Hip Osteoarthritis in Patients with Cerebral Palsy: Pathogenesis, Prevention, and Treatment

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Learning Objectives:

1. The reader will be able to define osteoarthritis.
2. The reader will be able to define cerebral palsy.
3. The reader will understand the pathogenesis of hip osteoarthritis in those with cerebral palsy.
4. The reader will be able to recognize common symptoms of hip osteoarthritis in those with cerebral palsy.
5. The reader will be able to differentiate between conservative and surgical treatment options for hip osteoarthritis in those with cerebral palsy.

Introduction:

Cerebral palsy (CP) and osteoarthritis (OA) are two distinctly different conditions with a unique connection. Many patients with CP will develop secondary complications or conditions such as OA of the hip or other joints.[1,2] Healthcare professionals will benefit from understanding the conditions of CP and OA, the pathogenesis and symptoms of OA, prevention tactics, as well as the conservative and surgical treatments commonly used to treat OA of the hip joint.

CP is a non-progressive neurological disorder[1] characterized by a non-progressive lesion in the immature brain that can cause a permanent movement and/or posture disorder.[2] This condition affects 2–3/1000 live births and is considered to be the most common disabling condition in children.[3] A wide range of conditions and skeletal problems can be associated with CP such as crouched gait, muscular tightness, abnormal bone development, low bone density, impaired vision, and malnutrition.[1] Also, malformation of the acetabulum, hip subluxation and dislocation, elevation of the tibial tubercle, fragmentation of the patella, increased femoral anteversion of the proximal femur, tibial torsion, and differing rates of skeletal maturation can be associated with this disorder.[1] Because CP is a life-long disorder, secondary complications may result. These include decreased range of motion, decreased cardiovascular endurance, muscle weakness, obesity, osteoporosis, chronic pain, fatigue, increased muscle tone, and OA.[1,2,4]

The condition of OA involves destruction of articular cartilage (AC) and is commonly the result of shear stress.[1,5] Every year, more people are affected by this condition. Nearly 27 million Americans were estimated to have OA in 2008, which was

almost 9% of the population at the time. This number is up from 21 million Americans in 1995.[6] More recently in 2012, 52.5 million adults were found to have self-reported doctor-diagnosed arthritis.[7] However, the percentage of people with OA is much greater among those with CP. For those people with CP between the ages of 16 and 84 years of age, 57% are estimated to have OA.[8]

Idiopathic OA Pathogenesis

In a properly functioning joint, AC is able to protect subchondral bone because of its high water content and the collagen ultrastructure of its ground substance.[5,9] Water accounts for between 60 and 80% of the composition of AC.[5,9] This large amount of water results in low permeability and a prolonged time of deformation for this tissue as compared to other tissues that have lower water content. Consider Newton's second law of motion (Force=mass*Δvelocity/Δtime or F=m*Δv/Δt). When permeability is low, Δt is increased which will attenuate contact force and help to protect the subchondral bone.

AC is organized into four layers with collagen fibers of differing shapes and orientations in order to optimize its function.[5,9,10] The outermost superficial zone has elongated chondrocytes with fibers resting parallel to the surface so that it resists shear forces from the roll, spin, and glide of joint motions.[9,10] This layer contains numerous metabolically inactive chondrocytes, thus this layer does not repair itself after injury and subsequently the other layers are exposed to greater shear stress.[9,10] The transitional zone is more metabolically active than the superficial zone, and its collagen fibers are randomly oriented which minimizes the stress concentration between the layers.[9,10] The next two layers are the deep zone and calcified cartilage; they have larger collagen fibers, high proteoglycan content, and low water content. The chondrocytes in these layers are oriented perpendicularly to the AC, or vertically, allowing them to resist compressive and

tensile stresses.[9,10] When force is applied to AC, as when weight is applied through a joint, the meshwork of collagen fibrils and proteoglycans realigns and subsequently undergoes tensile stress as the fibers stretch from the load. With age or degeneration (as can occur with CP), collagen fibers exhibit decreased density and can only withstand a fraction of the load that healthy AC can withstand.[10]

