Case Report

Nodular elastosis with cysts and comedones, a severe unilateral presentation

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Abstract
Case presentation. A 73-year-old patient with a history of chronic occupational exposure to solar radiation and smoking for 54 years; he consulted for a unilateral dermatosis on the face with a yellowish multicystic tumor appearance, accompanied by open and circumscribed comedones, involving zygomatic, infraorbital, and left cheek regions, of 25 years of evolution, with little pruriginosity and mild body dysmorphic symptoms.**Treatment.** Clinical and dermatoscopic findings and a skin biopsy made the diagnosis. Management was performed on an outpatient basis, including education regarding the disease, as well as measures to avoid, abandon, or reduce aggravating factors, in addition, permanent sun protection and a combination of alpha hydroxy acids with topical retinoids were prescribed. **Outcome.** This dermatosis is characterized by a chronic, non-resolving course with no tendency to malignant transformation. The main approaches are surgical and pharmacological; the former for severe cases, as the one described in this report. In the Plastic Surgery Department, surgical excision of the lesions was performed in two stages, with ambulatory follow-up in dermatology due to the high incidence rate of malignant neoplasms, inherent in skin with chronic actinic damage.
Keywords
Favre-Racouchot Syndrome, Nodular Elastoidosis, Photosensitivity Disorders.

Resumen
Presentación del caso. Paciente de 73 años con antecedentes de exposición crónica ocupacional a radiación solar y tabaquismo durante 54 años; quien acudió a consulta por una dermatosis unilateral en el rostro, con aspecto tumoral multiquístico amarillento, acompañada de comedones abiertos y de tipo circunscrito, que comprometía regiones cigomática, infraorbitaria y mejilla izquierda. La lesión tenía 25 años de evolución, era pruriginosa y presentaba síntomas dismórficos corporales leves. **Intervención terapéutica.** El diagnóstico se estableció a partir de los hallazgos clínicos, dermatoscópicos y una biopsia cutánea. El manejo se realizó en modalidad ambulatoria, incluyendo educación sobre la enfermedad, así como medidas para evitar, abandonar o reducir factores agravantes. Además, se prescribió fotoprotección solar permanente y el uso de una combinación de alfa hidroxiácidos y retinoides tópicos. **Evolución clínica.** Esta dermatosis se caracteriza por un curso crónico, no resolutivo y sin tendencia a la transformación maligna. Los abordajes principales son el quirúrgico y el farmacológico; siendo el primero para los casos severos, como el descrito en este reporte. En el Servicio de Cirugía Plástica se realizó la extirpación quirúrgica de las lesiones en dos tiempos, con seguimiento ambulatorio en dermatología debido a la elevada tasa de incidencia de neoplasias malignas asociado al daño actínico crónico en la piel.

Palabras clave
Síndrome de Favre-Racouchot, Elastoidosis Nodular, Dermatosis Ocupacional.

Introduction

Dermatoheliosis refers to skin changes induced by damage due to chronic exposure to ultraviolet radiation (UVR) from the sun. The clinical manifestations depend on the total cumulative lifetime UVR dose and genetic predispositions.[i] The most significant manifestations include skin cancer, premalignant or pigmented lesions, wrinkles, uneven skin tone, rhomboid skin of the nape of the neck, purpura and actinic elastosis (AE). The

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latter is used in two contexts: to describe a set of clinical findings and, in histopathology, to denote alterations in the elastic fibers.

Coincidentally, the clinical manifestations are a consequence of histopathological alterations.[i,ii]

In the clinical aspect, AD encompasses manifestations such as: skin thickening, xerosis, yellowish or "citrine skin", intensely demarcated wrinkles and comedones. In the histopathological context, AD involves chronic sun damage to the elastic and collagenous tissue of the dermis, culminating in its degeneration and loss of function.[i,ii]

AD can present syndromically, with a characteristic topography and morphology, as in the case of nodular elastosis with cysts and comedones, Favre-Racouchot syndrome (FRS), which affects the periorbital and malar region, the form known as actinic comedonal plaque and elastic nodules, which are usually located on the ear.[i,iii]

