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Oral manifestations in adolescent patients with pediatric - onset multiple sclerosis: a pilot study

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Abstract

Objectives: To identify and characterize oral manifestations in adolescents with pediatric-onset multiple sclerosis (POMS).

Methods: Nine adolescents with newly diagnosed POMS (8 females, 1 male; mean age 16.1 ± 1.3 years; mean EDSS 1.66) underwent neurological and standardized dental examinations. The oral assessment included a structured interview, clinical evaluation of oral mucosa, gingival inflammation (GI), periodontal pocket depth and caries recording using ICDAS-II criteria. Physicochemical salivary parameters were assessed with the Saliva-Check Buffer Test. In five patients, panoramic and occlusal radiographs were obtained.

Results: All participants exhibited pronounced mucosal vascularization with linear/punctate telangiectasias, enlargement/erythema of the incisive papilla and dry lips with fissures/erosions. Lingual varices were observed in 3/9. Gingivitis and dental caries were present in all patients (GI mean 0.623 ± 0.391; DMFT mean 4.625 ± 2.781). Resting salivary flow was reduced in 6/9 and stimulated flow in 5/9; buffering capacity was normal in only 1/9, while resting salivary pH remained within the normal range. Radiographic evaluation (n=5) consistently showed widening of the incisive and mandibular canals, periodontal ligament space irregularities with a wavy lamina dura, trabecular radiolucencies, root abnormalities and pulp stones and/or pulp chamber obliteration.

Conclusions: Adolescents with POMS demonstrated a consistent pattern of oral vascular alterations accompanied by impaired salivary defense, gingivitis and caries.

Clinical Relevance: Early dental surveillance in pediatric-onset MS may help identify vascular mucosal changes and salivary dysfunction that could increase caries and gingivitis risk. Incorporating salivary testing and targeted radiographic assessment into interdisciplinary care may support timely preventive and therapeutic interventions.

Introduction

Multiple sclerosis (MS) is a chronic, autoimmune inflammatory disease of the central nervous system (CNS) with complex, incompletely understood etiology [1]. It is considered to result from the interplay of environmental exposures (including Epstein–Barr virus infection, infectious mononucleosis, tobacco smoking, and vitamin D deficiency) in individuals with genetic susceptibility [2]. The pathological hallmark of MS is demyelination accompanied by axonal injury and neurodegeneration, driven by activated T and B lymphocytes and cells of the innate immune system [1].

MS predominantly affects individuals aged 20–40 years and occurs more frequently in women. However, it may also present in childhood, in which case it is referred to as pediatric-onset multiple sclerosis (POMS).. It is estimated that at least 5% of the overall MS population consists of children [3]. Between 3% and 10% of patients develop symptoms before the age of 16 years and < 1% before the age of 10 years [4]. Population-based studies, case series and case–control studies indicate that individuals younger than 18 years account for approximately 1.7% to 5.6% of the MS population [3, 5]. In recent years, there has been increasing interest in POMS, its underlying determinants and prodromal manifestations preceding the first neurological symptoms [6].

The clinical presentation of MS is heterogeneous and depends on the CNS regions involved including the brain, spinal cord and optic nerves. The formation of inflammatory lesions leads to demyelination, axonal damage and loss, resulting in motor deficits, sensory disturbances, visual impairment and cognitive dysfunction. MS most commonly follows a relapsing–remitting course (approximately 80% of the overall MS population and > 98% of patients with POMS), characterized by episodes of acute neurological dysfunction followed by periods of partial or complete recovery. Disability in MS patients typically accumulates over time [6]. The clinical phenotype of POMS differs from that of adult-onset disease. In children, onset is generally more aggressive and often presents with a multifocal pattern [3]. Although disease progression tends to be slower, early relapse rates are higher [7]. Compared with adults with MS, patients with POMS reach disability milestones at a younger age, have a higher annualized relapse rate, greater lesion burden and brain atrophy on magnetic resonance imaging and more rapidly evolving cognitive impairment [8].

