Diagnosis and Management: Surgical excision was performed. Macroscopically, the lesion consisted of a well-circumscribed, lobulated, submucosal mass measuring 2.3 cm in diameter. Histopathologic examination revealed a well-circumscribed, but nonencapsulated neoplasm consisting of slightly polymorphous cells that exhibited small, round to ovoid nuclei and eosinophilic cytoplasm set in prominent myxoid stroma containing pseudocystic spaces. Lesional cells were arranged in broad lobules separated by slender fibrous septae (Figures 3A and 3B). The overlying mucosa was unremarkable. Immunohistochemical analysis showed diffuse, strong positivity of lesional cells to vimentin and S100, with more focal, strong expression of glial fibrillary acidic protein (GFAP) (Figures 3C and 3D). Pancytokeratin, p63, smooth muscle actin, CD31, and CD34 were negative, with appropriate reactivity in control tissues. Given the histomorphology and immunohistochemical profile, a diagnosis of ectomesenchymal chondromyxoid tumor was rendered.

Discussion: ECT is a relatively rare neoplasm initially described by Smith et al. in 1995.[9] Two recent extensive reviews of the English language and German language literature confirmed at least 60 reported cases of ECTs, showing a striking predilection for the dorsal tongue, with few extralingual cases reported.[4,10] Age at diagnosis ranges from 7 to 78 years, with no sex predilection. Lesions generally present as a slow-growing, painless mass with minimal chance for recurrence after surgical excision.

Histopathologic features of ECT characteristically show demarcated, lobular sheets of fairly uniform round to ovoid, polygonal, or spindle-shaped cells with small, uniform nuclei. Although mild nuclear atypia can be observed, mitotic figures are rarely noted. Cytoplasmic boundaries may be indistinct, with variable proportions of myxoid to chondroid stroma admixed with cellular zones.[9]

While the origin of ECTs is uncertain, this neoplasm is thought to derive from pluripotent ectomesenchymal cells from the neural crest.[9] This impression is corroborated by tumor expression of GFAP. To further substantiate the neural crest origin, cells derived from ECTs have been shown to be similar to neuronal cells in cell cultures, with expression of homeobox protein-transcription factor (Nanog), GFAP, and microtubule-associated protein 2 (MAP2), and with mRNA real-time polymerase chain reaction analysis demonstrating positivity for octamer binding protein 3/4, transcription factor Sox2, Nanog, MAP2, and CD105.[5]

The *EWSR1* gene rearrangement has been described in a subset of ECTs.[11] This rearrangement has also been reported in soft tissue myoepitheliomas and clear cell hyalinizing tumors of the tongue and, therefore, does not discriminate between these entities.[11,12]

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CLINICAL PATHOLOGY CONFERENCE CASE 2: GINGIVAL OVERGROWTH AROUND A BADLY CARIOUS FIRST MOLAR

Natheer H. Al-Rawi, PhD,[a] Basheer M. Salman, MSc,[b] and Ana Ortega-Pinto, DDS, MSc[c], [a] Associate Professor, Consultant Oral Pathologist, College of Dental Medicine, University of Sharjah, United Arab Emirates, [b] Lecturer, Oral Surgeon, College of Dental Medicine, University of Sharjah, United Arab Emirates, and [c] Associate Professor, School Dentistry, University of Chile, Chile

Clinical Presentation: A 10-year-old girl was referred to the oral surgery clinic with a chief complaint of easily bleeding gingival overgrowth of 3 months' duration in the lower right quadrant (Figure 1). The associated right first molar was badly carious, with grade 2 mobility along with the adjacent premolars. On palpation, the buccal and lingual cortical bones associated with first molar were missing. On radiographic evaluation, a periradicular radiolucency with external root resorption was evident (Figure 2). The patient showed developmental delay and had a history of tonsillectomy and chronic cough. Enlarged upper anterior gingiva and enlarged, palpable bilateral submandibular lymph nodes were also noted during clinical examination (Figures 3 and 4).

