Clinical, Cosmetic and Investigational Dermatology

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CASE REPORT

Cutis verticis gyrata in a Patient with Familial presenile sebaceous hyperplasia Presenting as "Leonine facies": A Rare Case

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Abstract: Cutis verticis gyrata (CVG) is a rare dermatological disorder characterized by thickened, gyriform folds and ridges of the skin, predominantly affecting the head and face. Based on etiology, it is classified into primary and secondary forms. Familial presenile sebaceous hyperplasia (FPSH), a rare variant of sebaceous hyperplasia, follows an autosomal dominant inheritance pattern with incomplete penetrance and variable severity among family members. We report a case of a 60-year-old male who presented to the Dermatology and Venereology Department, Hasan Sadikin General Hospital, Bandung, with thick plaques and deep furrows on the forehead, giving a "leonine facies" appearance. These lesions first appeared 25 years ago and progressively increased in number and spread to the neck and chest. Similar symptoms were observed in his twin brother and son. Investigations including skin slit smears, blood tests, serum biochemistry, imaging, and testosterone levels were unremarkable. Histopathological examination of facial papules and nodules showed enlarged sebaceous glands in the dermis, supporting the diagnosis of sebaceous hyperplasia. The patient was diagnosed with FPSH associated with CVG. Although rare, both conditions may be linked to systemic abnormalities, highlighting the importance of comprehensive evaluation. There is no definitive treatment for FPSH or CVG; current management is mainly cosmetic. Isotretinoin, with its sebocyte-suppressing effect, may reduce gland size but has a high recurrence rate upon discontinuation. Early recognition and a multidisciplinary approach are crucial to prevent further progression, particularly in older patients.

Keywords: cutis verticis gyrata, familial presenile sebaceous hyperplasia, leonine facies

Introduction

Cutis verticis gyrata (CVG) is a rare dermatological condition primarily involving the head and face, characterized by thickened, gyriform folds and ridges.[1] Initially described by Unna in 1907, CVG is now classified based on its etiology as either primary or secondary.[2] Male patients are disproportionately affected.[3,4] The condition is further subdivided into primary essential, primary nonessential, and secondary forms.[5] Primary CVG often coexists with neuropsychiatric disorders,[6] whereas secondary CVG arises from systemic diseases, medications, or inflammatory and neoplastic processes, including sebaceous hyperplasia (SH).[7]

Sebaceous hyperplasia is a common, benign proliferation of sebaceous glands, typically observed on the faces of middle-aged to elderly adults.[8,9] Clinically, it presents as small (<3 mm), yellowish papules, predominantly on the forehead.[9,10] This condition is driven by intrinsic (genetic or chronological) aging and extrinsic factors.[11,12] In some cases, sebaceous hyperplasia may occur in association with genetic syndromes, such as Muir-Torre syndrome, or in familial forms.[12]

Familial presenile sebaceous hyperplasia (FPSH) is a rare variant of SH that manifests during puberty and progressively worsens with age.[13,14] Dupre et al[15] in 1980 described its key clinical features: onset during or shortly after puberty, prominent sebaceous hyperplasia localized to the face, neck, and upper thorax without periorificial involvement, absence of acneiform lesions, slow progression, resistance to conventional acne treatments, and histopathological

Received: 8 March 2025
Accepted: 31 May 2025
Published: 3 June 2025

findings of markedly enlarged sebaceous glands with minimal inflammation. Histopathologically, FPSH typically shows hypertrophic sebaceous glands with numerous lobules surrounding a dilated sebaceous duct.[13]

The treatment approach for CVG ranges from conservative management to surgical intervention, with the latter being the most effective for addressing the pronounced skin folds.[1,2] Conversely, FPSH is a benign condition that requires treatment for cosmetic purposes only.[9] Treatment modalities include electrosurgery, laser therapy, photodynamic therapy, and, in severe cases, isotretinoin, though recurrence is common post-treatment.[13,16,17]

Herein, we present a rare and unique case of CVG associated with FPSH, manifesting as "leonine facies", to highlight the clinical and histopathological features and the challenges in its management.