Craniofacial manifestations may include trigeminal neuralgia–type pain, paresthesia, hemifacial spasms with dysarthria, tonic facial spasms, muscle weakness and temporomandibular joint disorders [9, 10]. Oral manifestations include xerostomia, gingivitis and dental caries, toothache, dentin hypersensitivity and taste disturbances [3, 10]. Bruxism and increased pulpal sensory threshold to electrical and cold stimuli have also been reported. Oral symptoms in patients with MS may arise from the disease process itself or as adverse effects of pharmacotherapy [11–13].

Studies evaluating oral health in MS have predominantly focused on adult patients and have examined associations not only with disease duration but also with the type of treatment received. Only a few investigations have included pediatric participants, however results were typically reported for the entire cohort without separate analyses for pediatric patients [14, 15].

The aim of this study was to identify and characterize oral manifestations observed in adolescent patients with POMS.

Materials and methods

Ethical approval

was obtained from the Bioethics Committee of the Medical University of Warsaw (approval no. KB/40/A 2025) and the study protocol complied with international etical standards of the Declaration of Helsinki. The study was conducted within the framework of project no. 2024/ABM/03/KPO/KPOD.07.07-IW.07-0192/24 – 00. Eligible participants were patients with a recent diagnosis of multiple sclerosis established by a neurologist, without prior neurological treatment, who provided written informed consent signed by the patient and/or a parent/legal guardian. Patients with other systemic diseases, those receiving chronic medication and those with a history of previous neurological treatment were excluded.

All enrolled participants underwent neurological and dental examinations. The dental assessment comprised a structured interview (e.x. craniofacial pain and sensory disturbances, as well as difficulties with swallowing and speech) and a clinical examination (assessment of functional disorders and parafunctional habits of the stomatognathic system; evaluation of the lips and oral mucosa; assessment of the gingiva and dentition, including dental caries and the presence of dental calculus). In addition, physicochemical salivary parameters were assessed (consistency, pH, buffering capacity and flow rate). In five patients, the clinical examination was supplemented with radiological assessment (panoramic radiography and occlusal radiographs). All dental examinations were performed by two specialists in pediatric dentistry in a dental office setting.

During the oral mucosal examination, color, moisture and the presence of lesions, including their type and location, were recorded [16, 17]. Gingival inflammation was assessed using the Gingival Index (GI) according to Löe and Silness [18]. The gingival condition was evaluated on four surfaces—mesiobuccal, buccal, distobuccal, and palatal/lingual—of six index teeth: 16, 12, 24, 36, 32, and 44. Based on the GI values, gingivitis was classified as mild (GI = 0.1–1.0), moderate (GI = 1.1–2.0), or severe (GI = 2.1–3.0). Using a WHO CPI periodontal probe, periodontal pocket depth was measured at six sites per tooth for teeth 16, 12, 11, 21, 22, 26, 36, 32, 31, 41, 42, and 46. For dental status assessment, each surface of every tooth was examined and caries lesions were recorded according to the ICDAS-II criteria [19]. The number of teeth present in the oral cavity was determined, as well as the number of teeth (T) and tooth surfaces (S) with non-cavitated lesions (D1–2T), cavitated caries lesions (D ≥ 3T), teeth missing due to caries (MT), and teeth with fillings (FT). The indices D1–2T, D ≥ 3MFT, D1–2MFS, and D ≥ 3MFS were calculated and expressed as mean ± SD. In addition, the number and percentage of patients with caries (D1–2T > 0 and/or D ≥ 3MFT > 0) were determined.

The Saliva-Check Buffer Test (GC Co., Tokyo, Japan) was used for salivary assessment. The examination was performed in accordance with the manufacturer's instructions. Salivary tests were conducted in the morning, at least 2 h after breakfast. Participants were instructed not to brush their teeth or use mouthwash for at least 1 h prior to the examination. The following parameters were assessed: resting salivary flow rate, salivary consistency, resting salivary pH, stimulated salivary flow rate, and salivary buffering capacity.

