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Rare Gingival Manifestation of Sarcoidosis in a 13-Year-Old Patient: A Case Report

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Case Report

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Abstract

Background

Oral sarcoidosis is an uncommon granulomatous disorder, and isolated gingival involvement as the initial manifestation is exceptionally rare, particularly in children.

Case presentation:

A 13-year-old male presented with a three-year history of progressive gingival enlargement and upper lip swelling. Medical history revealed allergic rhinitis, with no medication use or familial predisposition. Extraoral examination showed symmetric upper lip enlargement with taut, dehydrated mucosa. Intraorally, erythematous, predominantly fibrotic gingival overgrowth involved the anterior regions of both arches and was disproportionate to local plaque deposits. External bevel gingivectomy was performed in two stages. Histopathological examination revealed non-caseating epithelioid granulomas with multinucleated giant cells, prompting systemic evaluation. Laboratory investigations showed leukocytosis (10.31 × 10^3/μL), eosinophilia (7.10 × 10^3/μL), elevated serum angiotensin-converting enzyme (95 IU/mL), raised ESR (24 mm/hour), and hypergammaglobulinemia with increased IgG, IgA, and IgE. Upper lip swelling was treated with intralesional triamcinolone acetonide (40 mg/mL), resulting in gradual resolution. The patient was followed for 24 months without recurrence and with uneventful healing.

Conclusion

Gingival sarcoidosis may present as the sole clinical manifestation in children. Histopathological assessment and systemic evaluation are essential for early diagnosis and appropriate multidisciplinary management.

Background

Sarcoidosis represents a multi-organ granulomatous condition of undetermined origin, marked histopathologically by the deposition of non-caseating epithelioid granulomas within the affected tissues.[1] The disease can impact virtually any organ system, with the lungs, lymph nodes, integument, eyes, hepatic tissue, spleen, brain, and cardiac muscle being among the most commonly affected.[2] Establishing a definitive diagnosis of sarcoidosis poses a considerable clinical challenge, as the disorder is widely regarded as a "disease of uncertainties" with respect to its underlying etiology, clinical manifestations, and natural progression.[3,4] Furthermore, no single diagnostic investigation reliably confirms its presence.[3] Its clinical trajectory remains difficult to predict and can occasionally be severe

or even fatal, with therapeutic interventions frequently failing to alter the natural course of the illness.[4] Consequently, sarcoidosis continues to confound both researchers and treating clinicians alike.

Within the maxillofacial region, the salivary glands are commonly implicated, and clinical features such as xerostomia, bilateral enlargement of the parotid glands, or facial nerve palsy may occasionally be observed.[5] Oral sarcoidosis, which refers to lesions developing within the soft tissues of the oral cavity, is regarded as uncommon, and its true prevalence has not been clearly established. When sarcoidosis does involve the oral cavity, the lips, hard and soft palate, buccal mucosa, gingivae, tongue, and tonsils are the most frequently reported sites.[6] Sarcoid involvement of the gingivae as the primary or inaugural presenting lesion is regarded as exceptionally uncommon[5], with only a limited number of such reports existing in the literature.[7–10]Available evidence indicates that gingival involvement generally arises in patients already known to have long-standing multisystem sarcoidosis, affecting approximately one-third to as many as two-thirds of such individuals.[9,10] The kind of gingival sarcoidosis presentation that may constitute the first and sole clinical manifestation of the disease is extremely rare. Given its rarity, the documentation of such cases is critical in alerting clinicians to the importance of early recognition and the timely implementation of appropriate management for both the systemic and dental dimensions of the disease. The case described below illustrates one such atypical presentation, in which the gingiva represented the only site of sarcoid involvement, manifesting as diffuse gingival overgrowth and upper lip swelling as the sole presenting sign and symptom.