Case Illustration

A 60-year-old male presented with thick plaque on the face with deep furrows, especially on the forehead, which looked like "leonine facies". Since 25 years ago, he has complained of numerous yellowish papules on his face. The lesions gradually increased in number and extended to the neck and chest wall. The patient complained about the thickness and protrusion of the skin on their left cheek and consequently requested the removal of the skin disorder. He was from West Java, Indonesia, and a miner by occupation. He did not have any medical problems or take any medication regularly. He was the third of 9 siblings, of whom 3 (2 males and 1 female) had similar skin disorders after the age of 30 (Figure 1). His twin brother had the same lesions as him (Figure 2). He had five children and three of whom, two sons and one daughter, reported similar skin changes, but less severe (Figure 3). There was no significant medical problem or consanguineous marriage in his family.

On physical examination, a marked thickening and coarsening of the facial features, with accentuation of skin furrows, were found and it gave the appearance of leonine face. There was skin protruding on his left cheek, measuring 3 cm long, 1 cm wide and 1 cm thick. The facial skin felt greasy to touch (Figure 2). His periorbital, perinasal, perioral, and periauricular areas were spared. Hair, nails, and teeth were normal. No comedones were present. No telangiectasia associated with rosacea was noted.

A facial papule and nodule biopsy showed multiple-enlarged mature sebaceous glands in the dermis, consistent with the possible clinical diagnoses of SH (Figure 4). The results of complete blood count, serum biochemistry, chest radiography, computed tomography scans, and testosterone levels were normal.

A shave excision was performed on the lesion located on the left cheek of the patient due to perceived interference (Figure 5). Presently, the patient declines further therapy as they consider other skin abnormalities to be non-disruptive.

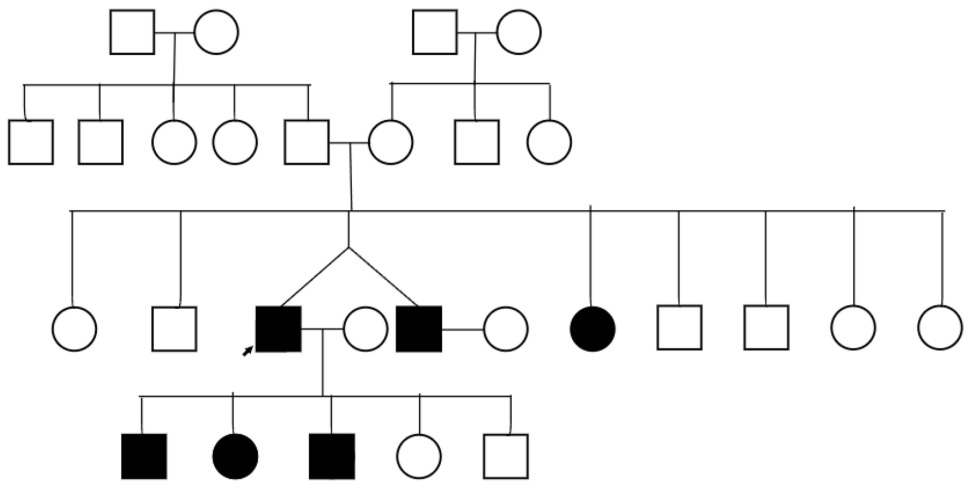

Figure 1 Family pedigree of the patient.

