A large mandibular soft tissue lesion in an 8-year-old boy

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CLINICAL PRESENTATION

An 8-year-old male patient was referred to an oral surgery clinic for evaluation of a slow-growing soft tissue mass, which was located posterior to the lower right permanent first molar and had been enlarging for the past 6 to 9 months (Figure 1). The patient had no significant medical conditions or current medications. No known drug allergies were reported, and the patient had a noncontributory family medical history. Clinical examination revealed a nonmobile, firm, pink to red, asymmetric, slightly tender mass, which measured 3.5 cm in greatest diameter and interfered with mastication. Extraoral examination revealed no facial swelling, asymmetry, or regional lymphadenopathy. A panoramic radiograph revealed a radiolucent soft tissue growth over the right mandibular alveolar ridge. Cupping effect was also noted superior to the developing tooth #31 (Figure 2). An excisional biopsy of the tumor was performed with the patient under general anesthesia. The patient was lost to follow-up after the diagnosis.

DIFFERENTIAL DIAGNOSIS

Because this was a soft tissue overgrowth causing cupping of bone, the differential diagnoses did not include any central lesions. The differential diagnoses for a slow-growing soft tissue mass of the alveolar ridge includes reactive lesions, such as peripheral giant cell granuloma, pyogenic granuloma, and peripheral ossifying fibroma; peripheral odontogenic tumors, such as peripheral odontogenic fibroma and peripheral ameloblastoma; and, even less commonly, mesenchymal tumors, such as myofibroma, leiomyoma, and neurofibroma. In a young age group, aggressive soft tissue tumors, such as desmoplastic fibroma, Langerhans histiocytosis (LCH), rhabdomyosarcoma, and metastatic neoplasms, should also be considered. However, lack of an ill-defined or moth-eaten radiographic appearance of the mass, along with the absence of a history of rapid growth, argued against an aggressive lesion in this case.

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Reactive Oral Lesions

Peripheral ossifying fibroma (POF) was a possibility. POF is an isolated reactive overgrowth that exclusively affects the gingiva.[1] It can occur at any age, with a peak prevalence in the second decade.[2] The anterior maxillary gingival region is the most common location, and POF demonstrates a female predilection.[1,2] The etiology of POF is uncertain; however, it has been attributed to local irritation or trauma. Clinically, it presents as a pink to red, solitary, asymptomatic, slow-growing, sessile or pedunculated nodular mass. It can grow up to 5 cm in diameter.[1]

Peripheral giant cell granuloma (PGCG) is a reactive lesion occurring as a bump on the gingiva or the alveolar ridge.[3] It affects a wide age range, with a slight female predilection and an affinity for the mandible.[4] A recurrence rate of 9.5% after simple excision and curettage has been reported for PGCG.[4] The largest size ever reported for a PGCG is 6.2 cm.[4] Cupping erosion of underlying bone is a common manifestation of PGCG, described in one-third of the cases. This feature closely mimics the radiographic presentation of the lesion in our case.[4] Thus, the asymptomatic nature and the radiographic presentation of the lesion seen in our case correlated well with PGCG.

Pyogenic granuloma (PG, which is another consideration in the clinical differential diagnosis, shows some similarity to PGCG and to the lesion in our case. PG affects patients of all age groups, particularly young adult females in the second decade of life.[5] PG presents as an exophytic tumor-like growth of soft tissue, with a pedunculated or sessile base that results from a reactive response to local irritation caused by trauma, hormonal changes, or drug-induced hyperplasia.[1,5] PG present as a red, erythematous nodule that bleeds easily and can sometimes reach a maximum size up to 2.5 cm, closely resembling the mass in the present case. In our case, spontaneous bleeding upon probing was neither reported in patient history nor found on clinical examination. Clinically, PGCG may exhibit a more purple-blue hue in comparison with the more erythematous color of PG. PGCG, POF, and PG can only be definitively distinguished on the basis of histologic examination findings. PGCG demonstrates multiple osteoclast-like multinucleated giant cells distributed in a highly cellular and well-vascularized mesenchymal background.[3,4] In contrast, POF consists of highly cellular connective tissue stroma composed of plump, spindled fibroblasts proliferating in

