RESTORATION OF BAREFOOT GAIT IN A 75-YEAR OLD FEMALE WITH CERVICAL SPONDYLOTIC MYELOPATHY: A CASE REPORT UTILIZING CHIROPRACTIC BIOPHYSICS (CBP®) TECHNIQUE

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ABSTRACT

Objective: To present a case of the restoration of barefoot gait in a patient with cervical spondylotic myelopathy (CSM), balance issues with an inability to barefoot walk, neck pain, and poor cervical spinal posture utilizing Chiropractic BioPhysics (CBP®) technique.

Clinical Features: A 75-year old female patient could not walk barefooted and had fallen frequently. She reported neck pain, decreased flexibility and numbness down her arms, legs and feet. She had been previously diagnosed with CSM. Digital radiography showed cervical hypolordosis and forward head position with spondylosis consistent with CSM.

Intervention & Outcome: The patient received CBP® care including mirror-image cervical extension exercises, prone drop-table adjustments, and cervical extension traction. At the 26th treatment she reported no neck pain or numbness, and could walk barefoot. Cervical x-ray showed marked improvement in posture.

Conclusion: We believe that increasing the cervical lordosis by extension traction decreases cord tension by shortening canal length, relaxing the pons-cord tissue tract and releasing its intermittent impingement upon the spondylotic ridges that caused the neurological symptoms in this patient with CSM. (Chiropr J Australia 2017;45:16-27)

Key Indexing Terms: Chiropractic; Cervical Spine

INTRODUCTION

Cervical spondylotic myelopathy (CSM) is a neurologic entity consisting of cervical cord symptoms and signs, with or without either brachialgia or local neck pain, in association with degenerative or spondylotic changes of the cervical spine (1-3). It is estimated that, at a minimum, the incidence of CSM is 41/1,000,000, and its prevalence is 605/1,000,000 (4).

Radiographic abnormalities can include elements of spinal canal narrowing by osteophytes and by vertebral subluxation, as well as alterations in spinal curvature resulting from degenerative changes of the intervertebral discs and joints and of ligamentous and muscle tissue. Overall, CSM is the most common cause of spinal cord dysfunction (5). There is evidence to suggest that 20-60% of patients will deteriorate neurologically without surgical intervention (6).

Since there is a paucity of evidence for nonoperative treatment of CSM (7), and because of its progressive nature, it is considered a surgical disorder (8). Surgical approaches to CSM typically involve decompression (5,9). Decompressive procedures include laminectomy or anterior procedures of osteophyte removal with or without fusion – occasionally, both are used (10,11). Post-surgical late deterioration is also seen after decompression operation (12,13).

The actual pathobiology of symptom production in CSM is poorly understood (5). Myelopathy is thought to result from static compression, spinal malalignment leading to altered cord tension and vascular supply, and dynamic injury mechanisms (4). Regarding surgical outcomes, Batzdorf et al. (14) determined that the best surgical outcomes in terms of improvement in signs and symptoms in patients getting decompression surgery are seen in the patients with "relatively normal curvature." Similarly, better surgical outcomes have been noted in patients having lordotic cervical curvature post-surgery (15).

We hypothesize that restoration of the cervical lordosis, by Chiropractic Biophysics (CBP®) procedures in patients with loss of lordosis and CSM would result in a favorable outcome due to the relationship of the spinal cord to the cervical spine posture. This case discusses the successful outcome in a 75-year old female patient suffering from CSM and corresponding balance issues and a lack of ability to perform barefoot gait as treated using CBP® technique.

CASE REPORT

Clinical Features

A 75-year-old female sought care for a primary complaint of balance issues and a lack of ability to barefoot walk. She was originally only seeking orthopedic shoes. After consultation it was felt she needed a complete check-up including x-rays of her spine.

Her reason for seeking custom shoes was because that was the only way she could walk effectively; by wearing shoes that were heel-raised she was able to walk freely. Without the raised heel she lost balance; being unstable, she had fallen repeatedly over the last 5 years. She had a great fear of falling as she had fallen several times, hurting herself, and had broken a finger and sprained a wrist. This unsteady gait was only present while barefoot.

She also complained about neck pain, reporting it to vary between a 3-6/10 on the numeric rating scale (NRS: 0=no pain; 10=bed-ridden) and scoring a 42% on the Neck Disability Index (NDI) (16). Visual rating of range of motion demonstrated decreased neck flexibility in all directions. She was unable to heel

walk, she had a positive Romberg's sign, and multiple sensory deficits to soft touch along dermatomes on the legs and arms. She had not driven a car in 3 years because of a lack of confidence, and had numbness down her arms and legs as well as tingling on the bottoms of her feet, bilaterally.