Case Presentation

A 13-year-old male was brought to the Department of Periodontology at Government Dental College, Raipur, India with a three-year history of progressive gingival enlargement along with swelling of the upper lip. At the time of consultation, the gingival and lip disfigurement was adversely affecting the patient's psychological state, with the chief concern relating to facial appearance, although he reported only minimal physical discomfort. He additionally described occasional gingival bleeding and a sensation of oral dryness attributed to a habit of mouth breathing. There was no history of spontaneous bleeding from the gingivae, nor was there any history of medication use.

The patient's medical background was notable for allergic rhinitis. No hereditary predisposition was identified. His immunization record was complete, with no associated adverse events. The history was unremarkable for gastrointestinal, cardiovascular, neurological or fungal disorders. The dental history revealed no delayed eruption of teeth and no history of dental fractures. There was no history of consanguinity between the parents, and no familial occurrence of similar complaints was reported.

Extraoral assessment demonstrated a competent, symmetrically enlarged upper lip. The overlying mucosa appeared stretched and dehydrated, with no alteration in pigmentation. Mild fissuring was observed around the lip region. Neurological examination confirmed intact facial nerve function. Palpation of the upper lip revealed a soft, edematous, and non-tender consistency.

Intraoral examination identified erythematous and hyperplastic gingival tissues affecting the labial aspect of the maxillary right canine extending to the maxillary left canine, involving the marginal, interdental, and attached gingiva up to the mucogingival junction. The overgrowth extended coronally to cover the crowns up to the middle and incisal thirds in the maxilla. In the mandibular arch, enlargement was evident in relation to the right lateral incisor and canine, extending to the mucogingival junction and covering the middle third of the crowns. The enlarged gingivae appeared erythematous, smooth, and shiny, with loss of stippling and loss of the normal gingival contour (Fig. 1a, 1b, 1c). Palpation revealed that the enlargement was predominantly fibrotic in nature. Periodontal probing elicited bleeding from the base of the pseudo-pockets, with mild local deposits evident but no clinical attachment loss. A panoramic radiograph demonstrated no alveolar bone loss in the maxillary or mandibular anterior segments (Fig. 1d). The dorsum of the tongue appeared normal, and the oral cavity was adequately lubricated, with clear saliva.

The etiology of gingival overgrowth encompasses a range of factors, including suboptimal oral hygiene, systemic diseases and conditions, metabolic disturbances, hematologic abnormalities, nutritional deficiencies, drug-induced reactions, hereditary causes, or idiopathic origins. In the present instance, the patient's oral hygiene was satisfactory, and the quantity of plaque deposits did not correlate with the magnitude of gingival enlargement. Given the absence of a family history and no history of medication usage, hereditary and drug-induced forms of gingival overgrowth were excluded. Furthermore, as the lip swelling showed no regression or recurrence and was not accompanied by facial paralysis or fissured tongue, Melkersson-Rosenthal syndrome was also ruled out.

A provisional diagnosis of idiopathic gingival enlargement was established, and treatment was initiated. The initial therapy comprised oral prophylaxis, including scaling and root planing, along with oral hygiene reinforcement. The patient was instructed to rinse twice daily with 0.2% chlorhexidine mouthwash, using 10 ml in a 1:1 dilution, for a duration of two weeks. Upon re-evaluation after one month, the gingival enlargement remained unchanged in severity, dimensions, clinical features, and consistency. Consequently, an inflammatory etiology was excluded, and the patient was scheduled for surgical excision of the hyperplastic tissue.

Informed consent

was obtained from the parent, and the requisite baseline laboratory investigations were conducted prior to surgery. The maxillary anterior region was selected for the first surgical appointment, as the enlargement was most pronounced in this area. Surgical excision was carried out under local anaesthesia employing the external bevel gingivectomy technique (Fig. 1e). The residual gingival enlargement in the mandibular anterior region was addressed one week later using the same external bevel gingivectomy technique. The patient remained under follow-up for two years following the completion of gingivectomy, during which healing was uneventful and no recurrence of gingival enlargement was observed (Fig. 1f, 1g).

