Local Coverage Determination (LCD): Corneal Hysteresis (L38026)

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Contractor Information

CONTRACTOR NAME	CONTRACT TYPE	CONTRACT NUMBER	JURISDICTION	STATE(S)
Palmetto GBA	A and B MAC	10111 - MAC A	J - J	Alabama
Palmetto GBA	A and B MAC	10112 - MAC B	J - J	Alabama
Palmetto GBA	A and B MAC	10211 - MAC A	J - J	Georgia
Palmetto GBA	A and B MAC	10212 - MAC B	J - J	Georgia
Palmetto GBA	A and B MAC	10311 - MAC A	J - J	Tennessee
Palmetto GBA	A and B MAC	10312 - MAC B	J - J	Tennessee
Palmetto GBA	A and B and HHH MAC	11201 - MAC A	J - M	South Carolina
Palmetto GBA	A and B and HHH MAC	11202 - MAC B	J - M	South Carolina
Palmetto GBA	A and B and HHH MAC	11301 - MAC A	J - M	Virginia
Palmetto GBA	A and B and HHH MAC	11302 - MAC B	J - M	Virginia
Palmetto GBA	A and B and HHH MAC	11401 - MAC A	J - M	West Virginia
Palmetto GBA	A and B and HHH MAC	11402 - MAC B	J - M	West Virginia
Palmetto GBA	A and B and HHH MAC	11501 - MAC A	J - M	North Carolina
Palmetto GBA	A and B and HHH MAC	11502 - MAC B	J - M	North Carolina

LCD Information

Document Information

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L38026

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Corneal Hysteresis

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Notice Period Start Date
09/05/2019

Notice Period End Date
10/20/2019

CMS National Coverage Policy

Title XVIII of the Social Security Act, §1862(a)(1)A) allows coverage and payment for only those services that are considered to be reasonable and necessary for the diagnosis or treatment of illness or injury or to improve the functioning of a malformed body member.

Title XVIII of the Social Security Act, §1862(a)(1)(D) addresses services that are determined to be investigational or experimental.

Title XVIII of the Social Security Act §1833(e) prohibits Medicare payment for any claim which lacks the necessary information to process the claim.

CMS Internet-Only Manual, Pub 100-02, Medicare Benefit Policy Manual, Chapter 16, §10 General Exclusions from Coverage

CMS Internet-Only Manual, Pub 100-04, Medicare Claims Processing Manual, Chapter 23, §30A Services Paid Under the Medicare Physician's Fee Schedule CMS Internet-Only Manual, Pub 100-04, Medicare Claims Processing Manual, Chapter 30, §50.3.1 Mandatory ABN Uses

Coverage Guidance

Coverage Indications, Limitations, and/or Medical Necessity

This is a NON-coverage policy for all corneal hysteresis assessments as a means of risk assessment or monitoring for progression of ophthalmic disease activity.

Summary of Evidence

Hysteresis is a measure of resistance to deformation to an applied force. Corneal hysteresis (CH) is a measure of the viscoelastic dampening property of the cornea and is postulated to be a surrogate for the viscoelastic dampening properties of the posterior sclera and lamina cribrosa through which the retinal ganglion cell axons pass as they exit the eye. It has been theorized that glaucomatous damage to the retinal ganglion cell axons occurs at the lamina cribrosa and that viscoelastic differences in the lamina cribrosa are responsible for differential effects of intraocular pressure within these tissues and contribute to the susceptibility to intraocular pressure (IOP)-mediated damage. Studies show an association between a lower CH and glaucoma or glaucoma risk, and it has been proposed as a risk stratification tool for use in the treatment of glaucoma, glaucoma suspect, and ocular hypertension. CH is not itself a modifiable risk factor for glaucoma, but theoretically could signal the need for more aggressive IOP reduction.

The Ocular Response Analyzer (ORA) (Reichert Inc., Buffalo, New York, USA) is a non-contact tonometer that measures CH. The ORA received FDA 510(k) clearance in 2004 for the intended use of measurement of intraocular pressure and biomechanical response of the cornea "for the purpose of aiding in the diagnosis and monitoring of glaucoma."[1] This device measures CH by measuring the difference of two applanation event pressures taken during the inward and outward movement of the cornea following delivery of a metered pulse of air.