She had seen multiple healthcare providers including a medical doctor, a neurologist and a physiotherapist over the last year with no change in her symptoms. She felt her condition had worsened over the last year. The neurologist had confirmed the diagnosis of CSM. The only improvement to her balance and gait ability had been with the use of shoes having a thicker heel with a downward-sloping orientation that she relied on wearing constantly, including indoors.

Radiographic Exam

Cervical and lumbar radiographic series were taken. The x-rays were analyzed by the posterior tangent method, which has been determined to be reliable and repeatable (as has patient positioning), and has a standard error of measure <2° (17-20).

Analysis of cervical x-rays (Figure 1) revealed spondylosis, consistent with CSM from C3-T1, forward head posture (FHP) of 35mm (normal = 0-15mm (21,22)), reduced cervical lordosis absolute rotation angle (ARA C2-C7) of -22.4° (normal = 31-42° (21-23)), and reduced atlas plane line (APL) of -17.8° (normal = -24- -29° (21,22)). Lumbar spine revealed a slight hypolordotic lumbar curve (ARA L1-L5) of -35° (normal = -40° (24,25)), and sacral base angle of -35° (normal = -40°(24)) with a posterior thoracic cage translation of 34mm (normal = 0mm).

Intervention and Outcome

The patient elected to start care. Initially she received instrument adjusting throughout the paraspinal muscles, prone cervical spine mirror-image extension drop table adjustments, heat before treatment, and ice after treatment. On the 4th treatment and thereafter, she stood on a PowerPlate® for 1-minute while performing moderate level knee squats while hanging on for support not going beyond a 90° knee bend. On the seventh treatment and thereafter, she performed cervical extension traction as per CBP® technique (26-35), on the PosturePump® (Figure 2).

Initially the patient was treated on a 3 times per week basis as per CBP protocol (26-27). Upon each visit, she filled out a numerical rating scale (NRS= 0-10) for pain in the neck as well as had her posture visually checked. By the beginning of the twelfth treatment (7/16/12) she reported a 0/10 for neck pain where it remained a 0/10 for the remainder of her treatments.

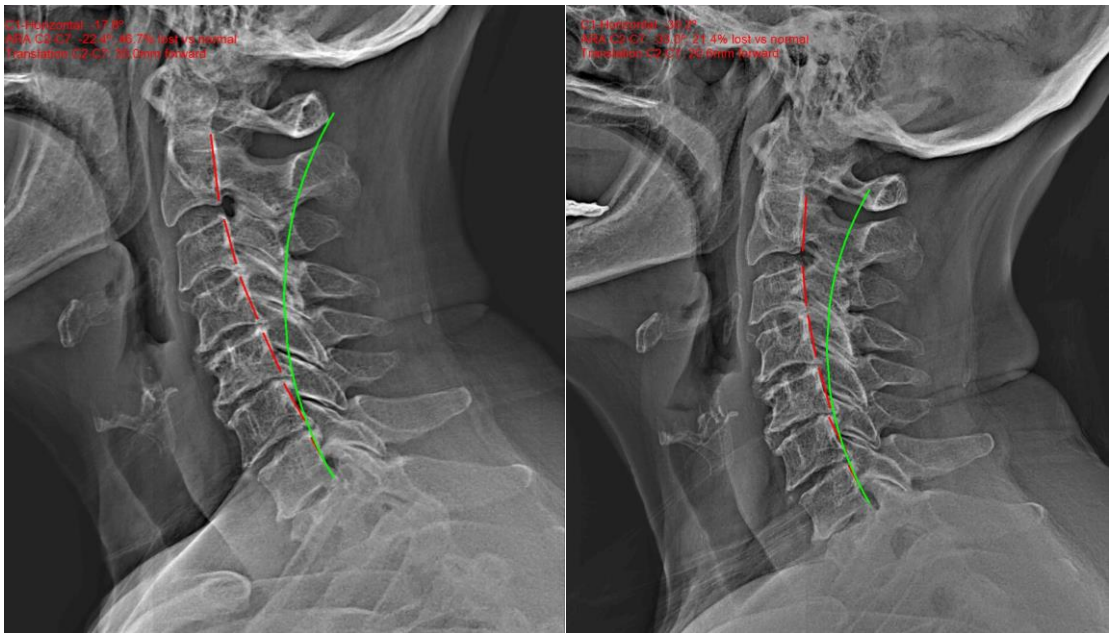

Figure 1. Lateral cervical radiograph views in standing neutral position. Left: Initial view (6/6/2012) demonstrating 35mm forward head posture (FHP), absolute rotation angle (ARA C2-C7) of -22°, and an atlas plane line (APL) of -18°; Right: 10.5-week follow-up (8/23/12) showing reduction of FHP (21mm), increase in cervical lordosis (ARAC2-C7=-33°), and increased APL (-30°).

