Clinical Policy Title: COLARIS® testing for Lynch syndrome

Clinical Policy Number: 02.01.10

Effective Date: October 1, 2014
Initial Review Date: March 19, 2014
Most Recent Review Date: April 19, 2017
Next Review Date: April 2018

Policy contains:
- COLARIS® testing.
- Colorectal cancer (CRC).
- Lynch syndrome (LS).

Related policies:

CP# 02.01.08 Familial polyposis gene testing
CP# 08.01.09 Colorectal cancer screening

ABOUT THIS POLICY: AmeriHealth Caritas Northeast has developed clinical policies to assist with making coverage determinations. AmeriHealth Caritas Northeast’s clinical policies are based on guidelines from established industry sources, such as the Centers for Medicare & Medicaid Services (CMS), state regulatory agencies, the American Medical Association (AMA), medical specialty professional societies, and peer-reviewed professional literature. These clinical policies along with other sources, such as plan benefits and state and federal laws and regulatory requirements, including any state- or plan-specific definition of “medically necessary,” and the specific facts of the particular situation are considered by AmeriHealth Caritas Northeast when making coverage determinations. In the event of conflict between this clinical policy and plan benefits and/or state or federal laws and/or regulatory requirements, the plan benefits and/or state and federal laws shall control. AmeriHealth Caritas Northeast’s clinical policies are for informational purposes only and not intended as medical advice or to direct treatment. Physicians and other health care providers are solely responsible for the treatment decisions for their patients. AmeriHealth Caritas Northeast’s clinical policies are reflective of evidence-based medicine at the time of review. As medical science evolves, AmeriHealth Caritas Northeast will update its clinical policies as necessary. AmeriHealth Caritas Northeast’s clinical policies are not guarantees of payment.

Coverage policy

AmeriHealth Caritas Northeast considers the use of COLARIS® testing, also known as genetic testing for the germline mutations MLH1, MSH2, or MSH6, which causes hereditary nonpolyposis colorectal cancer (HNPCC), or Lynch syndrome (LS), to be clinically proven and, therefore, medically necessary in members who meet the following criteria:

The initial screening for LS to evaluate tissue tumor must use microsatellite instability (MSI) or immunohistochemistry (IHC) with or without BRAF/MLH1 promoter methylation testing.

Those who are candidates for COLARIS® testing include:
- Members with colorectal cancer (CRC) diagnosed in the past.
- Members with at-risk relatives diagnosed with LS with a known mismatch repair mechanism (MMR) mutation.
- Members with endometrial cancer and one first-degree relative diagnosed with a Lynch-associated cancer, with one of the cancers being diagnosed before age 50 for the diagnosis of LS.
- Members who have been diagnosed, before age 60, with CRC or endometrial cancer and a high MSI in the tumor.
- Members with a differential diagnosis of attenuated familial adenomatous polyposis (FAP) versus (mutY homolog) s (MUTYH-) associated polyposis versus LS.
- Members without CRC but with a family history meeting the Amsterdam or revised Bethesda criteria, when no affected family members have been tested for MMR mutations; members referred for genetic testing if family member meets Amsterdam II or revised Bethesda criteria.
  a. Amsterdam II clinical criteria (all criteria must be fulfilled) for defining families at high risk for LS:
     - Three or more relatives with a histologically verified HNPCC-associated cancer (CRC or cancer of the endometrium, small intestine, ureter, or renal pelvis), one of whom is a first-degree relative of the other two.
     - HNPCC-associated cancer involving at least two generations.*
     - Cancer in one or more affected relatives diagnosed before age 50.
     - Familial adenomatous polyposis excluded in any cases of CRC.
     - Tumors should be verified by pathologic examination. (Note: Recognizing the difficulty of obtaining pathology reports of family members, pathology reports should be verified whenever possible.)

