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Clinical Relationship Between Cervicogenic Headache and Leg Length Discrepancy

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Abstract

Background: Cervicogenic headache (CGH) is a secondary classification of headache theorized to originate from degenerative pathology and/or nociceptive irritation of the cervical spine. The purpose of this study was to assess if a relationship existed between CGH and clinically relevant leg length discrepancy.

Methods: Participant demographic data were collected [age (years), gender, height (cm), body mass (kg), and body mass index (BMI) ($kg \bullet m^{-2}$)] on 37 individuals between the ages of 18 and 65 years. Participants completed a CGH diagnostic questionnaire, were assessed for blood pressure, leg length discrepancy, cervical range of motion, cervical posture, cervical facet pathological degeneration through palpation, and underwent the cervical flexion-rotation test.

Results: Twenty-three of thirty-seven participants met the CGH diagnostic criteria. Significant differences were demonstrated between participants who met and those who did not meet CGH diagnostic criteria regarding LLD ($\bar{x}$=32.82, SD ±6.19 mm and $\bar{x}$=7.49, SD±5.70 mm, respectively) (p<0.0001), flexion-rotation test side-to-side difference mean ($\bar{x}$=27.74, SD±9.09 degrees and $\bar{x}$=3.43, SD±1.90 degrees, respectively) (p<0.0001), and headache frequency mean ($\bar{x}$=3.34, SD±2.45 days per week and $\bar{x}$=0.42, SD ±0.72 days per week, respectively) (p=0.004). Relationships were investigated between LLD >20mm and; diagnostic flexion-rotation test (r=1.00 p=2.22E-16), CGH questionnaire (r=0.91 p=1.52E-12), additional diagnostic criteria (r=0.79 p=1.68E-07), flexion-rotation test side-to-side difference (r=0.72 p=7.69E-06), and headache frequency (r=0.56 p=0.001).

Conclusion: A significant association was found between participants who positively met diagnostic criteria for cervicogenic headache and leg length discrepancy >20 mm, significantly different from participants with frequent headache who did not meet the diagnostic criteria for cervicogenic headache. Cervicogenic headache is a secondary classification of headache whose etiology is derived from cervical spine pathology. Treatment for cervicogenic headache has focused on mediation of cervical spine pathology with inattention for the etiology of unilateral symptom presentation. Cervicogenic headache treatment outcomes have been effective regarding symptom mediation, yet fleeting. Leg length discrepancy >20mm should be considered in the examination and treatment regarding cervical spine pathological degeneration as found to be responsible for the presentation of cervicogenic headache.

Keywords: Cervical spine, cervicogenic headache, cervicogenic migraine, flexion-rotation test, leg length discrepancy, cervicogenic headache diagnosis

Introduction

Headache, historically associated with pathology of the nervous system, is a common disorder experienced by more than half of the adult population each year in the United States (US) [1]. It is one of the most common causes of chronic pain [2] and has been identified as the leading cause for outpatient neurologist

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visits in the US [3,4]. Approximately 14.2% of US adults suffered from migraine-like, or severe headache in 2012 [5]. Greater than 3% of the US adult population suffered headaches daily and approximately 36% experienced multiple headaches each month [1]. Headache has commonly been under-diagnosed, under-estimated, and under-treated due, in-part, to ambiguous and over-lapping diagnostic criteria [1,6,7]. The International Headache Society [8] has established three headache classifications; 1) primary (migraine, tension-type headache, trigeminal autonomic cephalalgias, and other primary headache disorders), 2) secondary (headache attributed to trauma or injury to the head and/or neck, headache attributed to cranial and/or cervical vascular disorder, headache attributed to non-vascular intracranial disorder, headache attributed to a substance or its withdrawal, headache attributed to infection, headache attributed to disorder of homeostasis, headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cervical structure, and headache attributed to psychiatric disorder), and 3) tertiary (painful cranial neuropathies, other facial pain and other headaches) [8]. Headache disorders have presented with recurrent frequency and have been associated with societal, financial, and personal burdens related to pain, reduced quality of life (QOL) [9], disability [1], and increased cardiovascular and respiratory disease risk [2]. There is a vital need for health initiatives dedicated towards improved headache prevention and treatment as headache costs a minimum of 100 million dollars per million citizens each year [4].

