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*RESEARCH ARTICLE*

# CONGENITAL INFILTRATING LIPOMATOSIS OF THE CHEEK IN AN ADOLESCENT: A CASE REPORT AND LITERATURE REVIEW

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## Abstract

**Background:** Congenital infiltrating lipomatosis of the face (CILF) is a rare, histologically benign but locally infiltrative disorder characterised by a diffuse proliferation of mature, non-encapsulated adipose tissue that invades the facial soft tissues and, in some patients, the underlying bone and teeth. It belongs to the PIK3CA-related overgrowth spectrum (PROS).

**Methods:** We report a case managed at Oued Eddahab Military Hospital, Agadir, Morocco. Clinical, radiological, operative and histopathological data were reviewed and compared with the literature, with particular attention to molecular diagnosis, dentoskeletal assessment and emerging targeted therapies.

**Results**: A 15-year-old boy presented with a slowly progressive swelling of the left cheek, first noticed at approximately 10 years of age and responsible for facial asymmetry. Magnetic resonance imaging (MRI) showed diffuse fatty infiltration of the left buccal soft tissues involving the buccal fat pad and extending into the masticator space, without significant enhancement. No computed tomography or panoramic radiograph of the facial skeleton and no molecular testing for PIK3CA mutations were performed. Conservative partial excision was performed through an extended pretragal approach combined with a nasolabial fold incision. Histopathology showed mature adipose tissue without atypia or malignancy. The postoperative course was uneventful, with no facial nerve deficit and no salivary fistula, and no clinical regrowth was observed at the third month of follow-up.

**Conclusion:** CILF should be considered in any soft, ill-defined, unilateral cheek swelling that has progressed since childhood. MRI is essential for mapping the lesion, and surgery should be conservative and individualised, aiming at aesthetic and functional improvement while preserving the facial nerve and the parotid duct. A dentoskeletal assessment and, when available, molecular confirmation of a PIK3CA mutation in lesional tissue complete the work-up and may guide targeted therapy in extensive or recurrent forms. Long-term follow-up is mandatory because of the risk of recurrence.



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## Introduction:-

Congenital infiltrating lipomatosis of the face (CILF) is a rare entity first described by Slavin et al. in 1983 [1]. It consists of a diffuse proliferation of mature, non-encapsulated adipocytes that infiltrate the adjacent muscles and soft tissues, without evidence of malignancy [1,2]. Unlike a conventional lipoma, it has ill-defined margins, tends to extend progressively and is rarely amenable to complete excision. The cheek is one of the most frequently involved sites, and the disease usually presents as a unilateral facial asymmetry noticed at birth or in early childhood [2,3]. Depending on the extent of the lesion, dental, osseous, mucosal or parotid abnormalities may be associated [4–6]. Since the identification of activating somatic mutations of PIK3CA in lesional tissue, CILF has been classified within the PIK3CA-related overgrowth spectrum (PROS), a group of mosaic segmental overgrowth disorders that also includes CLOVES syndrome, fibroadipose hyperplasia and some forms of hemifacial hyperplasia [10,11,15]. The diagnosis relies on the correlation of clinical, radiological and histopathological findings, magnetic resonance imaging (MRI) being the reference investigation for characterising the fatty component and its extension [3].
We report a case of CILF of the left cheek in an adolescent and discuss its diagnostic, therapeutic and prognostic aspects in the light of the current literature, with emphasis on the place of molecular testing, dentoskeletal evaluation and targeted PI3K inhibition.

## Materials and Methods:-

This work is a single-patient case report combined with a narrative review of the literature, prepared in accordance with the CARE (CAseREport) guidelines. The patient was managed in the Department of Oral and Maxillofacial Surgery of Oued Eddahab Military Hospital, Agadir, Morocco. Data were retrieved from the medical record and included the history of the disease, extraoral and intraoral examination, MRI findings, the operative report, the postoperative course, the duration and findings of postoperative follow-up and the histopathological report.

