Peritonsillar Abscess: Complication of Acute Tonsillitis or Weber’s Glands Infection?

Tejs Ehlers Klug, MD1, Maria Rusan, MD1,2, Kurt Fuursted, DMsc3, and Therese Ovesen, DMsc1

Abstract

Objective. To review the literature concerning the 2 primary hypotheses put forth to explain the pathogenesis of peritonsillar abscess: “the acute tonsillitis hypothesis” (peritonsillar abscess is a complication of acute tonsillitis) and “the Weber gland hypothesis” (peritonsillar abscess is an infection of Weber’s glands).

Data Sources. PubMed, EMBASE.

Review Methods. Data supporting or negating one hypothesis or the other were elicited from the literature.

Conclusions. Several findings support the acute tonsillitis hypothesis. First, the 2 main pathogens in peritonsillar abscess have been recovered from pus aspirates and bilateral tonsillar tissues with high concordance rates, suggesting that both tonsils are infected in patients with peritonsillar abscess. Second, studies report signs of acute tonsillitis in the days prior to and at the time of peritonsillar abscess. Third, antibiotic treatment reduces the risk of abscess development in patients with acute tonsillitis. However, some findings suggest involvement of the Weber’s glands in peritonsillar abscess pathogenesis. First, high amylase levels have been found in peritonsillar pus. Second, the majority of peritonsillar abscesses are located at the superior tonsillar pole in proximity of the Weber’s glands. We propose a unified hypothesis whereby bacteria initially infect the tonsillar mucosa and spread via the salivary duct system to the peritonsillar space, where an abscess is formed.

Implications for Practice. Our findings support the rationale for antibiotic treatment of patients with severe acute tonsillitis to reduce the risk of abscess development. Improved understanding of peritonsillar abscess pathogenesis is important for the development of efficient prevention strategies.

Keywords

pathogenesis, peritonsillar abscess, acute tonsillitis

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the common duct from the Weber’s glands results in a localized, suppurative salivary gland infection—a PTA. It has furthermore been proposed that a blockage of the duct could be secondary to recurrent tonsillitis or other inflammatory processes.12,13

Antibiotic treatment is generally recommended for patients with GAS-positive acute tonsillitis. The benefits of treatment are reduced duration of symptoms, halting of infectious spread, and decreased risk of immunologic and suppurrative complications.14-16 The mean reduction of symptoms is approximately only 1 day, and immunologic complications have become rare in Western countries. Hence, a major reason for antibiotic treatment of patients with acute tonsillitis is based on the belief that PTA is a complication of acute tonsillitis, which can be averted by timely antibiotic treatment of the causative bacteria. If PTA is rather an infection of the Weber’s glands, the reasoning behind antibiotic treatment of acute tonsillitis may be questioned.

Recent studies have added important new information regarding these 2 hypotheses. The Weber gland hypothesis has gained support by researchers over the last few years,13,17-19 while several findings supportive of the acute tonsillitis hypothesis have been disproved. This shift prompted the current review of the literature reporting on findings supporting or contradicting these 2 leading hypotheses of PTA pathogenesis.

Methods

The current narrative review is based on a comprehensive literature search in PubMed and EMBASE. No restrictions on publication year were used. Only articles in English were considered. The last search was performed September 9, 2015, using MeSH terms including free text—peritonsillar abscess, quinsy, acute tonsillitis, pathogenesis, and Weber’s glands—along with others in text format: hypothesis, theory, etiology, antibiotics, histology, location, amylase, recurrence, findings, pathogens, and epidemiology. Furthermore, an extensive manual search was performed through the reference lists (from articles included). Articles were read with the aim to identify and elicit data supporting or negating the 2 hypotheses. Given review of the available data, we synthesized a unified hypothesis.

Discussion

Findings Possibly Supportive of the Weber Gland Hypothesis

Detection of amylase in pus from PTA. El-Saied et al reported significantly elevated (median, 62 U/L) and occasionally highly elevated (>500 U/L) amylase levels in PTA pus compared with pus from neck abscesses with other locations.18 Amylase levels >20 U/L were found in 31 of 41 (76%) PTA pus specimens, compared with none of 6 control pus specimens.18 As a PTA is bound by the tonsillar capsule and the pharyngeal musculature (and not an impermeable abscess capsule), it can be argued that amylase may enter the abscess following abscess formation from the adjacent salivary glands. Hence, the finding of elevated amylase levels may not signify that the Weber’s glands are the source of the infection or even that these are part of the route of bacterial spread.