Differential Diagnosis: Given the clinical presentation of gingival overgrowth around a badly carious tooth, the differential diagnosis for this case had to include either reactive lesions, such as pyogenic granuloma, peripheral giant cell granuloma, local-

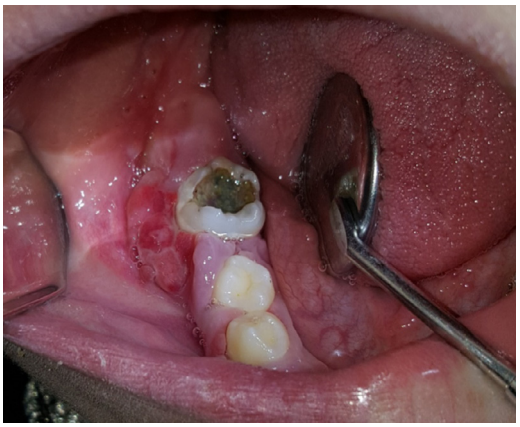

Fig. 1. Easily bleeding gingival overgrowth on buccal surface of tooth #30.

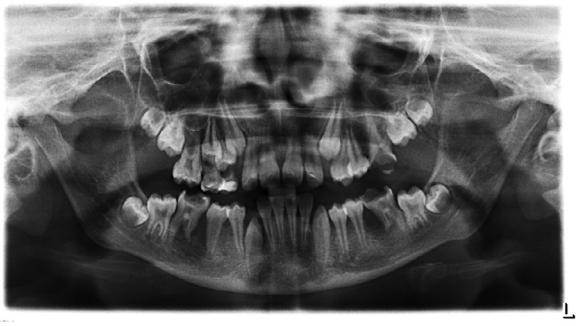

Fig. 2. Periradicular radiolucency with external root resorption evident on the radiograph.

ized spongiotic gingival hyperplasia, and, less likely, manifestations of systemic or generalized diseases when the clinical findings were correlated with radiologic findings.

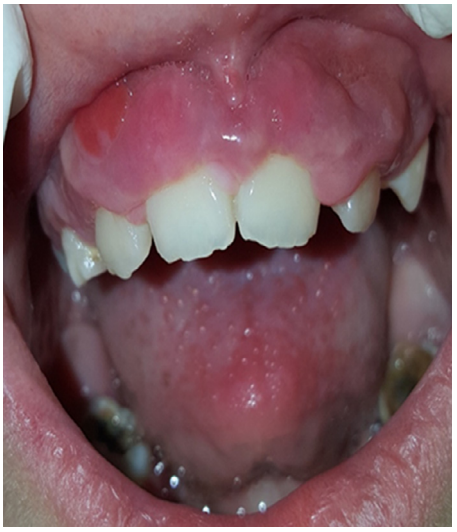

Fig. 3. Enlarged upper anterior gingiva.

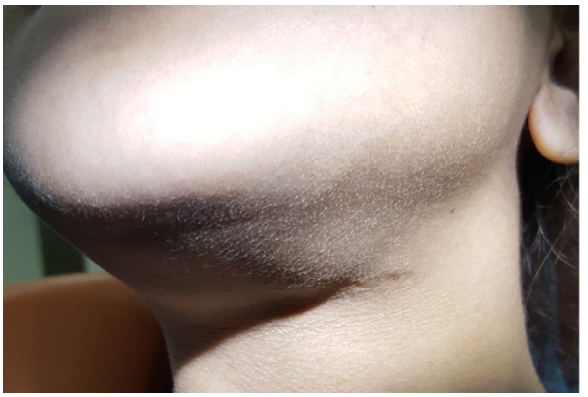

Fig. 4. Enlarged, palpable bilateral submandibular lymph nodes.

The pyogenic granuloma is a common, tumor-like lesion that manifests as plenteous tissue in response to trauma or local irritation. It is a form of granulation tissue and is characterized by proliferating capillaries and fibrocellular connective tissue with dispersed inflammatory cells. The lesion exhibits a lobular, bright red, pedunculated or sessile mass with surface ulceration. The gingiva is the most common site of pyogenic granuloma, particularly around the upper anterior teeth. The lips, dorsum of the tongue, and buccal mucosa could also be affected. Badly carious teeth and poor oral hygiene are the most common etiologic factors of pyogenic granuloma.[1] Pyogenic granuloma can occur at any age, but young adults are most frequently affected. Fibrous maturation could be seen as a sign of healed pyogenic granuloma.