Results

The study included eight girls and one boy (mean age, 16.1 ± 1.3 years). The characteristics of the enrolled participants are presented in Table 1.

Table 1
Overall characteristics of included patients.

Subjects (n)	**9**
Female (n)	**8**
Male (n)	**1**
Mean age (years)	16,2
Age at diagnosis (years)	13–17 (mean 15,5)
BMI (mean)	22,58
Vitamin D, mean serum concentration (mg/dl)	23,11
Allergy history (n)	1
Positive	8
Negative	
Expanded Disability Status Scale (EDSS) (mean)	1,66

None of the participants reported numbness, pain, burning sensations, dysarthria, loss of oral sensation, toothache or dentin hypersensitivity, or difficulties with swallowing or speech. One patient reported paresthesia involving the tongue and the floor of the mouth. All patients presented with dry, fissured lips with cracks and/or erosions, prominent mucosal vascularization, and linear and punctate telangiectasias located in various oral regions and on the vermilion border, as well as enlargement and erythema of the incisive papilla (Figs. 1–3). In addition, gingivitis and dental caries were observed in all patients (Tables 2 and 3). In three patients, vascular dilatation resulted in a clinical appearance consistent with lingual varices (Fig. 4). Frequent findings included the presence of dental calculus, linea alba, and yellowish discoloration of the palate (Fig. 1).

Figure 1. An enlarged and erythematous incisive papilla, yellowish discoloration of the palate, and telangiectasias.

Figure 2. Marked vascularization of the vestibular oral mucosa with vascular macules.

Figure 3. Vascular macules on the vermilion border

Figure 4. Lingual varices and prominent telangiectasias on the floor of the mouth and the ventral surface of the tongue.

Table 2
Extra- and intraoral manifestations and physicochemical salivary parameters in patients with MS.

Extraoral findings	**Number of patients/ Result**
Bruxism (n)	2
Dry lips (n)	8
Intraoral findings	
Cracks and erosions of the lips and/or oral mucosa (n)	8
Petechiae (macules) on the vermilion border and/or the oral mucosa (n)	8
Linear vascular dilatations (telangiectasias) (n)	8
Erythema of the vestibular oral mucosa and the incisive papilla (n)	8
Yellowish discoloration of the oral mucosa (n)	6
White lesions of the oral mucosa (n)	8
Coated tongue (n)	5
Gingivitis (GI ≥0,1) (n)	8
GI (mean ± SD)	0,623±0,391
Dental calculus (n)	6
Caries (DMFT ≥ 1)	8
DMFT (mean ± SD)	4,625±2,781
DT (mean ± SD)	9,38±4,06
DMFS (mean ± SD)	6,375±5,266
DS (mean ± SD)	11,0± 6,97
Physicochemical properties of saliva	
Resting salivary flow > 60 s (n)	2
Saliva consistency – watery (n)	5
Saliva consistency – bubly (n)	3
pH of resting saliva (mean ± SD)	6,93±0,36
Range 6.0–6.6 (n)	2
Range 6.8–7.8 (n)	6
Stimulated salivary flow 3.5-5.0ml/5 min (n)	5
Stimulated salivary flow > 5.0ml/5 min (n)	3
Normal buffering capacity (n)	1
Low buffering capacity (n)	5
Very low buffering capacity (n)	2

Table 3
Oral health status in individual patients with MS, including the distribution of oral mucosal lesions and the severity of dental caries and gingivitis.

