

Policy: MP304

Section: Medical Benefit Policy

Subject: Genetic Testing for Inherited Cardiomyopathies and Channelopathies

Applicable Lines of Business

Commercial	X	CHIP	X
Medicare	X	ACA	X
Medicaid	X		

I. Policy: Genetic Testing for Inherited Cardiomyopathies and Channelopathies

II. Purpose/Objective:

To provide a policy of coverage regarding Genetic Testing for Inherited Cardiomyopathies and Channelopathies

III. Responsibility:

- A. Medical Directors
- B. Medical Management

IV. Required Definitions

1. Attachment – a supporting document that is developed and maintained by the policy writer or department requiring/authoring the policy.
2. Exhibit – a supporting document developed and maintained in a department other than the department requiring/authoring the policy.
3. Devised – the date the policy was implemented.
4. Revised – the date of every revision to the policy, including typographical and grammatical changes.
5. Reviewed – the date documenting the annual review if the policy has no revisions necessary.

V. Additional Definitions

Medical Necessity or Medically Necessary means Covered Services rendered by a Health Care Provider that the Plan determines are:

- a. appropriate for the symptoms and diagnosis or treatment of the Member's condition, illness, disease or injury;
- b. provided for the diagnosis, and the direct care and treatment of the Member's condition, illness disease or injury;
- c. in accordance with current standards of good medical treatment practiced by the general medical community.
- d. not primarily for the convenience of the Member, or the Member's Health Care Provider; and
- e. the most appropriate source or level of service that can safely be provided to the Member. When applied to hospitalization, this further means that the Member requires acute care as an inpatient due to the nature of the services rendered or the Member's condition, and the Member cannot receive safe or adequate care as an outpatient.

Medicaid Business Segment

Medically Necessary — A service, item, procedure, or level of care that is necessary for the proper treatment or management of an illness, injury, or disability is one that:

- Will, or is reasonably expected to, prevent the onset of an illness, condition, injury or disability.
- Will, or is reasonably expected to, reduce or ameliorate the physical, mental or developmental effects of an illness, condition, injury or disability.
- Will assist the Member to achieve or maintain maximum functional capacity in performing daily activities, taking

into account both the functional capacity of the Member and those functional capacities that are appropriate for Members of the same age

DESCRIPTION:

Risk for structural heart disease and arrhythmia can run in families. Cardiomyopathies are diseases of the heart muscle and include hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy, restrictive cardiomyopathy (RCM), left ventricular noncompaction (LVNC), arrhythmogenic right ventricular cardiomyopathy (ARVC). HCM is a common genetic heart disease reported in pan-ethnic populations. Among patients with HCM, 30% to 60% have a genetic etiology. There are 8 genes associated with HCM with a strong degree of evidence supporting association. Around 30% of patients with DCM are thought to have a hereditary cause for disease. 10-60% of cases with restrictive cardiomyopathy will have an underlying genetic etiology, and the genes responsible for this condition share significant overlap with HCM and DCM. ARVC and CPVT, while rare, have a high likelihood of a genetic etiology. Among patients with ARVC and CPVT, current evidence suggests up to 66% and 75% of cases, respectively, will have an identifiable genetic variant

Cardiac channelopathies, also called arrhythmias, are disorders involving cardiac cells membranes that allow passage of specific ions. These pathways regulate the flow of ions through the cells and are necessary to conduct electrical impulses across the heart. Cardiac channelopathies include long QT syndrome (LQTS), Brugada syndrome (BrS) (also referred to as sudden unexpected nocturnal death syndrome), short QT syndrome (SQTS) and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT). Cardiac channelopathies are characterized by delayed repolarization of the myocardium and QT interval alteration, resulting in increased risk for syncope, seizures, and sudden cardiac death (SCD) in the setting of a structurally normal heart and otherwise healthy individual.

Among patients with long QT syndrome 75-80% have a genetic etiology. In short QT syndrome, up to 20% have a genetic etiology. Brugada syndrome can be caused by pathogenic or likely pathogenic variants in SCN5A in 30% of cases, and there are at least 40 other genes associated with the disease that account for <1% of causes. CPVT is rare, and there are two known genes involving this syndrome: RYR2, CASQ2.