Idiopathic OA is characterized by destruction of AC in joints.[1] Throughout the disease process the AC thins out and eventually wears away as it loses its mechanical integrity.[1] This condition is commonly associated with aging.[1,10] Two common mechanisms that occur with aging can lead to OA; these include subchondral endochondral ossification and surface wear of AC.[1]

Subchondral endochondral ossification is a natural process that occurs in the body with aging.[1,5,9] Joint loading is a chondroprotective mechanism as it creates hydrostatic pressure within the cartilage.[1] A lack of mechanical joint pressure can accelerate subchondral endochondral ossification.[1] As demonstrated in Figure 1, when there is improper or reduced loading of joints, areas of AC will experience low hydrostatic pressure which promotes the endochondral ossification process.[1]

Surface wear of the AC occurs mechanically as forces rip or tear away pieces of AC.[1] This process occurs quickly and can be accelerated when joint surfaces are incongruent and there is resulting shear stress. Degradation of cartilage can also be accelerated by physical damage to the superficial cartilage, blunt impact, and joint laxity further accentuating mechanical destruction of the AC.[1]

Effects of CP on Joint Development and Aging:

CP can alter the typical processes of joint development and aging which predisposes a person with CP to OA.[1] These processes are altered to a greater degree in persons with more severe CP making them prone to OA and causing them to develop OA at a higher rate than those with less severe CP.[1] With diplegic or quadriplegic involvement, as is common with more severe CP, patients present with compromised ambulation, reduced muscular activity, and restricted range of motion.[1] As a result of impaired ambulation, patients are "unable to provide sufficient cyclic loads to different areas of the hip that are necessary to maintain the cartilage."[1] Additionally, abnormal joint loading and hip subluxation and dislocation commonly occur in this population and contribute to the development of OA.[1] Some children with CP will require surgery such as a femoral varus osteotomy to prevent further hip subluxation and dislocation.[11,12] Poor outcomes after such surgeries may result in the development of OA in some children.[11,12]

Carter and Tse present a four step osteoarthritic cascade that outlines how abnormal loading conditions can lead to abnormal joint morphology in patients with CP and how these events can lead to early osteoarthritis.[1] First, the altered muscular activity and restricted range of motion that are common with CP can lead to abnormal joint structure, joint subluxation, and poor coverage of AC on the femoral head.[1] The adductor, internal rotator, and hip-flexor muscles are commonly overactive in people with CP due to spasticity and therefore overpower their antagonistic muscle groups.[1] This causes the lesser trochanter to become the functional axis of the femur and directs the femoral head superolaterally making the joint prone to subluxation.[1,13] Subluxation of the hip causes morphological changes such as deformation of the femoral head with flattening of the

epiphysis on the medial side resulting in a wedge-shaped femoral head [1] After repetitive subluxation and the morphological changes that result, the hip may eventually dislocate.[1]

Next, stress concentrations can result from joint incongruities and can cause mechanical damage to the AC of patients with CP.[1] The acetabular labrum creates this stress concentration and a deep notch in the AC located on the superolateral aspect of the femoral head may result (Fig. 2).[1] Deterioration of the AC around the femoral head can be seen on radiographs or with direct visualization during surgeries or autopsies.[1] Because there is high stress at the superolateral aspect of the femoral head, the other areas (particularly the medial side) of the femoral head experience reduced loading which makes them vulnerable to subchondral endochondral ossification and OA.[1] Further, focal shear stresses are created in the notch which leads to damage of the AC and OA.[1]

Muscles also create abnormal loading conditions as they do not produce sufficient force thereby reducing the contact pressures at the hip joint and leading to thin AC and osteopenia.[1,14] Osteopenia and low bone mass density will lead to further deformation of the femoral head and subchondral bone collapse. Because of this, the joint continues to destroy the AC due to the persisting joint incongruity. In the final step of the osteoarthritic cascade, the thinner cartilage degenerates faster/earlier in life and subchondral bone collapse contributes to the mechanical destruction of the remaining AC.[1]

Symptoms:

Because OA is a common secondary condition associated with CP, it is important to be aware of common symptoms of this disease. Common symptoms of OA can include joint pain, stiffness, swelling, instability, inefficient muscle control, and ligamentous

laxity.[6,15] Joint pain and stiffness are also associated with CP; therefore, it can be difficult to tell if those symptoms are related to CP, OA, or both. Pain and inflammation in joints with movement[1] and progressive loss of functional weight bearing mobility[16] have been identified as symptoms of OA that are commonly present in those with CP.