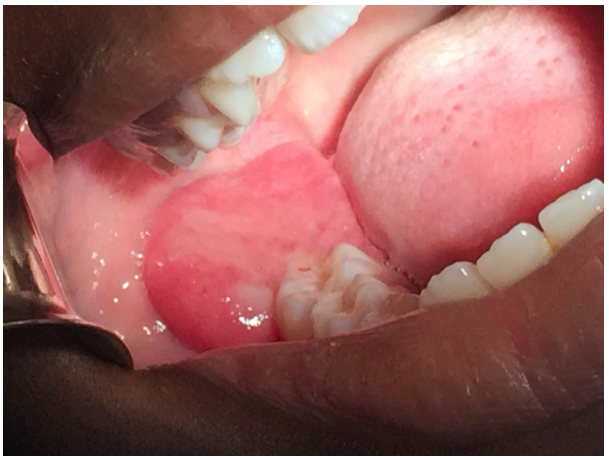

Fig. 1. Clinical appearance of a massive nodular mass in the posterior region of the right alveolar ridge covered by a smooth surface and reddish vascular mucosa.

a patternless pattern.[2] It shows areas of variable amount mineralized components consisting of bone trabeculae, cementum-like material, or dystrophic calcifications.[2] PG shows a highly vascular proliferation resembling granulation tissue with numerous proliferative endothelial cells in a delicate collagenous background.[3,5] Management of reactive gingival lesions requires conservative surgical excision and elimination of local irritants. The recurrence rate of PG is estimated to be 16%.[5]

Peripheral Odontogenic Tumors

Peripheral odontogenic fibroma (POdF) is another crucial entity to be considered in the differential diagnosis of this exophytic mass. POdF is a rare benign neoplasm of odontogenic origin.[6] This entity affects a broad age range. A large case series of this entity reported the peak incidence of POdF in the second to fourth decades of life, with mean patient age of 35 years.[7] However, cases have been reported in newborns and other patients as young as 5 years of age.[7] Clinically, the lesion appears as an exophytic soft tissue mass that varies in size, and a lesion measuring 3.4 cm has been reported.[7] Microscopic examination of POdF shows a background of fibroblastic proliferation infiltrated with inactive-appearing odontogenic epithelial islands. Calcified materials in the form of bone, cementum, or dentinoid spherules can be seen.[7] Surgical excision or excisional biopsy with peripheral osteotomy is the recommended management approach for POdF. A significant recurrence rate of 50% has been reported.[7]

Peripheral ameloblastoma (PA) is a rare entity that appears clinically as an exophytic soft tissue growth on the tooth-bearing areas of the jaws.[8] PA is a nonaggressive subtype of ameloblastoma that usually does not

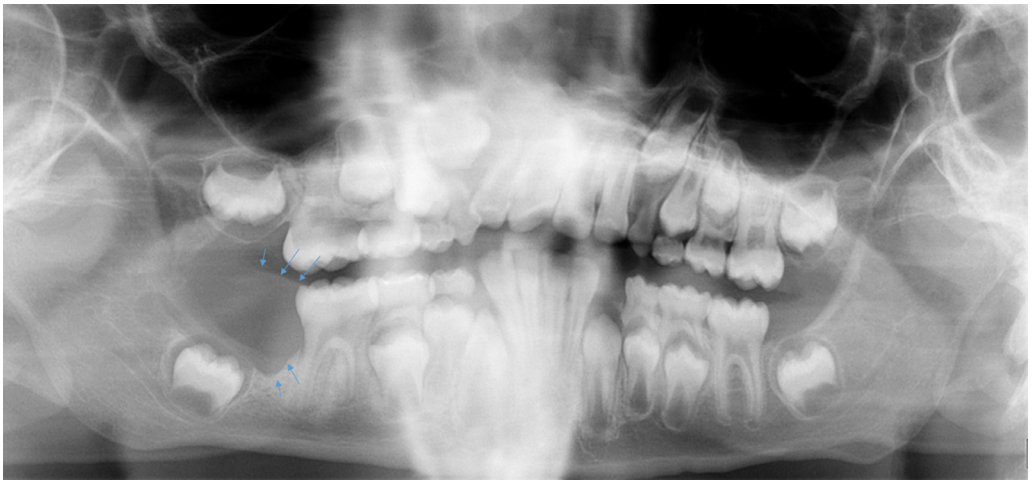

Fig. 2. Panoramic radiograph is depicting superficial resorption (cupping) of the alveolar bone crest produced by a peripheral posterior to the first permanent molar.