Supratonsillar location of PTAs. In a retrospective review of 100 consecutive PTA patients, Passy reported that 99% of abscesses were located in the supratonsillar space, and he argued that this finding supported the involvement of the Weber’s glands.12 Only a few researchers have described the precise location of PTAs in relation to the tonsillar poles.20-26 A meta-analysis of the literature confirms that the most frequent location for PTAs is behind the upper tonsillar pole (Table 1). However, 36% (95% confidence interval [95% CI]: 31.7%-40.4%) of PTAs were located behind the midportion or lower tonsillar pole. In contrast to the acute tonsillitis hypothesis, the Weber gland hypothesis can explain why the abscess is most commonly located at the superior tonsillar pole. However, the Weber gland hypothesis cannot explain how or why 36% of PTAs arise inferior to the upper tonsillar pole, far from the Weber’s glands. Kraitrakul et al examined the distribution of minor salivary glands in 55 tonsils.27 They found salivary glands at the upper (in 82% of patients), middle (80%), and lower (82%) parts of the peritonsillar space. Hence, an extended version of the Weber gland hypothesis, taking all minor salivary glands in the tonsils into account, could explain the distribution of PTAs. This more general minor salivary gland hypothesis remains to be more thoroughly investigated.

Concurrent PTA and inflammation and fibrosis of the Weber’s glands. Passy argued that the histologic finding of destructive and inflammatory changes of the salivary glands surrounding the PTA supported the Weber gland hypothesis.12 However, Passy studied only 1 patient, and the findings were not compared with tonsils removed for other reasons. According to Powell et al,13 Chen and colleagues28 found smooth and healthy tonsils but inflammation and minor fibrosis of the Weber’s glands in acutely removed tonsils because of PTA. More studies are needed concerning the

<table>
<thead>
<tr>
<th>Study</th>
<th>Upper Pole</th>
<th>Middle</th>
<th>Lower Pole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bateman (1959)21</td>
<td>57 (72)</td>
<td>12 (14)</td>
<td>13 (14)</td>
</tr>
<tr>
<td>Beeden (1970)22</td>
<td>35 (56)</td>
<td>9 (14)</td>
<td>19 (30)</td>
</tr>
<tr>
<td>Brandow (1973)23</td>
<td>109 (70)</td>
<td>34 (22)</td>
<td>13 (8)</td>
</tr>
<tr>
<td>Bonding (1973)24</td>
<td>30 (35)</td>
<td>24 (28)</td>
<td>31 (37)</td>
</tr>
<tr>
<td>Yung (1976)25</td>
<td>38 (76)</td>
<td>6 (12)</td>
<td>6 (12)</td>
</tr>
<tr>
<td>Maisel (1982)26</td>
<td>38 (85)</td>
<td>7 (15)</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>307 (64)</td>
<td>92 (19)</td>
<td>82 (17)</td>
</tr>
</tbody>
</table>

*aValues presented in n (%). Dash (—) indicates none.
histologic findings of the Weber’s glands and ducts during infection.

**PTA is associated with previous PTA and recurrent tonsillitis.** Passy\(^{12}\) speculated that chronic infection of the Weber’s glands or attacks of peritonsillar cellulitis induced fibrosis of the salivary duct system, leading to increased risk of PTA. To our knowledge, selective chronic or recurrent infection of Weber’s glands has never been described. We suggest that fibrosis could also be secondary to recurrent tonsillitis or previous PTA, which could lead to increased risk of PTA.

PTA recurrence rates (excluding patients with residual disease) were in the range of 1.8% to 25.3% in 14 studies reporting on the question (Table 2),\(^{29-42}\) with a cumulative average of 12.2% (95% CI: 11.2%-13.2%). The true number may be higher because recurrences can develop years after the initial PTA and the mean follow-up period (when defined) ranged from 18 months to 17 years.\(^{29-31,33,35,39,40}\) Gavirol et al reported a mean time of 14 months between 2 PTA episodes.\(^{43}\)

