J Oral Med Oral Surg 2023;29:2
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https://doi.org/10.1051/mbcb/2022035

https://www.jomos.org

JOMOS
Journal of Oral Medicine and Oral Surgery

Original Research Article

Acute periapical dental abscesses and increased risk for extracranial carotid artery aneurysms

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(Received: 2 November 2022, accepted: 12 December 2022)

Keywords:
Aneurysm / apical abscess / carotid artery / endodontics / periapical abscess

Abstract – Aim: To assess the prevalence of acute periapical abscesses (PAs) in patients with a history of extracranial carotid artery aneurysms (ECAA). **Materials and methods**: History of acute PAs and ECAA diagnosis was retrieved by searching the appropriate query in the database. All cases were diagnosed for acute PAs by calibrated dentists for patients admitted to urgent care. The odds ratio (OR) for the prevalence of acute PAs and its association with history of ECAA were then calculated. **Results**: The prevalence of acute PAs in patients with a history ECAA was significantly higher as compared to the general hospital patient population ($p < 0.0001$). The OR was 5.5. Females were more affected than males by 1.7 folds ($p < 0.001$). Whites were more affected than African Americans by 1.6 folds ($p < 0.00001$). **Conclusions**: The high prevalence of acute PAs in patients with a history of ECAA may suggest an association between these two conditions warranting a thorough medical examination.

Introduction

Aneurysm is a significant dilation of an artery of at least 1.5 times the adjacent normal segment. It is found in up to 5% of patients undergoing coronary angiography and is caused by weakening of the arterial wall [1]. Aneurysms of the extracranial carotid artery (ECCA) are infrequent but can pose a serious risk to the patient when ruptured [2–4]. Rupture of an ECAA can be life-threatening and require immediate surgical care. It has been reported that the incidence may reach up to 5% of all carotid artery surgeries [5].

Attigah *et al.* [6], described five types of morphological classifications based on the location of the ECAA along the carotid artery. Type I is an isolated and short aneurysm of the internal carotid artery superior to the carotid bulb; Type II is a long aneurysm of the internal carotid artery, ranging from the carotid bulb up to the line of Blais- dell; Type III is an aneurysm of the proximal internal carotid artery and the carotid bifurcation; Type IV is an Aneurysm involving the common carotid artery and internal carotid artery, extending far more distally and proximally; and Type V is an Isolated aneurysms of the common carotid artery.

The etiology of ECAA is controversial. Atherosclerotic degeneration, traumatic injury, radiation, and local infection have been suggested as potential causes [2–5,7]. Increasing evidence in the literature points to the possible association between infection and ECAA [8–11]. Several reports implicated infections associated with the oro-dental cavity [12,13].

A dental infection of clinical concern is acute periapical abscess (PA). Acute PA is an inflammatory reaction to dental pulp infection and necrosis characterized by rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation and swelling of the associated tissues [14]. Periapical abscesses (PAs) present a serious health risk to patients and require expeditious and efficient treatment. In the United States, more than 400,000 emergency patient visits were due to either pulpal or periapical dental infections resulting in medical charges totaling more than $160 million [15]. More than 60,000 hospitalizations were primarily attributed to acute PAs [16].

To date, there are no reports in the literature on the possible association between acute PAs and ECAAs. Therefore, the aim of this cross-sectional, hospital-based study was to assess the prevalence of acute PAs in patients with a history of ECAA.

Materials and methods

The University of Florida (UF) Integrated Data (IDR) i2b2, supported by the National Institute of Health (NIH) and the UF Health Office of the Chief Data Officer, for the period from October 2015 to May 2022 was used. The study was in

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Table I. Demographics of the patient population studied.

