Introduction

Hypertrophic cardiomyopathy is a condition where the muscle cells of the heart become big. This can make the walls of the heart thicker than normal, and because the walls surrounding the heart’s pumping chambers get thicker, the chambers become smaller than they should be. Hypertrophic cardiomyopathy is usually inherited, and is caused by changes to one or more of the person’s genes. The muscle problems eventually lead to enlargement of the heart and possible problems with the heart’s rhythm and valves. This policy discusses when genetic testing for this form of cardiomyopathy may be considered medically necessary.

Note: The Introduction section is for your general knowledge and is not to be taken as policy coverage criteria. The rest of the policy uses specific words and concepts familiar to medical professionals. It is intended for providers. A provider can be a person, such as a doctor, nurse, psychologist, or dentist. A provider also can be a place where medical care is given, like a hospital, clinic, or lab. This policy informs them about when a service may be covered.
<table>
<thead>
<tr>
<th>Testing</th>
<th>Medical Necessity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic testing for predisposition to HCM</td>
<td>Genetic testing for predisposition to HCM may be considered medically necessary for individuals who are at risk for developing HCM.</td>
</tr>
<tr>
<td></td>
<td>• “At risk” is defined as having a first-degree relative who has a confirmed diagnosis of HCM and the relative has a documented pathogenic gene variant. (See below)</td>
</tr>
<tr>
<td>Genetic testing for HCM gene variants</td>
<td>Genetic testing for HCM gene variants may be considered medically necessary for the index patient with confirmed clinical HCM, when used to assist unaffected first degree family members. (See Benefit Application section)</td>
</tr>
<tr>
<td>Genetic testing for predisposition to HCM</td>
<td>Genetic testing for predisposition to HCM is considered not medically necessary for patients with a family history of HCM in which a first-degree relative has tested negative for pathologic variants.</td>
</tr>
</tbody>
</table>

Due to the complexity of genetic testing for hypertrophic cardiomyopathy (HCM) and the potential for misinterpretation of results, the decision to test and the interpretation of test results should be performed by, or in consultation with, an expert in the area of medical genetics and/or HCM.

To inform and direct genetic testing for at-risk individuals, genetic testing should initially be performed in at least one close relative with a confirmed diagnosis of HCM (index case), if possible. See Practice Guidelines and Position Statements and Benefit Application section for information regarding testing of the index case.

Because there are varying degrees of penetrance for different HCM variants, consideration for testing of second- or third-degree relatives may be appropriate in certain circumstances. Some judgment should be allowed for these decisions, for example, in the case of a small family pedigree. Consultation with an expert in medical genetics and/or the genetics of HCM, in conjunction with a detailed pedigree analysis, is appropriate when testing of second- or third-degree relatives is considered.
<table>
<thead>
<tr>
<th>CPT</th>
<th>Description</th>
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<tr>
<td>81405</td>
<td>Molecular pathology procedure, Level 6 (e.g., analysis of 6-10 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 11-25 exons, regionally targeted cytogenomic array analysis). This code includes:</td>
</tr>
<tr>
<td></td>
<td>• ACTC1 (actin, alpha, cardiac muscle 1) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• MYL2 (myosin, light chain 2, regulatory, cardiac, slow) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• MYL3 (myosin, light chain 3, alkali, ventricular, skeletal, slow) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• TNNC1 (troponin C type 1 [slow]) (e.g., hypertrophic cardiomyopathy or dilated cardiomyopathy), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• TNNI3 (troponin I, type 3 [cardiac]) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• TPM1 (tropomyosin 1 [alpha]) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td>81406</td>
<td>Molecular pathology procedure, Level 7 (e.g., analysis of 11-25 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 26-50 exons, cytogenomic array analysis for neoplasia). This code includes:</td>
</tr>
<tr>
<td></td>
<td>• TNNT2 (troponin T, type 2 [cardiac]) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td>81407</td>
<td>Molecular pathology procedure, Level 8 (e.g., analysis of 26-50 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of &gt;50 exons, sequence analysis of multiple genes on one platform). This code includes:</td>
</tr>
<tr>
<td></td>
<td>• MYBPC3 (myosin binding protein C, cardiac) (e.g., familial HCM), full gene sequence</td>
</tr>
<tr>
<td></td>
<td>• MYH7 (myosin, heavy chain 7, cardiac muscle, beta) (e.g., familial HCM, Liang distal myopathy), full gene sequence</td>
</tr>
<tr>
<td>81439</td>
<td>Inherited cardiomyopathy (e.g., hypertrophic cardiomyopathy, dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) genomic sequence analysis panel, must include sequencing of at least 5 genes, including DSG2, MYBPC3, MYH7, PKP2, and TTN (new code effective 1/1/17)</td>
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</table>

