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CLINICAL CASE

Hyperpigmented lesions with acquired atrophy following Blaschko lines in a patient diagnosed with localized scleroderma

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Abstract

Background: *Linear atrophoderma of Moulin (LAM) is a dermatosis that affects children and adolescents characterized by hyperpigmented and atrophic linear lesions following Blaschko lines. So far, less than 50 cases have been published. Therefore, it is a rare entity with unknown etiology and a chronic and self-limited course. Histologically, it is described as hyperpigmentation of the basal layer without the involvement of the dermis and subcutaneous tissue. No specific treatment exists currently. Localized scleroderma is a chronic and progressive autoimmune connective tissue disorder that affects the skin and adjacent tissues, characterized by abnormal collagen deposition and alteration in elastic fibers, blood vessels, and annexes. No reports have been published on the coexistence of localized scleroderma and LAM.* **Case report:** *We describe the case of an 11-year-old male with a clinical diagnosis of linear scleroderma since 5 years of age. Four years later, the patient developed atrophic and hyperpigmented lesions following Blaschko lines in the posterior trunk. A biopsy of both dermatoses was performed: the trunk showed epidermis with hyperpigmentation of the basal layer, and within the dermis, no alteration in the collagen bundles; the forearm biopsy corroborated scleroderma. Based on the clinical-pathological correlation, LAM coinciding with localized linear scleroderma was diagnosed.* **Conclusions:** *LAM is an infrequent entity by itself. Moreover, its coexistence with sclerodermiform syndrome has not been reported in the indexed literature.*

Keywords: *Atrophoderma of Moulin. Scleroderma. Linear. Blaschko.*

Lesiones hiperpigmentadas con atrofia adquiridas siguiendo las líneas de Blaschko en un paciente con diagnóstico de esclerodermia localizada

Resumen

Introducción: *La atrofodermia lineal de Moulin (ALM) es una dermatosis que afecta a niños y adolescentes, caracterizada por lesiones lineales hiperpigmentadas y atróficas que siguen las líneas de Blaschko. Al día de hoy, se conocen menos de 50 casos en la literatura, por lo que se considera una enfermedad rara, de etología aún desconocida, que presenta un curso crónico y autolimitado. Histológicamente se describe hiperpigmentación de la capa basal sin afección de las dermis ni*

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del tejido subcutáneo. No existe tratamiento específico. La esclerodermia localizada es un trastorno autoinmunitario del tejido conectivo, de curso crónico y progresivo, que afecta la piel y los tejidos adyacentes, caracterizada por un depósito anómalo de colágeno y una alteración en las fibras elásticas, los vasos sanguíneos y los anexos. No existen informes sobre la coexistencia de esclerodermia localizada y ALM. **Caso clínico:** *Se describe el caso de un paciente de sexo masculino de 11 años con diagnóstico clínico de esclerodermia lineal desde los 5 años. Cuatro años después desarrolló lesiones atróficas e hiperpigmentadas que siguen las líneas de Blaschko en la parte posterior del tronco. Se realizó una biopsia de ambas dermatosis: la de tronco mostró epidermis con hiperpigmentación de la capa basal; la de dermis, sin alteración en los haces de colágeno; y la de antebrazo corroboró la esclerodermia. Basándose en la correlación clínico-patológica, se concluyó el diagnóstico de ALM en coexistencia con esclerodermia localizada lineal.* **Conclusiones:** *La ALM es una afección infrecuente por sí misma. La coexistencia con procesos esclerodermiformes no ha sido reportada en la literatura indexada.*

Palabras clave: *Atrofodermia de Moulin. Esclerodermia. Lineal. Blaschko.*

Introduction

Localized scleroderma or morphea is a chronic, slowly progressive, autoimmune connective tissue disorder that affects the skin and adjacent tissues[1]. The etiopathogenesis is not yet fully understood, but multiple factors that increase pro-inflammatory cytokines are most likely involved, leading to increased collagen production and extracellular matrix deposition.

Furthermore, in the first two decades of life, hyperpigmented lesions and skin atrophy following Blaschko lines are distinctive features of linear atrophoderma of Moulin (LAM)[2]. LAM is a rare condition of unknown etiology and chronic and self-limited course, with no specific treatment to date.

We report the case of a male patient with a diagnosis of linear scleroderma, who subsequently developed atrophic and hyperpigmented lesions following Blaschko lines.

