Volume 11, Issue 3, March – 2026 International Journal of Innovative Science and Research Technology
ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

Tuberous Sclerosis Complex Presenting with Oral Lesions: A Periodontist's Perspective

Dr. Aastha Gajavalli[1*]; Dr. Suchetha Aghanashini[2]; Dr. Sapna Nadiger[3]; Dr. Apoorva S. K.[4]; Dr. Shreekrishna K.[5]

[1]Post Graduate, Department of Periodontics D. A. P. M. R. V. Dental College Bangalore
[2]MDS, Professor, Department of Periodontics, D. A. P. M. R. V. Dental College Bangalore
[3]MDS, Reader, Department of Periodontics, D. A. P. M. R. V. Dental College Bangalore
[4]Post Graduate, Department of Oral Medicine and Radiology, D. A. P. M. R. V. Dental College Bangalore
[5]Post Graduate, Department of Conservative Dentistry and Endodontics, D. A. P. M. R. V. Dental College Bangalore

Corresponding Author: Dr. Aastha Gajavalli[1*]

Publication Date: 2026/03/30

Abstract:

- ***Aim***

To describe a rare presentation of gingival angiofibroma in a patient with tuberous sclerosis complex, highlight the diagnostic relevance of oral manifestations in TSC, and demonstrate the clinical and histopathological outcomes following conventional gingivectomy in managing associated gingival overgrowth.

- ***Background***

Tuberous sclerosis complex (TSC) is a neurocutaneous autosomal dominant genetic disorder characterized by mutations in the TSC1 and TSC2 genes which leads to dysregulation of the mechanistic target of rapamycin (mTOR) pathway resulting in cellular hyperplasia and the formation of benign tumors in various organs. Oral manifestations, although less frequently emphasized, may serve as important diagnostic indicators.

- ***Case Description***

This report presents the case of a 33-year-old female with TSC who exhibited prominent gingival overgrowth in the maxillary anterior region, contributing to aesthetic and functional concerns. Conventional gingivectomy using a scalpel was performed to excise the gingival enlargements in maxillary anterior region to re-establish normal contour. The postoperative period was uneventful, and significant improvement in gingival architecture and facial appearance was observed. Microscopic examination revealed parakeratinized stratified squamous epithelium with short rete ridges and numerous pigmented melanocytes. These features were consistent with angiofibroma of the maxillary anterior gingiva, correlating with the clinical presentation.

- ***Conclusion***

Management of TSC requires a multidisciplinary approach, given its varied systemic and oral manifestations. Early diagnosis, seizure control with appropriate antiepileptics, and timely intervention for oral lesions are critical in improving long-term outcomes.

- ***Clinical Significance***

This case emphasizes that oral angiofibromas and gingival enlargements can serve as early, easily overlooked markers of tuberous sclerosis. Recognizing these characteristic oral lesions helps differentiate them from other gingival overgrowths and supports timely diagnosis, appropriate management, and multidisciplinary care.

Keywords: *Tuberous Sclerosis, Seizure, Gingival Enlargement, Gingivectomy, Genetic Condition.*

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

How to Cite: Dr. Aastha Gajavalli; Dr. Suchetha Aghanashini; Dr. Sapna Nadiger; Dr. Apoorva S. K.; Dr. Shreekrishna K. (2026) Tuberous Sclerosis Complex Presenting with Oral Lesions: A Periodontist's Perspective. *International Journal of Innovative Science and Research Technology,* 11(3), 2489-2494. https://doi.org/10.38124/ijisrt/26mar1240

I. INTRODUCTION

Tuberous sclerosis complex (TSC) is a rare autosomal dominant genetic condition that affects several organ systems- the brain, kidney, heart and skin. It is caused by pathogenic mutations in the TSC1 gene (9p34) or TSC2 gene (16p13), which encode the proteins hamartin and tuberin. [1]These proteins interact within the mTOR-signalling pathway, which is the key regulator of central nervous and dermatologic systems. Abnormalities in these processes contribute to the epilepsy and neurocognitive issues found in TSC. The hyperactivity of mTOR also inhibits melanogenesis, leading to hypopigmented areas.[2,3]

Tuberous sclerosis complex (TSC) leads to tumor development in multiple organs, which leads to the wide variability of disease severity among individuals.

