Corporate Medical Policy

Cardiac Biomarkers for Myocardial Infarction AHS – G2150

Description of Procedure or Service

Cardiac biomarkers are the biochemical markers released in blood from the injured myocardial tissue. They become elevated in blood after a certain period and can be measured. The examples of cardiac biomarkers commonly used in clinical setting include Troponin and Creatine Kinase MB isoenzyme (CKMB) (Thygesen, Alpert, & White, 2007).

***Note: This Medical Policy is complex and technical. For questions concerning the technical language and/or specific clinical indications for its use, please consult your physician.

Policy

BCBSNC will provide coverage for cardiac biomarkers for myocardial infarction when it is determined the medical criteria or reimbursement guidelines below are met.

Benefits Application

This medical policy relates only to the services or supplies described herein. Please refer to the Member’s Benefit Booklet for availability of benefits. Member’s benefits may vary according to benefit design; therefore member benefit language should be reviewed before applying the terms of this medical policy.

When Cardiac Biomarkers For Myocardial Infarction is covered

Reimbursement is allowed for measurement of cardiac troponin (troponin T or I) for the diagnosis of myocardial infarction (MI) is considered medically necessary in all patients presenting with signs and symptoms of acute coronary syndrome* (please see Note 1)

When Cardiac Biomarkers For Myocardial Infarction is not covered

1. Reimbursement is not allowed for measurement of the following cardiac biomarkers for the diagnosis and/or prognosis of MI in patients presenting with signs and symptoms of acute coronary syndrome*:
   a) Aspartate aminotransferase (AST/SGOT)
   b) Cardiac creatine kinase isoenzyme MB (CKMB)
   c) Creatine kinase (CK)
   d) Creatine kinase Isoenzymes
   e) Lactate Dehydrogenase (LD, LDH)
   f) Myoglobin
2. Reimbursement is not allowed for measurement of cardiac biomarkers in patients presenting with signs and symptoms of acute coronary syndrome* in an outpatient setting which is not capable of performing adequate clinical MI evaluation (eg. independent lab or physician’s office).

3. Reimbursement is not allowed for measurement of the following cardiac biomarkers for the diagnosis and/or prognosis of MI in patients presenting with signs and symptoms of acute coronary syndrome*:
   a) Copeptin
   b) Troponin C
   c) C-reactive protein
   d) Heart-type fatty acid binding protein (H-FABP)
   e) any other cardiac biomarkers not listed above

*Note 1:  
Acute Coronary Syndrome/Myocardial Infarction Common Signs and Symptoms (Reeder, 2018):  
Ischemic chest pain with radiation to an upper extremity, radiation to both arms, and pain associated with diaphoresis or with nausea and vomiting.
Squeezing, tightness, pressure, constriction, crushing, strangling, burning, heartburn, fullness in the chest, band-like sensation, knot in the center of the chest, lump in throat, ache, heavy weight on chest and toothache (when there is radiation to the lower jaw).
Ischemic pain often radiates to other parts of the body including the upper abdomen (epigastrium), shoulders, arms (upper and forearm), wrist, fingers, neck and throat, lower jaw and teeth (but not upper jaw), and not infrequently to the back (specifically the interscapular region).
Shortness of breath, belching, nausea, indigestion, vomiting, diaphoresis, dizziness, lightheadedness, clamminess, and fatigue.

Atypical Signs and Symptoms (Reeder, 2018):  
Dyspnea alone, weakness, nausea and/or vomiting, epigastric pain or discomfort, palpitations, syncope, or cardiac arrest.

Reimbursement Limitations:  
Maximum of 4 serial troponin tests will be reimbursed (eg. Repeat troponin measurements) in the first 24-72 hours after presentation.