Multi-gene panel testing is the most cost-effective and accurate approach to characterize familial cardiomyopathy and channelopathies (arrhythmia) because there is considerable phenotypic overlap among these disorders. Therapeutic interventions may be tailored based on genetic findings. The value of genetic testing among individuals with risk for cardiomyopathy is threefold: to understand disease prognosis, to facilitate identification and subsequent screening recommendations for at-risk relatives, and to guide therapeutic options (eg: transthyretin amyloidosis, experimental treatments, need for ICD placement).

One new study of >700 patients with TTN variants, Mukherjee (2024) notes that following a negative baseline screening, 11% developed DCM during a median follow up of 11 years.

Disease-specific panels may change from year to year based on available evidence and technological advancements. Test methodology should include sequencing and full deletion and duplication analysis (i.e., detection of large genomic rearrangements) with a benefit of once per lifetime, dependent upon advances in testing technology.

INDICATIONS:

When ordered by a cardiologist/electrophysiologist, medical geneticist, or board-certified and licensed (where required) genetic counselor, the following tests including multigene panels are considered to be medically necessary in ANY of the following scenarios:

Channelopathies (Arrhythmias)

The member has a personal history of ANY of the following clinical features or diagnoses:

One or more signs or symptoms of LQTS, SQTS, CPVT, or Brugada, AND a definitive diagnosis cannot be made without genetic testing:

1. Prolonged QT/QTc interval on resting electrocardiogram of >470 msec in males or >480 msec in females without additional risk factors (eg: heart failure, bradycardia, electrolyte imbalance, recent history of QT prolonging drugs); or
1. Short QT/QTc interval of <350 msec; or
2. EKG pattern suggestive of Brugada with or without additional symptoms; or
3. Schwartz score of 2-3 or greater+ (see below); or
4. History of aborted sudden cardiac arrest.
5. History of isolated cardiac conduction disease or with concomitant structural heart disease or extracardiac disease when there is an early age of dx or a suspicion of laminopathy.

The member has a family history of ANY of the following:

1. One first- or second- degree relative with a known history of sudden cardiac arrest or death
2. Two or more relatives on the same side of the family with history sudden cardiac arrest or death
3. A first, second, or third degree relative with a known mutation in an LQTS, SQTS, CPVT, Brugada, or channelopathy gene for which medical management may impact the member's ongoing cardiac surveillance.
4. Two or more first, second, or third degree relatives with a reported clinical history of LQTS, SQTS, CPVT, Brugada, or hereditary arrhythmia but genetic testing may have not been completed or the report is unavailable to direct testing.

Cardiomyopathies:

The member has a personal history of ANY of the following clinical features or diagnoses:

1. Hypertrophic Cardiomyopathy (HCM)
2. Dilated Cardiomyopathy (DCM)
3. Restrictive Cardiomyopathy (RCM)
4. Arrhythmogenic cardiomyopathy (ACM) not secondary to ischemic, hypertensive, or valvular heart disease, such as arrhythmogenic right/left ventricular cardiomyopathy (ARVC/ALVC), cardiac amyloidosis, sarcoidosis, and left ventricular noncompaction (LVNC).
5. Member has sub-clinical signs suggestive of HCM, DCM, RCM, or ACM AND a first degree or second-degree relative with a cardiomyopathy.

The member has a family history of ANY of the following diagnoses:

1. One first- or second- degree relative with a known history of sudden cardiac arrest or death <40y; or
2. Two or more relatives on the same side of the family with history sudden cardiac arrest or death at any age; OR
3. A first, second, or third degree relative with a known mutation in a cardiomyopathy gene for which medical management may impact the member's cardiac surveillance strategy.
4. Two or more first, second, or third degree relatives with a reported cardiomyopathy but genetic testing may have not been completed or the report is unavailable to direct testing.

***Swartz Score Calculator for Clinical Diagnosis of Long QT Syndrome**

Findings		Points
	QTc ² ≥480 ms	3
	QTc ² =460-479 ms	2
	QTc ² =450-459 ms (in males)	1
ECG ¹	≥480 ms during 4 th minute of recovery from exercise stress test	1
	Torsade de pointes	2
	T wave alternans	1
	Notched T wave in 3 leads	1
	Low heart rate for age	0.5
Clinical history	Syncope ³ With stress	2
	Without stress	1
Family history	Family member(s) with definite LQTS	1
	Unexplained sudden cardiac death at age <30 years in immediate family	<u>0.5</u>
Total score		

