Case Report

Progressive osteoarthritis during pregnancy several years following hip arthroscopy for femoroacetabular impingement

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ABSTRACT

Case: A 28-year-old with borderline left hip dysplasia who underwent arthroscopic acetabuloplasty, femoral osteochondroplasty, and labral repair for femoroacetabular impingement. She did well for 8 years, though required arthroscopic capsulolabral adhesion release 2 years after the initial procedure. After this period of stability, she developed left hip pain during pregnancy. Radiographs demonstrated progressive osteoarthritis that lead to total hip arthroplasty at age 37.

Conclusion: The physiologic and hormonal changes during pregnancy leading to increased ligamentous laxity may put vulnerable patients with hip dysplasia and iatrogenic instability at increased risk for progression of osteoarthritis.

1. Introduction

One of the primary etiologies of hip osteoarthritis (OA) is thought to be mechanical overload. Joint instability is becoming increasingly evident as a contributor to this process.^{1–4} Hip stability is provided by osseous constraints as well as static and dynamic soft tissues. Due to undercoverage of the femoral head, acetabular dysplasia predisposes a hip to instability and early OA.⁴ In the unstable dysplastic hip, increased shear forces and mechanical edge loading contribute to labral, capsular, and chondral damage.^{4–6} Hip arthroscopy in dysplastic patients is controversial due to the risk of further destabilizing an already shallow and vulnerable joint. Parvizi et al. noted lateral migration of the femoral head in 36% of patients who underwent arthroscopy in the setting of developmental hip dysplasia.⁷ Additionally, hip dysplasia and subsequent instability may be caused iatrogenically from acetabular over-resection. Several case reports of patients with mild to moderate hip dysplasia have shown gross instability and rapid OA progression after hip arthroscopy.^{8–10} Additionally, hormonal changes during pregnancy, including increased relaxin, estrogen and progesterone, increase joint ligamentous laxity through collagenolytic process; increasing motion and potentially contributing to joint instability.^{11–14}

To our knowledge, this case study documents the first reported rapid development of pregnancy related osteoarthritis in a patient who had previously undergone hip arthroscopy and been clinically stable for a number of years. While underlying hip dysplasia and iatrogenic laxity

also attributed to the risk of instability, we believe that the pregnancy caused transient joint laxity, further worsening the instability and subsequent arthritis. The patient provided written informed consent to having her case submitted for publication.

2. Case report

A 28-year-old, highly-active female presented to an outside institution with left hip pain after a skiing accident. Clinical records and radiographs from the outside institution were unavailable. An operative report suggests she failed conservative therapies and she eventually underwent hip arthroscopy to treat femoroacetabular impingement (FAI). The procedure included femoral osteochondroplasty, acetabuloplasty and labral repair. She was also left with an area of focal dysplasia (Fig. 2). Two years later, the patient underwent a revision procedure at the same facility for pain attributed to post-operative capsulolabral adhesions. Intra-operative reports note a well-healed chondrolabral junction, mild chondromalacia at the anterior acetabular rim, fraying at the adjacent labrum, and a large capsulolabral adhesion with adjacent synovitis. In addition to adhesion release, a minor synovectomy, chondroplasty, and labral debridement were performed.

The patient did well for 6 years following her second surgery with only occasional hip discomfort during athletic activities. In 2014, now 36 years-old, she suffered a missed pregnancy at seven weeks gestational age. She presented to our clinic for the first time with several

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Fig. 1. A–B. Pre-pregnancy Radiographs of the Pelvis. AP pelvis and left hip false profile from February 2015. Films were obtained just prior to pregnancy.

months of insidious left hip pain and associated popping and clicking that began during the missed pregnancy. Athletics and rising from sitting worsened the pain. Anterior impingement and FABER tests were positive for pain while axial distraction caused pain and apprehension. Beighton criteria score was 0. Radiographs revealed slight lateral and superior/anterior subluxation of the left hip compared to the right hip. The lateral center edge angle (LCEA) to the sclerotic sourcil measured 2° on the left compared to 27° on the right (Fig. 1). Three-dimensional computed tomography (CT) reconstruction and magnetic resonance

Fig. 2. A–B. Left Hip Computed Tomography with 3D Reconstruction. Left hip CT 3D reconstruction demonstrating the bony anatomy with relative anterosuperior acetabular dysplasia and previous femoral osteoplasty.

arthrogram (MRA) were ordered to evaluate her bony and soft tissue architecture.