Seizures, intellectual disability, and facial angiofibroma make up the traditional clinical triad (also known as the Vogt triad), but less than 50% of patients have all three of these conditions. Gingival hyperplasia, and intra-oral fibroma on gingiva, buccal mucosa, tongue and lip are the most common and primary oral manifestations in patients suffering from TSC.

Other secondary oral manifestations that were frequently found were intra-oral hypopigmented macule, enamel hypoplasia of teeth, dental anomalies like enamel pearl, pitting of teeth, odontogenic tumors like desmoplastic fibromas, odontogenic myxoma in the jaws, hemangioma of the tongue.

➢ *Clinical Features*

- *Skin and Nail Findings:*

Dermatologic involvement is common and includes several characteristic lesions. Although these rarely cause serious medical issues, they may have cosmetic implications. Typical skin findings include facial angiofibromas (seen in about 75% of cases), shagreen patches (over 50%), ungual fibromas (20%–80%), hypopigmented macules (around 90%), and fibrous cephalic plaques (about 25%).

- *Eye Involvement:*

Retinal astrocytic hamartomas appear in 40%–50% of patients, particularly in those with genetic mutations.

- *Neurological Manifestations:*

Brain lesions are a major source of symptoms. Seizures affect nearly 80%–90% of individuals with TSC. Cortical, subcortical, and subependymal tubers are common, and several studies show that a higher tuber count correlates with greater cognitive impairment and seizure burden. Subependymal nodules occur in 80%–90% of patients, while 10%–20% develop subependymal giant cell astrocytomas (SEGAs). Neurodevelopmental delays and behavioral challenges are seen in up to 90% of affected patients.

- *Renal Manifestations:*

Kidney involvement includes angiomyolipomas and cysts, which may affect function and lead to pain, bleeding, or renal failure. Angiomyolipomas occur in up to 75% of patients. More severe but less frequent complications include polycystic kidney disease (around 5%) and renal cell carcinoma (about 3%). Because the TSC2 and PKD1 genes lie adjacent to each other, a mutation in TSC2 may extend into PKD1, resulting in a contiguous gene syndrome with more aggressive kidney disease.

- *Cardiac Findings:*

Cardiac rhabdomyomas are extremely common in foetuses with TSC. Although these tumors usually regress during early childhood, they can occasionally cause neonatal death. They may also recur during adolescence, especially in females. Approximately 20% of adults with TSC have rhabdomyomas, though most remain asymptomatic.

- *Pulmonary Involvement:*

Lung involvement may include cysts and lymphangioleiomyomatosis (LAM), the latter being a major cause of mortality in patients who develop it. About 30% of individuals with TSC develop LAM, with diagnosis typically around age 35. It occurs almost exclusively in women of reproductive age and worsens with elevated estrogen levels, such as during pregnancy.

- *Liver Manifestations:*

Hepatic lesions such as angiomyolipomas and cysts occur in roughly 30% of patients. These growths may enlarge over time, but most of them are asymptomatic.[4-10]

➢ *Oral Manifestations*

Oral fibromas are the second most common manifestation of tuberous sclerosis. They are localized most often on the maxillary anterior gingiva but can be observed on the cheeks, lips, edge of the lip, tongue, or palate[11-15]. According to the studies, their prevalence is 11%–69% and average diameter is 5 mm.[16] These fibromas can achieve gingival growth, which can be confused with a drug-related etiology when anticonvulsant therapy, particularly phenytoin, is concomitantly prescribed. [17-20]According to Curi *et al*., the differential diagnosis is based on the purely gingival involvement in cases of increased drug dose without lesions affecting any other mucous membranes contrary to the tuberous sclerosis. Removal of these fibromas is indicated in case of an increase in size or aesthetic or functional discomfort with associated bleeding. [21]

