finding is unknown by most clinicians. In our opinion, these types of lesions described as pseudo-striae could indicate the possible relationship of CRP with PC, which was pointed out in 1957 in five African American patients [2]. However, in the study from Bogotá, the biopsy sites were not clearly stated and one does not know if these were taken from pseudo-striae or from typical lesions of CRP. Histopathological findings varied from acanthosis to atrophy. Their histopathological image illustrated the classical features of CRP.

In another case recently reported as CRP an 18-year-old man was described with a vertically rippled and keratotic eruption on the trunk, with brown and rippled streaks on the epigastric area that spread to the chest, back, and waist [23]. The clinical and histopathological figures mostly resembled those of PC, in our opinion, rather than CRP. It has been stated that CRP can also manifest as atrophic macules with a shiny appearance resembling cigarette paper [15], with clumped or frayed elastic fibers in the lower dermis in some cases [24].

Treatment

Spontaneous improvement has been described in one case, as well as temporary response to ultraviolet light [2], or total improvement after the use of vitamin A in doses of 50,000U/day [10,11]. Partial improvement with topical treatment with 5% lactic acid has been observed [3]. In our experience, two cases improved after intake of minocycline or lymecycline. In both, improvement was noted within the first month of treatment.

Conclusion

The original presentation of PC was in the neck, hence its designation as pseudoatrophoderma colli. The term has persisted, although other body areas can be affected. The process bears some similarities with confluent and reticular papillomatosis. Like the latter, it has been proposed that PC is related to an acquired keratinization disorder of unknown origin. But in some cases, other distinctive features may be the fragmentation and irregular thickness of dermal elastic fibers. Nevertheless, these abnormal elastic fibers could explain the fine wrinkling appearence of the skin in this rare entity. Until now, it has not been clearly demonstrated whether or not PC and CRP are separate processes, albeit their clinical and histopathological differences. We also do not believe that it is clear if PC is a distinct entity or just a variant of CRP, or if they could occur simultaneously with different clinical/histopathological features. We suggest that further studies comparing PC and CRP that should include biopsies of both papular and atrophic lesions with staining for elastic fibers. Minocycline and lymecycline should be considered as the first choice of treatment.

Potential conflicts of interest

The authors declare no conflicts of interest.

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Table 1. *Summary of cases which may be considered or described as pseudoatrophoderma colli.*

Ref	Sex Age	Location of lesions	Clinical description	Histopathology	Treatment
[6]	ND	Upper trunk	Numerous brilliant macules without desquamation. Initial lesion was a brilliant point which evolved to scaly rose elements defined as 'brilliant macules'	Not done	No response to iodide, chrysarobin, salicylic acid, mercury
[7]	F ?	Upper trunk, abdomen	Brilliant rounded or oval macules, 2-10 mm, isolated or confluent, no desquamation, depression or atrophy. Persistent and stable	Slight hyperkeratosis without parakeratosis. Slight epidermal atrophy, dermal sclerosis. No fungus	No
[8]	F 29	Upper trunk	Multiple lesions Upper trunk. Oval lesions 0.5-5 mm, 10-20 mm with confluence	Hyperkeratosis without parakeratosis. Epidermal atrophy. Partial destruction of elastic tissue. Condensation of collagen fibers	No
[1]	F 19	Neck, upper thorax	Atrophic appearance, the apparently depigmented portions were glossy, and apparently pigmented regions were epidermal thrown up into tiny folds. Under tension dull areas were covered with fine scales; upon release wrinkling appeared again. Shiny pseudo-atrophic plaques were arranged in vertical direction	Stratum corneum thick and loose, granulosum one layer, stratum mucosum appeared thickened and thinned in different sections. Elastic fibers less conspicuous in areas of loosened collagen	Cleared once with UV Recurrence 2 months later
[5]	F 20 F 19	1. Neck, thorax 2. Breasts	1. Dark brown macules with fine wrinkles like collodion 2. Brown macules with wrinkles which disappeared on tension. Pale patches with slight atrophy in the center of involved areas	Waviness of the epidermis. Loosely adherent stratum corneum. Moderate edema in corium. No fungus	No response to local remedies