Her CT scan confirmed the previous surgical acetabuloplasty and relative dysplasia in the anterosuperior quadrant (Fig. 2). The MRA revealed capsular tissue that did not show any defect or extravasation as well as focal acetabular and femoral cartilage defects (Fig. 3). Prior imaging is unavailable, making the timing and progression of the lesions unclear. Conservative management was pursued, including physical therapy, activity modification and NSAIDs.

The patient was seen again 8 months later, now 28 weeks pregnant, with progressive left hip symptoms despite adherence to conservative measures. Squatting, sitting, and walking all caused left thigh and groin pain. Radiographs were forgone at that visit due to pregnancy. She began pool therapy and underwent intra-articular steroid and hyaluronic acid injections providing 4 months of moderate pain relief.

Five months after giving birth and 15 months after her initial clinic visit, she returned with progressive left hip pain. She had stopped recreational activities and pain was limiting daily life. Radiographs revealed advanced arthritic changes and lateral subluxation of the femoral head (Fig. 4). She was referred to an arthroplasty specialist and subsequently underwent a total hip arthroplasty (THA).

3. Discussion

We report a case of rapidly progressive hip OA during pregnancy in

Fig. 3. A–B. Magnetic Resonance Arthrogram of the Left Hip. MRA left hip, sagittal cut showing intact capsule and focal acetabular and femoral cartilage defects.

a patient with anterosuperior quadrant iatrogenic acetabular dysplasia. The hip showed mild chondral changes and was minimally symptomatic without instability upon presentation, eight years after initial arthroscopy. She initially presented with mild left hip pain after a miscarriage. Her pain continued to progress during her subsequent pregnancy.

Serum concentrations of relaxin, estrogen, and progesterone are all elevated during pregnancy, with relaxin peaking at 12 weeks and estrogen and progesterone at parturition.^{13,15,16} The influence of relaxin, estrogen, and progesterone on ligamentous tissue in the peripartum period is incompletely understood. It is thought that relaxin plays a role in remodeling ligaments via a collagenolytic pathway, thereby increasing laxity.¹⁷ Estrogen and progesterone may bolster the effect of relaxin on collagen through increased relaxin receptor expression.¹⁸ The

Fig. 4. A–B. Post-Pregnancy Radiographs of the Pelvis. AP pelvis and false profile left hip taken after pregnancy demonstrating subluxation and progressive arthritis.

ligamentous changes allow for parturition through increased laxity of pelvic ligaments.¹⁹ Other major joints experience increased laxity during pregnancy.^{1–14,20,21} Increased ACL laxity during pregnancy has been correlated with increased estrogen and progesterone and reports have documented transient increased ACL graft laxity during pregnancy.^{21,22} Additionally, elevated relaxin has been noted in female athletes who sustain ACL tears.²³ While no studies have addressed peripartum hip laxity, ligamentous changes have been hypothesized to contribute to some peripartum labral tears.^{24,25}

Pregnancy is a previously undescribed risk factor for post-arthroscopic hip instability. Matsuda and Khatod reported on a patient who developed hip OA after arthroscopy during pregnancy.² The patient had severe acetabular dysplasia (LCEA 11°) and refused a periacetabular osteotomy so an arthroscopic cam decompression and labral repair were performed. After surgery, she saw 6 weeks of improved pain and function. She became pregnant thereafter and developed progressive

OA and lateral migration of the femoral head, requiring THA within 1 year of her arthroscopy.² The authors believed her course was likely due to the instability of her native hip morphology, without mentioning the potential role of pregnancy-related factors.