II. CASE DESCRIPTION

A 33-year-old female patient reported to the Department of Periodontics of D A P M R V Dental college, Bengaluru

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

with a chief complaint of enlarged gums in upper left front teeth region which led to compromised aesthetics. Patient presented with medical history of tuberous sclerosis complex (TSC) associated with epilepsy since the age of 10 years. Patient has also been diagnosed with ADHD by NIMHANS (National institute of mental health and neurosciences) for 20 years. She is currently residing under the care of a charitable trust. She had been referred for further evaluation on academic grounds. As part of her ongoing management, the patient was advised to undergo MRI of the brain (with and without contrast), EEG, and an ultrasound of the abdomen and pelvis to evaluate the neurological and systemic manifestations commonly associated with tuberous sclerosis complex. The EEG performed as part of this workup revealed normal background activity and normal sleep elements, but it also showed rare interictal epileptiform discharges predominantly originating from the right frontal and left temporal regions. These findings were consistent with her known history of epilepsy secondary to TSC, supporting the need for continued neurological monitoring and correlation with neuroimaging studies to assess for cortical or subependymal lesions that may contribute to seizure activity. She was prescribed levetiracetam (Tab. Levipil 500 mg) as part of her seizure management plan.

On extraoral examination, multiple facial angiofibromas, adenoma sebaceum (acne like lesions) along with ash leaf patch was seen on the cheek.

On intra-oral examination, the buccal mucosa showed scattered small melanotic macules which were well circumscribed, smooth without ulceration and non-tender. No discharge, or surface changes were evident.

Localized plaque and calculus deposits were noted, particularly in the maxillary anterior region, contributing to marginal gingival inflammation.

Well-defined, localized, exophytic gingival swelling on the maxillary anterior labial gingiva, extending from approximately the region of the right maxillary central incisor to the canine was observed. The lesion appeared as multiple nodular to lobulated masses, with a smooth, shiny surface. The color was predominantly dark purple to bluish, suggestive of a vascular or pigmented soft-tissue lesion. The nodules appeared pedunculated and varied slightly in size. Surrounding gingiva showed signs of inflammation, with diffuse hyperpigmentation of the upper and lower gingiva.

Overall, the intraoral findings were suggestive of multiple gingival fibromas with generalized mucosal pigmentation in a patient with tuberous sclerosis.

Additional findings were dental caries noted wrt 16(maxillary right 1st molar), root stumps and grossly decayed tooth wrt 26,36 and 46 (maxillary left 1st molar, mandibular left and right 1st molar respectively). Patient also presented with a pronounced deep bite, reduced lower facial height, and traumatic occlusion of the maxillary incisors against the mandibular mucosa.

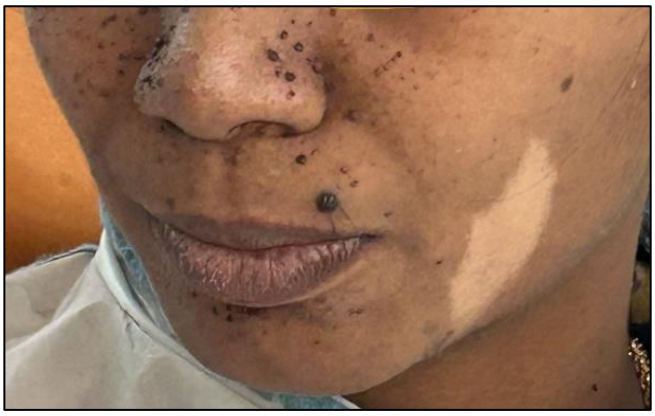

Fig 1 Facial Image Showing Multiple Dark Papules Over the Nose and Cheeks Along with a Well-Defined Hypopigmented Patch on the Right Cheek, Features Suggestive of Cutaneous Manifestations of Tuberous Sclerosis

