

Breastfeeding and *Helicobacter Pylori* Infection in Children with Digestive Symptoms

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Received: Jul 22, 2009; Final Revision: Dec 27, 2009; Accepted: Apr 30, 2010

Abstract

Objective: This study aims to evaluate the role of breastfeeding in the acquisition of *Helicobacter pylori* (*H. pylori*) infection in Iran and to compare the histopathologic changes occurring in children feeding on breast milk with those in infants feeding on formula.

Methods: In a case-control study parents of children with and without *H. pylori* infection who had undergone endoscopic survey and gastric biopsy in the Children's Medical Center, Tehran, were asked about their feeding practices during the first 6 months after birth, the duration of breastfeeding period, the symptoms, and the duration of symptoms and concomitant diseases.

Findings: A total of 154 children were included in this study. From this sample, 77 children formed the case group and 77 children formed the control group. A significant difference was found between *H. pylori* infection and feeding with formula ($P=0.045$). In case group, a significant difference was found between breastfeeding and age of the infected child ($P=0.034$), shorter duration of symptoms ($P=0.016$), and finally degree of *H. pylori* colonization ($P=0.021$).

Conclusion: It appears that breastfeeding in the first 6 months after birth can decrease the degree of *H. pylori* colonization, postpone infection until older age, shorten the duration of symptoms, and be concomitant with milder gastritis.

Iranian Journal of Pediatrics, Volume 20 (Number 3), September 2010, Pages: 330-334

Key Words: Breastfeeding; Gastritis; *Helicobacter pylori*; Formula

Introduction

Helicobacter pylori, a gram negative bacillus, is recognized as the main etiological agent of

several gastroduodenal diseases including peptic ulcer and gastric malignancies. Breastfeeding, while providing protection against many

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infective agents, can facilitate the acquisition of *H. pylori* infection. This infection occurs as a result of the close contact between breastfeeding child and the infected mother.

Some studies have shown breastfeeding's protective effect against the acquisition of *H. pylori* infection^[1-4]. Contrastingly, other studies have reported that breastfeeding does not have a protective effect against the acquisition of *H. pylori* infection^[5-7]. Furthermore, these studies have suggested that breastfeeding can even increase the rate of infection, especially in children breastfed for longer than 6 months^[8].

Such different results might stem from the differences in the social and cultural practices of breastfeeding. Despite this plausible explanation, no studies have yet explored the role of breastfeeding in the acquisition of *H. pylori* infection in Iran and also most studies did not pay attention to histopathological changes in stomach. Therefore, this study aimed to evaluate the role of breastfeeding in the acquisition of *H. pylori* infection in Iran and to compare the histopathologic changes occurring in children feeding on breast milk and in those feeding on formula.

Subjects and Methods

This is a case-control study carried out from March 2007 to February 2009. Participants consisted of children aged 2-14 years who were diagnosed with digestive symptoms, especially abdominal pain and undergone endoscopic survey and gastric biopsy in the Children's Medical Center. Patients with a history of chronic debilitating disease were excluded and all cases and controls suffered mainly from abdominal pain but were in good general condition according to weight, history and absence of chronic disease stigmata. Children whose breastfeeding status was unknown or feeding consisted of a mixture of breast milk and formula were excluded from the study. The *H. pylori* infection was detected by Gimsa staining of the gastric biopsy and scored by Borshyang Sheu's scoring^[9]. Feeding practice was

defined as breastfeeding exclusively in the first 6 months of life and formula feeding in this time period. Patients were placed in each group according to their history. We also conducted a pilot study in order to estimate the prevalence of formula consumption in Iran in infected and non-infected children by *H. pylori*, and discovered that in infected children 33.3% and in children who were not infected 14% consumed formula. We used these findings to determine the volume of our case and control groups. Hence, our case group consisted of 77 consecutive children infected by *H. pylori* and the control group consisted of 77 consecutive children not infected by this germ. We also asked the parents about their breastfeeding practices during the first 6 months after birth, the duration of breastfeeding period, the symptoms, and the duration of symptoms and concomitant diseases.