The clinical variables recorded were age, sex, medical history, age at onset and duration of progression, characteristics and morphological impact of the swelling, condition of the oral mucosa, patency of the parotid (Stensen's) duct orifice, state of the dentition and occlusion, and examination of the parotid gland, cranial nerves and cervical lymph nodes. MRI was analysed for signal intensity, suppression on fat-saturated sequences, enhancement after gadolinium administration, and the relationship of the lesion with the buccal fat pad and the masticator space. Whether hard-tissue imaging (panoramic radiograph or computed tomography) and molecular analysis of lesional tissue had been performed was also recorded. The literature review covered clinical series, imaging features, dentoskeletal abnormalities, genetic data, the differential diagnosis within the PROS and therapeutic options, including targeted medical therapy, in CILF. Written informed consent for publication, including clinical images, was obtained from the patient's legal representative, and all images were anonymised.

**Results: Case Presentation:-**

**Patient information:-**

A 15-year-old boy with no relevant medical history presented with a swelling of the left cheek that had first been noticed by his parents at approximately 10 years of age and had progressed slowly thereafter, without any period of rapid growth. There had been no inflammatory episode, spontaneous pain, bleeding or sign of infection. He reported no sensory disturbance, no feeding difficulty and no episode of facial palsy. The main complaint was facial asymmetry, which had become more conspicuous with growth.

**Clinical findings:-**

Extraoral examination revealed a soft, ill-defined, painless, non-inflammatory, non-pulsatile and non-compressible swelling of the lower left cheek, measuring approximately 3 × 6.5 cm, with downward displacement of the left labial commissure (Figure 1). The overlying skin was normal, with no erythema, local warmth or visible superficial venous network, and no epidermal naevus. Intraoral examination showed bulging of the left buccal mucosa without ulceration or mucosal infiltration; the parotid duct orifice was patent and non-inflamed. The tongue was of normal size and symmetrical. Inspection of the dentition showed no clinically apparent macrodontia, premature eruption or asymmetry of the dental arches, and the occlusion was preserved. The parotid gland was not enlarged. Examination of the cranial nerves, including facial nerve function, was normal, and the cervical lymph node areas were free.

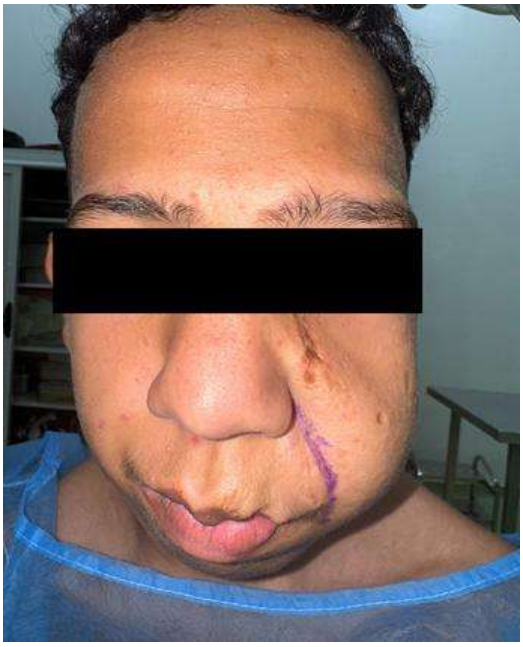

**Figure 1. Preoperative frontal view showing infiltrative hypertrophy of the lower left cheek responsible for facial asymmetry and downward displacement of the left labial commissure; the planned nasolabial fold incision is marked on the skin. The periorbital region has been masked to preserve anonymity.**

**Diagnostic assessment:-**

Facial MRI showed an ill-defined lesion with a fatty signal, hyperintense on T1- and T2-weighted images (Figure 2A), with signal suppression on fat-saturated sequences (Figure 2B) and no significant enhancement after gadolinium injection. The lesion diffusely infiltrated the left buccal soft tissues, involved the buccal fat pad and extended into the masticator space (Figure 2B, C). These features favoured infiltrating lipomatosis over a vascular or lymphatic malformation. No panoramic radiograph (orthopantomogram) or computed tomography (CT) of the facial skeleton was performed before surgery, so that subclinical osseous hypertrophy of the maxilla or mandible and tooth root morphology could not be assessed. No molecular analysis of the lesional tissue for PIK3CA mutations was carried out, as tissue-based genetic testing is not routinely available at our institution.

