# *Helicobacter Pylori* as an Etiologic Factor for Development of Nasal Polyposis in Patients with Extraesophageal Reflux

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## SUMMARY

The aim of this cross-sectional study was to identify *Helicobacter pylori* (HP) as an etiologic factor for the development of nasal polyposis (NP) in patients with extraesophageal reflux (EER). The study enrolled 35 cases with NP and 30 controls with bullous middle nasal concha (CB) undergoing endoscopic sinus surgery (ESS). All subjects underwent esophagogastroendoscopy with routine antrum and corpus biopsies and filled out two questionnaires on EER and nasal symptoms (NS) prior to ESS. All gastric and nasal biopsy specimens were histopathologically and real time polymerase chain reaction (RT PCR) examined for HP. All subjects, cases and controls, had positive results for HP in gastric mucosa and 33 (50.8%) subjects had EER. The proportion of patients with reported EER was significantly higher among patients with NP (70.0% *vs*. 30.0%; $\chi^2$-test, p=0.003). HP was identified in nasal mucosa in 10 (30.3%) patients with NP and all 10 patients had EER and NS. HP was not detected in any control patient with CB. HP was not present in healthy nasal mucosa. HP can be detected in nasal mucosa in about 40% of patients with nasal polyps and EER. HP can be considered as an etiologic factor in patients with NP and EER although further studies are needed to evaluate this correlation.

## KEYWORDS

*Helicobacter pylori; Nasal polyposis; Extraesophageal reflux; Chronic rhinosinusitis*

## Introduction

*Helicobacter pylori* (HP) is the cause of one of the most common chronic bacterial infections in human population, present in countries all over the world. It is estimated that more than 50% of adult population in developed countries suffer from this infection[1]. The mode of HP infection spread has not yet been fully clarified[2]. Given the evidence of high HP seroprevalence and infection rates, this organism may also play role in the etiology of extra-gastrointestinal disease such as low respiratory disorders (e.g., chronic obstructive pulmonary disease, bronchiectasis, bronchial asthma), upper aerodigestive disorders (chronic rhinosinusitis, nasal polyps, otitis media with effusion), vascular disorders, autoimmune disorders, and other conditions[3-5]. Major scientific efforts are invested all over the world in exploring the possibilities of diagnosing and treating this infection[6].

Chronic rhinosinusitis is one of the most frequently encountered diseases throughout the world. The pathophysiology of chronic rhinosinusitis involves inflammatory mucosal changes, which result in mucosal edema, ostial obstruction, mucus stasis, and subsequent infection. Chronic rhinosinusitis is a multifactorial disease with well recognized predisposing factors, which include viral, bacterial and/or fungal infections, inhalation of allergens, and pollution. Nasal polyposis (NP) is considered to result from chronic inflammation and there are two significant factors to NP increasing prevalence. One is advancement of diagnostics, nasal and paranasal cavity endoscopy, and computer tomography (CT) scans of those areas. The other factor is the increasing incidence of chronic inflammatory diseases of the respiratory system which nasal polyps are frequently in conjunction with (aspirin intolerance, asthma, chronic sinusitis, cystic fibrosis, allergic fungal sinusitis, primary ciliary dyskinesia)[7]. Despite the fact that NP has been known for so long, it still remains a great enigma and the exact cause of the condition is unknown. However, there are two basic stand points on the emergence of polyps, i.e., allergy and infection[8].

Extraesophageal reflux (EER) is a result of retrograde flow of gastric contents through insufficient upper esophageal sphincter where it comes in contact with the upper aerodigestive tract tissue. Increasing evidence has demonstrated that EER is contributing factor in some cases of hoarseness, voice fatigue, benign vocal cord lesions, subglottic stenosis, chronic cough, asthma, pneumonia, otitis media with effusion, and chronic rhinosinusitis with NP. The mechanism of how EER may contribute to NP is still a controversial topic[9-11].

Recently, some reports on the high prevalence of HP in chronic rhinosinusitis and NP have been published[12-17]. Although intranasal colonization and association of HP with a number of pharyngeal and laryngeal diseases have been previously investigated, false-negative and/or positive results have to be considered due to insufficient specificity and sensitivity of the commonly used testing methods[18-20].