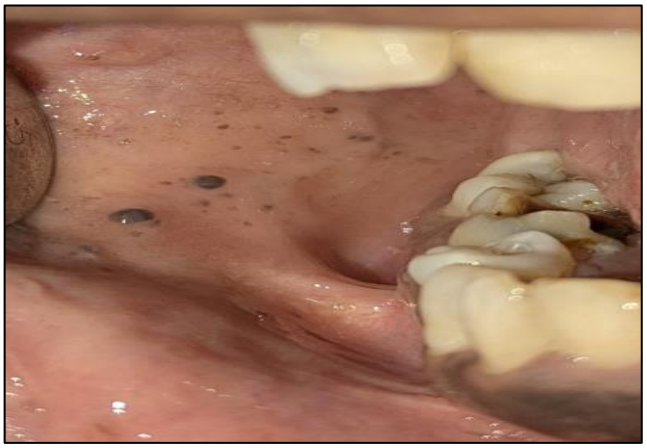

Fig 2 Intraoperative Image of Buccal Mucosa

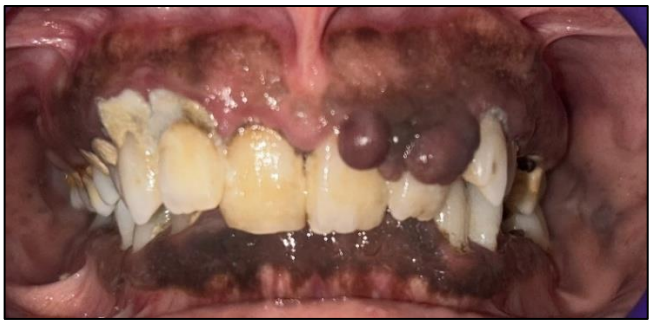

Fig 3 Pre-Operative View of Teeth Before Ultrasonic Scaling

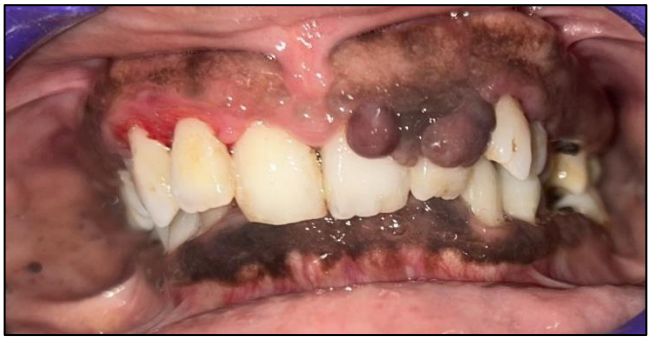

Fig 4 Post Operative View of Teeth After Ultrasonic Scaling

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

➢ *Treatment*

The patient first underwent Phase I periodontal therapy, which included thorough scaling and root planing to remove heavy calculus deposits and reduce local inflammatory burden. Detailed oral hygiene instructions were provided, emphasizing effective plaque control measures to minimize further irritation of the gingiva. The patient was re-evaluated following initial therapy, and a reduction in generalized inflammation was noted, although the localized gingival overgrowths in the maxillary anterior region persisted.

Subsequently, Phase II periodontal therapy was initiated. Under local anesthesia, a surgical excision of the gingival enlargements was performed. Gingivectomy procedure was carried out on the labial aspects of the maxillary anterior teeth to remove the nodular, hyperplastic tissue and re-establish normal gingival contours. Hemostasis was achieved, and the excised tissue was sent for histopathological examination to confirm the clinical diagnosis and rule out any neoplastic or vascular pathologies. The patient was recalled after one week for post-operative evaluation. Healing was satisfactory, and no signs of infection or recurrence were observed.

Post-surgical instructions were reinforced, and the patient was advised to maintain meticulous oral hygiene, including proper brushing techniques and the use of adjuncts such as chlorhexidine mouthrinse if indicated. Regular follow-up appointments were scheduled to monitor healing and ensure stability of the periodontal tissues.

Given the presence of additional dental concerns, the patient was referred to relevant dental specialties for comprehensive management:

- Endodontist for root canal therapy of teeth with deep caries and pulpal involvement,
- Orthodontist for correction of malalignment and deep bite, which may contribute to abnormal forces and periodontal stress,
- Oral and maxillofacial surgeon for extraction of root stumps and grossly decayed, non-restorable teeth.

