Blood tests in the management of *Helicobacter pylori* infection

D Vaira, J Holton, M Menegatti, F Landi, C Ricci, A Ali’, L Gatta, S Farinelli, C Acciardi, B Massardi, M Miglioli and the Italian *Helicobacter pylori* Study Group

**Summary**
There are three main types of blood test available for the management of *Helicobacter pylori* infection: those that detect an antibody response; tests of the pathophysiological state of the stomach; and those that indicate an active infection. Enzyme linked immunosorbent assay (ELISA) based kits are the most numerous of the commercially available tests. Originally the kits used crude antigen preparations but many of the newer kits use a more purified antigen preparation giving increased specificity but a lower sensitivity. The sensitivity, specificity, and predictive values of the tests can also be affected by the population under test and coexistent disease in the patients. Near patient test kits are based on either latex agglutination or immunochromatography. Generally, they have low sensitivities compared with laboratory tests. Commerical western blotting kits have also been developed and are used to detect the presence of specific virulence markers. The exact role of serology in the management of *Helicobacter* infection has still to be defined, although there is evidence that, used as a screening procedure, it can reduce endoscopy cost and workload. Gastrin and pepsinogen blood concentrations may provide valuable information on the pathophysiological state of the stomach—for example, the presence of inflammation or gastric atrophy. A combination of serology and serum concentrations of gastrin and pepsinogen may be used effectively to detect serious gastroduodenal disease in patients.

**Introduction**

*Helicobacter pylori* has been linked with an increasing number of conditions since its first suggested association with gastritis, now well established. Knowledge about the pathogenesis of peptic ulcer disease (PUD) has been revolutionised by the isolation of *H pylori*, and, as a natural consequence of this, the management of PUD has completely changed from one of primarily acid suppression to one of primarily bacterial eradication. The causal association between colonisation by *H pylori*, PUD, and gastric cancer places *H pylori* as an important human pathogen. The link between colonisation by *H pylori* and the risk of developing various forms of gastric neoplasm raises the exciting prospect of reducing the risk of neoplasm development by some form of eradication therapy at a population or even individual level. A natural corollary of this is the requirement of an effective screening procedure to determine colonisation status. In addition, the possible association between the widespread pathophysiological effects of chronic inflammation in the stomach and the risk of ischaemic heart disease, growth retardation, and gall stones also suggests the need for cost-effective management protocols.

Generally, there are a number of factors that must be included in an effective protocol for all aspects of disease management. For example, there should be an accurate diagnostic procedure at an individual level and a cost-effective population screening procedure. Also, the ability to determine prognostic indicators may influence management of the disease and there should be an effective follow up protocol to establish efficacy of treatment and recurrence of disease. More specifically, with respect to determining colonisation by *H pylori* and associated disease, blood tests are one of the two non-invasive techniques available; the other is the urea breath test (UBT).

**Antibody tests for *H pylori***

**EVALUATION OF ANTIGEN**

Several different antigen preparations have been tested. Initially, crude sonicates were used, and, although the sensitivity of the test was high, the specificity was relatively low compared with other diagnostic tests such as culture or histology, because of false positives caused by non-specific cross reactions with other organisms—for example, *Campylobacter* sp.

Comparison of a whole cell preparation and an acid-glycine extract showed enrichment of some immunodiagnostic antigens in the acid-glycine extract (the 54 and 69 kDa proteins) but complete loss of others (the 29 and 120 kDa proteins). Further, although the intention was to reduce non-specific cross reactions, they were still detectable when assessed by western blotting. A more extensive study compared crude sonicates with ultracentrifuged whole cell sonicates and acid-glycine extracts and antigen fractions separated by sodium dodecyl sulphate-polyacrylamide gel electrophoresis. The greatest discrimination between *H pylori* positive and negative sera was found with high molecular mass fraction antigens, but whole cell sonicates were better than acid-glycine extracted antigens. A further comparison of four different antigen preparations (crude sonicate, acid-glycine extract, acid-glycine extract of a flagellate organism, and urease enriched fraction) showed the crude sonicate to have the highest sensitivity but the lowest specificity. A study using more purified antigen prepared by fast protein liquid chromatography (FPLC) or monoclonal antibody capture generally showed lower sensitivities of the purified antigens compared with an acid-glycine extract, with specificities of 100%
Table 1  Sensitivity and specificity of the different antigens

