Local Coverage Determination (LCD): Helicobacter Pylori Infection Testing (L37624)

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Contractor Information

CONTRACTOR NAME	CONTRACT TYPE	CONTRACT NUMBER	JURISDICTION	STATE(S)
Noridian Healthcare Solutions, LLC	A and B MAC	01111 - MAC A	J - E	California - Entire State
Noridian Healthcare Solutions, LLC	A and B MAC	01112 - MAC B	J - E	California - Northern
Noridian Healthcare Solutions, LLC	A and B MAC	01182 - MAC B	J - E	California - Southern
Noridian Healthcare Solutions, LLC	A and B MAC	01211 - MAC A	J - E	American Samoa Guam Hawaii Northern Mariana Islands
Noridian Healthcare Solutions, LLC	A and B MAC	01212 - MAC B	J - E	American Samoa Guam Hawaii Northern Mariana Islands
Noridian Healthcare Solutions, LLC	A and B MAC	01311 - MAC A	J - E	Nevada
Noridian Healthcare Solutions, LLC	A and B MAC	01312 - MAC B	J - E	Nevada
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LCD Information

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Helicobacter Pylori Infection Testing

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CMS National Coverage Policy

Title XVIII of the Social Security Act, §1862(a)(1)(A). Allows coverage and payment for only those services that are

considered to be reasonable and necessary.

Title XVIII of the Social Security Act, §1833(e). Prohibits Medicare payment for any claim which lacks the necessary information to process the claim.

42 Code of Federal Regulations (CFR) 410.32(a). Diagnostic x-ray tests, diagnostic laboratory tests, and other diagnostic tests: Conditions.

CMS On-Line Manual, Publication 100-02, Medicare Benefit Policy Manual, Chapter 15, §§80.0, 80.1.1, 80.2. Clinical Laboratory services.

CMS Internet-Only Manuals, Publication 100-04, Medicare Claims Processing Manual, Chapter 16, §50.5 Jurisdiction of Laboratory Claims, 60.12 Independent Laboratory Specimen Drawing, 60.2. Travel Allowance.

CMS Internet Online Manual Pub. 100-04 (Medicare Claims Processing Manual), Chapter 23 (Section 10) "Reporting ICD Diagnosis and Procedure Codes"

Coverage Guidance

Coverage Indications, Limitations, and/or Medical Necessity

This policy provides limited coverage for Helicobacter pylori (H. pylori) infection testing by carbon isotope (^{13}C or ^{14}C) urea breath testing or stool antigen testing. This policy also denies coverage for H. pylori serology testing, TZAM H. pylori multiplex PCR testing, plasma pepsinogen II testing, tonsillar H. pylori colonization, IL1B-31>T polymorphism testing for H. pylori, tumor necrosis factor-alpha (TNF*α*), and AmHPR *Helicobacter* antibiotic resistance next generation sequencing panel testing.

Summary of Evidence

This policy is consistent with guidelines of the American Gastroenterological Association and the American College of Gastroenterology.[3,4] in younger patients without "alarm" symptoms (e.g., weight loss, progressive dysphagia, recurrent vomiting, evidence of GI bleeding, or family history of UGI cancer)[20]. Endoscopy with biopsy is recommended for patients >55 years of age and younger patients with alarm symptoms.[2,5]

Multiple Food and Drug Administration (FDA) cleared urea place.[6,8] (Halyard Health, Alpharetta, GA).

A stool antigen test, cleared by the FDA, may be used for initial diagnosis, therapeutic monitoring and eradication confirmation in adults and children. The HpSA® test (Meridian Bioscience, Cincinnati, OH) is the only FDA cleared stool antigen test in the US. All others use analyte specific reagents (ASR) or are laboratory developed tests (LDTs). The stool antigen test is based on the passage of H. pylori bacteria and H. pylori antigens in the GI tract, and their detection by immunoassay which translates into the detection of an active infection. The test does not require fasting or an instrument for analysis, does not have adverse effects, nor does it depend on a by-product of H. pylori and, has the additional advantage that testing can be performed while patients are on proton pump inhibitor (PPI),

bismuth or H2 blockers.