Adapted from: Schwartz PJ, Crotti L. QTc behavior during exercise and genetic testing for the long-QT syndrome. Circulation. 2011;124:2181-4

Scoring:

- ≤1.0 point = low probability of LQTS
- 1.5-3.0 points = intermediate probability of LQTS
- ≥3.5 points = high probability of LQTS

EXCLUSIONS:

Per MoDx A54685, Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/C) genetic testing (CPT Code 81439) may be performed in panels of 5-7 of these genes and disease-causing mutation is expected to be identified in 42-55% of cases. Testing would be performed to confirm an established diagnosis or on individuals already diagnosed with

ARVD/C to identify family members at risk. Therefore, MoIDX has determined that testing for ARVD/C is a statutorily excluded test for the Medicare/Medicaid population.

Genetic testing for inherited cardiomyopathies and channelopathies not meeting the criteria described above is considered to be experimental, investigational or unproven and therefore **NOT COVERED**. There is insufficient evidence in the published, peer-reviewed medical literature to support the use of this testing outside of the indications listed above.

Medicaid Business Segment:

Any requests for services that do not meet criteria set in the PARP may be evaluated on a case by case basis.

CODING ASSOCIATED WITH:

The coding listed in this document may not represent the comprehensive range of codes that may be associated with this service.

- 81161 – DMD (dystrophin) (eg, Duchenne/Becker muscular dystrophy) deletion analysis and duplication analysis, if performed
- 81170 – ABL1 (ABL proto-oncogene 1, non-receptor tyrosine kinase)(eg, acquired imatinib tyrosine kinase inhibitor resistance), gene analysis, variants in the kinase domain
- 81280 Long QT Syndrome gene analysis (eg, KCNQ1, KCNH2, SCN5A, KCNE1, KCNE2, KCNJ2, CACNA1C, CAV3, SCN4B, AKAP, SNTA1, and ANK2); full sequence analysis
- 81281 Long QT Syndrome gene analysis (eg, KCNQ1, KCNH2, SCN5A, KCNE1, KCNE2, KCNJ2, CACNA1C, CAV3, SCN4B, AKAP, SNTA1, and ANK2); known familial sequence variant
- 81282 Long QT Syndrome gene analysis (eg, KCNQ1, KCNH2, SCN5A, KCNE1, KCNE2, KCNJ2, CACNA1C, CAV3, SCN4B, AKAP, SNTA1, and ANK2); duplication / deletion variants
- 81400 – Molecular pathology procedure, Level 1 (eg, identification of single germline variant [eg, SNP] by techniques such as restriction enzyme digestion or melt curve analysis)
- 81401 - Molecular pathology procedure, Level 2 (eg, 2-10 SNP's, 1 methylated variant, or 1 somatic variant [typically using nonsequencing target variant analysis], or detection of a dynamic mutation disorder/triplet repeat)
- 81403 - Molecular pathology procedure, Level 4 (eg, analysis of single exon by DNA sequence analysis, analysis of >10 amplicons using multiplex PCR in 2 or more independent reactions, mutation scanning or duplication/deletion variants of 2-5 exons)
- 81404 - Molecular pathology procedure, Level 5 (eg, analysis of 2-5 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 6-10 exons, or characterization of a dynamic mutation disorder/triplet repeat by Southern blot analysis)
- 81405 - Molecular pathology procedure, Level 6 (eg, analysis of 6-10 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 11-25 exons, regionally targeted cytogenomic array analysis)
- 81406 - Molecular pathology procedure, Level 7 (eg, analysis of 11-25 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 26-50 exons)
- 81407 - Molecular pathology procedure, Level 8 (eg, analysis of 26-50 exons in a single gene by DNA sequence analysis, mutation scanning or duplication/deletion variants of >50 exons, sequence analysis of multiple genes on one platform)
- 81408 - Molecular pathology procedure, Level 9 (eg, analysis of >50 exons in a single gene by DNA sequence analysis)
- 81413 (Cardiac ion channelopathies (eg, Brugada syndrome, long QT syndrome, short QT syndrome, catecholaminergic polymorphic ventricular tachycardia); genomic sequence analysis panel, must include sequencing of at least 10 genes, including ANK2, CASQ2, CAV3, KCNE1, KCNE2, KCNH2, KCNJ2, KCNQ1, RYR2, and SCN5A)
- 81414 (Cardiac ion channelopathies (eg, Brugada syndrome, long QT syndrome, short QT syndrome, catecholaminergic polymorphic ventricular tachycardia); duplication/deletion gene analysis panel, must include analysis of at least 2 genes, including KCNH2 and KCNQ1)
- 81439 Hereditary cardiomyopathy (eg, hypertrophic cardiomyopathy, dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy), genomic sequence analysis panel, must include sequencing of at least 5 cardiomyopathy-related genes
- 0231U CACNA1A (calcium voltage-gated channel subunit alpha 1A) (eg, spinocerebellar ataxia), full gene analysis, including small sequence changes in exonic and intronic regions, deletions, duplications, short tandem repeat (STR) gene expansions, mobile element insertions, and variants in non-uniquely mappable regions
- 0237U Cardiac ion channelopathies (eg, Brugada syndrome, long QT syndrome, short QT syndrome, catecholaminergic polymorphic ventricular tachycardia), genomic sequence analysis panel including ANK2, CASQ2, CAV3, KCNE1, KCNE2, KCNH2, KCNJ2, KCNQ1, RYR2, and SCN5A, including small sequence changes in exonic and intronic regions, deletions, duplications, mobile element insertions, and variants in non-uniquely mappable regions