show radiographic evidence of underlying bone involvement.[8,9] However, saucerization or cupping effect from tumor pressure on bone has been reported rarely.[8] Clinically, PA presents as a painless, sessile, firm, smooth, pink to red overgrowth with a mean diameter of 1.3 cm.[9] PA has been reported in patients in their 5th to 6th decades but can occur at any age, with a male-to female-ratio of 1.9:1.[9] PA exhibits a preference for the mandible, with a rate of 70.9% reported in a large series of 160 cases.[9] Histologically, PA is composed of odontogenic epithelial islands exhibiting a hyperchromatic, palisaded basal cell layer with reverse polarity and apical vacuolization. Unlike conventional ameloblastoma, the involvement of bone or periosteum is not evident.[8] As PA does not typically show aggressive behavior compared with conventional ameloblastoma, a conservative surgical excision with free margins is the treatment of choice. Philipsen et al. reported a recurrence range of 16% to 19%.[9]

Mesenchymal Tumors

Another entity considered in the differential diagnosis is neurofibroma, which is a benign tumor of peripheral nerve sheath origin. It can be solitary or occur in association with neurofibromatosis type 1, which often presents with multiple lesions. Solitary neurofibroma represents nearly 6.7% of neural tumors affecting the oral cavity.[10] Clinically, it may present as an asymptomatic, slow-growing soft tissue mass with a preference for the tongue, buccal mucosa, and lips.[11] The solitary variant affects younger adults and shows a female predilection, which was not found in the present case.[10,11] Central counterparts within the jaws have been reported rarely. Histologically, it exhibits a well-circumscribed mass composed of interlacing bundles of spindle-shaped cells and a myxoid or delicate collagenous stroma.[11] Mast cells may be seen but are nondiagnostic. Immunohistochemical reactivity to S-100 or neurofilament protein confirms the diagnosis.[12] CD34 and epithelial membrane antigen staining may be variably positive in isolated cases of neurofibroma.[11] Solitary neurofibromas not associated with neurofibromatosis type 1 generally show low recurrence rates after conservative surgical excision.[10,11]

Leiomyoma is a benign smooth muscle neoplasm that commonly arises in the genitourinary and gastrointestinal tract of females and rarely affects the oral cavity.[13,14] Many histologic variants of leiomyoma, including the solid, vascular, and epithelioid types, have been reported in the literature. The most commonly reported type in the oral cavity is vascular leiomyoma.[13,15] Oral leiomyoma is most commonly a submucosal lesion of the lip, tongue, palate, or buccal mucosa. It is frequently seen in older male patients (mean age 42.5 years at diagnosis). This was not consistent with the age of the patient in the present case.[15] The majority of oral leiomyomas present clinically as asymptomatic, slow-growing, firm masses that show a pinkish-to-red hue, depending on the lesion vasculature. Microscopically, it shows bundles of elongated spindle cells with cigar-shaped, blunt-ended nuclei arranged in streaming fascicles around endothelium-lined vascular spaces. The spindle cells intersect at right angles and show perinuclear vacuolization. No mitotic activity or cytologic atypia is seen.[15] Immunohistochemical stains help distinguish this tumor from other spindle cell tumors of muscle origin. Leiomyoma shows high positive reactivity to smooth muscle actin (SMA) and desmin. The management of large lesions includes extensive surgical resection; however, the prognosis of leiomyoma is favorable because of its low recurrence rate.[15]

Among myofibroblastic tumors, desmoplastic fibroma and myofibroma must be considered in the differential diagnosis. Although both tumors occur in a wide age range, they are common in those younger than age 20 years. Desmoplastic fibroma is a benign, locally aggressive tumor that can show clinical behavior similar to the tumor in the present case. The age range is wide (mean age 14 years), and there is no gender predilection. The majority of the cases present sporadically, and familial presentation seen frequently in association with familial adenomatous polyposis or Gardner syndrome.[16] Desmoplastic fibroma presents in the mandibular posterior area in 80% of the cases compared with 16% in the maxilla.[17,18] Patients frequently present with painless, slow, or, if aggressive, fast-growing swellings, which may be associated with tooth mobility, bone destruction, and tooth displacement.[17] Radiographic presentation varies from unilocular to multilocular radiolucency and can be well defined or ill defined. Although desmoplastic fibroma is predominantly an intraosseous tumor, it is commonly associated with a soft tissue component. Microscopically, it is composed of small, elongated fibroblasts in interlacing patterns, with a background of collagenized connective tissue. Immunohistochemistry (IHC) is positive for vimentin, SMA (77%), variable β-catenin, and definitively negative for S100, the muscle-specific actin.[18] The lesion may exhibit a relatively high recurrence rate with conservative excision, and a wide resection minimizes the recurrence rate to 5%.[17]

