

EFFECTIVE DATE: 02|01|2027

POLICY LAST REVIEWED: 10|21|2026

OVERVIEW

Measurable residual disease (MRD) testing for cancer is rapidly becoming a sensitive and specific method for monitoring the relative amounts of tumor-derived genetic material circulating in the blood of cancer patients. These tests leverage new genomic technologies that allow detection of extremely dilute tumor material, yielding an extremely sensitive method for determining the continued presence of tumor material or, by serially testing the same individual, tracking the relative increase or decrease of tumor material being deposited in the blood. Although it is a relatively new application of novel genomic technologies, it has rapidly demonstrated its ability to impact patient care in several ways in cancer diagnosis and treatment.

The following tests are addressed in this policy:

- clonoSEQ (Adaptive Biotechnologies) – CPT code 0364U

MEDICAL CRITERIA

Medicare Advantage Plans and Commercial Products

Effective 2/1/2027, the following test(s) are considered medically necessary when the medical criteria in the online authorization tool for participating providers is met:

- Guardant Reveal (Guardant Health) – (CPT code 0569U)
- Guardant Response (Guardant Health) – (CPT code 0422U)

PRIOR AUTHORIZATION

Medicare Advantage Plans and Commercial Products

Prior authorization is required for Medicare Advantage Plans and recommended for Commercial Products via the online tool for participating providers for the following tests:

- Guardant Reveal
- Guardant Response

Medicare Advantage Plans and Commercial Products

Prior authorization is required for Medicare Advantage Plans and recommended for Commercial Products and is obtained via the online tool for participating providers. See the Related Policies section.

Note: Laboratories are not allowed to obtain clinical authorization or participate in the authorization process on behalf of the ordering physician. Only the ordering physician shall be involved in the authorization, appeal or other administrative processes related to prior authorization/medical necessity.

In no circumstance shall a laboratory or a physician/provider use a representative of a laboratory or anyone with a relationship to a laboratory and/or a third party to obtain authorization on behalf of the ordering physician, to facilitate any portion of the authorization process or any subsequent appeal of a claim where the authorization process was not followed and/or a denial for clinical appropriateness was issued, including any element of the preparation of necessary documentation of clinical appropriateness. If a laboratory or a third party is found to be supporting any portion of the authorization process, BCBSRI will deem the action a violation of this policy and severe action will be taken up to and including termination from the BCBSRI provider network. If a laboratory provides a laboratory service that has not been authorized, the service will be denied as the financial liability of the participating laboratory and may not be billed to the member.

POLICY STATEMENT

Medicare Advantage Plans and Commercial Products

Effective 2/1/2027, the following test(s) may be considered medically necessary when the medical criteria in the online authorization tool for participating providers is met:

- Guardant Reveal
- Guardant Response

Medicare Advantage Plans and Commercial Products

The following test may be considered medically necessary for Acute lymphoblastic leukemia (ALL), Multiple myeloma (MM), and Chronic lymphocytic leukemia (CLL), and Diffuse Large B-Cell Lymphoma (DLBCL). See Coding section below for details.

- clonoSEQ

Commercial Products

Some genetic testing services are not covered and a contract exclusion for any self-funded group that has excluded the expanded coverage of biomarker testing related to the state mandate, R.I.G.L. §27-19-81 described in the Biomarker Testing Mandate policy. For these groups, a list of which genetic testing services are covered with prior authorization, are not medically necessary or are not covered because they are a contract exclusion can be found in the Coding section of the Genetic Testing Services or Proprietary Laboratory Analyses policies. Please refer to the appropriate Benefit Booklet to determine whether the member's plan has customized benefit coverage. Please refer to the list of Related Policies for more information.

COVERAGE

Benefits may vary between groups and contracts. Please refer to the appropriate section of the Benefit Booklet, Evidence of Coverage, or Subscriber Agreement for applicable genetic testing and not medically necessary/not covered benefits/coverage.

BACKGROUND

Minimal Residual Disease testing can be used to:

- diagnose cancer progression, recurrence, or relapse before there is clinical, biological, or radiographical evidence of progression, recurrence, or relapse.
- detect tumor response to therapy by measuring the proportional changes in the amount of available tumor DNA.

Both above uses may enable physicians to better assign risk stratification, deploy alternate treatment strategies, or preclude the use of unnecessary adjuvant therapies.