Cracks and erosions	**WL**	**WK**	**HK**	**MP**	**AK**	**TG**	**AV**	**WŚ**
	lips	**Hard palate, tip of the tongue**	**Lips, cheeks**	**lips**	**Lips, dorsal part of the tongue**	**Lower lip**	**Lips, left palatal arch**	**lips**
Petechiae (macules)	Left cheek mucosa, lips	Hard palate, right cheek mucosa, floor of the mouth, ventral surface of the tongue	Attached gingiva, hard palate, mouth angle	Attached gingiva	lips, ventral surface of the tongue	lips, hard palate, floor of the mouth	lips, attached gingiva	lips, marginal and attached gingiva, tongue
Linear vascular dilatations	vestibulum, soft palate, floor of the mouth, lips	vestibulum, floor of the mouth, ventral surface of the tongue	vestibulum, palate, floor of the mouth, ventral surface of the tongue	vestibulum, floor of the mouth	vestibulum, floor of the mouth, ventral surface of the tongue	vestibulum, soft palate, floor of the mouth	vestibulum, soft palate	vestibulum, floor of the mouth
Yellowish discoloration of the oral mucosa	palate	palate	palate, tip of the tongue	no	palate	no	palate	tongue, palate
White lesions of the oral mucosa	attached gingiva, linea alba	attached gingiva, linea alba	attached gingiva, linea alba	linea alba	linea alba	attached gingiva	linea alba	attached gingiva linea alba
Coated tongue	yes	yes	no	no	no	yes	yes	yes
Gingivitis (GI)	mild (1,0)	mild (0,6)	mild (0,2)	mild (0,2)	mild (1,0)	moderate (1,2)	moderate (1,2)	mild (0,3)
Dental calculus	+	+	-	+	+	+	+	+
DMFT	9	7	2	3	3	4	1	8
DT	8	17	9	10	4	4	13	10
DMFS	18	8	2	3	3	6	1	8
DS	8	22	9	10	4	4	21	10
Stimulated salivary flow ml/5 min	6	5	5	5	5	8	4	10
Buffering capacity of stimulated saliva	very low	very low	low	normal	low	low	low	low

On radiological assessment, all examined patients showed widening of the incisive canal and the mandibular canal, irregularities of the periodontal ligament space often accompanied by a wavy appearance of the lamina dura, radiolucencies within trabecular bone, root-related abnormalities (dilacerations and/or root resorption), and pulp stones and/or obliteration of the pulp chambers (Fig. 5). One patient exhibited agenesis of tooth 35, and one patient showed a localized radiopaque area in the apical region of tooth 36. Distribution of trabecular bone rarefactions and root-related changes on radiographic imaging in particular patients are presented in Table 4.

Resting salivary flow was reduced (> 60 s to visible salivation) in six patients, and five also demonstrated decreased stimulated salivary flow. Only one patient exhibited normal buffering capacity, whereas the remaining patients had low or very low buffering capacity (Table 3).

Figure 5. Panoramic radiograph (OPG) of a 17.6-year-old female patient with MS: a well-delineated incisive canal, a distinct funnel-shaped incisive fossa, and an incomplete intermaxillary suture (a); irregular periodontal ligament spaces with a wavy lamina dura (b); radiolucencies of the interproximal cancellous bone (c); widening of the periodontal ligament space (d); root canal obliteration, pulp stones (e); and condylar resorption (f).

Table 4
Distribution of trabecular bone rarefactions and root-related changes on radiographic imaging.

Interproximal rarefaction of the cancellous (trabecular) bone.	**MP**	**AK**	**TG**	**AV**	**WŚ**
	16; 12; 11; 22; 23; 33; 32; 42	**16; 26**	**16; 15; 14; 12; 11; 22; 25; 26; 46**	**16; 15; 14; 12; 11; 21; 22; 23; 24; 25; 26; 27; 37; 42**	**12; 21; 37**
Root abnormalities	Root resorpion 12; 11; 21; 22	Internal resorption of canal DB in 26, resorption of buccal canals in 16; 26	dilaceratio 15; taurodontism 17; cementoma 36; resorption of buccal canals in 16; shortened root in 21with bone rarefaction	taurodontism 17; 27; 37; 47; root resorption 15; 12; 11; 21; 22; 47	Dilaceratio 26; resorption of canal DB in 26; root resorption 11; 21

Discussion

The most commonly reported oral manifestations of MS include xerostomia (reduced salivary secretion), periodontal disease and gingivitis, increased susceptibility to dental caries (higher DMFT values), toothache, dentin hypersensitivity and taste disturbances [10, 14]. Consistent with these observations, our cohort demonstrated a uniform pattern of impaired salivary secretion, dental caries and mild-to-moderate gingivitis across all participants.