Clinical case

We report the case of an 11-year-old male with no relevant clinical history. At 5 years of age, dermatosis appeared in the extremities of the right hemibody, with hyperpigmented plaque-type lesions and sclerosis. Local rheumatology and dermatology services evaluated the patient and diagnosed clinical linear scleroderma. Subsequently, topical and systemic steroid treatment was given with partial improvement. Then, the patient was referred to the pediatric rheumatology of our unit. Laboratory studies, including rheumatoid factor, complement C3, C4, and antinuclear antibodies, were made, which were negative. Treatment with methotrexate and colchicine was started.

In 2017, the patient was evaluated for the first time in the pediatric dermatology department, clinically confirming the diagnosis of linear scleroderma. Antibodies for *Borrelia* were requested but reported negative. Due to limited mobility secondary to scleroderma, he was referred to physical medicine and rehabilitation. At 9 years of age, during the clinical follow-up, dermatosis localized to the trunk affecting the posterior and right lateral part of the patient was observed, characterized by discretely atrophic, hyperpigmented linear plaques that followed Blaschko lines, with an absence of induration or evidence of sclerosis (Fig. 1). These lesions were asymptomatic, without inflammation. A biopsy of both dermatoses was taken with the diagnostic proposal of linear atrophoderma of Moulin in a patient with localized scleroderma (Fig. 2). The diagnosis was confirmed. To date, the trunk lesions have remained without progression, clinically without inflammation or sclerosis.

Concerning scleroderma, no more lesions have appeared. Immunosuppressive-based treatment was suspended in December 2019. Finally, the patient continues with rehabilitation therapy, obtaining a functional improvement in the elbow joint and right foot mobility.

Discussion

LAM is a rare disease first described by Moulin in 1992, who published a series of five cases describing hyperpigmented and atrophic lesions[3]. It is a rare, self-limited dermatological disorder that manifests in childhood and adolescence, with no family history of involvement. The exact etiology is currently unknown, but it is theorized to be a mosaic resulting from a post-zygotic somatic mutational event during early embryogenesis[3]. This predisposition, together with external factors not yet well established, is responsible for this dermatosis. Clinically, LAM is characterized by a unilateral band-like or linear dermatosis of variable size following the Blaschko lines, hyperpigmented and atrophic[4]. The lesion does not present induration or sclerosis, it is unilateral, and its usual topography is the