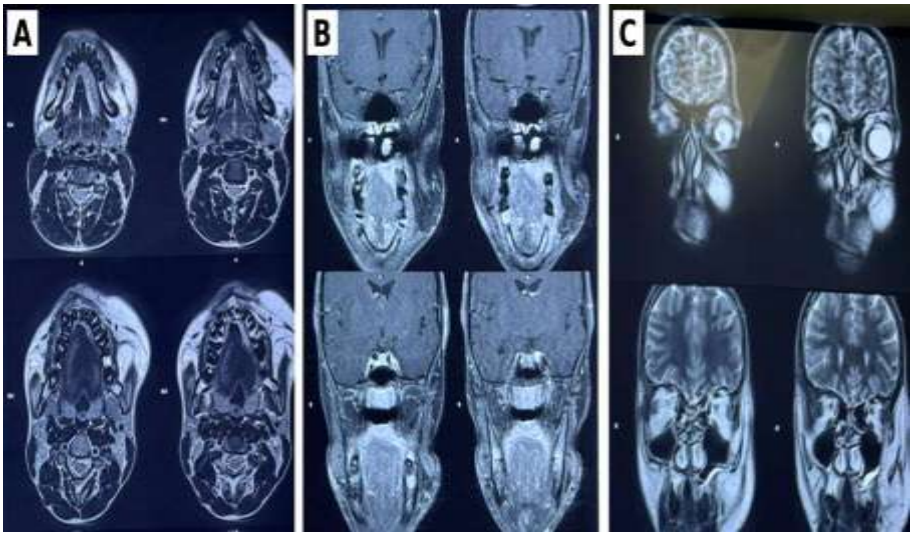


**Figure 2. Facial MRI. (A) Axial T1-weighted slices showing diffuse fatty infiltration of the left buccal soft tissues, hyperintense and ill-defined. (B) Coronal fat-suppressed slices showing signal suppression of the lesion and its extension towards the deep spaces of the face (buccal fat pad and masticator space). (C) Additional coronal sequences confirming the buccal location and the fatty nature of the lesion and the absence of significant enhancement.**

**Therapeutic intervention:-**

Surgery was performed under general anaesthesia through two approaches: a left extended pretragal (preauricular) incision, providing access to the posterior buccal region and the masseteric space, and a nasolabial fold incision to control the anterior component. After dissection in the subcutaneous plane superficial to the superficial musculoaponeurotic system (SMAS), the lesion appeared as yellowish, diffuse, non-encapsulated adipose tissue infiltrating the buccal muscle planes without a cleavage plane (Figure 3). A partial, stepwise, piecemeal resection was carried out; complete deep excision was deliberately not attempted in order to preserve the branches of the facial nerve, the parotid duct and perioral mobility.

