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Stylohyoid Ligament Calcification and Its Association With Dental Diseases

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ABSTRACT

Objectives: This research aimed at investigating the relationship between calcification of the stylohyoid ligaments (SHLs) and odontogenic inflammatory events.

Methods: Cone-beam computed tomography scans of 175 patients were retrospectively assessed for presence of SHL calcifications. Frequencies of 4 types of odontogenic inflammatory events were assessed using the same scans, namely dental extractions, root canal treatments, furcation lesions, and periapical lesions. Patients were telephone-interviewed and asked about histories of tonsillectomy and head and neck trauma. Mann–Whitney *U* and binary logistic regression tests were used to assess associations between SHL calcification and mean ranks of odontogenic inflammatory events.

Results: Calcification of SHLs was detected in 71 (41%) and 58 (33%) patients on the right and left sides, respectively. Tonsillectomy and trauma were reported in 14% and 10% of the sample, respectively. Amongst all inflammatory events, root canal treatments were significantly associated with SHL calcification ($U = 2755$; $Z = -2.1$; $P = .035$). This association was independent from patient age, tonsillectomy, trauma, and other inflammatory events (odds ratio, 1.2; 95% confidence interval, 1.0–1.4; $P = .036$).

Conclusions: This study presents new evidence that intra-oral inflammatory events, particularly related to root canal treatments, have a stronger association with SHL dystrophic calcification than that associated with traditional predisposing factors (ie, old age, tonsillectomy, and trauma).

Introduction

Dystrophic calcification represents a situation in which insoluble mineral salts are deposited in soft tissues, in the absence of an increase in serum calcium and phosphate. This type of calcification has been incidentally detected in the head and neck region in more than 33% of the population.[1,2]

Although asymptomatic in most patients, dystrophic calcifications in the neck have occasionally been associated with conditions that range in their severity from mild to life-threatening.[3] Examples of those conditions include ischemic stroke, which has been linked to carotid artery calcification,[4] and Eagle syndrome, which results from elongated styloid process (SP) and/or calcified stylohyoid ligaments (SHLs).[5]

In a recent systematic review, SHL calcification, as an incidental finding, was reported in 27% of cone-beam computed tomography (CBCT) scans.[2] Whilst the exact cause of SHL calcification remains unknown, associations with predisposing factors including old age,[6-9] tonsillectomy,[10,11] and trauma to the head and neck[5,12] have been suggested.

Donders et al reported an association between the number of missing teeth and higher scores of carotid artery calcification,[13] suggesting that dental pathologies play a role in the occurrence of dystrophic calcification in the neck. The increasing prevalence of dental diseases at the global scale[14] proposes the question whether dental and periodontal diseases are associated with dystrophic calcification in nearby soft tissues in the neck.

In this retrospective radiological study, we aimed at investigating the relationship between incidentally detected SHL calcifications and radiologic manifestations of 4 types of odontogenic inflammatory events, namely missing teeth, root canal–treated teeth, furcation lesions, and periapical lesions.

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Methods

This study was part of a large project that investigated the clinical significance of stylohyoid apparatus.[15] The study arms were approved by the University of Sharjah Research Ethics Committee (approval number REC-19-12-16-01). The said committee works in accordance with the Helsinki Declaration of 1964 and its later amendments. All patients gave their consent to participate in this research.

Sample size

Sample size was calculated using the following formula: $N = \{1.962 \times p \times (1 - p)\}/ME^2$. Based on a recent systematic review that assessed the association between Eagle syndrome and trauma,[11] p was set at 12.5%. This rate represented the percentage of Eagle syndrome cases associated with dental extractions vs other forms of trauma.[11] With a margin of error set at 5% and with a 95% confidence level, the minimum sample size needed was 167.

Radiologic assessment

All CBCT scans archived in the radiology unit of the University Dental Hospital Sharjah were retrieved and used in this study. All scans had been performed previously for clinical reasons related to patients' assessment and treatment planning, such as dental implantology, orthodontic treatments, and wisdom tooth extractions. Our radiology studies were conducted using a field of view of 17 × 13 cm. All scans were done using Sirona GALILEOS® (Dentsply Sirona).

