The association between periodontal disease and peritonsillar infection: A prospective study

CHRISTOS GEORGALAS, MD, MRCS, **JEEVE KANAGALINGAM,** MA, MRCS, DLO, **AZIDA ZAINAL,** MBBS, **HAMID AHMED,** FDS, **ARVIND SINGH,** MB BS, and **KALPESH S. PATEL,** BSC, FRCS(ORL), London, United Kingdom

OBJECTIVE: **To study the relationship between periodontal status and peritonsillar disease/recurrent tonsillitis.**

STUDY DESIGN AND SETTING: **A total of 158 patients presenting over a 3-year period with peritonsillar abscess (PTA) confirmed by needle aspiration and a control group of 112 patients booked for elective tonsillectomy for recurrent tonsillitis (RT) were examined in terms of their periodontal status using the WHO Community Periodontal Index of Treatment Needs (CPITN).**

RESULTS: **The mean CPITN index was 2.81 (Standard Deviation (SD), 1.10) in patients with PTA and 1.41 (SD, 0.92) in patients with RT. One hundred seven of 158 patients with PTA had significant periodontal pathology (CPITN, 3 or 4) compared with 12 of 112 patients with RT. These differences were statistically significant.**

CONCLUSION: **Patients with peritonsillar abscess had an increased prevalence of periodontal disease as compared with patients with recurrent tonsillitis.**

SIGNIFICANCE: **There is a need to further explore this correlation and determine its nature, although it could be the result of common pathogenic factors, a causal relation cannot be excluded.** (Otolaryngol Head Neck Surg 2002;126:91-4.)

Peritonsillar abscess (PTA) is probably the most common deep neck infection and has been associated with significant morbidity and occasional mortality. Indeed, George Washington, America's first president is thought to have succumbed to this condition.

From the Department of Otolaryngology–Head and Neck Surgery (Drs Georgalas, Kanagalingam, Zainal, Singh, and Patel) and the Department of Oral Surgery (Dr Ahmed), St Mary's Hospital, London.

Reprint requests: Dr Christos Georgalas, Department of Otolaryngology–Head and Neck Surgery, St Mary's Hospital, Praed Street, London, W2 1NY, United Kingdom; e-mail, cgeorgalas@hotmail.com.

0194-5998/2002/$35.00 + 0 **23/77/121318**
doi:10.1067/mhn.2002.121318

A number of studies have looked at the microbial pathogenesis of PTA and found that bacterial synergy between obligate anaerobes and aerobic organisms plays an important role in the development of an abscess.[1,2] Conversely, the pathoetiology of periodontal disease involves the formation of plaque and subsequent infiltration by microorganisms. These in turn produce a variety of metabolites and toxins, which overcome host defenses and lead to further tissue invasion.[3] The microbial profiles of peritonsillar abscess fluid and that from periodontal pockets share a number of similarities. It has been suggested that the plaque can act as a reservoir for oral pathogens especially in patients with poor oral hygiene.

A study was set up to determine whether an association existed between peritonsillar infection and the prevalence of periodontal disease.

PATIENTS AND METHODS

One hundred fifty-eight patients presenting to a tertiary otolaryngology department with peritonsillar abscess were studied over a 3-year period. All patients were admitted for treatment with intravenous fluids and antibiotics. Drainage of purulate exudate was invariably by 3-point needle aspiration.

After recovery from the acute episode, an oral surgeon (H. A.) examined the patients before discharge. Periodontal status was assessed on the basis of the Community Periodontal Index of Treatment Needs (CPITN).[4] This system requires the dentition to be divided into sextants and use of a periodontal probe is essential. The standard probe has a ball end of 0.5-mm diameter. A color-coded area extends from 3.5 to 5.5 mm. Probing force should not exceed 20 to 25 g. The probe tip was gently inserted into the gingival pocket, and the depth of insertion read against the color coding. The total extent of the probe was measured, and at least 6 points on each tooth were examined. For each sextant, the highest score was recorded. Observations were documented and averaged using a 0-4 scale (Table 1).

A control group of 112 patients admitted for elective tonsillectomy for recurrent or chronic tonsillitis were similarly examined on admission in respect of their periodontal status. Results were averaged for each patient. The Shapiro-Wilk test for normality of data showed that CPITN scores were not normally distrib-

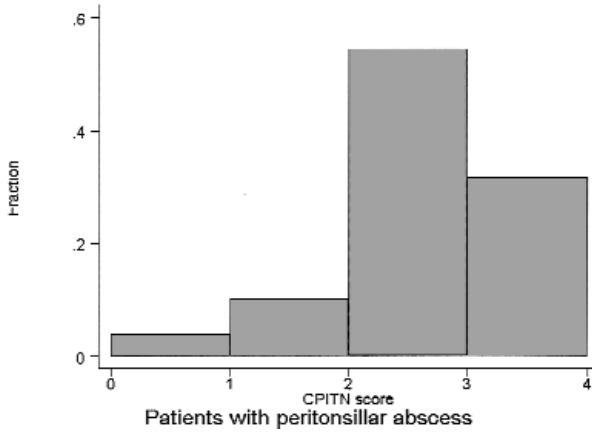

Fig 1. Distribution of CPITN scores in the peritonsillar abscess group.