Skeletal muscle tumors such as rhabdomyosarcoma (RMS) may also be a consideration in the differential diagnosis. RMS is considered the most common soft tissue sarcoma in pediatric patients. RMS shows a slight male predilection, and 30% of the cases occur in the head and neck region,[19] but RMS rarely occurs in the oral cavity. If seen within the oral cavity, the most common sites are the tongue, lip, and palate. RMS usually presents as a well-demarcated, nodular or polypoid

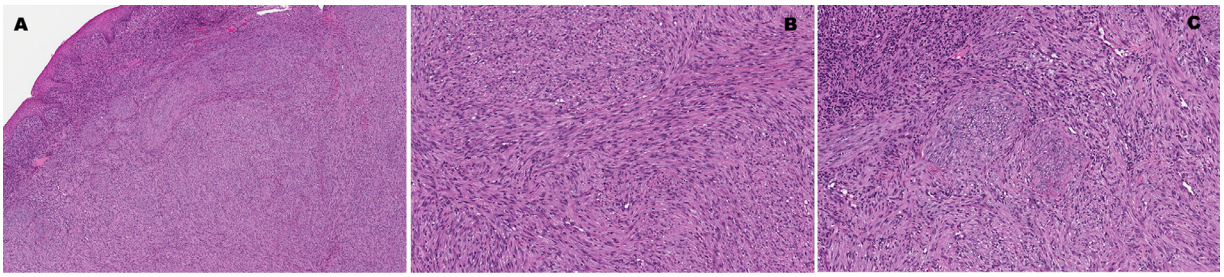

Fig. 3. **A,** Hematoxylin and eosin (H&E) staining shows the characteristic "zoning phenomenon" biphasic cellular and acellular zoning seen in myofibromas (magnification × 5). **B,** A short, streaming pattern of fascicles composed of spindled cells with cigar-shaped nuclei was demonstrated (magnification × 20). **C,** Distinct features typical to myofibroma are demonstrated with the formation of myoid balls, with a densely packed, cellular central portion surrounded by an acellular and myxoid area at the periphery (magnification × 20).

lesion that has a soft consistency and exhibits a smooth or lobulated surface. Clinical signs are variable and include neural deficit, loss of teeth, trismus, and pain.[20] The histologic variants encountered are the alveolar, pleomorphic, and embryonal types. Among pediatric patients, the most common variant is the embryonal type, which has a somewhat favorable prognosis in comparison with the other variants.[19] However, the present case was devoid of any signs or symptoms that may raise suspicions of malignancy.

Langerhans Cell Histiocytosis

LCH is a rare neoplasm that is encountered primarily in pediatric patients age 1 to 15 years.[21] LCH commonly affects the mandible with a prevalence rate of 10% to 20%.[22] Radiographically, it can present as a unilocular or multilocular radiolucency. It is generally well demarcated but can be ill demarcated, depending on the level of medullary or cortical bone destruction. It causes significant destruction that is often limited to the alveolar process, creating the characteristic "tooth floating in air" appearance. Mucosal involvement has been noted if the central lesion breaks out of bone.[21,22] The radiographic features mentioned for this entity was absent in our current case. For monostatic jaw lesions, surgical excision offers a favorable prognosis. Radiotherapy and chemotherapy should be reserved for inoperable cases or those with polyostotic involvement.[21,22]

DIAGNOSIS AND MANAGEMENT

The current patient underwent excision under general anesthesia. Gross examination revealed multiple, tanned, cauliflower-shaped pieces of soft tissue measuring 3.5 × 2.1 × 0.9 cm in aggregate, sectioned longitudinally. Microscopic examination revealed an ulcerated benign neoplastic proliferation composed of elongated spindle−shaped cells, with eosinophilic cytoplasm (smooth muscle morphology) arranged in swirls and interlacing fascicles distributed in a delicate fibrous stroma. Cellular and nuclear pleomorphisms were seen in this framework. Scattered mitoses were noted. Also noted was the formation of pseudochondroid "myoid balls," which were scattered throughout the specimen. Dense cellularity was seen in most areas. The neoplasm appeared myxomatous in many areas (Figures 3 Ato 3C).