<table>
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<tr>
<th>HCPCS</th>
<th>Description</th>
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</thead>
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<tr>
<td>S3865</td>
<td>Comprehensive gene sequence analysis for hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>S3866</td>
<td>Genetic analysis for a specific gene mutation for hypertrophic cardiomyopathy (HCM) in an individual with a known HCM mutation in the family</td>
</tr>
</tbody>
</table>

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Genetic Counseling

Genetic counseling is primarily aimed at patients who are at risk for inherited disorders, and experts recommend formal genetic counseling in most cases when genetic testing for an inherited condition is considered. The interpretation of the results of genetic tests and the understanding of risk factors can be very difficult and complex. Therefore, genetic counseling will assist individuals in understanding the possible benefits and harms of genetic testing, including the possible impact of the information on the individual’s family. Genetic counseling may alter the utilization of genetic testing substantially and may reduce inappropriate testing. Genetic counseling should be performed by an individual with experience and expertise in genetic medicine and genetic testing methods.

Benefit Application

Some Plans may have contract or benefit exclusions for genetic testing.

Local plan information:

Professional society recommendations indicate that, when possible, the family member who has been diagnosed with HCM undergo genetic testing for hypertrophic cardiomyopathy (HCM). This will allow unaffected, at-risk family members to undergo genetic testing for the specific HCM variant found in the index case. This testing is intended to document whether a known pathologic variant is present in the family and optimize the predictive value of predisposition testing for at-risk relatives.

However, coverage for testing of the affected index case is dependent on contract benefit language when there is no conclusive evidence of clinical benefit to the index case from testing.

Specific contract language must be reviewed and considered when determining coverage for testing. In some cases, coverage for testing the index case may be available through the contract that covers the unaffected, at-risk individual who will benefit from knowing the results of the genetic test.

Evidence Review
Description

Familial hypertrophic cardiomyopathy (HCM) is an inherited condition that is caused by a disease-associated variant in one or more of the cardiac sarcomere genes. HCM is associated with numerous cardiac abnormalities, the most serious of which is sudden cardiac death. Genetic testing for HCM-associated variants is currently available through a number of commercial laboratories.

Background

**Familial Hypertrophic Cardiomyopathy (HCM)**

Familial hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular condition, with a phenotypic prevalence of approximately 1 in 500 adults (0.2%).

It is the most common cause of sudden cardiac death (SCD) in adults younger than 35 years of age and is probably also the most common cause of death in young athletes. The overall death rate for patients with HCM is estimated to be 1% per year in the adult population.