Lending to the deep nature and conformity of the femoral head in the acetabulum, the hip is inherently stable. The labrum, capsuloligamentous complex, and surrounding musculotendinous structures add further support.^{1,26–29} In the dysplastic hip, hypoplasia of the acetabular rim leads to increased articular cartilage contact pressures, resulting in labral hypertrophy, myxoid degeneration, and tears.^{5,6} Lateral translation of the femoral head from the loss of normal joint conformity further increases rim shear stresses, predisposing the hip to chondrolabral degeneration and OA.^{5,30–32} The etiologies of hip dysplasia, defined as a LCEA of less than 25°, include developmental dysplasia, Perthes, acetabular retroversion, or iatrogenic.²⁹ Arthroscopic osteochondroplasty or labral debridement may destabilize a hip with mild acetabular dysplasia and cause rapid degenerative changes in the joint.

There have been several reports of progressive OA after arthroscopy in dysplastic patients. Benali and Katthagen reported on a patient with borderline hip dysplasia (LCEA 23°) that underwent debridement of a labral tear and acetabular sided exostosis.⁹ Within 3 months, the patient developed subluxation of the femoral head and severe OA requiring THA. Mei-Dan et al. reported on a patient with an LCEA of 22° who underwent arthroscopic labral repair.¹⁰ The patient also developed subluxation and significant OA within 3 months, requiring a THA at 1 year. They attributed this rapid progression to instability from trimming the ligamentum teres and unrepaired capsulotomy in the setting of borderline dysplasia. Both Parvizi and Ross showed improved outcomes with periacetabular osteotomy in dysplastic patients after failed hip arthroscopy.^{7,33}

Outcomes of hip arthroscopy in mildly dysplastic patients are not universally poor. Six studies showed good results with preservation of the labrum and meticulous capsular repair.^{34–39} The patient in our report had severe focal post-acetabuloplasty dysplasia. We believe that this dysplasia was a contributing factor to the rapid progression of OA, but required secondary destabilization to initiate rapid progression – in this case, becoming pregnant.

Pregnancy increases not only the laxity of the ligamentous structures supporting the hip joint, but also leads to weight gain. This patient gained 10.9 kg (24 lbs.) during her pregnancy which is marginally less than the recommended weight gain of 11.3–15.9 kgs (25–35 lbs.) with a pre-pregnancy body mass index (BMI) of 21.3 kg/m².⁴⁰ Although the forces on the hip joint and soft tissue are increased with the weight of pregnancy, BMI alone has not been conclusively shown to be an independent risk factor for hip OA in women.^{41,42}

While not attributable to pregnancy, other causes of ligamentous laxity have been shown to play a role in hip stability after arthroscopy. Ranawat et al. reported on a patient with generalized ligamentous laxity that suffered an anterior dislocation two months after arthroscopic femoral osteochondroplasty, labral repair, and capsular closure.⁴³ Larson et al. reported on 16 cases with Ehlers-Danlos syndrome.⁴⁴ Soft tissue management was considered critical as 13 had labral repairs and all underwent capsular plication at closure. Only one had continued post-operative instability.⁴⁴

Potentially confounding factors in this report are the intra-articular corticosteroid and hyaluronic acid injections that the patient received during the third trimester of pregnancy. While hyaluronic acid injections have been shown to be safe,⁴⁵ corticosteroid injections are known to cause avascular necrosis of the femoral head⁴⁶ and there is conflicting evidence regarding the risk of chondrolysis and progressive osteoarthritis, as well as limited research specific to the hip. In vitro and in vivo studies of the knee have shown conflicting evidence of the risk of chondrolysis and subsequent worsening osteoarthritis symptoms.^{47–50} A recent randomized controlled study compared intra-articular injections of 40 mg triamcinolone acetonide every 3 months to

normal saline; In the steroid group they found decreased cartilage volume and no improvement of symptoms.⁵¹ While this patient's hip showed no evidence of avascular necrosis, the etiology of her rapidly progressive symptoms could be attributed to her corticosteroid injections. However, the patient's hip was symptomatic for months prior to the hyaluronic acid and corticosteroid injections. Additionally, the worsening symptoms coincided with the lateral subluxation of the hip, that was stable during the 6 years post-op until pregnancy.