An immunohistochemical panel, including SMA (smooth muscle marker); desmin (skeletal muscle marker); SOX-l 0 (neural crest marker); and S-100 protein (neural origin marker), was performed with adequate controls. The lesional cells were strongly reactive

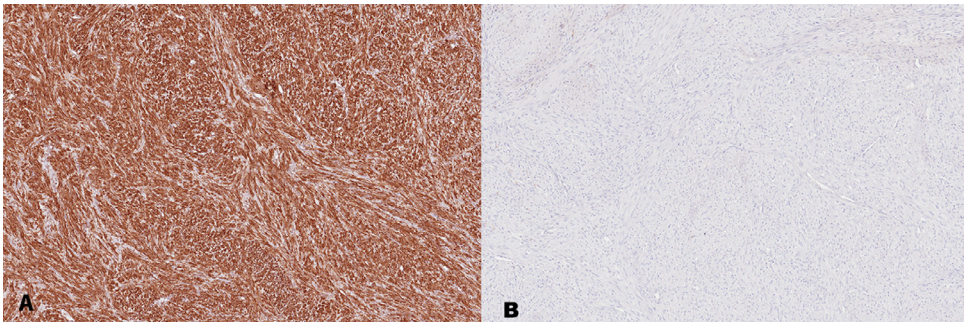

Fig. 4. **A,** Positive diffuse reactivity to α-smooth muscle actin (α-SMA) underlining the variable cellularity all over the lesion (magnification × 20). **B,** No reactivity to desmin illustrated in the highly cellular area of the lesion (**A,** magnification × 4; **B,** magnification × 20).

for SMA and negative for desmin (Figure 4), indicating a smooth muscle/myofibroblastic origin (Figure 3D). Histomorphology, when combined with the IHC results and the clinical background, supported the final diagnosis of myofibroma. No follow-up information was available for this case.

DISCUSSION

Initially, myofibroma was considered a benign pediatric tumor exclusively.[23] Later, it was re-classified as an uncommon myofibroblastic tumor that affects patients of a wide age range (mean age 23 years). These lesions exhibit a slight male predilection, with a ratio of 1.2:1.[24] The most common intraosseous location in the head and neck region is the mandible, and soft tissue lesions occur most commonly in the buccal mucosa, retromolar area, and tongue, followed by the labial mucosa.

Among patients age less than 20 years, 58% of myofibromas occur on the attached gingiva and 38% on the movable mucosa.[24] In contrast, a relatively high incidence is seen in the movable mucosa (67.8%) in patients older than 40 years of age. Smith et al. reported that a vast number of myofibromas involving the posterior alveolar mucosa appeared to be overlying an unerupted molar or associated with an unerupted tooth.[24] This finding is similar to the present case, in which the lesion would have interfered with the eruption of the underlying tooth #31.

In comparison with myofibroma in adults, that in pediatric patients tends to show greater bone involvement.[25] Solitary myofibromas have a relatively low recurrence rate. Foss et al. reported recurrence of the tumor in 4 of 79 patients, with an average follow-up of 12 to 15 months.[25] Myofibromatosis is histologically identical to myofibroma, but the difference is in the involvement of multiple sites, the rarity, and the most common involvement in infants.[16,23,25]

Microscopically, myofibroma demonstrates a characteristic biphasic histologic pattern of cellular and acellular areas and a nodular and bundled arrangement of spindle cells. In addition, diffuse positive reactivity to SMA and muscle-specific actin and negative or weak reactivity to desmin are very useful in establishing the diagnosis. Oudijk et al. reported SMA positivity in 95% of cases and desmin negativity in 90% (n = 114).[26] IHC is not useful in differentiating between myofibroma and myofibromatosis. Myofibroma can demonstrate a worrisome clinical growth pattern that can lead to its being misinterpreted as a malignant or aggressive entity. It also has quasi-malignant microscopic features, such as mitotic figures, but a mitotic activity of less than 5 per × 10 high-power fields is acceptable in myofibromas.[25]

Microscopic differential diagnosis for solitary myofibroma includes inflammatory myofibroblastic tumor (IMT), leiomyoma, low-grade fibromyxoid sarcoma, solitary fibrous tumor (SFT) and fibrous histiocytoma.