We investigated the relationship between HP colonization of healthy nasal mucosa and nasal polyps in patients with HP infections in gastric mucosa and EER.

## Patients and Methods

A cross-sectional study with a control group was performed at the Department of Otorhinolaryngology, Head and Neck Surgery, Osijek University Hospital Center, Osijek, Croatia. Study design was approved by the Ethics Committees of the Osijek University Hospital Center and Osijek Faculty of Medicine, Josip Juraj Strossmayer University in Osijek (no. 29-1:3388-9/2005).

Case group consisted of 35 patients with clinically, radiologically (CT scan) and histologically diagnosed NP. Control group included 30 patients with bullous middle nasal concha (CB) confirmed with CT scan. Patients with other nasal breathing impairments were not included in the study. Patients with ulcer disease, chronic atrophic gastritis with a history of taking H2 blockers, antacids or proton pump inhibitors one week prior to surgical procedures or antibiotics within four weeks were excluded from the research.

After clinical examination, all study subjects, cases and controls, filled out two assessment tools. The first one contained queries on the symptoms of EER. We used a common assessment tool, Reflux Symptom Index (RSI), in diagnosis and treatment. Patients were asked to rate how 9 problems had affected them over the past month on a scale of 0 (no problem) to 5 (severe problem), with a maximum total score of 45. A total score of more than 13 is considered positive for diagnosis of EER (Table 1). The second assessment tool, Sino-Nasal Outcome Test (SNOT-20), contains 20 questions. In this study, we included questions related to problems caused by NS, i.e., nasal breathing difficulties, nasal secretion, postnasal drip, sneezing, loss of smell, and snoring. The SNOT-20 is a validated instrument which is scored using a Likert scale where 0=No problem, 1=Very mild problem, 2=Mild or slight problem, 3=Moderate problem, 4=Severe problem, and 5=Problem as bad as it can be.

All subjects, both with NP and with CB, underwent esophagogastroduodenoscopy with routine gastric antrum and corpus biopsies prior to endoscopic sinus surgery (ESS). ESS procedures, polypectomy and conchotomy, were performed in local intensified anesthesia and multiple biopsies of nasal mucosa were taken during the procedure in both study groups for histopathologic analysis and real time polymerase chain reaction (RT PCR) test. All specimens were examined by one experienced pathologist from the Department of Pathology, Osijek University Hospital Center, Osijek, Croatia, who was unaware of the clinical diagnosis and RT PCR test results.

**TABLE 1.** Reflux symptom index (adopted from Belafsky *et al*.[23])

| | | |
|---|---|---|
| 1 | Hoarseness or a problem with your voice | 0 1 2 3 4 5 |
| 2 | Clearing your throat | 0 1 2 3 4 5 |
| 3 | Excess throat mucous or postnasal drip | 0 1 2 3 4 5 |
| 4 | Difficulty swallowing food, liquids, or pills | 0 1 2 3 4 5 |
| 5 | Coughing after you ate or after lying down | 0 1 2 3 4 5 |
| 6 | Breathing difficulties or choking episodes | 0 1 2 3 4 5 |
| 7 | Troublesome or annoying cough | 0 1 2 3 4 5 |
| 8 | Sensation of something sticking in your throat or a lump in your throat | 0 1 2 3 4 5 |
| 9 | Heartburn, chest pain, indigestion or stomach acid coming up | 0 1 2 3 4 5 |

## Real time polymerase chain reaction

DNA was isolated from nasal polyp tissue samples, mucosa of bullous middle nasal concha, and mucosa of stomach antrum and corpus using the QIAGEN DNeasy tissue kit mini-spin columns according to the manufacturer's instructions (QIAGEN, Hilden, Germany). Leachates potentially containing HP DNA were analyzed using the standard fluorescent ABI Helicobacter plus-minus PCR assay, and final fluorescence values were read using the ABI Prism 7000 RT PCR device and analyzed with the appertaining software[18].

### Ethical statement

Before participation, each study subject signed an informed written consent form that was approved by the Ethics Committee of the Osijek University Hospital Center.

### Statistical analysis

Statistical analysis was performed with SPSS for Windows, version 15.0. (Chicago, IL, USA) using Pearson $\chi^2$-test. The level of statistical significance was set at $p<0.05$.