Each scan was assessed for the presence and number of 4 types of odontogenic inflammatory events, namely number of missing teeth (excluding third molars), number of root canal–treated teeth, number of furcation lesions, and number of periapical lesions. Presence or absence of calcification of SHLs, length of the right and left SPs, and length of calcification of the right and left SHL, were measured using Sirona GALAXIES software (version 1.9.5605.25519, Dentsply Sirona). Interruptions of calcification of SHLs (ie, skip areas) were included in the overall calcification length. SHL calcification patterns were not assessed. Assessment was performed in the sagittal plane and verified in both coronal and axial planes. All scans were assessed by the same oral and maxillofacial radiologist (see Figure).

Demographic data and medical history

Patients' age and sex were collected from their electronic medical records. Patients with valid telephone numbers were contacted and asked for their consent to use their archived radiographs for this research. Patients who consented were asked about self-reported histories of tonsillectomy and major trauma to the head and neck, such as car accidents, sports injuries, and falls.

Statistical analysis

SPSS Statistics package (IBM Corp. Released 2021. IBM SPSS Statistics for Macintosh, Version 28.0) was used for statistical analysis. Calcification of SHLs was assessed both as a continuous variable and as a nominal variable (present or absent). Odontogenic inflammatory events were assessed as ordinal variables. Correlations amongst those events themselves and between them and lengths of SPs and SHL calcifications were assessed using Spearman and Pearson coefficient tests, respectively. Chi-square test was used to assess associations between history of tonsillectomy and trauma and presence of SHL calcification, whilst Mann–Whitney *U* test was used for comparing the mean ranks of odontogenic inflammatory events against the SHL calcification. Binary logistic regression model was used to assess the strength of association between calcification of

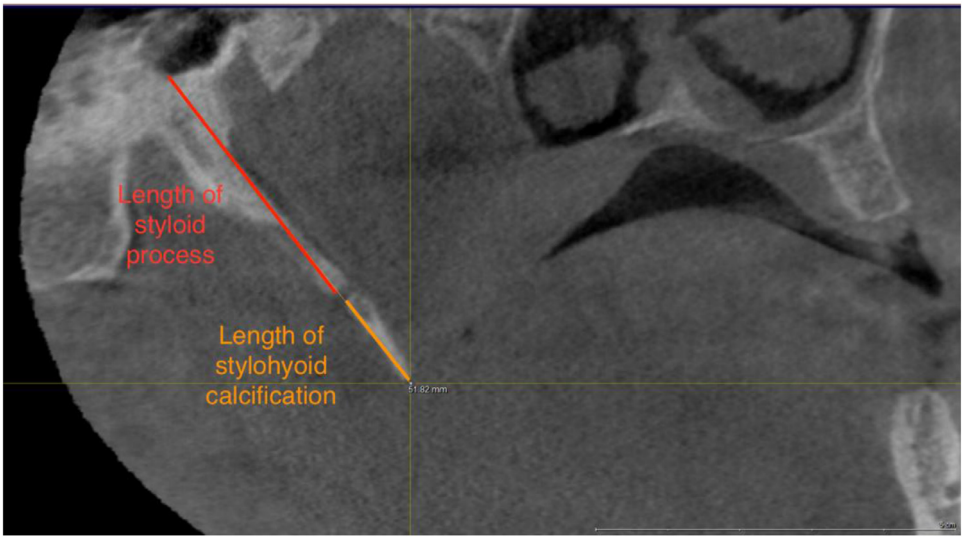

Fig – Cone beam computed tomography image in the sagittal plane showing the length of styloid process and length of stylohyoid ligament calcification.

SHLs with various types of inflammatory events, adjusted for traditional aetiologies (ie, old age, tonsillectomy, and trauma).*P* value was considered significant at <.05.

Results

The mean age of our sample was 40.7 (SD = 13.0) years, ranging from 20 to 76 years. Male to female ratio was almost 1:1, with 55% being male. Tonsillectomy and trauma to the head and neck were reported in 14% and 10% of the sample, respectively. Calcification of the SHL was detected in 71 (41%) and 58 (33%) patients on the right and left sides, respectively (Table 1).

The most common odontogenic inflammatory event was dental extractions (90%). The mean number of extracted teeth throughout the sample was almost 5 (Table 2). Table 3 shows the results of Mann–Whitney *U* test wherein higher mean ranks of odontogenic inflammatory events were seen in patients with SHL calcifications. Despite this, the only statistically significant difference in mean ranks was related to root canal treatments; patients with SHL calcification had a greater number of root canal–treated teeth ($U = 2755.00$; $Z = -2.1$; $P = .035$; Table 3).