Published literature emphasizes the need for histopathological confirmation of the excised tissue, as well as systemic evaluation to rule out associated involvement and establish a differential diagnosis. Accordingly, excised gingival tissue from both the maxillary and mandibular regions was submitted for histopathologic assessment. Microscopic examination revealed parakeratinized to non-keratinized stratified squamous epithelium of varying thickness, with areas of atrophy alongside regions demonstrating hypertrophy and pseudoepitheliomatous hyperplasia. Dyskeratotic features, koilocyte-like cells, acantholysis, and inflammatory cell infiltration were also noted. The underlying connective tissue was fibrocellular in nature, harbouring moderate to severe chronic inflammatory cell infiltration and ill-defined non-caseating epithelioid cell granulomatous regions containing multinucleated giant cells distributed throughout the granulomatous formations (Fig. 2).

In light of these histopathological findings, it became evident that the enlargement was attributable to an underlying chronic granulomatous condition, with possible diagnoses including orofacial granulomatosis, Crohn's disease, sarcoidosis, Wegener’s granulomatosis or tuberculosis. A battery of specialized investigations was therefore undertaken to differentiate between these granulomatous disorders. Laboratory investigations encompassed a basic metabolic panel, complete blood count with differential leukocyte count, erythrocyte sedimentation rate (ESR), hepatic function panel, renal function tests, HAV antibody, HCV antibody, HBV envelope antibody and antigen, C-reactive protein, immunoglobulin panel, C4 complement, C3 complement, glomerular basement membrane antibodies, cytoplasmic neutrophil antibodies, and serum angiotensin-converting enzyme (sACE) levels. Additional investigations as requested by the referred physician included the Mantoux test, sputum analysis, chest radiograph (posteroanterior view), high-resolution computed tomography (HRCT) lungs, intestinal contrast ultrasonography, electrocardiography (ECG), cardiac and brain magnetic resonance imaging (MRI).

Acid-fast bacilli were not identified on Ehrlich-Ziehl-Neelsen staining of the oral tissue specimen, thereby excluding mycobacterial infection. The Mantoux and sputum tests were negative for tuberculosis. The chest radiograph and HRCT scans appeared normal, with no evidence of hilar lymphadenopathy. Following a comprehensive medical history review along with intestinal contrast ultrasonography, Crohn's disease was excluded by the treating physician. Similarly, the absence of systemic signs, pathognomic ‘strawberry gingivitis’ and normal p-ANCA and c-ANCA excluded the possibility of granulomatosis with polyangiitis (historically known as Wegener’s granulomatosis).

The complete blood count revealed a mildly elevated leukocyte count (10.31 × 10^3/μL) with mild eosinophilia (7.10 × 10^3/μL), moderately elevated sACE levels (95 IU/mL), raised ESR (24 mm/ hour), slightly decreased transferrin% (15.1%), normal serum and urine calcium, and an altered immunological profile (T4:T8 ratio 3.8) with raised immunoglobulin levels- IgG (19.4g/L), IgA (4.69g/L), IgE(680IU/ml), C4(0.41g/L). The cumulative clinicopathological and biochemical evidence strongly supported a diagnosis of gingival sarcoidosis, with detailed findings presented in Table 1.