## Results

Median age in the case and control groups was 54 (range 27-78) and 43 (range 19-75) years, respectively. There were 71% of men in the case group and 40% in the control group. All subjects, cases and controls, had nasal breathing difficulties. Patients with NP reported significantly more NS than patients with CB except for loss of smell (Table 2).

Overall, 21 (38.1%) subjects had a history of allergies in the upper respiratory tract, 18 (29%) were smokers, and 33 (50.8%) had EER (Table 3). The occurrence of allergies was significantly

**TABLE 2.** Difference in nasal symptoms between case and control group

| Nasal symptom | Case (%) | Control (%) | p |
|---|---|---|---|
| Nasal breathing difficulty | 35 (100) | 30 (100) | - |
| Nasal secretion | 20 (57.1) | 6 (20) | 0.003 |
| Sneezing | 30 (85.7) | 3 (13.3) | <0.001 |
| Postnasal drip | 25 (71.4) | 3 (10) | <0.001 |
| Loss smell | 30 (85.7) | 30 (100) | 0.035 |
| Snoring | 30 (85.7) | 5 (16.7) | <0.001 |

**TABLE 3.** Difference in clinical data and histopathologic and RT PCR results of study cases and controls

| | Case (%) | Control (%) | p |
|---|---|---|---|
| EER (RSI) | 24 (70.0) | 9 (30.0) | 0.003 |
| History of allergies | 18 (51.4) | 3 (10.0) | 0.001 |
| Smoking | 8 (22.8) | 8 (26.6) | 0.723 |
| HP PCR positive nasal mucosa | 10 (28.8) | 0 | 0.001 |
| HP PCR positive gastric mucosa | 35 (100) | 30 (100) | - |
| HP histology positive gastric mucosa | 26 (74.3) | 18 (60.0) | 0.224 |

EER (RSI) = extraesophageal reflux (Reflux Symptom Index); HP = *Helicobacter pylori*; PCR = polymerase chain reaction

more frequently reported by patients with NP than in control group (51.4% *vs*. 10.0%; $\chi^2$-test, p=0.001). The proportion of patients with reported EER was significantly higher among patients with NP (70.0% *vs*. 30.0%; $\chi^2$-test, p=0.003) (Table 3). In 10 (28.8%) of 35 patients with NP, HP was identified in nasal polyp tissue. All these patients had both NS and EER. In control group, HP was not detected in the mucosa of the middle concha specimens (Table 3). All subjects, patients and controls, had positive RT PCR results for HP in gastric mucosa (Table 3). In 74.3% of patients with NP, histologic analysis of the gastric biopsy material was positive for HP and chronic active gastritis. In control group, that proportion was 60%, and this difference was not statistically significant ($\chi^2$-test, p=0.224) (Table 3).

## Discussion

Our study confirmed the presence of HP in nasal polyp mucosa in patients with HP positive gastric mucosa and EER. In patients with NP and HP positive gastric mucosa without EER we did not detect the presence of HP in nasal polyp specimens. These results suggest correlation between the presence of EER and pathologic process, which may cause bacterial colonization. The presence of HP in nasal polyp mucosa may be a cofactor in various inflammatory processes and may have implications for the possible role in the development of nasal polyp. Upper aerodigestive tract has reduced defense to gastric contents and direct reflux irritation causes mucosal damage. The major aggressors are acid, pepsin, and bacteria. The resultant mucosal edema and ciliary dysfunction cause mucus stasis and accumulation, which produces postnasal drip sensation and provokes various local persistent or progressive inflammatory processes. The presence of HP in vulnerable nasal mucosa can induce hypoxia and acid environment which facilitate stronger growth of this microorganism. HP may also play an antigenic role which evocates infiltration of inflammatory cells and release of chemical and inflammatory mediators[16]. Results of our study also demonstrate that healthy nasal mucosa is not a preferential site for HP colonization. In all patients with CB with and without EER, the RT PCR results were negative for HP in nasal mucosa. These negative results suggest that normal nasal mucosa acts as a barrier to HP infections. Dinis and Subtil investigated the potential role of EER content in chronic sinus disease. They found similar presence of HP in healthy and diseased sinus mucosa[16]. Nemati *et al.* believe that HP colonization in NP is an accidental finding, and there is no relationship between this microorganism and NP in cases without gastroesophageal reflux disease (GERD)[21]. Ozmen *et al.* investigated the relationship between chronic rhinosinusitis and laryngopharyngeal reflux (LPR) using nasal pepsin assay. The results indicated that patients with chronic rhinosinusitis had a higher incidence of LPR than control patients.