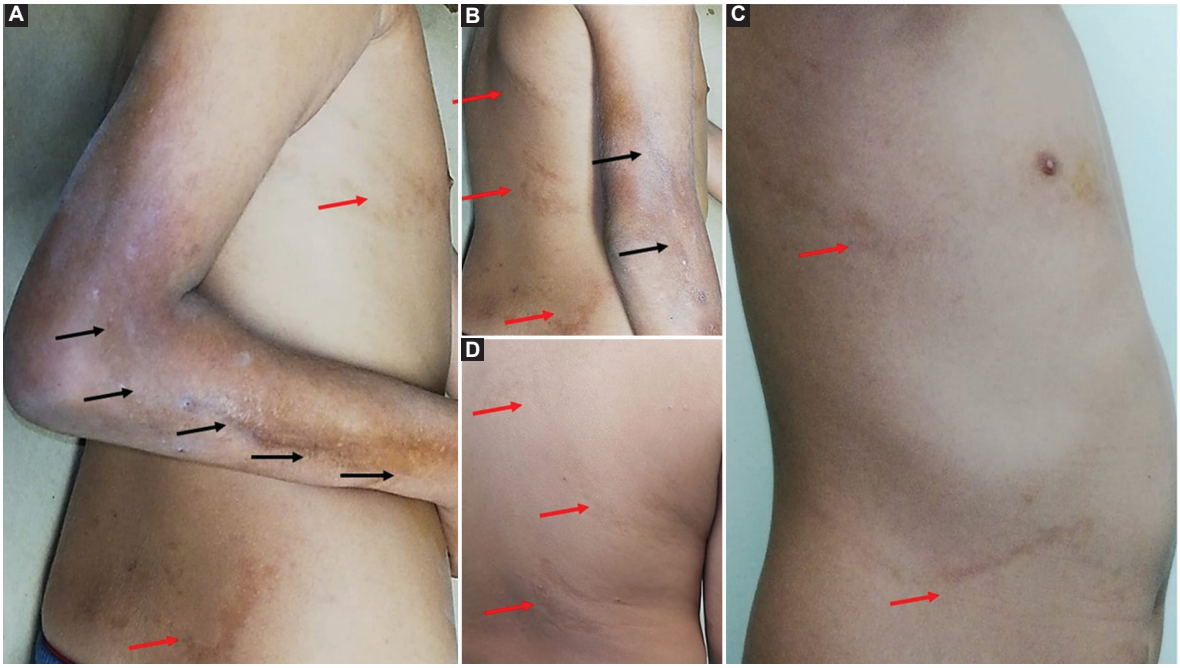

Figure 1. A. Indurated, hyperpigmented plaque with sclerosis and atrophy on right arm and forearm, following a linear trajectory (Black arrows) **B.** Limitation of full elbow extension is observed. **C and D.** Hyperpigmented linear plaques, slightly atrophic following Blaschko lines in the posterior and lateral trunk in the right hemibody (red arrows).

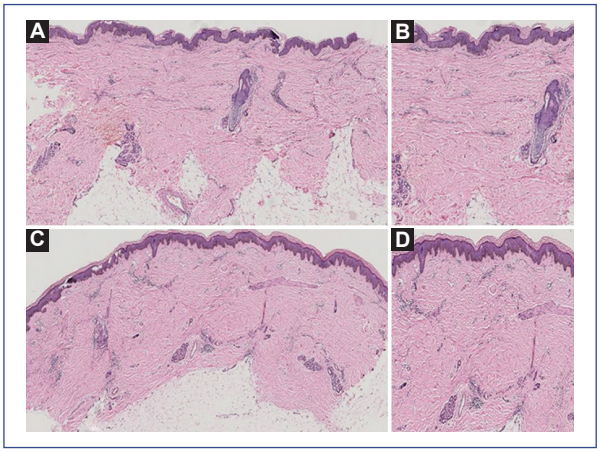

Figure 2. A and B. Epidermis with hyperpigmentation of the basal layer; dermis with moderate perivascular inflammatory infiltrate, no alteration in the collagen bundles. **C and D.** Epidermis with hyperpigmentation of the basal layer dermis with lymphocytic and neutrophilic inflammatory infiltrate at the perivascular level, irregular dense collagen foci with atrophy, and scarcity of adjacent tissue. The changes extend to the subcutaneous cellular tissue.

trunk and extremities[3,5]. Its course is asymptomatic with an absence of systemic involvement or progression[6]. Histopathological findings are controversial. Originally it was described in five cases; only three had a biopsy, where hyperpigmentation was identified without other changes in the epidermis or dermis[7]. Later reports described hyperpigmentation in the lower epidermis, with perivascular lymphocytic infiltrate in the dermis, slightly thickened collagen bundles, or compact dermis[5]. The recommendation is to perform a skin biopsy of both healthy skin and the lesion to establish the histopathological diagnosis since, in some cases, the histopathological findings are very subtle[8].

In 2008, López et al.[9] conducted a review of reported cases up to that date, proposing the following diagnostic criteria:

1. Onset from infancy to adolescence
2. Development of hyperpigmented, slightly atrophic, unilateral lesions following Blaschko lines on the trunk or extremities
3. Lack of previous inflammation and subsequent absence of scleroderma
4. Stable and non-progressive clinical course with no pattern of remission
5. Histologic findings showing hyperpigmentation of the epidermis' basal layer and normal dermis with unaffected connective tissue and elastic fibers

López et al. reviewed 23 cases, of which ten had collagenization of the dermis, and four had no biopsy, meaning that less than 50% of the cases described were those expressing changes at the epidermal level[9].

In the literature, we found 46 cases. Only five patients (10.6%) strictly met the criteria proposed in 2008. We identified reports that showed an atypical clinical expression due to the presence of erythematous plaques, telangiectasias, dissemination to trunk and extremities, and histopathological changes in 34 cases (72%). In these cases, findings in the dermis were described, such as thickening and compacted collagen bundles (Table 1)[2-4,6-37].