Figure 2. Cervical extension traction via PosturePump®. The patient's neck is placed over an air bladder, where the patient is required to squeezing a bulb to inflate and hyper-extend their neck. She worked up to 10-minutes as maximally inflated as possible over the 19 treatments that included that procedure before x-ray follow-up (visits 7-25).

Prior to the 26th treatment the patient filled out a follow-up NDI, where she scored a 0%. She also reported no neck pain, had increased neck flexibility in all directions, and reported her barefoot gait had improved dramatically, she was able to walk freely and at will without her orthopedic shoes. She reported that she had no more numbness down her arms and legs and that she had admitted to be confident enough and drove her car for the first time in 3 years. She was able to heel walk; all orthopedic tests were normal.

At this time the patient also had a follow-up lateral cervical x-ray to assess changes expected from cervical extension traction (26-35) (Figure 1, right). Mensuration demonstrated improvements as compared to the initial view taken 10.5 weeks earlier, for FHP (21mm vs. 35mm), ARA C2-C7 (-33° vs. -22°), and APL (-30° vs. -18°).

After this assessment, the patient had initially continued a 3 times per week frequency of treatments to continue postural improvement as would be expected from the initial results, though due to personal reasons choose to go to a 'maintenance' frequency of once per week. This was continued until the last time she came for treatment as she moved away, she refused any sort of exit examination as she reported to be 'very well.' In total, the patient received 44 treatments over almost 5-months.

DISCUSSION

This report documents the successful outcome in a 75-year old patient with cervical spondylotic myelopathy and concomitant poor cervical spine posture. The correction of cervical spine alignment and reduction of forward head posture by CBP® extension traction methods correlated with the improvement in symptoms.

Whenever there are comparative x-rays used for treatment outcome assessment, the issue of repeatability needs to be considered. Lateral cervical spine posture repeatability has been substantiated in the literature (20). The patient was given the instructions: "stand in your normal, natural, comfortable standing position and look straight ahead," prior to both lateral cervical images being taken. We note that the patient had a slight head extension on the follow-up radiograph compared to the initial one prior to treatment. Although every attempt was made to duplicate the patient positioning, this slight difference is likely due to the correction in her posture. More importantly, however, it has been determined that mild head extension only affects the upper cervical spine alignment estimated at less than 5° from C2-4 for a head extension of up to 14° (36). Since there was an 11° improvement in the patient, even if the head position affected the lordosis

(11°-5°=6°), most of the increase in lordosis as measured from the follow-up x-ray is a treatment effect.

There have been 2 other reports documenting treatment of CSM patients by non-surgically, increasing the cervical spine curvature in the chiropractic and physiotherapy literature. McAlpine (1991) reported on the improvement in cervical lordosis in ten patients with CSM (37). There was an average improvement in lordosis of 7.8° after a single treatment using Grostic procedure; that is, with "the Laney hand held adjusting instrument on a Grostic, mastoid support, side posture table." The force was delivered to the atlas transverse process according to its' determined laterality. No information was given, however, as to any symptomatic improvement in the CSM treated group.

Moustafa et al. (2011) in a randomized clinical trial reported on the simultaneous improvement in cervical lordosis in 15 patients with cervical spondylosis and the alleviation of radiculopathy (32,33). The treatment consisted of 30 sessions over a 2.5-month timeframe involving stretching exercises, infrared radiation, as well as a 3-point bending cervical extension traction. The control group consisted of 15 patients treated identically with the exception of not having the cervical spine extension traction. Documentation of VAS, peak to peak amplitude of dermatomal somatosensory evoked potentials, and cervical spine flexion-extension kinematic analysis was done initially, post 10-weeks treatment, and at 3-months follow-up. Both groups had initial symptom reduction at the end of treatment (10 weeks); however, the controls' symptoms returned to baseline, whereas, the treatment group receiving the cervical extension neck traction was the only group to have increase in lordosis and remained significantly symptomatically improved at the 3-month follow-up, as well as at the 2-year follow-up (33).

To understand the plausible rationale for success of the patients having improved lordosis and reduction of neurological symptoms in the Moustafa trial (32,33), as well as our own case, one needs to consider the normal biomechanics of the cervical spine and spinal cord functional anatomy. Normal, non-pathological cervical spine flexion causes an 'unfolding' of the cord tissue, referred to as 'physiologic movement (38).' When there are pathologic changes present in the anatomy of the cervical spinal canal however, as in CSM, then cervical spine flexion will cause 'pathologic movement' of the cord; that is, irritate it by exerting compression, tensile, and/or shearing stresses (39,40).