Evaluation of pepsin in nasal lavage supported these results. Our study suggests that EER may have significant implications in colonization of nasal mucosa with HP. The role of EER was not considered in many other studies as an exclusion criterion and in interpreting the results. The signs and symptoms are not specific and it is sometimes difficult to distinguish EER from GERD. Up to 50% of patients presenting with EER may not have classic reflux symptoms such as heartburn and regurgitation. EER includes LPR and reflux that occurs at other sites of upper aerodigestive tract. Belafsky *et al.* developed a useful self-administered tool, the RSI that can help clinically assess the relative degree of LPR during initial evaluation and treatment[22]. The relationship between HP and GERD has been well studied but opposite data are continuing to be gathered. Saruc

*et al.* suggest that HP status has no effect on the development of LPR[23].

Recently, several techniques have been introduced into the HP diagnostic procedures, with different sensitivity and specificity. It is difficult to diagnose HP when low quantities of organisms or unusual forms exist or when antibiotics or other drugs are used[24]. Therefore, multiple diagnostic methods may be necessary to prevent false results or we have to apply highly specific method to demonstrate HP, and that is PCR. Koc *et al.* demonstrated a significant statistical difference between the cases with NP and controls, and found HP present only in 20% of cases based on immunohistochemistry[25]. Szczygelski *et al.* did not find HP in NP using urease test[26]. Kaviani *et al.* detected HP in only 8% of NP cases, with weak correlation between HP colonization and NP. Kaviani *et al.* suggest that culture and PCR are the best methods for HP determination[27]. Huang *et al.* emphasize the importance of objective measurements, PCR and ELISA results, and immunohistochemical evaluation as diagnostic tools[28]. The advantage of PCR is that it can detect even dead bacteria in the specimen, while the bacterium in the specimen must be alive for positivity of culture. Ozyurt *et al.*[29] clearly indicated the superiority of RT PCR systems over standard one-step in-house PCR for the detection of HP DNA from tissues.

## Conclusion

Our data strongly suggest that the stomach is the primary HP reservoir and HP is transferred to the nasal cavity by EER and injury of nasal mucosa. Damaged nasal mucosa colonization by HP can be considered as an etiologic factor for nasal polyposis. In patients with healthy nasal mucosa, HP is not a normal commensal regardless of EER. The clinical perspective of this study is the importance of HP eradication. The treatment of HP and EER can block the harmful effects on nasal mucosa and reduce the possibility of nasal polyposis development. ■

## References


1. Eusebi LH, Zagari RM, Bazzoli F. Epidemiology of *Helicobacter pylori* infection. Helicobacter. 2014;1:1-5.
2. Kayali S, Manfredi M, Gaiani F, Bianchi L, Bizzarri B, Leandro G, *et al. Helicobacter pylori*, transmission routes and recurrence of infection state of art. Acta Biomed. 2018;89(8):72-6. doi: 10.23750/abm.v89i8-S.7947.
3. Ambartsumyan L, Nurko S, Rosen R. Gastrointestinal dysmotility and the implications for respiratory disease. Curr Treat Options Pediatr. 2019;5(2):197-214.
4. Song Q, Lange T, Spahr A, Adler G, Bode G. Characteristic distribution pattern of *Helicobacter pylori* in dental plaque and saliva detected with nested PCR. J Med Microbiol. 2000;49(4):349-53. doi: 10.1099/0022-1317-49-4-349.
5. Pajić-Penavić I, Danic D, Maslovara S, Gall-Trošelj. Absence of *Helicobacter pylori* in healthy laryngeal mucosa. J Laryngol Otol. 2012;126(2):196-9. doi: 10.1017/S0022215111002799.
6. Fischbach F, Malfertheiner P. *Helicobacter pylori* infection. Dtsch Arztebl Int. 2018;115:(25):429-36. doi: 10.3238/arztebl.2018.0429.
7. Chaaban MR, Walsh EM, Woodworth BA. Epidemiology and differential diagnosis of nasal polyps. Am J Rhinol. 2013;27(6):473-8. doi: 10.2500/ajra.2013.27.3981.
8. Bachert C, Pawankar R, Zhang L, Bunnag C, Fokkens WJ, Hamilos DL, *et al.* ICON: chronic