LINE OF BUSINESS:

Eligibility and contract specific benefit limitations and/or exclusions will apply. Coverage statements found in the line of business specific benefit document will supercede this policy.

REFERENCES:

- Kapplinger JD, Tester DJ, Alders M, et al. An international compendium of mutations in the SCN5A-encoded cardiac sodium channel in patients referred for Brugada syndrome genetic testing. *Heart Rhythm*. 2010;7(1):33-46.
- Probst V, Veltmann C, Eckardt L, et al. Long-term prognosis of patients diagnosed with Brugada syndrome: results from the FINGER Brugada Syndrome Registry. *Circulation*. 2010;121(5):635-643
- Priori SG, Napolitano C, Gasparini M, et al. Natural history of Brugada syndrome: insights for risk stratification and management. *Circulation*. 2002;105(11):1342-1347
- Bai R, Napolitano C, Bloise R, Monteforte N, Priori SG. Yield of genetic screening in inherited cardiac channelopathies: how to prioritize access to genetic testing. *Circ Arrhythm Electrophysiol*. 2009;2(1):6-15
- Sacher F, Probst V, Iesaka Y, et al. Outcome after implantation of a cardioverter-defibrillator in patients with Brugada syndrome: a multicenter study. *Circulation*. 2006;114(22):2317-2324
- Sinner MF, Pfeufer A, Akyol M, et al. The non-synonymous coding IKr-channel variant KCNH2-K897T is associated with atrial fibrillation: results from a systematic candidate gene-based analysis of KCNH2 (HERG). *Eur Heart J*. 2008;29(7):907-914.
- Goldenberg I, Moss AJ, Bradley J, et al. Long-QT syndrome after age 40. *Circulation*. 2008;117(17):2192-2201
- Sauer AJ, Moss AJ, McNitt S, et al. Long QT syndrome in adults. *J Am Coll Cardiol*. 2007;49(3):329-337.
- Moss AJ, Shimizu W, Wilde AA, et al. Clinical aspects of type-1 long-QT syndrome by location, coding type, and biophysical function of mutations involving the KCNQ1 gene. *Circulation*. 2007;115(19):2481-2489
- Phillips KA, Ackerman MJ, Sakowski J, Berul CI. Cost-effectiveness analysis of genetic testing for familial long QT syndrome in symptomatic index cases. *Heart Rhythm*. 2005;2(12):1294-1300
- Postma AV, Denjoy I, Kamblock J, et al. Catecholaminergic polymorphic ventricular tachycardia: RYR2 mutations, bradycardia, and follow up of the patients. *J Med Genet*. 2005;42(11):863-870.
- Priori SG, Napolitano C, Memmi M, et al. Clinical and molecular characterization of patients with catecholaminergic polymorphic ventricular tachycardia. *Circulation*. 2002;106(1):69-74.
- Medeiros-Domingo A, Bhuiyan ZA, Tester DJ, et al. The RYR2-encoded ryanodine receptor/calcium release channel in patients diagnosed previously with either catecholaminergic polymorphic ventricular tachycardia or genotype negative, exercise-induced long QT syndrome: a comprehensive open reading frame mutational analysis. *J Am Coll Cardiol*. 2009;54(22):2065-2074.
- di Barletta MR, Viatchenko-Karpinski S, Nori A, et al. Clinical phenotype and functional characterization of CASQ2 mutations associated with catecholaminergic polymorphic ventricular tachycardia. *Circulation*. 2006;114(10):1012-1019.
- Zipes DP, Camm AJ, Borggrefe M, et al.; American College of Cardiology/American Heart Association Task Force; European Society of Cardiology Committee for Practice Guidelines; European Heart Rhythm Association; Heart Rhythm Society. ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Circulation*. 2006;114(10):e385-e484.
- Tester DJ, Will ML, Haglund CM, Ackerman MJ. Compendium of cardiac channel mutations in 541 consecutive unrelated patients referred for long QT syndrome genetic testing. *Heart Rhythm*. 2005;2(5):507-517.
- Tester DJ, Salisbury BA, Carr JL, et al. The effect of mutation class on QTC in unrelated patients referred for the Familion™ genetic test for long QT syndrome. Presented at: Heart Rhythm Society 28th Annual Scientific Sessions; May