In different reports of LAM, we identified a diversity of manifestations that do not fall under the criteria for the disease. For example, Yan et al., in 2016, described a patient with different alterations, including positive antinuclear antibodies, ribonucleoprotein, and anti-SM antibodies[36]. In 2012, Afshar et al.[32] reported that the clinical presentation progressed over 46 years. Other patients were adults[3,7,32] presenting with inflammatory-type[11,18,34], bilateral[17,20,26] lesions associated with lentiginosis[26,33], which is atypical for the clinical picture in LAM. From the histopathological point of view, collagen alterations have been identified in a large percentage of cases. This information raises the question of whether the reports were LAM cases or other conditions.

The case described here presented localized linear scleroderma with subsequent appearance of hyperpigmented and atrophic lesions, asymptomatic, with histology supporting the diagnosis of LAM. This coexistence is exceptional, with no other previous report to date.

Regarding treatment, various drugs have been used, ranging from topical steroids, platelet-rich plasma, PUVA therapy [psoralen (P) and long-wavelength ultraviolet radiation (UVA)], oral potassium, and high-dose penicillin, without optimal results[1,6]. A case treated with methotrexate at a dose of 20 mg per week was reported, where improvement in skin color and texture was clinically demonstrated after 6 months of treatment[1]. However, further studies are required for its recommendation.

The prognosis of this disease is excellent for life and self-limited. There is no evidence of long-term progression[3].

Differential diagnoses of LAM include conditions affecting Blaschko's lines such as linear and whorled nevoid hypermelanosis, *incontinentia pigmenti*, lichen striatus, epidermal nevi, and atrophoderma of Pasini and Pierini (APP)[5,6,37]. A review of the transcendent conditions to establish the diagnosis of LAM is made.

Scleroderma is a fibrosing dermatosis limited to the skin and subcutaneous tissue. Two subtypes are distinguished: systemic scleroderma with associated visceral involvement and sclerodactyly with Raynaud's phenomenon or localized scleroderma or morphea limited to the skin and underlying tissues[38]. It occurs predominantly in females and affects children and adults similarly. The peak incidence is between 2 and 14 years of age in children; in adults, between 40 and 50 years of age. Patients with scleroderma usually have a family history of this pathology or other autoimmune disorders[39]. The etiology is not yet well established, but several elements involved in the fibrosis pathway are recognized. Among these elements are vascular damage or anomalous activation of T lymphocytes with abnormal tissue production by fibroblasts, which could interact with external triggering factors, such as trauma, insect bites, vaccinations, and exposure to X-rays, resulting in the development of sclerosis[7,40].

The clinical classification depends on the consulted literature, but it is usually divided into circumscribed morphea with linear expression (including trunk and extremities, *en coup de sabre*, and Parry-Romberg syndrome), generalized, pansclerotic, and the mixed variant[38].

In the early stages of the disease, differentiation of scleroderma and systemic sclerosis is impossible, as both present perivascular lymphocytic infiltration in the reticular dermis and endothelial inflammation. They also share standard features such as thickening of collagen bundles extending into the subcutaneous tissue, loss of eccrine glands, and involvement of blood vessels in later stages[38].

When there is head and neck involvement, a periodic ophthalmologic examination is indicated to rule out third cranial nerve damage since it may be irreversible[38].

The clinical picture consists of an indurated plaque of insidious onset, with an active border of violaceous or erythematous coloration and a slightly whitish center that progresses to sclerotic tissue[41]. Later, the plaque becomes pigmented with loss of adjacent tissue. Extracutaneous involvement is exceptional. However, it may be associated with myalgias, arthralgias, fatigue, and central nervous system fibrosis, present in up to 22% of cases[38]. The evolution is variable and tends to progress, especially in childhood-onset cases, although it usually becomes inactive after 3-5 years[42].

Multiple treatment options include phototherapy, topical or systemic steroids, topical calcipotriol, oral

Table 1. Characteristics of linear atrophoderma of Moulin cases reported in the literature