Table 1
Results of the blood parameters and other investigations done

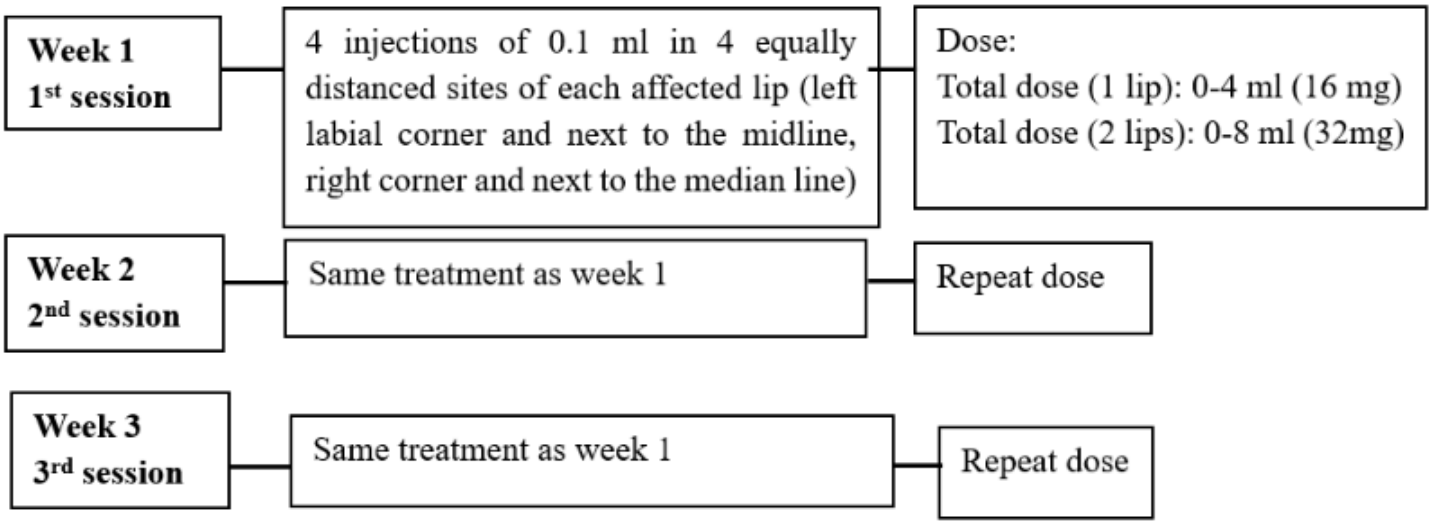
Examinations	**Results**
Haemoglobin	12.5 g/dl
Red blood cell count	4.57 mill/cmm
Total white blood cell count	10.31 x 10^3/uL*
Neutrophil count	2.46 x 10^3/uL
Eosinophil count	7.10 × 10^3/μL*
Lymphocyte count	1.35 x 10^3/uL
Platelet count	192,000/uL
Erythrocyte sedimentation rate	24mm/hour *
High sensitive C-Reactive protein	0.19 mg/dL
Leukocyte phagocytic activity	> 40%
Neutrophil alkaline phosphatase score (40 to 100 is normal)	97
IgE	680 IU/ML*
Iron deficiency profile	Normal except transferrin% 15.1 (low)*
Vitamin B12, Folic acid, vitamin D	Normal
Serum Angiotensin converting enzyme (ACE)	95 U/L*
T4:T8	3.8
IgM, IgG, IgA, C3, C4 (g/L)	Normal,19.4* 4.69* Normal, 0.41*
Antibodies (human immunodeficiency virus, hepatitis B, A, tuberculosis, cytoplasmic anti-neutrophil cytoplasmic, peri-nuclear anti-neutrophil cytoplasmic)	Negative
Serum calcium, liver function test, kidney function test, urine analysis, stool examination	Normal
Blood glucose (fasting/postprandial)	Normal
Intestinal contrast ultrasonography	Normal
Cardiac and brain MRI	Normal
Electrocardiogram	Normal
Bold font with * represents elevated/ altered levels compared to normal values	

Examinations	Results
Posterior–anterior of the chest	Normal
HRCT lungs	Normal
Mantoux test	Negative
Histopathology	Negative for PAS, Congo red
Allergy test	Allergic to cocoa, tomatoes, inorganic dust
Bold font with * represents elevated/ altered levels compared to normal values	

The patient was referred to a physician for further systemic evaluation and management. In the absence of any systemic features suggestive of sarcoidosis, together with normal chest radiograph, HRCT lungs, ECG, cardiac and brain MRI and intestinal contrast ultrasonography, systemic sarcoid involvement was excluded. No pharmacological therapy for sarcoidosis was prescribed, and the patient remained under regular physician follow-up for two years.