Peripheral giant cell granuloma (PGCG) is another reactive, tumor-like lesion that mainly affects the gingiva and/or alveolar crest. It arises from the cells of the periodontal ligament or the periosteum. PGCG is clinically very similar to pyogenic granuloma. However, the high content of multinucleated giant cells in PGCG make the lesion appear more bluish compared with pyogenic granuloma.[2] Widening of the periodontal ligament space and tooth mobility are other clinical findings associated with PGCG and mainly occur around the mandibular premolars and molars. Resorption of the interdental bone level and alveolar crest region of associated teeth is also frequently seen.[3] The histology of PGCG consists of abundant multinucleated giant cells in highly vascularized fibrocellular stroma with numerous capillaries.[4] Children can demonstrate a rapid growth rate pattern of PGCG, which may reach a large size within several months after initial diagnosis. This soft tissue growth may stimulate bone resorption, which could interfere with tooth eruption and may lead to tooth mobility.[5]

Localized juvenile spongiotic gingival hyperplasia is a recently described benign lesion that affects the gingiva of children and young adults.[6] Clinically localized juvenile spongiotic gingival hyperplasia presents as a pedunculated, papillary gingival overgrowth that bleeds easily and occurs mainly on the anterior maxillary gingiva. The histologic presentation of this lesion is characterized by prominent intercellular edema (spongiosis) and extravascular neutrophilic infiltrate. It represents a gingival overgrowth rather than a pure inflammatory process and shows minimal tissue swelling. The etiology is unknown, and it is not a plaque-associated lesion.[7]

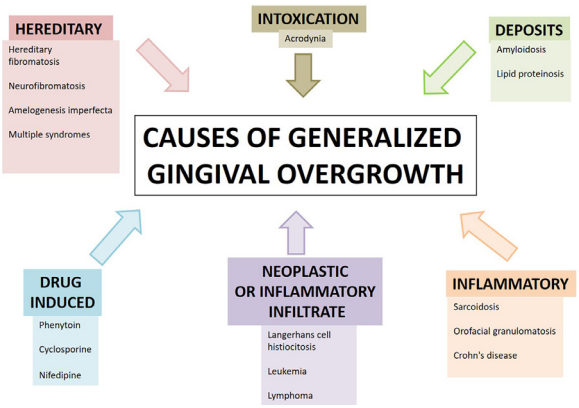

Fig. 5. Causes of generalized gingival overgrowth.

Generalized sources of gingival overgrowth are presented in Figure 5. Rare causes of gingival overgrowth include hereditary conditions, heavy metal toxicity disorders, and deposition disorders, such as amyloidosis and lipoid proteinosis.[8-10] Medication-related gingival hyperplasia may be seen most commonly in conjunction with use of phenytoin, cyclosporine, and nifedipine.[9] Inflammation-related diseases resulting in chronic granulomatous inflammation of oral tissues include sarcoidosis; inflammatory bowel diseases, such as Crohn disease; and orofacial granulomatosis.[8,9] Neoplastic causes of gingival overgrowth include Langerhans cell histiocytosis and leukemia. The former is most frequently seen in young patients and typically presents with tooth mobility, gingival overgrowth and bleeding, and bony destruction resulting in a "teeth floating in air" appearance on radiograph.[11] The latter may present with gingival enlargement and bleeding, oral ulceration, petechiae, and lymphadenopathy.[12]

Diagnosis and Management: After administration of local anesthesia (lidocaine 2% with adrenaline 1:100000), excisional biopsy was performed in the oral surgery clinic at the University Dental Hospital Sharjah (UDHS), and a 1.5 × 1.0 cm specimen of the granulomatous lesion was excised from the buccal side by making an elliptical incision around the lesion. Tooth #30, which was mobile, was then removed by using extraction forceps. The whole area was then curetted of any remaining granulation tissue. There was profuse bleeding of the area during surgery. A gelatin sponge was applied in the extraction socket, and 2 sutures and a pressure pack were applied. The surgical site was rechecked after 30 minutes for hemostasis. The patient was recalled 1 week after the surgery for review of the surgical site and removal of sutures. The surgical site demonstrated good healing by formation of granulation tissue, which filled the socket site. No sign of bleeding, swelling, or trismus was seen.

Histologic examination by hematoxylin and eosin staining revealed noncaseating epithelioid cell granulomas (Figure 6A). Typical Langerhans multinucleated giant cells with Howship lacunae were evident throughout the granuloma (Figure 6B). To rule out an infective cause for the granuloma, Ziehl Neelsen staining for acid-fast bacilli and Gomori methenamine silver and periodic acid−Schiff staining for fungal infection were done and yielded negative results. Chest radiography and serum angiotensin-converting enzyme (S-ACE) were ordered. Bilateral hilar lymphadenopathy and elevated S-ACE (>90 U/L) were detected in the present case. On the basis of the histologic examination results, elevated serum ACE level, and bilateral hilar lymph nodes infiltration, a diagnosis of oral sarcoidosis was made.