<table>
<thead>
<tr>
<th>Antigen preparation</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude sonicate</td>
<td>94-100</td>
<td>60-100</td>
</tr>
<tr>
<td>Ultra centrifuged sonicate</td>
<td>84-97</td>
<td>95-100</td>
</tr>
<tr>
<td>Surface antigen</td>
<td>82</td>
<td>92</td>
</tr>
<tr>
<td>Acid-glycine extract</td>
<td>82-95</td>
<td>83-98</td>
</tr>
<tr>
<td>Acid-glycine extract*</td>
<td>89</td>
<td>96</td>
</tr>
<tr>
<td>Urea preparation</td>
<td>81-97</td>
<td>89-90</td>
</tr>
<tr>
<td>120 kDa protein (Caga)</td>
<td>84-96</td>
<td>92-98</td>
</tr>
<tr>
<td>Recombinant caga</td>
<td>96</td>
<td>96</td>
</tr>
<tr>
<td>Caga + ultra centrifuged sonicate</td>
<td>97</td>
<td>100</td>
</tr>
<tr>
<td>Caga + acid-glycine extract</td>
<td>97</td>
<td>100</td>
</tr>
<tr>
<td>FPLC purified urease</td>
<td>91</td>
<td>91</td>
</tr>
<tr>
<td>FPLC purified flagella</td>
<td>78</td>
<td>100</td>
</tr>
<tr>
<td>MAB purified urease</td>
<td>83</td>
<td>93</td>
</tr>
</tbody>
</table>

Prepared from references 1–11.

*Acid-glycine extract from an aflagellate organism.

FPLC, fast protein liquid chromatography; MAB, monoclonal antibody.

EVALUATION OF COMMERCIALLY AVAILABLE SEROLOGICAL ASSAYS

ELISA kits

Many publications have compared either single or many kits one against another in a defined population, usually patients with dyspepsia or PUD or symptomatic individuals. Table 2 lists the main commercial serological assays available for the detection of *H pylori*. A comparison of the use of three kits for 76 patients using known culture positive cases showed comparable sensitivity and specificity of between 88 and 96% and 86 and 96% respectively. The inter- and intra-laboratory assay variation was low. The three kits used antigens of different purity: Pyloristat (enriched fractions), HelicoG (acid-glycine extract), Premier HP (high molecular mass cell associated proteins). In a further test on 95 dyspeptic patients, the Cobas Core anti- *H pylori* immunoglobulin EIA-G, which uses an FPLC purified antigen, had a sensitivity and specificity of 94 and 98% respectively and was superior to the rapid urease test (RUT) (89 and 96%) and culture (70 and 98%) when compared with histology. A laboratory comparison of the three kits was carried out, which included modified Pyloriset EIA-G update kit and Malakit EIA-G, on serum samples from 154 dyspeptic patients. Serological results were compared with those using culture/histology/RUT as the “gold standard”. The updated Pyloriset showed an improved sensitivity but reduced specificity compared with previous results for this kit from other studies on equivalent groups of patients. A single laboratory comparison of eight kits was undertaken on 84 dyspeptic patients and compared with histology and UBT. The results showed that all the kits had comparable sensitivity (90–100%) but more variable and lower specificity (76–96%). Indeterminate (grey zone) results occurred with some kits in up to 12% of the readings, although Premier HP, Pyloriset EIA-G, and HelicoG were calibrated so as not to give grey zone results (the latest version of the last of these kits, HelicoG2, however, does not have a grey zone range). The kit giving the highest percentage of grey zone results was GAP IgG. In this study Pyloriset ELISA II and Premier HP were particularly effective. A multilaboratory comparison of eight kits also showed that all the kits tested were broadly comparable. Some laboratories experienced difficulties with some kits and some kits showed high inter-laboratory variation. Overall the Pyloriset EIA-G and Roche MTP kits seemed to be the best. Again most of the kits produced some indeterminate results but this varied between kits (Roche, 0.9%; Hel-p Test, 13%). Table 3 gives the published sensitivity, specificity, positive, and negative values of these kits.
Data taken from references 12–23, 36, 37.