This is the first report of a delayed onset, rapid development of hip OA in a pregnant patient who had previously been stable after hip arthroscopy. While there is a clear concern for iatrogenic hip dysplasia secondary to an acetabuloplasty, her hip was clinically asymptomatic for six years. This case highlights the challenging surgical management of the dysplastic hip and suggests that pregnancy induced ligamentous laxity and related hip instability may contribute to the rapid progression of hip osteoarthritis in the setting of an iatrogenic focal hip dysplasia.

Conflict-of-interest statement

None of the authors report a conflict-of-interest related to the submitted work.

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References

- Crawford MJ, Dy CJ, Alexander JW, et al. The 2007 Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. *Clin Orthop Relat Res*. 2007;465(December):16–22.
- Matsuda DK, Khatod M. Rapidly progressive osteoarthritis after arthroscopic labral repair in patients with hip dysplasia. *Arthroscopy*. 2012;28(November (11)):1738–1743.
- Cerezal L, Arnaiz J, Canga A, et al. Emerging topics on the hip: ligamentum teres and hip microinstability. *Eur J Radiol*. 2012;81(December (12)):3745–3754.
- Kalisvaart MM, Safran MR. Microinstability of the hip-it does exist: etiology, diagnosis and treatment. *J Hip Preserv Surg*. 2015;2(July (2)):123–135.
- Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. *J Bone Jt Surg Br*. 1991;73(May (3)):423–429.
- Henak CR, Abraham CL, Anderson AE, et al. Patient-specific analysis of cartilage and labrum mechanics in human hips with acetabular dysplasia. *Osteoarthritis Cartilage*. 2014;22(February (2)):210–217.
- Parvizi J, Bican O, Bender B, et al. Arthroscopy for labral tears in patients with developmental dysplasia of the hip: a cautionary note. *J Arthroplasty*. 2009;24(September (6)):110–113.
- Cuellar A, Ruiz-Iban MA, Marin-Pena O, Cuellar R. Rapid development of osteoarthritis following arthroscopic resection of an "os acetabuli" in a mildly dysplastic hip—a case report: author's reply. *Acta Orthop*. 2015;86(6):760.
- Benali Y, Katthagen BD. Hip subluxation as a complication of arthroscopic debridement. *Arthroscopy*. 2009;25(April (4)):405–407.
- Mei-Dan O, McConkey MO, Brick M. Catastrophic failure of hip arthroscopy due to iatrogenic instability: can partial division of the ligamentum teres and iliofemoral ligament cause subluxation? *Arthroscopy*. 2012;28(March (3)):440–445.
- Aldabe D, Milosavljevic S, Bussey MD. Is pregnancy related pelvic girdle pain associated with altered kinematic, kinetic and motor control of the pelvis? A systematic review. *Eur Spine J*. 2012;21(September (9)):1777–1787.
- Calguneri M, Bird HA, Wright V. Changes in joint laxity occurring during pregnancy. *Ann Rheum Dis*. 1982;41(April (2)):126–128.
- Schauberg CW, Rooney BL, Goldsmith L, Shenton D, Silva PD, Schaper A. Peripheral joint laxity increases in pregnancy but does not correlate with serum relaxin levels. *Am J Obstet Gynecol*. 1996;174(February (2)):667–671.
- Dumas GA, Reid JG. Laxity of knee cruciate ligaments during pregnancy. *J Orthop Sports Phys Ther*. 1997;26(July (1)):2–6.
- Kristiansson P, Svarsudd K, von Schoultz B. Serum relaxin, symphyseal pain, and back pain during pregnancy. *Am J Obstet Gynecol*. 1996;175(November (5)):1342–1347.
- Costanzo LS. *Physiology*. 4th ed. Philadelphia, PA: Saunders/Elsevier; 2010 xi, 493 p. p.
- Dehghan F, Haerian BS, Muniandy S, Yusof A, Dragoo JL, Salleh N. The effect of relaxin on the musculoskeletal system. *Scand J Med Sci Sports*. 2014;24(August (4)):e220–e229.
- Dehghan F, Yusof A, Muniandy S, Salleh N. Estrogen receptor (ER)-alpha, beta and progesterone receptor (PR) mediates changes in relaxin receptor (RXFP1 and RXFP2)