The mean lengths of the right and left SPs were 30.2 mm (SD = 10.8) and 31.2 mm (SD = 10.5), respectively, whilst the mean lengths of calcification of the SHLs on the right and left side were 8.3 mm (SD = 11.7) and 7.2 mm (SD = 11.8) mm, respectively. Pearson correlation coefficient test showed a positive correlation between the length of SP on the right and left side ($r = 0.58$; $P < .001$) and between calcification of the SHL on the right and left sides ($r = 0.63$; $P < .001$). A significant positive correlation was also observed between the number of root canal–treated teeth and dental extractions ($r = 0.167$; $P < .05$), periapical lesions ($r = 0.525$; $P < .05$), and furcation lesions ($r = 0.153$; $P < .05$; based on Spearman correlation).

Table 1 – Demographic and medical data and radiologic findings related to odontogenic inflammatory events and calcification of SHLs.

Variable		n (%)
Sex	Male	96 (54.9)
	Female	79 (45.1)
History of tonsillectomy	No	150 (85.7)
	Yes	25 (14.3)
History of trauma	No	155 (89.6)
	Yes	18 (10.4)
Extracted teeth	Absent	18 (10.3)
	Present	157 (89.7)
Root canal–treated teeth	Absent	74 (42.3)
	Present	101 (57.7)
Furcation lesions	Absent	151 (86.3)
	Present	24 (13.7)
Periapical lesions	Absent	101 (57.7)
	Present	74 (42.3)
Calcification of the SHL on the right side	Absent	104 (59.4)
	Present	71 (40.6)
Calcification of the SHL on the left side	Absent	117 (66.9)
	Present	58 (33.1)

SHL, stylohyoid ligament.

Table 2 – Descriptive statistics of the sample's odontogenic inflammatory events.

Dental pathologies	Range	Mean	Median (SD)
Number of extracted teeth*	0–28	4.6	2 (6.1)
Number of root canal–treated teeth	2–20	2.1	1 (2.8)
Number of periapical lesions	0–8	0.9	0 (1.3)
Number of furcation lesions	0–4	0.3	0 (0.7)

* Excluding third molars.

No difference was observed between SHL calcification and patients' sex, or self-reported histories of tonsillectomy and trauma (Table 4). Patients with SHL calcification were slightly older than those without calcification: 41.7 (SD = 13.0) years vs 40.0 (SD = 13.0) years on the right side and 41.5 (SD = 13.4) years vs 40.3 (SD = 12.9) years on the left side, respectively. However, the difference between means was not statistically significant ($P = .393$ and $P = .581$ on the right and left sides, respectively; based on independent-samples *t* test). A binary logistic regression model showed that root canal treatment is a significant predictor of calcification of the SHL that is independent from patient age, histories of trauma and tonsillectomy, and other dental pathologies (Table 5).

Discussion

Dystrophic calcifications of the SHLs, with or without elongation of the SP, have been linked to nonspecific symptoms of dizziness, dysphagia, and pain in the head and neck region in what is collectively known as Eagle syndrome.[16]

Causes of calcifications in the SHLs have not been fully established. However, associations with various inflammatory events have been reported. Those include old age,[6-9] tonsillectomy,[17-19] and trauma to the head and neck.[11,16] Our study showed that—amongst our sample—none of those 3 predisposing factors had an association with the presence of SHL calcification.

Since dental pathologies occur at close anatomic proximity to SHLs, we investigated potential associations between intra-oral signs of odontogenic inflammatory events and SHL calcifications, with the assumption that dental inflammatory foci have an impact on the nearby stylohyoid apparatus.

Our findings showed that SHL calcifications were associated with greater number of dental extractions and periapical lesions, but those associations were not statistically significant. A significant association, however, was found between those calcifications and the number of root canal–treated teeth.

Compared to dental extractions, root canal treatments are complex and carry greater inflammatory impact on adjacent tissues for 2 reasons. First, root canal treatments are done to treat necrotic or intensely inflamed pulpal and periapical tissues, both of which represent inflammatory foci in the jaws. Second, completing a root canal treatment necessitates multiple visits, with the potential for procedural inflammation during and after each visit.