	ECAAs (n = 547)	PAs (n = 7763)	ECAAs & PAs (n = 18)	Total hospital patients (n = 1,314,925)
Males	184 (33.6%)	3,441 (44.3%)	5 (27.8%)	607,571 (46.2%)
Females	363 (66.4%)	4,322 (55.7)	13 (72.2%)	707,354 (53.8%)
Whites	425 (77.7%)	4,398 (67.3%)	12 (66.7%)	623,741 (47.4%)
African Americans	88 (16.1%)	2,730 (19.5%)	6 (33.3%)	134,327 (10.2%)
Other Ethnicities	34 (6.2%)	6,35 (13.2%)	0 (0%)	556,857 (42.4%)
Adults (>18 years)	547 (100%)	6,748 (13.2%)	18 (100%)	1,131,087 (86%)
Children (<18 years)	0 (0%)	1,015 (86.8%)	0 (0%)	183,838 (14%)

ECAAs = Extracranial Carotid Artery Aneurysms; PAs = Periapical Abscesses.

compliance with the UF Institutional Review Board (IRB), ethics, and privacy rules.

Data aggregate from inpatients and outpatients visiting the UF Health Center were recorded using the electronic patient record Epic (epic.com). Epic is the preferred electronic medical record system used by more than 250 health care organizations in the USA. More than 50% of the USA population have their medical records in an Epic system.

The different diagnoses were coded using the international coding systems ICD 10. The ICD system requires that all HIPAA-covered entities implement the ICD-10-CM diagnosis code set. Medicaid is a HIPAA entity, as are medical and dental clinics. The ICD coding system has also been adopted by the American Dental Association.

The patient population analyzed was mixed, presenting with different disease conditions including acute PAs without sinus (ICD 10 K04.7). All cases were diagnosed for acute PAs by calibrated dentists in a hospital setting for patients admitted to urgent care. Diagnosis was made based on clinical examination and imaging data confirming the diagnoses of acute PAs without sinus tract.

Inclusion criteria included the corresponding diagnostic code for PAs without sinus (ICD 10 K04.7) and for carotid aneurysm (I72.0). There were no exclusion criteria since all codes were computerized, and specific diagnoses of acute PAs in the total hospital patient population were searched using the appropriate ICD 10 code. History of ECAA diagnosis was retrieved by searching the appropriate query in the database.

Cases of chronic PA with sinus tract or symptomatic and asymptomatic apical periodontitis or combined PA and periodontal disease were not included. as they have a different ICD10 codes.

The odds ratio (OR) for the prevalence of acute PAs and its association with history of ECAA were calculated with a 95% confidence interval and the statistical difference between the study groups was assessed using MedCalc software Version 20.110 (MedCalc Software Ltd. Odds ratio calculator. https://www.medcalc.org/calc/odds_ratio.php). A standard normal deviate (z-value) was calculated as follows: ln(OR)/SE{ln (OR)}. The p-value was the area of the normal distribution that falls outside $\pm z$ [17]. A value of $p < 0.05$ was considered statistically significant.

Table II. Prevalence of acute periapical abscesses (PAs) in extracranial carotid artery aneurysms (ECAAs) patients. OR = odds ratio; CI = confidential interval.

	ECAAs	Total hospital patients
Number of acute PAs	18	7,763
Number of patients	547	1,314,925
OR	5.5	
95% CI	3.48 to 8.91	
p-value	<0.0001	

Results

The total hospital patient population studied was 1,314,925; 46.2% males and 53.8%% females (Tab. I). Out of the total hospital patient population, 547 (0.04%) individuals were diagnosed with a history of ECAA. Females were more affected than males by 2 folds. Whites were more affected than African Americans by almost 5 folds. Whites were more affected than African Americans combined with other ethnicities by circa 3.5 folds. Patients over the age of 18 were exclusively affected (Tab. I). The average age of the patients in the PA group was 42 yrs.

Out of the total hospital patient population, 77,630 (0.59%) were diagnosed with acute PAs. Females were more affected than males by 1.7 folds. The chi-square statistic was 10.9754. The p-value was 0.000923; Significant at $p < 0.05$. Whites were more affected than African Americans by 1.6 folds. The chi-square statistic is 2034.1494. The p-value was 0.000009; Significant at $p < 0.05$.

Out of the total number of patients with a history of ECAA, 18 patients (3.29%) had PAs (Tab. II). The OR was 5.5 and the difference in prevalence was statistically significant ($p < 0.0001$). White females were significantly more affected by both ECAA and PA ($p < 0.0001$).