Studies report that patients with recurrent tonsillitis at the time of initial PTA development are at increased risk of PTA recurrence.\(^{33,39,44}\) Kronenberg et al found that recurrent PTA was 4 times more frequent in patients with previous recurrent tonsillitis (29 of 72, 40%) compared with patients without (21 of 218, 9.6%).\(^{33}\) Similarly, in a study by Savolainen et al, 8 of 14 (57%) patients with ≥3 episodes of tonsillitis had recurrence of PTA, compared with 13 of 77 (17%) patients with ≤3 acute tonsillitis episodes.\(^{44}\)

Hence, a previous episode of PTA significantly increases the risk of recurrent PTA to approximately 12% (compared with a 2%-3% lifetime risk of PTA in Denmark). Patients who also suffer from recurrent tonsillitis at the time of PTA are at even greater risk of recurrent PTA, especially if <40 years old.\(^{32,34,39}\)

These findings suggest that scarring or other anatomic changes of the tonsil or the Weber’s glands, as inflicted by infection within the tonsil or the peritonsillar tissues, increase the risk of recurrent tonsillar disease in general and PTA in particular. However, there is a lack of histologic studies supporting this hypothesis and indicating the precise location of significant histologic alterations.

There may be an increased risk of PTA in patients with recurrent tonsillitis (without previous PTA), but the magnitude of this association is not clear from the current literature (Table 3).

Nevertheless, only a minority of PTA patients has had a history of recurrent tonsillitis, peritonsillar cellulitis, or previous PTA.

**Tonsillectomy prevents future PTA.** The risk of PTA is markedly reduced after tonsillectomy. Passy argued that the Weber gland hypothesis explains the reduced PTA risk among tonsillectomized individuals, presumably due to the removal of Weber’s glands at the time of tonsillectomy.\(^{12}\) However, it can be similarly argued that the removal of the tonsillar tissue is the reason for the reduced PTA risk.

**Findings Supporting the Acute Tonsillitis Hypothesis**

**Concurrent acute tonsillitis in PTA patients.** Passy claimed that only 4 of 100 consecutive patients with PTA had tonsillar exudates.\(^{12}\) Accordingly, the majority of PTA patients did not have acute tonsillitis at the time of their PTA diagnosis. This argument against the acute tonsillitis hypothesis was made on the basis of a retrospective chart review, which raises

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**Table 2. Patients with Recurrent PTA after Treatment With Aspiration or Incision.**

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients Treated with Aspiration or Incision, n</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muller (1978)(^{38})</td>
<td>42</td>
<td>3</td>
<td>7.1</td>
</tr>
<tr>
<td>Herbild (1981)(^{32})</td>
<td>166</td>
<td>37</td>
<td>22.3</td>
</tr>
<tr>
<td>Fried (1981)(^{31})</td>
<td>57</td>
<td>1</td>
<td>1.8</td>
</tr>
<tr>
<td>Nielsen (1981)(^{34})</td>
<td>44</td>
<td>10</td>
<td>22.7</td>
</tr>
<tr>
<td>Litman (1987)(^{37})</td>
<td>87</td>
<td>22</td>
<td>25.3</td>
</tr>
<tr>
<td>Kronenberg (1987)(^{33})</td>
<td>218</td>
<td>50</td>
<td>22.9</td>
</tr>
<tr>
<td>Harris (1991)(^{39})</td>
<td>36</td>
<td>8</td>
<td>22.2</td>
</tr>
<tr>
<td>Sorensen (1991)(^{36})</td>
<td>536</td>
<td>33</td>
<td>6.1</td>
</tr>
<tr>
<td>Wolf (1994)(^{35})</td>
<td>160</td>
<td>14</td>
<td>14.4</td>
</tr>
<tr>
<td>Ophir (1988)(^{29})</td>
<td>75</td>
<td>11</td>
<td>14.7</td>
</tr>
<tr>
<td>Ong (2004)(^{30})</td>
<td>185</td>
<td>14</td>
<td>7.6</td>
</tr>
<tr>
<td>Chung (2014)(^{41})</td>
<td>172</td>
<td>24</td>
<td>13.9</td>
</tr>
<tr>
<td>Bovo (2015)(^{42})</td>
<td>2667</td>
<td>312</td>
<td>11.7</td>
</tr>
<tr>
<td>Shaul (2015)(^{40})</td>
<td>117</td>
<td>17</td>
<td>14.5</td>
</tr>
<tr>
<td>Total</td>
<td>4562</td>
<td>556</td>
<td>12.2</td>
</tr>
</tbody>
</table>