Policy Guidelines
Background

Acute coronary syndromes (ACS) represent continuous events starting with angina, reversible injury, progressing to unstable angina, frequently associated with minor myocardial damage, and myocardial infarction (MI) that results in extensive tissue necrosis (Thygesen et al., 2007). Patients with ACS usually present with chest pain and associated signs and symptoms. These patients are subdivided into two major categories based on the 12-lead electrocardiogram (ECG). If ST-segment elevations observed on the ECG, they are indicative of acute ST-elevation myocardial infarction (STEMI) type of ACS. If the ECG shows ST-segment depression, T-wave changes, or no ECG abnormalities, it is indicative of non-ST elevation myocardial infarction (NSTEMI) and unstable angina. The ACS is a complex syndrome. However, the most common cause is atherosclerotic coronary artery disease with rupture of atherosclerotic plaque.
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(Amsterdam et al., 2014). First documented definition of acute MI was established in 1979 by World Health Organization. It included in the criteria for MI diagnosis the recommendation to use the rise or fall patterns of cardiac biomarkers such as CK, CKMB, LDH or AST activities (WHO, 1979). Since then, other societies proposed their own criteria for diagnosis. The third universal definition of MI includes typical clinical symptoms, suggestive ECG changes, or imaging evidence of new loss of viable myocardium or new regional wall abnormality with a rise and/or fall of cardiac biomarkers (Thygesen et al., 2012). Nonetheless, the universal criteria are being refined by cardiovascular societies and will likely change with scientific progress and better understanding of MI pathophysiology.

Myocardial infarction results in cardiac injury and extensive tissue necrosis. The cellular membranes become compromised and release structural proteins and other macromolecules into cardiac interstitial, called cardiac biomarkers. The levels of these cardiac biomarkers in blood will rise and fall with time after MI (Thygesen et al., 2007). The first cardiac biomarker, aspartate aminotransferase (AST), was used for MI diagnosis in 1954. AST is present in human tissues as two isoenzymes: cytoplasmic and mitochondrial. AST is a non-specific biomarker and its activity could also be elevated in other conditions such as hepatic congestion secondary to congestive heart failure. Since then, other cardiac biomarkers were used as an aid in diagnosis of MI, but due to their non-specificity and other reasons, many of them are no longer used in clinical practice or their use remains very limited (Danese & Montagnana, 2016). The most common cardiac biomarkers and their characteristics are well summarized in the table from Danese & Montagnana, 2016:

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>First assay development, year</th>
<th>Molecular weight, Da</th>
<th>First detection, hours</th>
<th>Maximum value, hours</th>
<th>Return to normal values, days</th>
<th>Sensitivity for myocardial necrosis</th>
<th>Specificity for myocardial necrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>1954</td>
<td>105,000</td>
<td>3-4</td>
<td>15-28</td>
<td>5</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>LDH</td>
<td>1955</td>
<td>140,000</td>
<td>5-10</td>
<td>60-114</td>
<td>12</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>CK total enzyme activity</td>
<td>1960</td>
<td>83,000</td>
<td>3-9</td>
<td>10-20</td>
<td>3</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>CK-MB activity</td>
<td>1972</td>
<td>83,000</td>
<td>3-8</td>
<td>10-20</td>
<td>3</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1978</td>
<td>17,800</td>
<td>1-3</td>
<td>4-7</td>
<td>1-1.5</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>CK-MB mass</td>
<td>1985</td>
<td>83,000</td>
<td>3-12</td>
<td>12-18</td>
<td>2-3</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>cTnI</td>
<td>1987</td>
<td>23,900</td>
<td>3-7</td>
<td>10-20</td>
<td>10</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>cTnT</td>
<td>1989</td>
<td>37,000</td>
<td>3-8</td>
<td>12-120</td>
<td>14</td>
<td>++++</td>
<td>++++</td>
</tr>
</tbody>
</table>

Lactate Dehydrogenase (LDH, also known as LD)

Lactate dehydrogenase is a cytoplasmic enzyme present in many different tissues such as skeletal muscle, liver, heart, kidney and red blood cells. There are five isoenzymes that has been identified by gel electrophoresis and other techniques (Marshall, Williams, & Williams, 1991). The heart isoenzymes, LD1 and LD2, activity increases in blood after five to ten hours after MI symptoms onset and remains elevated for up to ten days (Danese & Montagnana, 2016). LD has poor specificity for cardiac tissue and is not recommended as a biomarker for the diagnosis of MI (Amsterdam et al., 2014), its use as cardiac biomarker is discouraged (Jaffe & Morrow, 2018).