Several studies showed higher levels of proinflammatory cytokines tumour necrosis factor, interleukin (IL)-6, and IL-1B

Table 3 – Comparison of the mean ranks of the dental pathologies amongst patients with and without calcification of the SHLs.

Dental pathology	Presence of calcification	Right SHL		Left SHL	
		Mean rank	U value (*P* value*)	Mean rank	U value (*P* value*)
Extracted teeth	Not detected	86.03	3487.00 (.529)	87.33	3314.50 (.802)
	Detected	90.89		89.35	
Root canal–treated teeth	Not detected	83.99	3275.00 (.186)	82.55	2755.00 (.035)
	Detected	93.87		99.00	
Furcation lesions	Not detected	88.48	3642.50 (.801)	87.86	3376.50 (.930)
	Detected	87.30		88.28	
Periapical lesions	Not detected	86.53	3539.00 (.603)	86.68	3239.00 (.585)
	Detected	90.15		90.66	

* Based on Mann–Whitney *U* test.SHL, stylohyoid ligament.

Table 4 – Association between SHL calcification and self-reported history of tonsillectomy and trauma

Variables		Calcification of the SHL on the right side		*P* value*	Calcification of the SHL on the left side		*P* value*
		Absent, n (%)	Present, n (%)		Absent, n (%)	Present, n (%)	
Sex	Male	60 (62.5)	36 (37.5)	.362	67 (69.8)	29 (30.2)	.363
	Female	44 (55.7)	35 (44.3)		50 (63.3)	29 (36.7)	
History of tonsillectomy	No	87 (58.0)	63 (42.0)	.346	99 (66.0)	51 (34.0)	.555
	Yes	17 (68.0)	8 (32.0)		18 (72.0)	7 (28.0)	
History of trauma	No	92 (59.4)	63 (40.6)	.756	103 (66.5)	52 (33.5)	.985
	Yes	10 (55.6)	8 (44.4)		12 (66.7)	6 (33.3)	

* Based on chi-square test.SHL, stylohyoid ligament.

during and after root canal treatments.[20-22] The same cytokines have similarly been associated with dystrophic calcification.[23] The mutual presence of proinflammatory cytokines in both situations support the assumption that intraoral inflammatory events, which result from dental pathologies and advanced dental treatments, are associated with SHL calcifications.

More than half of our sample (58%) had at least one root canal–treated tooth. This prevalence rate is similar to recently published reports[24,25] but higher than that reported regionally[26] and globally.[27,28] The higher rates of root canal –treated teeth amongst our sample can be explained by nature of our patients themselves, the majority of whom are disadvantaged persons who attended our teaching institute (study site) for free dental treatments. Previous studies have shown higher rates of dental diseases and pulpal pathologies amongst people of lower socioeconomic status.[29,30] The positive correlation amongst root canal treatments, dental extractions, periapical lesions, and furcation lesions implies that root canal treatment is not singularly high, but represents the sample's overall poor dental health.

Prevalence of SHL calcification has been reported to range from 2% to 68%.[1,31-34] The prevalence in our study was 33% and 41% in the right and left sides, respectively, which is similar to that reported elsewhere.[2,34]

The wide range of prevalence rates reported in the literature can be explained by the tool used to assess SHL calcification. In most studies, 2-dimensional panoramic radiographs were used, which—although conveniently available—are limited by perspective distortion in which anatomic structures

Table 5 – Binary logistic regression model assessing the strength of association between root canal treatment and presence of calcification of the SHL on the left side.

Variable	B	SE	Sig.	Exp(B)	95% CI	
					Lower	Upper
Age	−0.003	0.015	0.836	0.997	0.967	1.027
History of trauma	−0.076	0.553	0.890	0.927	0.313	2.741
History of tonsillectomy	−0.147	0.488	0.764	0.864	0.332	2.249
Extracted teeth	0.018	0.031	0.569	1.018	0.958	1.081
Root canal–treated teeth	0.154	0.073	0.036	1.167	1.010	1.347
Furcation lesions	0.095	0.230	0.680	1.100	0.700	1.727
Periapical lesions	−0.147	0.154	0.340	0.864	0.639	1.167

SHL, stylohyoid ligament.

that are deep in the medial and posterior directions are not visible in their true dimensions or not visible at all. Three-dimensional CBCT scans overcome this limitation and provide an accurate assessment of the stylohyoid apparatus that is free from any dimensional distortion.[35,36] Hence, we consider the prevalence rate reported here to be of higher accuracy than that reported using 2-dimensional radiographs.

