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Maxillofacial Surgeons Beware: Some American Heart Association "Moderate Risk" Patients Develop Endocarditis After Exodontia

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Given the absence of randomized controlled trials the scientific validity of the association between the performance of invasive dental procedures (IDP) and subsequent development of infectious endocarditis (IE) has been debated for more than 50 years. In 2007, the American Heart Association (AHA) restricted its antibiotic prophylaxis (AP) recommendations to only a very few cardiac abnormalities (eg, prosthetic cardiac valve) categorizing patients having same as being at high risk for IE and in need of medication.[1] Given these precepts it is critically important that oral and maxillofacial surgeons be keenly aware of the findings of a recently published study which demonstrated a temporal relationship between the development of IE and the performance of IDP (ie, exodontia) and that this enhanced risk of cardiac infection affected not only high risk patients but also those previously deemed moderate risk by the AHA.[2]

In a recent issue of the Journal of the American College of Cardiology, Thornhill et al[2] described how they extracted data from the commercial/Medicare Supplemental prescription database and the IBM MarketScan Dental databases to perform a case-crossover analysis and cohort study to determine if there was an association between the development of IE and performance of IDP as well as the efficacy of AP to reduce IE. Identified among the almost 8 million records of hospitalized Americans over age 18 were 3,774 patients who developed IE after IDP. Analysis of this cohort using AHA categorization demonstrated that 34% were high risk (eg, prosthetic heart valve, unrepaired congenital heart defect), 22% moderate risk (eg, rheumatic heart disease, hypertrophic cardiomyopathy), and 44% low/unknown risk.

The results of this observational study (case-crossover analysis) demonstrated that among individuals described by AHA at high IE risk, there was a significant (odds ratio [OR] = 11.08; 95% confidence interval [CI], 7.34 to 16.74; $P < .0001$) temporal association between IE and dental extractions in the preceding 4 weeks. AP was associated with a significant reduction in IE incidence following IDP (OR = 0.49; 95% CI, 0.29 to 0.85; $P = .01$). In addition, there was a significant positive association between IE and dental extractions in those at moderate IE risk (OR = 2.05; 95% CI, 1.42 to 2.95; $P = .003$).[2]

The specific rationale for providing this Perspectives are the 831 patients who developed IE after IDP yet were classified by the AHA as being only at moderate risk and not in need of AP. Of note, these moderate risk patients evidenced less florid symptoms of IE and were not diagnosed with the cardiac infection for approximately 120 days post extraction, whereas those in the high risk group had a more virulent onset of IE and were identified as being afflicted with the disorder within 30 days. These findings, specifically the development of IE encountered by patients with a cardiac condition deemed by the AHA

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as of only moderate risk, should not be ignored by members of our profession.

The results of Thornhill's study are somewhat perplexing given that a large percentage of AHA-classified moderate risk patients not provided with AP went on to develop IE after IDP. Further compounding the complexity of this issue is that the efficacy of AP to prevent IE among moderate risk patients approached but did not quite attain statistical significance.

So, what do we do now? We suggest that until the AHA revises its AP guidelines, maxillofacial surgeons adhering to them should also recommend to their patients that they follow-up with their primary care physician or cardiologist to have their cardiac health monitored for 4 months after IDP. Our allusion to the possible benefit of AP for moderate risk patients (and our strong belief that they be prescribed) arises from a Commentary that accompanied the publication written by Bach.[3] Specifically, Bach noted that the study evaluated whether AP affected the likelihood of the patient developing IE rather than the medication's effectiveness on the severity of resultant cardiac disease. Thus, the study results seem to support, and we fully agree with, the need to revise the AHA's guidelines to include AP for all patients at high lifetime risk of developing IE rather than restricting AP only to those at the highest risk of an adverse outcome. Bach's analysis of Thornhill's data is similar to ours and recognizes that the study's results relative to AP effectiveness in preventing IE among moderate risk patients having IDP did not quite reach statistical significance, but he attributed this outcome to a function of the study's power. Furthermore, he opined that there was an absence of data supporting an association between AP and adverse drug reactions or antibiotic resistance, therefore suggesting that revision of the AHA's guidelines to include AP for all patients at increased risk of IE may be appropriate.[3]

Finally, one more admonition. Sadly, Thornhill's results also demonstrated that large numbers of dentists are not providing even their high risk patients with the AHA's recommended prescriptions for AP. And while it is true that during the study's period of observation only a small percentage of these unprotected patients went on to develop IE, this void, if continuous, may over time represent a significant public health problem. This problematic issue therefore demands additional continuing educational efforts by our various dental organizations to impress on their membership the need to appropriately prescribe AP, after carefully obtaining a drug allergy history, in order to avoid adverse medication reactions.

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