This multidisciplinary approach ensured complete rehabilitation of the patient's oral health, addressing both periodontal and non-periodontal issues to achieve long-term functional and esthetic outcomes.

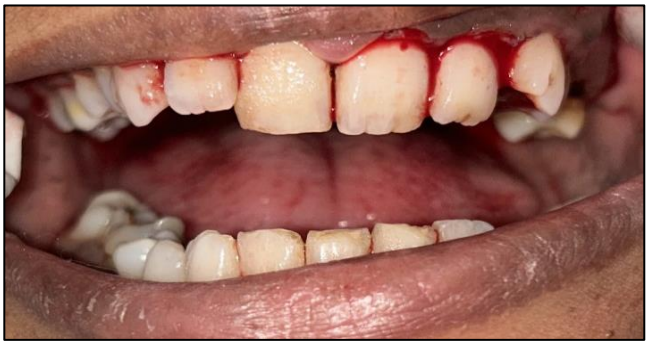

Fig 5 Immediate Post-Operative View of Teeth After Gingivectomy

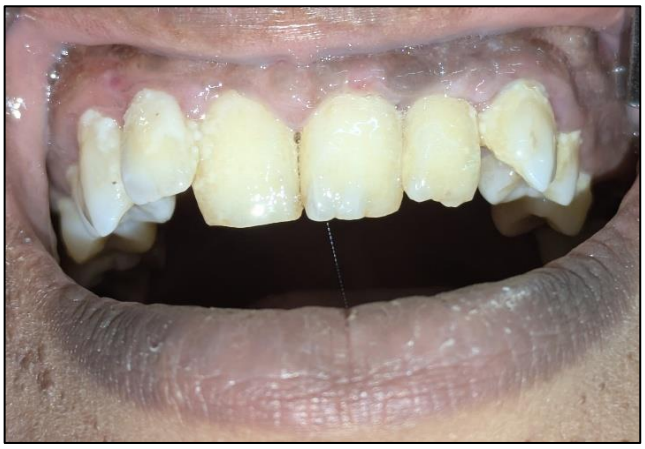

Fig 6 1week Postoperative View of Teeth

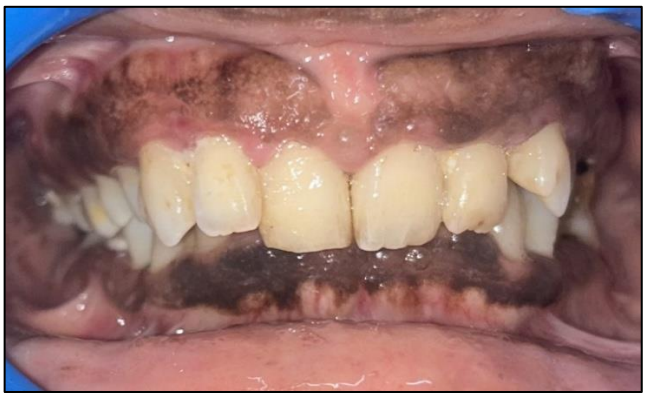

Fig 7 3 Months Follow up Post-Operative View of Teeth

➢ *Differential Diagnosis*

In this patient, the differential diagnosis was considered based on the clinical presentation of multiple gingival enlargements and mucocutaneous lesions. Conditions such as neurofibromatosis type 1 (NF1) may appear similar and were therefore included in the initial differential list. However, NF1 and tuberous sclerosis complex (TSC), though both neurocutaneous disorders with autosomal dominant inheritance, differ significantly in their systemic involvement. Unlike TSC, NF1 typically does not present with seizures or neurocognitive impairment, both of which are commonly seen in patients with TSC, including the present case.

Drug-induced gingival enlargement was considered, particularly due to the patient's ongoing levetiracetam therapy; however, the localized nodular morphology favored a reactive lesion.