- expression and passive range of motion of rats' knee. *Environ Toxicol Pharmacol*. 2015;40(November (3)):785–791.
19. Marnach ML, Ramin KD, Ramsey PS, Song SW, Stensland JJ, An KN. Characterization of the relationship between joint laxity and maternal hormones in pregnancy. *Obstet Gynecol*. 2003;101(February (2)):331–335.
 20. Aldabe D, Ribeiro DC, Milosavljevic S, Dawn Bussey M. Pregnancy-related pelvic girdle pain and its relationship with relaxin levels during pregnancy: a systematic review. *Eur Spine J*. 2012;21(September (9)):1769–1776.
 21. Blecher AM, Richmond JC. Transient laxity of an anterior cruciate ligament-reconstructed knee related to pregnancy. *Arthroscopy*. 1998;14(January-February (1)):77–79.
 22. Charlton WP, Coslett-Charlton LM, Cicciotti MG. Correlation of estradiol in pregnancy and anterior cruciate ligament laxity. *Clin Orthop Relat Res*. 2001;387(June):165–170.
 23. Dragoo JL, Castillo TN, Braun HJ, Ridley BA, Kennedy AC, Golish SR. Prospective correlation between serum relaxin concentration and anterior cruciate ligament tears among elite collegiate female athletes. *Am J Sports Med*. 2011;39(October (10)):2175–2180.
 24. Baker JF, McGuire CM, Mulhally KJ. Acetabular labral tears following pregnancy. *Acta Orthop Belg*. 2010;76(June (3)):325–328.
 25. Brooks AG, Domb BG. Acetabular labral tear and postpartum hip pain. *Obstet Gynecol*. 2012;120(November (5)):1093–1098.
 26. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. *J Biomech*. 2003;36(February (2)):171–178.
 27. Myers CA, Register BC, Lertwanich P, et al. Role of the acetabular labrum and the iliofemoral ligament in hip stability: an in vitro biplane fluoroscopy study. *Am J Sports Med*. 2011;39(July):85S–91S.
 28. Bayne CO, Stanley R, Simon P, et al. Effect of capsulotomy on hip stability—a consideration during hip arthroscopy. *Am J Orthop (Belle Mead NJ)*. 2014;43(April (4)):160–165.
 29. Shu B, Safran MR. Hip instability: anatomic and clinical considerations of traumatic and atraumatic instability. *Clin Sports Med*. 2011;30(April (2)):349–367.
 30. Chegini S, Beck M, Ferguson SJ. The effects of impingement and dysplasia on stress distributions in the hip joint during sitting and walking: a finite element analysis. *J Orthop Res*. 2009;27(February (2)):195–201.
 31. Philippon MJ. New frontiers in hip arthroscopy: the role of arthroscopic hip labral repair and capsulorrhaphy in the treatment of hip disorders. *Instr Course Lect*. 2006;55:309–316.
 32. Murphy SB, Ganz R, Muller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. *J Bone Jt Surg Am*. 1995;77(July (7)):985–989.
 33. Ross JR, Clohisy JC, Baca G, Sink E, Investigators A. Patient and disease characteristics associated with hip arthroscopy failure in acetabular dysplasia. *J Arthroplasty*. 2014;29(September (9)):160–163.
 34. Uchida S, Utsunomiya H, Mori T, et al. Clinical and radiographic predictors for worsened clinical outcomes after hip arthroscopic labral preservation and capsular closure in developmental dysplasia of the hip. *Am J Sports Med*. 2016;44(January (1)):28–38.