FRS predominates in Caucasian adults aged 40 to 60 years, mainly in men with the presence of predisposing factors. The incidence increases with age, and the worldwide prevalence ranges from 1. 5 % to 6 %..[ii,iii] The most commonly identified predisposing factors are chronic sun exposure, whether occupational or recreational, and smoking.[ii,iv]

FRS manifests clinically as comedones and cystic lesions secondary to cutaneous elastosis, with bilateral and symmetrical distribution. In most cases, it is associated with actinic keratosis and rhomboidal cutis of the nape of the neck, both entities produced by photodamage.[v]

Diagnosis is made by identifying typical lesions accentuated in areas of skin with chronic actinic damage, evaluated during physical examination, and correlating these findings with the clinical history and the presence of risk factors. Dermoscopic findings have been described as diagnostic support; however, there is no official consensus. Some authors describe SFR as follows: perifollicular black central rings against a yellowish white background (open comedo); small homogeneous yellowish brown and circular areas surrounding keratin plugs (closed comedo); areas without intersectional structures of brown to ocher hue, or even skin-colored; concomitant findings of actinic damage (areas without structure or yellowish halos); and branching or arborizing linear vessels (corresponding to telangiectasias).[vi,vii]

In case of diagnostic doubt in atypical presentations, the diagnosis is confirmed by histopathological analysis. Characteristic findings in the dermis consist of the presence of thick, "matted" elastic fibers, the homogeneous eosinophilic or blue-grayish accumulation of amorphous material derived from the degeneration of these fibers, and the dilatation of hair follicles and formation of small epidermal cysts, containing keratinocyte detritus.[viii,ix]

Case presentation

A 73-year-old man came to the dermatology office due to a localized dermatosis on the head, affecting the malar region, infraorbital and left cheek (unilateral and asymmetrical). A circumscribed yellowish lesion was observed, with a tumor-like appearance and poorly defined borders; composed of multiple nodulocystic structures, telangiectasias, open comedones and some hairy stalks. The lesion measured 7 x 6 cm (Figure 1).

In the rest of the skin and adnexa, chronic actinic damage was evident on the face and neck (Figure 1).

During the interrogation, the patient reported having had the described lesion for 25 years, with discrete enlargement in the last three months and mild body dysmorphic symptoms. He denied previous dermatologic consultation or treatment.

Medical-social history: chronic arterial hypertension, chronic occupational actinic exposure (due to driving) and smoking for 54 years (without specifically remembering the number of packs per year). There was no documented family history of first degree of consanguinity with the same condition.

Dermoscopy was performed as diagnostic support. Panoramic view showed an asymmetric lesion of multilobulated appearance with multiple white (suggestive of keratin in the superficial dermis), and yellow (suggestive of actinic elastosis) structureless areas. Irregular linear and arborizing vessels (corresponding to telangiectasias), intersectional pink structureless areas, follicular openings with horny plugs (open and closed comedones) and some hair stalks (Figures 2 and 3).

A clinical-dermatoscopic correlation was performed to identify features suggestive of inflammatory comedogenic nevus, giant pilomatrixoma, and squamous cell carcinoma. Given the possibility of malignancy detected by application of the dermoscopic algorithm (prediction without pigment),[x] an elliptical incisional specimen (2. 5 × 2 cm) was taken for biopsy.

As initial therapeutic measures, sun protection recommendations were given while awaiting the histopathological report, in order to establish the specific diagnosis and treatment.

Figure 1. Macroscopic appearance of the dermatosis. A. Frontal view, skin with actinic damage: thickened skin, wrinkles with exaggerated demarcation, "citrine" hue, desquamation, and solar lentigines with seborrheic keratosis on the neck. B. Left lateral view, showing the circumscribed tumor-like lesion. C. Close-up of Figure 1B. Note the nodulocystic and lobulated morphology, hairy stalks, telangiectasias, and some open comedones.

At ten weeks, the histopathologic report revealed multiple cystically dilated hair follicles with horny plugs; epidermal invaginations filled with orthokeratotic keratin in lamellar form and remnants of hair shafts. Some cysts showed superficial opening into the epidermis. In the dermis there was marked AD, edema and discrete chronic inflammatory infiltrate. No malignancy was evident (Figure 4 and 5). Based on these findings, the specific diagnosis was established and the corresponding management proceeded.