Discussion

The results of this cross-sectional study show that, overall, the OR for prevalence of acute PAs is significantly higher in patients with a history of ECAA as compared to patients without this condition. Calcified atherosclerotic plaques are often visualized incidentally on extra-oral dental imaging [18]. Due to the relatively close proximity of the carotid arteries to the dental alveolar ridge, an inflammatory involvement from dental infection may have a direct or indirect effect on the wall of the carotid artery [19].

Although not common, infection triggering an inflammatory process can be associated with ECAA [19,20]. Primary mycotic or infected aneurysms of the aorta represent 0.65% to 1.3% of all aortic aneurysms [21]. Most microorganisms cultured from blood and aortic specimens were staphylococcal species while other microorganisms such as streptococcus species, salmonella, and syphilis were less frequent [21]. Fungal infections have also been documented but are rare [21].

It has been suggested that the inflammatory process weakens the arterial wall leading to aneurysmal degeneration [22]. The role of inflammation in atherosclerosis has also been demonstrated in relation to infection originating from the oral cavity [12,13,23,24]. It has been proposed that periodontitis enhances the levels of systemic inflammatory mediators that pose risk factors for atherosclerotic diseases [24]. An inflammatory cell infiltrate, neovascularization, and production and activation of various proteases and cytokines can contribute to the development of aneurysm. Although the underlying mechanisms has not been fully clarified, there is a strong clinical association between tobacco smoking and aneurysm development [25].

The present study is the first report on the possible association between a dental infection and an aneurysm of the carotid artery. However, several limitations should be considered. First, this is a cross-sectional study and therefore does not necessarily indicate causality. Second, chronic endodontic infections, periodontal pockets and other periodontal related components could further contribute to the development of ECAA. However, these conditions were not included in our study. Third, socio-economic reasons may influence the decision of certain patients when seeking a location for their medical and dental care. Therefore, the prevalence of PAs in this study may also reflect social-economic disparities. However, no questionnaire was used to assess the socio-economic status of these patients. Fourth, out of the total number of patients with a history of ECAA, only 18 patients had PAs. This sample size should be taken into consideration when extrapolating the results of this study to the general population. Although sample size is relatively small, the OR was high and the difference in prevalence was statistically significant. Fifth, the study design did not allow for simultaneous multivariant analysis of all possible covariates.

Conclusion

The prevalence of acute PAs in patients with a history ECAA appears significantly higher as compared to patients without a history of ECAA. Although causality cannot be attributed to cross sectional studies, the high prevalence of acute PAs in patients with ECAA warrants further studies to fully elucidate this association.

Author contribution statement

Joseph Katz: Conceptualization, Methodology, Investigation, Reviewing and Editing. Ilan Rotstein: Conceptualization, Methodology, Investigation, Writing original draft.

Conflict of interest

Authors declare no conflict of interest.

Informed consent

This study did not require informed consent.

Ethics approval and informed consent

All the procedures performed in our study followed the ethical standards of the institutional research committee and Privacy rules for research on IRB approved de-identified data sets. Approval date: April 6, 2021.

Funding

This research did not receive any specific funding.

References

1. Kawsara A, Núñez Gil IJ, Alqahtani F, Moreland J, Rihal CS, *et al.* Management of coronary artery aneurysms. JACC Cardiovasc Interv 2018;11:1211–1223.
2. Shimizu T, Sakakura Y, Yamagiwa M, Hori M, Yuasa H, *et al.* Aneurysm of the extracranial carotid artery. Arch Otolaryngol Head Neck Surg 1986;112:203–206.
3. Zwolak RM, Whitehouse WM Jr, Knake JE, Bernfeld BD, Zelenock GB, *et al.* Atherosclerotic extracranial carotid artery aneurysms. J Vasc Surg 1984;1:415–422.
4. Chen Z, Chen L, Zhang J, Chen Y, Liu C, *et al.* Management of extracranial carotid artery aneurysms: A 6-year case series. Med Sci Monit 2019;25:4933–4940.
5. Garg K, Rockman CB, Lee V, Maldonado TS, Jacobowitz GR, *et al.* Presentation and management of carotid artery aneurysms and pseudoaneurysms. J Vasc Surg 2012;55:1618–1622.
6. Attigah N, Külkens S, Zausig N, Hansmann J, Ringleb P, *et al.* Surgical therapy of extracranial carotid artery aneurysms: long-term results over a 24-year period. Eur J Vasc Endovasc Surg 2009;37:127–133.
7. Choi E, Gwon JG, Kwon SU, Lee DH, Kwon TW, *et al.* Management strategy for extracranial carotid artery aneurysms: A single-center experience. Medicine (Baltimore). 2022;101:e29327.
8. Sundarrajan C, Isa SA, Caruso JP, Ban VS, Shah GB, *et al.* Treatment of large infectious extracranial carotid artery pseudoaneurysms in children: a systematic review of the literature. Childs Nerv Syst 2021;37:1461–1470.