In this study the Pearson's χ^2 test and the Fisher's exact test were both employed to assess the relationship between *H. pylori* infection and variables such as age, gender, kind of feeding, duration of breastfeeding, symptoms and signs, duration of symptoms and signs, concomitant diseases, histopathologic changes in the stomach, concomitant histopathologic changes and the degree of *H. pylori* colonization.

Statistical analysis was performed using SPSS 14 software (SPSS Inc., Chicago, IL, USA).

Findings

A total of 154 children were included in this study. From this sample, 77 children formed the case group (40 boys and 37 girls) and 77 the control group (42 boys and 35 girls). Children whose breastfeeding status was unknown or consisted of a mixture of breast milk and formula were excluded from the study.

Table 1 and 2 show gender, mean age, kind of feeding, duration of breastfeeding, symptoms and signs, concomitant diseases, histopathologic changes in the stomach, concomitant histopathologic changes and the degree of *H. pylori* colonization in the case and control groups.

Table 1: Gender, mean age, kind of feeding, duration of breastfeeding, symptoms and signs, concomitant diseases in the case and control groups

		Case group (HP positive)		Control group (HP negative)		Total	P value or Exact
Kind of feeding		Breast milk 64 (83.1%)	Formula 13 (16.9%)	Breast milk 72 (93.5%)	Formula 5 (6.5%)	154	0.04 ¹
Gender	Female	31 (20.1%)	6 (3.9%)	33(21.4%)	2(1.3%)	72(46.75%)	0.7 ¹
	Male	33 (21.4%)	7 (4.5%)	39(25.3%)	3(1.9%)	82(53.25%)	
Mean age (year)		8.1	6	6.2	8		0.006 ¹
		P=0.03		P=0.2			
Duration of breastfeeding (months)		15		11.75			0.4 ¹
Symptoms and signs	Abdominal pain	49	8	45	3	105	0.2 ¹
	GI bleeding	3	0	4	0	7	0.7 ¹
	Vomiting	10	2	18	0	30	0.2 ¹
	FTT	6	1	21	0	28	0.049 ¹
Presence of concomitant non-debilitating diseases		5	1	10	2	18	0.5 ²

¹ Pearson's χ^2 test² Fisher's exact testFTT: Failure to Thrive / HP: *Helicobacter pylori*

A significant association was found between *H. pylori* infection and feeding with formula ($P=0.04$; Odd's ratio=2.92) and association of *H. pylori* infection and age ($P=0.006$). In case group, a significant difference was found between breastfeeding and the age of infected child ($P=0.03$), breastfeeding and duration of symptoms ($P=0.02$), and finally, breastfeeding and *H. pylori* colonization degree (Exact value=0.02).

The more common gastric histopathologic changes in the case group were moderate gastritis ($P=0.03$), active gastritis ($P=0.006$) and follicular gastritis ($P<0.001$).

More common histopathologic changes of stomach in control group were normal stomach ($P<0.001$) and mild gastritis ($P=0.001$).

There was no significant difference between case and control group in the duration of breastfeeding ($P=0.4$), sex ($P=0.7$), type of symptoms, concomitant diseases (Exact value=0.5), duration of symptoms ($P=0.7$), severe gastritis (Exact value=0.12), peptic ulcer (Exact value=1), esophagitis ($P=0.4$), duodenitis ($P=0.676$), duodenal villous atrophy ($P=0.4$), Giardia infection (Exact value=1) and duodenal ulcer (Exact value=1).

Discussion

Acquisition of *H. pylori* takes place within the first five years of life^[10] and can produce severe inflammatory and immune responses, chronic gastritis, duodenal ulcer and mucosa-associated lymphoid tissue (MALT). Even though breastfeeding has a protective effect on a variety of infant illnesses, it also has the potential to increase the risk for *H. pylori* infection by placing the breastfed children in close contact with the infected mother. Such paradoxical findings motivated us to specifically investigate the association between type of feeding in infancy (breastfeeding or formula) and *H. pylori* infection.