Discussion: Sarcoidosis is inflammatory disease of unknown etiology involving many systems, although it predominantly affects the lungs and intrathoracic lymph nodes. Dermal and ocular lesions can also occur.[13] Sarcoidosis affects the head and neck region in about 10% to 15% of patients.[14,15] Salivary glands, particularly the parotid gland, are the most commonly affected areas in the head and neck.[16] Minor salivary gland involvement seems to be less common in sarcoidosis.[17] Oral involvement in sarcoidosis is relatively rare.

The first reported case of oral sarcoidosis was described by Schroff in 1942. Ever since, more than 70 well-documented cases of oral sarcoidosis have been reported in literature (Table I) The buccal mucosa, gingiva, lips, floor of the mouth, tongue, hard palate, and soft palate can be affected. Single or multiple nodular lesions, gingival ulceration, and ulcers of the buccal mucosa, labial mucosa, and palate are the most common manifestations.[18-20] After the buccal mucosa, the gingiva is the second most common oral soft tissue site affected by sarcoidosis, with 17 cases reported in the English language literature

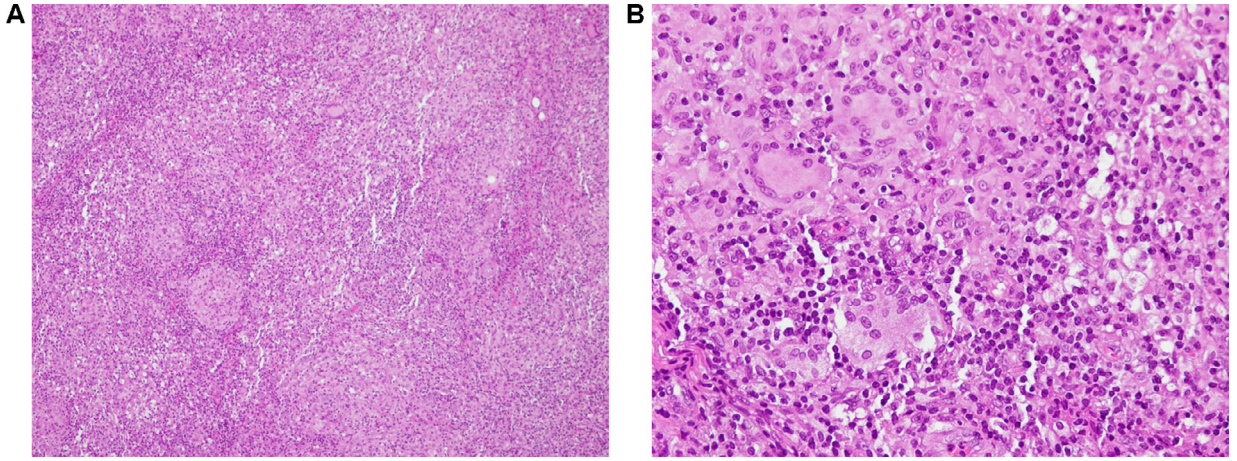

Fig. 6. **(A)** Photomicrograph of gingival biopsy showing noncaseating epithelioid granuloma (hematoxylin and eosin [H&E]; original magnification: × 100). **(B)** Many Langerhans multinucleated giant cells with inflammatory cells infiltrate (H&E; original magnification: × 400).

Table I. Distribution and appearance of oral sarcoidosis according to the site (adapted from Bouaziz et al. 2012)[26]