Cervicogenic headache (CGH) is a secondary classification of headache, or headache syndrome, [8] theorized to originate from nociceptive irritation of the cervical spine [10-13]. CGH incidence rates ranged from 0.7% and 13.8% [11] to 17.8% of the population, similar in prevalence and presentation as migraine headache [6,7,14]. CGH has been referenced as symptoms that initially present at the neck, or the occipital/sub-occipital region with pain distribution to the oculo-frontal-temporal region, primarily noted as unilateral symptom presentation with potential ipsilateral shoulder and/or arm pain [11,15,16]. CGH onset appeared to be stimulated by provocative cervical posturing or movements, cervical strain, and/or pressure application to proximal cervical tender-points [11,16]. The etiology of CGH has included theories that have suggested irritation, or pathological mechanics of the myodural bridge [17,18], convergence of the trigeminal-cervical nucleus with nociceptive C_1-C_3 afferents and the nociceptive afferents from the trigeminal nucleus and/or the greater occipital nerve [13,18], C_2-C_3 intervertebral disc pathology [19], pathology of C_1-C_3 (potentially C_4), cervical paraspinal muscles, zygapophyseal (facet) joints, cervical vasculature, lymphatics, and cervical ligamentous instability [18,20-23]. CGH associated cervical spine pathology presented with inflammatory cytokine expression, primarily IL-1β and TNF-α, which resulted in hyper-sensitized regional pain fibers [22]. Bogduk et al. [13] suggested the strongest evidence of CGH etiology was C_2-C_3 (facet) zygapophyseal joint pathology/degeneration. Roh et al. [22] reported excessive zygapophyseal joint loading commonly yielded regional expression of inflammatory cytokines with resultant spondylosis and neural stimulation secondary to inflammatory cytokine expression.

CGH diagnostic criteria were developed by Sjaastad et al. in 1990 and revised in 1998 [10,15]. The primary CGH diagnostic criteria included symptom unilaterality, symptom provocation through cervical movement and/or awkward cervical postural, external pressure application through palpation to associated posterior cervical spine segments with resultant symptom reproduction, reduced cervical range of motion (ROM), pain originating at the neck and radiating to oculo-frontal-temporal regions, possible ipsilateral shoulder and/or arm pain of vague reference, autonomic symptoms and signs which included nausea, vomiting, dizziness, phono and photophobia, and anesthetic blockade of the greater occipital nerve, C_2 root, or other ipsilateral involved cervical structures with symptom mediation [6,10]. Clinical presentation of symptom unilaterality triggered by head or neck movements, unilateral headache triggered by manual pressure on cervical trigger points, and pain initiated at the neck and spreading to the oculo-frontal-temporal region satisfied diagnostic criteria for CGH with the strength of diagnosis promoted through the addition of specific additional positive diagnostic criteria [24,25]. Diagnostic imaging did not reliably confirm CGH [26]. Zito et al. [27] determined that individuals with CGH demonstrated reduced cervical flexion/extension range of motion (P=0.048), higher incidences of proximal cervical (facet) zygapophyseal joint dysfunction ($p<0.05$) as determined through manual examination compared to individuals with migraine with aura and controls. Hanten et al. [28] found cervical spine mobility testing, which included cervical spine palpation, to be a reliable method for identifying CGH. The flexion-rotation test has been utilized as a CGH diagnostic tool with sensitivity of 90% [29] and 91% [30], specificity of 89% [29] and 90% [30], and diagnostic accuracy of 89% [29].

CGH treatment has typically involved pain mediation through analgesic medication such as non-steroidal anti-inflammatory drugs (NSAIDs) and acetaminophen, ergotamines, serotonin 5-$HT_{1B/D}$ agonists, Infliximab, and botulinum toxin injections [11]. Other treatments commonly employed to treat CGH have been chiropractic, manual physical therapy, transcutaneous electrical nerve stimulation (TENS), glucocorticoid and analgesic local injections, and attempts at surgical symptom mediation/decompression [11,18]. Pulsed radiofrequency neurotomy to involved structures appeared to mediate symptoms, yet relief was short-term [31-33]. Orthobiological treatments such as platelet rich plasma (PRP) and other regenerative treatments have demonstrated promising results [20].

