Cardiac Biomarkers for Myocardial Infarction

Initial Presentation Date: 7/01/2020
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Policy Description

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).

Related Policies

<table>
<thead>
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<th>Policy Number</th>
<th>Policy Title</th>
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<tbody>
<tr>
<td>APEA-G2050</td>
<td>Cardiovascular Disease Risk Assessment</td>
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</table>

Indications and/or Limitations of Coverage

Application of coverage criteria is dependent upon an individual’s benefit coverage at the time of the request

1. Measurement of cardiac troponin (troponin T or I) for the diagnosis of myocardial infarction (MI) MEETS COVERAGE CRITERIA in all patients presenting with signs and symptoms of acute coronary syndrome* (please see Note 1)

2. Measurement of following cardiac biomarkers for the diagnosis and/or prognosis of MI DOES NOT MEET COVERAGE CRITERIA 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
3. 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) DOES NOT MEET COVERAGE CRITERIA.

The following does not meet coverage criteria due to a lack of available published scientific literature confirming that the test(s) is/are required and beneficial for the diagnosis and treatment of a patient’s illness.

4. Measurement of the following cardiac biomarkers for the diagnosis and/or prognosis of MI DOES NOT MEET COVERAGE CRITERIA 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, 2020):

- 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, 2020):

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

Scientific Background

Acute coronary syndromes (ACS) represent continuous events starting with angina, reversible injury, progressing to unstable angina, are 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 an ST-segment elevation is observed on the ECG, it is 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. ACS are complex. However, the most common cause is atherosclerotic coronary artery disease with rupture of atherosclerotic plaque (Amsterdam et al., 2014). The first documented definition of acute MI was established in 1979 by the World Health Organization (WHO). It included in the criteria for MI diagnosis the recommendation to use the rise or fall patterns of cardiac biomarkers, such as creatine kinase (CK), creatine kinase’s MB isoenzyme (CK-MB), lactate dehydrogenase (LDH) or aspartate aminotransferase (AST) activities (WHO, 1979). Since then, other societies have 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 summarized in the table from (Danese & Montagnana, 2016):
Proprietary tests for various biomarkers are available in several clinical settings. Platforms including Roche’s “CARDIAC Trop T Sensitive test” and Responsebio’s battery of cardiac tests emphasize their speed (on the scale of minutes) and versatility (ResponseBio, 2020; Roche, 2020).

**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. Five isoenzymes have been identified by gel electrophoresis and other techniques (Marshall, Williams, & Williams, 1991). The heart isoenzymes, LD1 and LD2, have activity increases in blood 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 generally not recommended as a biomarker for the diagnosis of MI (Amsterdam et al., 2014; Jaffe & Morrow, 2017).

**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 a 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 myoglobin to be used in the diagnosis of MI (Amsterdam et al., 2014), and its use as cardiac biomarker is discouraged (Jaffe & Morrow, 2017).

**Creatine Kinase (CK) Isoenzymes and Isoform MB (CKMB)**
The cytosolic enzyme creatine kinase (CK), formerly known as creatine phosphokinase (Danese & Montagnana, 2016), 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, but 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). CKMB mass (CKMB protein concentration measurements) was once 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. 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; consequently, troponin is the recommended test for MI diagnosis now (Amsterdam et al., 2014; Jaffe & Morrow, 2017).

**Troponins**
The regulatory protein troponin in the troponin complex is composed of three isoforms. 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 because 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 is not expressed in normal, 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, but 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).

Neumann et al evaluated high-sensitivity troponin (troponin I and T)'s ability to predict myocardial infarction and subsequent 30-day outcomes. The authors developed a risk assessment tool based on patients presenting to the emergency department with “symptoms suggestive of myocardial infarction”. Concentrations of troponin I or T were measured at presentation and after early or late serial sampling. Cutoffs were then determined to create cutoffs for risk assessment. Among the 22651 patients (9604 in derivation cohort, 13047 in validation cohort), the total prevalence of myocardial infarction was 15.3%. The authors found that “lower high-sensitivity troponin concentrations at presentation and smaller absolute changes during serial sampling were associated with a lower likelihood of myocardial infarction and a lower short-term risk of cardiovascular events” (Neumann et al., 2019).

Anand et al evaluated the adoption rate of the universal definition of myocardial infarction and the corresponding recommendations. 1902 medical centers over 23 countries were surveyed, and the authors obtained answers regarding the primary biomarker, diagnostic thresholds, and clinical pathways used to identify myocardial infarction. The authors found that cardiac troponin was the primary biomarker used at 96% of surveyed sites, with 41% of these sites using high-sensitivity troponins. The sites using high-sensitivity assays were also more likely to use serial sampling (91% vs 78% using “contemporary” sensitivity troponin) and the 99% percentile diagnostic threshold (74% vs 66%). Use of creatine kinase-MB (CKMB) was “very limited” outside of Latin America (Anand, Shah, Beshiri, Jaffe, & Mills, 2019).

In addition, other cardiac biomarkers, such as heart-type fatty acid binding protein (H-FABP) and copeptin, have been reported in the scientific literature. However, they are not commonly used in clinical settings (Jaffe & Morrow, 2017).

**Heart-type fatty acid binding protein (H-FABP)**
H-FABP, a small cytoplasmic protein present in cardiomyocytes, 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 compared the use of H-FABP with rapid troponin in 371 patients with acute chest pain (Seino et al., 2003). Their study demonstrated that H-FABP had significantly higher sensitivity (89%) than troponin T (22%) and myoglobin (38%), but it has 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. H-FABP is not encouraged for assessment
of MI as troponins are generally superior (Jaffe & Morrow, 2017).