[10]	F 35	Chest, breasts, back, arms, neck, upper abdomen	Pink, fine scaly macules with superficial atrophy	Slight thickening of stratum corneum. Waviness of the epidermis. Slight thinning of stratum mucosum. Perivascular infiltration of corium. No fungus	No
[11]	F 31	Neck, upper trunk	Brownish-gray oval area, hyperkeratotic appearance. Discrete and grouped macules. Reticulated appearance. Slight brownish. Crinkly, atrophic surface. Darker brown beneath breast	Marked unevenness of epidermis. Acanthosis and thinning in some areas. Loose hyperkeratosis. Marked vacuolization. Moderate subpapillary edema. Perivascular lymphocytic infiltration	Disappearance with X-ray and oral vitamin A. Relapse treated similarly, without response
[9]	F 45	Neck, back, chest, arms, mons veneris	Wrinkled, atrophic, whitish buff, glossy surface	Thinned epidermis. Parakeratotic corneal layer. Minimal lymphocytic infiltrate. Elastic fibers without changes. No fungus	No effects after cortisone ointment and high-potency estrogen cream
[12]	M 34	Neck, upper trunk	Atrophic circumscribed pink macules, 3-7 mm oval; coalescent in arcuate patches Erythematous patches, fine scales, oval glossy plaques, pink wrinkled surface	Loosely woven hyperkeratosis. Vacuolization of epidermal cells. Perivascular lymphocytic infiltrate. Elastic fibers were normal	No
[3]	F 22	Neck and back (similar lesions in her father and sister)	Hyper and hypopigmented atrophic lesions, similar to her sister. Father with fine macules, papules and scaling of the neck	Hyperkeratosis, irregular mild acanthosis, thin granular layer, mild perivascular monocytoid infiltration in upper corium	10% lactic acid ointment. Relief of pruritus. Treated lesions flat and smooth
[4]	M 20	Neck, trunk, axillae	Neck: grayish papules. Trunk: lenticular, squamous and brilliant lesions, like cigarette paper. Axillae: new lesions with pink-grayish color and atrophic appearance. Confluent intermammary and interscapular lesions, similar to pityriasis versicolor	Hyperkeratosis, papillomatosis, acanthosis. Papillar dermis with dilated vessels and slight inflammatory perivascular mononuclear infiltrate. No fungus	No

ND: not described.

Table 2. *Cases of Pseudoatrophoderma colli in which elastic fibers were altered.*

Ref	Sex, age	Clinical features	Histopathology
[1]	F, 19	The condition looked like a pigmentary disturbance. The lesions appeared atrophic. On close examination the depigmented areas were glossy, whereas the apparently pigmented zones consisted of tiny folds. Under tension the lesions were less contrasted between the shiny and dull areas, which were covered with fine scales; when released the wrinkling reappeared	Thick and loose stratum corneum. One layer of cells in stratum granulosum. Irregular thickness of epidermis stratum mucosum. Elastic fibers less noticeable in areas of loosened collagen
Present Case 2	F, 27	Multiple whitish, scaly lesions with atrophic appeareance, mainly arranged in a vertical orientation. They were present and stable for 10 years and were located in abdomen and axillae	Irregular acanthosis, loose stratum corneum. Irregular thickness and fragmentation of elastic fibers

Table 3. *Main differences between pseudoatrophoderma colli (PC), confluent and reticulated papillomatosis (CRP) and acanthosis nigricans (AN).*

	PC	CRP	AN
Age	15-36	Late teens or early twenties	Any age
Sex	Women predominantly	Male predominance 1.4 to 2.6/1	Both sexes
Distribution	Neck, upper trunk, breast, axillary pillars, scapularregions, outer arms	Upper trunk, axillae, submammary creases, interescapular area, nape of the neck. Sometimes antecubital or popliteal fossae	Axillae and neck
Morphology of lesions	Brown macules with atrophic appearance. Thin scaly surface as cigarette paper. Sometimes dirty - looking skin with grayish pigmentation. Shiny pseudo-atrophic plaques arranged in vertical direction	Brown keratotic papules and plaques, centrally confluent and reticulated peripherally. Lesions often attenuated towards healthy skin	Hyperpigmented velvety appearance
Histopathology	Loose thickened stratum corneum. Spinous stratum with variable thickness. Irregular acanthosis. Thin granular layer. Dermis with slight lymphocytic infiltrate	Basket-weave hyperkeratosis. Papillomatosis with finger-like upward proyection of dermal papillae, focal acanthosis limited to areas of rete ridge elongation. Increased basal melanin pigmentation. Increased number of melanosomes in the horny layers. Invagination of hyperkeratosis into the epidermis. Mild perivascular mononuclear infiltration	Higher degree of acanthosis and papillomatosis. Hyperpigmentation related to increased melanogenesis
Other	Good response to minocycline	Good response to minocycline	Increased body mass index, insulin resistance or diabetes