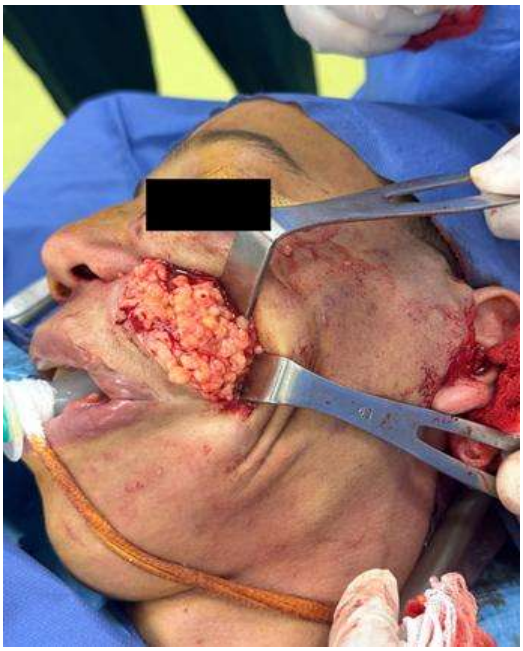

**Figure 3. Intraoperative view showing diffuse infiltration of the left buccal region by yellowish, non-encapsulated adipose tissue.**

**Follow-up, outcome and histopathology:-**

The immediate postoperative course was uneventful, without facial nerve deficit, salivary fistula or infection. Histopathological examination of the specimen showed a proliferation of mature adipocytes without lipoblasts or cytological atypia, consistent with a lipomatous lesion and with no evidence of malignancy. Given the diffuse, non-encapsulated, infiltrative and long-standing nature of the lesion, correlation of the clinical, radiological, operative and histological findings established the diagnosis of CILF of the left cheek. At the last follow-up visit, three months after surgery, the aesthetic improvement was stable, facial nerve function and perioral mobility were preserved and there was no clinical evidence of regrowth. Clinical follow-up every six months until the end of growth, with MRI in the event of clinical suspicion of recurrence, and a dental and orthodontic assessment have been planned. The timeline of the case is summarised in Table 1.

**Table 1. Timeline of the patient's management (CARE guidelines).**

| Period | Clinical event |
|---|---|
| **Early childhood** | Onset of a swelling of the lower left cheek, first noticed at approximately ten years of age. |
| **Childhood – adolescence** | Slow, painless progression without inflammatory or vascular signs; worsening facial asymmetry with growth. |
| **Presentation (age 15 years)** | Soft, ill-defined, painless, non-pulsatile mass (≈ 3 × 6.5 cm) with downward displacement of the left labial commissure. |
| **Diagnostic work-up** | MRI: diffuse fatty infiltration of the left buccal soft tissues, buccal fat pad and masticator space, without significant enhancement. No orthopantomogram or CT; no molecular testing. |

| Period | Clinical event |
|---|---|
| **Surgery** | Conservative partial piecemeal excision under general anaesthesia through extended pretragal and nasolabial fold approaches. |
| **Early postoperative course** | Uneventful: no facial nerve deficit, no salivary fistula, no infection. |
| **Histopathology** | Mature adipose proliferation without lipoblasts or atypia; no evidence of malignancy. |
| **Follow-up at 3 months** | Stable aesthetic result, preserved facial nerve and perioral function, no clinical regrowth. |
| **Final diagnosis and follow-up plan** | CILF of the left cheek; six-monthly clinical follow-up until the end of growth, MRI if regrowth is suspected, dental/orthodontic assessment; molecular testing of lesional tissue if reoperation or targeted therapy is considered. |

**CILF: congenital infiltrating lipomatosis of the face; CT: computed tomography; MRI: magnetic resonance imaging.**

## Discussion:-

CILF occupies a particular place among adipose lesions of the head and neck: it is histologically benign but locally infiltrative, which explains the difficulty of its treatment. Its classic histological description comprises mature, non-encapsulated adipocytes infiltrating muscles and soft tissues, without lipoblasts or cytological atypia, sometimes accompanied by nerve fibres, fibrous septa and increased vascularity [1,2]. A pathology report of "lipoma" is therefore insufficient on its own and must be interpreted together with the clinical, radiological and operative findings. In our patient, the tissue was mature and benign but diffuse, ill-defined and infiltrative, which fits the spectrum of CILF rather than an encapsulated lipoma.The age at diagnosis and the natural history of our case are also characteristic. Most patients present with facial asymmetry at birth or in early childhood, followed by slow progression during growth [2,4]. Paucisymptomatic forms may be neglected for a long time and managed only in adolescence, when the aesthetic impact becomes more pronounced. In our patient, the swelling was first noticed at approximately ten years of age and progressed slowly without pain, inflammatory signs or vascular manifestations. This chronic, silent course is consistent with published reports, in which facial asymmetry rather than functional impairment is the main complaint [4,7].