Dental caries and gingival/periodontal diseases in MS are plausibly multifactorial. Physical disability may compromise manual dexterity and thereby limit the effectiveness of daily oral hygiene procedures, while reduced salivary flow diminishes saliva's protective functions, including lubrication, buffering, antimicrobial activity and mechanical self-cleansing. The association between dental caries in MS and decreased salivary flow has been reported previously [14, 20]. Kiranatli et al. [20] documented lower salivary flow rates in patients with MS than in controls and identified a statistically significant, negative, moderate correlation between DMFT and salivary flow rate. Importantly, in our cohort, salivary dysfunction extended beyond reduced secretion and included marked impairment of buffering capacity: in seven of eight participants buffering capacity was classified as low or very low. In contrast, salivary pH remained within an unfavorable range in none of the patients, which aligns with the findings of Mortazavi et al. [14]. Mortazavi and colleagues also reported that lower salivary flow was associated with a higher caries burden and additionally noted increased salivary calcium and phosphorus concentrations in MS [14].

The caries burden observed in our cohort—likely driven in part by impaired salivary function—appeared higher than that reported in epidemiological studies of Polish adolescents aged 15 and 18 years (D≥3MFT mean ± SD = 5.88 ± 4.15; D1–2T mean ± SD = 0.59 ± 1.34; D≥3MFS mean ± SD = 8.50 ± 7.66) [21]. Notably, our patients exhibited a substantial number of non-cavitated caries lesions, which may reflect early disease activity in a context of reduced salivary defense. With respect to periodontal status, all adolescents demonstrated mild or moderate gingivitis based on the GI index. Overall, signs of gingival inflammation have been reported more frequently in patients with MS than in the general population [15]. In Poland, gingivitis prevalence among adolescents is estimated at approximately 30% [22, 23].

Previous studies suggest that the severity of periodontal and gingival inflammation in MS may correlate with broader clinical factors. Hatipoğlu et al. [24] linked gingival/periodontal inflammation and oral hygiene neglect in adults with MS to the degree of disability, which is particularly relevant in long-standing disease. Labuz-Roszak et al. [25] further highlighted the role of disease duration, reporting that oral manifestations—especially gingivitis—were more pronounced with longer disease course. They also observed associations between oral dryness and relapsing–remitting MS, as well as prior exposure to at least one course of corticosteroids, while not confirming associations with patient age or the use of disease-modifying therapies. In contrast, our patients had newly diagnosed MS

and were not receiving any medications, suggesting that the oral findings in this cohort cannot be readily attributed to long-term disability, cumulative treatment exposure or chronic disease duration.

While plaque-induced gingivitis remains fundamentally related to bacterial biofilm, it is important to consider additional mechanisms that may modulate clinical inflammatory signs such as swelling, erythema and bleeding. First, immune dysregulation—affecting both humoral and cellular pathways—plays a central role in MS pathophysiology and may plausibly influence periodontal inflammatory responses. Second, vascular alterations may substantially contribute to the oral phenotype observed in our patients [20, 26].