Site	*Number of cases*	*Age range*	*Male*	*Female*	*Not Mentioned*	*Chief complaint*		*Management*	
Buccal Mucosa	21	15-71	6	10	5	Swelling	8	No treatment	5
						Mass or nodule	5	Excision	4
						Ulcer	2	Steroids	3
						Pain	2	Oxygen	1
						Cystic lesion	1	Not mentioned	8
						Not mentioned	3		
Palate	6	23-40	2	4		Nodules	2	No treatment	3
						Papules	1	Steroids	2
						Erythema	1	Methotrexate & steroid	1
						Perforation	1		
						Swelling	1		
Lip	11	5-66	4	7		Nodules	6	No Treatment	3
						Swelling	3	Excision	3
						Ulcer	1	Steroids	2
						Cystic lesion	1	Not mentioned	3
Tongue	11	25-75	3	8		Nodules	3	No treatment	2
						Swelling	4	Excision	2
						Ulcer	2	Steroids	4
						Plaque	1	Hydroxychloroquine & steroids	1
						Bullous lesion	1	Hydroxychloroquine	1
Gingiva	14	18-57	5	9		Gingivitis	5	No treatment	4
						Hyperplasia	4	Steroids	5
						Swelling	2	Excision	3
						Ulcers	2	Antiseptics	1
						Recession	1	Spontaneous remission	1
Multiple sites	7	26-47	1	5	1	Ulcers	4	Steroids	3
						Gingivitis & recession	1	Steroids & Methotrexate	1
						Lip & palatal swelling	1	Not mentioned	3

so far[21-37](Table II). Most reports included Caucasian patients (mean age 41 years; range 16−66 years) and a nearly equal female-to-male ratio. The clinical presentation is variable and includes predominantly gingival hyperplasia (8 cases), gingival redness/ inflammation (5 cases), and gingival ulcers (4 cases) frequently associated with pain, bleeding, or other gingival symptoms.[25]

Diagnosis of sarcoidosis is made on the basis of 3 criteria: (1) a compatible clinical and radiologic presentation; (2) histopathologic presence of noncaseating granulomas; and (3) exclusion of other diseases with similar findings, such as infections or malignancy.[26] In the majority of cases of sarcoidosis, pulmonary infiltration and hilar lymph nodes enlargement are seen.[38] Blood chemistry may show hypercalcemia, increased liver enzymes, and elevated S-ACE. S-ACE is elevated in greater than 80% of cases.[39] Bilateral hilar lymphadenopathy and elevated S-ACE were recorded in this case.

The etiology of sarcoidosis is not well established. Bacterial and viral infections, such as those by mycobacteria, *Propionibacterium*, Epstein-Barr virus, and human herpes virus 8,and occupational exposures, such as those to wood dust, pollen, mold, and insecticides, have also been proposed as etiologic factors.[22,40] It has been suggested that the pathogenesis of sarcoidosis involves stimulation of T-helper 1 lymphocytes, after antigen presentation through macrophages and dendritic cells, with consequent development of the granulomatous reaction. It seems that the disease has a genetic predisposing mechanism because an association with human leukocyte antigen (HLA)-A1, HLA-B8, and HLA-DR3 has been described.[22,40,41]

The clinical course and severity of the disease can manifest as elevated S-ACE levels. Ueda et al.[42] stated that "ACE could be released from the epithelioid cells present in the noncaseating granuloma of the lesion resulting in high serum levels in 80% to 90% of patients with sarcoidosis." The choice of treatment of oral lesions is ranging from nonspecific therapy to surgical excision.[26] Corticosteroids are considered the primary treatment for sarcoidosis. A combination of antimalarial agents, such as hydroxychloroquine, with low-dose corticosteroids and specific immunosuppressants, such as tumor necrosis factor-α inhibitors, have also been proposed in the treatment of symptomatic systemic sarcoidosis. The prognosis of sarcoidosis correlates with the initial clinical course, host immune response, and severity of disease.[43] The clinical presence of hilar lymphadenopathy is good prognostic sign in this disease; however, the presence of radiologic signs of irreversible fibrosis may worsen the prognosis. Management of the gingival lesions, as reported in the literature, is extremely variable, ranging from no treatment to steroid therapy. Some cases have shown spontaneous remission after diagnosis. There are a few reports of aggressive and/or severe periodontitis in patients with sarcoidosis.[44,45] Local oral hygiene measures and conventional periodontal therapy are usually advised.

In the present case, periradicular alveolar bone loss was present in the location of the gingival lesion. Gingival tissues typically present a mixed inflammatory infiltrate, and this pattern can hinder the interpretation of gingival sarcoidosis specimens by masking the presence of the granulomas. However, it is important to rule out other gingival lesions that are characterized histologically by a chronic granulomatous inflammation, such as orofacial granulomatosis, foreign body granulomatous reactions, granulomatosis with polyangitis (Wegener granulomatosis), and Crohn disease, as well as specific infections, such as oral tuberculosis.[15,22,46-49]

Table II. Summary of literature reports on biopsy proven sarcoidosis involving the gingiva (adapted from Antunes et al. 2008)[25]