Regarding the lip swelling, the patient was referred to a dermatologist who prescribed clofazimine 100 mg twice daily for ten days, followed by twice weekly for one month; however, no improvement was observed. Subsequently, intralesional corticosteroid therapy with triamcinolone (40 mg/mL) was administered into the lips on a monthly basis for three weeks (Table 2, Fig. 3).[11] The swelling gradually resolved, and regular review over two years revealed no recurrence.

Table 2. Treatment protocol of Intralesional injection of triamcinolone acetonide (40 mg/ml) for lip enlargement



Discussion

Sarcoidosis is a systemic granulomatous condition with worldwide distribution affecting all ethnic groups.[12] In contrast to western countries, sarcoidosis constitutes highest burden among all the interstitial lung diseases in India.[13] A slight female predominance has been documented, with males typically presenting during the third to fifth decade and females during the fourth to sixth decade of life.[12,13] However, the current case involved an adolescent male aged 13 years. A literature review conducted by Suresh et al. noted that only four such cases had been recorded in children between the ages of 5 and 16 years.[9]

The clinical manifestations of sarcoidosis are highly heterogeneous, and disease onset may be acute (≤ 2 years) or sub-acute/chronic (≥ 3–5 years).[4] Sarcoid lesions can manifest in three principal patterns: (a) widespread involvement of one or more organ systems, (b) isolated local lesions that may later evolve into disseminated sarcoidosis over years, and (c) reactions to specific local agents such as beryllium.[8] Pulmonary and intrathoracic lymph node involvement is observed in more than 90% of cases. Involvement of other organ systems, including the hepatic, cardiac, and neurological systems, as well as glandular structures, occurs less frequently.[4] The present case appears to represent the sub-acute/chronic form of the disease manifested as ‘b’ kind of pattern.

Head and neck manifestations of sarcoidosis are reported in 10% to 15% of patients.[4] The first documented case of oral sarcoidosis was published by Poe et al in 1943.[14] Oral sarcoid involvement remains uncommon, with the buccal mucosa, lips, gingivae, tongue, and palate being the most frequently

affected sites.[15] Oral sarcoid lesions have been described as multiple, nodular, painless, and poorly demarcated ulcerations of the gingiva, buccal mucosa, labial mucosa, and palate.[7,16] Suresh et al reviewed 68 cases of oral sarcoidosis, of which only 10 demonstrated gingival involvement.[9] The clinical features of gingival sarcoidosis include gingival hyperplasia, gingival recession, mucosal swelling, and ulceration, with persistent swelling of the affected gingiva representing the most common presentation.[17]

In the present case, diffuse enlargement of the anterior gingiva was accompanied by swelling of the lips. Comparable cases have been reported in the literature.[9,10,17–20] However, these cases either involved older patients or were documented in individuals already diagnosed with sarcoidosis and concurrent multisystem involvement. The incidental discovery of sarcoid involvement confined to the gingiva in an adolescent is a rarely reported phenomenon. A similar case was reported by Sharma et al in an 11year old girl of Dravidian origin.[7] Watts et al also presented an isolated gingival sarcoid lesion but the patient was a middle aged 42-year female.[8]

The precise etiology of sarcoidosis remains elusive, although from a pathobiological perspective, the disease is believed to arise from exposure to unidentified antigenic triggers in genetically susceptible individuals.[4] A characteristic "immune paradox" has been described, wherein peripheral anergy (reduced delayed-type hypersensitivity to common antigens) coexists paradoxically with an exaggerated inflammatory response in the affected organs. Epidemiological investigations have suggested that a common genetic or environmental determinant may play a contributory role in cases of familial sarcoidosis.[4] Granuloma formation is understood as a pathological response to an antigenic agent that is insoluble or poorly degradable, whereby the granuloma serves as a physical barrier that sequesters the triggering substance and protects the surrounding tissues from injury.[4] The initiation and evolution of sarcoid granulomas depend on both innate and adaptive immune pathways.[4] Furthermore, research indicates that even inhaled materials — such as silicone or inorganic dust— may trigger an autoimmune response accompanied by the onset of local and systemic symptoms of sarcoidosis. In susceptible patients, giant cell granulomas containing exogenous material have been identified.[21] In the present case, the patient was found to be allergic to inorganic dust which could have triggered the autoimmune response to sarcoidosis.