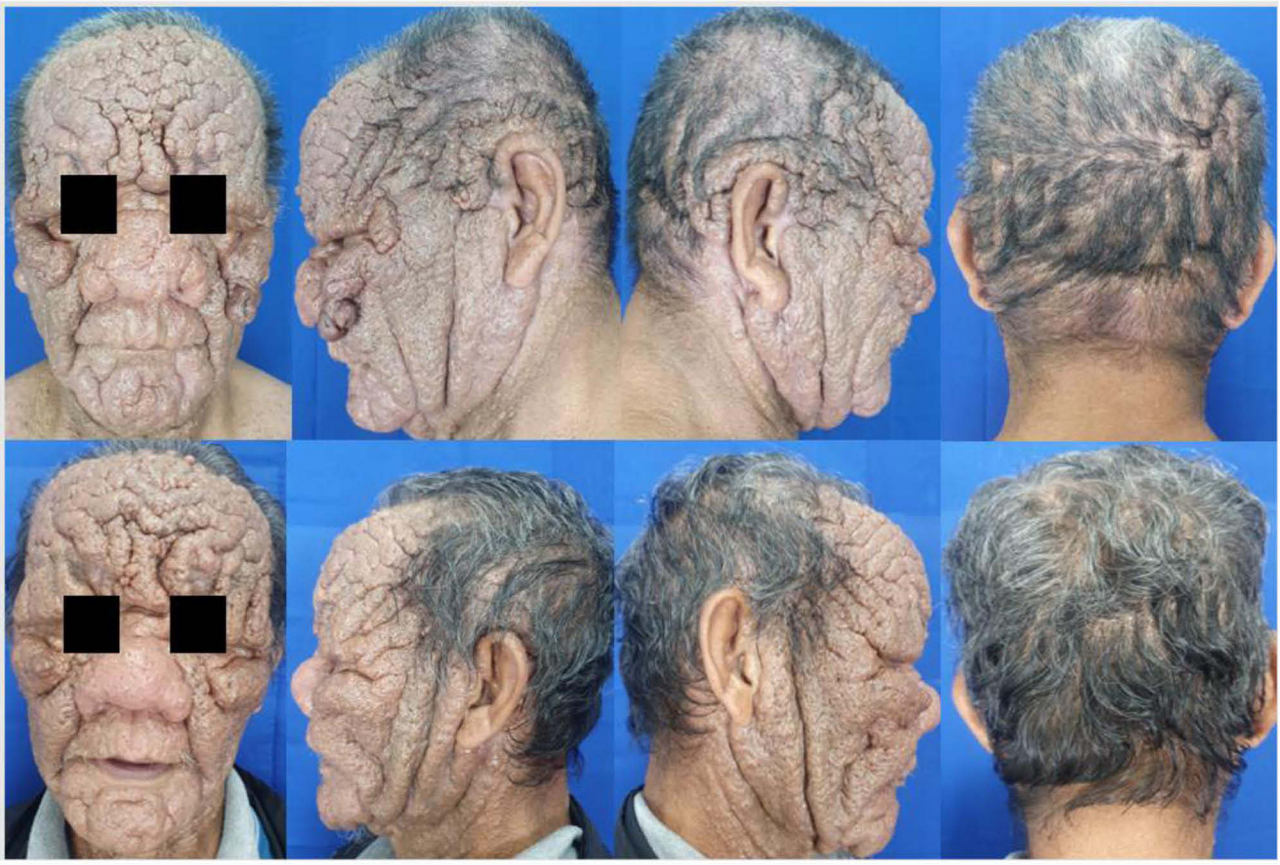

Figure 2 Multiple diffuse, greasy, dome-shaped umbilicated papules on his face sparing the periorificial and nasolabial areas of FPSH with CVG patient (upper) and his twin brother (lower).

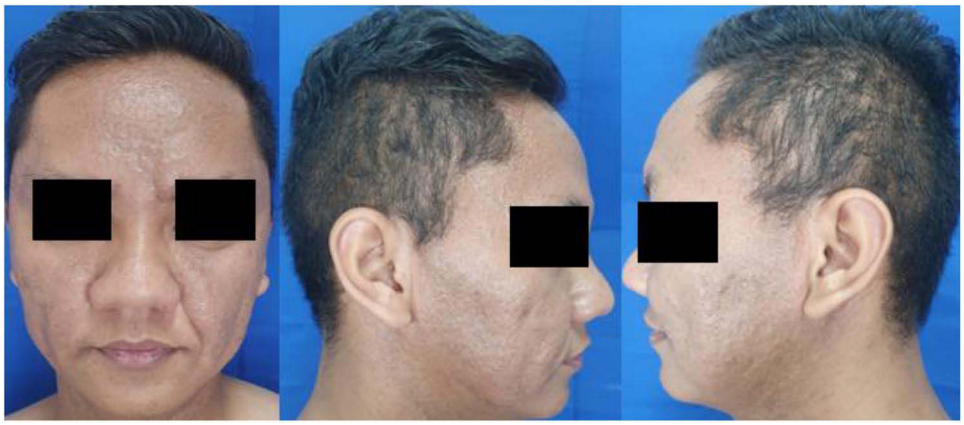

Figure 3 Clinical manifestation of his son.