Linetsky et al. [20] reported clinical findings associated with CGH to be postural abnormalities, functional asymmetries, and combinations of kyphoscoliosis, flattening of the lumbar

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and/or cervical lordosis, excessive or restricted active and/or passive cervical ROM. However, limited research has been performed regarding postural anomalies potentially associated with CGH origin, perturbation, and unilateral cervical spine degeneration. Ahern et al. [34] found more than 60% of study participants with chronic low back pain (LBP) demonstrated concomitant headache. Headache occurrence prior to LBP onset was demonstrated in greater than 24% of study participants, which indicated a degree of causal commonality [34]. Fishbain et al. [7] reported a common relationship between the occurrence of LBP and debilitating headache. Christie et al. [35] reported a relationship between postural anomalies and low back pain (LBP). Cervical spine mechanics, posture, and pain have been shown to be altered by anomalous movement or posture of more inferior regions, such as the shoulder girdle, pelvis, and thoracic and lumbar spine [36-38]. McDonnell et al. [39] linked altered lumbar posture and mobility to CGH, with CGH pain mediated through correction of lumbar mechanics. A direct relationship between pelvic, lumbar, and cervical spine posture has been established [40]. Compensatory cervical posture has been linked with postural anomalies at the lumbar spine and pelvis [41,42]. This compensatory cervical spine postural has been shown to alter (facet) zygapophyseal joint load/compression with resultant pathological degeneration [41-43], inflammatory cytokine expression [22], and the environment/clinical presentation associated with CGH.

Leg length discrepancy (LLD) greater than or equal to 15mm [44] to 20mm [45] has been shown to alter lumbar posture [45-48], has been associated with cervical pathology [49], and has been referenced as a risk factor for CGH [50], yet no studies to date have specifically studied the existence of a relationship between LLD and CGH. Therefore, the purpose of this study was to assess if a relationship existed between CGH and clinically relevant LLD ≥20 mm. The hypothesis that directed this study stated that individuals who positively fulfilled CGH diagnostic criteria [7,15] would demonstrate a positive and significant LLD ≥ 20mm [45,46].

Methods

Participants

This observational study was conducted at Southern Connecticut State University (SCSU) in the greater New Haven County of Connecticut, USA and was approved by the Southern Connecticut State University Institutional Review Board. Thirty–seven (37) participants from New Haven County, Connecticut between the ages of 18 and 65 years [51] with complaint of headache were recruited via word-of-mouth for voluntary test protocol participation, were informed of the study purpose, procedures, known risks, and provided written informed consent prior to study participation.

Study participant inclusion criteria were: 1) participants had to be between the ages of 18–65 years [51], and 2) demonstrate frequent headache, preferably based on diagnostic criteria documented by Fishbain et al. [7] for cervicogenic headache, including headache symptom unilaterality (symptoms on one side of the head) associated with concurrent neck pain and aggravated by neck postures or head movements, proximal cervical (facet) zygapophyseal joint tenderness assessed through manual palpation, with complaint of symptom regularity, greater than or equal to one occurrence monthly.

Exclusion criteria included medically explained headache syndrome rationale including: 1) hypertension (known uncontrolled HTN or blood pressure reading ≥ either 130 mmHg systolic/80 mmHg diastolic or both), 2) cancer-related chemotherapy, 3) post-cerebral vascular accident/TBI, 4) recent concussion, 5) any condition that would contraindicate cervical palpation or range of motion assessment such as Chiari malformation, 6) known vertebral/carotid artery pathology, 7) cervical vertebral fracture or known instability, and/or 8) chiropractic or manual physical therapy treatment in the past year for an associated diagnosis [51].

