Prevalence of H Pylori in tonsillar tissue of patients with adenotonsillar hypertrophy using Rapid Urease Test in a tertiary hospital in a sub-Saharan country

Peter Otieno Ochungo, Peter Mugwe, Wairimu Waweru
University of Nairobi; University of Nairobi; University of Nairobi

ABSTRACT

This article attempts to review the prevalence of H Pylori in the tonsillar tissue of patients with adenotonsillar hypertrophy. Rapid urease test is used to identify the presence of H Pylori. H pylori has the ability to cause / alter many disease conditions. Its ability to cause hypertrophy of extragastric tissues like tonsil is not clear.

Colonization of human palatine tonsils by H Pylori is a potentially exciting new frontier which could radically alter the management approach to patients with adenotonsillar hypertrophy.

Introduction:

H. pylori is a gram negative microaerophilic bacteria whose optimum growth is at an oxygen level of 2-5% with a high humidity level.[1,2]. It was discovered by Marshall and Warren in 1983 and is one of the most successful human pathogens[3].

The transmission of H. pylori bacterium is not yet well understood, but the oral–oral and the fecal–oral are the most common routes of transmission [4,5].

Although culture of H.pylori from the oral cavity has been inconsistent. Studies have reported the presence of H. pylori in the oral cavity in samples from, dental plaque, supragingival plaque and saliva by polymerase chain reaction [5,6].
H. pylori is known to be involved in the pathogenesis of various disease conditions, including duodenal ulcers, gastric conditions, IgA nephropathy and gastric adenomas with various host reservoirs acting as a nidus for continued persistence of the organism.[3] The human palatine tonsil has been proposed as one of the extra gastric reservoirs of H. pylori and various studies have been undertaken to study its role[7-11]. Other studies dispute the fact that the human palatine tonsil may be an extragastric reservoir of H. pylori[12,13]

Pathogenesis

Although beyond the scope of this article, a brief mention may be warranted. Various theories have been advanced to explain how H. pylori is able to colonise, adapt and persist in host tissue.[14,15] These include the fact that it may adhere to epithelial cells and induce a strong inflammatory response which does not lead to elimination of the organism but causes a chronic inflammation which leads to hyperkeratosis which makes the penetration of antibiotics difficult in the affected tissue [14,15].

The above mechanisms not only explain the complexity of the H. pylori organism in the human body but also show how much the organism is not fully understood due to its variable nature.

Material and methods.

This prospective , cross-sectional study was first approved by our local institutional review board before any patient enrolment.

Thirty-nine patients were referred to the otolaryngology clinic at Kenyatta National Hospital with a history of adenotonsillar hypertrophy.

Each patient was then diagnosed as having adenotonsillar hypertrophy by the clinic consultant and booked for tonsillectomy.

All enrolled patients met the criteria for elective tonsillectomy due to adenotonsillar hypertrophy and consent was sought from the guardians of the patient.

Exclusion criteria included any patient who declined to participate in the study, had used a full course of antibiotics during the last two weeks prior to the study, was on triple therapy for peptic ulcers or who had an indication for tonsillectomy of a diagnosis other than adenotonsillar hypertrophy.

Preparation of tonsillar tissues

Once tonsillectomy operation was done, one tonsil per patient was collected and taken to the pathology lab of the University of Nairobi.

A 2mm gross specimen was cut out using a sterile blade and gloves for each tonsillar tissue harvested by the laboratory technician.

Each specimen was then placed in a test well containing rapid urease (Cambridge life science limited U.K, Batch 311161 ) and an initial colour read at 0 minutes.

Subsequent colour changes were read at 30 minutes,6 hours and 24 hours. Any color change from the initial yellow colour to either pink or red was recorded as positive.

Any test well that remained yellow after 24 hours was recorded as negative. No readings were taken after 24 hours.
Use of core tonsillar tissue is recommended due to its sensitivity compared to a surface swab [16] RUT is a preferred method of examining tissue as it has high sensitivity and specificity[17,18,19].

Principles of color change

Any color change is based on detection of urease, a hydrolase produced by H pylori.

The test system is a test well filled with a urea containing gel and this is where the tonsillar tissue is inoculated and allowed to incubate.

Urease which is found in H pylori will hydrolyse the area in the gel. This will lead to accumulation of ammonium ion.

This will then cause a rise in the pH and this is detected in the PH indicators by a color change in the system from yellow to pink or red.

Results

There were 39 patients who were recruited for this study. The age range was from 4-14 years.

The average age for patients with ATH was 4.3 (Table 1). 19 patients were male while 20 were female, presence of H pylori by rapid urease test and based on color change was 23.1% (95% CI, 11.1-39.3%).(Table 3)

<table>
<thead>
<tr>
<th>Table 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline Characteristics:</strong></td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Age</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presence of H pylori by Rapid urease test:</strong></td>
</tr>
<tr>
<td>Variable:</td>
</tr>
<tr>
<td>Rapid Urease Test:</td>
</tr>
<tr>
<td>Positive</td>
</tr>
<tr>
<td>Negative</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3: Overall Presence of H. pylori</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prevalence</strong></td>
</tr>
<tr>
<td>Adenotonsillar hypertrophy (ATH)</td>
</tr>
</tbody>
</table>
Discussion

There is an increasing attention to H. pylori based on its ability to cause and even alter many disease conditions.

But its ability to cause hypertrophy of extra gastric tissues including the palatine tonsil is not clear[22].

Current studies are focused on analyzing for the presence of H. pylori in various disease states. This particular study attempts to analyze for the presence of H. pylori bacteria in tonsillar tissue of patients presenting for tonsillectomy secondary to adenotonsillar hypertrophy.

Various studies have attempted to identify H pylon in tonsillar tissue by using various experimental methods from polyclonal chain reactions (PCR) to Rapid urease test (RUT) with varying results. The studies have suggested that H pylori may exist in extra–gastric reservoirs.[20-22]

Cho K.K. et al[20] in his study on 38 patients who underwent adenotonsillectomy or tonsillectomy, found a prevalence of 21.1% for H.pylori based on the campylobacter like organism (CLO)test which is similar in principle to the Rapid urease test.

Moghaddam et al,[21] in their prospective study of H. pylori colonization in 258 children, found an overall prevalence of 14% by Rapid urease test.

Similarly, a study by Lin et al [22] on 94 patients recruited with chronic recurrent tonsillitis and adenotonsillar hypertrophy, found that 48% of the patients with chronic recurrent tonsillitis were positive for H.pylori, compared with 24% for the group with adenotonsillar hypertrophy.

Other studies have disputed the presence of H pylori in extra gastric reservoirs, including tonsil tissue. [23,24]

This may be as a result of geographical variations or the sensitivity of the method used [25]

Conclusion

In conclusion colonisation of the human palatine tonsils by H pylori is a potentially exciting new frontier which could radically alter the management approach to patients with adenotonsillar hypertrophy.

It may also be useful to design specific diagnostic tests for the detection of H pylori in adenotonsillar tissue.

Further studies may be needed to clarify the possible role of H pylori in the pathogenesis of adenotonsillar hypertrophy.

Acknowledgements

Part of this work was presented at the 20th Annual Kenya Ear, Nose and Throat Annual Scientific Conference, May 16th-20th 2012 held at the Watamu Resort, Malindi, Kenya.
REFERENCES