The genetic variant associated with HCM causes a defect in the cardiac sarcomere, which is the basic contractile unit of cardiac myocytes and is composed of a number of different protein structures. Nearly 1,400 individual variants in at least 18 different genes have been identified to date. Approximately 90% of pathogenic variants are missense (ie, one amino acid is replaced for another), and the strongest evidence for pathogenicity is available for 11 genes coding for thick filament proteins (MYH7, MYL2, MYL3), thin filament proteins (TNNT2, TNNI3, TNNC1, TPM1, ACTC), intermediate filament proteins (MYBPC3), and the Z-disc adjoining the sarcomere (ACTN2, MYOZ2). Variants in myosin heavy chain (MYH7) and myosin-binding protein C (MYBPC3) are the most common and account for roughly 80% of sarcomeric HCM. These genetic defects are inherited in an autosomal dominant pattern with rare exceptions. Genetic abnormalities can be identified in approximately 60% of patients with clinically documented HCM. Most of these patients have a familial pattern, although some exceptions are found presumably due to de novo variants.

Diagnosis

The clinical diagnosis of HCM depends on the presence of left ventricular hypertrophy (LVH, or thickening of the walls of the ventricle). This may be measured by echocardiography or magnetic resonance imaging, in the absence of other known causative factors such as valvular
disease, long-standing hypertension, or other myocardial disease. In addition to primary cardiac disorders, there are systemic diseases that can lead to LVH and thus “mimic” HCM. These include infiltrative diseases such as amyloidosis, glycogen storage diseases (eg, Fabry disease, Pompe disease), and neuromuscular disorders (eg, Noonan syndrome, Friedreich ataxia). These disorders need to be excluded before a diagnosis of familial HCM is made.

HCM is a very heterogeneous disorder. Manifestations range from subclinical, asymptomatic disease to severe life-threatening disease. Wide phenotypic variability exists among individuals, even when an identical variant is present, including among affected family members. This variability in clinical expression may be related to environmental factors and modifier genes. A large percentage of patients with HCM, perhaps the majority of all HCM patients, are asymptomatic or have minimal symptoms. These patients do not require treatment and are not generally at high risk for SCD. A subset of patients has severe disease that causes a major impact on quality of life and life expectancy. Severe disease can lead to disabling symptoms, as well as complications of HCM, including heart failure and malignant ventricular arrhythmias. Symptoms and presentation may include SCD due to unpredictable ventricular tachyarrhythmias, heart failure, or atrial fibrillation, or some combination. Management of patients with HCM involves treating cardiac comorbidities, avoiding therapies that may worsen obstructive symptoms, treating obstructive symptoms with β-blockers, calcium channel blockers, and (if symptoms persist) invasive therapy with surgical myectomy or alcohol ablation, optimizing treatment for heart failure, if present, and SCD risk stratification. ICD implantation may be indicated if there is a family history of SCD.

Diagnostic screening of first-degree relatives and other family members is an important component of HCM management. Guidelines have been established for screening in clinically unaffected relatives of affected individuals. Screening with physical examination, electrocardiography, and echocardiography is recommended every 12 to 18 months for individuals age 12 to 18 years and every 3 to 5 years for adults. Additional screening is recommended for any change in symptoms that might indicate the development of HCM.

**Genetic Testing**

Genetic testing has been proposed as a component of screening at-risk individuals to determine their predisposition to HCM. Patients at risk for HCM are defined as individuals who have a close family member with established HCM. Results of genetic testing may influence management of at-risk individuals, which may in turn lead to improved outcomes. Furthermore, results of genetic testing may have implications for decision making in the areas of reproduction, employment, and leisure activities.
Commercial testing has been available since May 2003, and numerous companies currently offer genetic testing for HCM. Testing is performed either as comprehensive testing or targeted gene testing. Comprehensive testing, which is done for an individual without a known genetic variant in the family, analyzes the genes that are most commonly associated with genetic variants for HCM and evaluates whether any potentially pathogenic variants are present. Some available panels include testing for multisystem storage diseases that may include cardiac hypertrophy, such as Fabry disease (GLA), familial transthyretin amyloidosis (TTR), X-linked Danon disease (LAMP2).