PPV, positive predictive value; NPV, negative predictive value.

Table 3 Comparison of commercially available ELISA kits for detection of Helicobacter pylori infection

<table>
<thead>
<tr>
<th>Kit</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Helori-test</td>
<td>95–99</td>
<td>88</td>
<td>94</td>
<td>88</td>
</tr>
<tr>
<td>Pylori 1</td>
<td>91–99</td>
<td>70–94</td>
<td>80</td>
<td>84</td>
</tr>
<tr>
<td>Pylori ELISA II</td>
<td>100</td>
<td>96</td>
<td>97</td>
<td>100</td>
</tr>
<tr>
<td>Helico C2</td>
<td>85</td>
<td>76</td>
<td>74</td>
<td>87</td>
</tr>
<tr>
<td>Premier HP</td>
<td>85–100</td>
<td>80–100</td>
<td>76–100</td>
<td>88–100</td>
</tr>
<tr>
<td>Cobas Core</td>
<td>87–98</td>
<td>83–98</td>
<td>87</td>
<td>86</td>
</tr>
<tr>
<td>Pylori test update</td>
<td>100</td>
<td>79</td>
<td>95</td>
<td>100</td>
</tr>
<tr>
<td>Hel-p Test</td>
<td>89–100</td>
<td>62–93</td>
<td>65–90</td>
<td>91–100</td>
</tr>
<tr>
<td>Malakit</td>
<td>79–87</td>
<td>86–98</td>
<td>96</td>
<td>60</td>
</tr>
<tr>
<td>GAP IgG</td>
<td>76–100</td>
<td>26–99</td>
<td>76–100</td>
<td>71–100</td>
</tr>
<tr>
<td>HP Stat Radim</td>
<td>84</td>
<td>90</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Roche MTP</td>
<td>94–98</td>
<td>83–86</td>
<td>86</td>
<td>90</td>
</tr>
<tr>
<td>HpG screen</td>
<td>83–93</td>
<td>68–91</td>
<td>66–84</td>
<td>84–100</td>
</tr>
<tr>
<td>Microstar</td>
<td>97</td>
<td>76</td>
<td>80</td>
<td>98</td>
</tr>
<tr>
<td>SIA Sigma</td>
<td>85–90</td>
<td>80–98</td>
<td>76–96</td>
<td>88–100</td>
</tr>
<tr>
<td>HM Sigma EIA</td>
<td>85–98</td>
<td>80–96</td>
<td>76</td>
<td>86</td>
</tr>
<tr>
<td>Autokine</td>
<td>89</td>
<td>52</td>
<td>58</td>
<td>87</td>
</tr>
<tr>
<td>Pyloragen</td>
<td>79</td>
<td>75</td>
<td>71</td>
<td>83</td>
</tr>
<tr>
<td>Enzygnost</td>
<td>80</td>
<td>74</td>
<td>70</td>
<td>83</td>
</tr>
<tr>
<td>Quidel EIA</td>
<td>89</td>
<td>66</td>
<td>68</td>
<td>89</td>
</tr>
<tr>
<td>Enzywell</td>
<td>90</td>
<td>71</td>
<td>71</td>
<td>91</td>
</tr>
<tr>
<td>Color Vue</td>
<td>88</td>
<td>86</td>
<td>63</td>
<td>87</td>
</tr>
</tbody>
</table>

Data taken from references 24–37.

PPV, positive predictive value; NPV, negative predictive value.

Near patient testing

There is a perceived unmet clinical need for near patient testing of patients for H pylori infection, and several companies have developed rapid tests. Most of the tests consist of one step using whole blood, but others require serum separation, which diminishes their usefulness as near patient kits. With one test kit, variation in sensitivity and specificity were noted depending on whether capillary or venous blood was used. Comparatively few assessments have been published.34–36 The Helisal rapid blood test (Helisal RBT, now superseded by Helisal One Step) had a sensitivity and specificity of 88 and 91% and a positive predictive value and negative predictive value of 92 and 86% when compared with histology, culture, RUT, and UBT in 154 dyspeptic patients.37 These results compared with 93 and 87% sensitivity and specificity respectively for an ELISA test (HelicoG) in 109 of the patients. Another assessment on 203 patients44 compared with RUT/histology gave a sensitivity and specificity of 82 and 91%, and in this study there was concurrence of results whether venous or capillary blood was used. When Helisal was compared with a laboratory ELISA, its sensitivity and specificity were 83 and 78% respectively.39 Other studies have found much lower specificity (55%) when compared with RUT/histology/culture36–37 (table 4). Inter- and intra-laboratory comparisons have not yet been performed and published for these kits, but a recent publication does not support the use of these kits as presently formulated for near patient testing.40