Sarcoidosis is fundamentally a diagnosis of exclusion, mimicking several other granulomatous conditions including Crohn's disease, orofacial granulomatosis, Wegener's granulomatosis, mycobacterial infections, and foreign body reactions.[1] Owing to the lack of definitive diagnostic criteria, establishing the diagnosis remains a considerable challenge. Rather, the diagnosis rests upon the histologic identification of characteristic non-caseating epithelioid granulomas, in conjunction with clinical manifestations that vary according to the organ involved and may include fatigue, fever, weight loss, erythema nodosum, arthralgia, and dyspnea.[3,4] There exists no single laboratory investigation or clinicopathological feature that is pathognomonic for sarcoidosis; however, elevated serum angiotensin-converting enzyme (sACE) levels have been documented in active untreated disease.[22] The erythrocyte

sedimentation rate may be elevated, and disturbances in calcium metabolism may lead to hypercalcemia, hypercalciuria, and decreased bone density. An increased T4:T8 lymphocyte ratio is observed, and hyperglobulinemia is a frequent finding, reflecting an altered immune state with notable elevation of IgG.[23] In the present case, the diagnosis of gingival sarcoidosis was established based on histopathological findings from gingival biopsy, supported by elevated sACE levels, raised ESR, altered T4:T8 cell ratios, and hypergammaglobulinemia with elevated IgG, IgA and IgE. Increased IgE is usually seen in patients with sarcoidosis co-existing with atopic conditions like allergic rhinitis. The patient in the present case gave a medical history of allergic rhinitis.

Systemic evaluation did not reveal sarcoid involvement beyond the oral cavity, suggesting that the gingival and lip involvement might represent the initial and sole clinical manifestation of the disease. The biochemical abnormalities noted prior to treatment returned to within normal limits upon re-evaluation three months post-surgery. Nonetheless, the possibility of generalized sarcoidosis with multiorgan involvement in subsequent years cannot be entirely discounted.[8]

The literature offers limited guidance regarding the management of oral sarcoidosis, with therapeutic approaches ranging from observation to surgical excision. As the lesion in this case was confined to the gingiva, surgical excision was performed without the need for systemic pharmacotherapy. Management of the lip swelling was similarly limited to intralesional corticosteroid therapy, to which the patient responded favourably. However, given the genetic underpinnings of the disease and its associated morbidity, extended long-term follow-up remains essential. The patient was monitored regularly for 24 months, with no recurrence of gingival enlargement or lip swelling. No new complaints pertaining to general or oral health emerged, and the overall well-being of the child remained satisfactory.

Conclusion

Although oral sarcoidosis remains an uncommon presentation, it is essential to recognize that gingival findings may offer valuable diagnostic insights into a serious underlying medical condition. Healthcare providers across disciplines, including dentists and clinicians of various specialties, should endeavour to broaden their specialist knowledge, as early diagnosis represents a critical step in preventing and delivering appropriate, comprehensive care to children at risk of abominable sequelae and the potential progression of systemic, life-threatening **sarcoid-related disease.**

Abbreviations

ESR
erythrocyte sedimentation rate
sACE
serum angiotensin-converting enzyme
HRCT
high-resolution computed tomography

ECG
electrocardiography
MRI
magnetic resonance imaging

Declarations

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Informed consent to participate: Written informed consent for the publication of clinical details and clinical images was obtained from the parent/guardian of the patient.