Patient	Author (year)	Sex	Age of onset (years)	Clinical picture	Topography	Histology	DXC (X/5) and OC
1	Moulin et al. (1992)[7]	M	8		Left trunk	Basal hyperpigmentation	5/5
2		F	7		Right trunk	Basal hyperpigmentation	5/5
3		M	15		Right trunk	Basal hyperpigmentation	5/5
4		M	20		Left trunk	No biopsy	4/5
5		M	6		Trunk and left arm	No biopsy	4/5
6	Baumann et al. (1994)[10]	M	22		Trunk and right arm	Epidermis: basal ballonization Dermis: perivascular lymphocytic infiltrate, increased collagen	3/5
7	Larregue et al. (1995)[11]	M	15		Left trunk	Dermis: collagenization	4/5
8	Wollenberg et al. (1996)[12]	F	5		Right trunk and arm	Epidermis: atrophy Dermis: perivascular lymphocytic infiltrate and increased collagen	4/5
9	Artola-Igarza et al. (1996)[13]	F	16		Left trunk	Epidermis: acanthosis and basal hyperpigmentation Dermis: perivascular lymphocytic infiltrate and increased collagen	4/5
10	Braun and Saurat (1996)[14]	M	16		Trunk and left abdomen	Increased collagen fibers	4/5
11	Cecchi and Giomi (1997)[15]	F	12	*Classic	Trunk and right arm	Basal hyperpigmentation	5/5
12	Rompel et al. (2000)[16]	F	17		Trunk and right gluteal region	Epidermis: vacuolar degeneration of the basal area Dermis: increased collagen	4/5
13	Browne and Fisher (2000)[17]	M	10	Classic, also erythematous patches with papules with linear distribution	Trunk and extremities, bilateral	Epidermis: thinned Dermis: prominent vessels and increased collagen	3/5
14	Martin et al. (2002)[18]	M	9		Left trunk	Increased collagen	4/5
15	Miteva and Obreshkova (2002)[19]	F	16	Classic + telangiectasia	Arm, gluteal region, and right leg	Increased collagen	4/5

(Continues)

Table 1. Characteristics of linear atrophoderma of Moulin cases reported in the literature *(continued)*

Patient	Author (year)	Sex	Age of onset (years)	Clinical picture	Topography	Histology	DXC (X/5) and OC
16	Utikal et al. (2003)[20]	M	23	Classic + telangiectasia	Trunk and extremities, bilateral	Perivascular lymphocytic infiltrate and edema	2/5
17		F	13	Erythematous lesions with atrophy and telangiectasia following Blaschko lines	Trunk and extremities, bilateral	Dermis: edema, perivascular infiltrate	3/5
18	Danarti et al. (2003)[21]	F	14		Left hemibody	Perivascular lymphocytic infiltrate	4/5
19		F	24		Left arm, trunk, and abdomen	No biopsy	3/5
20		F	38		Left thigh	Epidermis and dermis without relevant findings	4/5
21	Miteva et al. (2005)[22]	F	15		Gluteal region and left iliac crest	No biopsy	4/5
22	Atasoy et al. (2006)[23]	M	9		Trunk and left arm	Increased collagen	4/5
23	Peching et al. (2005)[4]	F	19	Classic	Trunk and left thigh	Epidermis: hyperkeratosis, orthokeratosis, basal hyperpigmentation. Dermis: disorganized collagen with loss of configuration.	4/5
24	Atasoy et al. (2006)[23]	M	16	Classic + telangiectasia	Arm and right trunk	Epidermis: atrophy Dermis: destruction of collagen fibers, vascular ectasia, perivascular lymphocytic infiltrate.	3/5
25	Zampetti et al. (2008)[24]	F	37		Arm and left trunk	Basal hyperpigmentation, thickening of collagen bundles	3/5
26	Cecchi et al. (2008)[25]	M	9		Neck	Basal hyperpigmentation, perivascular lymphocytic infiltrate	4/5
27	López et al. (2008)[9]	M	16	Classic	Right arm	Basal hyperpigmentation	5/5
28	Özkaya et al. (2010)[26]	F	18	Classic + lentiginosis	Bilateral	Epidermis: acanthosis, basal hyperpigmentation, decrease of elastic fibers.	3/5
29	Ripert and Vabres (2010)[27]	F	14		Left trunk	Basal hyperpigmentation, perivascular lymphocytic infiltrate	4/5

(Continues)

Table 1. Characteristics of linear atrophoderma of Moulin cases reported in the literature *(continued)*