A 2006 retrospective, observational study compared CH and central corneal thickness (CCT) on various indices of glaucomatous damage in 230 patients (mean 65 years), 85% diagnosed with primary open angle glaucoma (POAG) or glaucoma suspect, and 15% with angle-closure glaucoma, suspected angle-closure glaucoma, or secondary glaucoma.[2] Multivariate analysis found CCT, but not CH to be predictive of higher cup-to-disk ratio (CDR) (P=0.02 vs. P=0.36). Multivariate analysis found lower CH but not CCT to be predictive of visual field progression (P = 0.30), but not after factoring in axial length (P = 0.09). Neither CH nor CCT were significantly associated with worsening mean deviation (MD) or pattern standard deviation (PSD). A 2011 prospective observational study of 162 POAG subjects found no statistical difference in CH compared with 150 normal subjects.[3] A small (57 patients), 2012 retrospective study found both CH and IOP to be independent, statistically significant predictors of response to topical prostaglandin treatment.[4]

A 2012 prospective cohort study of 153 patients (153 eyes) with established glaucoma evaluated the relationship between CCT and CH and their correlation with progressive visual field (VF) loss.[5] Baseline measurements included age, race, sex, CH, MD, PSD, CCT, and IOP (calculated by averaging the first 4 measurements following the baseline VF), peak IOP, and corneal compensated IOP (IOPcc). Progression of glaucoma was determined by an automated pointwise linear regression analysis of visual field tests. Progression occurred in 25 enrolled eyes (16%) and demonstrated significantly lower CCT and CH compared with non-progressed eyes ($p=0.04$ and $p<0.01$, respectively). There was significant correlation between CH and CCT ($r=0.33$, $P<0.01$). After multivariate analysis,

peak IOP, age, and CH were demonstrated to be significantly associated with glaucomatous visual field progression. The authors conclude that "as CH may describe corneal properties more completely than thickness alone, it may be a parameter that is better associated with progression." However, of the 25 subjects that demonstrated glaucomatous progression, 9 had either secondary glaucoma, juvenile glaucoma, or angle closure glaucoma. No subgroup analysis was performed. Other confounders include the use of CH measurements obtained during a non-standardized episode in the care continuum (not at baseline), as well as the non-standardized treatment and follow-up (provider discretion) protocol.

A 2013 retrospective study of 131 glaucoma patients investigated the correlation between CH and other structural markers of glaucomatous damage on spectral domain optical coherence tomography (SDOCT).[6] In a multivariable analysis including MD, age, average retinal nerve fiber layer (RNFL) thickness, and glaucoma status, only MD ($p = 0.001$) and age ($p < 0.001$) retained significant associations with CH. The authors conclude that "in patients under evaluation and treatment for glaucoma, CH was more closely related to visual field MD than to structural markers of glaucoma damage as measured by SDOCT."

A 2017 cross sectional study compared single CH measurements among 123 patients (123 eyes) previously diagnosed with either glaucoma (high tension glaucoma, N=37; pseudoexfoliative glaucoma, N=12; normal tension glaucoma, N=24), ocular hypertension (OHT) (N=28), or glaucoma-like optic discs (GLD) (N=22).[7] A One-way Analysis of Covariance (ANCOVA), correcting for differences in age and IOP, found mean CH to be significantly lower in patients with glaucoma versus those with OHT and GLD ($p < 0.001$). The authors hypothesize there may be greater viscoelasticity in ocular tissues of GLD and OHT which may have a protective role against glaucomatous nerve damage.

The following four studies by the same principal investigator included subjects who were part of the larger Diagnostic Innovations in Glaucoma Study (DIGS).[8-11] DIGS is a single-center, prospective, longitudinal cohort study of the relationships between optic nerve structure and glaucomatous vision loss, and the assessment of new diagnostic and monitoring modalities that could be used to mitigate functional vision loss by identifying at-risk patients through earlier detection and intervention.

A 2012 observational cross-sectional study of the association between CH and severity of glaucoma, as defined by automated visual field deficits and RNFL thickness, among 299 eyes in 191 glaucoma or glaucoma suspect patients.[8] In multivariable regression models, after adjusting for central corneal thickness, age, and axial length, the relationship of CH to RNFL thickness was not statistically significant. The authors conclude they found only "a weak relationship between corneal biomechanical parameters and measures of structural and functional damage in glaucoma."