Confirmation of the presence of H. pylori bacterium can be determined invasively on endoscopic biopsy followed by rapid urease testing (CLOtest™ PyloriTek™, Hpfast™), by histology which on occasion may require special stains or immunohistochemistry, or culture.

More than 90% of gastroduodenal ulcers are associated with H. pylori infection. The ACG guidelines recommend that all person suspected of having peptic ulcer disease should be tested for H. pylori regardless of whether they are concurrently taking non-steroidal anti-inflammatory drugs (NSAIDS), as H. pylori and NSAIDs are independent risk factors for the development of peptic ulcer disease. Antibiotic therapy is indicated for all H. pylori infected ulcer patients together with acid-suppressing drugs to facilitate symptom relief and healing. The ACG also recommend post-treatment testing, by the stool antigen test or the urea breath test, in ALL patients treated for H. pylori infection [3].

With an H. pylori prevalence of up to 30-40% in the US, it is not surprising that 30-40% of patients undergoing bariatric surgery are infected with H. pylori.[9] Because H. pylori infection may increase the risk of post-operative marginal ulcers, noninvasive H. pylori infection testing is recommended as part of the routine pre-operative evaluation of patients before bariatric surgery.

The AGA and ACG no longer recommend H. pylori serology testing because it is not a test of active infection. Although a negative serology for H. pylori antibody can be used to rule out infection, a positive serology indicates H. pylori exposure at some time in the past, not whether the patient has current infection. Studies suggest that nearly 50% of person with positive H. pylori serology do not have active infection.[3] Furthermore, serology cannot be used to show that H. pylori infection has been successfully eradicated after treatment. Antibody levels commonly remain elevated for months to years after treatment.

A reliable diagnosis is mandatory for the identification of infection and to confirm eradication of infection. Although bacterial culture from the gastric biopsy is the "gold" standard technique for H. pylori identification, and is recommended for antibiotic susceptibility testing, it is not practical for all patients. Although infrequently indicated, quantitative polymerase chain reaction (PCR) on gastric biopsies can be used to detect low bacterial loads, the use of the testing is limited by its high cost.[10] Others have suggested the measurement of decreased plasma pepsinogen II may be a reliable biomarker to confirm successful eradiation of H. pylori infection.[11] However, studies are with limited numbers of patients, and inconclusive findings.

Others have suggested that H. pylori infection plays a role in the development of other conditions. Hwang et al[12], in a systematic review and meta-analysis, found no evidence that H. pylori infection plays a role in the pathogenesis or development of chronic tonsillitis. Gomes et al[13] concluded that recurrent aphthous stomatitis (RAS) ulcers are not associated with the presence of bacteria in the oral cavity and there is no evidence that H. pylori infection drives RAS development. Sun et al[14] hypothesized that host genetic factors that control the production of cytokines, including interleukin -1ß, which affect susceptibility to many H. pylori-related diseases. The authors concluded that the findings of their meta-analysis showed that IL1ß-31C>T polymorphism might increase H. pylori risk in Asian and Latin American populations, that TNF*a*-308G>A and -1031T>C polymorphisms may be protective factors against H. pylori infection[15], and that -863C>A may be a risk factor in Asian populations. However, they indicate further studies with different ethnicities and larger samples size are needed to validate their findings.

AmHPR H. pylori antibiotic resistance panel testing examines antibiotic resistance to 6 antibiotic types that are currently used in H. pylori treatment by means of NGS: 23S rRNA for clarithromycin; gyrA for fluoroquinolones; rdxA for metronidazole; pbp1 for amoxicillin; 16S rRNA for tetracycline, and rpoB for rifabutin. Binh et al[16] stated that metronidazole resistance is a key factor associated with H. pylori failure. The authors confirmed that the mutations in

rdxA were mainly associated with metronidazole resistance, and mutations in frxA were able to enhance H. pylori resistance only in the presence of rdxA mutations. These authors conclude that further work is needed to identify the role of mutations associated with treatment failure. In a large pilot study by[17] and colleagues on 849 Indonesian dyspeptic patients, authors showed a high prevalence of metronidazole and levofloxacin resistance with low prevalence of clarithromycin, amoxicillin and tetracycline resistance, largely related to local antibiotic consumption. They noted that resistance is primarily due to the H. pylori genotype, rather than the human genotype.