MRD testing often requires 2 types of assays to be performed as part of the service. First, a sample is taken from tumor diagnostic material to establish a baseline (solid and/or liquid) tumor signature as defined by the test methodology. This is followed by a series of assays run on a minimally invasive specimen (i.e., liquid biopsy or bone marrow aspirate) to detect the presence or recurrence of tumor, based on the measured biomarkers, expression, or other analytes over various timepoints. Other approaches are also acceptable, based on the validity established for the individual test comprising the service. This series of assays comprises a single test when the patient is known to have cancer.

When the patient is NOT known to have cancer (specifically when there is no clinical, radiographical, or other biological evidence that tumor cells remain post treatment and subsequently the patient is no longer being subjected to therapeutic interventions for cancer), a second kind of test may exist wherein a single timepoint may constitute a single test. In such patients, the frequency of MRD testing is in accordance with national or society guidelines or recommendations.

For patients with or without cancer (as defined above), established standard-of-care MRD tests using single-gene PCR (i.e., BCR-ABL1) are covered under this policy according to testing schedules outlined in national

(i.e., NCCN) or society guidelines. MRD testing in accordance with this policy can be performed using PCR and/or sequencing-based technologies and is not restricted to a single type of biological material or defined number of genes.

Colorectal Cancer and Solid Tumors

Colorectal cancer (CRC) is the second leading cause of cancer-related mortality, with an estimated 145,600 newly diagnosed cases in the United States (U.S.) The current standard of care for patients with localized or regionally advanced CRC involves surgical resection, possibly followed by adjuvant radiation or adjuvant chemotherapy (ACT). It is generally accepted that earlier recurrences are more likely to be treated with curative intent and that these patients have improved overall survival after such interventions. Existing consensus guidelines for the treatment of colon cancer recommend surgical resection as a key treatment for Stage II cancer with the consideration of adjuvant chemotherapy, but notes that patients should be counseled that the absolute benefit is not more than 5% in colon cancer. The evidentiary review that appears to underlie the recommendation in colon cancer is based on a number of studies, among them a meta-analysis by Böckelman, et al.

The NCCN's colon cancer guideline reviews these findings in addition to a number of other studies and concludes that for patients with average risk stage II colorectal cancer the benefit of adjuvant therapy is small, and patients with high risk features have been considered more likely to benefit from adjuvant chemotherapy, although data is lacking. Uniform patient stratification based on risk features is lacking, resulting in physician discretion likely being a major factor in ACT use. In NCCN guideline concerning rectal cancer, ACT vs observation is the recommended treatment for pT3N0M0 tumors. Current surveillance methods in CRC include history taking and physical exams, periodic chest/pelvic imaging, colonoscopies, and serial carcinoembryonic antigen (CEA) monitoring at intervals dependent on patient stage. Serial CEA elevations result in a suspicion of recurrence, resulting in a subsequent workup.

MRD testing may be beneficial for patients in that it may be a more sensitive and specific method for detecting or predicting recurrent disease than current surveillance methods; furthermore, it may help risk-stratify patients that may or may not benefit from ACT because although they may not have radiographical evidence of disease, they may have residual microscopic tumor detected at the molecular level that may require additional ACT treatment. Multiple studies have to date been performed to evaluate these scenarios.

MRD in other solid tumor types

In addition to CRC, MRD testing has been performed in multiple cancer types. Validation studies for the use of MRD testing have been conducted in lung cancer, breast cancer, bladder cancer, and esophageal cancer, among others. MRD testing across multiple cancer types demonstrates consistent sensitivity and specificity (ranging from 88-100% sensitivity and 98-100% specificity). Of the mentioned studies, approximately 400 unique patients were evaluated. Moreover, in these studies, circulating tumor DNA (ctDNA) has shown significant lead time over radiographic imaging for the detection of relapse. In TRACERx, a prospective study phylogenetically profiling and monitoring (from diagnosis to death) the clonal evolution of tumors in 100 NSCLC patients, the median interval between ctDNA detection and detection of relapse by imaging was 70 days (range 10 to 346 days); in some of these cases, lead times of more than 6 months were observed. In some cases, further subclonal analysis revealed targetable mutations and amplification events implicated in driving the relapse, thereby also impacting the therapeutic options available to a given patient. Another longitudinal study in breast cancer patients found that plasma ctDNA was detected before clinical or radiologic relapse in 16 of 18 relapsed patients; moreover, ctDNA predicted metastatic relapse with a lead time of up to 2 years. A prospective study evaluating ctDNA before and after surgery and during chemotherapy in patients with locally advanced bladder cancer found that the dynamics of ctDNA during treatment is a good predictor of outcome and a better predictor of treatment efficacy than pathologic downstaging. Moreover, in this study, patients without clearance of ctDNA had a response rate of 0%. ctDNA has also been shown to accurately monitor the activity and diagnose recurrence of endometrial cancer, and multiple studies have found it to be highly sensitive for monitoring and predicting disease progression and response to therapy in patients with metastatic melanoma.