Procedures

Upon voluntary acceptance of study participation

1. Participants completed a CGH diagnostic questionnaire with question clarification assistance provided by tester [7,15].
2. Participant demographic data were collected [age (years), gender, height (cm), body mass (kg), blood pressure, and body mass index (BMI) ($kg \cdot m^{-2}$)]. Height and weight (mass) were assessed using a medical scale (Detecto, Webb City, MO).
3. Palpation: Participants' cervical spine were evaluated through standard cervical spine palpation methodology [52].
4. Postural Inspection: Cervical, thoracic, and lumbar spine posture was observationally analyzed as per a standard physical therapy inspection/examination [52].
5. Cervical Flexion-Rotation Test: This test of upper cervical (neck) dysfunction [29] was performed while the participant was relaxed in the supine position on a standard physical therapy treatment table. The participant's cervical spine was passively and maximally flexed. The participant's head was passively rotated to the left and the right with end range of motion determined via firm end feel or the onset of pain, which ever occurred first. Range of motion was assessed in each direction through digital goniometry using the EasyAngle (Meloq AB, Stockholm, Sweden). Asymmetrical range of motion greater than or equal to 10 degrees has been considered clinically significant and indicative of C_1-C_2 (facet) zygapophyseal pathology [29]. Please see **Figure 1** for flexion-rotation test image.
6. Leg Length Assessment: Leg length was determined using a common clinical technique [53]. Each participant was supine lying on a standard physical therapy treatment table. The examiner gently grasped and elevated (approximately four inches in height from the table)

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Figure 1. Flexion-Rotation Test.

the participant's ankles and gently applied a distraction force (traction) to the lumbo-pelvic region until tissue resistance to traction was encountered (referred to as "taking up the slack"). The participant's feet were then slowly lowered to the table and the participant was instructed not to move and remain relaxed. A standard tape measure was used to assess the length from each participant's pubic symphysis to the medial malleolus bilaterally (for both legs). The participant securely held the static portion of the tape measure to his/her pubic symphysis, meanwhile, the examiner approximated the dynamic end of the tape measure to the most prominent aspect of the participant's medial malleolus and recorded this measure. Each measurement was taken twice and recorded for each measure for each leg.

7. Pearson's product-moment coefficient of correlation (r) descriptors were derived from Hinkle et al.'s Rule of Thumb for Interpreting the Size of a Correlation Coefficient [**54**], referenced as the following descriptors:
   - 90 to 1.00 (-.90 to –1.00) Very high positive (negative) correlation
   - 70 to .90 (-.70 to -.90) High positive (negative) correlation
   - 50 to .70 (-.50 to -.70) Moderate positive (negative) correlation
   - 30 to .50 (-.30 to -.50) Low positive (negative) correlation
   - 00 to .30 (.00 to -.30) Little if any correlation

Statistical Analysis

Pearson's Product-Moment Coefficient of Correlation (r) was calculated to describe the relationship between leg length discrepancy (LLD) ≥20 mm and CGH diagnostic criteria which included; flexion-rotation test, flexion-rotation test side-to-side difference, CGH diagnostic questionnaire, additional CGH diagnostic criteria, and headache frequency. Coefficient of Determination (r^2) was calculated to analyze the proportion of variation in each CGH diagnostic criteria that was predicted by LLD ≥20 mm . Linearity of data was determined from scatter diagram analysis. Two-tailed t-tests were performed to determine differences between participants with positive CGH diagnoses and those participants with negative CGH diagnoses for variables that included LLD, headache frequency, and flexion-rotation test side-to-side differences. A single-factor analysis of variance (ANOVA) was calculated using IBM SPSS Statistics (version 21, IBM Corp. Armonk, New York, USA) to attain correlation data regarding; LLD and CGH questionnaire, LLD and flexion-rotation test side-to-side difference, LLD and flexion-rotation test diagnosis, LLD and HA frequency, flexion-rotation test and HA frequency, and LLD and additional CGH diagnostic criteria. Regression was used to assess the relationship between LLD, flexion-rotation test diagnosis, and flexion-rotation test side-to-side difference. Statistical significance was determined at an alpha value of less than 0.05 ($\alpha<0.05$).

Results

Thirty-seven (37) volunteers met the study's inclusion criteria and successfully completed the testing protocol. See **Table 1** for participant characteristics.

Twenty-three of thirty-seven participants who met the study's inclusion criteria demonstrated a positive CGH diagnosis based on CGH diagnostic parameters [**6**,**15**,**29**]. Pearson Product-Moment Coefficient of Correlation (r) and Coefficient of Determination (r^2) described the relationship and analyzed the proportion of variation in CGH diagnostic criteria between participants with LLD >20mm and 1) the diagnostic flexion-rotation test (r=1.00 p=2.22E-16 r^2=1.00), 2) CGH questionnaire score (r=0.91 p=1.52E-12 r^2=0.84), 3) additional diagnostic criteria [**24**,**25**] (r=0.79 p=1.68E-07 r^2 = 0.63), 4) flexion-rotation test side-to-side difference (r=0.72 p=7.69E-06 r^2=0.52), and 5) headache frequency (r=0.56 p=0.001 r^2=0.32). Please see **Table 2** for Pearson Product-Moment Coefficient of Correlation and

Table 1. Participant Characteristics.