Vascular features were a distinctive component of the clinical presentation. On non-keratinized oral mucosal surfaces—such as the vestibular mucosa, floor of the mouth and ventral tongue—vascular structures are physiologically more visible; however, in our cohort the vascular pattern was markedly accentuated, with multiple dilated vessels consistent with telangiectasias. Telangiectatic lesions can present as macular (flat) or papular (raised) and may be linear or round. In our patients, classic round telangiectasias (1–2 mm macules) were only sporadically observed, whereas dilated linear vessels—often tortuous—predominated on the vestibular mucosa and palate, accompanied by raised lesions in the sublingual region. Enlargement and erythema of the incisive papilla and intense erythema of the alveolar mucosa were consistently observed and contrasted sharply with the relatively pale appearance of attached gingiva; in four cases, subtle hyperkeratotic changes further accentuated this contrast. Erythema of the free marginal and interdental gingiva was also present and could reflect inflammatory changes, non-inflammatory vascular alterations, or their coexistence. These findings should be interpreted in the context of established determinants of mucosal color, namely the degree of keratinization and vascularization: greater keratinization reduces vascular visibility and produces a lighter appearance, whereas increased vascularity results in deeper erythema [27, 28].

The most prominent vascular manifestation in our cohort was lingual varices (caviar tongue). Lingual varices are typically associated with aging but have also been linked to tobacco smoking and a range of systemic conditions, including liver disease, scleroderma, cardiovascular disorders, arterial hypertension, rosacea, HIV infection, and type 2 diabetes mellitus [29, 30]. Clinically, they are classically described as multiple bluish or violaceous elevations, most often on the ventral surface of the tongue where the mucosa is thin and translucent. In the “caviar tongue” phenotype, vessels become markedly dilated and tortuous, with rounded, dark lesions resembling caviar [29, 31]. Additional support for vascular involvement in our patients was the presence of red macules in multiple intraoral sites, including the vermilion border, buccal mucosa, floor of the mouth, palate, and even the attached gingiva.

Telangiectasias are a hallmark feature of hereditary hemorrhagic telangiectasia (HHT), but they can also be encountered in other conditions, including liver failure and autoimmune connective tissue diseases such as systemic sclerosis, dermatomyositis and lupus erythematosus [32, 33]. Although our patients were evaluated within the context of MS, these differential considerations underscore the need to interpret telangiectatic changes in a broad systemic framework. Notably, the vascular phenotype extended beyond the soft tissues and was reflected in radiological findings. The incisive papilla overlies the incisive foramen, which constitutes the oral opening of the incisive canal. The canal contains nerve fibers (including the pterygopalatine nerves, the nasopalatine nerves, and palatine branches related to the trigeminal system) as well as vascular structures (the sphenopalatine artery and sphenopalatine vein). In our cohort, panoramic radiographs demonstrated a well-delineated incisive canal and a funnel-shaped incisive fossa/foramen; in one patient, an incomplete intermaxillary suture was noted, potentially reflecting vascular enlargement. Additional findings included trabecular bone radiolucencies of varying size, widening of the mandibular canal and root resorption. Radiographic descriptions of intraosseous vascular lesions support the plausibility of such patterns. According to Li X et al. [34], vascular malformations may present in the maxilla as an indistinct maxillary sinus with mild ground-glass changes or as a well-defined oval radiolucency, whereas mandibular lesions may appear as oval or irregular radiolucencies aligned with the mandibular canal, sometimes associated with root resorption and canal widening, as well as “soap-bubble”–like changes or extensive multiloculated osteolytic lesions with displacement and deformation of roots and expansion of the involved region [34].

External apical root resorption should not be viewed exclusively through the lens of inflammation or trauma. Pressure-related mechanisms have also been proposed, including effects from cysts, tumors, fibro-osseous lesions, or intraosseous vascular anomalies. Although the biological mechanism linking vascular anomalies or vascular proliferation to root resorption remains uncertain, clinical reports support this association. Noreau et al. [35] described root resorption of a permanent molar adjacent to a mandibular vascular malformation in a 9-year-old girl. Fernández et al. [36] reported five cases of root resorption associated with intraosseous hemangiomas and emphasized that risk appears higher when the lesion lies close to the root apex [36]. In addition, external root resorption related to telangiectasias on the hard palate near incisors has been reported in a patient with HHT [37]. Histopathology in that case demonstrated multiple thin-walled vascular elements immediately beneath the surface epithelium, deeper large dilated

vessels with thickened muscular layers and only minimal inflammation, comprised mainly of scattered lymphocytes, plasma cells and occasional mast cells.