The cheek is a frequent site of the disease, but involvement may extend to the lip, tongue, parotid gland, masticatory muscles, soft palate, maxilla, mandible or zygoma [5,6,8]. In recent series, involvement is usually hemifacial and generally respects the midline; the most extensive forms may be associated with macroglossia, labial thickening, epidermal naevi, bone hypertrophy and dental abnormalities (Figure 4) [8,9]. Our case is distinguished by a predominantly buccal presentation without macroglossia, visible mucosal involvement or neurological abnormality. This localised form nevertheless remains typical because of the unilateral, progressive and infiltrative nature of the mass.


**Figure 4. Clinical presentations of facial infiltrating lipomatosis illustrating phenotypic variability: epidermal naevi in the trigeminal dermatomes (A, B), labial thickening (C) and hemimacroglossia with lower lip thickening (D). Reproduced from Chen et al. [8], an open-access article distributed under the Creative Commons Attribution 4.0 International License (CC BY 4.0).**

Imaging is decisive for the diagnosis. Ultrasonography may be performed first, but it hardly distinguishes pathological fatty infiltration from normal buccal fat and is of limited value for assessing deep extension [3]. Computed tomography (CT) is useful for evaluating bony structures, dental abnormalities and skeletal hyperplasia. MRI is the most informative investigation for soft tissues: the lesion is hyperintense on T1- and T2-weighted images, with signal suppression on fat-saturated sequences and absent or faint enhancement after gadolinium administration [3]. These criteria help to exclude lymphatic or venous malformations, which may mimic a cheek swelling in a child. The MRI of our patient showed exactly this profile, with extension to the buccal fat pad and the masticator space.

Dental and osseous abnormalities deserve particular attention even when they are not obvious on initial examination. Accelerated tooth formation or eruption, macrodontia, root hypoplasia, early loss of deciduous or permanent teeth and hypertrophy of the maxilla or mandible on the affected side have been reported [6,9]; Sun et al. showed that dental abnormalities are a frequent marker of the disease [6]. Osseous hyperplasia underlying the fatty infiltration is well demonstrated by CT, as in a Moroccan case reported by Harouna et al. (Figure 5) [7]. In our patient, these manifestations were not clinically prominent, but no orthopantomogram or CT was obtained before surgery, so that subclinical bony expansion, macrodontia or root abnormalities on the affected side cannot be formally excluded. This is a limitation of our report: because MRI was sufficient to characterise the soft-tissue lesion and to plan a conservative procedure, hard-tissue imaging was not considered necessary at the time, whereas the literature indicates that dentoskeletal involvement is frequent and often underestimated [6,9]. A panoramic radiograph and, when bone hypertrophy is suspected, a three-dimensional CT should therefore be part of the initial work-up of every child or adolescent with CILF; a complete dental and osseous assessment is also advisable during follow-up in order to detect underestimated involvement and to plan orthodontic treatment if required. Such an assessment has been scheduled for our patient.