Patient	Author (year)	Sex	Age of onset (years)	Clinical picture	Topography	Histology	DXC (X/5) and OC
30	Schepis et al. (2010)[28]	M	14	Classic	Left trunk	Basal hyperpigmentation, dilated vessels, edema, perivascular lymphocytic infiltrate	4/5
31	Tukenmez Demirci et al. (2011)[29]	F	39		Left side of the neck	Basal hyperpigmentation, proliferation of vessels, macrophages, and inflammatory infiltrate	3/5
32	Norisugi et al. (2011)[30]	M	26	Classic	Trunk and right leg	Basal hyperpigmentation, thickening of collagen bundles, perivascular inflammatory infiltrate	3/5
33	Patsatsi et al. (2013)[31]	F	17	Classic	Left trunk	Epidermis: thinned, basal hyperpigmentation, increased collagen fibers	3/5
34	Zaouak et al. (2014)[2]	F	11	Classic	Arm, trunk, and right leg	Basal hyperpigmentation, perivascular lymphocytic infiltrate	4/5 Treated with methotrexate with clinical improvement
35	Afshar et al. (2012)[32]	M	20	Pruritic pink areas that later become hyperpigmented and atrophic while remaining asymptomatic	Trunk and left arm	Epidermal atrophy, Thinned collagen bundles and fragmented elastic fibers	3/5 Lesions progressed over 46 years
36	Yücel et al. (2013)[33]	F	23	Classic + lentiginosis	Right leg	Melanophages and perivascular lymphocytic infiltrate	3/5
37	Villani et al. (2013)[3]	M	8	Classic	Thigh and right gluteal area	Basal hyperpigmentation, perivascular inflammatory infiltrate, thickened collagen bundles	4/5
38		F	6	Classic	Abdomen and right leg	No biopsy	4/5
39		F	9	Classic	Left back	No biopsy	4/5
40		M	20	Classic	Left arm	Pigmentation of the basal layer, perivascular inflammatory infiltrate, thickened collagen bundles	3/5

(Continues)

Table 1. Characteristics of linear atrophoderma of Moulin cases reported in the literature *(continued)*

Patient	Author (year)	Sex	Age of onset (years)	Clinical picture	Topography	Histology	DXC (X/5) and OC
41	De Golian et al. (2014)[34]	M	10	Classic	Left side	Normal epidermis Thickening of collagen bundles, Plasma cells, perivascular lymphocytic infiltrate	4/5
42	Zahedi et al. (2015)[35]	F	10	Classic	Right arm	Basal hyperpigmentation, perivascular inflammation	4/5
43	Yan et al. (2016)[36]	M	15	Classic	Arm and trunk, ipsilateral	Epidermis: acanthosis, basal hyperpigmentation, perivascular lymphocytic infiltrate, and increased collagen	4/5 Antibodies (+)
44	Tan and Tay (2016)[8]	F	11	Classic	Arm, trunk, and right leg	Perivascular lymphocytic infiltrate, collagen bundle compaction	4/5
45	Zhang et al. (2020)[6]	F	10	Classic	Trunk and leg	Basal hyperpigmentation, perivascular inflammatory infiltrate	4/5
46	Present case (2020)	M	9	Classic	Trunk	Basal hyperpigmentation, perivascular lymphocytic infiltrate	5/5

*Classic: hyperpigmented, atrophic skin areas following the Blaschko lines.
DXC, diagnostic criteria; F, female; M, male; OC, other characteristics.

calcitriol, tacrolimus, topical pimecrolimus, methotrexate, mycophenolate mofetil, intralesional interferon-gamma, cyclosporine, D-penicillamine, imiquimod, and penicillin. In pediatric patients, the therapies with the highest degree of evidence are phototherapy and pulse regimen with corticosteroids and methotrexate[42].

Moreover, APP is a rare dermatosis characterized by mild dermal atrophy affecting adolescent and young adult women[43,44]. It was first reported in 1923 by Pasini as "progressive idiopathic atrophoderma" and was described later in 1936[45]. It predominates in young women in the second or third decade of life, has a predilection for the trunk, and clinically presents as a plaque, single or multiple, atrophic with well-defined borders, hyperpigmented, non-indurated, which can vary in size and tone, and tends to be bilateral. APP may be accompanied by pruritus, pain, or paresthesias[45]. About 100 cases of APP have been described in the literature. Although the cause is still unknown and no genetic factors have been identified, Pasini and Pierini reported familial atrophoderma[43,45]. Some authors have related it to *Borrelia* infection[44].