Given the coexistence of inflammatory and vascular phenomena, cytokine-mediated pathways warrant consideration. Cytokines modulate inflammatory responses and exert broad systemic effects that depend on cellular sources, cytokine profiles, and complex cytokine–cytokine interactions. In MS, expression of multifunctional mediators such as transforming growth factor beta (TGF-β) and bone morphogenetic protein (BMP) is altered; beyond their roles in CNS and immune regulation, they are key regulators of skeletal homeostasis through effects on chondrocytes, osteoblasts, osteocytes, and osteoclasts [38]. TGF-β may also promote angiogenesis, potentially via induction of vascular endothelial growth factor (VEGF). VEGF is a potent pro-angiogenic factor and has been reported to increase during MS relapses both in inflammatory lesions and in serum compared with healthy controls or patients in remission [39, 40]. The extent to which these pathways contribute to the oral and dental findings in our cohort remains speculative and should be addressed in future studies.

In our cohort, root pathology was not limited to external resorption; root dilacerations and internal resorption within the distobuccal root of the first molar were also observed. These abnormalities could reflect MS-associated inflammatory mechanisms and/or vascular-related tissue alterations. Internal resorption and pulpal calcifications typically indicate pulpal pathology. Pulp stones may be true (containing dentin and lined by odontoblasts) or false (mineralized degenerative pulpal cells), and radiographic methods cannot reliably differentiate between these types [41]. The concomitant tendency toward pulp canal obliteration may be more consistent with calcific response to inflammatory stimuli.

Future investigations should also consider the reported elevation of calcium and phosphate in saliva—and potentially blood—among patients with MS, as such alterations may contribute to mineralization phenomena and could be relevant to tooth hypersensitivity and pain frequently described in MS. Finally, attention should be given to periapical changes resembling cemento-osseous dysplasia (COD). Su et al. [42] noted that approximately 70% of COD cases are closely associated with the tooth apex. COD is considered to arise within periodontal ligament tissues given its relationship to the apical foramen and the frequent histopathological appearance of cementum-like proliferation [43]. Periapical cemento-osseous dysplasia (PCOD) is a benign fibro-osseous lesion characterized by replacement of normal bone by cementum-like material and fibrous tissue, most commonly in the mandible [44, 45]. Focal cemento-osseous dysplasia (FCOD) is typically confined to the apical region of a single tooth in posterior jaw segments and has historically been interpreted as idiopathic, reactive, or hamartomatous; more recently, genetic contributions have been increasingly discussed [46]. At present, there is no evidence indicating a predisposition to PCOD in MS; however, FCOD has been reported in patients with Osler–Weber–Rendu syndrome (HHT) [47, 48], supporting a potential link between fibro-osseous changes and systemic vascular dysregulation in selected contexts.

Declarations

Funding

declaration

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Author Contribution

Conceptualization – DOK, ATS, KS, MB, JG, PPZMethodology – DOK, ATS, KS, MB, JG, PPZFormal Analysis – DOK, ATS, KS, MB, JG, PPZInvestigation – DOK, ATS, KS, MB, JG, PPZData Curation – DOK, ATS, KS, MB, HCR, JG, PPZWriting – Original Draft – DOK, ATS, KS, MB, HCR, JG, PPZWriting – Review & Editing – DOK, ATS, KS, MB, HCR, JG, PPZSupervision – DOK, ATS, KS, JGProject Administration – DOK, ATS, KS, JGFunding Acquisition – DOK, ATS, KS, MB, JG, PPZ

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Data Availability

The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request. Data are located in controlled access data storage at Medical University of Warsaw.