Prevention:

In addition to monitoring symptoms that may indicate the presence of OA, it is important to take measures to prevent the development of OA in patients with CP.[1,16] Particularly in children, it is important to prevent hip subluxation and dislocation which could lead to morphological changes and stress concentrations as discussed previously.[1,16] After 18 months of age, screening radiographs may be used every 6-12 months to detect subluxation or dysplasia early and to allow for proper treatment.[16] Stretching of the hip flexors and adductors should be done daily in order to prevent contractures. Avoiding contractures of more than 20˚ of hip flexion and more than 45-60˚ of functional hip adduction should be sufficient for preventing hip subluxation.[16] If excessive hip flexion or adduction does occur, Botulinum neurotoxin type A (BoNTA) injections can be used to reduce soft tissue tightness.[16]

By reducing contractures with stretching and BoNTA injections, one is also promoting proper alignment which has been shown to prevent OA in persons with CP.[16] Similarly, spasticity reduction with intrathecal baclofen will improve soft tissue extensibility and allow for better musculoskeletal alignment. Using a night-time hip abduction orthosis also promotes proper alignment and prevents subluxation by positioning the proximal femoral head in a more central location.[16] Taking measures to protect AC throughout the lifespan of someone with CP is also an important component

in the prevention of OA.[5,9] This concept will be discussed further along with treatment strategies.

Treatment Strategies:

Both conservative and surgical options are available for the treatment of OA in patients with CP. Because high contact pressures, high contact forces, repetitive impact loading, and frictional abrasion all destroy AC, conservative treatments should strive to minimize these circumstances.[5,9,10] Because contact force=contact pressure/contact area, anything that aims to decrease contact force will also decrease contact pressure.[5]

Treatments that aim to decrease contact force include shock absorption, weight reduction, and protected weight bearing. Shock absorption can be achieved by providing a patient with shoes or orthotics with increased padding which will help to prolong the time of deformation upon loading.[5,17] Properly fitting shoes or orthotics may also increase the contact area and result in decreased contact force. Similarly, encouraging a patient to walk on a softer surface such as a track or grass will help to prolong the time of deformation and decrease contact force.[5] Strengthening muscles around the hip joint will provide shock absorption and improve strength, reduce pain, and improve physical function.[17] In patients with CP, some muscles such as the hip flexors and adductors may be too tight and causing increased contact force.[2,16] Stretching these muscles will reduce the contact forces in the hip.[2] Gait training (with or without assistive devices) to limit trunk movement or leaning has been shown to reduce hip and knee joint loading and improve shock absorption in children with CP.[18]

Reducing body weight is another way to decrease contact force and can be important for patients with CP that suffer from obesity as a secondary complication.[5,17,19]

When a person has a higher body weight, joints are exposed to greater forces every time they are loaded which leads to a faster rate of AC destruction. Weight loss can help prevent OA from developing as well as reduce the symptoms associated with OA. Evidence shows that reducing body weight by 10% can decrease symptoms of OA symptoms by 28%.[19]

Correcting musculoskeletal malalignments with orthotics may be effective for some patients with CP, unless their malalignments are the result of severe spasticity. If their malalignments can be corrected with orthotics, they will benefit from increased contact area at their joint surfaces which will in turn reduce contact force.[5,17] However, it is important to recognize that musculoskeletal malalignments cannot always be corrected conservatively, especially in those with CP.