https://doi.org/10.2147/CCID.S519136

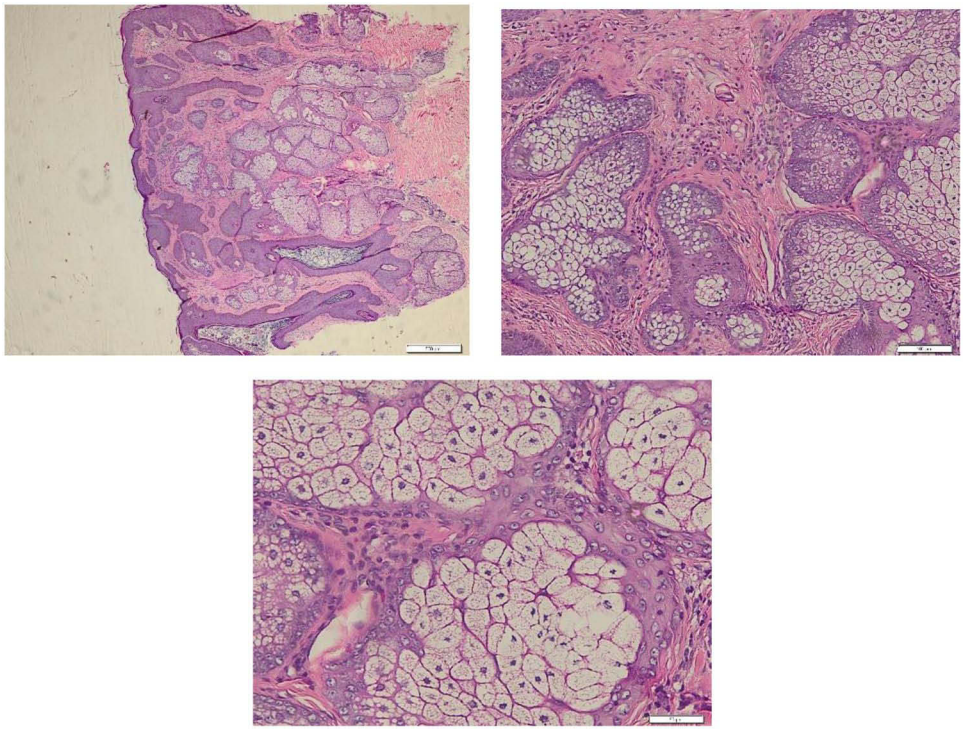

Figure 4 Histopathology results confirmed the diagnosis of SH. Histopathology showed differentiated enlarged mature sebaceous glands in the dermis and one or two layers of undifferentiated basaloid cells at the periphery of sebaceous lobules.

Figure 5 A shave excision was performed on the lesion located on the left cheek. Here is the appearance two weeks post-procedure.

Discussion

Cutis verticis gyrata (CVG) is a rare dermatological disorder characterized by thick, undulating folds of skin resembling cerebral convolutions. These folds are separated by deep grooves and predominantly affect the scalp and face.[18] Reported measurements range from 2 to 30 folds, with each fold being 0.5–2 cm wide and furrows approximately 1 cm deep.[3,19] The condition is more prevalent in males, with an incidence of 1 in 100,000, compared to 0.026 in 100,000 for females. Most patients have lesions on the scalp, and only a few cases with forehead lesions have been described.[5] Some previous reported cases are summarized in Table 1. Our patient exhibits CVG lesions on the scalp and face.