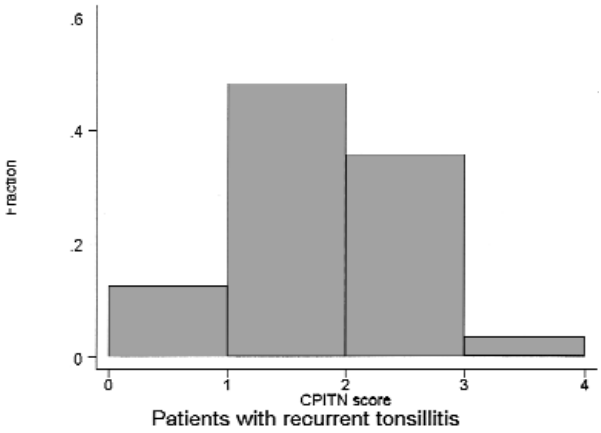

Fig 2. Distribution of CPITN scores in the recurrent tonsillitis group.

Table 1. Community Periodontal Index of Treatment Needs Scoring System

0	Healthy gingival tissues with no bleeding after gentle probing
1	Colored area of probe remains completely visible in the deepest pocket in the sextant. No calculus or defective margins detected. There is bleeding after gentle probing.
2	Colored area remains completely visible in the deepest pocket in the sextant. Either a supra- or subgingival calculus or the defective margin of a filling or crown is detected.
3	Colored area of probe remains partly visible in the deepest pocket in the sextant.
4	Colored area of probe disappears into the pocket indicating probing depth of at least 5 mm.

Table 2. Causative organisms in peritonsillar abscess

Aerobic
Streptococcus pyogenes
Streptococcus millieri
Haemophilus influenzae
Viridans streptococci
Anaerobic
Fusobacterrium necrophorum
*Prevotella melaninogenica**
*Prevotella intermedia**
*Peptostreptococcus micros**
*Fusobacterium nucleatum**
Actinomyces odontolyticus
*Campylobacter rectus**
*Eubacterium nodatum**

*Present in both periodontitis and peritonsillar abscess.

uted (the null hypothesis of normal distribution was rejected at $P = 0.0003$ for the PTA group and $P = 0.01$ for the RT group, respectively). As a consequence, the CPITN scores in the 2 groups were compared using the 2-sample Wilcoxon rank sum test. Chi-square for equal proportions was used for comparing the 2 groups for the presence or absence of periodontal disease (CPITN scores, >2).

RESULTS

There were 158 patients in the PTA group; 91 were male and 67 were female. Mean age at presentation was 19.4 years. One hundred twelve patients presented for tonsillectomy (recurrent/chronic tonsillitis) 62 of which were male and 50 were female with a mean age of 22.6. The 2 groups did not differ significantly with respect to their age and sex. In the PTA group, the CPITN values had a mean of 2.81 (SD, 1.10). In the RT group, the values had a mean of 1.41 (SD, 0.92).

The 2 groups were compared with respect to their mean CPITN score with the use of 2-sample Wilcoxon rank-sum test. The difference in scores was statistically significant at $P = 0.000$ level ($z = 9.28$).

Twelve (11%) of 112 patients with RT had moderate-to-severe periodontal disease (CPITN, 3 or 4); 107 (68%) of 158 patients with RTA had similarly elevated CPITN scores. A contingency table and the Chi-square for equality of proportions showed that the increased prevalence of periodontal disease in the PTA group was statistically significant ($P = 0.000$). More precisely, the odds ratio between the 2 groups was 17.48 (95% CI, 8.87 to 34.39); in other words, a patient with PTA was 17 times more likely to have periodontitis compared with someone with recurrent tonsillitis.

DISCUSSION

A peritonsillar abscess is traditionally considered to be a complication of tonsillitis, peritonsillitis or (rarely)

infective mononucleosis. It usually affects adolescents and young adults and its cause is polymicrobial. In a recent study[2] of 143 peritonsillar abscesses, 16 different aerobic species and 40 different anaerobic bacteria were cultured, an average of 4.4 species per abscess. Six anaerobic and 4 aerobic species accounted for 80% and 65% of all aerobic and anaerobic isolates, respectively (Table 2).

Periodontal disease is an inflammatory disease of the gingiva, the supporting connective tissues and alveolar bone that anchor the teeth to the jaw.[3] Genetic factors, smoking, and diabetes as well as specific microbial factors have been implicated in its pathogenesis. The bacteria linked with this disease with moderate or strong evidence[5] are displayed in Table 3. The overlapping species between periodontitis and peritonsillar abscess are noted.