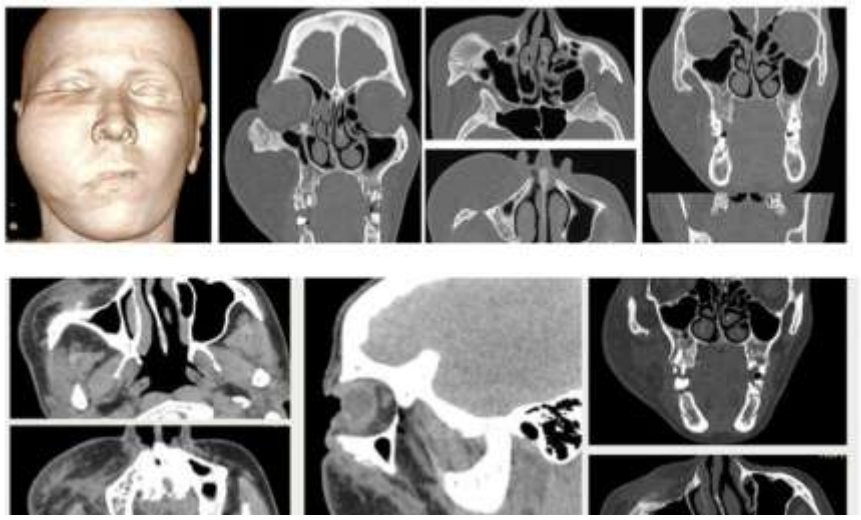

**Figure 5. Moroccan case of facial infiltrating lipomatosis: three-dimensional CT reconstruction and CT slices showing soft tissue thickening of the right hemiface and hyperplasia of the underlying bones. Reproduced from Harouna et al. [7], an open-access article distributed under the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (CC BY-NC-ND 4.0).**

The differential diagnosis includes simple lipoma, lipoblastomatosis, vascular or lymphatic malformations, hemifacial hyperplasia, Proteus syndrome, other lesions of the PIK3CA-related overgrowth spectrum (PROS) and, more rarely, malignant adipose tumours [9–11]. Slow progression, soft consistency, absence of pulsatility, fatty signal on MRI and absence of histological atypia point towards a benign lesion, whereas the absence of a capsule and the infiltration of the deep planes distinguish infiltrating lipomatosis from a conventional lipoma. In our case, the lesion was neither compressible nor pulsatile, showed no vascular enhancement and infiltrated the buccal planes, making a vascular malformation unlikely. The distinction between CILF and the other overgrowth phenotypes with which it may be confused is summarised in Table 2. Isolated hemifacial hyperplasia involves all tissue layers of the hemiface, including bone and teeth, in a proportionate manner, whereas in CILF the overgrowth is predominantly adipose and infiltrative on MRI; some authors nevertheless regard CILF as a subtype of hemifacial hyperplasia, both entities sharing PIK3CA mutations [9]. CLOVES syndrome is a PIK3CA-related disorder characterised by truncal lipomatous overgrowth, vascular malformations, epidermal naevi and scoliosis or skeletal anomalies, which were

absent in our patient [11,15]. Proteus syndrome, caused by a mosaic activating mutation of AKT1 rather than PIK3CA, is distinguished by its postnatal onset, relentless and disproportionate progression, cerebriform connective tissue naevi and progressive skeletal overgrowth [15]. In our patient, the strictly unilateral, adipose, buccal overgrowth without vascular, truncal, acral or skeletal anomalies was consistent with an isolated form of CILF.

**Table 2. Differential diagnosis of CILF within the PIK3CA-related overgrowth spectrum and related phenotypes [9–11,15].**

| Entity | Gene (mosaic) | Typical features | Distinguishing points from CILF |
|---|---|---|---|
| **Congenital infiltrating lipomatosis of the face** | PIK3CA (somatic) | Unilateral, infiltrative overgrowth of mature adipose tissue of the cheek and hemiface; ± ipsilateral maxillary/mandibular hypertrophy, macrodontia, early eruption, macroglossia, epidermal naevi; present at birth or early childhood, slow progression | Reference entity: overgrowth predominantly adipose, infiltrative on MRI, respecting the midline |
| **Hemifacial hyperplasia (isolated)** | Mostly idiopathic; PIK3CA in some cases | Proportionate enlargement of all tissues of the hemiface (skin, soft tissue, bone, teeth) present at birth; bone and dental involvement prominent | Bone and dental overgrowth dominate; some authors consider CILF a subtype of hemifacial hyperplasia |
| **CLOVES syndrome** | PIK3CA (somatic) | Congenital lipomatous overgrowth of the trunk, vascular malformations (capillary, venous, lymphatic), epidermal naevi, scoliosis/skeletal and spinal anomalies, acral deformities | Truncal predominance and vascular malformations; facial involvement unusual |
| **Proteus syndrome** | AKT1 (somatic, p.Glu17Lys) | Postnatal onset with rapid, relentless and disproportionate overgrowth; cerebriform connective tissue naevus, progressive skeletal overgrowth, lipomas and lipoatrophy, vascular malformations | Different gene; progressive and asymmetric multisystem overgrowth, not confined to the face |