Treatment

The diagnosis of the presented case was established by correlation of clinical, dermatoscopic and histopathologic findings. Conservative treatment was chosen due to the non-neoplastic nature of the disease and the patient's lack of interest in cosmetic refinement. The main approaches included providing clear information about the benign nature of the lesion, topical pharmacologic therapy, a joint approach with plastic surgery, and long-term outpatient follow-up by dermatology to monitor therapeutic response and early detection of malignancies associated with actinic damage.

With regard to patient education, emphasis was placed on the avoidance, reduction or abandonment of associated factors. The importance of sun photoprotection and smoking cessation was reinforced, a habit that the patient had managed to abandon a year before the consultation.

Topical treatment consisted of broad-spectrum sun photoprotection in cream, with a sun protection factor ≥ 50, applied every three hours to photoexposed areas. In addition, 0. 05 % retinoic acid gel was prescribed as a keratolytic and keratinization regulator, applied in a thin layer at night on the face. The application of 12 % glycolic acid cream in the morning on the lesion area was also indicated. All medications were indicated for continuous use, with

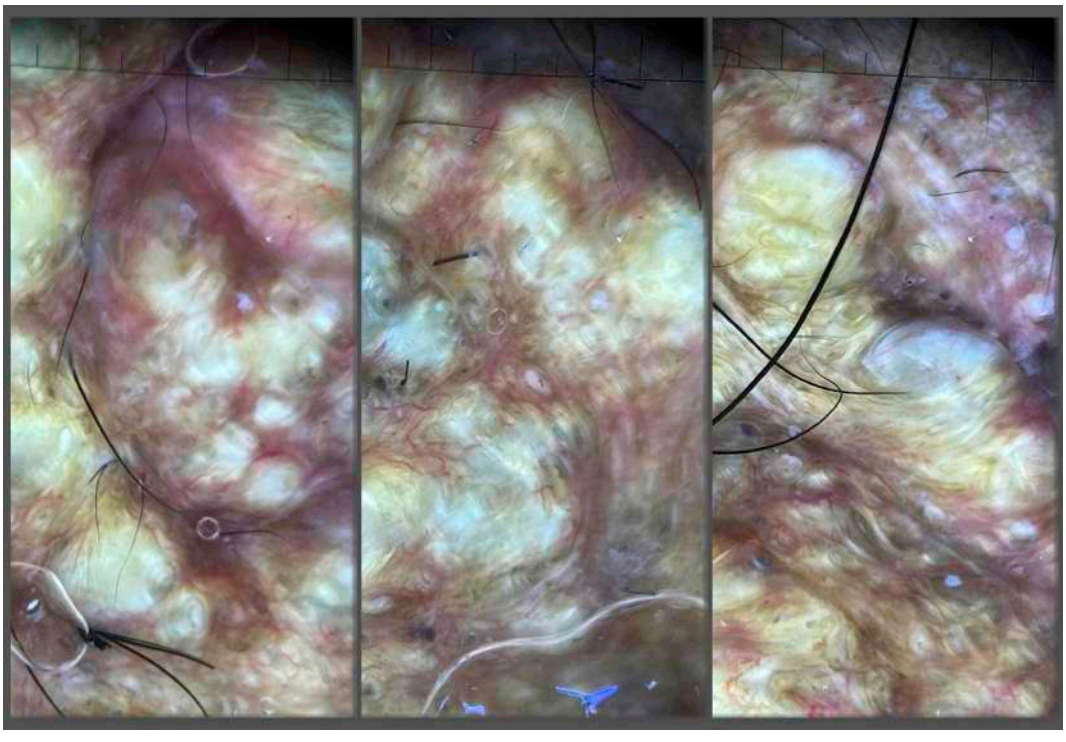

Figure 2. Dermatoscopy with DermLite handyscope® 10x device. Central black rings and/or light to dark brown homogeneous areas surrounding hair follicles and/or horny plugs against a white background (green arrows), yellowish brown circular structureless homogeneous areas surrounding keratin plugs (blue arrows), brown to pink intersectional areas containing vascular structures dividing white and yellowish structureless areas (blue stars) giving a lobulated appearance and irregular linear arborizing blood vessels (red arrows).

the purpose of limiting as much as possible the extension of the lesion and causing some degree of reduction of the lesion. In severe cases of extensive tumor appearance, a surgical approach is recommended as the ideal treatment. For this reason, the patient was referred to the plastic surgery department, where long-term outpatient management was recommended.