*Modifications allow for small HNPCC families: Either these families must have two CRCs in first-degree relatives involving at least two generations, with at least one individual diagnosed by age 55, or, in families with two first-degree relatives affected by CRC, the presence of a third relative with an unusual early-onset neoplasm or endometrial cancer is sufficient.

b. Revised Bethesda guidelines: Meeting any of the following is sufficient for consideration of MSI/IHC testing:
   - CRC diagnosed before age 50.
   - Presence of synchronous or metachronous CRC or other Lynch-associated tumor, regardless of age.
   - CRC with MSI-H histology diagnosed in an individual who is under age 60.
   - CRC diagnosed with one or more first-degree relatives with a Lynch-related tumor, with one of the cancers diagnosed before age 50.
   - CRC diagnosed in two or more first- or second-degree relatives with a Lynch-related tumor, regardless of age.
   - There has been a recommendation for testing made after genetic counseling performed by a specialist or other physician equivalent to that provided by a genetic counselor and also be appropriate for the test being requested.
*Note: Family history should include information on cancers in first-degree (parents, siblings, children) and second-degree (grandparents, aunts/uncles, nieces/nephews, grandchildren) relatives on maternal and paternal sides, including cancer type and age of person when diagnosed.

**Limitations:**

All other uses of COLARIS testing, also known as genetic testing for LS, are not clinically proven and, therefore not medically necessary.

- Testing must be performed at a participating laboratory facility when available.

**Alternative covered services:**

- Colonoscopy for colorectal screening.
- Network provider evaluation and surveillance.

**Background**

CRC is the second most deadly cancer (behind only lung/bronchus cancer) of men and women in the United States; in 2016, an estimated 134,490 Americans were diagnosed with the disease, and 49,190 died from it (Howlader, 2016). When CRC is detected early, illness and death can be prevented. The U.S. Department of Health and Human Services is committed to boosting public awareness about the importance of screening and treatment for CRC.

CRC poses elevated risk to adults over age 50, and the United States Preventive Services Task Force (USPSTF) recommends that all individuals ages 50 to 75 be screened for CRC as part of routine preventive health care (USPSTF, 2016). Currently, about one in three adults between the ages of 50 and 75 are not receiving recommended screening (CDC, 2013).

LS is an autosomal dominant familial cancer syndrome caused by mutations in multiple susceptibility genes (e.g., MLH1, MSH2, MSH6, PMS2, EPCAM) and is associated with an increased lifetime risk for CRC and other malignancies within the tumor spectrum including at least endometrial, ovarian, gastric, small bowel, urothelial, hepatobiliary tract, sebaceous, and pancreatic cancers. LS prevalence in CRC and endometrial cancer is estimated at 1 percent to 3 percent, and annual incidence ranges between 1 of 660 and 1 of 2,000 (de la Chapelle, 2005). In individuals with LS, the lifetime risk of CRC may be as high as 80 percent (cancer.net, 2014). While the incidence of adenomas in individuals with LS is similar to that in the general population, the high rate of CRC is due to an acceleration of the adenoma to carcinoma sequence. Cancer risks associated with LS are largely derived from family studies. Mutations in MLH1 and MSH2 account for 70 percent to 90 percent of families with LS. The risk of colon and endometrial cancer is lower in MSH6 and PMS2 mutation carriers, although the cancer risk may not be lower for MSH6 carriers.
if one takes the data out to age 80. While individuals with a single MLH1, MSH2, MSH6, and PMS2 mutation develop cancers in midlife, individuals with biallelic MLH1, MSH2, MSH6, and PMS2 mutations have a distinctive phenotype and tumor spectrum, and often develop cancer as early as the first decade of life.

First-degree relatives of mutation carriers have a 50 percent probability of having the same germline mutation. Despite the high penetrance of CRC and endometrial cancer and recommendations of consideration for screening unaffected first-degree relatives following diagnosis of LS proband, testing of genetic carriers who are unaffected with a Lynch-related cancer is not a Medicare benefit and is statutorily excluded from coverage.