Multiple regimens are available for treating H. pylori infection. The first-line regimen for H. *pylori* eradication includes proton pump inhibitor (PPI), clarithromycin (CAM), and amoxicillin (AMX), or metronidazole. Proton pump inhibitors (PPIs) suppress acid production in combination with antibiotic treatment. However, the failure rate of triple anti-H. pylori therapies has increased up to 30%. The known factors for therapy failure include antibiotic resistance, poor compliance, high gastric acidity, and high bacterial load.

Studies suggest that cytochrome P450 CYP2C19 polymorphism may also play a role in therapy failure. CYP2C19 is implicated in the metabolism of PPIs. What is known is that differences in PPI metabolism lead to variability in gastric acid suppression, with associated variability in gastric pH, and that CYP2C19 polymorphism is highly varied among different ethnic populations. Observational studies suggest that extensive metabolizers (EM) of PPIs have lower eradication rates following standard treatment for H. pylori compared to poor metabolizers (PM). Studies suggest that CTP2C19 genotype is a cardinal factor for H. pylori eradication in patients taking omeprazole-based or lansoprazole-based triple therapies. In contrast, this polymorphism has no significant effect on the rabeprazole-based or esomeprazole-based triple therapies. However, overall there is conflicting data and meta-analyses that conflict with one another. At the current time, the existing scientific data is insufficient to demonstrate a causal effect.

Analysis of Evidence
(Rationale for Determination)

Level of Evidence

Quality of evidence: Mixed
Strength of evidence: Strong
Weight of evidence: Sufficient

Based upon the American College of Gastroenterology 2017 Guidelines, Noridian establishes the following Criteria for coverage for urea breath testing **or** stool antigen testing for active H pylori infection are:

- Evaluation of new onset, uninvestigated dyspepsia in persons younger than 60 years of age without alarm symptoms; or
- All patients with active peptic ulcer disease (PUD), a past history of PUD (unless previous cure of H. Pylori infection is documented); or
- Patients with low grade gastric mucosa-associated lymphoid tissue (MALT); or
- Patients with a history of endoscopic resection of early gastric cancer; or
- Patients taking long term low dose aspirin may be considered for testing to reduce the risk of ulcer bleeding; or
- Patients initiating chronic treatment with nonsteroidal anti-inflammatory drugs; or
- Patients with unexplained iron deficiency despite an appropriate workup; or
- Adults with idiopathic thrombocytopenic purpura; or
- Recurrent dyspeptic symptoms suggest reinfection with H. pylori; or
- Re-evaluation to assess success of eradication of H. pylori infection (no sooner than 4 weeks post-treatment

and after PPI threapy has been withheld for 1-2 weeks).

All other H. pylori testing for any other etiology is not reasonable and necessary, and not a Medicare benefit. Some non-covered etiologies including but not limited to the risk of developing dementia, dyspepsia associated with "alarm" markers, recurrent aphthous stomatitis (RAS), onset of new dyspepsia in person aged 55 years or older, and screening of asymptomatic person for H. pylori infection. Upper GI endoscopy is indicated for persons aged 55 years or older because of increased concern for gastric neoplasia.

Note: Either urea breath testing or stool antigen testing for H. pylori is medically indicated; not both tests. Serology is no longer an acceptable non-invasive test H. pylori infection.

Coding Information

Bill Type Codes:

Contractors may specify Bill Types to help providers identify those Bill Types typically used to report this service. Absence of a Bill Type does not guarantee that the policy does not apply to that Bill Type.Complete absence of all Bill Types indicates that coverage is not influenced by Bill Type and the policy should be assumed to apply equally to all claims.

N/A

Revenue Codes:

Contractors may specify Revenue Codes to help providers identify those Revenue Codes typically used to report this service. In most instances Revenue Codes are purely advisory. Unless specified in the policy, services reported under other Revenue Codes are equally subject to this coverage determination. Complete absence of all Revenue Codes indicates that coverage is not influenced by Revenue Code and the policy should be assumed to apply equally to all Revenue Codes.

N/A

CPT/HCPCS Codes

Group 1 Paragraph:

Note: CPT 87338 is to be used for FDA cleared HpSA stool antigen testing only. All other stool antigen tests must use CPT 84999.