Outcome

Treatment was conservative through topical therapy with keratolytics in order to reduce and limit the extension of the lesion. Continuous outpatient monitoring of the patient was the main objective in the evolution for the prevention of new actinic damage and early detection of malignant neoplasms, frequent in skin with chronic photoexposure. At the date of completion of the case report, two outpatient dermatology controls were performed, where a discrete reduction in the size of the lesion was evidenced and no malignant lesions were observed.

Clinical diagnosis

The presumptive diagnosis was made by correlation of risk factors such as the work as a cab driver for several years (greater photoexposure of the left hemiface), history of smoking and dermoscopic evaluation. By integrating the patient's history with the clinical findings and the skin biopsy, the definitive diagnosis of severe unilateral Favre-Racouchot syndrome was established, due to the tumor appearance.

Discussion

The etiopathogenesis of FFR is not known exactly, but involves changes produced by cumulative damage from UVR A and B, smoking, corticosteroid use, radiotherapy and infrared radiation. These factors induce phenomena such as reduction of tensile strength in the dermis,[ii,iv] due to degeneration of the elastic tissue, which causes loss of support and, as an infundibular distension in the pilosebaceous unit. This triggers keratinization of the hair follicle, eventual formation of extensive comedones and keratin-filled epidermal invaginations, and skin atrophy with reduction of collagen fibers.

As a consequence of permanent elastic fiber degeneration, the treatment of RMSF is difficult, invasive and with a risk of recurrences. This condition, like other dermatoses, can generate variable egodystonic self-perception among patients. Furthermore, it is important to note that these lesions do not present an increased risk of malignancy, ulceration or infections. Since there is no offi-