rhinosinusitis. World Allergy Organ J. 2014;7(1):25. doi: 10.1186/1939-4551-7-25.
9. Cvorovic L, Brajovic D, Strbac M, Milutinovic Z, Cvorovic V. Detection of *Helicobacter pylori* in nasal polyps: preliminary report. J Otolaryngol Head Neck Surg. 2008;37(2):192-5.
10. Zeleník K, Matoušek P, Formánek M, Urban O, Komínek P. Patients with chronic rhinosinusitis and simultaneous bronchial asthma suffer from significant extraesophageal reflux. Int Forum Allergy Rhinol. 2015;5(10):944-9.
11. Pearson JP, Parikh S. Review article: nature and properties of gastro-oesophageal and extra-oesophageal refluxate. Aliment Pharmacol Ther. 2011;33(1):1-71. doi: 10.1111/j.1365-2036.2011.04581.
12. Zika G, Fousekis FS, Exarchakos G, *et al.* Detection of *Helicobacter pylori* in nasal polyps: an epidemiological study. Indian J Otolaryngol Head Neck Surg. 2023;75(Suppl 1):1084-9. https://doi.org/10.1007/s12070-023-03585-9.
13. Siupsinskiene N, Katutiene I, Jonikiene V, Jancauskas D, Vaitkus S. Intranasal *Helicobacter pylori* infection in patients with chronic rhinosinusitis with polyposis. J Laryngol Otol. 2018;132(9) 816-21.
14. Kariya S, Okano M, Nishizaki K. An association between *Helicobacter pylori* and upper respiratory tract disease: fact or fiction? World J Gastroenterol. 2014;20(6):1470-84.
15. Sahin E, Katar MK, Can IH. Impact of gastric *Helicobacter pylori* infection on nasal mucociliary clearance. Eur Arch Otorhinolaryngol. 2020;277(10):2761-5. doi: 10.1007/s00405-020-06089-2.
16. Dinis PB, Subtil J. *Helicobacter pylori* and laryngopharyngeal reflux in chronic rhinosinusitis. Otolaryngol Head Neck Surg. 2006;134:67-72.
17. Ozdek A, Cirak MY, Samim E, Bayiz U, Safak MA, Turet S. A possible role of *Helicobacter pylori* in chronic rhinosinusitis: a preliminary report. Laryngoscope 2003; 113(1):679-82. doi: 10.1016/j.otohns.2005.10.013.
18. Megraud F, Lehours P. *Helicobacter pylori* detection and antimicrobial susceptibility testing. Clin Microbiol Rev. 2007;20(2):280-322. doi: 10.1128/CMR.00033-06.
19. Akbayir N, Basak T, Seven H, Sungun A, Erdem L. Investigation of *Helicobacter pylori* colonization in laryngeal neoplasia. Eur Arch Otorhinolaryngol. 2005;262(3):170-2. doi: 10.1007/s00405-004-0794-0.
20. Eyigor M, Eyigor H, Gultekin B, Aydin N. Detection of *Helicobacter pylori* in adenotonsillar tissue specimens by rapid urease test and polymerase chain reaction. Eur Arch Otorhinolaryngol. 2009;266(10):1611-3. doi: 10.1007/s00405-008-0903-6.
21. Nemati S, Mojtahedi A, Naghavi SE, Banan R, ZiaF. Investigating *Helicobacter pylori* in nasal polyposis using polymerase chain reaction, urease test and culture. Eur Arch Otohrinolaryngol. 2011;268(5):1848-8. doi: 10.1007/s00405-011-1848-8.
22. Belafsky PC, Postma GN, Koufman JA. The validity and reliability of the reflux finding score (RFS). Laryngoscope. 2001;111(8):1313-7. doi: 10.1097/00005537-200108000-00001.
23. Saruc M, Aksoy EA, Vardeteli E, Karaaslan M, Cicek B, Ince U, Oz F, Tozun N. Risk factors for laryngopharyngeal reflux. Eur Arch Otorhinolaryngol. 2012;269(4):1189-94. doi: 10.1007/s00405-011-1905-3.
24. Morinaka S, Ichimiya M, Nakamura H. Detection of *Helicobacter pylori* in nasal and maxillary sinus specimens from patients with chronic sinusitis. Laryngoscope. 2003;113(9):1557-63. doi: 10.1097/00005537-200309000-00027.
25. Koc C, Arikan OK, Atasoy P, Aksoy A. Prevalence of *Helicobacter pylori* in patient with nasal polyps: a preliminary report. Laryngoscope. 2004;114(11):1941-4. doi: 10.1097/01.mlg.0000147924.96980.34.
26. Szczygielski K, Jurkiewicz D, Rapiejko P. Detection of *Helicobacter pylori* in nasal polyps specimens using urease test GUT plus. Pol Merkuriusz Lek. 2005;19(111):309-11.
27. Kaviani M, Khademi B, Mousavi SA, Azarpira N, Mohammadjavad A. Determination of *Helicobacter pylori* in nasal polyposis with use of rapid urease test and ELISA. Iran J Otorhinolaryngol. 2009;20:189-93.
28. Huang Y, Gu M, Wu Q, Zhu J, Wu J, Wang P, Wang M, Luo J. Is laryngeal squamous cell carcinoma related to *Helicobacter pylori*? Front Oncol. 2022;28(12):790997. doi: 10.3389/fonc.2022.790997.
29. Ozyurt M, Gungor A, Ergunay K, Cekin E, Erkul E, Haznedaroglu T. Real-time PCR detection of *Helicobacter pylori* and virulence-associated cagA in nasal polyps and laryngeal disorders. Otolaryngol Head Neck Surg. 2009;141(1):131-5. doi: 10.1016/j.otohns.2009.04.005.

