Standard Scientific Research and Essays Vol 3(10): 298-301, October 2015 (ISSN: 2310-7502)
http://www.standresjournals.org/journals/SSRE

Research Article

Sensitivity and Specificity Techniques for Diagnosis of *Helicobacter pylori* in Middle Jordan, non-invasive techniques based study

***Nawal S Faris (PhD) and Hasem A Abu-Huriarh**

Department of Allied medical sciences, Zarqa University, Jordan

*Corresponding Author's E-mail: nawal_fares94@hotmail.com

Accepted 27 October 2015

Abstract

***Helicobacter pylori* is a ubiquitous organism that is present in about 50% of the global population. Chronic infection with *H pylori* causes atrophic and even metaplastic changes in the stomach, and it has a known association with peptic ulcer disease. *H. pylori* causes chronic gastritis and has been associated with several serious diseases of the gastrointestinal tract, including duodenal ulcer and gastric cancer The most common route of *H pylori* infection is either oral-to-oral or fecal-to-oral contact. The discovery of *Helicobacter pylori* in 1982 was the starting point of a revolution concerning the concepts and management of gastroduodenal diseases. Most of the many different techniques involved in diagnosis of *H. pylori* infection are performed in clinical laboratories. This research will be carried out in several hospitals and private lab in Jordan. The aim of this study is determine comparison between the sensitivity and specificity of these methods and their application. Noninvasive techniques will be reviewed because the noninvasive techniques are the most common tests applied in private labs and hospitals**

Keywords: *H. pylori,* sensitivity, specificity and non-invasive techniques

INTRODUCTION

At least half the world's population is infected by the bacterium, making it the most widespread infection in the world (Allaker et al., 2002). Actual infection rates vary from nation to nation; the developing world has much higher infection rates than the West (Western Europe, North America, Australasia), where rates are estimated to be around 25%(Allaker et al., 2002). The age at which this bacterium is acquired seems to influence the possible pathologic outcome of the infection: people infected with it at an early age are likely to develop more intense inflammation that may be followed by atrophic gastritis with a higher subsequent risk of gastric ulcer, gastric cancer, or both. Acquisition at an older age brings different gastric changes more likely to lead to duodenal ulcer (Frenck and Clemens,2003). Infections are usually acquired in early childhood in all countries (Zhang et al., 2006). However, the infection rate of children in developing nations is higher than in industrialized nations, probably due to poor sanitary conditions, perhaps combined with lower antibiotics usage for unrelated pathologies. In developed nations, it is currently uncommon to find infected children, but the percentage of infected people increases with age, with about 50% infected for those over the age of 60 compared with around 10% between 18 and 30 years (Zhang et al., 2006). The higher prevalence among the elderly reflects higher infection rates in the past when the individuals were children rather than more recent infection at a later age of the individual.[13] In the United States, prevalence appears to be higher in African-American and Hispanic populations, most likely due to socioeconomic factors (Zhang et al., 2006; IDSA, 2013). The lower rate of infection in the West is largely attributed to higher hygiene standards and widespread use of antibiotics. Despite high rates of infection in certain areas of the world, the overall frequency of *H. pylori* infection is declining (Bina et al., 2000). However, antibiotic resistance is

appearing in *H. pylori*; many metronidazole- and clarithromycin-resistant strains are found in most parts of the world.

H. pylori is contagious, although the exact route of transmission is not known(Allaker et al., 2002). Person-to-person transmission by either the oral-oral or fecal-oral route is most likely. Consistent with these transmission routes, the bacteria have been isolated from feces, saliva, and dental plaque of some infected people. Findings suggest *H. pylori* is more easily transmitted by gastric mucus than saliva (Calvet et al., 2004). Transmission occurs mainly within families in developed nations, yet can also be acquired from the community in developing countries (Calvet et al., 2004). *H. pylori* may also be transmitted orally by means of fecal matter through the ingestion of waste-tainted water, so a hygienic environment could help decrease the risk of *H. pylori* infection

In developing countries, diseases due to *H. pylori*, e.g., peptic ulcer and gastric cancer, are very common. In children, *H. pylori* infection may be a risk factor for diarrheal diseases (Allaker et al., 2002). Two criteria are crucial in the decision making for diagnosing *H. pylori* infection: the prevalence of the infection and the cost of the tests in relation to the health resources available (Feldman et al., 1995). Recent data have shown that the number of bleeding ulcers has not decreased these last years despite the decrease in *H. pylori* infection. This may be due to a higher consumption of nonsteroidal anti-inflammatory drugs, including aspirin, and anticlotting agents(Faulde et al.,1992). The diagnosis of *H. pylori* infection is important, but there are limited possibilities to take biopsy specimens, and diagnostic tests are considered less accurate in this context. Urease test, culture, and histology, as well as UBT(Bilardi et al., 2002). Lack sensitivity, while the polyclonal stool antigen test has a lower specificity than usual conditions(Faulde et al.,1992). Serology should remain the preferred method to be used in this context because it is not affected by the local environment (Demirturk et al., 2003).