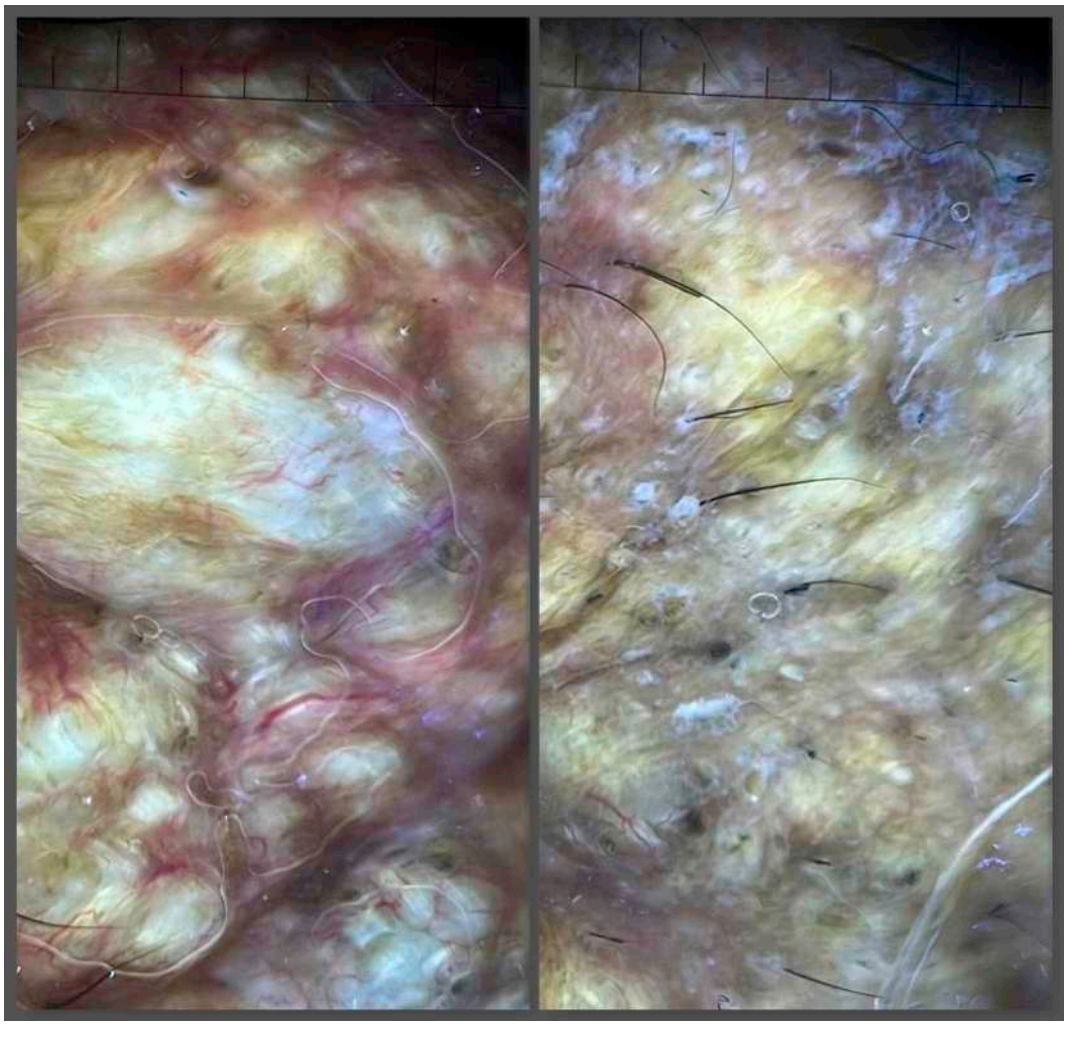

Figure 3. Dermatoscopy with DermLite handyscope® 10x device. Central black rings and/or homogeneous light to dark brown areas surrounding hair follicles and/or horny plugs against a white background (green arrows), homogeneous areas without circular, yellowish-brown structures surrounding keratin plugs (blue arrows), irregular linear and arborizing blood vessels (red arrows), and yellowish areas without structure (blue stars).

cial consensus for their treatment, the most important factor to consider is the patient's concern, which allows an individualized therapeutic approach to be offered.[ii,xi]

Current evidence recommends preventive measures that include abandoning, reducing or avoiding predisposing or aggravating risk factors, as well as the permanent use of broad-spectrum topical photoprotectors. In addition, emollients and alpha hydroxy acids are suggested to improve skin appearance as aesthetic adjunctive measures.[i,ii,iv]

Therapeutic modalities are divided into pharmacological (topical and systemic) and surgical (minor or major).[xi] In topical pharmacological therapies, retinoids are the most commonly used options; however, there are other documented but not standardized alternatives such as chemical dermoexfoliation with salicylic acid, among others.[xi,xiii]

In systemic pharmacological treatment, retinoids such as isotretinoin are used.[ii] Minor surgical treatment for small lesions involves traditional excision, dermabrasion, curettage and manual removal of comedones, pulsed carbon dioxide laser and plasma exeresis.[ii,xi]

While, for major surgical treatment it is indicated in cases of extensive and/or numerous plaques and nodules, with functional compromise, cosmetic discomfort and suspicion of malignancy.[ii,xi,xiv] In addition, decortication plus electro curettage[xiv] or plastic surgery with displacement autoplasty of the neighborhood has been used.[xv]

The most satisfactory results are the combined modalities. Carbon dioxide laser in combination with topical retinoids has shown the best results.[ii,xi,xiv]

A typical FFS presents with thick yellowish plaques accentuated on actinically damaged skin, covered with comedones (punctiform, waxy and non-inflammatory), open or closed, and cystic dilatations filled with keratin. Its distribution is predominantly bilateral, symmetrical and localized to the head, with a predilection for the periorbital skin (adjacent to the lateral ridges)