**SAŽETAK**

## *Helicobacter pylori* kao etiološki čimbenik razvoja nosne polipoze u bolesnika s ekstraezofagusnim refluksom

Andrijana Včeva, Ivan Abičić, Tihana Mendeš, Martina Smolić, Robert Smolić, Aleksandar Včev i Željko Zubčić

Cilj ove presječne studije bio je identificirati *Helicobacter pylori* (HP) kao etiološki čimbenik za razvoj nosne polipoze (NP) u bolesnika s ekstraezofagusnim refluksom (EER). U istraživanje je bilo uključeno 35 bolesnika s NP i 30 ispitanika s buloznom srednjom nosnom školjkom (CB) u kontrolnoj skupini kojima je bila indicirana endoskopska operacija sinusa (ESS). Svi ispitanici su podvrgnuti ezofagogastroendoskopiji s rutinskom biopsijom antruma i tijela želuca te su ispunili dva upitnika, o EER-u i nazalnim simptomima prije ESS-a. Svi uzorci biopsije želuca i nosa su patohistološki i pomoću RT PCR pregledani na HP. Svi ispitanici u obje skupine imali su pozitivan nalaz na HP u sluznici želuca, a 33 (50,8%) ispitanika imalo je EER. Udio bolesnika s prijavljenim EER bio je značajno veći među bolesnicima s NP (70,0% prema 30,0%; $\chi^2$-test, p=0,003). HP je identificiran u sluznici nosa u 10 (30,3%) bolesnika s NP, a svih 10 bolesnika imalo je EER i nazalne simptome. HP nije otkriven niti kod jednog bolesnika u kontrolnoj skupini s CB. HP nije normalna pojava u zdravoj sluznici nosa. U oko 40% bolesnika s polipima nosa i EER HP se može detektirati u sluznici nosa. HP se može smatrati etiološkim čimbenikom u bolesnika s nosnom polipozom i EER-om, iako su potrebne daljnje studije za procjenu ove povezanosti.

**KLJUČNE RIJEČI**

*Helicobacter pylori; Nosna polipoza; Ekstraezofagusni refluks; Kronični rinosinuitis*