Abbreviation: PTA, peritonsillar abscess.
Table 3. Studies of Patients with PTA and Information Concerning Previous Episodes of Acute Tonsillitis, Recurrent Tonsillitis, Chronic Tonsillitis, and Tonsillar Diseases in General.4

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grahn (1958)54</td>
<td>79% (of 725) of patients had a history of ≥1 previous episodes of tonsillitis or peritonsillitis</td>
</tr>
<tr>
<td>Bateman (1959)21</td>
<td>34 of 120 (28%) patients had repeated tonsillitis in the past</td>
</tr>
<tr>
<td>Beeden (1970)22</td>
<td>56 of 111 (50%) patients had previous episodes of tonsillitis</td>
</tr>
<tr>
<td>Brandow (1973)23</td>
<td>79 of 156 (51%) patients previously had ≥1 attacks of tonsillitis</td>
</tr>
<tr>
<td>Bonding (1973)24</td>
<td>83 of 317 (26%) patients previously had repeated tonsillitis with fever ≥3 times per year; 38 of 317 (12%) patients had symptoms of chronic tonsil affection or previously had 1 or 2 episodes of acute tonsillitis per year</td>
</tr>
<tr>
<td>Herbild (1981)32</td>
<td>51 of 256 (20%) patients previously had recurrent episodes of tonsillitis and 7% previously had symptoms of pharyngitis in varying degrees</td>
</tr>
<tr>
<td>Fried (1981)31</td>
<td>12 of 41 (29%) patients had had ≥1 episodes of sore throat symptoms</td>
</tr>
<tr>
<td>Schechter (1982)91</td>
<td>29 of 74 (39%) patients had a history of tonsillar disease, including 4 patients with previous PTA</td>
</tr>
<tr>
<td>Stegehuis (1986)92</td>
<td>22 of 83 (27%) patients previously had ≥2 attacks of tonsillitis per year over the last 2 y</td>
</tr>
<tr>
<td>Stringer (1988)93</td>
<td>10 (36%) patients had a history of tonsillitis, including 5 patients with a history of PTA</td>
</tr>
<tr>
<td>Savolainen (1993)94</td>
<td>34 of 98 (35%) and 21 of 98 (21%) patients previously had 1-3 and ≥3 episodes of tonsillitis, respectively</td>
</tr>
<tr>
<td>Wolf (1994)35</td>
<td>34 of 160 (21%) patients had a medical history of recurrent tonsillitis</td>
</tr>
<tr>
<td>Matsuda (2002)95</td>
<td>75 of 724 (10%) patients had a history of tonsillar disease, including 48 with prior PTA</td>
</tr>
<tr>
<td>Ong (2004)30</td>
<td>23 of 185 (12%) patients gave a history of recurrent tonsillitis</td>
</tr>
<tr>
<td>Segal (2009)96</td>
<td>45 of 127 (35%) children previously had tonsillar infection in the past</td>
</tr>
</tbody>
</table>

Abbreviation: PTA, peritonsillar abscess.

The wording used by the original study authors is employed.

concerns regarding the accuracy and completeness of the tonsillar mucosa description in the patient records. An accurate description of the tonsillar mucosa in PTA patients is not included in the majority of published PTA studies. However, in contrast to Passy, Spires et al reported tonsillar exudates in 51% of patients with PTA.4 In a study of 275 acutely removed tonsils due to PTA, acute or chronic inflammation was described in 68% of cases in routine histologic examination.45 Blair et al conducted histopathologic examinations of tonsillar specimens from 6 patients with PTA, 4 with acute tonsillitis, and 2 with intratonsillar abscess.11 In all 6 specimens from PTA patients, the authors found erosion of the tonsillar surface epithelium and invasion of neutrophils, as observed in acute tonsillitis specimens. The limited evidence regarding signs of acute tonsillar infection in patients with PTA favors the acute tonsillitis hypothesis.

Common bacterial pathogens. GAS is commonly recognized as a significant pathogen in acute tonsillitis, and PTA.46 Recent studies point to a major role of F necrophorum in PTA,4,9,10 and this anaerobe may also play a pathogenic role in acute tonsillitis.49-52 This concordance of 2 significant pathogens between PTA and acute tonsillitis lends further support to the acute tonsillitis hypothesis. However, it can be argued that bacteria can exert their pathogenic potential at different anatomic sites without causal relationship.