Consent for publication: informed consent for publication of clinical details and/or clinical images was obtained from the parents of the patient

Data availability: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Author contribution: SM: Conceptualization of the case report, clinical evaluation and management, drafting the manuscript, data analysis, manuscript review and editing KP: literature search, acquisition and interpretation of data, manuscript review and editing SJS: acquisition and interpretation of data, supervision, manuscript drafting, review and editing SVS: literature search, manuscript drafting, review and editing. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Clinical relevance: Oral sarcoidosis remains an uncommon presentation; it is essential to recognize that gingival findings may offer valuable diagnostic insights into a serious underlying medical condition. Healthcare providers across disciplines, including dentists and clinicians of various specialties, should endeavour to broaden their specialist knowledge, as early diagnosis represents a critical step in preventing and delivering appropriate, comprehensive care to children at risk of abominable sequelae and the potential progression of systemic, life-threatening sarcoid-related disease.

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Figures



Figure 1

a. Pre operative diffuse gingival enlargement (frontal view) b. Pre operative diffuse gingival enlargement (left lateral view) c. Pre operative diffuse gingival enlargement (right lateral view) d. Orthopantamograph of the patient e. External bevel gingivectomy done f. 2 year follow up (maxillary) g. 2 year follow up (mandibular)

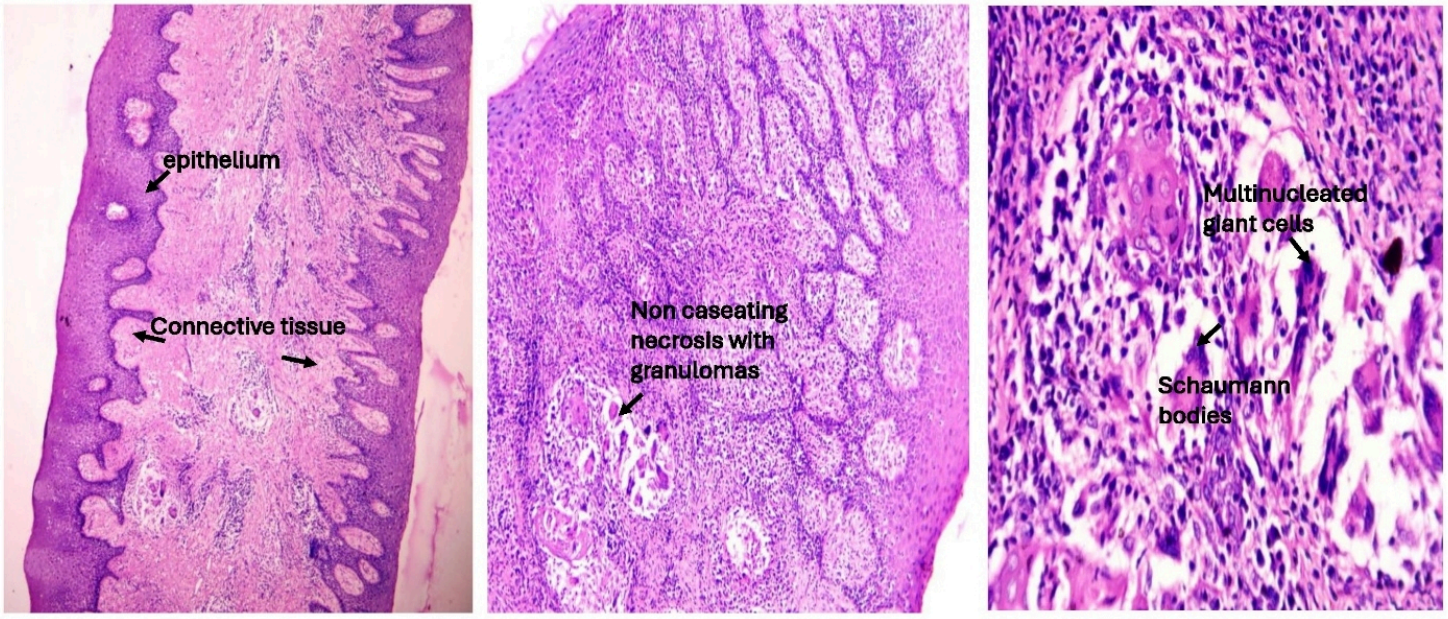


Figure 2

H-E staining of the excised tissue viewed at 5x, 10x and 40 x magnification showing a. epithelium and connective tissue b. non- caseating necrosis with granulomas c. granulomatous nest of epithelioid histiocytes, multinucleated Langerhans giant cells and Schaumann bodies

