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Chiropractic Management of Low Back Pain in a 75-Year-Old Man With Bilateral Developmental Hip Dysplasia

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Abstract

Objective: The purpose of this case report is to describe chiropractic management of an elderly man with untreated bilateral hip joint dysplasia presenting with mild acute mechanical low back pain.

Clinical Features: A 75-year-old man presented with an insidious-onset intermittent low back pain of 3 days' duration. Physical examination findings supported a mechanical cause for mild acute low back pain. Plain radiography revealed dysplasia of hip joints with absence of femoral heads and necks and bilateral high dislocation.

Intervention and Outcome: Chiropractic management included vibration, mobilization, light drop-piece adjustments of the lower lumbar and sacroiliac joints, and recommendation of the use of heat at home. Treatments were given 3 times over the course of 1 week. The low back pain intensity over this period dropped from 5 to 0 on an 11-point numerical rating scale, and the patient was discharged.

Conclusion: This patient with substantial postural and gait abnormalities as a result of severe bilateral hip dysplasia associated with an unusual pattern of osteoarthritic change in the spine responded favorably to a short course of chiropractic care.

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Introduction

Developmental dysplasia of the hip (DDH), also known as *congenital hip dislocation* or *congenital hip dysplasia*, represents a broad range of severities from mild transient neonatal hip instability to total hip dislocation.[1] Dysplasia is an alteration in cell growth resulting in cells that differ in size, shape, and

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appearance producing abnormal growth and development. In developed countries, DDH is encountered in 1 per 100 newborns, making it a commonly diagnosed and treated neonatal condition.[2] This is in contrast to developing countries where untreated cases are common.[3] Developmental dysplasia of the hip was commonly diagnosed by plain radiography or computed tomographic scans in those older than 6 months. More recently, ultrasound imaging has become the preferred method for DDH diagnosis.[1,4]

The normal development of the hip is dependent on proper formation of the triradiate cartilages forming the acetabulum and a spherical femoral head that is well centered. This normal development provides stability to the femoral head in the acetabular fossa and is partly dependent on the absence of abnormal laxity in the hip joint capsule.[5] However, in the presence of hip joint instability, the femoral head can subulxate or dislocate and may even form a pseudoarthrosis.[5] High dislocation of the femoral head and a fibrous pseudoacetabular formation can occur, preventing further superior migration of the femur during weight bearing.[6] In less severe cases of dysplasia that do not involve dislocation of the femoral head, DDH causes hip joint degeneration. It is interesting that acetabular dysplasia was reported to be responsible for approximately 80% of osteoarthritis (OA) of the hip in Japan.[7] Similarly, a recent study of a population-based sample of 842 participants in France has confirmed that even mild acetabular dysplasia is related to hip OA.[8] Risk factors for DDH include family history, female sex, congenital calcaneovalgus foot deformity, breech presentations, and swaddling of infants.[1,9]

It is important to note that untreated DDH, particularly when unilateral, may cause asymmetrical joint loading manifesting as leg length discrepancy (LLD), gait abnormalities, and biomechanical effects such as muscle weakness, abnormal joint movement, and knee and foot disorders.[10] There is evidence suggesting that, without correction, functional impairment due to DDH is common and worsens with age.[10] However, there are instances where cases of untreated dislocation are without notable complications and remain pain free.[11] Generally, in situations of high dislocation due to DDH, the acetabulum is small, is porotic, and may be poorly shaped. The superiorly migrated femoral head is small and dysplastic, and the femoral neck is anteverted.[12]

At present, there is only one other published case series that describes the chiropractic management of low back pain in a patient with hip dysplasia.[13] Therefore, the purpose of this case report is to describe chiropractic management of an elderly man with untreated bilateral hip joint dysplasia presenting with mild acute mechanical low back pain.

Case Report

A 75-year-old retired white man presented with an insidious-onset intermittent low back pain of 3 days' duration. It was described as 5/10 on the numerical rating scale-11 in terms of severity at its worst and was reported as 0 (absent) at best. He felt pain in the midline at the lumbosacral level, which was aggravated by lumbar flexion as when doing up his shoelaces. No radiation or referral of the pain was reported; and the patient denied any paresthesia, numbness, or muscle weakness in the legs. The patient had a history of mild intermittent spinal pain since he was in his thirties lasting no more than a day or two and had not produced any impairment or disability. However, this time, his low back pain had persisted for 3 days and was more severe; hence, he sought chiropractic care. The pain was dull and relieved by rest or heat and aggravated by pulling himself up into the bus that he drove on occasion. He also mentioned that he had had an abnormality with his hip joints since birth. Nevertheless, he reported that his hip condition had not impaired his activities and therefore he had not sought to have it treated. He stated that he had participated in physical activities while growing up without any impairment or pain. In addition, he had worked as an insurance salesman in adulthood (an occupation that required a lot of driving) until retirement. He had no relevant family or medical history, had not suffered any trauma in the past, had not been hospitalized, and was not taking any medications.

The patient was 155 cm tall and weighed 70 kg (body mass index = 29.1). The patient's thighs appeared short, and the gluteal muscles seemed round and bulky. A severe LLD of approximately 45 mm was measured on prone leg length check, with the short leg being on the right side. The patient stood and walked with almost maximal plantar flexion of the right ankle, which compensated for the LLD. Moderate joint stiffness and muscular hypertonicity were found at L4/5 and L5/S1 spinal levels. Lumbar ranges of motion (ROM) were full and only painful at end-ROM in lateral flexion or rotation to either side. Hip ROM on both sides were within the normal range and pain free. Result of orthopedic and neurologic examination of the lumbar spine and lower extremities was unremarkable.

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