A 2013 prospective, observational study looked at the relationship between baseline CH and visual field progression in 68 patients (114 eyes) with confirmed diagnosis of open angle glaucoma.[9] CH measurements were obtained at the baseline study visit. Subjects underwent baseline and every 6 month follow-up examinations which included examination and assessment of Goldmann applanation tonometer (GAT) IOP, CCT, Humphrey visual field, stereoscopic disc photos, and axial length measurements. Treatment was uncontrolled and at the discretion of the treating physician. Subjects were followed for an average of 4 years (range, 2.0-6.6 years), during which visual fields were assessed for evidence of progression using the visual field index (VFI) method. Univariable analysis found that each 1 mmHg lower baseline CH was significantly associated with a 0.25%/year faster rate of visual field progression ($p<0.001$). The multivariable model showed an interaction between IOP and CH; eyes with high IOP and low CH were at increased risk for having fast rates of disease progression. CH explained a larger proportion of the variation in VFI change than CCT (17.4% vs. 5.2%, respectively). The authors conclude: "The prospective longitudinal design of this study supports the role of CH as an important factor to be considered in the assessment of the risk of progression in glaucoma patients."

In a 2016 prospective, observational cohort study, the relationship between CH and progressive loss of the retinal nerve fiber layer (RNFL) was analyzed in 133 patients (186 eyes) with confirmed diagnosis of open angle glaucoma.[10] CH measurements were obtained at the baseline study visit. Subjects underwent baseline and every 6 month follow-up examinations which included examination and assessment of GAT IOP, CCT, Humphrey visual field, stereoscopic disc photos, and circumpapillary retinal nerve fiber layer thickness measurements with the spectral domain optical coherence tomography (SD-OCT). Treatment was uncontrolled and at the discretion of the treating physician. Subjects were followed for an average of 3.8 years (range, 2.0-5.2 years) during which time average circumpapillary RNFL thickness measurements and stereo disc photos were assessed for evidence of glaucomatous progression. Univariable analysis found that each 1 mmHg lower baseline CH was significantly associated with a 0.13 um/year faster loss of RNFL (p = 0.011). In multivariable analysis adjusting for age, race, average GAT IOP and CTT, CH was still associated with a faster rate of RNFL loss (p=0.015). The authors conclude that "the prospective longitudinal design of this study supports a role for CH as a risk factor for progression in glaucoma." Both the Medeiros and Zhang studies were small and confounded by the fact that treatment was not controlled. Though findings were suggestive, the use of a complex regression model that was not clearly developed from a-priori hypothesized relationships and not validated following development do not allow firm conclusions about the generalizability of the results.

A 2018 prospective, observational study investigated the predictive role of CH as a risk factor for the development of glaucoma in a cohort of glaucoma suspect patients.[11] The study included 199 patients (287 eyes) recruited from a single site. Treatment for glaucoma suspect was uncontrolled and subject to discretion of the treating physician. Baseline measurements included CH, GAT IOP, CCT, Humphrey visual field, and stereoscopic disc examination. Subjects were examined every six months for an average follow-up period of 3.9 years during which time glaucoma developed in 19% of enrolled eyes (54 eyes in 48 patients). Baseline CH and age was significantly lower in those who developed glaucoma vs. those who did not (9.5 ±1.5 mm Hg vs. 10.2 ±2.0 mm Hg; p = 0.012). Baseline MD and PSD were significantly different between the two groups. CH was found to be predictive of glaucoma development in a multivariable model (hazard ratio = 1.20; 95% CI: 1.01-1.42; p = 0.04), while baseline IOP, CCT, and treatment were not. Each 1 mmHg lower CH was associated with a 21% increase in risk of glaucoma development (95% CI 1.04-1.41; p = 0.013). The authors acknowledged that "because the impact of CCT on risk of glaucoma development is now widely known, it is likely that physicians may have treated more aggressively eyes of glaucoma suspects who had thin corneas, also artificially reducing the impact of CCT as a predictive factor," and that, "the higher predictive value of CH compared to CCT in our study should be seen with caution." Additionally, the multivariable analysis included only some of the known risk factors of glaucoma development, specifically, age, IOP, CCT, PSD, and treatment, but excluded others such as race, family history, and optic disc morphology (CDR). The authors conclude that "future studies including randomization protocols controlling for treatment should be performed to clarify the relative importance of these predictive factors."

A 2017 meta-analysis included 19 studies that assessed CH in 1213 eyes with glaucoma and 1055 healthy eyes.[12] Mean CH was 1.5 mm Hg lower, and mean CCT 8.5 micrometer less thick, in eyes with glaucoma (P < 0.0001 and P < 0.001, respectively). The authors conclude that there are differences in corneal properties such as CH and CCT between patients with glaucoma and healthy controls "and support further studies on the influence of CH and CCT in glaucoma screening and diagnosis." In a 2018 prospective cross-sectional study of CH as a potential glaucoma screening tool in 46 patients (76 eyes) on routine eye exam, the 21 eyes (27.6%) found to have normal tension glaucoma (NTG) did not differ statistically in CH (P = 0.19).[13]