The interaction between the pathogens is generally synergistic and may be due to the mutual protection from phagocytosis and intracellular killing, production of essential growth factors (such as succinate for *Bacteroides sp*), and the lowering of oxidation-reduction in host tissues. Another feature of mixed infection is the ability of organisms resistant to an antimicrobial agent to protect an organism susceptible to an agent by the production of an antibiotic-degrading enzyme that is secreted into the tissues. In addition, an avirulent colonizer can become a virulent pathogen under certain conditions, especially in the chronic stage of the disease. The presence of capsular material external to the cell wall is a known virulence factor that prevents phagocytosis.[6]

Over the last years it has become clear that tooth infection is a chronic low-grade infection with multiple systemic effects: it has been linked to acute myocardial infarction[7] and chronic and acute respiratory disease.[8,9] Bacteriologic as well as epidemiologic data have shown a relationship between attacks of tonsillitis and pericoronitis.[10]

In 1981, Fried and Forrest[11] were the first to associate peritonsillar abscess with severe dental carious and periodontal disease. Although there is anecdotal evidence of poor dentition in patients with tonsillitis and peritonsillar infection, it could be argued that poor dentition serves as a surrogate marker for smoking, low socioeconomic status, and diabetes, conditions that are associated with upper respiratory tract infections. However, if this is the case, one would expect increased prevalence of periodontal disease in patients with recurrent tonsillitis. A recent article[12] puts forward a new theory about the pathogenesis of peritonsillar abscess. It suggests that it starts as an infection that follows the obstruction of Weber's glands, small accessory salivary glands located in the supratonsillar space. This hypothesis would explain the different pathogens in the abscess and the ease of contamination from the periodontal fluid. A similar bacteriological and epidemiological association have been demonstrated between pericoronitis and peritonsillar disease.[10]

Table 3. Causative organisms in periodontitis

Actinobacillus actinomycetemcomitans
Porphyromonas gingivalis
Bacteroides forsythus
*Campylobacter rectus**
*Eubacterium nodatum**
*Fusobacterium nucleatum**
*Prevotella intermedia**
*Prevotella melaninogenica**
*Peptostreptococcus micros**
Streptococcus intermedius
Treponema denticola

*Present in both periodontitis and peritonsillar abscess.

This study has shown an association between PTA and periodontal disease that warrants further investigation. Although periodontal disease may only be an indicator of health care practices and overall health status, a causal relationship cannot be excluded and at least the fact remains that they often affect the same individual. Our study has shown an increased prevalence of periodontal disease in patients with peritonsillar abscess compared with a group of patients admitted for elective tonsillectomy. Both groups of patients came from the same catchment area, and the age of the patients did not differ significantly between the 2 groups; however, the 2 groups were not formally matched in any other respect. As 1 group contained patients that were admitted electively and the other emergency admissions, one could argue that they had different characteristics. However, as all of these patients were National Health Service patients belonging to the same catchment area, one would expect them to share the same socioeconomic level, although this is by no way proven. The otolaryngologist managing peritonsillar infection should remain vigilant for such associated conditions and refer to an appropriate specialist for treatment, as one could speculate that the treatment of periodontal disease could also have a prophylactic effect against peritonsillar abscess. At the same time, more studies are needed that will further elaborate in this link and provide the answers about its nature.

REFERENCES

1. Brook I. The clinical microbiology of Waldeyer's ring. Otolaryngol Clin North Am 1987;20:259-72.
2. Jousimies-Somer H, Savolainen S, Makitie A, et al. Bacteriologic findings in peritonsillar abscesses in young adults. Clin Infect Dis 1993;16(Suppl 4):S292-8.

3. Williams RC. Periodontal disease. N Engl J Med 1990;322: 373-82.
4. Ainamo J, Barmes D, Beagrie G, et al. Development of the World Health Organization Community Periodontal Index of Treatment Needs (CPITN). Int Dent J 1982;32:281-91.
5. Consensus report. Periodontal diseases: pathogenesis and microbial factors. Ann Periodontol 1996;1:926-32. Review.
6. The role of encapsulated anaerobic bacteria in synergistic infections. FEMS Microbiol Rev 1994;13:65-74.
7. De Stefano F, Anda RF, Kahn HS, et al. Dental disease and risk of coronary heart disease and mortality. Br Med J 1993;306: 688-91.
8. Meurman JH, Rajasuo A, Murtomaa H, et al. Respiratory tract infections and concomitant pericoronitis of the wisdom teeth. Br Med J 1995;310:834-6.
9. Scannapieco FA, Papandonatos GD, Dunford RG. Associations between oral conditions and respiratory disease in a national sample survey population. Ann Periodontol 1998;3:251-6.
10. Rajasuo A, Jousimies-Somer H, Savolainen S, et al. Bacteriologic findings in tonsillitis and pericoronitis. Clin Infect Dis 1996;23:51-60.
11. Fried MP, Forrest JL. Peritonsillitis: evaluation of current therapy. Arch Otolaryngol 1981;107:283-6.
12. Passy V. Pathogenesis of peritonsillar abscess. Laryngoscope 1994;104:185-90.