For those who are skeletally mature and have joint space incongruity and OA, intra-articular injections of a long-acting steroid and anesthetic can be done under fluoroscopy with an arthrogram. Murphy reports that these injections can provide relief for up to, or possibly more than, six months.[16] These individuals might also benefit from low resistance, high velocity activities such as stationary biking with no resistance or deep water, non-impact running in order to take advantage of fluid-film lubrication to reduce friction within the joint.[5]

Repetitive impact loading can be damaging to AC and should be avoided in those with OA.[5,16,17] Aquatic therapy has been shown to be an effective intervention for reducing pain and improving function in persons with OA.[17] Further, this intervention has been found to improve muscle strength, gait velocity, energy expenditure, endurance, pain, and range of motion in children and adults CP.[2,20,21]

Water has unique physical properties that make aquatic exercise less painful than land-based exercise and ideal for those with CP and OA. The property of buoyancy reduces the effects of gravity, decreases weight bearing and joint loading by decreasing body weight, and can also provide assistance for strengthening activities.[2,19,22,23] Buoyancy also reduces the weight bearing requirements and the amount of trunk control needed for support which is beneficial for patients with CP.[23] Turbulence and hydrostatic pressure create resistance to movement in the water that helps increase intensity and allow for equal muscle strengthening around a joint.[2,22,24] The hydrostatic pressure helps to reduce swelling and contributes to pain reduction.[24] The warm water temperatures can also decrease pain, ease soft tissue contractures, and relieve muscle spasms and fatigue.[25]

Overall, aquatic exercise is an effective alternative to land-based exercise for the those with OA and other common conditions associated with CP such as lower limb joint weakness, increased body weight, and poor balance.[2] It promotes aerobic conditioning and strengthening while maintaining joint integrity, which is key in preventing and limiting damage to AC.[1,2] Some studies have even shown that isokinetic aquatic exercises can provide equal or greater strength benefits when compared to land-based isokinetic exercises.[2] This form of exercise can be a life-long fitness activity that may address some of the secondary conditions associated with CP while also helping to manage the symptoms and progression of OA.[2,17]

If conservative treatment is not effective in managing OA in patients with CP, surgical intervention may be warranted. Procedures such as a vascularized iliac graft, rotational acetabular osteotomy, and a total hip arthroplasty (THA) have been used to

treat OA in persons with CP and have been found to be effective in relieving pain, protecting AC, and slowing the progression of OA.[26–28]

The vascularized iliac graft procedure is used to treat early osteoarthritis in adolescents and young adults with subluxed hips. Evidence shows that this procedure may delay the need for a total joint replacement in this population.[26] The results from a study by Fernandez-Palazzi et al show that this procedure effectively improves pain and acetabular inclination angle.[26] The authors concluded that based on their results, the vascularized iliac graft procedure is the procedure of choice to delay the need for a total joint replacement in young adults with early OA.[26]

The rotational acetabular osteotomy has been used to treat acetabular dysplasia and OA in adults with CP since the 1980s.[27] As discussed earlier, correcting musculoskeletal malalignment is beneficial in protecting AC and preventing OA. Because OA symptoms can take decades to appear, it is important to make corrections in a timely manner before significant damage occurs.[27] After performing rotational acetabular osteotomies on five adults with CP and OA, Nagoya et al reported pain relief and no progression of joint degeneration in all of the subjects up to 10 years after the procedure.[27]

A THA is a common treatment for those with severe hip OA, however such a surgery can be challenging in patients with spasticity.[28,29] In their study, Alosh and colleagues evaluated the effectiveness and safety of THA in patients with upper motor neuron disease, such as those with CP.[28] The results indicated statistically significant improvements in pain, Harris Hip scores, and range of motion after THA. The authors determined that THA may provide pain relief and improved mobility for patients with

CP. Further, they warned that these patients will more be more likely to require hip adductor release and hip flexor lengthening at the time of THA because of their spaticity.[28] Yoon et al retrospectively reviewed five adult CP patients who underwent THA using contemporary ceramic-on-ceramic bearings, a less common approach.[30] Every patient in this study experienced pain relief without a decline in mobility postoperatively which suggests that this specific procedure can effectively treat advanced degenerative hip OA in ambulatory adults with CP.[30]

Other studies report that THA is safe and effective for some individuals with CP, but not always for those with more severe involvement.[16,31] Complications such as hip dislocations and loosening of the implant are rare but can occur as a result of this procedure.[32,33] Sanders et al suggest the use of dual-mobility cup to possible reduce the risk of hip dislocation.[34]