Myoglobin

Myoglobin is an oxygen-binding, cytoplasmic, heme protein. It is one of the first cardiac biomarkers measurable in the serum that appears between one and three hours after MI symptoms onset. Myoglobin is present in skeletal and cardiac muscles and is cleared by the kidneys (Vaidya, 1994). Its clinical utility is limited by its poor specificity. The main reason of using myoglobin in
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clinical setting was its sensitivity for MI (Danese & Montagnana, 2016), but with appearance of sensitive troponin assays, myoglobin use offers little advantage for the diagnosis of MI (Eggers, Oldgren, Nordenskjold, & Lindahl, 2004; Kavsak et al., 2007). Currently, there are no recommendations for the myoglobin to be used in the diagnosis of MI (Amsterdam et al., 2014) and its use as cardiac biomarker is discouraged (Jaffe & Morrow, 2018).

Creatine Kinase Isoenzymes and Isoform MB (CKMB)

The cytosolic enzyme creatine kinase (CK) formerly was known as creatine phosphokinase (Danese & Montagnana, 2016). The CK is present as three cytosolic isoenzymes and one mitochondrial isoenzyme. These isoenzymes are dimers of M (muscle) and B (brain) chains that exist in three combinations: MM, MB and BB (Bessman & Carpenter, 1985). The CKMM is predominant in both heart and skeletal muscle, but CKMB is more specific for the myocardium. The total CK activity could be detected in blood 3-9 hours after MI, it reaches the maximum levels in blood in 10-20 hours and returns to normal in about 72 hours (Penttila, Penttila, & Rantanen, 2000). The measurement of total CK activity is not specific to MI because it also increases in liver, biliary tract, kidneys and skeletal muscle disease and its measurement is problematic in older individuals with lower muscle mass (Dillon et al., 1982; Heller, Blaustein, & Wei, 1983; Yusuf et al., 1987). Once, CKMB mass (CKMB protein concentration measurements) was the cardiac biomarker of the choice that replaced CK, CKMB activity, AST and LDH (Danese & Montagnana, 2016). However, with arrival of cardiac troponin assays, the use of CKMB became less popular. Yet, some clinicians advocate for the use of CKMB for diagnosis and prognosis of MI, but cardiac troponins have shown either equally reliable or superior results compared to CKMB (Jaffe & Morrow, 2018) and troponin is the recommended test for MI diagnosis now (Amsterdam et al., 2014).

Troponins

There are three isoforms of regulatory protein troponin in troponin complex. Troponin C (TnC) is responsible for calcium binding and has no role to play as a cardiac biomarker. Troponin I (TnI) and Troponin T (TnT) are responsible for inhibition of ATPase activity and tropomyosin binding respectively (Greaser & Gergely, 1971). Contrary to all previously used cardiac biomarkers, cardiac troponins have high specificity for cardiac tissue. The cardiac troponins have a specific pattern of expression, they have different amino sequences encoded by different genes for skeletal and cardiac muscles. Cardiac TnI has an additional 31-amino acid residue compared to skeletal muscle. This protein has never been found to be expressed in normal or regenerating, or diseased skeletal muscle from human or animal origin (Bodor, Porterfield, Voss, Smith, & Apple, 1995). Cardiac TnT has an additional 11-amino acid residue, except this protein was also found in regenerating rat skeletal muscle, during human fetal development, and in diseased human skeletal muscle (Anderson, Malouf, Oakeley, Pagani, & Allen, 1991; Bodor et al., 1997; Saggin, Gorza, Ausoni, & Schiaffino, 1990). In addition, cardiac TnT was also found in skeletal muscle specimens from patients with muscular dystrophy, polymyositis, and chronic renal disease (Bodor et al., 1997; McLaurin, Apple, Voss, Herzog, & Sharkey, 1997).

In addition, other cardiac biomarkers keep appearing in the field such as heart-type fatty acid binding protein (H-FABP) and Copeptin. However, they are not commonly used in clinical setting because they still require additional research to prove their clinical utility.