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Figures



Figure 1

An enlarged and erythematous incisive papilla, yellowish discoloration of the palate, and telangiectasias.



Figure 2

Marked vascularization of the vestibular oral mucosa with vascular macules.



Figure 3

Vascular macules on the vermilion border

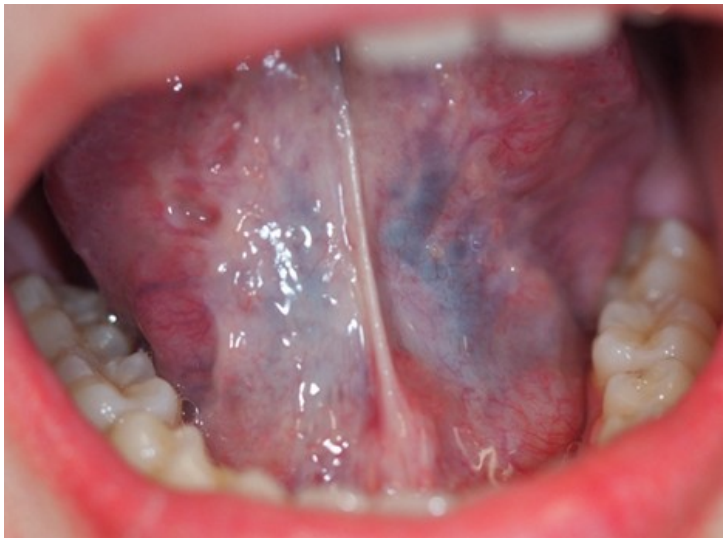


Figure 4

Lingual varices and prominent telangiectasias on the floor of the mouth and the ventral surface of the tongue.

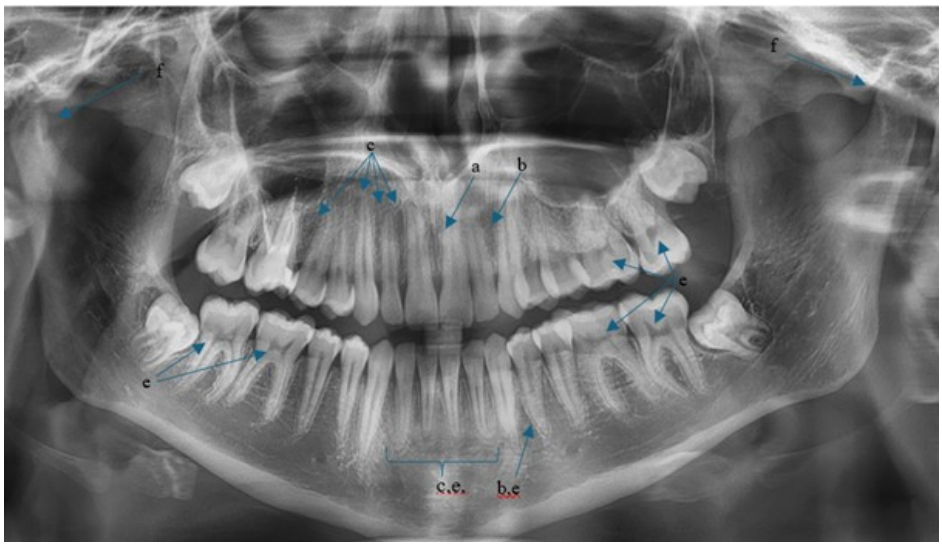


Figure 5

Panoramic radiograph (OPG) of a 17.6-year-old female patient with MS: a well-delineated incisive canal, a distinct funnel-shaped incisive fossa, and an incomplete intermaxillary suture (a); irregular periodontal ligament spaces with a wavy lamina dura (b); radiolucencies of the interproximal cancellous bone (c); widening of the periodontal ligament space (d); root canal obliteration, pulp stones (e); and condylar resorption (f).