CILF: congenital infiltrating lipomatosis of the face; CLOVES: congenital lipomatous overgrowth, vascular malformations, epidermal naevi, scoliosis/skeletal and spinal anomalies; MRI: magnetic resonance imaging.

Understanding of the pathophysiology has advanced considerably through genetic studies. Activating somatic mutations of PIK3CA, the gene encoding the catalytic alpha subunit of phosphatidylinositol 3-kinase (PI3K), have been identified in affected tissues [10]. These mutations activate the PI3K–AKT–mTOR pathway, which drives cell growth and tissue proliferation, and place the disease within the PROS [8,11]. This explains the variability of presentations, ranging from localised buccal involvement to extensive hemifacial forms with bone abnormalities, neurological involvement or associated malformations. The mutations are post-zygotic and mosaic, confined to the affected tissue, so that they are usually undetectable in peripheral blood; their identification requires targeted next-generation sequencing (NGS) of DNA extracted from lesional tissue, ideally fresh or frozen, with sufficient depth to detect low variant allele fractions [10,11,15]. According to the diagnostic framework proposed by Keppler-Noreuil et al., molecular confirmation of PROS relies on the identification of a somatic PIK3CA mutation. When no mutation is identified despite compatible clinical features, the diagnosis may be considered presumptive PROS [15]. In our patient, molecular analysis was not performed because tissue-based NGS is not routinely available in our institution, and because the diagnosis of CILF was established on concordant clinical, radiological, operative and histological findings, which remain the cornerstone of diagnosis. Our case therefore remains clinically compatible with the PROS spectrum, without molecular confirmation. We nevertheless consider that molecular testing of

lesional tissue should be requested (i) in the event of recurrence or extension requiring reoperation, when fresh tissue can be sampled; (ii) in the presence of syndromic features (truncal lipomatous overgrowth, vascular malformations, macrodactyly, megalencephaly) suggesting CLOVES or another PROS phenotype; and (iii) whenever targeted medical therapy is contemplated, as molecular confirmation of a PIK3CA mutation is important for establishing eligibility for targeted PI3K inhibition [15–17]. Formalin-fixed, paraffin-embedded tissue from the initial resection can also be analysed retrospectively, albeit with a lower sensitivity.

Treatment is essentially surgical, but it should not be conceived as an oncological resection. The realistic objective is to reduce volume, improve symmetry and preserve function. Complete excision is often impossible because the pathological fat infiltrates muscles, salivary glands and the deep regions of the face, with a risk of injury to the facial nerve, the parotid duct or vascular structures [4,12,13]. Depending on age, extent of disease and the patient's wishes, the options include partial reduction, liposuction, soft tissue remodelling, cheiloplasty, bone contouring and orthodontic treatment, often in several stages [6,13,14]. In our case, partial piecemeal excision through a double approach was adapted to the buccal topography and to the need to preserve vital structures; the absence of facial palsy and salivary fistula supports this cautious strategy.