Heart-type fatty acid binding protein (H-FABP)

H-FABP is a small cytoplasmic protein present in cardiomyocytes. It is believed to have a function in myocardial lipid homeostasis (Glatz & van der Vusse, 1990). Because of its small size, this protein appears in the blood after MI almost as early as myoglobin, but it has better specificity than myoglobin for cardiac tissue (Van Nieuwenhoven et al., 1995). Seino et al. have compared the use of H-FABP with rapid troponin in 371 patients with acute chest pain (Seino et al., 2003). Their study have demonstrated that H-FABP had significantly higher sensitivity (89%)
than troponin T (22%) and myoglobin (38%) with lower specificity (52%) than troponin (94%). Other studies were performed to compare H-FABP to troponins, however they were unable to demonstrate superior results compared to troponins. In addition, currently, H-FABP is not approved in the United States for clinical use (Jaffe & Morrow, 2018).

Copeptin

Copeptin is a peptide of 39 amino acids which is the C-terminal part of pro-arginine vasopressin (AVP). After MI, copeptin levels increase rapidly and decline over the next two to five days (Khan et al., 2007). In the Copeptin Helps in the Early Detection of Patients With Acute Myocardial Infarction (CHOPIN), 16-site study, involving 1,967 patients presenting within 6 hours of pain onset, copeptin was shown to have a potential value in ruling-out MI with a negative predictive value greater than 99% when combined with TnI measurements (Maisel et al., 2013). The Advantageous Predictors of Acute Coronary Syndrome Evaluation (APACE) multicenter study, involving 1,439 patients presenting with MI symptoms have demonstrated no benefit in using copeptin as an early rule-out cardiac biomarker for MI (Hillinger et al., 2015). Finally, there was no recommendation from professional societies to use copeptin in the diagnosis of MI (Jaffe & Morrow, 2018).

Applicable Federal Regulations

Cardiac biomarkers testing is performed in laboratories meeting Clinical Laboratory Improvement Act (CLIA) quality standards. There is a multitude of FDA-approved cardiac biomarkers tests for Troponin and CKMB currently available in high and moderate complexity formats.

Guidelines and Recommendations

2012 ESC/ACCF/AHA/WHF Third Universal Definition of Myocardial Infarction (Thygesen et al., 2012)

Myocardial Infarction was previously defined by the presence of clinical features such as ECG findings, elevated cardiac markers and imaging studies. However, scientific progress leading to myocardial tissue-specific cardiac biomarkers and more sensitive imaging techniques prompted the revision of the Universal Definition of MI by the Third Global MI Task Force which is the unified effort of the European Society of Cardiology (ESC), the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA) and the World Heart Federation (WHF). In their new definition of MI, the Task Force recognized that the myocardial injury could be detected when there is an increase in cardiac biomarkers such as CKMB or troponin. According to the Task Force, their preferred biomarker for each category of MI is cardiac troponin cTn (I or T) because it has high myocardial tissue specificity and high clinical sensitivity. They recognized rise and/or fall of the levels of cTn as “essential to the diagnosis of acute MI”. The 99th percentile of a normal reference population increase was designated by the Task Force as the decision level for the diagnosis of MI. They recommended performing cTn measurements at first assessment, repeated 3-6 hours later. In addition, they suggested that later samples are required in cases where further ischemic episodes occur or when the timing of initial symptoms is unclear. It is the rise and/or fall in cardiac biomarkers which was recognized as an important indication for MI diagnosis. The elevated troponin values, without rise/fall pattern, were recognized to be indicative of MI in the high pre-test risk of MI setting. Finally, in the absence of troponin assay, the CKMB test was recognized as the best alternative and it is the measurement above the 99th percentile URL which was designated as the decision level for the diagnosis of MI. For the CKMB, gender-specific values were recommended.