➢ *Histopathological Findings*

The gingival tissue excised from different areas during surgery was fixed in 10% buffered formaldehyde solution soon after excision and sent for histopathologic examination. It was processed and embedded in wax. Multiple sections were prepared, stained with hematoxylin and eosin, and viewed under a microscope.

The haematoxylin and eosin-stained tissue sections revealed a prominent vascular network comprised of numerous dilated thin-walled blood vessels of variable size. Few endothelial cells lining the vessels appeared to be plump

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

and immature. The remaining stroma was composed of fibroblasts and unorganized delicate and coarse collagenic fibres along with sprinkled chronic inflammatory cells. The overlying epithelium was parakeratinized stratified squamous type with short rete pegs and abundant pigmented melanocytes. Foci of melanin incontinence were observed in the connective tissue.

Features were suggestive of angiofibroma of the maxillary anterior gingiva, on clinical correlation.

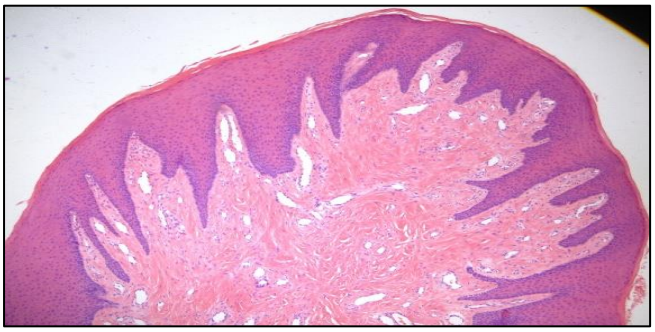

Fig 8 Histopathological Section of Excised Tissue

➢ *Final Diagnosis*

Considering the clinical presentation of multiple gingival enlargements along with mucocutaneous lesions, and correlating these findings with the patient's history of seizures, neurocognitive involvement along with histological findings, the final diagnosis is oral angiofibromas associated with Tuberous Sclerosis Complex (TSC).

III. DISCUSSION

Tuberous sclerosis complex (TSC) is a rare genetic condition with an estimated prevalence of 1 in 6,000–10,000 live births, characterized by hamartomatous growths in multiple organs. Diagnosis is primarily clinical, supported by neuroimaging and, when available, genetic testing. Because the manifestations vary widely, continuous and individualized surveillance involving MRI, EEG, renal imaging, and dermatologic assessment is recommended throughout life.

Oral lesions are an important but frequently overlooked component of TSC. Gingival fibromas are the second most common oral finding and may present as localized, nodular overgrowths predominantly on the anterior maxillary gingiva. In the present case, the patient demonstrated multiple gingival enlargements accompanied by generalized mucosal pigmentation — features consistent with oral involvement of TSC. Histopathology confirmed the presence of an angiofibroma, a finding well aligned with documented oral manifestations of the disease.

An important clinical consideration in this patient was her ongoing use of levetiracetam for seizure control. Although classic anticonvulsants such as phenytoin are well-known causes of drug-induced gingival enlargement, emerging evidence indicates that levetiracetam may also contribute to gingival overgrowth. James et al. (2022) reported that gingival hyperplasia, though rare with newer antiepileptics, can occur with levetiracetam and may resolve upon discontinuation, emphasizing the importance of dental monitoring in such patients. While the localized nodular morphology in the present case was more consistent with TSC-associated fibromas than generalized drug-related enlargement, the patient's levetiracetam therapy may have acted as a secondary exacerbating factor. This underscores the need for clinicians to evaluate all potential etiologies when assessing gingival hyperplasia in medically compromised individuals. Moreover, the absence of recurrence on follow-up further reduces the likelihood of a drug-induced etiology, supporting a diagnosis more consistent with the underlying syndrome. This underscores the importance of carefully differentiating syndromic lesions from medication-related changes when evaluating gingival overgrowth in medically compromised individuals.