EFFECT OF POPULATION ON SEROLOGICAL RESULTS

Most assessments have been made in adults who were dyspeptic or asymptomatic. It is recognised from sero-epidemiological studies that different ethnic populations have widely differing prevalences of infection and that the assay cut-off value may have to vary to reflect this. Similarly the positive and negative predictive values of the various serological tests may vary according to age, drug administration, or coexistent disease in the population or individual under investigation. Studies in a group of children and in the elderly have shown decreased specificity of the serological tests when compared with culture and histology.35 In the elderly this can often be due to atrophic gastritis and reduction in colonisation by H pylori. Apart from this age related effect on the accuracy of serological tests, the use of non-steroid anti-inflammatory drugs41 can also affect the test accuracy, as can coexistent disease such as HIV infection,42 cystic fibrosis,43 and cirrhosis.44

SEROLOGY IN DIAGNOSIS AND SCREENING

Serology can only give evidence of contact with H pylori and does not necessarily indicate a current infection. This is more accurately diagnosed using a UBT. Serology, however, like the UBT, is a global test and is not affected by sampling errors, as are the biopsy based tests. In a study comparing all the diagnostic methods available, serology had a sensitivity and specificity of 98 and 95% respectively compared with 98 and 100% (culture), 96 and 100% (PCR), 98 and 98% (histology), 90 and 100% (RUT), and 100 and 100% (13C-UBT),45 although lower values for the sensitivity and specificity have been obtained in other studies. For example, comparing serology with RUT, sensitivities of 74 and 90% and specificities of 89 and 96% respectively were obtained in one study46 and sensitivities and specificities of 96 and 88% for serology were obtained in another study when compared with UBT (96 and 100%), RUT (92 and 92%), and histology (96 and 91%).46 The relative sensitivity and specificity of serology obtained in another study, when compared with other diagnostic methods, depended on the population studied, the number of individuals investigated, and the type of serological assay.
used. Serological assays for *H pylori* infection may have value in both diagnosis and screening and to monitor the effect of eradication treatment. The decision to use one test rather than another depends on the clinical circumstances, the reported test parameters (sensitivity, specificity, positive predictive value, and negative predictive value), cost, and convenience.

There are several possible management algorithms: to treat empirically; to use a screening test and either treat or proceed to endoscopy on the basis of the results; to examine every symptomatic patient by endoscopy. As the latter is an expensive option, various screening strategies have evolved to decrease the number of endoscopies performed. Other factors that need to be taken into account in a management algorithm are age (if over 45 years the patient should proceed to endoscopy without necessarily having a serological test), use of non-steroid anti-inflammatory drugs, and worrying symptoms. Several studies have shown that screening dyspeptic patients using serological tests can be cost-effective in reducing the endoscopy workload by up to 30% without missing significant pathology.52 53 Patients who are positive on serological testing can then go on to endoscopy to verify the presence of PUD and hence be started on treatment, or may proceed directly to treatment. However, other studies have shown that if a screening strategy is adopted, significant pathology in some populations can be missed,49 and we do not recommend it as a routine practice.

An alternative screening strategy is the use of a symptom questionnaire, and these have been reported to save a similar percentage of endoscopies as serological screening. In one direct comparison of symptom questionnaires with serological screening in 315 patients,54 the latter detected more PUD than the questionnaires, but one of the questionnaires was more cost-effective in avoiding unneeded endoscopies. In addition, a cost analysis51 of adopting a screening protocol using serological tests compared with empirical treatment with H2 receptor antagonists, this was cheaper than suppressive treatment with non-steroid anti-inflammatory drugs, and worrisome symptoms. Several studies have shown that screening dyspeptic patients using serological tests can be cost-effective in reducing the endoscopy workload by up to 30% without missing significant pathology.52 53 Patients who are positive on serological testing can then go on to endoscopy to verify the presence of PUD and hence be started on treatment, or may proceed directly to treatment. However, other studies have shown that if a screening strategy is adopted, significant pathology in some populations can be missed,49 and we do not recommend it as a routine practice.