Non-covered CPT codes:

0008U H. pylori detection and antibiotic resistance, …

83009 H. pylori, blood test analysis for urease activity, non-radioactive isotope

83519 IA for analyte other than infectious agent antibody {plasma pepsinogen II testing}

86318 IA for infectious agent antibody, qual or semiquant, egg, reagent strip {office-based serology}

86677 Antibody, H. pylori {lab-based}

Group 1 Codes:

CODE	DESCRIPTION
78267	UREA BREATH TEST, C-14 (ISOTOPIC); ACQUISITION FOR ANALYSIS
78268	UREA BREATH TEST, C-14 (ISOTOPIC); ANALYSIS
83013	HELICOBACTER PYLORI; BREATH TEST ANALYSIS FOR UREASE ACTIVITY, NON-RADIOACTIVE ISOTOPE (EG, C-13)
83014	HELICOBACTER PYLORI; DRUG ADMINISTRATION
87338	INFECTIOUS AGENT ANTIGEN DETECTION BY IMMUNOASSAY TECHNIQUE, (EG, ENZYME IMMUNOASSAY [EIA], ENZYME-LINKED IMMUNOSORBENT ASSAY [ELISA], IMMUNOCHEMILUMINOMETRIC ASSAY [IMCA]) QUALITATIVE OR SEMIQUANTITATIVE, MULTIPLE-STEP METHOD; HELICOBACTER PYLORI, STOOL

ICD-10 Codes that Support Medical Necessity

Group 1 Paragraph:

All other ICD10 Codes are not covered.

Group 1 Codes:

ICD-10 CODE	DESCRIPTION
B96.81	Helicobacter pylori [H. pylori] as the cause of diseases classified elsewhere
C16.0	Malignant neoplasm of cardia
C16.1	Malignant neoplasm of fundus of stomach
C16.2	Malignant neoplasm of body of stomach
C16.3	Malignant neoplasm of pyloric antrum
C16.4	Malignant neoplasm of pylorus
C16.5	Malignant neoplasm of lesser curvature of stomach, unspecified
C16.6	Malignant neoplasm of greater curvature of stomach, unspecified
C16.8	Malignant neoplasm of overlapping sites of stomach
C16.9	Malignant neoplasm of stomach, unspecified
C83.30	Diffuse large B-cell lymphoma, unspecified site
C83.31	Diffuse large B-cell lymphoma, lymph nodes of head, face, and neck

ICD-10 CODE	DESCRIPTION
C83.32	Diffuse large B-cell lymphoma, intrathoracic lymph nodes
C83.33	Diffuse large B-cell lymphoma, intra-abdominal lymph nodes
C83.34	Diffuse large B-cell lymphoma, lymph nodes of axilla and upper limb
C83.35	Diffuse large B-cell lymphoma, lymph nodes of inguinal region and lower limb
C83.36	Diffuse large B-cell lymphoma, intrapelvic lymph nodes
C83.37	Diffuse large B-cell lymphoma, spleen
C83.38	Diffuse large B-cell lymphoma, lymph nodes of multiple sites
C83.39	Diffuse large B-cell lymphoma, extranodal and solid organ sites
C88.4	Extranodal marginal zone B-cell lymphoma of mucosa-associated lymphoid tissue [MALT-lymphoma]
E66.01	Morbid (severe) obesity due to excess calories
E66.09	Other obesity due to excess calories
E66.1	Drug-induced obesity
E66.2	Morbid (severe) obesity with alveolar hypoventilation
E66.3	Overweight
E66.8	Other obesity
E66.9	Obesity, unspecified
K25.0	Acute gastric ulcer with hemorrhage
K25.1	Acute gastric ulcer with perforation
K25.2	Acute gastric ulcer with both hemorrhage and perforation
K25.3	Acute gastric ulcer without hemorrhage or perforation
K25.4	Chronic or unspecified gastric ulcer with hemorrhage
K25.5	Chronic or unspecified gastric ulcer with perforation
K25.6	Chronic or unspecified gastric ulcer with both hemorrhage and perforation
K25.7	Chronic gastric ulcer without hemorrhage or perforation
K25.9	Gastric ulcer, unspecified as acute or chronic, without hemorrhage or perforation
K26.0	Acute duodenal ulcer with hemorrhage
K26.1	Acute duodenal ulcer with perforation
K26.2	Acute duodenal ulcer with both hemorrhage and perforation
K26.3	Acute duodenal ulcer without hemorrhage or perforation
K26.4	Chronic or unspecified duodenal ulcer with hemorrhage
K26.5	Chronic or unspecified duodenal ulcer with perforation