**Copeptin**
Copeptin is the 39 amino acid C-terminal fragment cleaved from 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, demonstrated no
benefit in using copeptin as a an early rule-out cardiac biomarker for MI (Hillinger et al., 2015).
Copeptin is not encouraged for assessment of MI as troponins are generally superior (Jaffe &

**Guidelines and Recommendations**

**2018 ESC/ACC/AHA/WHF Fourth Universal Definition of Myocardial Infarction (Jaffe et al., 2018)**
Both cTnI and cTnT are recommended for evaluation of myocardial injury, and high-sensitivity
cTn assays are recommended for routine clinical use. An acute MI is designated when a
rising/falling pattern is seen with cTn levels and if there is at least one measurement greater
than the 99th percentile of the upper reference limit (URL) (Jaffe et al., 2018).

CKMB is considered less sensitive and specific than either troponin. However, in the absence of
cTn assay, CK-MB is considered the best alternative. A measurement of CK-MB above the 99th
percentile of the URL should be “designated as the decision level for the diagnosis of MI”. Sex-
specific CK-MB values should be used (Jaffe et al., 2018).

In the 2019 AHA guideline discussing the “Contemporary Diagnosis and Management of
Patients With Myocardial Infarction in the Absence of Obstructive Coronary Artery Disease
[MINOCA]”, the AHA notes that the diagnostic criteria of MINOCA follows the “Fourth Universal
Definition of Myocardial Infarction” above, specifically the rise or fall of cardiac troponin levels
with at least one value above the 99th percentile of the reference limit. The guideline considers
this definition “fundamental” to identifying and defining MINOCA (Tamis-Holland Jacqueline et
al., 2019).

**2014 AHA/ACC Guideline for the Management of Patients with Non-ST-Elevation
Acute Coronary Syndromes (NSTE-ACS) (Amsterdam et al., 2014)**
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, the AHA/ACC panel
has 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 5](image)

**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 the 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

In their review of previously issued guidelines, the expert panel 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, nor did they gave any recommendations regarding CKMB (O’Connor Robert et al., 2015).

European Society of Cardiology (ESC, 2015)

The ESC notes measurement of cardiac troponins as “mandatory” in all patients with suspected non-ST-elevation acute coronary syndromes. The guidelines assert that cardiac troponins are more sensitive and specific biomarkers of cardiomyocyte injury than CK, CKMB, and myoglobin. However, if troponin measurement is not possible, measurement of copeptin is recommended.

The ESC also acknowledges the natriuretic peptides (B-type natriuretic peptide, N-terminal pro-B-type natriuretic peptide and midregional pro-A-type natriuretic peptide) as providing useful prognostic information along with the troponins. The ESC mentions other biomarkers such as midregional pro-adrenomedullin, growth differentiation factor 15 and copeptin, but they cannot recommend them at this time as their added value in risk assessment seems “marginal” (Gencer et al., 2016).

The 2019 ESC guidelines focusing on chronic coronary syndromes states that for “clinical suspicion of coronary artery disease instability...management should follow the Guidelines for ACS without persistent ST-segment elevation”, which is discussed above (Knuuti et al., 2019).


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 follow 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.

American Society for Clinical Pathology (ASCP, 2015)

The ASCP recommends against testing CK-MB or myoglobin to diagnose an acute myocardial infarction. Instead, they recommend testing either troponin I or T. They also assert that both troponins are specific to cardiac injury and that there is much support for relying solely on troponin (ASCP, 2015).

National Institute for Health and Care Excellence (NICE, 2016)

NICE recommends diagnosis of MI using the "detection of rise and/or fall of cardiac biomarkers values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile of the upper reference limit and at least one of the following:

- symptoms of ischaemia
- new or presumed new significant ST-segment-T wave (ST-T) changes or new left bundle branch block (LBBB)
- development of pathological Q waves in the ECG
- imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- identification of an intracoronary thrombus by angiography” (NICE, 2016).

State and Federal Regulations, as applicable

There is a multitude of FDA-approved cardiac biomarkers tests for Troponin and CKMB currently available in high and moderate complexity formats. Additionally, many labs have developed specific tests that they must validate and perform in house. These laboratory-developed tests (LDTs) are regulated by the Centers for Medicare and Medicaid (CMS) as high-complexity tests under the Clinical Laboratory Improvement Amendments of 1988 (CLIA ‘88). As an LDT, the U. S. Food and Drug Administration has not approved or cleared this test; however, FDA clearance or approval is not currently required for clinical use.

Applicable CPT/HCPCS Procedure Codes

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Procedure codes appearing in Medical Policy documents are included only as a general reference tool for each policy. They may not be all-inclusive.

Evidence-based Scientific References


Policy Implementation/Update Information
7/1/20 New Policy

State and Federal mandates and health plan contract language, including specific provisions/exclusions, take precedence over Medical Policy and must be considered first in determining eligibility for coverage. The medical policies contained herein are for informational purposes. The medical policies do not constitute medical advice or medical care. Treating health care providers are independent contractors and are neither employees nor agents Blue KC and are solely responsible for diagnosis, treatment and medical advice. No part of this publication may be reproduced, stored in a retrieval system or transmitted, in any form or by any means, electronic, photocopying, or otherwise, without permission from Blue KC.