The management of TSC requires a coordinated, multidisciplinary approach. In the present case, periodontal therapy successfully addressed the gingival enlargements and improved the patient's oral hygiene. Additional referrals were essential due to the presence of dental caries, traumatic occlusion, malalignment, and deep bite — conditions that could further contribute to periodontal stress and functional discomfort. Collaboration between periodontists, endodontists, orthodontists, oral surgeons, neurologists, dermatologists, and radiologists is vital for optimizing outcomes in individuals with TSC.

Continued research into mTOR inhibitors and other targeted therapies holds promise for improving control of both neurological manifestations and dermatologic lesions. Early recognition, long-term follow-up, and interprofessional care remain fundamental to enhancing quality of life in patients with TSC.[22-25]

IV. CONCLUSION

This case highlights the importance of recognizing oral manifestations as significant clinical features of tuberous sclerosis complex. Gingival overgrowths, particularly in the anterior maxillary region, may be among the earliest or most noticeable signs and can impact both function and esthetics. Histopathological confirmation is essential to differentiate TSC-associated fibromas from other reactive or drug-induced enlargements. Although rare, the potential contribution of levetiracetam to gingival enlargement further underscores the need for careful evaluation of all possible etiologic factors in medically compromised patients. Early identification, appropriate periodontal intervention, and coordinated multidisciplinary management are crucial for achieving favorable outcomes and improving overall quality of life in individuals with TSC.

CLINICAL SIGNIFICANCE

This case highlights the importance of recognizing oral angiofibromas and gingival enlargements as potential mucocutaneous manifestations of tuberous sclerosis complex (TSC). Because oral lesions may be subtle, recurrent, or

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26mar1240

mimic other benign growths, they can easily be overlooked or misdiagnosed as inflammatory or drug-induced enlargements. Early identification of these oral findings can support timely diagnosis or reinforcement of a suspected diagnosis of TSC, especially in patients with neurological features such as seizures. Furthermore, awareness of the recurrence tendency and the characteristic histopathological features of oral angiofibromas helps clinicians to differentiate them from other gingival overgrowths and guide appropriate management and long-term follow-up. This case therefore underscores the role of dental professionals in multidisciplinary care and early detection of systemic conditions such as TSC.

REFERENCES

[1]. Gomes I, Jesus Ribeiro J, Palavra F. Monitoring and Managing Patients with Tuberous Sclerosis Complex: Current State of Knowledge. J Multidiscip Healthc. 2022 Jul 14; 15:1469-1480.

[2]. Gruber V, Scholl T, Samueli S, Gröppel G, Mühlebner A, Hainfellner JA, Feucht M. Pathophysiology of neurodevelopmental mTOR pathway-associated epileptic conditions: Current status of biomedical research. Clin Neuropathol. 2019 Sep/Oct;38(5):210-224.

[3]. Luo C, Ye WR, Shi W, Yin P, Chen C, He YB, Chen MF, Zu XB, Cai Y. Perfect match: mTOR inhibitors and tuberous sclerosis complex. Orphanet J Rare Dis. 2022 Mar 04;17(1):106.

[4]. Northrup H, Aronow ME, Bebin EM, Bissler J, Darling TN, de Vries PJ, Frost MD, Fuchs Z, Gosnell ES, Gupta N, Jansen AC, Jóźwiak S, Kingswood JC, Knilans TK, McCormack FX, Pounders A, Roberds SL, Rodriguez-Buritica DF, Roth J, Sampson JR, Sparagana S, Thiele EA, Weiner HL, Wheless JW, Towbin AJ, Krueger DA., International Tuberous Sclerosis Complex Consensus Group. Updated International Tuberous Sclerosis Complex Diagnostic Criteria and Surveillance and Management Recommendations. Pediatr Neurol. 2021 Oct; 123:50-66.

[5]. Cascarino M, Leclerc-Mercier S. Histological Patterns of Skin Lesions in Tuberous Sclerosis Complex: A Panorama. Dermatopathology (Basel). 2021 Jul 04;8(3):236-252.