ICD-10 CODE	DESCRIPTION
K26.6	Chronic or unspecified duodenal ulcer with both hemorrhage and perforation
K26.7	Chronic duodenal ulcer without hemorrhage or perforation
K26.9	Duodenal ulcer, unspecified as acute or chronic, without hemorrhage or perforation
K27.0	Acute peptic ulcer, site unspecified, with hemorrhage
K27.1	Acute peptic ulcer, site unspecified, with perforation
K27.2	Acute peptic ulcer, site unspecified, with both hemorrhage and perforation
K27.3	Acute peptic ulcer, site unspecified, without hemorrhage or perforation
K27.4	Chronic or unspecified peptic ulcer, site unspecified, with hemorrhage
K27.5	Chronic or unspecified peptic ulcer, site unspecified, with perforation
K27.6	Chronic or unspecified peptic ulcer, site unspecified, with both hemorrhage and perforation
K27.7	Chronic peptic ulcer, site unspecified, without hemorrhage or perforation
K27.9	Peptic ulcer, site unspecified, unspecified as acute or chronic, without hemorrhage or perforation
K28.0	Acute gastrojejunal ulcer with hemorrhage
K28.1	Acute gastrojejunal ulcer with perforation
K28.2	Acute gastrojejunal ulcer with both hemorrhage and perforation
K28.3	Acute gastrojejunal ulcer without hemorrhage or perforation
K28.4	Chronic or unspecified gastrojejunal ulcer with hemorrhage
K28.5	Chronic or unspecified gastrojejunal ulcer with perforation
K28.6	Chronic or unspecified gastrojejunal ulcer with both hemorrhage and perforation
K28.7	Chronic gastrojejunal ulcer without hemorrhage or perforation
K28.9	Gastrojejunal ulcer, unspecified as acute or chronic, without hemorrhage or perforation
K29.00	Acute gastritis without bleeding
K29.01	Acute gastritis with bleeding
K29.20	Alcoholic gastritis without bleeding
K29.21	Alcoholic gastritis with bleeding
K29.30	Chronic superficial gastritis without bleeding
K29.31	Chronic superficial gastritis with bleeding
K29.40	Chronic atrophic gastritis without bleeding
K29.41	Chronic atrophic gastritis with bleeding
K29.50	Unspecified chronic gastritis without bleeding

ICD-10 CODE	DESCRIPTION
K29.51	Unspecified chronic gastritis with bleeding
K29.60	Other gastritis without bleeding
K29.61	Other gastritis with bleeding
K29.70	Gastritis, unspecified, without bleeding
K29.71	Gastritis, unspecified, with bleeding
K29.80	Duodenitis without bleeding
K29.81	Duodenitis with bleeding
K29.90	Gastroduodenitis, unspecified, without bleeding
K29.91	Gastroduodenitis, unspecified, with bleeding
K30	Functional dyspepsia
K31.89	Other diseases of stomach and duodenum
R10.13	Epigastric pain
Z87.11	Personal history of peptic ulcer disease

ICD-10 Codes that DO NOT Support Medical Necessity

N/A

Additional ICD-10 Information

N/A

General Information

Associated Information

N/A

Sources of Information

N/A

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05/27/2019	R1	04/04/2019: Corrected typographical error	• Typographical

REVISION HISTORY DATE	REVISION HISTORY NUMBER	REVISION HISTORY EXPLANATION	REASON(S) FOR CHANGE
		in title of LCD.	Error

Associated Documents

Attachments

N/A

Related Local Coverage Documents

Article(s)
A56381 - Response to Comments: Helicobacter Pylori Infection Testing
LCD(s)
DL37624 - Heliocobacter Pylori Infection Testing

Related National Coverage Documents

N/A

Public Version(s)

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