Other panels include testing for genes related to HCM but also those associated with other cardiac disorders. For example, the Comprehensive Cardiomyopathy panel (ApolloGen, Irvine, CA) is an next-generation sequencing (NGS) panel of 44 genes associated with HCM, dilated cardiomyopathy, restrictive cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia, left ventricular noncompaction syndrome, Danon syndrome, Fabry disease, Barth syndrome, and transthyretin amyloidosis.

For a patient with a known variant in the family, targeted testing is performed. Targeted variant testing evaluates for the presence or absence of a single variant known to exist in a close relative.

It can be difficult to determine the pathogenicity of genetic variants associated with HCM. Some studies have reported that assignment of pathogenicity has a relatively high error rate and that classification changes over time. With NGS and whole-exome sequencing techniques, the sensitivity of identifying variants on the specified genes has increased substantially. At the same time, the number of variants of uncertain significance is also increased with NGS. In addition, the percentage of individuals who have more than 1 variant that is thought to be pathogenic is increasing. A 2013 study reported that 9.5% (19/200) patients from China with HCM had multiple pathogenic variants and that the number of variants correlated with severity of disease.

Summary of Evidence

For individuals who are asymptomatic with risk for hypertrophic cardiomyopathy (HCM) because of a positive family history who receive testing for a specific HCM-related variant identified in affected family member(s), the evidence includes studies reporting on the analytic and clinical validity of testing. Relevant outcomes are overall survival, test accuracy and validity, changes in reproductive decision making, symptoms, and morbid events. For individuals at risk for HCM (first-degree relatives), genetic testing is most useful when there is a known disease-associated
variant in the family. In this situation, genetic testing will establish the presence or absence of
the same variant in a close relative with a high degree of certainty. Absence of this variant will
establish that the individual has not inherited the familial predisposition to HCM and thus has a
similar risk of developing HCM as the general population. Such patients will no longer need
ongoing surveillance for the presence of clinical signs of HCM. Although no direct evidence
comparing outcomes for at-risk individuals managed with and without genetic testing was
identified, there is a strong chain of evidence that management changes can improve outcomes
with genetic testing when there is a known familial variant. The evidence is sufficient to
determine that the technology results in a meaningful improvement in the net health outcome.

For individuals who are asymptomatic with risk for HCM because of a positive family history who
receive nonspecific testing for a HCM-related variant, the evidence includes studies reporting on
the analytic and clinical validity of testing. Relevant outcomes are overall survival, test accuracy
and validity, changes in reproductive decision making, symptoms, and morbid events. Given the
wide genetic variation in HCM and the likelihood that not all causative variants have been
identified, there is imperfect clinical sensitivity. Therefore, a negative test is not sufficient to rule
out a disease-associated variant in patients without a known family variant. For at-risk
individuals without a known variant in the family, there is no clear relation between testing and
improved outcomes. The evidence is insufficient to determine the effects of the technology on
health outcomes.

Ongoing and Unpublished Clinical Trials

Some trials that might influence this review are listed below.

Table 1. Summary of Key Trials

<table>
<thead>
<tr>
<th>NCT No.</th>
<th>Trial Name</th>
<th>Planned Enrollment</th>
<th>Completion Date</th>
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<tr>
<td>NCT01915615</td>
<td>HCMR - Novel Markers of Prognosis in Hypertrophic Cardiomyopathy</td>
<td>2750</td>
<td>Nov 2018</td>
</tr>
<tr>
<td>NCT00156429</td>
<td>Genetic Predictors of Outcome in HCM Patients</td>
<td>540</td>
<td>May 2020</td>
</tr>
</tbody>
</table>

NCT: national clinical trial.
Clinical Input Received From Physician Specialty Societies and Academic Medical Centers

While the various physician specialty societies and academic medical centers may provide appropriate reviewers who collaborate with and make recommendations during this process, input received does not represent an endorsement or position statement by the physician specialty societies or academic medical centers, unless otherwise noted.