This study is the first in showing an association between root canal treatments and SHL calcifications. Our results are strengthened by the inclusion of old age, tonsillectomy, and trauma as confounding factors that have been adjusted for. Although the association reported here does not establish a causal relationship between intra-oral inflammatory events and SHL calcifications, those results form grounds for future prospective cohort studies aimed at investigating chronological causative associations.

Despite those findings, our study is limited by the following: First, tonsillectomy and trauma to the head and neck were self-reported without objectively verifying patients' recalls using medical records. Second, imbalances in serum minerals were not assessed as a possible cause of SHL calcification. Third, the association reported between root canal treatments and SHL calcification is based on the mere radiographic occurrence of root canal treatments, without assessing the effectiveness of those treatments.

Conflict of interest

None disclosed.

REFERENCES

1. Yalcin ED, Ararat E. Prevalence of soft tissue calcifications in the head and neck region: a cone-beam computed tomography study. Niger J Clin Pract 2020;23(6):759–63.
2. Dief S, Veitz-Keenan A, Amintavakoli N, McGowan R. A systematic review on incidental findings in cone beam computed tomography (CBCT) scans. Dentomaxillofac Radiol 2019;48(7):20180396.
3. Gupta A, Aggrawal A, Setia P. A rare fatality due to calcified stylohyoid ligament (Eagle syndrome). Med Leg J 2017;85(2):103–4.
4. Wu XH, Chen XY, Wang LJ, Wong KS. Intracranial artery calcification and its clinical significance. J Clin Neurol 2016;12(3):253–61.
5. Gonzalez-Garcia N, Porta-Etessam J, Garcia-Azorin D. Eagle syndrome: toward a clinical delimitation. Neurologia (Engl Ed) 2021;36(6):412–7.
6. Bruno G, De Stefani A, Barone M, Costa G, Saccomanno S, Gracco A. The validity of panoramic radiograph as a diagnostic method for elongated styloid process: a systematic review. Cranio 2019:1–8.
7. AlZarea BK. Prevalence and pattern of the elongated styloid process among geriatric patients in Saudi Arabia. Clin Interv Aging 2017;12:611–7.
8. Shaik MA, Naheeda Kaleem SM, Wahab A, Hameed S. Prevalence of elongated styloid process in Saudi population of Aseer region. Eur J Dent 2013;7(4):449–54.
9. Sudhakara Reddy R, Sai Kiran C, Sai Madhavi N, Raghavendra MN, Satish A. Prevalence of elongation and calcification patterns of elongated styloid process in south India. J Clin Exp Dent 2013;5(1):30.
10. Al-Khateeb TH, al Dajani TM, Al Jamal GA. Mineralization of the stylohyoid ligament complex in a Jordanian sample: a clinicoradiographic study. J Oral Maxillofac Surg 2010;68(6):1242–51.
11. Saccomanno S, Quinzi V, D'Andrea N, Albani A, Coceani Paskay L, Marzo G. Traumatic events and Eagle syndrome: is there any correlation? A systematic review. Healthcare (Basel) 2021;9(7):825.
12. Maki D, Okami K, Ebisumoto K, Sakai A. Chronological progression of an enlarged styloid process: a case report of Eagle syndrome. Case Rep Otolaryngol 2018;2018:9207264.
13. Donders HCM, IJzerman LM, Soffner M, van't Hof AWJ, Loos BG, de Lange J. Elevated coronary artery calcium scores are associated with tooth loss. PLoS One 2020;15(12):e0243232.
14. Kassebaum NJ, Smith AGC, Bernabé E, et al. Global, regional, and national prevalence, incidence, and disability-adjusted life years for oral conditions for 195 countries, 1990–2015: a systematic analysis for the global burden of diseases, injuries, and risk factors. J Dent Res 2017;96(4):380–7.
15. Al-Amad SH, Al Bayatti S, Alshamsi HA. The effect of styloid process medial angulation on Eagle's syndrome's self-reported symptoms: a preliminary cross-sectional study. Oral Surg Oral Med Oral Pathol Oral Radiol 2021;132(1):52–6.
16. Piagkou M, Anagnostopoulou S, Kouladouros K, Piagkos G. Eagle's syndrome: a review of the literature. Clin Anat 2009;22(5):545–58.
17. Leong SC, Karkos PD, Papouliakos SM, Apostolidou MT. Unusual complications of tonsillectomy: a systematic review. Am J Otolaryngol 2007;28(6):419–22.
18. Waters CM, Ho S, Luginbuhl A, Curry JM, Cognetti DM. Surgical management of stylohyoid pain (Eagle's) syndrome: a 5-year experience. Ann Otol Rhinol Laryngol 2019;128(3):220–6.
19. Bafaqeeh SA. Eagle syndrome: classic and carotid artery types. J Otolaryngol 2000;29(2):88–94.
20. Virdee SS, Butt K, Grant M, Camilleri J, Cooper PR, Tomson PL. A systematic review of methods used to sample and analyse periradicular tissue fluid during root canal treatment. Int Endod J 2019;52(8):1108–27.
21. Lu Y, Liu Z, Huang J, Liu C. Therapeutic effect of one-time root canal treatment for irreversible pulpitis. J Int Med Res 2019;48(2):1–11.
22. Sousa ELR, Martinho FC, Leite FRM, Nascimento GG, Gomes BPFA. Macrophage cell activation with acute apical abscess contents determined by interleukin-1 Beta and tumor necrosis factor alpha production. J Endod 2014;40(11):1752–7.
23. Dellegrottaglie S, Sanz J, Rajagopalan S. Molecular determinants of vascular calcification: a bench to bedside view. Curr Mol Med 2006;6(5):515–24.
24. Bürklein S, Schäfer E, Jöhren HP, Donnermeyer D. Quality of root canal fillings and prevalence of apical radiolucencies in a German population: a CBCT analysis. Clin Oral Investig 2020;24(3):1217–27.
25. Tibúrcio-Machado CS, Michelon C, Zanatta FB, Gomes MS, Marin JA, Bier CA. The global prevalence of apical periodontitis: a systematic review and meta-analysis. Int Endod J 2021;54(5):712–35.
26. Al-Nazhan SA, Alsaeed SA, Al-Attas HA, Dohaithem AJ, Al-Serhan MS, Al-Maflehi NS. Prevalence of apical periodontitis and quality of root canal treatment in an adult Saudi population. Saudi Med J 2017;38(4):413–21.
27. Pak JG, Fayazi S, White SN. Prevalence of periapical radiolucency and root canal treatment: a systematic review of cross-sectional studies. J Endod 2012;38(9):1170–6.
28. Meirinhos J, Martins JNR, Pereira B, et al. Prevalence of apical periodontitis and its association with previous root canal treatment, root canal filling length and type of coronal restoration - a cross-sectional study. Int Endod J 2020;53(4):573–84.

