

Prevalence of *H. Pylori* in Tonsillar Tissue of Patients with Chronic Recurrent Tonsillitis Using Rapid Urease Test in a Tertiary Referral Hospital in Sub Saharan Africa

O. Peter Ochung'o · P. Mugwe · P. Masinde · W. Waweru

Received: 26 April 2014 / Accepted: 23 July 2014 / Published online: 6 August 2014
© Association of Otolaryngologists of India 2014

Abstract There has been conflicting results regarding the presence of *H. pylori* in tonsillar tissue. Our objective was to analyze for the presence of *H. pylori* in tonsillar tissue in patients undergoing tonsillectomy for chronic recurrent tonsillitis using rapid urease test in a Tertiary care academic medical center in a sub Saharan hospital. A prospective cross-sectional analysis of 39 consecutive cases of patients undergoing tonsillectomy secondary to chronic recurrent tonsillitis was done. Rapid urease test was conducted on each tonsillectomy tissue and results were determined using color change at specific time intervals within 24 h. Average age of the patients was 4.3 years. Among the 39 tonsillar tissues analysed using rapid urease test, *H. pylori* was present in 30.5 % of the samples. Colonisation by *H. pylori* of the palatine tonsils is a new frontier with conflicting results depending on the accuracy of the test method used and population studied. More studies need to be performed to ascertain the different rates of colonisation based on geographical regions.

Keywords *H. pylori* · Chronic recurrent tonsillitis · Rapid urease test

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 is warranted. Various theories have been advanced to explain how *H. pylori* is able to colonise, adapt and persist

O. P. Ochung'o · P. Mugwe · P. Masinde
Department of Ear, Nose and Throat Head and Neck Surgery,
University of Nairobi, Uhuru Highway, Nairobi 00100, Kenya
e-mail: drmugwep@gmail.com

P. Masinde
e-mail: masinde@wananchi.com

O. P. Ochung'o (✉)
Department of Otolaryngology-Head and Neck Surgery,
University of Nairobi, P.O. Box 580-00600, Nairobi, Kenya
e-mail: peter.ochungo@gmail.com

W. Waweru
Department of Pathology, University of Nairobi, Uhuru
Highway, Nairobi 00100, Kenya
e-mail: wairimu@uonbi.co.ke

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].

Direct injury to host tissues through production of urease, lipopolysaccharide and cytotoxins have also been advanced as another theory by which *H. pylori* may gain direct entry into host tissue.

The *H. pylori* may be able to avoid detection by the hosts immune response by reducing its production of flagellin and thus avoiding detection by the hosts toll like receptor system [16]. It may also remain viable in macrophage phagosomes by inhibiting phagosome maturation [17].

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.

Materials 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 suggestive of recurrent tonsillitis. Each patient was then diagnosed as having chronic recurrent tonsillitis by the clinic consultant and booked for tonsillectomy.

All enrolled patients met the criteria for elective tonsillectomy due to chronic recurrent tonsillitis 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 2 weeks prior to the study, was on triple therapy for peptic ulcers or who had an indication for tonsillectomy of a diagnosis other than chronic recurrent tonsillitis.

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 2 mm 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 min. Subsequent colour changes

were read at 30 min, 6 and 24 h. 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 h was recorded as negative. No readings were taken after 24 h.

Use of core tonsillar tissue is recommended due to its sensitivity compared to a surface swab [18]. RUT is a preferred method of examining tissue as it has high sensitivity and specificity [19–21].

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 urea 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

Thirty nine patients were recruited for this study. The age ranged from 4 to 14 years.

The median age for patients with chronic recurrent tonsillitis was 6.0, 21 patients were male while 18 were female, presence of *H. pylori* by rapid urease test and based on color change was 38.5 % Tables 1, 2 and 3.

Table 1 Baseline characteristics

Male	21 (53.8 %)
Female	18 (46.2 %)
Age	6.0

Table 2 Presence of *H. pylori* by rapid urease test

Variable:	Indication for tonsillectomy:
Rapid urease test:	Chronic recurrent tonsillitis
Positive	15 (38.5 %)
Negative	24 (61.5 %)

Table 3 Presence of *H. pylori* with confidence intervals

Chronic recurrent tonsillitis	Prevalence	95 % CI
	38.5 %	23.4–55.5 %

Discussion

There is an increasing concern about the rate of *H. pylori* in causing various disease states.

The *H. pylori* is also attracting increased attention because it is increasingly found in various parts of the body due to its ability to change the micro-environment.

Various studies have attempted to identify *H. pylori* in tonsillar tissue by using various experimental methods from polyctonal chain reactions (PCR) to rapid urease test (RUT) with varying results. The studies have suggested that *H. pylori* may exist in extra gastric reservoirs [9, 10, 22–24].

The current study compares with Monem et al. [10] who studied 30 children who were diagnosed with chronic recurrent tonsillitis and 53.3 % were found to be positive for *H. pylori* using RUT. The overall prevalence rate was higher than this present study.

A study by Nam et al. [22] on 98 patients with recurrent tonsillitis found an overall prevalence of 62 % positivity for *H. pylori* using RUT.

Cho et al. [23] 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 RUT.

Moghaddam et al. [9] in their prospective study of *H. pylori* colonization in 258 children, found an overall prevalence of 14 % by RUT. Similarly, a study by Lin et al. [24] 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 [12, 13].

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 chronic recurrent tonsillitis.

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 chronic recurrent tonsillitis.