Hip OA is a common condition that occurs in patients with CP.[1,30] Hip related conditions and limitations such as degenerative OA, gait disturbances, and difficulty with perineal hygiene occur in 50 to 75% of adult patients with CP.[30] The condition of CP can alter the typical processes of joint development and aging leading to abnormal joint loading and morphology, decreased range of motion, gait abnormalities, and stress concentrations which may contribute to the development of hip OA in this population.[1] Healthcare providers should be aware of the common symptoms of OA and those unique to patients with CP such as pain and inflammation in joints with movement[1] and progressive loss of functional weight bearing mobility.[16] Practitioners should have a solid understanding of preventionstrategies including preventing hip subluxation and dislocation, reducing soft tissue tightness, preventing contractures, and aquatic

exercise.[2,5,16] Additionally, they should have an understanding of interventions that are supported by evidence for the treatment of OA in patients with CP including decreasing contact force, weight reduction, protected weight bearing, vascularized iliac graft, rotational acetabular osteotomy, and THA.[5,26–28] Understanding of these concepts and strategies will allow for high-quality care for patients with CP and hip OA.

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Appendix 1:

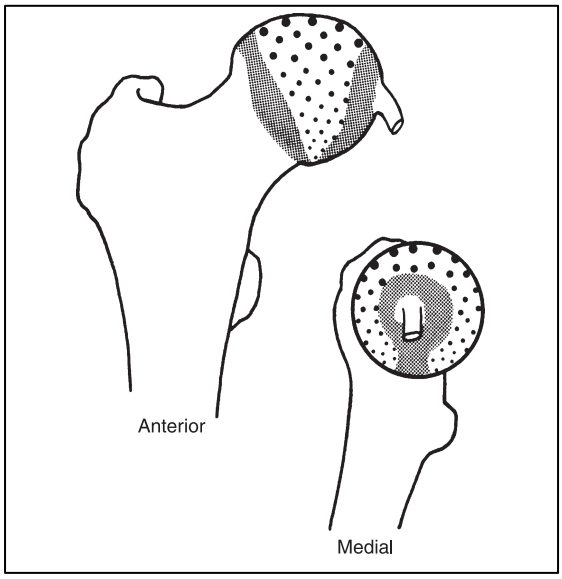

Figure 1: The shaded areas indicate initial degeneration in osteoarthritis. The dotted areas represent when there is surface contact. The larger the dot, the greater and more frequent the contact pressure. Areas with reduced or no loading will experience low hydrostatic pressure and endochondral ossification leading to osteoarthritis. (Source: Carter et al[1]).

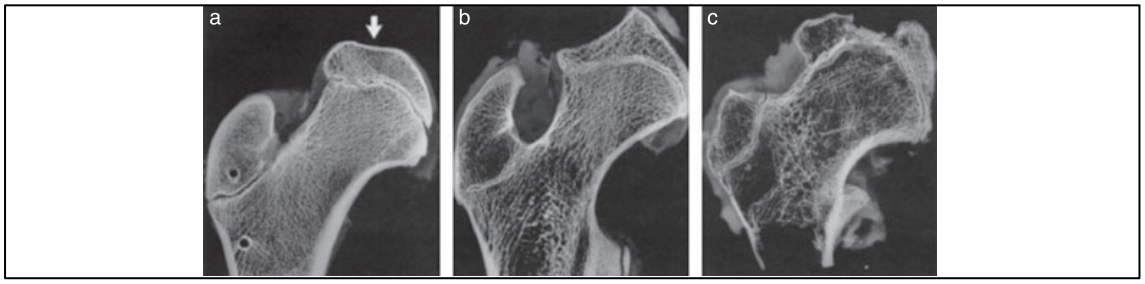

Figure 2: Mechanical destruction of the articular cartilage due to joint incongruities. The progressive stages of degeneration of three femurs cut in the coronal plane as shown by radiographs. In all three cases, the formal head is deformed. (a) Slight cupping, or a notch, is evident in the superolateral aspect of the epiphysis. (b) A deep concave cut and sharp pointed surface on the superior aspect of the lateral femoral head. (c) Extensive articular cartilage destruction and subchondral and trabecular bone collapse shown. (Source: Carter et al[1]).