Clinical input was solicited in January 2011 regarding general agreement with the policy. This was followed up by a second round of focused clinical vetting in October 2011 to address specific questions raised after the first round of vetting. The initial vetting indicated uniform agreement with the medically necessary indication for individuals with a first-degree relative who has a known pathologic mutation. This vetting also asked whether testing should be restricted to first-degree relatives. For this question, there was a mixed response, with two reviewers indicating that they agree with testing only first-degree relatives, two reviewers indicating that testing should be offered to non-first-degree relatives, and one reviewer who was unsure.

The second round of clinical vetting focused on the changes in management that could result from genetic testing. Reviewers were uniform in responding that a positive test will result in heightened surveillance. All but one reviewer indicated that a negative test will eliminate the need for future surveillance in all cases. There was general agreement that the surveillance schedule used in clinical practice was that proposed by Maron et al.\textsuperscript{11}

Practice Guidelines and Position Statements

*European Society of Cardiology (ESC)*

In 2014, the European Society of Cardiology issued guidelines on the diagnosis and management of hypertrophic cardiomyopathy (HCM), which included the following recommendations related to genetic testing\textsuperscript{36}

Class I Recommendations

- Genetic counseling is recommended for all patients with HCM when their disease cannot be explained solely by a non-genetic cause, whether or not clinical or genetic testing will be used to screen family members. (Level of Evidence: B)
Genetic testing is recommended in patients fulfilling diagnostic criteria for HCM, when it enables cascade genetic screening of their relatives. (Level of Evidence: B)

It is recommended that genetic testing be performed in certified diagnostic laboratories with expertise in the interpretation of cardiomyopathy-related variants. (Level of Evidence: C).

In the presence of symptoms and signs of disease suggestive of specific causes of HCM, genetic testing is recommended to confirm the diagnosis. (Level of Evidence: B)

Cascade genetic screening, after pre-test counseling, is recommended in first-degree adult relatives of patients with a definite disease-causing mutation. (Level of Evidence: B)

Clinical evaluation, employing ECG and echocardiography and long-term follow-up, is recommended in first-degree relatives who have the same definite disease-causing mutation as the proband (index patient). (Level of Evidence: C)

**Class IIa Recommendations**

- Genetic counselling should be performed by professionals trained for this specific task working within a multidisciplinary specialist team. (Level of Evidence: C)

- Genetic testing in patients with a borderline diagnosis of HCM should be performed only after detailed assessment by specialist teams. (Level of Evidence: C)

- Post-mortem genetic analysis of stored tissue or DNA should be considered in deceased patients with pathologically confirmed HCM, to enable cascade genetic screening of their relatives. (Level of Evidence: C)

- First-degree relatives who do not have the same definite disease-causing mutation as the proband (index patient) should be discharged from further follow-up but advised to seek reassessment if they develop symptoms or when new clinically relevant data emerge in the family. (Level of Evidence: B)

- When no definite genetic mutation is identified in the proband (index patient) or genetic testing is not performed, clinical evaluation with ECG and echocardiography should be considered in first-degree adult relatives and repeated every 2–5 years (or 6–12 monthly if non-diagnostic abnormalities are present). (Level of Evidence: C)

- The children of patients with a definite disease-causing mutation should be considered for predictive genetic testing—following pre-test family counseling—when they are aged 10 or
more years and this should be carried out in accordance with international guidelines for genetic testing in children. (Level of Evidence: C)

- In first-degree child relatives aged 10 or more years, in whom the genetic status is unknown, clinical assessment with ECG and echocardiography should be considered every 1–2 years between 10 and 20 years of age, and then every 2–5 years thereafter. (Level of Evidence: C)

Class IIb Recommendations

- If requested by the parent(s) or legal representative(s), clinical assessment with ECG and echocardiography may precede or be substituted for genetic evaluation after counselling by experienced physicians and when it is agreed to be in the best interests of the child. (Level of Evidence: C)