MRD in monitoring of therapeutic interventions

Immune check point inhibitors (ICI) have emerged as an effective therapy and have been approved for various types of solid tumor malignancies. However, in most settings only a minority of patients respond to immunotherapy. FDA labels for ICI therapies call for treatment until disease progression or unacceptable toxicity, however there is no definitive guidance on the method for evaluation of disease progression, which leaves this determination up to the judgement of clinicians prescribing these drugs.

The determination as to whether a tumor is progressing is currently based largely on repeated radiographic evaluation of the tumor. While tumor growth is often associated with progression, this is complicated by pseudo-progression, where immune cell infiltration may cause the tumor to initially appear larger on a scan prior to shrinking, making it difficult to ascertain in a timely fashion who is responding to treatment and who is not responding based on radiographic imaging and complicating patient management.

Numerous peer-reviewed studies have reported that monitoring of ctDNA levels, in conjunction with radiological assessment, may be a clinically valid method of assessing the efficacy of ICI, and may help differentiate between progression and pseudo-progression. These studies have ranged across many cancer types and multiple different types of immunotherapy, and have shown that a decreasing level of ctDNA during treatment (“molecular response”) is a potential indicator of treatment response, while an increasing level of ctDNA during treatment (“molecular progression”) is a potential indicator of treatment non-response.

MRD in hematopoietic malignancies

MRD use in certain hematological malignancies has been well established in the scientific literature and is used as a patient risk stratification tool and to guide treatment decisions. This is true in both the myeloid and lymphoid leukemias, and is increasingly apparent in certain lymphomas. In chronic myeloid leukemia (CML), BCR-ABL PCR tests are well-known to reliably detect the presence of leukemic cells at levels as low as 1 tumor cell per 100,000 normal cells. In acute lymphoblastic leukemia (ALL), Multiple Myeloma (MM), and Chronic Lymphocytic Leukemia (CLL), some MRD tests have demonstrated the ability to reliably detect and monitor tumor DNA from as little as 200-500ng DNA.

The goal of treatment in many hematologic malignancies has been to achieve a complete response (CR) based on morphologic or surrogate markers, and/or imaging. However, it is well-established that conventional CR is an insufficient definition of response, as many patients who achieve CR using conventional methods still harbor MRD, which can be significantly more predictive of poor outcomes.

Acute Myeloid Leukemia (AML) and Myelodysplastic Syndrome (MDS)

In AML, the goal for patients after chemotherapy is to achieve complete remission (CR) without evidence of MRD. However, it is well-known that conventional morphologic techniques may miss MRD that is below the threshold of detection, and approximately 50% of patients relapse, despite having achieved CR by standard morphologic criteria. The presence of MRD in AML indicates worse prognosis, and lower survival and relapse-free survival. Further, various therapeutic interventions may be differentially considered, depending on the molecular MRD risk assessment. For example, allogeneic hematopoietic stem cell transplant (HSCT) for persistent MRD in certain types of AML, such as t(8;21), may improve survival compared with continuation of standard therapy. Therefore, MRD testing can be useful in determining whether a patient should be referred for allogeneic HSCT in cases of persistent MRD. Moreover, in acute promyelocytic leukemia (APL), the detectable presence of the promyelocytic leukemia-retinoic receptor alpha (PML-RARA) fusion has been shown to predict relapse, and therapy at the time of molecular relapse has been reported to improve survival compared with therapy at the time of hematologic relapse. Finally, the relapse prevention with azacitidine (RELAZA2) clinical trial found that pre-emptive therapy was able to prevent or substantially delay relapse in high-risk MRD-positive patients with MDS or AML.