Cutis verticis gyrata can present as a rare primary form, arising independently without any underlying disease, or as a secondary form associated with systemic conditions.[4,19] The etiology of CVG is complex, involving various cell types that contribute to its distinctive convoluted skin appearance, which may result from inflammatory processes,

Table 1 Summary of Previous Reported Case

Author	Case						Treatment
	Number of Cases	Age / Gender	Familial Similar Skin Lesions	Age of Onset (Years)	Lesion	Supporting Examinations	
Tan and Ergen (2006)[4]	One case of CVG	51-year-old female	No family history of similar disorder	30	Many folds located diffusely on the vertex, occipital and biparietal areas of the scalp that could not be obliterated by pressure.	• Cranial CT showed well-demonstrated deep skin folds of the scalp. • Normal laboratory findings. • Histopathological showed epidermal thinning and conjunction of the epi-dermal rates in a hyperkeratotic epithelium	No surgical intervention was considered due to no cosmetic or functional complaint
Radwanski et al (2008)[6]	One case of CVG	30-year-old male	Grandfather	18	Severe large skin folds of the scalp, in the vertical and horizontal axis.	• Normal laboratory findings.	Surgery was done with limited incision to one side of the demarcation, down to the galea, with supragaleal undermining of the remaining demarcated area. The recovery was uneventful and the patient was discharged the next day.
Liu et al (2016)[13]	Two cases of presenile diffuse familial sebaceous hyperplasia (PDFSH)	39-year-old female	Mother and son	36	Diffuse skin-colored erythematous umbilicated papules on cheeks, forehead, chin and neck with seborrhea and inflammatory acne.	A skin biopsy on the left cheek revealed sebaceous hyperplasia with keratin plugged dilated pilosebaceous ducts and chronic granulomatous inflammation over the upper dermis	Underwent 7-month treatment with a cumulative isotretinoin dose of 41mg/kg and reported no recurrence in a 2-year follow up
		29-year-old female	Grandmother	27	Progressive facial lesions with hot sensations. Multiple diffuse yellowish to erythematous dome-shaped umbilicated papules on face, except for the periorificial area and nasolabial folds.	Histopathology showed typical sebaceous hyperplasia.	No recurrence for 19 months after a cumulative isotretinoin dose of 64 mg/kg.
Trakoolwannachai and Kootiratrakarn (2016)[20]	One case of DFSH	44-year-old female	First and second younger sisters	29	• Numerous yellowish dome-shaped papules with central umbilication, con-fluent to thick plaques with deep fur-rows on the face, except periorbital and perioral areas, neck, and chest. • Lesions on face turned to thick plaques with deep furrow especially on forehead similar to leonine faces.	• Dermoscopic examination showed multiple yellow white globules and thin non-arborizing blood vessels • Histopathological examination showed multiple enlarged mature sebaceous gland in dermis	Improved symptoms after 12 weeks of using isotretinoind 20mg/day (0.4mgkg/day).
Gupta et al (2016)[21]	One case of sebaceous hyperplasia and sebaceous adenoma	64-year-old male	No familial history	58	• Marked thickening and coarsening of the facial features with accentuation of skin furrows, similar to leonine faces • Skin-coloured to yellowish umbilicated papules, clustered on the nasal tip, cheeks, and forehead along with a few skin-coloured to erythematous, lobu-lated nodules.	• Skin biopsy showed features consis-tent with the possible clinical diag-noses of sebaceous hyperplasia and sebaceous adenoma • Normal laboratory and radiological findings	Flattening of the lesions in the next 3 months with oral 20mg isotretinoin daily.

hamartomatous growths, or neoplastic proliferations. CVG is classified into primary (idiopathic) and secondary types, with the secondary type often described as "scalp diseases resembling CVG".[22] The primary essential form occurs without association with other conditions, while the primary nonessential form is linked to mental disabilities, neurological disorders, and ophthalmic abnormalities.[6] Secondary CVG, on the other hand, can be triggered by factors such as inflammatory dermatoses, systemic illnesses, infiltrative disorders, or cutaneous neoplasms.[22] There was no systemic disease involvement in this patient.