9-12, 2007; Colorado, AZ. Abstract AB28-2. Heart Rhythm Society. Scientific Sessions. Past Meetings. Heart Rhythm 2007. Abstracts. Search: author (requires registration). Available at: <http://www.abstracts2view.com/hrs07/sessionindex.php>.

Tester DJ, Salisbury BA, Carr JL, et al. Clinical phenotype and the yield of the Familion™ genetic test for congenital long QT syndrome. Presented at: Heart Rhythm Society 28th Annual Scientific Sessions; May 9-12, 2007; Colorado, AZ. Abstract AB10-3. Heart Rhythm Society. Scientific Sessions. Past Meetings. Heart Rhythm 2007. Abstracts. Search: author (requires registration). Available at: <http://www.abstracts2view.com/hrs07/sessionindex.php>.

Phillips KA, Ackerman MJ, Sakowski J, Berul CI. Cost-effectiveness analysis of genetic testing for familial long QT syndrome in symptomatic index cases. *Heart Rhythm*. 2005;2(12):1294-1300.

Kumar, S, Peters, S, Thompson, T, Morgan, et al. Familial cardiological and targeted genetic evaluation: low yield in sudden unexplained death and high yield in unexplained cardiac arrest syndromes. *Heart Rhythm*. 2013;10(11):1653-1660.

Schwartz PJ, Crotti L. QTc behavior during exercise and genetic testing for the long-QT syndrome. *Circulation*. 2011;124:2181-4

Arking, DE, Juntila, MJ, Goyette, P, et al. Identification of a sudden cardiac death susceptibility locus at 2q24.2 through genome-wide association in European ancestry individuals. *PLoS Genet*. 2011;7(6):e1002158.

Magi, S., Lariccia, V., Maiolino, M., Amoroso, S., & Gratteri, S. Sudden cardiac death: focus on the genetics of channelopathies and cardiomyopathies. *J Biomed Sci*, 2017; 24.

Kalia, S. S., Adelman, K., Bale, S. J., Chung, W. K., Eng, C., Evans, J. P. et al. Recommendations for reporting of secondary findings in clinical exome and genome sequencing, 2016 update (ACMG SF v2.0): a policy statement of the American College of Medical Genetics and Genomics. *Genetics In Medicine*, 2016;19, 249.

Hershberger, R. E., Givertz, M. M., Ho, C. Y., Judge, D. P., Kantor, P. F., McBride, K. L. et al. Genetic evaluation of cardiomyopathy: a clinical practice resource of the American College of Medical Genetics and Genomics (ACMG). *Genet Med*. 2018 doi:10.1038/s41436-018-0039-z

van Lint, F. H. M., Mook, O. R. F., Alders, M., Bikker, H., Lekanne Dit Deprez, R. H., & Christiaans, I. Large next-generation sequencing gene panels in genetic heart disease: yield of pathogenic variants and variants of unknown significance. *Neth Heart J*, 2019;27(6), 304-309.

Adler A, Novelli V, Amin AS, Abiusi E, Care M, Nannenberg EA, et al. an international, multicentered, evidence-based reappraisal of genes reported to cause congenital long QT syndrome. *Circulation*. 2020 Feb 11;141(6):418-428

Parker, L. E., & Landstrom, A. P. The clinical utility of pediatric cardiomyopathy genetic testing: From diagnosis to a precision medicine-based approach to care. *Prog Pediatr Cardiol*, 2021;62.