COLARIS is a genetic test that assesses a person’s risk of developing hereditary colorectal cancer and a woman’s risk of developing hereditary uterine cancer. COLARIS\textsuperscript{PLUS} detects disease-causing mutations in the MLH1, MSH2, MSH6, PMS2, EPCAM, and MYH genes that are responsible for the majority of LS and MYH-associated polyposis (MAP) cases.

LS, also known as HNPCC, are the most common of the hereditary colon cancer syndromes and is believed to account for 3 percent to 5 percent of all CRCs (NLM, 2017). COLARIS is a test for LS. Knowing the results may help patients and their physicians take steps to prevent cancer before it has a chance to develop.

MAP is caused by mutations in the mutY homolog (MYH) gene. Individuals with MAP have mutations in both of their MYH genes — one from each parent — often referred to as "biallelic MYH mutations." Patients often have no family history of CRC or polyps in parents, although siblings may be affected. The MYH gene is an important part of the base excision repair (BER) pathway, which allows for repair of DNA mutations caused by oxidative damage to cells.

COLARIS is a simple blood test or oral rinse sample used to find out if a patient has MLH1, MSH2, MSH6, PMS2, EPCAM, or MYH mutations. Knowing the results may help patients and their physicians act before cancer has a chance to develop.

Searches

AmeriHealth Caritas Northeast searched PubMed and the databases of:

- UK National Health Services Centre for Reviews and Dissemination.
- Agency for Healthcare Research and Quality Guideline Clearinghouse and evidence-based practice centers.
- The Centers for Medicare & Medicaid Services (CMS).

We conducted searches on March 6, 2017. Search terms were: “Colorectal Cancer,” “Gene testing,” and “Lynch Syndrome.”
We included:

- **Systematic reviews**, which pool results from multiple studies to achieve larger sample sizes and greater precision of effect estimation than in smaller primary studies. Systematic reviews use predetermined transparent methods to minimize bias, effectively treating the review as a scientific endeavor, and are thus rated highest in evidence-grading hierarchies.

- **Guidelines based on systematic reviews.**

- **Economic analyses**, such as cost-effectiveness, and benefit or utility studies (but not simple cost studies), reporting both costs and outcomes — sometimes referred to as efficiency studies — which also rank near the top of evidence hierarchies.

**Findings**

Various professional societies and groups have endorsed universal genetic testing for LS for all persons diagnosed with CRC. An early analysis conducted for the Agency for Healthcare Research and Quality of 104 studies screening CRC patients included 40 studies assessed for clinical validity; presence of age <50 years; history of CRC or endometrial cancer in a first-degree relative; or multiple colorectal or endometrial cancers. IHC or MSI testing of tumor tissue were each as effective as more complex tests. In the 61 other studies, no long-term harm of testing was found, while genetic counseling was efficacious (Bonis, 2007).

The Evaluation of Genomic Applications in Practice and Prevention Working Group, an independent panel of experts established by the Centers for Disease Control and Prevention (CDC), recommended genetic testing for LS in all newly diagnosed CRC patients, but failed to find a specific genetic testing strategy (EGAPP, 2009; Palomaki, 2009).

A 2010 workshop in Jerusalem recommended that all CRC patients under age 70 should be screened with IHC or MSI for MMR proteins. Any abnormality means patients should be offered genetic counseling plus genetic testing for LS. Patients with LS should receive an annual colonoscopy (Boland, 2010).

The U.S. Multi-Society Task Force on Colorectal Cancer consists of the American Society for Gastroendoscopy, American Gastroenterological Association, American College of Gastroenterology, and the American Society of Colon and Rectal Surgeons. The group recommended universal testing for LS in all newly diagnosed CRC patients in persons under age 70 and persons over age 70 with a family history of the syndrome. Testing can be done by IHC and/or MSI. In addition, the panel recommends genetic evaluation for LS for persons with a history of a tumor with MMR deficiency; with uterine cancer diagnosed by age 50; with a known family gene mutation; or with fulfillment of the Amsterdam criteria or Bethesda guidelines (USMSTFCC, 2014).
An article whose primary author was an official from the CDC recommended preliminary screening tests for CRC patients, including MSI, IHC, and BRAF and/or MLH1 promoter hypermethylation testing for cases with no IHC staining for MLH1. This approach will improve efficiency and reduce costs in patients most likely to have MMR mutations (Coates, 2011).