Characteristics	Female	Male	Female/Male Combined
Mean Age (Years)	32.4±16.5	37.1±14.2	34.4±16.3
Height (cm)	162.3±7.4	180.0±6.9	167.1±10.2
Weight (lbs.)	134.6±15.3	179.8±18.5	147.4±26.2
BMI ($kg•m^{-2}$)	23.2±3.3	25.2±2.7	23.8±3.2

Participant data presented as mean ± standard deviation

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Table 2. Pearson's Product Moment Correlation Coefficients (r) with Coefficient of Determination (r^2) Between Leg Length Discrepancy >20mm and Cervicogenic Headache Diagnostic Variables.

Diagnostic Variables	Leg Length Discrepancy >20mm		
	r	p	r^2
Flexion-Rotation Test	r=1	p=2.22E-16	r^2=1
CGH Diagnostic Questionnaire	r=0.91	p=1.52E-12	r^2=0.84
Additional Diagnostic Criteria	r=0.79	p=1.68E-7	r^2=0.63
Flexion-Rotation Test Side To-Side Difference	r=0.72	p=7.69E-06	r^2=0.52
Headache Frequency	r=0.56	p=0.001	r^2=0.32

Coefficient of Determination data. Multiple regression analysis was performed between participants with LLD ≥20mm, the flexion rotation diagnostic test, and flexion rotation test side-to-side difference (r=0.92 p=2.23E-11).

A two-sample T-test compared participant research variables between participants who met positive CGH diagnostic criteria (positive group) and those who did not meet CGH diagnostic criteria (negative group). See **Table 3** for comparative data. Comparison of LLD mean ($\bar{x}$) using a two-sample T-test indicated a significant difference between the negative ($\bar{x}$=7.49, SD ±5.70 mm) and positive ($\bar{x}$=32.82, SD ±6.19 mm) CGH diagnosis groups (p=000000000216). Comparison of side-to-side flexion-rotation test difference mean using a two-sample T-test indicated a significant difference between negative ($\bar{x}$=3.43, SD ±1.90 degrees) and positive ($\bar{x}$=27.74, SD ±9.09 degrees) CGH diagnostic groups (p=000000149). Comparison of headache frequency mean using a two-sample T-test indicated a significant difference between the negative ($\bar{x}$=0.42, SD ±0.72 days per week) and the positive ($\bar{x}$=3.34, SD ±2.45 days per week) CGH diagnostic groups (p=0.004). Please see **Table 3** for cervicogenic headache criteria comparison between participants with positive and negative CGH diagnoses.

Discussion

Headache is a common disorder experienced by more than half of the adult population each year in the United States [1] and is one of the most common causes of chronic pain [2]. Greater than 3% of the US adult population suffered headaches daily and approximately 36% experienced multiple headaches each month [1]. Headache has been commonly under-diagnosed, under-estimated, and under-treated due, in-part, to ambiguous, over-lapping diagnostic criteria [1,6,7]. Cervicogenic headache is a secondary headache classification that originates from pathological degeneration of cervical spine structures [11,24,26]. The physiological etiology of cervicogenic headache is multi-factorial, yet prevalent theories have suggested excessive cervical facet loading and dysfunction with resultant inflammatory cytokine expression which yielded neural activation along occipital nerve pain afferents and/or the trigeminocervical nucleus [11,18,20-23,27]. Unilateral cervical facet pathological degeneration was most notable with CGH [42].