References

1. Sidebotham RL, Worku ML, Karim QN et al (2003) How *Helicobacter pylori* urease may affect external pH and influence growth and motility in the mucus environment: evidence from in vitro studies. *Eur J Gastroenterol Hepatol* 15(4):395–401
2. O'Toole PW, Lane MC, Porwollik S (2000) *Helicobacter pylori* motility. *Microbes Infect* 2:1207–1214
3. Kusters J, Arnoud H, Kuipers E et al (2006) Pathogenesis of *Helicobacter pylori* infection. *Clin Microbiol Rev* 19(3):449–490
4. Azevedo NF, Guimaraes N, Figueiredo C, Keevil CW, Vieira MJ (2007) A new model for the transmission of *Helicobacter pylori*: role of environmental reservoirs as gene pools to increase strain diversity. *Crit Rev Microbiol* 33:157–169
5. Azevedo NF, Huntington J, Goodman KJ (2009) The epidemiology of *Helicobacter pylori* and public health implications. *Helicobacter* 14(Suppl. 1):1–7
6. Czesnikiewicz-Guzik M, Bielanski W, Guzik TJ, Loster B, Konturek SJ (2005) *Helicobacter pylori* in the oral cavity and its implications for gastric infection, periodontal health, immunology and dyspepsia. *J Physiol Pharmacol* 56(Suppl. 6):77–89
7. Zahedi M, Darvish Moghadam S, Ahmadi Mosavi M et al (2009) *Helicobacter pylori* colonization in biopsies of the adenotonsillectomy specimens. *Am J Appl Sci* 6(12):2050–2053
8. Skinner LJ, Winter DC, Curran AJ et al (2001) *Helicobacter pylori* and tonsillectomy. *Clin Otolaryngol Allied Sci* 26(6):505–509
9. Moghaddam Y, Rafeey M, Radfar R et al (2009) Comparative assessment of *Helicobacter pylori* colonization in children tonsillar tissues. *Int J Pediatr Otorhinolaryngol* 73(9):1199–1201 Epub 2009 Jun 12
10. Abdel-Monem MH, Magdy EA, Nour YA et al (2011) Detection of *Helicobacter pylori* in adenotonsillar tissue of children with chronic adenotonsillitis using rapid urease test, PCR and blood serology: a prospective study. *Int J Pediatr Otorhinolaryngol* 75(4):568–572 Epub 2011 Feb 15
11. Dağtekin-Ergür EN, Eren F, Ustün MB et al (2008) Investigation of *Helicobacter pylori* colonization in pharyngeal and palatine tonsils with rapid urease test. *Kulak Burun Bogaz Ihtis Derg* 18(2):85–89
12. Vilarinho S, Guimarães NM, Ferreira RM (2010) *Helicobacter pylori* colonization of the adenotonsillar tissue: fact or fiction? *Int J Pediatr Otorhinolaryngol* 74(7):807–811 Epub 2010
13. Vayisoglu Y, Ozcan C, Polat A et al (2008) Does *Helicobacter pylori* play a role in the development of chronic adenotonsillitis? *Int J Pediatr Otorhinolaryngol* 72(10):1497–1501
14. Ramarao N, Meyer T (2001) *Helicobacter pylori* resists phagocytosis by macrophages: quantitative assessment by confocal microscopy and fluorescence-activated cell sorting. *Infect Immun* 69(4):2604–2611
15. Crabtree JE (1996) Immune and inflammatory responses to *Helicobacter pylori* infection. *Scand J Gastroenterol Suppl* 215:3–10
16. Gewirtz AT, Yu Y, Krishna US (2004) *Helicobacter pylori* flagellin evades toll-like receptor 5-mediated innate immunity. *J Infect Dis* 189(10):1914–1920
17. Blanchard T, Drakes M, Czinn S (2004) *Helicobacter* infection: pathogenesis. *Curr Opin Gastroenterol* 20:10–15
18. Di Bonaventura G, Neff M, Neri G et al (2001) Do tonsils represent an extragastric reservoir for *Helicobacter pylori* infection? *J Infect* 42(3):221–222
19. Khademi B, Imanieh MH, Gandomi B et al (2005) Investigation of *H. pylori* colonization in adenotonsillectomy specimens by means of rapid urease (CLO) test. *Iran J Med* 138 Sci 30(3):54–65

20. Dye KD, Marshall BJ, Frierson HF et al (1988) Is CLO test alone adequate to diagnose *Campylobacter pylori*? [abstract]. Am J Gastroenterol 83:1032
21. Schnell GA, Schubert TT, Barnes WG et al (1988) Comparison of urease, H&E and culture tests for *Campylobacter pylori*. Gastroenterology 94:A410
22. Nam JK, Park KC, Kwon JK (2007) Is *Helicobacter Pylori* the pathogen of chronic tonsillitis? Korean J Otorhinolaryngol-Head Neck Surg 50(7):616–621
23. Cho KK, Lee SS, Shim KN (2007) Effect of palatine tonsil and adenoid tissue on gastric infection of *Helicobacter pylori*. Korean J Otorhinolaryngol-Head Neck Surg 50(10):907–912
24. Lin Hsin-Ching, Pei-Yin Wu, Friedman Michael et al (2010) Difference of *Helicobacter pylori* colonization in recurrent inflammatory and simple hyperplastic tonsil tissues. Arch Otolaryngol Head Neck Surg 136(5):468–470
25. Rowland M, Daly L, Vaughan M et al (2006) Age-specific incidence of *Helicobacter pylori*. Gastroenterology 130:65–72