NCCN and the European Leukemia Net (ELN) MRD Working Party guidelines support the use of MRD testing in AML. MRD assessment methodologies in AML/APL include the use of flow cytometric methods

(FC) as well as cytogenetic and molecular methods. Molecular MRD assessment includes single-gene real-time quantitative PCR (RT-qPCR) for patients that harbor 'suitable' gene mutations, rearrangements and fusions. These include the nucleophosmin (NPM1) mutations and PML-RARA fusion in APL, as well as the Runt-related transcription factor 1 fusion (RUNX1-RUNX1T1), core binding factor-myosin heavy chain 11 fusion (CBFB-MYH11), and NPM1 mutations in AML; therefore, molecular MRD is routinely assessed in APL, CBF-AML, and NPM1-mutated AML. Many other gene aberrations commonly diagnosed in AML, such as fms-like tyrosine kinase 3- internal tandem duplication (FLT3-ITD), are not appropriate for MRD testing, as they may not be stable at relapse due to frequent gains and losses. However, MRD monitoring is expanding and is expected to become routinely used in additional types of AML and for more suitable gene mutations. Molecular MRD assessment may also include multi-gene sequencing (i.e., NGS); however, targeted PCR-based assays for MRD testing in AML have historically demonstrated superior sensitivity and are not confounded by clonal hematopoiesis of indeterminate potential (CHIP, discussed in further detail below) making them less challenging to interpret than NGS. NGS, however, has recently demonstrated increased sensitivity such that NGS is expected to become a more commonly used and versatile approach to MRD testing in AML.

Chronic Myeloid Leukemia (CML)

RT-qPCR for the Breakpoint Cluster Region-Tyrosine Kinase ABL1 (BCR-ABL1) gene fusion, also known as the Philadelphia Chromosome (Ph+), is the current gold standard for MRD testing in CML. Results of BCR-ABL1 provide information regarding molecular response (MR) as well as the potential for tyrosine-kinase (TKI)-resistant disease, allowing for subsequent changes to patient management. In CML, there exist well-established targets and molecular response milestones over time such that testing can guide therapeutic decision-making, including the need to switch therapies, refer for allogeneic HSCT, or discontinue TKI therapy. Moreover, discontinuation of TKI therapy is reliant on a qPCR with a sensitivity of detection of at least BCR-ABL1 international scale (IS) $\leq 0.0032\%$ (molecular response (MR)4.5), with a time to results of less than 2 weeks. For patients who do not achieve response milestones, BCR-ABL1 kinase domain mutational analysis and bone marrow cytogenetic analysis may be performed.

Studies have shown that CML patients with certain rare variant transcripts of BCR-ABL1 have a worse outcome than patients with the most common BCR-ABL1 fusion, and that this may also be relevant for TKI therapy outcomes. However, most laboratories do not perform testing for atypical transcripts; moreover, standardization to the IS is not available for these transcripts. Though monitoring in such cases may be performed by fluorescence in situ hybridization (FISH), amplification-based approaches, such as multiplex PCR, have shown promise for MRD testing in CML patients with atypical BCR-ABL1 transcripts.

Resistance mutations in non-BCR-ABL1 genes have also been associated with poor outcomes and resistance to TKIs. The NEXT-in-CML study, a prospective multicenter study evaluating 236 CML patients with failure or warning response to TKI therapy, found that NGS detected mutations in 47% of patients, while sanger sequencing (SS) detected mutations in only 25%; NGS additionally detected clonally complex mutations (including compound mutants) that were missed by SS. Therefore, for patients who do not achieve response milestones, as well as for patients with no identifiable BCR-ABL1 mutations, NGS with a myeloid mutation panel is recommended by the ELN and NCCN guidelines.

Acute Lymphocytic Leukemia (ALL)

MRD is used in ALL patients to monitor complete response (CR) duration and to make treatment decisions. NCCN guidelines recommend the use of MRD testing in these patients. The most common genetic aberration in these patients is the Philadelphia Chromosome (Ph+) and patients with this alteration are less likely to respond to chemotherapies and have traditionally had worse outcomes. Selective targeted therapies have been created that have resulted in improved outcomes for these patients, but about one third of these patients will relapse. A systematic literature review in late 2019 of MRD use in adult B-cell acute lymphoblastic leukemia (B-ALL), the most common subtype of ALL, included 23 articles and abstracts and describes the common uses of MRD and describes the clinical validity and utility of the test across these studies. The most frequently employed MRD tests includes flow cytometry, RT-qPCR for fusion genes (such as BCR-ABL1) and NGS-based assays to detect clonal rearrangements in immunoglobulin heavy chain (IgH)

genes and/or T-cell receptor (TCR) genes. The review found that MRD status was consistently demonstrated to be predictive of overall survival and could be used to assess patient risk stratification and treatment response. MRD was shown to be useful after induction chemotherapy to identify the quality of response when morphological remission is obtained, as well as a predictor for pending relapse in these patients. The predictive ability of MRD is present regardless of Ph status.