- When there is a malignant family history in childhood or early-onset disease or when children have cardiac symptoms or are involved in particularly demanding physical activity, clinical or genetic testing of first-degree child relatives before the age of 10 years may be considered. (Level of Evidence: C)

- In definite mutation carriers who have no evidence of disease expression, sports activity may be allowed after taking into account the underlying mutation and the type of sport activity, and the results of regular and repeated cardiac examinations. (Level of Evidence: C)

American College of Cardiology (ACC) Foundation and the American Heart Association (AHA)

The American College of Cardiology Foundation and the American Heart Association issued joint guidelines on the diagnosis and treatment of HCM in 2011.12

Class I Recommendations

- Evaluation of familial inheritance and genetic counseling is recommended as part of the assessment of patients with HCM. (Level of Evidence: B)

- Patients who undergo genetic testing should also undergo counseling by someone knowledgeable in the genetics of cardiovascular disease so that results and their clinical significance can be appropriately reviewed with the patient. (Level of Evidence: B)
• Screening (clinical, with or without genetic testing) is recommended in first-degree relatives of patients with HCM. (Level of Evidence: B)

• Genetic testing for HCM and other genetic causes of unexplained cardiac hypertrophy is recommended in patients with an atypical clinical presentation of HCM or when another genetic condition is suspected to be the cause. (Level of Evidence: B)

**Class IIa Recommendations**

• Genetic testing is reasonable in the index patient to facilitate the identification of first-degree family members at risk for developing HCM (Level of Evidence: B).

**Class IIb Recommendations**

• The usefulness of genetic testing in the assessment of risk of SCD in HCM is uncertain (Level of Evidence: B).

**Class III indications: No Benefit**

• Genetic testing is not indicated in relatives when the index patient does not have a definitive pathogenic mutation (Level of Evidence: B).

• Ongoing clinical screening is not indicated in genotype-negative relatives in families with HCM (Level of Evidence: B).

*Heart Rhythm Society (HRS) and European Heart Rhythm Association (EHRA)*

The HRS and the EHRA published recommendations for genetic testing for cardiac channelopathies and cardiomyopathies in 2011. For hypertrophic cardiomyopathy, the following recommendations were made:

• Comprehensive or targeted HCM genetic testing is recommended for any patient in whom a cardiologist has established a clinical diagnosis of HCM based on examination of the
patient’s clinical history, family history, and electrocardiographic/echocardiographic phenotype

- Mutation-specific testing is recommended for family members and appropriate relatives following the identification of the HCM-causative mutation in an index case.

**Medicare National Coverage**

There is no national coverage determination (NCD). In the absence of an NCD, coverage decisions are left to the discretion of local Medicare carriers.

**Regulatory Status**

Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; laboratory-developed tests (LDTs) must meet the general regulatory standards of the Clinical Laboratory Improvement Act (CLIA). Sequencing tests for hypertrophic cardiomyopathy (HCM) are available under the auspices of CLIA. Laboratories that offer LDTs must be licensed by CLIA for high-complexity testing.

To date, the U.S. Food and Drug Administration (FDA) has chosen not to require any regulatory review of this test.

There are no assay kits approved by FDA for genetic testing for HCM.