29. Schwendicke F, Dörfer CE, Schlattmann P, Foster Page L, Thomson WM, Paris S. Socioeconomic inequality and caries: a systematic review and meta-analysis. J Dent Res 2015;94(1):10–8.
30. Mejia GC, Elani HW, Harper S, et al. Socioeconomic status, oral health and dental disease in Australia, Canada, New Zealand and the United States. BMC Oral Health 2018;18(1) 176–3.
31. Alpoz E, Akar GC, Celik S, Govsa F, Lomcali G. Prevalence and pattern of stylohyoid chain complex patterns detected by panoramic radiographs among Turkish population. Surg Radiol Anat 2014;36(1):39–46.
32. MacDonald-Jankowski DS. Calcification of the stylohyoid complex in Londoners and Hong Kong Chinese. Dentomaxillofac Radiol 2001;30(1):35–9.
33. Oztas B, Orhan K. Investigation of the incidence of stylohyoid ligament calcifications with panoramic radiographs. J Investig Clin Dent 2012;3(1):30–5.
34. Guimarães A, Pozza D, Guimarães A. Prevalence of morphological and structural changes in the stylohyoid chain. J Clin Exp Dent 2020;12(11):e1027–32.
35. Benninger B, Peterson A, Cook V. Assessing validity of actual tooth height and width from cone beam images of cadavers with subsequent dissection to aid oral surgery. J Oral Maxillofac Surg 2012;70(2):302–6.
36. Donmez M, Okumus O, Pekiner FN. Cone beam computed tomographic evaluation of styloid process: a retrospective study of 1000 patients. Eur J Dent 2017;11(2):210–5.