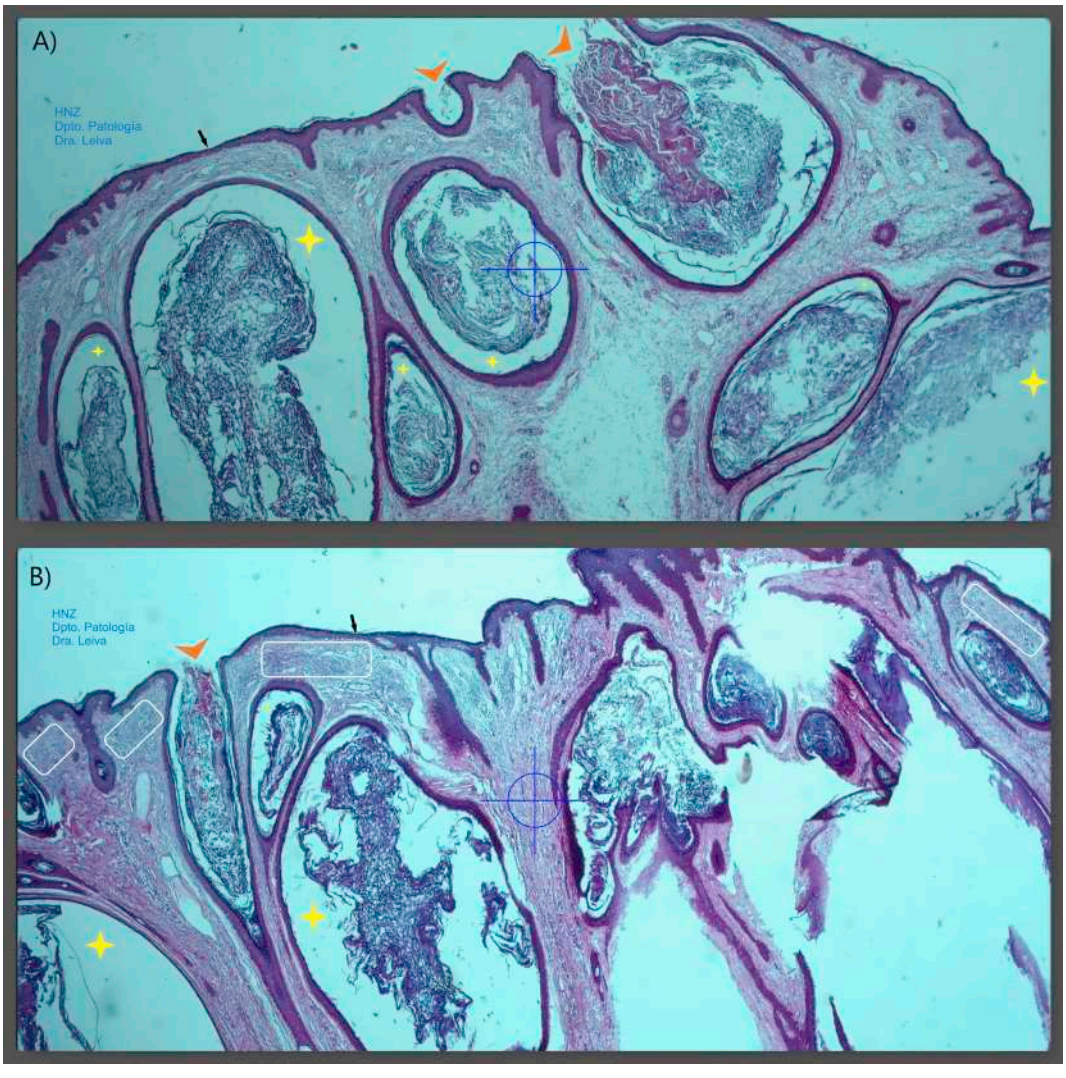

Figura 4. 10X hematoxylin and eosin stain of skin biopsy. A and B show epidermal atrophy (black arrows), widely dilated follicular openings filled with lamellar keratin (yellow stars); epidermal invaginations, some of which open into the epidermis (orange arrowheads). AE corresponds to blue-gray material in the dermis (white squares).

Figura 5. 20X hematoxylin and eosin stain of skin biopsy (enlargement of Figure 4-B). In the dermis, an amorphous blue-gray material and a thickening of elastic fibers, corresponding to AE (black squares), can be identified..

and the malar eminences. In sporadic cases it has been observed in the frontal or temporal regions.[i,iv]

However, there are documented unilateral cases. In a systematic review only fourteen cases were reported.[xi] These are attributed to chronic asymmetric exposure to light radiation (UV or infrared) in occupational settings, such as drivers, miners, secretaries. Other cases were associated with radiotherapy, while in the rest the cause was not determined.