**References**


37. Ackerman MJ, Priori SG, Willems S, et al. HRS/EHRA expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies this document was developed as a partnership between the Heart Rhythm Society (HRS) and the European Heart Rhythm Association (EHRA). Heart Rhythm. Aug 2011;8(8):1308-1339. PMID 21787999
<table>
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<th>Date</th>
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<td>02/11/13</td>
<td>Replace policy. Policy updated with literature search through October 2012. References 6, 26, and 27. No change to policy statement. CPT codes 83890 – 83913 deleted as of 12/31/12; CPT codes 81405 - 81407 and 81599, effective 1/1/13, are added to the policy.</td>
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<td>05/14/13</td>
<td>Update Related Policies. Add 12.04.91.</td>
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<tr>
<td>02/24/14</td>
<td>Replace policy. Policy update with literature search through October 31, 2013. References 15-17 and 30-33 added. The policy statements are unchanged. Deleted CPT codes 83890 – 83912 and 81599 (no specific to this testing) removed from the policy; ICD-9 Diagnosis codes are not used to adjudicate the policy and have been removed.</td>
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<td>03/21/14</td>
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<td>02/25/15</td>
<td>Annual Review. Policy updated with literature review through October 29, 2014. Added list of commercially available testing companies to Policy Guidelines from the Description section found in Table 1. Added TNNC1 (troponin C type 1 [slow]) full gene sequence to CPT 81405 description in Policy Guidelines. Benefit Application section rewritten to match the plan variance. Policy Statement for testing of the index patient as medically necessary. References 11, 28-29, 32-33, 36-37, 41, and 43 added, broken hyperlinks repaired; others renumbered/removed. Policy statements unchanged.</td>
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<td>02/09/16</td>
<td>Annual Review. Genetic Counseling information added to Guidelines section. Policy updated with literature review; references added. Policy statements unchanged.</td>
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<tr>
<td>01/01/17</td>
<td>Coding update; added new CPT code 81439 effective 1/1/17. Reformatted coding table, removed duplicate coding table near end of policy. Other minor formatting corrections were made.</td>
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  - Written information in other formats (large print, audio, accessible electronic formats, other formats)
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  - Qualified interpreters
  - Information written in other languages

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Email AppealsDepartmentinquines@Premera.com

You can file a grievance in person or by mail, fax, or email. If you need help filing a grievance, the Civil Rights Coordinator is available to help you.

You can also file a civil rights complaint with the U.S. Department of Health and Human Services, Office for Civil Rights, electronically through the Office for Civil Rights Complaint Portal, available at https://ocrportal.hhs.gov/ocr/portal/lobby.jsf, or by mail or phone at:

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Oromo (Cushite):

Français (French):

Kreyòl ayisyen (Creole):
Avi sila a gen Enfòmasyon Empòtan ladan. Avis sila a kapab genyen enfòmasyon empòtan konsènan aplikayon w la osa konseán kouvèti asirans lan atravè Premera Blue Cross. Kapab genyen dat ki enpòtan nan avis sila a. Ou ka gen pou tran kék aksyon avan sèten dat limit pou ka tenbe kouvèti asirans sante w la osa pou yo ka ede w avèk depans yo. Se dwa w pou resewa enfòmasyon sa a ak asistans nan lang ou pale a, san ou pa gen pou peyey pou sa. Rate nan 800-722-1471 (TTY: 800-842-5357).

Deutsche (German):

Hmoob (Hmong):

Ilokto (Ilocano):
Daytoy a Pakdaar ket naglao iti Napateg nga Impomarson. Daytoy a pakdaar mabalbin nga adda ket naglao iti napateg nga impomarson maipangepp ngi aplikayonu yenno coverage babaen iti Premera Blue Cross. Daytoy ket mabalbin dagiti importante a pelta iti daytoy a pakdaar. Mabalbin nga adda rumbeng nga aramidenyo nga addang sakbay dagiti partikular a naituding nga aldaw tapno mapogalataydo a coverage ti salun-atyo yenno tulong kadaygig gastos. Adda karbenganyo a mangala iti daytoy nga impomarson ken tulong iti bukodyo a pagasasao nga awan ti bayadanyo. Tumawag ti numero nga 800-722-1471 (TTY: 800-842-5357).

Italiano (Italian):

中文 (Chinese):
通知有重要的信息。本通知可能有关於您透过 Premera Blue Cross 提交的申请或保险的重要讯息。本通知可能有关於重要日期。您可能需要在截止日期之前採取行动，以保留您的健康保障或者費用補貼。您有權利免費以您的母語得到本訊息和幫助。請接電話 800-722-1471 (TTY: 800-842-5357).

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