Sun L, Pennells L, Kaptoge S, et al. Polygenic risk scores in cardiovascular risk prediction: a cohort study and modelling analysis. *PLoS Med*. 2021 Jan; 18(1):e1003498.

Viskin S, Chorin E, et al. Polymorphic Ventricular Tachycardia: Terminology, Mechanism, Diagnosis, and Emergency Therapy. *Circulation* 2021;144(10):823-839.

Towbin JA, McKenna WJ, Abrams DJ, et al. 2019 HRS expert consensus statement on evaluation, risk stratification, and management of arrhythmogenic cardiomyopathy. *Heart Rhythm*. 2019 Nov;16(11):e301-e372.

Musunuru K, Hershberger RE, Day SM, et al. American Heart Association Council on Genomic and Precision Medicine; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; and Council on Clinical Cardiology. Genetic Testing for Inherited Cardiovascular Diseases: A Scientific Statement From the American Heart Association. *Circ Genom Precis Med*. 2020 Aug;13(4):e000067.

Wilde AAM, Semsarian C, Márquez MF, et al.; Document Reviewers. European Heart Rhythm Association (EHRA)/Heart Rhythm Society (HRS)/Asia Pacific Heart Rhythm Society (APHRS)/Latin American Heart Rhythm Society (LAHRS) Expert Consensus Statement on the state of genetic testing for cardiac diseases. *Heart Rhythm*. 2022 Apr 1:S1547-5271(22)01697-6

Vissing CR, Espersen K, Mills HL, et al. Family Screening in Dilated Cardiomyopathy: Prevalence, Incidence, and Potential for Limiting Follow-Up. JACC Heart Fail. Nov 2022; 10(11): 792-803.

Dellefave-Castillo LM, Cirino AL, et al. Assessment of the Diagnostic Yield of Combined Cardiomyopathy and Arrhythmia Genetic Testing. JAMA Cardiol. 2022;7(9):966-974.

MolDx. Billing and Coding: MolDX: Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/C) Testing A55235

Ommen SR, Mital S, Burke MA, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 2020;76:e159-e240.

2023 ESC Guidelines for the Management of Cardiomyopathies: Developed by the Task Force on the Management of Cardiomyopathies of the European Society of Cardiology. Eur Heart J 2023;Aug 25:[Epub ahead of print].

Penetrance of Dilated Cardiomyopathy in Genotype-Positive Relatives. J Am Coll Cardiol 2024;83:1640-1651.

2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol 2024;May 8:[Epub ahead of print].

This policy will be revised as necessary and reviewed no less than annually.

Devised: 7/21

Revised: 7/22 (add indication for CPVT), 7/23 (grammatical revisions), 7/24 (add MolDx exclusion)

Reviewed:

CMS UM Oversight Committee Approval: 12/23, 7/24

Geisinger Health Plan may refer collectively to health care coverage sponsors Geisinger Health Plan, Geisinger Quality Options, Inc., and Geisinger Indemnity Insurance Company, unless otherwise noted. Geisinger Health Plan is part of Geisinger, an integrated health care delivery and coverage organization.

Coverage for experimental or investigational treatments, services and procedures is specifically excluded under the member's certificate with Geisinger Health Plan. Unproven services outside of an approved clinical trial are also specifically excluded under the member's certificate with Geisinger Health Plan. This policy does not expand coverage to services or items specifically excluded from coverage in the member's certificate with Geisinger Health Plan. Additional information can be found in MP015 Experimental, Investigational or Unproven Services.

Prior authorization and/or pre-certification requirements for services or items may apply. Pre-certification lists may be found in the member's contract specific benefit document. Prior authorization requirements can be found at <https://www.geisinger.org/health-plan/providers/ghp-clinical-policies>

Please be advised that the use of the logos, service marks or names of Geisinger Health Plan, Geisinger Quality Options, Inc. and Geisinger Indemnity Insurance Company on a marketing, press releases or any communication piece regarding the contents of this medical policy is strictly prohibited without the prior written consent of Geisinger Health Plan. Additionally, the above medical policy does not confer any endorsement by Geisinger Health Plan, Geisinger Quality Options, Inc. and Geisinger Indemnity Insurance Company regarding the medical service, medical device or medical lab test described under this medical policy.