2014 AHA/ACC Guideline for the Management of Patients with Non-ST-Elevation Acute Coronary Syndromes (NSTEMI) (Amsterdam et al., 2014)
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The American College of Cardiology (ACC) and the American Heart Association (AHA) have developed clinical practice guidelines to provide recommendations applicable to patients with or at risk of developing cardiovascular disease and to provide guidance to clinicians on optimal management of patients with NSTE-ACS. In their comprehensive document, AHA/ACC panel have provided recommendations for initial evaluation and management of patients presenting with ACS symptoms, for the early hospital care, myocardial revascularization, late hospital care, hospital discharge and posthospital discharge care, special patient groups and quality of care and outcomes for ACS. The Task Force recommended to stratify patients with suspected ACS based on the likelihood of ACS and those with high-risk features should be referred immediately to the emergency department (ED). They have provided specific recommendations for the use of cardiac biomarkers in the diagnosis and prognosis of MI. They specifically recommended using troponin (troponin I or T when contemporary assay is used) for the diagnosis of MI. According to AHA/ACC guidelines, the cardiac troponin is recommended and should be measured at presentation and 3 to 6 hours after symptom onset in all patients who present with ACS symptoms. The panelists recommended identifying rising and/or falling pattern of troponin. In addition, they recommended measuring troponin levels beyond 6 hours after symptom onset in patients with normal troponins on serial examination when ECG changes and/or clinical presentation suggests ACS. If the onset of symptoms is not clearly identified, they recommended using the time of presentation as the time of onset for measuring troponin. The AHA/ACC guideline clearly highlighted that CKMB or myoglobin should not be used for the diagnosis of ACS. All recommendations for the use of cardiac biomarkers in the diagnosis of MI were level A evidence.

The AHA/ACC guideline considered all recommendations in the use of cardiac biomarkers for ACS prognosis as level of evidence B. They considered the presence and magnitude of troponin elevations useful for short and long-term prognosis. The re-measurement of troponin once on day 3 or day 4 in patients with MI was considered reasonable to estimate the infarct size and dynamics of necrosis. Finally, they considered the use of B-type natriuretic peptide to be reasonable for additional prognostic information.

The recommendations for the use of cardiac biomarkers in the diagnosis and prognosis of MI was well summarized in Table from 2014 AHA/ACC guidelines p.2655 (Amsterdam et al, 2014):

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Measure cardiac-specific troponin (troponin I or T) at presentation and 3–6 h after symptom onset in all patients with suspected ACS to identify pattern of values</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Obtain additional troponin levels beyond 6 h in patients with initial normal serial troponins with electrocardiographic changes and/or intermediate/high risk clinical features</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Consider time of presentation the time of onset with ambiguous symptom onset for assessing troponin values</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>With contemporary assays, CK-MB and myoglobin are not useful for diagnosis of ACS</td>
<td>III: No Benefit</td>
<td>A</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Troponin elevations are useful for short- and long-term prognosis</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Remeasurement of troponin value once on day 3 or 4 in patients with MI may be reasonable as an index of infarct size and dynamics of necrosis</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>BNP may be reasonable for additional prognostic information</td>
<td>IIb</td>
<td>B</td>
</tr>
</tbody>
</table>

ACS indicates acute coronary syndromes; BNP, B-type natriuretic peptide; CK-MB, creatine kinase myocardial isoenzyme; COR, Class of Recommendation; LOE, Level of Evidence; and MI, myocardial infarction.

2013 (published 2014) Society for Cardiovascular Angiography and Interventions (SCAI) (Moussa et al., 2013)
In their expert consensus document titled Consideration of a New Definition of Clinically Relevant Myocardial Infarction After Coronary Revascularization, the SCAI expert panel introduced a new definition of clinically relevant MI after coronary revascularization percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). In their definition of clinically relevant MI after both PCI and CABG procedures, authors gave recommendations according to 3 different types of clinical presentation. In the first case, when patient has a normal CKMB baseline: “The peak CK-MB measured within 48 hours of the procedure rises to >10x the local laboratory ULN, or to >5x ULN with new pathologic Q-waves in >2 contiguous leads or new persistent LBBB. OR in the absence of CK-MB measurements and a normal baseline cTn, a cTn (I or T) level measured within 48 hours of the PCI rises to >70x the local laboratory ULN, or >35x ULN with new pathologic Q-waves in >2 contiguous leads or new persistent LBBB”. In case when patients have elevated baseline CKMB (or cTn) with stable or falling biomarkers levels, they issued the following recommendation: “The CK-MB (or cTn) rises by an absolute increment equal to those levels recommended above from the most recent pre-procedure level”. And, in patients with elevated CKMB (or cTn), but without stable or falling biomarkers level, the recommendation was: “The CK-MB (or cTn) rises by an absolute increment equal to those levels recommended above plus new ST-segment elevation or depression plus signs consistent with a clinically relevant MI, such as new onset or worsening heart failure or sustained hypotension”. The authors have expressed preference to use CKMB instead of cTn, but they have included cTn in their definition if CKMB was not available.