The National Comprehensive Care Network (NCCN) updated its recommendations for LS testing in 2014, supporting universal screening for CRC patients. NCCN also recommended that the preferred method of testing is IHC or MSI alone for those with a personal or family history, and IHC and MSI together for routine testing (Hampel, 2014). The CDC publication Healthy People 2020 includes a goal to increase the proportion of newly diagnosed CRC patients tested for LS (USDHHS, 2017).

A systematic review of 12 studies found that screening the general population for LS reduced CRC incidence by 68 percent and all-cause mortality by 78 percent. Universal screening beginning at age 20 resulted in the greatest gain, but was also not cost-effective. Authors concluded inadequate evidence exists to compare harms and benefits for such screening (Prince, 2017).

Authors of a study of 1,344 CRC patients under age 60 recommended that MSI be the initial test for population-based screening of LS in younger CRC patients (Schofield, 2009). A meta-analysis of 1,114 LS families concluded 1 of 71 males and 1 of 102 females with MLH1 or MSH2 mutations can expect a diagnosis of CRC in the next five years, and thus the standard for people in their 30s (colonoscopy every one to two years) might not be justifiable for those in their 20s (Jenkins, 2015).

A systematic review of five studies of women with HNPCC failed to identify an evidence-based means for surveilling members of families with LS mutations (Auranen, 2011). However, the IHC and MSI tests continue to be the standard for LS. A study of 1,566 patients with CRC found 2.8 percent with LS (each had at least three relatives with LS). It also found IHC to be nearly as sensitive to as MSI. Bethesda criteria failed to identify 28 percent of LS patients (Hampel, 2008). Analyzing BRAF mutations can help in diagnosing LS; in one study of subjects with at least one MMR absent, BRAF mutation was found in 55 percent, or 36 of 65, simplifying genetic testing for LS (Jin, 2013).

The Bethesda guidelines are helpful, but not in all cases. One study found 62 of 1,040 CRC patients had an abnormal IHC or MSI result, but 37 percent (23) of these abnormalities did not meet Bethesda criteria (Canard, 2012). Another review found that tumor MMR testing cases in persons under age 70 with CRC fulfilling Bethesda guidelines missed 4.9 percent of LS cases, but required 28.6 percent fewer cases undergoing mutational analysis than the universal approach (Moreira, 2012).

Detection of mutations is often good predictors of CRC risk. One study found that carriers of the EPCAM deletion had a 75 percent risk of CRC (Kempers, 2011), even though this deletion only accounts for 1.1 to 2.8 percent of confirmed LS families (Kuiper, 2011). Conversely, in some patient categories, testing for LS is not always effective. One study concluded that in CRC patients under age 50 (“apparently sporadic”), only 21 percent have features of LS (Goel, 2010).
While genetic testing patients and family members with CRC and other cancers is indicated, there has been only one meta-analysis of efficacy, which found that nine of 10 studies described a reduction of CRC incidence and mortality with registration and screening (Barrow, 2013).

Significant differences in the risk of developing cancer by age 70 were found between the mutations MH1, MSH2, and MSH6. The risks of developing colorectal cancer were 41 percent for MLH1, 48 percent for MLH2, and 12 percent for MSH6; for endometrial cancer, risks were 54, 21, and 16; and for ovarian cancer, risks were 20, 24, and 1 percent (Bonadona, 2011).