Due to the unusual manifestation of facial involvement observed in our patient, it was crucial to rule out other potential causes of "leonine facies", such as lepromatous leprosy, mycosis fungoides, leishmaniasis, and amyloidosis. Histopathological features in primary CVG cases are diverse, ranging from normal histology to notable findings such as connective tissue thickening, hypertrophy, or hyperplasia of hair follicles and sebaceous glands.[22,23] Biopsy specimen that was consistent with SH. In this case, CVG occurs secondary to SH, especially FPSH.

Sebaceous glands are holocrine glands composed of acini attached to a common excretory duct.[8] These glands may exist independently or as part of the pilosebaceous unit, which includes the hair shaft, hair follicle, and arrector pili muscle.[24] These units are distributed across most areas of the body, with the exception of the lower lip, soles, palms, and the tops of the feet.[25] Pilosebaceous units are predominantly on the face, upper neck, and chest. Sebaceous glands secrete sebum, a combination of wax esters, squalene, cholesterol esters, and triglycerides, through the sebaceous duct into the hair follicle, which helps keep the skin and hair supple.[26]

Sebaceous hyperplasia is a prevalent skin condition observed primarily in middle-aged to elderly individuals and is often misdiagnosed as basal cell carcinoma.[8,9] Most lesions are 2–4 mm in diameter, with a central umbilication representing the site of a ductal opening.[9] Telangiectasia may also be present, with some lesions resembling basal cell carcinomas.[27] These lesions are generally asymptomatic and present as soft, yellow, dome-shaped papules, commonly located on the forehead, cheeks, and nose.[8,9]

Familial presenile sebaceous hyperplasia (FPSH) is a rare subtype of sebaceous hyperplasia.[14] The initial case was reported by Dupre[15] in 1980. Subsequently, there have been limited instances of FPSH documented in the literature, comprising only 13 patients of diverse ethnic backgrounds in published studies since 1980.[13] Previous reports suggest that FPSH is an autosomal dominant inheritance with incomplete penetrance.[14] There is no sex predilection.[20] This disease occurs at an earlier age than SH in the general population.[13,20] It appears typically during puberty or soon afterward and slowly progresses.[28] The diffuse expanse and high function of the sebaceous gland lead to facial irregularity and excess sebum.[13] These lesions are distributed diffusely on the face and linearly on the neck and upper thorax. The periorificial regions, including the periorbital, perinasal, preauricular, and perioral areas, are characteristically unaffected.[13] The pedigree suggests autosomal dominant inheritance with incomplete penetrance.[13,14] It appears equally in both sexes, but lesions in the affected female family members seem to be less severe.[13] Family members must have the same presentation but vary in severity.[13,14] The patient in this case report is a 60-year-old male who presents with similar complaints to his male twin and their offspring.

The clinical differential diagnosis of FPSH encompasses rosacea, micropapular sarcoidosis, multiple sebaceous adenomas, multiple trichoepitheliomas, multiple follicular fibromas, and the angiofibromas of tuberous sclerosis.[28,29] The patient's complaints in this case report have been experienced since the age of 30 and have been progressively worsening up to the present time.

Histopathology of FPSH is identical to its senile counterpart, characterized by voluminous sebaceous glands with mature sebaceous lobules that empty into a dilated central sebaceous duct.[13] Histopathologically, SH should be differentiated from nevus sebaceous, sebaceous adenoma, basal cell carcinoma with sebaceous differentiation, and sebaceous carcinoma.[13,21] Biopsy from a facial papule and nodule in this patient showed multiple enlarged mature sebaceous glands in the dermis and no signs of malignancy.