[6]. Kassiri J, Snyder TJ, Bhargava R, Wheatley BM, Sinclair DB. Cortical tubers, cognition, and epilepsy in tuberous sclerosis. Pediatr Neurol. 2011 May;44(5):328-32

[7]. Goodman M, Lamm SH, Engel A, Shepherd CW, Houser OW, Gomez MR. Cortical tuber count: a biomarker indicating neurologic severity of tuberous sclerosis complex. J Child Neurol. 1997 Feb;12(2):85-90.

[8]. Rebaine Y, Nasser M, Girerd B, Leroux C, Cottin V. Tuberous sclerosis complex for the pulmonologist. Eur Respir Rev. 2021 Sep 30;30(161)

[9]. Gupta N, Henske EP. Pulmonary manifestations in tuberous sclerosis complex. Am J Med Genet C Semin Med Genet. 2018 Sep;178(3):326-337.

[10]. Uysal SP, Şahin M. Tuberous sclerosis: a review of the past, present, and future. Turk J Med Sci. 2020 Nov 03;50(SI-2):1665-1676.

[11]. López E, Escovich L, Vigna A. Tuberous sclerosis: presentation of a clinical case with oral manifestations. Med Oral 2003; 8:122–128.

[12]. Araújo Lde J, Muniz GB, Santos E, Ladeia JP, Martelli H Jr, Bonan PR. Tuberous sclerosis complex diagnosed from oral lesions. Sao Paulo Med J 2013; 131:351–355.

[13]. Damm DD, Tomich CE, White DK, Drummond JF. Intraosseous fibrous lesions of the jawsA manifestation of tuberous sclerosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 87:334–340.

[14]. Harutunian K, Figueiredo R, Gay-Escoda C. Tuberous sclerosis complex with oral manifestations: a case report and literature review. Med Oral Pathol Oral Cir Bucal 2011;16: e478–e481.

[15]. Scully C. Oral mucosal lesions in association with epilepsy and cutaneous lesions: the Pringle-Bourneville syndrome. Int J Oral Surg 1981; 10:68–72.

[16]. Nico MM, Hammerschmidt M, Lourenço SV. Oral mucosal manifestations in some genodermatoses: correlation with cutaneous lesions. Eur J Dermatol 2013; 23:581–591

[17]. Barron RP, Kainulainen VT, Forrest CR, Krafchik B, Mock D, Sandor GK. Tuberous sclerosis: clinicopathologic features and review of the literature. J Craniomaxillofac Surg 2002; 30:361–366.

[18]. López-López J, Rodriguez-de-Rivera-Campilo E, Marques-Soares MS, Finestres-Zubeldia F, Chimenos-Küstner E, Rosello-Llabres X. Tuberous sclerosis and its oral manifestations. A clinical case. Med Oral 2004; 9:216–223.

[19]. Sparling JD, Hong CH, Brahim JS, Moss J, Darling TN. Oral findings in 58 adults with tuberous sclerosis complex. J Am Acad Dermatol 2007; 56:786–790.

[20]. Lygidakis NA, Lindenbaum RH. Oral fibromatosis in tuberous sclerosis. Oral Surg Oral Med Oral Pathol 1989; 68:725–728.

[21]. Curi MM, Cardoso CL, Ikuta CRS, Koga DH, Zardetto C. Tuberous Sclerosis: A case report with oral manifestation. Int J Odontostomatol 2014; 8:185–189.

[22]. De Waele L, Lagae L, Mekahli D. Tuberous sclerosis complex: the past and the future. Pediatr Nephrol. 2015 Oct;30(10):1771-80.

[23]. Crino PB, Nathanson KL, Henske EP. The tuberous sclerosis complex. N Engl J Med. 2006 Sep 28;355(13):1345-56.

[24]. Ng KH, Ng SM, Parker A. Annual review of children with tuberous sclerosis. Arch Dis Child Educ Pract Ed. 2015 Jun;100(3):114-21.

[25]. James, J; Jose, J; Gafoor, VA. Levetiracetam-induced gingival hyperplasia. Journal of Postgraduate Medicine 68(3): p 168-169, Jul–Sep 2022.