Several cost-effectiveness analyses of testing for LS have been conducted. One study of screening three to four relatives of persons diagnosed with CRC, using IHC and BRAF mutation testing, found a cost per life-year gained of $44,000 for testing up to 70 years, while screening without an upper age limit cost $88,700 per life-year gained (Ladabaum, 2011). Testing women with endometrial cancer who had at least one first-degree relative with a Lynch-associated cancer yielded a favorable incremental cost-effectiveness ratio of $9,126 per life-year gained; testing all relatives of women with endometrial cancer would be more costly, i.e., $648,494 per life-year gained (Kwon, 2011).

Policy updates:

A total of 11 professional guidelines/other and 22 peer-reviewed references were added to this policy.

Summary of clinical evidence:

<table>
<thead>
<tr>
<th>Citation</th>
<th>Content, Methods, Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prince (2017)</td>
<td><strong>Key points:</strong></td>
</tr>
<tr>
<td>Screening the gen. population for LS with genetic testing</td>
<td>• Systematic review of 12 studies.</td>
</tr>
<tr>
<td></td>
<td>• No studies address risk/benefit of screening for LS in the general population.</td>
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<tr>
<td></td>
<td>• Meta-analyses find relative reductions in CRC incidence and mortality of 68% and 78%.</td>
</tr>
<tr>
<td></td>
<td>• Universal screening beginning at age 20 is most effective, but not cost-effective.</td>
</tr>
<tr>
<td>Barrow (2013)</td>
<td><strong>Key points</strong></td>
</tr>
<tr>
<td>Impact of screening on CRC incidence + mortality in LS</td>
<td>• Systematic review of 33 studies on familial adenomatous polyposis and LS.</td>
</tr>
<tr>
<td></td>
<td>• Nine of 10 studies found reduction of CRC incidence and mortality after registration and screening for LS.</td>
</tr>
<tr>
<td></td>
<td>• Evidence graded a level 2 (Grade B).</td>
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<tr>
<td>Bonadona (2011)</td>
<td><strong>Key points:</strong></td>
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<tr>
<td>Cancer risk by mutation type</td>
<td>• French study (2006–2009) of LS families with segregating mutated genes (248 with MLH1, 256 with MSH2, and 33 with MSH6).</td>
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<tr>
<td></td>
<td>• Cumulative risk of colorectal cancer by age 70 were 48%, 41%, and 12%.</td>
</tr>
<tr>
<td></td>
<td>• Cumulative risk of endometrial cancer by age 70 were 54%, 21%, and 16%.</td>
</tr>
<tr>
<td></td>
<td>• Cumulative risk of ovarian cancer by age 70 were 20%, 24%, and 1%.</td>
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</tbody>
</table>
Citation | Content, Methods, Recommendations
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Ladabaum (2011) Estimate of efficacy and cost effectiveness to identify LS | **Key points:**
- Literature review of persons newly diagnosed with CRC and their relatives.
- Current testing, screening, and surgical interventions reduce CRC deaths 7–42% and endometrial/ovarian cancer deaths 1–6%.
- IHC testing plus BRAF mutation testing had greatest cost-effectiveness ($36,200 per life-year gained); others have higher cost figure.
- Screening for LS up to age 70 cost $44,000 per incremental life-year gained, and $88,700 for all ages.

**References**

**Professional society guidelines/other:**


**Peer-reviewed references:**


**CMS National Coverage Determinations (NCDs):**

No NCDs identified as of the writing of this policy.

Under Medicare, genetic tests for cancer are a covered benefit only for a beneficiary with a personal history of an illness, injury or signs and/or symptoms thereof (i.e., clinically affected). A person with a personal history of a relevant cancer is a clinically affected person, even if the cancer is considered cured. Predictive or presymptomatic genetic tests and services, in the absence of past or present illness in the beneficiary, are not covered under national Medicare rules. CMS recognizes Lynch syndrome as “an autosomal dominant syndrome that accounts for about 3 percent to 5 percent of colorectal cancer cases. [Lynch] syndrome mutations occur in the following genes: *hMLH1, hMSH2, hMSH6, PMS2* and *EPCAM.*” CMS also recognizes FAP and MAP syndromes and their associated mutations.