METHODS

Between May 2015 and September 2015, all patients referred from the Gastro-esophageal Clinic to the private labs and hospitals at Amman capital of Jordan. For each patient blood sample was collected andBlood antibody test was performed. Blood tests are used to measure antibodies to H pylori A blood test checks to see whether your body has made antibodies to *H. pylori* bacteria. If you have antibodies to *H. pylori* in your blood, it means you either are currently infected or have been infected in the past. *H. pylori* infection almost constantly induces a specific systemic immune response which may reflect the antibodies produced at the gastric mucosal level, while only 2% of patients fail to seroconvert (276). The immune response varies according to the antigens present in the infecting strains and to the host. A partial purification to obtain surface antigens can be achieved by glycine acid extraction. ELISAs with these complex antigens, a second test is Urea breath test. A urea breath test checks to see if you have *H. pylori* bacteria in your stomach. This test can show if you have an *H. pylori* infection. It can also be used to see if treatment has worked to get rid of *H. pylori*.This was the first demonstration of the bacterial origin of a gastric urease. A solution of labeled urea ingested by the patient is rapidly hydrolyzed by *H. pylori* urease if this organism is present in the stomach; the labeled CO_2 is absorbed by the blood and exhaled in expired air. If the patient is not infected, no labeled CO_2 is produced and most of the isotope is eliminated in urine without modification. Athird test is Stool antigen test. A stool test detect traces of H pylori in the feces A stool antigen test checks to see if substances that trigger the immune system to fight an *H. pylori* infection (*H. pylori* antigens) are present in your feces (stool). Stool antigen testing done to help support a diagnosis of *H. pylori* infection or to find out whether treatment for an *H. pylori* infection has been successful. An enzyme-linked immunosorbent assay (ELISA) performed on stools, using polyclonal anti-*H. pylori* antibodies coated on microwells to capture *H. pylori* antigen and peroxidase-conjugated polyclonal antibodies to detect the immune complex. This test was named *H. pylori* stool antigen test (HpSA).

RESULTS

A total of 372 patients male and females with a mean age of 40 years were studied. *H. pylori* infection almost constantly induces a specific systemic immune response which may reflect the antibodies produced at the gastric mucosal level. A partial purification to obtain surface antigens can be achieved by glycine acid extraction. ELISAs with these complex antigens had a sensitivity of 85% with a specificity of 80% .Table 1

False-negative results may occur following a new infection before the antibody level is sufficiently elevated. The specificity of serology is satisfactory (90%) in developed countries, the risk of cross-reactions with other bacteria is limited. In developing countries, the specificity of the test may be altered by the presence of concomitant infections, especially with campylobacters or other related bacteria; furthermore, the host immune response may be lower due to malnutrition.

Table 1. Detection of Helicobacter pylori IgG antibodies in blood sample

Test	Lab	No of patients	Reference tests	Sensitivity (%)	Specificity (%)
Serology	Privet lab1	23	UBT, HpStAR	86	77
			UBT, HpStAR		
	Privet lab2	34	UBT, HpStAR	85	88
			UBT, HpStAR		
	Privet lab3	53	UBT, HpStAR	86	75
			UBT, HpStAR		
	Hospital 1	80		81	84
	Hospital 2	112		88	76
	Hospital 3	230		87	80

detection of *H. pylori* antigens in stools . an enzyme-linked immunosorbent assay (ELISA) performed on stools, using polyclonal anti-*H. pylori* antibodies coated on microwells to capture *H. pylori* antigen and peroxidase-conjugated polyclonal antibodies to detect the immune complex This test was named *H. pylori* stool antigen test (HpSA) . Its evaluation on stools obtained from 372 adult dyspeptic patients indicated 88.8% sensitivity and 94.5% specificity. Table 2.

Table 2. Evaluation of stool antigen tests using monoclonal antibodies performed pretreatment patients for detection of *H pylori*

Test	Lab	No of patients	Reference tests	Sensitivity (%)	Specificity (%)
HpStAR	Privet lab1	23	UBT,Sero	97	96
	Privet lab2	34	UBT,Sero	88	95
	Privet lab3	53	UBT,Sero	78	79
	Hospital 1	80	UBT,Sero	87	91
	Hospital 2	112	UBT,Sero	96	98
	Hospital 3	230	UBT,Sero	88	97

urease breath test (UBT):The breath test utilises the ability of *H. pylori* to produce large quantities of urease as a diagnostic characteristic, Breath testing has been found to have a high sensitivity and specificity (94-98%) .Table 3.