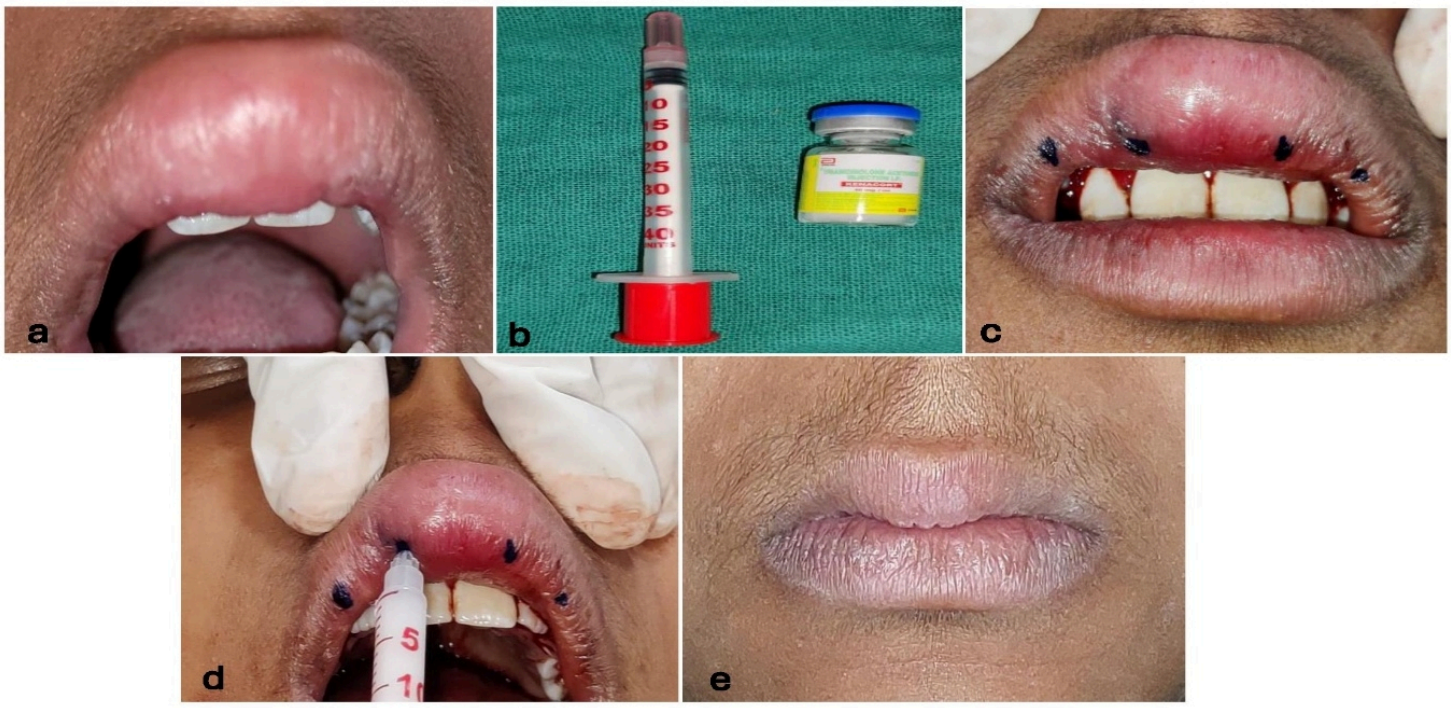


Figure 3

a. Upper lip swelling; b. Triamcinolone injection; c. Piercing points made; d. Application of injection; e. 2 year follow up