IMT is known to occur in patients of a wide age range but demonstrates a tendency to occur in children and young adults. IMT can arise anywhere in the body. Histologically, IMT demonstrates a dense inflammatory infiltrate, a proliferation of spindle-shaped myofibroblasts/fibroblasts distributed in a variably fibromyxoid background. Despite similarities to myofibroma with regard to histomorphology, positive immunohistochemical staining for anaplastic lymphoma protein kinase in IMT distinguishes it from myofibroma.[16,26] In addition, genetic analysis is useful in distinguishing between these 2 entities.[27,28]

As mentioned earlier, leiomyoma is an unusual benign smooth muscle tumor in the oral cavity. It is most frequently seen in older adults.[13] Leiomyoma shows histologic similarity to myofibroma; however, these 2 entities can be distinguished through immunohistochemical staining. In leiomyoma, neoplastic cells show high positive reactivity to SMA and desmin.[13-15] In contrast, myofibroma shows no reactivity to desmin.[26]

Solitary fibrous tumor (SFT) is an uncommon benign tumor of mesenchymal origin. SFT affects patients of a wide age range and shows a slight predilection for adults and females.[29] Clinically, it may mimic any reactive oral lesions and frequently involves the buccal mucosa, tongue, and palate, at a rate of 45.6%, 15.4%, 6.6%, respectively.[29] Microscopically, SFT exhibits variable cellularity of bland spindle to ovoid cell proliferation and haphazardly distributed staghorn thickened wall blood vessels in a collagenous or myxoid background.[29] Histologic variation within SFT can imitate other myofibroblastic tumors, thus immunohistochemical staining is crucial for final diagnosis. Commonly used immunohistochemical stains that show variable positive expression in SFT are CD34, CD99, and Bcl-2. Positive expression of the STAT6 protein is reported as high as 100% in benign SFTs. However, no expression of the STAT6 protein has been ever reported in myofibroma.[30] Cytogenic studies of SFT has shown unique chromosomal translocation affecting the *NAB2* and *STAT6* genes.[29]

Benign fibrous histiocytoma (BFH) is a soft tissue neoplasm that exhibits biphasic fibroblastic and histiocytic differentiation.[31] BFH presents anywhere in the body in cutaneous and subcutaneous areas and in bone.[31,32] To date, few cases in the oral cavity have been reported. BFH affects patients of a wide range of age, with a slight male predilection.[31] Clinically, oral BFH appears as a solitary, slow-growing, asymptomatic, well-circumscribed nodule that can reach up to 10 cm in size.[31,32] Microscopic examination of BFH demonstrates dual-cell populations composed of spindle-shaped fibroblasts with elongated nuclei and round histiocytes that

vary in size. The lesional cells are arranged haphazardly in storiform fashion within a mature fibrous connective tissue background that shows areas of focal hyalinization and myxoid changes.[31,32] The microscopic appearance of BFH can mimic that of myofibroma; however, pseudochondroid "myoid balls" are not seen in BFH, and the lack of multinucleated giant cells renders the diagnosis of myofibroma.

Low-grade fibromyxoid sarcoma usually affects patients younger than 18 years of age. These lesions arise superficially in the head and neck area. Microscopic examination exhibits alternate zones of myxofibrous areas and spindle cell proliferation; however, rare foci of cytologic atypia may be seen. Genetic analysis demonstrates a distinctive t(7;16) (q32−34;p11) translocation (2,63), which aids in the diagnosis of this low-grade malignancy.[16] In our case, the histomorphology and the immunohistochemical staining profile, namely, SMA positivity and negative reactivity to S-100, SOX-10, and desmin, were highly supportive of a diagnosis of myofibroma.

CONCLUSIONS

Myofibroma is generally a slow-growing lesion. Because of its high recurrence potential, long-term clinical follow-up is essential.

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