Some studies have shown breastfeeding's protective effect against the acquisition of *H. pylori* infection^[1-4], while other studies have reported that breastfeeding does not have a protective effect against the acquisition of *H. pylori* infection^[5-7]. Furthermore, these studies suggest that breastfeeding can even increase the rate of infection, especially in children breastfed for longer than 6 months^[8]. This study was successful in determining the protective effect of breastfeeding history on *H. pylori* infection in

Table 2: Histopathologic changes in the stomach, concomitant histopathologic changes and the degree of *H. pylori* colonization in the case and control groups

		Case group (HP positive)		Control group (HP negative)		Total	P-value or Exact
		Breast milk	Formula	Breast milk	Formula		
Histopathologic changes in stomach	Normal	1	0	24	0	24	<0.001 ¹
	Mild gastritis	11	1	27	4	42	0.001 ¹
	Intermediate gastritis	10	0	2	0	12	0.03 ¹
	Severe gastritis	3	0	0	0	4	0.1 ²
	Active gastritis	7	1	0	0	13	0.006 ¹
	Follicular gastritis	31	7	11	0	42	<0.001 ¹
	Peptic ulcer	0	0	1	0	1	1 ²
Concomitant histopathologic changes	Esophagitis	38	4	44	3	89	0.4 ¹
	Duodenitis	12	1	15	0	28	0.7 ¹
	Duodenal villous atrophy	4	1	8	0	13	0.4 ¹
	Giardia infection	1	0	0	0	1	1 ²
	Duodenal ulcer	1	0	0	0	1	1 ²
Degree of <i>H. pylori</i> colonization	Mild	31.25	33.33				
	Moderate	31.25	0				0.02 ²
	Severe	37.5	66.67				

¹ Pearson's χ^2 test² Fisher's exact testHP: *Helicobacter pylori*

children of Iran and was in parallel with Chak study in 2009^[11]. The possible mechanisms enrolled in this protection may be the lactoferrin in human milk which binds to *H. pylori* liposaccharide inactivating the organism^[12]. Also, Clyne et al showed that breast milk inhibits *H. pylori* adherence to gastric adenocarcinoma cell line *in vitro*^[13].

One important point of our study is the evaluation of histological changes and separating their severity into two groups. This is not done in other studies. We showed that it is more common to see more severe gastritis and also more *H. pylori* organisms (more colonization) in gastric biopsies of formula fed children which may be due to lactoferrin effect as mentioned above or it may be due to *H. pylori*-specific IgA present in breast milk^[14], which formula lacks.

Our study had some limitations: it was conducted on a small sample of children suffering from digestive symptoms; therefore, the results might have been different if healthy children were taken into the study. Additionally,

we used histopathologic changes in biopsy in order to evaluate *H. pylori* infection status in infants without having any knowledge regarding the *H. pylori* infection status of the mothers.

Conclusion

Our study reveals valuable information about the role of breastfeeding in protection against *H. pylori* infection. More specifically, it appears that breastfeeding in the first 6 months after birth may decrease the degree of colonization, postpone infection until older ages, shorten the duration of symptoms, and be concomitant with milder gastritis.

As a result, we can recommend breastfeeding as a possible reliable method to prevent the consequences of *H. pylori* infection: gastritis, duodenal ulcer and lymphoma. It must also be mentioned, that breastfeeding may have a

protective effect only in the first year of life and after that, it can increase *H. pylori* infection by facilitating mother-child transmission, especially if the mother doesn't have good hygienic habits.

To achieve more accurate results, it is recommended to do similar studies on a larger sample, which includes both healthy and infected children.

Acknowledgment

The financial support of this study was provided by Tehran University of Medical Sciences. The authors have no competing interests.

Conflict of Interest: None

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