Author	*Year*	*Gender*	*Age (Years)*	*Clinical Presentation*	*Treatment*
Tilman[27]	1964	Male	66	Normal gingiva	None
Watts[28]	1968	Female	42	Gingival hyperplasia	Steroids
Hogan[29]	1983	Female	37	Gingival hyperplasia	None
Sloan et al[24]	1983	Male	16	Gingival hyperplasia	Surgery + steroids + Oral hygiene
Altman & Robinson[30]	1984	Male	36	Gingival hyperplasia	Steroids
Zakrzeweska & Naily[31]	1985	Female	30	Gingivitis	Steroids
Zakrzeweska & Naily[31]	1985	Male	33	Gingivitis	None
Hayter & Robertson [32]	1988	Male	34	Gingival ulcer	None
Caudill[33]	1988	Female	57	Gingival recession	Surgery
Ho & Blair[23]	2003	Female	58	Gingival redness	None
Armstrong et al[21]	2004	Female	39	Gingival ulcers & nodules	Oral hygiene
Antunes et al[25]	2007	Female	57	Gingival erosion/ulcer	Steroids & oral hygiene
Aslangul et al[34]	2008	Female	28	Gingivitis, hyperplasia	Topical antiseptics
Poate et al[35]	2008	Female	41	Swelling	None
Saylor et al[36]	2010	female	28	Gingival enlargement	Surgery
Bouaziz et al[26]	2012	Male	26	-	Steroids
Tripathy et al[37]	2014	Male	42	Gingival enlargement	Surgery + Oral hygiene
Present case	2018	Female	10	Gingival hyperplasia	Surgery

Monitoring disease progress in patients with sarcoidosis is of paramount importance because some lesions may undergo spontaneous remission, whereas others may progress further. Although the present case is a rare type, this report supports findings from previous studies on the oral manifestation of sarcoidosis and stresses the importance of considering it in the differential diagnosis.

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CLINICAL PATHOLOGIC CONFERENCE CASE 3: A SLOW-GROWING EXPANSILE POSTERIOR MANDIBULAR SWELLING

Muhammed Yakin, BDS, MMedSci ,DClinDent,[a] Carlos Liceaga Escalera, DDS,[b] Luis A. Montoya Perez, DDS,[b] and Beatriz Aldape-Barrios, DDS, MS[c], [a] School of Dentistry and Health Sciences, Faculty of Science, Charles Sturt University, Orange, NSW, Australia, [b] Hospital Juarez de mÉxico, Mexico City, Mexico, and [c] Universidad Nacional Autónoma de MÉxico, Mexico City, Mexico

Clinical Presentation: A 54-year-old female patient presented with an asymptomatic swelling of 3 months' duration after dental extractions (Figure 1). The patient remembered having facial asymmetry 2 years before the dental extractions. The patient showed a cooperative attitude and consented to having laboratory tests and imaging performed. Clinically, there was an intraoral blue, solid, and diffuse swelling, with intact overlying mucosa (Figure 2).

The panoramic radiograph showed a relatively well-defined multilocular radiolucency extending form the area of the missing mandibular permanent right first molar anteriorly, toward the posterior part of the mandibular ramus posteriorly, past the normal location of the inferior dental canal, which was not seen in the radiograph (Figure 3). The radiolucency was clearly demarcated toward the anterior part of the right-hand side of the mandible but less so in the posterior and superior margins of the radiolucency. Inferiorly, the radiolucency extended toward the lower border of the body of the mandible. The inferior cortex appeared sound, and no periosteal reaction was detected. In addition, the area of the missing mandibular permanent right first molar appeared punched out, and there was loss of trabeculation anterior to the main radiolucent area and posterior to the right mental foramen. Close to the ascending ramus of the mandible, a soft tissue shadow accompanied by irregular radiolucent specks and trabeculae was seen.

On the panoramic radiograph taken 2 years before the dental extractions, root canal fillings were evident. There was no visible radiolucency, but there were signs of condensing osteitis (Figure 4).

Aspiration of the swelling yielded blood, and an incisional biopsy specimen (Figure 5) showed significant bleeding.

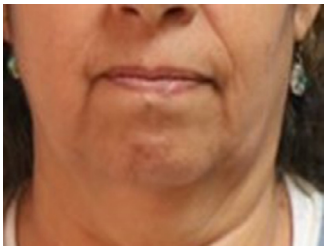

Fig. 1. Photograph of patient at presentation.