Multiple Myeloma (MM)

Immunomodulatory drugs, such as lenalidomide, and proteasome inhibitors have become available to MM patients and allow for a large percentage to achieve CR. However, many patients will relapse. A large-cohort (14 studies) meta-analysis in 2017 demonstrated the clinical validity of MRD testing to predict survival outcomes, including in patients that demonstrated CR, and utility in treatment selection. It was also demonstrated to be useful in monitoring maintenance therapy. As in ALL, CLL, and lymphoma, a primary molecular target for MRD assessment in MM is IgH. Use of MRD testing is required for the assessment of relapse in the 2021 NCCN Guidelines for MM.

Chronic Lymphocytic Leukemia (CLL)

Although many CLL patients have prolonged survival or cure after treatment with fludarabine, cyclophosphamide, and rituximab, the risk of relapse remains. Flow cytometry is an accepted method for risk stratification of patients and assessment for CR after treatment to assess residual disease, however NGS-based MRD was demonstrated to be more sensitive and a better predictor of patient outcomes, possibly because other methods are not sensitive enough to accurately predict CR. As in ALL, MM and lymphoma, a primary molecular target for MRD assessment in CLL is IgH. 2021 NCCN Guidelines describes MRD testing as an important predictor of treatment efficacy and describes NGS-based methods as more sensitive than PCR or flow cytometry-based testing.

Lymphoma

Although many patients with a B-cell lymphoma (BCL) experience prolonged remission or achieve cure after systemic therapy, many will relapse and suffer adverse outcomes. There is substantial evidence that measurement of MRD in different types of BCL using various cell free DNA (cfDNA) profiling assays can characterize risk and detect residual disease earlier than clinical relapse. MRD testing with cfDNA has shown improved sensitivity and specificity compared with computerized tomography (CT) and positron emission/CT (PET/CT) traditionally used in disease surveillance, with lead times of 3-6 months for the detection of relapse. The use of ctDNA MRD tests in lymphoma may therefore allow for a reduction in the frequency of surveillance imaging, with a consequent reduction in radiation exposure, a potential health risk emphasized by lymphoma investigators.

In addition to assessment of disease burden, prognostication, and monitoring for relapse, ctDNA tests for MRD in lymphoma may allow for the customization of therapy (“risk-adapted” strategies) and initiation of pre-emptive therapy designed to prevent overt clinical relapse. In 1 study monitoring 183 mantle cell lymphoma patients after autologous stem cell transplantation, pre-emptive rituximab was administered to patients with evidence of molecular relapse; the patients treated pre-emptively converted to MRD negativity. Though the results were uncontrolled, the authors acknowledge that this likely resulted in a delay in overt clinical relapse. Another study in Hodgkin lymphoma patients found that MRD testing using ctDNA can track clonal gene evolution and monitor residual disease during multiagent chemotherapy. The authors highlight that this approach to MRD testing can complement PET/CT and identify patients likely to be chemo refractory, with the goal of informing early treatment intensification (or de-escalation, in the case of unlikely chemoresistance).

Importantly, it is not uncommon for lymphoma patients who ultimately become ctDNA-positive to have had ≥ 1 prior ctDNA-negative results, highlighting the utility of serial disease monitoring over time. NCCN guidelines support the use of molecular analysis in the differential diagnosis of BCL, to detect IgH and TCR gene rearrangements. Various highly sensitive NGS-based techniques are available for detecting MRD in lymphoma. However, despite their high sensitivity for detecting MRD, the implementation of MRD testing into routine MRD protocols has not been optimized in B or T-cell lymphomas.