The primary purpose of this study was to assess if a relationship existed between cervicogenic headache and leg length discrepancy ≥20 mm, although this study did not address CGH causation. This study's secondary purposes were to analyze the proportion of variation in the dependent, diagnostic variables that were predicted by LLD ≥20 mm, to provide insight regarding CGH etiology, lend to appropriate examination and treatment parameters, and to compare study variables between participants who presented with positive and negative CGH diagnostic criteria. The results from the current study supported the hypothesis that individuals who positively fulfilled CGH diagnostic criteria delineated by Sjaastad et al. [15] and Fishbain et al. [7] demonstrated a positive and significant LLD ≥ 20 mm [45,46]. A very high positive correlation [54] was demonstrated between participants who demonstrated LLD ≥20 mm and fulfilled positive CGH diagnostic criteria including CGH Questionnaire (r=0.91 p=1.52E-12) with 84% of the variability in the CGH Questionnaire explained by LLD ≥20 mm. A high positive correlation [54] was demonstrated between participants with LLD ≥ 20mm and additional diagnostic criteria that included neck, shoulder, and/or ipsilateral arm pain, associated nausea, light provocation, sound provocation, associated dizziness, associated blurred vision, difficulty swallowing, fascial warmth, and/or neck warmth (r=0.79 p=1.68E-07) with 63% of the variability in additional diagnostic criteria explained by LLD ≥ 20 mm, and participants who fulfilled positive CGH diagnostic criteria including flexion-rotation test side-to-side difference

Table 3. Cervicogenic Headache Criteria Comparison Between Positive Diagnosis and Negative Diagnosis Participants.

	Positive CGH Diagnosis Group	Negative CGH Diagnosis Group	Statistical Difference
LLD Difference	32.82±6.19mm	7.49±5.70mm	P=2.16E10
Flexion-Rotation Test Side-To-Side Difference	27.74°±9.09 °	3.43°±1.90°	P=1.49E-07
Headache Frequency	3.34±2.45 days/week	0.42±0.72 days/week	P=0.004

Participant data presented as mean ± standard deviation.

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(r=0.72 p=7.69E-06), with LLD ≥20 mm representing 52% of the variability in flexion-rotation test side-to-side difference. A moderate positive correlation [54] was demonstrated between participants who demonstrated LLD ≥20 mm and headache frequency (r=0.56 p=0.001), however LLD ≥20 mm only explained 32% of the variability in participant's headache frequency. The flexion-rotation test served as the primary CGH diagnostic measure in participants who demonstrated positive CGH screening criteria to distinguish CGH from other headache classifications as the flexion-rotation test has been clinically utilized as a CGH diagnostic tool with sensitivity of 90% [29] and 91% [30], specificity of 89% [29] and 90% [30], and diagnostic accuracy of 89% [29]. A very high positive correlation [54] (r=1.00 p=2.22E-16) was demonstrated between participants with a LLD >20 mm and a positive flexion-rotation test with variability in the flexion-rotation test completely explained by LLD ≥20 mm. Linetsky et al. [20] reported LLD on clinical presentation in individuals with CGH, yet no studies to date had directly explored a relationship between LLD of a significant nature [45,46] and individuals with a positive CGH diagnosis.

CGH has been strongly associated with cervical spine pathology of various etiology often with unremarkable, insidious onset [18,20-23,27,36]. Several studies have reported on clinically relevant CGH risk factors including hypomobile cervical spine range of motion and altered cervical spine posture [6,10]. Cervical spine postural anomalies have been identified as potential provocative factors associated with cervical spine pathological degeneration [41-43], yet the etiological rationale for the associated postural anomalies present in CGH and the asymmetric cervical facet load have not been sufficiently explained or explored. Cervical spine provocative postural and/or movement anomalies commonly appeared in individuals with CGH [11,16,27]. Cervical spine abnormal compressive loading from abnormal postural alignment has been shown to result in pathological degenerative facet joint changes [13,18,20-22,36]. Cervical spine postural anomalies have been linked to inferior structural kinetic chain asymmetries [7,20,34-36,38-40]. Betsch et al. [46] demonstrated LLD of ≥20 mm resulted in compensatory pelvic asymmetry/obliquity with resultant frontal plane compensatory spinal deviations, presented as functional scoliosis. Abnormal lumbar posture, such as scoliotic deformities, has been associated with cervical spine mechanical abnormalities with improved CGH outcomes demonstrated in individuals with correction of those lumbar spine postural impairments [37-39]. LLD has been directly associated with pelvic obliquity and scoliotic spinal deformities [35,46,47]. Timgren & Soinila [44] reported pelvic asymmetry resulted in atlanto-occipital unilateral compressive dysfunction based on a unilateral narrowed gap between the mastoid process and transverse process of the atlas in study participants. This cervical posture is indicative of lateral cervical flexion with contralateral, compensatory head posturing. This abnormal cervical-head posture would lead to increased unilateral cervical facet loading which has been associated with pathological degenerative facet joint changes [13,18,20-22,36]. Pathological degenerative facet joint changes of the upper cervical spine, specifically C_2-C_3, have demonstrated the strongest association with CGH [13]. The current study found a positive high correlation [54] between participants who presented with CGH and demonstrated LLD >20 mm. LLD >20 mm has been found to result in pelvic asymmetries which alter lumber frontal plane posture resulting in lumbar side bending, found to result in compensatory cervical spine side bending, and to alter cervical spine facet joint loading. A hallmark finding with CGH is symptom unilaterality [15], indicative of provocative unilateral cervical facet joint degenerative changes. Forward head posture, often associated with CGH [37,38] would yield bilateral cervical facet degeneration and symptom bilaterality. LLD >20 mm has been shown to yield asymmetric proximal cervical spine facet joint loading due to asymmetric frontal plane cervical spine posture which has been shown to increase unilateral cervical facet loading and degenerative changes with cytokine expression [18,44].