9. Tsai TC, Barot N, Dalman R, Mihm F. Combined endovascular and open operative approach for mycotic carotid aneurysm. J Vasc Surg 2010;51:1514–1516.
10. Martens S, Beelen R, Degrieck I. Mycotic aneurysm of the extracranial carotid artery: a case report and review of the literature. Acta Chir Belg 2011;111:97–99.
11. Lui DH, Patel S, Khurram R, Joffe M, Constantinou J, *et al.* Mycotic internal carotid artery pseudoaneurysm secondary to mycobacterium tuberculosis. J Vasc Surg Cases Innov Tech 2022;8:251–255.
12. Abularrage CJ, Crawford RS, Durand ML, LaMuraglia GM. Extracranial infected carotid artery aneurysm. J Vasc Surg 2009;50:1484–1486.
13. Figuero E, Lindahl C, Marín MJ, Renvert S, Herrera D, Ohlsson O, *et al.* Quantification of periodontal pathogens in vascular, blood, and subgingival samples from patients with peripheral arterial disease or abdominal aortic aneurysms. J Periodontol 2014;85:1182–1193.
14. Glossary of endodontic terms. American Association of Endodontists, 10th edn., Chicago IL, 2020, p. 4.
15. Nalliah RP, Allareddy V, Elangovan S, Karimbux N, Lee MK, *et al.* Hospital emergency department visits attributed to pulpal and periapical disease in the United States in 2006. J Endod 2011;37:6–9.
16. Shah AC, Leong KK, Lee MK, Allareddy V. Outcomes of hospitalizations attributed to periapical abscess from 2000 to 2008: a longitudinal trend analysis. J Endod 2013;39:1104–1110.
17. Sheskin DJ. Handbook of parametric and nonparametric statistical procedures. 5th edn., Chapman & Hall /CRC, Boca Raton, FL, 2011, pp. 761–804.
18. Couto Souza PH, Berti-Couto SA, Majewski CNM, da Silva IC, Donaduzzi LC, *et al.* Association of calcified carotid artery plaque in panoramic images and diagonal earlobe crease. Dentomaxillofac Radiol 2019;48:20170256.
19. Stephen E, Sridhar R, Pradhan NR, Thomas SV, Narayan RL, *et al.* Tuberculous aneurysm of extracranial carotid artery. Eur J Vasc Endovasc Surg 2008;35:9–10.
20. Patel S, Sharma AK, Meena D, Garg PK, Tiwari S, *et al.* Extracranial carotid artery pseudoaneurysm due to Mycobacterium tuberculosis. Asian Cardiovasc Thorac Ann 2020;28:279–281.
21. Calero A, Illig KA. Overview of aortic aneurysm management in the endovascular era. Semin Vasc Surg 2016;29:3–17.
22. Long R, Guzman R, Greenberg H, Safneck J, Hershfield E. Tuberculous mycotic aneurysm of the aorta: review of published medical and surgical experience. Chest 1999;115:522–531.
23. Katz J, Marc H, Porter S, Ruskin J. Inflammation, periodontitis, and coronary heart disease. Lancet 2001;358:1998.
24. Schenkein HA, Papapanou PN, Genco R, Sanz M. Mechanisms underlying the association between periodontitis and atherosclerotic disease. Periodontology 2000 2020;83:90–106.
25. Sakalihasan N, Limet R, Defawe OD. Abdominal aortic aneurysm. Lancet 2005;365:1577–1589.