Occasional bilateral PTA. In 14 studies reporting on the prevalence of bilateral PTA in patients undergoing bilateral quinsy tonsillectomy, bilateral PTA was found in 2.0% to 24% of patients (average across studies, 5.5%; 95% CI: 4.7%-6.5%; Table 4).19,22,23,53-66 The majority of patients with bilateral PTA were thought to have unilateral PTA, but an additional, contralateral abscess was discovered at the time of bilateral acute tonsillectomy. Unfortunately, none of the studies report on bacterial findings from concurrent abscesses. However, the fact that approximately 5% of PTA patients have bilateral PTA suggests that PTA is secondary to bilateral acute tonsillitis, and the finding is difficult to explain based on the Weber gland hypothesis alone.

Antibiotic treatment prior to PTA diagnosis. Studies report that 25% to 79% of PTA patients (collected average, 41%; 95% CI: 39.2%-43.6%) received antibiotics prior to diagnosis of PTA (Table 5).3,4,30,67-76 The indications for antibiotic treatment were not described in any of the studies, and antibiotics may have been prescribed without signs and symptoms of acute tonsillitis. However, the fact that almost half the PTA patients sought medical consultation and initiated antibiotic treatment in the days prior to abscess formation suggests that acute tonsillitis prior to PTA development is common. The 60% of patients who did not take antibiotics prior to PTA development may have had less severe symptoms or a negative test result for the presence of GAS.

Antibiotic treatment prevents PTA development. The findings above indicate that antibiotic treatment does not prevent abscess formation in all cases. However, studies suggest that there could be a protective effect of appropriate antibiotic treatment to patients with bacterial tonsillitis with avoidance of suppurative complications.77,78 A Cochrane review
by Del Mar et al, based on 2433 patients from 8 studies, found a convincing 85% reduction in the risk of PTA if bacterial acute tonsillitis was treated with antibiotics. This percentage should be regarded with some reservation, as 72% (18 of 25) of the patients who developed PTA were included in 2 nonrandomized and non-double-blinded studies from 1951. At that time, the prevalence of PTA in untreated patients was much higher than it is today. Furthermore, some patients were treated with intramuscular penicillin or antibiotics not presently recommended for acute tonsillitis. Hence, it is largely unknown if the treatment of bacterial acute tonsillitis patients with the antibiotics used today reduces the risk of PTA. Nevertheless, the fact that a protective effect of antibiotics on PTA development exists is a strong indicator for the acute tonsillitis hypothesis.

**Development of PTA after well-described initial sore throat.** Recently, Little et al published the results of an impressive study including 14,610 patients treated for sore throat in 616 general practices. They found that 47 of 13,288 patients (1322 patients had missing data) with well-described initial symptoms and findings of acute tonsillitis developed PTA within a month of initial consultation. It can be argued that the development of PTA in a minority (0.5%) of patients could just be random or that the symptoms (throat pain and difficulty swallowing) in these 47 patients were due to incipient infection of the Weber’s glands. Hence, it is largely unknown if the treatment of bacterial acute tonsillitis patients with the antibiotics used today reduces the risk of PTA. Nevertheless, the fact that a protective effect of antibiotics on PTA development exists is a strong indicator for the acute tonsillitis hypothesis.

**Bacterial concordance rates between the 2 tonsils in patients with PTA.** The bacteriology of both tonsils (surface and core) and pus aspirates in patients with unilateral PTA undergoing acute bilateral tonsillectomy was previously studied. GAS was isolated from 7 of 36 patients. In all 7 patients, GAS was found in aspirated pus and the tonsillar core at the side of the abscess. Similarly, *F necrophorum* was found in both pus and the tonsillar cores at the side of the abscess. In addition, 2 *F necrophorum* isolates were recovered solely from aspirated pus, and 1 *F necrophorum* isolate was found in the tonsillar core only. These findings confirm the nearly perfect concordance between pus and ipsilateral tonsillar cores and show that the oropharyngeal infection in PTA patients affects both tonsils with concurrent pathogens.