Regarding histology, the most characteristic finding is a decrease in dermal thickness and collagen changes, including atrophy, sclerosis, fragmentation, and hyalinization. The elastic fibers show reduction and fragmentation. The adjacent tissues do not show alterations[32]. To date, no definitive treatment is available. However, when positive antibodies for *Borrelia* are present, it can be managed with tetracyclines, although the response is partial[43,45].

The coexistence of different sclerodermiform conditions is recognized. Most reports describe the association of morphea with lichen sclerosus and atrophic lichen[42,46-48] and of morphea with APP[26]. However, in the latter case, several authors share the theory that

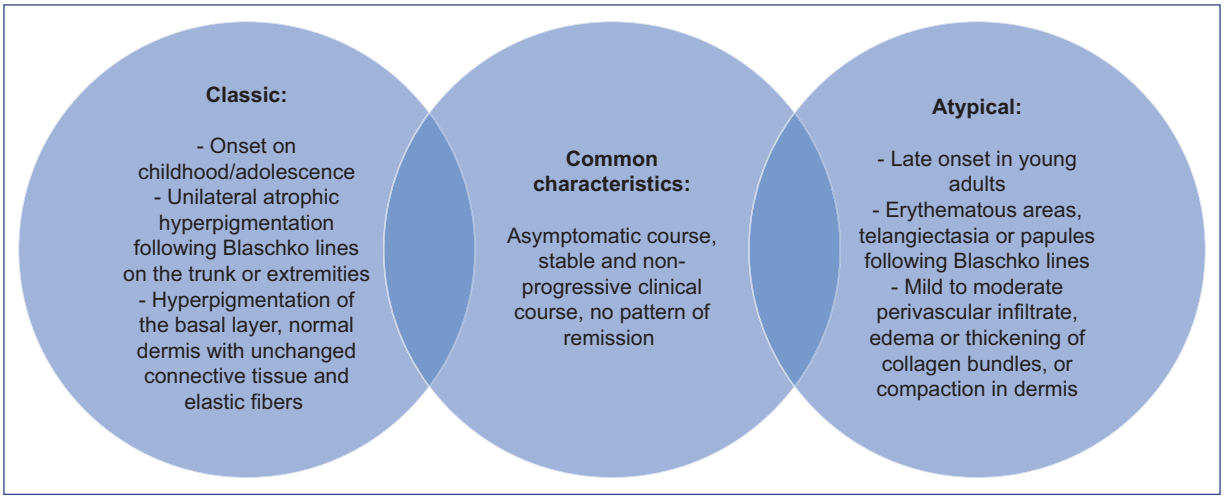

Figure 3. Interaction of features obtained from literature reports.

both conditions are part of the same spectrum or that APP is an abortive variant of morphea[37,49].

Currently, the possibility has been raised that LAM, APP, and linear scleroderma are part of the same spectrum, as they all share some clinical and histological manifestations[34,49].

Finally, from the reported cases, we compiled and analyzed the following common features: (a) dermatosis characterized by hyperpigmented patches and mild atrophy following Blaschko lines; (b) children and adolescents; (c) on trunk and extremities, unilateral; d) epidermis with hyperpigmentation, scarce perivascular lymphohistiocytic inflammatory infiltrate, dermis unchanged, or the slight thickening of collagen or compaction without alteration of elastic fibers; e) asymptomatic course, without clinical progression to induration or involution. This information represents an opportunity to expand the previously proposed diagnostic criteria for LAM, adding a subdivision according to the findings (Fig. 3).

LAM is a controversial entity. Of the 46 published cases, only five fully meet the diagnostic criteria already established. Furthermore, these criteria are exceeded, as they are not strictly met, mainly due to histopathological findings. In the literature, LAM is a dermatosis without changes in the dermis. However, more than 70% of the reports show alterations in the dermis: collagenization, thickening of bundles, edema, compacting of collagen bundles, or decrease of elastic fibers. Therefore, we face a dermatosis that shows the importance of the clinical-pathology-evolutionary correlation. Overall, the above provides a guideline to reconsider LAM, linear scleroderma, and APP as part of a clinical and histopathological spectrum, concurring in the manifestations of hyperpigmentation, the Blaschko lines pattern, the presence of thickening of the collagen bundles, or the compacting of the dermis, where LAM would be the mildest form, APP an intermediate form, and scleroderma as the significant clinical expression due to tissue damage.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author has this document.

Conflicts of interest

The authors declare no conflict of interest.

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