Table 3. Detection of Helicobacter pylori by produce large quantities of urease

Test	Lab	No of patients	Reference tests	Sensitivity (%)	Specificity (%)
UBT	Privet lab1	23	HpStAR,Sero	96	96
	Privet lab2	34	HpStAR,Sero	95	97
	Privet lab3	53	HpStAR,Sero	90	99
	Hospital 1	80	HpStAR,Sero	91	98
	Hospital 2	112	HpStAR,Sero	95	98
	Hospital 3	230	HpStAR,Sero	93	97

The characteristics dependent on the test are sensitivity and specificity and, when combined, determine the accuracy. In contrast, the positive and negative predictive values, which are important for clinical practice, are very dependent on the prevalence of the infection in the community or on the group of patients considered.Table 4.

Table 4. Comparison of performances of three noninvasive diagnostic tests for Helicobacter pylori detection 372

Test	Sensitivity	Specificity	PPV[b](%)	NPV[b](%)
UBT	94	98	94	98
Serology	85	80	85	80
Stool antigen test	88	94	88	94

PPV[b] : positive predictive value
NPVb: negative predictive value
Sensitivity and specificity value were obtained after defining the optimal cutoff

CONCLUSION

H. pylori detection is not an easy task due to the difficulty in accessing its ecological niche and the fragile nature of the bacterium. Analysis of a blood sample may reveal evidence of an active or previous *H. pylori* infection in your body.

However, breath and stool tests are better at detecting active H. pylori infections than is a blood test. This rapid noninvasive method allows a direct detection of the bacterium and is also a means to test its susceptibility to macrolides, the most important information currently needed for treatment. The lack of standardization should end with the availability of a kit.

However, many other aspects of *H. pylori* detection not covered in this research concern the host. It is clear that the evolution of *H. pylori* infection depends highly not only on the bacterial strain but also on the host's characteristics. Because of all of these possibilities, it is most likely that, in the near future, *H. pylori* detection will be a pioneer in the field of predictive medicine.

References

Allaker RP, Young KA, Hardie JM, Domizio P, Meadows N (2002). Prevalence of Helicobacter pylori at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. J. Med. Microbiol. 51:312-317.

Frenck RW, Clemens J (2003). Helicobacter in the developing world. Microbes Infect. 5:705-713.

Zhang L, Day A, McKenzie G, Mitchell H (2006). Nongastric Helicobacter species detected in the intestinal tract of children. J. Clin. Microbiol. 44:2276-2279.

Kosunen T U, Seppala K, Sarna S, Sipponen P(1992). Diagnostic value of decreasing IgG, IgA, and IgM antibody titres after eradication of Helicobacter pylori. Lancet. 339:893-895.

Feldman RA, Deeks JJ, Evans SJ W(1995). Multi-laboratory comparison of eight commercially available Helicobacter pylori serology kits. Eur. J. Clin. Microbiol. Infect. Dis. 14:428-433.

A Guide to Utilization of the Microbiology Laboratory for Diagnosis of Infectious Diseases: 2013 Recommendations by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM)a Clinical Infectious Diseases 2013 57(4): e22-e121

Faulde M, Schroder JP, Sobe D(1992). Serodiagnosis of Helicobacter pylori infections by detection of immunoglobulin G antibodies using an immunoblot technique and enzyme immunoassay. Eur. J. Clin. Microbiol. Infect. Dis. 11:589-594.

Bina JE, Alm RA, UriaNickelsen M, Thomas SR, Trust TJ, Hancock REW(2000). Helicobacter pylori uptake and efflux: basis for intrinsic susceptibility to antibiotics in vitro. Antimicrob. Agents Chemother. 44:248-254.

Bilardi C, Biagini R, Dulbecco P, Iiritano E, Gambaro C, Mele MR, Borro P, Tessieri L, Zentilin P, Mansi C, Vigneri S, Savarino V(2002). Stool antigen assay (HpSA) is less reliable than urea breath test for post-treatment diagnosis of Helicobacter pylori infection. Aliment. Pharmacol. Ther. 16:1733-1738.

Demirturk L, Yazgan Y, Tarcin O, Ozel M, Diler M, Oncul O, Yildirim S(2003). Does N-acetyl cystein affect the sensitivity and specificity of Helicobacter pylori stool antigen test? Helicobacter. 8:120-123.

Calvet X, Quesada M, Sanfeliu I, Montserrat A, Brullet E, Real J, Segura F, Campo R(2003). Evaluation of a rapid test (ImmunoCard STAT! HpSA) for Helicobacter pylori detection in stools. Gastroenterol. Hepatol. 26:531-534.

Calvet X, Quesada M, Sanfeliu I, Salceda F, Rosello M, Montserrat A, Brullet E, Segura F(2004). Diagnosis of Helicobacter pylori infection in dyspeptic patients by stool antigen detection-usefulness of a new monoclonal enzyme immunoassay test. Dig. Liv. Dis. 36: 450-454

Chisholm SA, Watson CL, Teare ELS, Saverymuttu S, Owen RJ (2004). Non-invasive diagnosis of Helicobacter pylori infection in adult dyspeptic patients by stool antigen detection: does the rapid immunochromatography test provide a reliable alternative to conventional ELISA kits? J. Med. Microbiol. 53:623-627.