Fourteen of thirty-seven participants with chronic headache presentation did not meet the CGH diagnostic criteria (negative CGH group) [15]. The negative CGH group reported symptom presentation resembling tension-type headache and/or migraine [1]. A significant difference in leg length discrepancy (p=000000000216), side-to-side flexion-rotation test (p=000000149), and headache frequency (p=0.004) was demonstrated between positive and negative CGH diagnostic group participants. Participants in the negative CGH diagnostic group did not achieve the 20 mm LLD threshold required for compensatory posturing of the pelvis, lumbar, thoracic, and cervical spine. The difference in right and left cervical rotation upon the flexion-rotation test was used as a measure of C_1-C_2 facet pathological degeneration [29,30]. LLD ≥20 mm, as present in the positive CGH diagnostic group, has shown compensatory, asymmetrical frontal plane spine posture, which has been found to result in asymmetrical proximal cervical facet load, has been shown to perpetuate associated facet degeneration, and has demonstrated a strong association with CGH [13].

Several factors may have limited the generalizability of the current study's results including urban/sub-urban participant population, a limited number of participants primary due to covid-19 restrictions, participant honesty when completing the CGH questionnaire, and despite methodological controls built into this research design, a degree of tester subjectivity should be considered. A future direction for this research line should investigate a causative relationship between CGH and LLD ≥20 mm.

Conclusion

A significant association was found between participants who positively met diagnostic criteria for cervicogenic headache and leg length discrepancy ≥20 mm, significantly different from participants with frequent headache who did not meet

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the diagnostic criteria for cervicogenic headache. Significant differences were found between participants with frequent headache who met and those who did not meet CGH diagnostic criteria regarding leg length discrepancy ≥ 20 mm, headache frequency, and side-to-side flexion rotation test difference, indicative of cervical facet degeneration. Cervicogenic headache is a secondary classification of headache whose etiology is derived from cervical spine pathology. Treatment for cervicogenic headache has focused on mediation of cervical spine pathology with inattention for the etiology of unilateral symptom presentation. Cervicogenic headache treatment outcomes have been effective regarding symptom mediation, yet fleeting. Leg length discrepancy ≥20mm should be considered in the examination and treatment regarding cervical spine pathological degeneration as found to be responsible for the presentation of cervicogenic headache.

List of abbreviations

ANOVA: Analysis of Variance
BMI: Body mass index
C: Cervical
CGH: Cervicogenic headache
cm: Centimeter
HTN: Hypertension
IL-1β: Interleukin 1 beta
IRB: Institutional review board
kg: Kilogram
$kg \cdot m^{-2}$: Kilograms per meters squared
mm: Millimeter
QOL: Quality of life
ROM: Range of motion
SD: Standard deviation
TNF-α: Tumor necrosis factor alpha
US: United States
USA: United States of America
$\bar{x}$: Statistical mean

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

Authors' contributions	MR	RG
Research concept and design	√	√
Collection and/or assembly of data	√	--
Data analysis and interpretation	√	√
Writing the article	√	--
Critical revision of the article	√	--
Final approval of article	√	√

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