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**Table 5 Reactivity against Helicobacter pylori lysate, CagA and VacA by RIBA-SIA**

<table>
<thead>
<tr>
<th>Reactivity (%)</th>
<th>Lysate positive</th>
<th>CagA positive</th>
<th>VacA positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood donor (n=999)</td>
<td>42</td>
<td>32</td>
<td>15</td>
</tr>
<tr>
<td>Non-ulcer dyspepsia (n=571)</td>
<td>42</td>
<td>36</td>
<td>17</td>
</tr>
<tr>
<td>Duodenal ulcer (n=275)</td>
<td>82</td>
<td>70</td>
<td>38</td>
</tr>
<tr>
<td>Gastric ulcer (n=71)</td>
<td>77</td>
<td>68</td>
<td>38</td>
</tr>
<tr>
<td>Gastric cancer (n=570)</td>
<td>78</td>
<td>61</td>
<td>33</td>
</tr>
<tr>
<td>Extragastric cancer (n=438)</td>
<td>63</td>
<td>38</td>
<td>21</td>
</tr>
</tbody>
</table>

**Table 6 Prevalence of CagA by ELISA in Helicobacter pylori positive patients according to endoscopic findings**

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Gastritis duodenitis</th>
<th>Gastric ulcer</th>
<th>Duodenal ulcer</th>
<th>Gastric cancer</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>CagA+</td>
<td>117 (55.7)</td>
<td>322 (59.6)</td>
<td>41 (80.4)</td>
<td>171 (85.1)</td>
<td>6 (85.7)</td>
<td>671 (64.7)</td>
</tr>
<tr>
<td>CagA−</td>
<td>93 (44.3)</td>
<td>219 (40.5)</td>
<td>10 (19.6)</td>
<td>30 (14.9)</td>
<td>1 (14.3)</td>
<td>366 (35.3)</td>
</tr>
<tr>
<td>Overall</td>
<td>210 (20.2)</td>
<td>541 (39.3)</td>
<td>51 (4.9)</td>
<td>201 (19.4)</td>
<td>7 (0.7)</td>
<td>1037 (100)</td>
</tr>
</tbody>
</table>

Values in parentheses are percentages.

Infection. It is therefore important to diagnose the type of infecting organism. This is serologically possible since CagA protein is highly immunogenic: in fact, more than 95% of subjects infected by *cagA* positive *H pylori* strains develop a serologically detectable response to the gene product (anti-CagA), compared with 0% of uninfected patients. However, both the structure of the VacA proteins and the serological response to it is only just being clarified and data are still scanty. These and other as yet undiscovered proteins could therefore lead to the identification of “bad”, “very bad”, “neutral”, or even “good” *H pylori* strains, as recently speculated by Blaser. The serological techniques currently available to determine the cytotoxic type of infecting strains are western blotting and ELISA. A novel recombinant immunoblot assay (RIBA-SIA; Chiron Corp., Emeryville, California, USA) has recently been proposed which contains individual bands for whole *H pylori* lysate, recombinant CagA, and VacA. In a recent evaluation of anti-CagA and anti-VacA reactivity by RIBA-SIA in large populations of both asymptomatic subjects and patients with different pathologies, anti-CagA mainly but also anti-VacA reactivities were found to be more prevalent in patients with severe gastroduodenal pathology (table 5). Similarly the seroprevalence of anti-CagA reactivity assessed by ELISA (Helori-CTX; Eurospital, Trieste, Italy) was confirmed as being higher in *H pylori* positive subjects with gastric or duodenal ulcer than asymptomatic subjects or patients with NUD. The results of a large multicentre study carried out in Italy involving over 3000 patients examined by endoscopy in more than 90 endoscopy units have recently been published. CagA prevalence was assessed by ELISA in this large population, and the preliminary results in over 1300 patients confirm the association between CagA and major gastroduodenal pathology (table 6).