The identification of the PI3K–AKT–mTOR pathway as the driver of the disease has opened the way to targeted medical treatment. Sirolimus, an mTOR inhibitor, was the first agent used in PROS, with partial and inconsistent results. Alpelisib, an oral selective inhibitor of the p110α catalytic subunit of PI3K, was shown by Venot et al. to reduce the volume of overgrowth lesions, to improve symptoms and to be well tolerated in patients with severe PROS, including facial infiltrating lipomatosis, at low doses (50 mg/day in children and 250 mg/day in adults in the initial series) [16]. On the basis of the retrospective EPIK-P1 study, in which 37.5% of evaluable patients achieved a ≥20% reduction in target lesion volume at 24 weeks, alpelisib received accelerated approval from the United States Food and Drug Administration in 2022 for patients aged two years and older with severe manifestations of PROS requiring systemic therapy [17]. The main adverse effects are hyperglycaemia, stomatitis, diarrhoea and, in children, a possible effect on growth, which requires monitoring [16,17]. In a localised form such as ours, adequately managed by conservative surgery, systemic alpelisib would not currently be warranted; it may, however, be considered in severe, extensive, functionally disabling or recurrent disease requiring systemic therapy [11,16,17]. This reinforces the interest of preserving frozen lesional tissue at the time of surgery whenever possible.

The risk of recurrence remains a major element of follow-up. Recurrence or regrowth is explained by the persistence of infiltrating tissue in the deep planes and by the progression of the disease during growth [2,4,12]. Some teams therefore recommend deferring aggressive procedures until the end of growth, except in the case of significant functional, social or psychological impact [6,13]. In our patient, no clinical regrowth was observed at three months, but this follow-up is short in relation to the remaining growth period, and regrowth during the adolescent growth spurt cannot be excluded. Our patient therefore requires prolonged follow-up, planned every six months until the end of growth, focused on the stability of the aesthetic result, perioral function, facial nerve integrity and the detection of recurrence, with MRI whenever regrowth is suspected.

The interest of this report lies in documenting an isolated buccal form of CILF in a Moroccan adolescent, with typical imaging findings and conservative management. Its limitations are the single-case design, the absence of genetic testing (and therefore the absence of molecular confirmation of PROS), the lack of an orthopantomogram or CT assessment of the bony and dental structures and the short postoperative follow-up of three months, which does not allow the risk of recurrence during the remaining growth period to be assessed. Despite these limitations, it is a reminder that CILF should be considered in any soft, unilateral, ill-defined cheek swelling progressing since childhood, especially when MRI shows diffuse fatty infiltration without significant enhancement.

## Conclusion:-

Congenital infiltrating lipomatosis of the cheek is a rare, benign but locally infiltrative condition responsible for progressive facial asymmetry. Our case underlines the value of MRI in confirming the fatty nature of the lesion, defining its deep extension and guiding the operative strategy. Treatment relies on cautious, often partial surgery aimed at aesthetic improvement while preserving the facial nerve and the parotid duct. A dentoskeletal assessment should be part of the initial work-up, and molecular confirmation of a PIK3CA mutation in lesional tissue, when available, is recommended in recurrent, extensive or syndromic forms, in which targeted PI3K inhibition may complement surgery. Prolonged follow-up remains essential because of the risk of recurrence and of progression with growth.

**Declarations:-**

**Ethics approval and consent to participate:-**

This work was conducted in accordance with the principles of the Declaration of Helsinki. As the patient was a minor at the time of management, both the patient and his legal representative were informed and gave their consent.

**Consent for publication:-**

Written informed consent for the publication of this case report and of the accompanying clinical images was obtained from the patient's legal representative. The images were anonymised to protect the patient's identity. A copy of the written consent is available for review by the Editor-in-Chief on request.

**Availability of data and materials:-**

All data generated or analysed during this study are included in this article. Further information is available from the corresponding author on reasonable request.

**Competing interests:-**

The authors declare that they have no competing interests.

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**Authors' contributions:-**

RB: conception of the report, clinical management, data collection and drafting of the manuscript. SK and AO: surgical management, acquisition of operative data and critical revision of the manuscript. OEG: literature review, drafting of the discussion and verification of references. ZS: supervision and critical revision of the manuscript. All authors read and approved the final version of the manuscript.

**Reporting guideline:-**

This case report was prepared in accordance with the CARE guidelines.

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