A limited body of evidence suggests there may be a role in the application of CH in the identification of corneal pathology or in preoperative assessment prior to refractive surgery. A 2007 study first described a statistically significant difference in the mean CH of 207 normal and 93 keratoconic eyes (10.7 + 2.0 mmHg vs. 9.6 + 2.2 mmHg; p<0.0001).[14] The study also revealed that CH values in the keratoconic eyes decreased with increasing severity of disease, though could not differentiate between eyes with mild keratoconus and normal controls. A subsequent study similarly found poor overall predictive accuracy for CH to detect mild keratoconus from age- and gender–matched controls.[15] A 2011 study investigated the ability of the ORA parameters to aid in diagnosis of

keratoconus in preoperative laser in situ keratomileusis (LASIK) patients.[16] Biomechanical measurements were acquired from 103 eyes with mild keratoconus and 97 control eyes and 12 parameters were analyzed. Though sensitivity and specificity of the parameters was low (66% and 67%, respectively, for CH), the authors concluded that some parameters offered high negative likelihood ratios and should be studied in a larger sample size.

Analysis of Evidence
(Rationale for Determination)

Neither the current (2015) nor most recent updates (2017) to the American Academy of Ophthalmology (AAO) Preferred Practice Pattern (PPP) guidelines for glaucoma recommend measurement of CH in the management or risk assessment of glaucoma, glaucoma suspect, or ocular hypertension.[17-19] Similarly, the Canadian Association of Optometrists (CAO), notes that "despite the association between CH and glaucoma onset and progression, there is still a paucity of clinical evidence to support adding CH measurement to the standard glaucoma workup.[20] The AAO PPP 2018 guidelines for corneal ectasia concede that while measures of corneal biomechanics, including CH, are likely altered in corneal ectasia, the parameters for use in the detection at the subclinical stage is currently being evaluated.[21]

A 2018 Hayes review of 16 qualifying studies found that CH testing has some capacity to detect presence of glaucoma, to predict risk for glaucoma progression, and to predict response of glaucoma to certain types of treatment.[22] However, "the evidence is mainly comprised of very poor quality correlation studies, which lack the rigor to determine diagnostic or prognostic accuracy. Most of these studies did not use reliable methods to determine the accuracy of diagnosis or prognosis. No studies were identified that directly assessed the clinical utility of CH measurement for selecting treatment for glaucoma or for impacting long-term health outcomes." This perspective was also reflected in the one recent meta-analysis; although CH was correlated with glaucoma, there were no findings related to the use of CH for the prognosis, diagnosis, or management of glaucoma.[12]

In summary, CH is promising as a risk assessment tool in the diagnosis and management of glaucoma or corneal pathology. However, while the body of evidence is large, the overall quality is low. The studies are relatively small, observational, often confounded by lack of treatment control, uniformly citing simple correlations, precluding cause-and-effect conclusions. Not only are there no Level I studies, none of the reviewed studies demonstrate that CH measurement alters clinical management and improves clinical outcomes. A wide array of tests are accepted for detection and monitoring of glaucoma (tonometry for IOP, perimetry to assess visual field, ophthalmoscopy to detect a glaucomatous optic nerve head (ONH) and RNFL changes, and pachymetry for CCT). It is still unclear whether CH provides useful additional information, much less its optimal role in any diagnostic, prognostic, and treatment algorithm. Randomized controlled trials (RCTs) that compare outcomes in patients whose treatment is selected based on CH are needed to determine definitive patient selection criteria and clinical utility. The lack of level I evidence, absence of proven clinical utility, no clinical practice guideline endorsement,[18-20,23] as well neither Medicare[24-27] nor commercial[28-30] coverage, argue strongly against current CH coverage as reasonable and necessary for treatment of Medicare patients.

General Information

Associated Information

N/A

Sources of Information

N/A

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Revision History Information

REVISION HISTORY DATE	REVISION HISTORY NUMBER	REVISION HISTORY EXPLANATION	REASON(S) FOR CHANGE
10/21/2019	R1	This LCD is being revised in order to adhere to CMS requirements per chapter 13, section 13.5.1 of the Program Integrity Manual, to remove all coding from LCDs. There has been no change in coverage with this LCD revision.	• Provider Education/Guidance

Associated Documents

Attachments

N/A

Related Local Coverage Documents

Article(s)
A56968 - Response to Comments: Corneal Hysteresis
LCD(s)
DL38026 - Corneal Hysteresis

Related National Coverage Documents

N/A

Public Version(s)

Updated on 09/26/2019 with effective dates 10/21/2019 - N/A
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- hysteresis