Many similar, although smaller, studies have previously shown the association between CagA as a marker for PUD and gastric cancer, although other studies have not found this association.

**Markers of Gastric Inflammation and *H pylori***

To distinguish PUD (in which the eradication of *H pylori* is recommended) from NUD (in which the role of *H pylori* is controversial), it is necessary to perform an endoscopy. Neither serological tests nor UBT give any quantitative information that would help to differentiate between these two conditions. However, measurement of blood markers of gastric inflammation may give some clinical information that is useful in the management of *H pylori* related disease.

**Gastrin**

*H pylori* infection is associated with a set of well recognised disturbances to normal gastric physiology. Plasma gastrin levels are elevated in *H pylori* infection (150 ng/ml) compared with control levels (50 ng/ml) as a consequence of inhibition of somatostatin production. After eradication of *H pylori* the plasma gastrin levels return to normal.

Higher levels of plasma gastrin are found in corpus gastritis than in antral gastritis, but there is no significant difference between the levels in gastritis compared with ulceration.

**Pepsinogen**

Variations in concentrations of pepsinogen (PG) I and II and the PGI:II ratio can occur with age, weight, smoking, and chronic renal failure. Increases in both PGI (73 ng/ml compared with 50 ng/ml) and PGII (24 ng/ml compared with 10 ng/ml) with a reduction in the PGI:II ratio (3.6 compared to 6.2) are found in *H pylori* associated gastritis compared with *H pylori* negative individuals. Some studies have shown that PGII levels are even further elevated in *H pylori* associated PUD compared with those without PUD, and the elevation correlates with the degree of inflammation. A high PGII ratio is found in ulceration associated with the Zollinger-Ellison syndrome. In *H pylori* associated gastritis the increase in PGI is least in corpus only gastritis and highest in predominantly antral gastritis. Reduction in both PGI and PGII and normalisation of the ratio can be used to confirm successful eradication of *H pylori*, although a decrease in PGI is the most accurate biomarker of eradication compared with PGII, serology, and serum gastrin.

Variation in the levels and ratio of PG can be used to predict the presence of more serious gastric pathology. Used as a screening test in an asymptomatic population, a low PGI compared with PUD, and the elevation correlates with the degree of inflammation. A high PGII ratio is found in ulceration associated with the Zollinger-Ellison syndrome. In *H pylori* associated gastritis the increase in PGI is least in corpus only gastritis and highest in predominantly antral gastritis. Reduction in both PGI and PGII and normalisation of the ratio can be used to confirm successful eradication of *H pylori*, although a decrease in PGI is the most accurate biomarker of eradication compared with PGII, serology, and serum gastrin.

A high serum IgA anti-*H pylori* antibody level associated with a...
decreased PGI (<50 ng/ml) correlates with an increased risk of gastric cancer, with an odds ratio of 5.95 in one study population.76

Conversely there is an inverse correlation between serum IgG anti-H pylori antibody levels and the extent of gastric metaplasia, but only in those individuals that have normal PGI levels.77 Screening strategies have been developed to detect gastric adenocarcinoma using a combination of H pylori positivity and PG and gastrin levels.77 78 A study of 686 patients, of which 150 had gastric adenocarcinoma, showed that age above 62 years, low PGI levels, low PGI x gastrin value, and low PGI:gastrin ratio were indicative of gastric adenocarci-
noma, with the value of serum PGI being the most important.

Conclusions

The exact role of serological testing in the management of H pylori infection is yet to be defined. However, used as a screening procedure, it can reduce endoscopy workload and cost, although the savings may take several years to accumulate.

Used in conjunction with blood levels of gastrin and PGs, these tests can suggest the presence of H pylori associated gastritis and be used to screen for serious gastroduodenal pathology, although further work is required to clarify their usefulness in this aspect.

The role of H pylori in NUD will affect how these blood tests are used in the management of Helicobacter infections. Currently, eradica-
tion of H pylori is only recommended in cases of PUD, and endoscopy is required to differentiate PUD from NUD. Therefore blood tests that could achieve this differentiation may reduce the endoscopy workload even further.

Italian Helicobacter pylori Study Group

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Blood tests in H pylori infection management


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