2015 AHA Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (O’Connor et al, 2015)

In their review of previously issued guidelines, the expert panel have introduced new recommendation for diagnostic interventions in ACS regarding cardiac biomarkers. They still recommended to use Troponin in following situations: “We recommend against using hs-cTnT and cTnI alone measured at 0 and 2 hours (without performing clinical risk stratification) to identify patients at low risk for ACS (Class III: Harm, LOE B-NR). We recommend that hs-cTnI measurements that are less than the 99th percentile, measured at 0 and 2 hours, may be used together with low-risk stratification (TIMI score of 0 or 1 or low risk per Vancouver rule) to predict a less than 1% chance of 30-day MACE (Class IIa, LOE B-NR). We recommend that negative cTnI or cTnT measurements at 0 and between 3 and 6 hours may be used together with very low-risk stratification (TIMI score of 0, low-risk score per Vancouver rule, North American Chest Pain score of 0 and age less than 50 years, or low-risk HEART score) to predict a less than 1% chance of 30-day MACE (Class IIa, LOE B-NR)””. They did not express a preference in cardiac biomarkers to use neither they gave any recommendations regarding CKMB.


In 2016 The American College of Cardiology (ACC), Society for Cardiovascular Angiography and Interventions (SCAI), Society of Thoracic Surgeons (STS), and American Association for Thoracic Surgery (AATS), along with key specialty and subspecialty societies created an Appropriate Use Task Force with the mission to revise the appropriate use criteria (AUC) for coronary revascularization. They have used clinical scenarios to mimic patient presentations seen in everyday clinical practice and included information on symptom status, presence of clinical instability or ongoing ischemic symptoms and other characteristics. They are following 2014 AHA/ACC recommendations for the use of cardiac biomarkers (Amsterdam et al., 2014).

2017 International Liaison Committee on Resuscitation (ILCOR) Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations Summary (Olasveengen et al., 2017). No recommendations were given regarding the use of cardiac markers.

Billing/Coding/Physician Documentation Information

This policy may apply to the following codes. Inclusion of a code in this section does not guarantee that it will be reimbursed. For further information on reimbursement guidelines, please see Administrative Policies on the Blue Cross Blue Shield of North Carolina web site at www.bcbsnc.com. They are listed in the Category Search on the Medical Policy search page.
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Applicable service codes: 82550, 82552, 82553, 82554, 82725, 83615, 83625, 83874, 84450, 84484, 84512, 84588, 86140, 84999

BCBSNC may request medical records for determination of medical necessity. When medical records are requested, letters of support and/or explanation are often useful, but are not sufficient documentation unless all specific information needed to make a medical necessity determination is included.

Scientific Background and Reference Sources


Cardiac Biomarkers for Myocardial Infarction AHS – G2150


http://www.uptodate.com
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Policy Implementation/Update Information

1/1/2019 New policy developed. BCBSNC will provide coverage for cardiac biomarkers for myocardial infarction when it is determined to be medically necessary because the criteria and guidelines are met. Medical Director review 1/1/2019. Policy noticed 1/1/2019 for effective date 4/1/2019. (jd)

10/01/19 Policy Statement revised to read: BCBSNC will provide coverage for cardiac biomarkers for myocardial infarction when it is determined the medical criteria or reimbursement guidelines noted below are met. Wording revised in the Not Covered section. “Not Medically Necessary” and “investigational” changed to read “Reimbursement is not allowed…” Deleted coding grid. Notification given 10/1/2019 for policy effective date 12/2/2019. (an)


Medical policy is not an authorization, certification, explanation of benefits or a contract. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the group contract and subscriber certificate that is in effect at the time services are rendered. This document is solely provided for informational purposes only and is based on research of current medical literature and review of common medical practices in the treatment and diagnosis of disease. Medical practices and knowledge are constantly changing and BCBSNC reserves the right to review and revise its medical policies periodically.