There are reports of cases with atypical unilateral extrafacial morphology. Siragusa M *et al.* describe an 80-year-old man with plaque SFR affecting the left shoulder and scapular area. This case was attributed to chronic asymmetric photoexposure and the occupational factor of a bricklayer, in which there was evidence of continuous asymmetric unilateral material loading and pressure.[xvi] In another case, Ibad S *et al.* describe a 60-year-old man with SFR on scalp skin, associated with the profession of a farmer since childhood and chronic sun exposure. In addition, they describe other lesions linked to actinic damage.[xvii] In both cases there are no details on the treatment or evolution of the patients.

Cases with unilateral atypical facial morphology have been described. Mavilia L *et al.*, describe two male smokers, 57 and 65 years old, with plaque SFR, affecting the left malar region, attributing the unilateral presentation to chronic occupational exposure to UVR.

Diagnosis was made by clinical evaluation and histopathology, and treatment consisted of fractionated carbon dioxide laser and manual extraction of comedones. However, the evolution of the patients was not reported.[xviii] On the other hand, Sobjanek M *et al.* describe a 71-year-old man, with a history of having been a bricklayer for several years, who presented FFS in plaque on the right malar region. They highlight other findings of concomitant actinic damage. The diagnosis was made on the basis of clinical and histopathologic findings, although neither the treatment nor the patient's evolution was described.[xix]

There are other types of cases that are considered severe due to their tumor appearance, either bilateral or unilateral. Enriquez MJ *et al.* describe a 63-year-old man with bilateral lesions in malar regions classified as severe, with a history of smoking since the age of 16. Due to the extent of the lesions and the inability of dermatology to resolve them, the patient was referred to surgery, where a complete excision was performed, with the aim of improving aesthetics. The diagnosis was made by histopathology, and the evolution was favorable.[xx] Pizzati A *et al.* report a 63-year-old man with left malar SFR with a tumor-like appearance. Among the findings, they highlight a history of smoking for 40 years and suggest that the etiology could be related to chronic exposure to radiation from incandescent light lamps used in his work. Treatment consisted of carbon dioxide laser, with complete resolution of the lesions.[xxi] Khouna A *et al.* described a 63-year-old man, a smoker with chronic sun exposure, with a tumor-like SFR on the right nasal ala, highlighting the concomitant presence of other lesions due to actinic damage, although they do not describe the treatment or the patient's evolution.[v]

The authors cited in previous paragraphs agree that the genesis of unilateral lesions in FFS is a combination of chronic asymmetric exposure to light radiation (predominantly in the left malar region), genetic predisposition, association with smoking, male patients and occupational factors.

In this case, as in the literature, the presentation of skin lesions is observed with a history of risk factors such as chronic exposure to UVR asymmetrically, male patient, occupational risk (driver, with greater exposure of the left hemiface to sunlight), lesion developed predominantly in the left malar region and chronic smoking habit. The clinical presentation of SFR was unilateral and atypical, presenting severely. In addition, in this patient, the presence of actinic damage lesions concomitant to RMS was identified, and the diagnosis was made mainly by histopathology due to the atypical presentation. This known relationship between risk factors may correspond to an opportunity to generate recommendations of practical utility, such as strengthening primary prevention measures, mainly sun photoprotection, in at-risk occupations or in patients with cigarette addiction. Likewise, secondary prevention, through continuous follow-up for the treatment of premalignant lesions and early detection of malignant lesions, is essential, given that these are frequently associated with SFR due to chronic actinic damage.

It is important to highlight that conservative therapeutic approaches are able to limit the process and avoid extensive surgical interventions, especially in patients who do not seek cosmetic refinement.[xx,xxii,xxiii]

Therefore, and because of the atypical and rare form of the presentation, we consider this case report to be of scientific relevance.

At the time of this case report, the first surgical intervention was pending, although it was classified as low priority because it did not present malignant nature or functional

alterations, being considered an aesthetic problem. This limited the evaluation of significant progress in the follow-ups performed, which did not show important changes, which was expected due to the severity of the condition and the limited response to topical therapy.

Ethical Aspects

This case is based on the Helsinki principles, which guarantee the confidentiality of the patient who authorizes publication of the clinical case and images through informed consent. Financing There are no sources of financing for the preparation of this publication.

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