**Findings Possibly Contradicting the Acute Tonsillitis Hypothesis**

**Acute tonsillitis is more frequent in childhood, and PTA is more prevalent among adults.** The majority of PTA patients are teenagers and young adults, while bacterial acute tonsillitis is more frequent among children. Researchers skeptical of the acute tonsillitis hypothesis argue that the age-
related incidence rates of acute tonsillitis and PTA should mirror each other if this hypothesis is true. However, factors other than age also contribute to the development of PTA. For instance, smoking is associated with an increased risk of PTA development. In a previous study of 847 PTA patients, *F. necrophorum* was significantly more prevalent among patients aged 15 to 24 years as compared with children and older adults. Similarly, *F. necrophorum* is more commonly associated with acute tonsillitis in patients aged 15 to 30 years. The observed differences in age distribution of acute tonsillitis and PTA cases in children vs adults can potentially be explained by additional risk factors for PTA development and susceptibility to specific bacterial pathogens, which are unequally distributed between children and adults.

**Conclusions**

More studies are needed before solid conclusions regarding the pathogenesis of PTA can be drawn. Based on the current literature, however, there are more findings in favor of the acute tonsillitis hypothesis. First, the 2 main pathogens in PTA—GAS and *F. necrophorum*—have been recovered from PTA pus aspirates and bilateral tonsillar tissues with high concordance rates. Second, also indicating bilateral tonsillar infection, bilateral PTA has been found in approximately 5% of PTA cases at the time of acute tonsillectomy. Third, signs of acute tonsillitis in the days prior to and at the time of PTA development have been described in multiple studies. These findings are in agreement with the finding that approximately 40% of PTA patients were treated with antibiotics in the days prior to PTA diagnosis, possibly due to signs and symptoms of acute tonsillitis. Last, antibiotic treatment has been found to reduce the risk of PTA development in patients with acute tonsillitis.

However, some findings are difficult to explain from the acute tonsillitis hypothesis alone but suggest a role of the minor salivary glands (Weber’s glands in particular) in PTA pathogenesis. First, elevated amylase levels have been found in PTA pus compared with other neck abscesses. Second, the majority of PTAs are located at the superior tonsillar pole. Yet, a significant proportion of PTAs are located at the midportion or lower tonsillar pole, and an extended version of the Weber gland hypothesis encompassing all minor salivary glands in the tonsils may explain this finding. In addition, the duct system of the minor salivary glands provides an explanation for how bacteria may penetrate the tonsillar capsule, when the infection progresses from the tonsillar mucosa to the peritonsillar tissues.

Previous researchers have, for unknown reasons, thought of these 2 hypotheses as opposing. We propose that, indeed, both hypotheses may be true and complementary. We find it likely that, in the majority of PTA cases, bacteria infect the tonsillar mucosa (including the crypt mucosa) and spread to the peritonsillar space via the salivary duct system, where an abscess is formed if the bacteria are not overcome by the immune system and (in some cases) antibiotics. With the variations seen in the clinical presentation of PTA patients, it is possible that PTA development in some patients is solely an infection of the Weber’s glands and in others a direct spread of the infection from the tonsillar mucosa.

**Implications for Practice**

PTA continues to be the most common deep neck abscess, and antibiotic treatment directed against GAS in patients with acute tonsillitis is currently the only intervention identified to reduce the incidence. However, the use of antibiotic...
therapy in patients with bacterial acute tonsillitis has recently been questioned by researchers skeptical to the acute tonsillitis hypothesis. The findings in the current review support the rationale for antibiotic treatment of patients with severe acute tonsillitis to reduce the risk of PTA development. Little et al reported that the symptoms and clinical findings in patients with acute tonsillitis were unable to predict those who developed complications, including PTA. 87 Hence, the number of acute tonsillitis patients needed to treat to avoid one case of PTA is currently high. Recent studies indicate a major role of *F. necrophorum* in acute tonsillitis and PTA, and it is plausible that early detection and antibiotic therapy directed against this anaerobe may prevent PTA development in many teenagers and young adults. However, more studies are needed to substantiate this assumption.

Improved understanding of the PTA pathogenesis is important for the development of more efficient prevention strategies. Studies of the anatomy, distribution, microbiology, and immunology of the minor salivary glands in the tonsils are likely to provide valuable information.

**Author Contributions**

Tejs Ehlers Klug, initiating, designing, and drafting of the work; identification and analysis of the studies; final approval of the manuscript; accountable for all aspects of the work; Maria Rusan, analysis and interpretation of the studies; critical revision and final approval of the manuscript; accountable for all aspects of the work; Kurt Fuursted, designing the review; critical revision and final approval of the manuscript; accountable for all aspects of the work; Therese Ovesen, designing the review; critical revision and final approval of the manuscript; accountable for all aspects of the work.

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