Panjabi and White (40) explain: "In normal anatomy, these physiological movements of the spinal column do not produce any abnormal stresses and strains in the nervous tissue. In pathologic situations, however, such as hypertrophy of the ligamentum flavum, abnormal disc bulge, osteophytic formations, ossification of posterior longitudinal ligament, and congenital narrow spinal canal, these same functional changes may result in abnormal stresses and strains in the spinal cord that lead to neurological problems."

To understand how treating the cervical spine can affect distant neurologically related sites, it is important to consider the 'histodynamic forces.' Breig explains how the pons-cord tissue tract is a 'self-contained compartment of biomechanics' (41). In the case of tensile stress on the cord, "there is a conductivity blocking concentration of stress within the intact nervous tissue only if this is under tension; neurological symptoms would then be elicited or aggravated when the head and vertebral column are flexed and would be reduced or eliminated on their extension (41)." The cervical spine is most important area of the spine in relaxing the spinal cord, and this has been seen to have effect all the way down to the sacral cone and lumbosacral nerve roots (42).

The exact cause of neurologic deficits in patients with CSM are thought to be a combination of tension in the cord and pathologic conditions (i.e. osteophytic formations) that cause intermittent cord vessel ischemia. Mechanical stresses of cervical flexion to the anterior of the cord over any spondylotic ridge deforms nervous tissue as well as blood vessels, such as the anterior spinal arteries (43). Breig found the lateral columns and anterior horns vessels most affected by mechanical stress produced by spondylotic bars during cervical flexion (43). Others have found that shear forces exert most cord deformation to the central portion (44); this is where the venules are located that happen to be especially vulnerable to mechanical damage (45).

The resulting temporary cord ischemia caused by forced pressure onto spondylotic ridges with cervical flexion need only be present for 10 minutes to produce injury (43). Upon extension the normal vessels lumina are re-established leaving no signs of blockage other than the 'residual parenchymal damage (43).' This process is ever repeated throughout daily cervical spine movements reflecting 'repeated episodes of hypoxia.' Simultaneous with these pathologic cord movements, axonal conduction may be impeded directly, by the over-stretching and/or squeezing to the axons themselves (43). Regardless, whether neurologic deficits result from intermittent blood vessel blockage or impingement of axonal conduction, ultimately, progressive deficits arise when the ability of the tissues to compensate for these repeated insults are exceeded (13).

Breig states that "Many older persons have extensive spondylotic changes with no evidence of myelopathy. This suggests that a variable, such as the number and location of the radicular arteries, may be highly relevant (43)." Another factor in the determination of whether an older person becomes symptomatic or not with cervical spondylosis may be in the presence of cervical spine hypolordosis or kyphosis. Support for this comes from surgical studies that point to the poor cervical spine alignment as significant causative factors in poor neurological outcomes in patients receiving decompression surgery for CSM (9,10,15).

The usual treatment for CSM is for decompression surgery, Biomechanically, if tension is put on to the spinal cord because of additive effects of

hypolordosis/forward head position and simultaneous lengthening of the spinal canal, and cord stress by spondylotic ridges, simple decompression via laminectomy may not cause a significant reduction in cord tension without extension of a hypolordotic/kyphotic spinal curvature (46). This probably explains why the success rate can be poor via surgical decompression approaches. It was pointed out by Aboulker (47), that surgical failure for this condition was ascribed to the development of post-operative cervical kyphosis.

It may be that in the condition of chronic neck flexion as in cervical hypolordosis, our patient's otherwise healthy cord tissue was undergoing relentless pathologic conductivity blocking in especially the neck flexion position from her neutral position as in movements required during normal activities of daily living, such as tying shoes, reading, cooking, cleaning, etc (48).

Increasing the cervical lordosis in patients with CSM will decrease cord tension by shortening the canal length. This would result in clinically relaxing the pons-cord tissue tract enough, past theoretical threshold, to release its impingement upon spondylotic ridges as well as to no longer exceed its ability of natural unfolding and elastic physiologic deformation, as opposed to its formerly pathologic deformation (as with normal head movements with hypolordosis/kyphosis). This is the plausible biomechanical explanation for the dramatic results seen in our patient. Since surgical decompression treatment is not offered until significant spondylotic changes have already occurred, non-surgical approaches such as what was performed on our patient and elsewhere (32-34), may prove useful in the earlier stages of the development of CSM.

Although we present a single case report, the results were consistent with the results obtained in the Moustafa trial results (32-34) and show promise for extension traction for patients with CSM. Although there is established evidence for better surgical outcomes with patients having a more normal cervical lordosis, both prior to (14), as well as after surgery (15), this case adds to the initial evidence (32-34) that this may also be true for non-surgical treatment of CSM.

CONCLUSION

Based on the biomechanical rationale for how cord tension can be reduced by increasing cervical lordotic curvature and reducing forward head posture, it would be prudent to continue study into the treatment of CSM by non-surgical postural cervical lordosis correction methods.

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