**Local Coverage Determinations (LCDs):**

Genetic Testing for Lynch syndrome. CMS Medicare Coverage Database website. 
https://www.cms.gov/medicare-coverage-database/search/search-results.aspx?CoverageSelection=Both&ArticleType=All&PolicyType=Final&s=All&KeyWord=Lynch&KeyWordLookUp=Title&KeyWordSearchType=And&bc=gAAAAAAAAAAAAA%3d%3d%&. Accessed March 10, 2017.

Includes:
- L34912 Genetic testing for Lynch syndrome – First Coast Service Options, Inc.
- L35349 MolDx: Genetic testing for Lynch syndrome – CGS Administrators LLC
- L36370 MolDx: Genetic testing for Lynch syndrome – Noridian Healthcare Solutions LLC
- L36374 MolDx: Genetic testing for Lynch syndrome – Noridian Healthcare Solutions LLC
- L35024 MolDx: Genetic testing for Lynch syndrome – Palmetto GBA
- L36793 MolDx: Genetic testing for Lynch syndrome – Wisconsin Physicians Service Insurance Corporation
- L35553 Pathology and Laboratory: Genetic testing for Lynch syndrome – Cahaba Government Benefit Administrators® LLC
Commonly submitted codes

Below are the most commonly submitted codes for the service(s)/item(s) subject to this policy. This is not an exhaustive list of codes. Providers are expected to consult the appropriate coding manuals and bill accordingly.

<table>
<thead>
<tr>
<th>CPT Code</th>
<th>Description</th>
<th>Comments</th>
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<tbody>
<tr>
<td>81292</td>
<td>MLH1 (mutL homolog 1, colon cancer, nonpolyposis type 2) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; full sequence analysis</td>
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<td>81294</td>
<td>MLH1 (mutL homolog 1, colon cancer, nonpolyposis type 2) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; duplication/deletion variants</td>
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<td>81295</td>
<td>MSH2 (mutS homolog 2, colon cancer, nonpolyposis type 1) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; full sequence analysis</td>
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<td>81297</td>
<td>MSH2 (mutS homolog 2, colon cancer, nonpolyposis type 1) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; duplication/deletion variants</td>
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<td>81298</td>
<td>MSH6 (mutS homolog 6 [E. coli]) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; full sequence analysis</td>
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<td>81300</td>
<td>MSH6 (mutS homolog 6 [E. coli]) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; duplication/deletion variants</td>
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<td>81317</td>
<td>PMS2 (postmeiotic segregation increased 2 [S. cerevisiae]) (e.g., hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; full sequence analysis</td>
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<tr>
<td>81319</td>
<td>PMS2 (postmeiotic segregation increased 2 [S. cerevisiae]) (hereditary non-polyposis colorectal cancer, Lynch syndrome) gene analysis; duplication/deletion variants</td>
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<tr>
<td>81403</td>
<td>Molecular pathology procedure, Level 4 (e.g., analysis of single exon by DNA sequence analysis, analysis of &gt;10 amplicons using multiplex PCR in 2 or more independent reactions, mutation scanning or duplication/deletion variants of 2-5 exons)</td>
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<thead>
<tr>
<th>ICD-10 Code</th>
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<td>C18.0</td>
<td>Malignant neoplasm of cecum</td>
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<td>C18.1</td>
<td>Malignant neoplasm of appendix</td>
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<td>C18.2</td>
<td>Malignant neoplasm of ascending colon</td>
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<td>C18.3</td>
<td>Malignant neoplasm of hepatic flexure</td>
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<td>C18.4</td>
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<td>C18.5</td>
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<td>Malignant neoplasm of colon, unspecified</td>
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<td>C19</td>
<td>Malignant neoplasm of rectosigmoid junction</td>
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<td>ICD-10 Code</td>
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<td>C54.1</td>
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<td>Personal history of other malignant neoplasm of rectum, rectosigmoid junction, and anus</td>
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<td>Z86.010</td>
<td>Personal history of colonic polyps</td>
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