Limitations of ctDNA for MRD assessment

MRD testing using ctDNA is not without limitations and challenges in interpretation. Discordance of mutations found between ctDNA and tissue is well-described and can occur in a significant proportion of cases. Reasons for this are varied and include tumor heterogeneity, clonal evolution, and time of sampling (i.e., contemporaneous vs remote sampling between ctDNA and tissue). Studies have also reported that ctDNA may be enriched for therapy resistance alterations as well as for variants associated with CHIP (sometimes referred to as age-related clonal hematopoiesis, or 'ARCH'). CHIP is known to increase with age and occurs in 10->30% of adults over 70 years of age. In 1 study of CRC patients, 17% of the pre-operative cell-free DNA mutations were determined to be CHIP and persisted post-operatively as well as after chemotherapy. In another study in advanced prostate cancer patients, CHIP variants accounted for nearly half of the cell-free somatic DNA repair gene variants detected. Importantly, many of these DNA repair genes are used for determination of eligibility for poly (ADP) ribose polymerase inhibitors (PARPi) therapy. In the study by Jensen et al., if a whole blood control had not been performed, 10% of patients would have been incorrectly considered eligible for PARPi therapy as a result of CHIP interference. Misclassification of CHIP as tumor-derived mutations can therefore lead to incorrect evaluation of residual disease as well as subsequent inappropriate treatment. For these reasons, some researchers have advocated for the use of paired ctDNA testing, using paired peripheral blood cell or tumor tissue as one potential approach to testing. There may be other approaches (i.e., algorithms and filters based on known population frequencies of genetic variants) to mitigate confounding by CHIP. This is an ongoing area of investigation.

Further, as ctDNA for MRD testing is specific to a given tumor, testing cannot be used to detect a second primary tumor, including 1 located within the same organ (i.e., 2 separate primary lung tumors). Testing by this methodology also requires the presence of sufficient ctDNA molecules in the plasma (or other tested compartment), a special consideration particularly in patients with smaller or less aggressive cancers. Finally, it is critical that MRD tests achieve high sensitivities at limits of detection significantly lower than those typically attained by NGS tests used for diagnosis.

Evidence supports that MRD testing can be used to accurately predict disease recurrence or progression before clinical or radiographical evidence is evident (establishing molecular recurrence) and performs better than other established methods for disease surveillance such as serial CEA monitoring, physical exams, imaging, or flow cytometry. Although this is a logical progression of the understanding of the development and evolution of cancer (that tumor cells grow and shed DNA at proportional levels until such a time there is macroscopic disease in organs or bone marrow), the evidence clearly establishes that MRD testing can demonstrate acceptable clinical validity in the determination of disease recurrence; a condition whose identification has pre-established utility as it is an event that in the proper clinical context requires altering or modifying patient management. Current medical practice, including as defined in the NCCN guidelines, clearly advocate for changing or re-establishing treatment when such a diagnosis is rendered. As such, determining molecular recurrence before there is clinical or radiographical evidence of it is likely to further improve patient outcomes and is consistent with current guidelines that advocate for early detection of and treatment for recurrence. Furthermore, additional uses of MRD have been established, such as for monitoring treatment response, although it is based on the same principle. Studies demonstrate the clinical validity of molecular progression as predictive of failure to respond to treatment and demonstrate futility in continued therapy. The utility of such testing in maintenance therapy monitoring to improve patient outcomes is therefore similarly inherent; preclusion of potentially hazardous compounds that are not likely to have clinical benefit and prevention of adverse events have demonstrated improved patient outcomes.

This remains a rapidly evolving field, and it is anticipated that new evidence may emerge either showing limitations of the clinical utility underlying MRD testing or additional strengths and new applications.

CODING

Medicare Advantage Plans and Commercial Products

The following CPT codes may be considered medically necessary for Medicare Advantage Plans and Commercial Products when the medical criteria in the online authorization tool for participating providers is met:

- Guardant Reveal (Guardant Health) – (CPT code 0569U)
- Guardant Response (Guardant Health) – (CPT code 0422U)

The following CPT code is medically necessary for Medicare Advantage Plans and Commercial Products when filed with an ICD-10 diagnosis code(s) listed below:

This code can be used for clonoSEQ:

0364U Oncology (hematolymphoid neoplasm), genomic sequence analysis using multiplex (PCR) and next-generation sequencing with algorithm, quantification of dominant clonal sequence(s), reported as presence or absence of minimal residual disease (MRD) with quantitation of disease burden, when appropriate

ICD-10 Diagnosis Code(s):

C83.10 – C83.1A

C83.30 - C83.3A

C90.00 - C90.02

C91.00 - C91.02

C91.10 - C91.12

Z85.6

Z85.72

Z85.79

RELATED POLICIES

Biomarker Testing Mandate

PUBLISHED

Provider Update, December 2026

Provider Update, March/June/September 2025

Provider Update, June 2024

Provider Update, April/September/November/December 2023

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