The precise etiology of FPSH remains unclear.[13] Several earlier literature suggest that this is associated with testosterone levels. However, serum levels of circulating androgens (testosterone, dehydroepiandrosterone, and androstenedione) and urinary 17-ketosteroids are normal in all reported patients, suggesting that the disease is not caused by overproduction of testosterone but possibly by congenital and abnormal hyperresponsiveness of the sebaceous gland itself or a hormonal receptor defect.[14] A few sporadic cases of FPSH, which had the same clinical characteristics of

diffuse functional papular eruptions over the face, were also reported. FPSH also occurs in patients with anhidrotic or hypohidrotic ectodermal dysplasia, those with Muir–Torre syndrome, and post-transplantation patients taking oral cyclosporin or azathioprine.[28,29] Single-lesion premature sebaceous hyperplasia (which has been postulated to be a hamartoma that originates from mature hair stem cells) is distinguished from FPSH by its distribution and number of lesions.[30] The testosterone examination results for the patient in this case report are within the normal range. The fact that the same condition are present in his brother and son, makes it suspected that the FPSH is associated with autosomal dominant inheritance.

Treatment strategies for CVG are contingent upon its underlying cause. Surgical scalp reduction procedures have been undertaken primarily for cosmetic purposes.[1,3] Surgical scalp reduction is the most common treatment, primarily performed for cosmetic improvement. Smaller furrows can often be managed with direct excision and primary closure, whereas larger, more extensive furrows typically require staged procedures, such as serial tissue expansion and flap advancements.[1] A variety of other treatment modalities have been explored for CVG, including pharmaceutical interventions like thyroid extract, corticosteroids, topical antiseptics, and antihistamines. Additionally, alternative approaches such as sleep therapy, irradiation, and psychotherapy have been trialed. However, these non-surgical methods have not demonstrated consistent or successful outcomes in managing the condition.[4]

Treatment for SH is predominantly aimed at cosmetic improvement.[8] Several therapeutic options have been described, including shave excision, isotretinoin therapy, bichloroacetic acid, cryotherapy, carbon dioxide laser ablation, electrodessication, erbium or yttrium aluminum garnet laser ablation, and pulsed-dye laser photothermolysis.[16] Prolonged remission of the lesions can be achieved following an adequate dosage of isotretinoin.[29] Isotretinoin works by exerting antiproliferative effects on sebocytes, reducing sebaceous gland size within one week of initiation. Its effects typically peak between 3 and 6 weeks, and perifollicular fibrosis can occur after 12 weeks of therapy.[14] The initial dosage typically ranged from 0.5 to 1 mg/kg per day for at least 3 weeks. Alternatively, initiating isotretinoin at a low dosage (10 mg/day) provides a safe, rational, and effective approach for addressing SH, with subsequent gradual tapering to a lower maintenance dose.[13,14] However, abrupt discontinuation of isotretinoin is associated with high recurrence rates. Maintenance therapy, achieving a cumulative dose of approximately 40–60 mg/kg, may be required to prevent recurrence. Topical treatments, such as tretinoin cream (0.05%) or isotretinoin gel (0.05%), can serve as adjunctive therapy. In female patients, antiandrogenic contraceptives, including cyproterone acetate and chlormadinone acetate, have been employed in some cases.[13] In this patient, we performed a shave excision on the protruding lesion on the left cheek. No further treatment was administered to the patient, as they declined it and felt that they were not bothered by the existing skin disorder.

Conclusion

In conclusion, we report an unique case of CVG in FPSH patient in twin brother with typical diffuse lesions, presenting as “leonine facies”. As a clinician, we must consider the diagnosis of FPSH because it tends to be underdiagnosed or misdiagnosed. A comprehensive examination is required for patients with this condition to establish a diagnosis and initiate early therapy to minimize the risk of progressing to a “leonine facies” appearance.

Ethics Statement

The publications of images were included in the consent from the patient’s and his son for publication of the case. Institutional approval has been obtained to publish the case details.

Consent Statement

The authors certify that they have obtained all appropriate patient consent forms from the patient and his son. The patient and his son signed a consent form for the publication of the case details and images.

Acknowledgments

The authors would like to thank the staff of the Department of Dermatology and Venereology, and Department of Pathological Anatomy, Faculty of Medicine, Universitas Padjadjaran, Bandung, West Java, Indonesia.

Funding

There is no funding to report.

Disclosure

The authors report no conflicts of interest in this work.

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