 35. Domb BG, Stake CE, Lindner D, El-Bitar Y, Jackson TJ. Arthroscopic capsular plication and labral preservation in borderline hip dysplasia: two-year clinical outcomes of a surgical approach to a challenging problem. *Am J Sports Med*. 2013;41(November (11)):2591–2598.
 36. Larson CM, Ross JR, Stone RM, et al. Arthroscopic management of dysplastic hip deformities: predictors of Success and failures with comparison to an arthroscopic FAI cohort. *Am J Sports Med*. 2016;44(February (2)):447–453.
 37. Nawabi DH, Degen RM, Fields KG, et al. Outcomes after arthroscopic treatment of femoroacetabular impingement for patients with borderline hip dysplasia. *Am J Sports Med*. 2016;44(April (4)):1017–1023.
 38. Fukui K, Briggs KK, Trindade CA, Philippon MJ. Outcomes after labral repair in patients with femoroacetabular impingement and borderline dysplasia. *Arthroscopy*. 2015;31(December (12)):2371–2379.
 39. Kalore NV, Jiranek WA. Save the torn labrum in hips with borderline acetabular coverage. *Clin Orthop Relat Res*. 2012;470(December (12)):3406–3413.
 40. American College of O, gynecologists. ACOG Committee opinion no. 548: weight gain during pregnancy. *Obstet Gynecol*. 2013;121(January (1)):210–212.
 41. Jacobsen S, Sonne-Holm S. Increased body mass index is a predisposition for treatment by total hip replacement. *Int Orthop*. 2005;29(August (4)):229–234.
 42. Franklin J, Ingvarsson T, Englund M, Lohmander LS. Sex differences in the association between body mass index and total hip or knee joint replacement resulting from osteoarthritis. *Ann Rheum Dis*. 2009;68(April (4)):536–540.
 43. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. *J Bone Jt Surg Am*. 2009;91(January (1)):192–197.
 44. Larson CM, Stone RM, Grossi EF, Giveans MR, Cornelsen GD. Ehlers-Danlos syndrome: arthroscopic management for extreme soft-tissue hip instability. *Arthroscopy*. 2015;31(December (12)):2287–2294.
 45. Strand V, McIntyre LF, Beach WR, Miller LE, Block JE. Safety and efficacy of US-approved viscosupplements for knee osteoarthritis: a systematic review and meta-analysis of randomized, saline-controlled trials. *J Pain Res*. 2015;8:217–228.
 46. Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. *J Bone Jt Surg Am*. 1995;77(March (3)):459–474.
 47. Milano G. Editorial commentary: new perspectives on the intra-articular use of local anesthetics: five weekly injections of 0.5% bupivacaine does not alter articular cartilage. *Arthroscopy*. 2016;32(October (10)):2037–2038.
 48. Iwasaki K, Sudo H, Kasahara Y, et al. Effects of multiple intra-articular injections of 0.5% bupivacaine on Normal and osteoarthritic joints in rats. *Arthroscopy*. 2016;32(October (10)):2026–2036.
 49. Buchko JZ, Gurney-Dunlop T, Shin JJ. Knee chondrolysis by infusion of bupivacaine with epinephrine through an intra-articular pain pump catheter after arthroscopic ACL reconstruction. *Am J Sports Med*. 2015;43(February (2)):337–344.
 50. Gomoll AH, Yanke AB, Kang RW, et al. Long-term effects of bupivacaine on cartilage in a rabbit shoulder model. *Am J Sports Med*. 2009;37(January (1)):72–77.
 51. McAlindon TE, LaValley MP, Harvey WF, Price LL, Driban JB, Zhang M, et al. Effect of intra-articular triamcinolone vs saline on knee cartilage volume and pain in patients with knee osteoarthritis: a randomized clinical trial. *JAMA*. 2017;317(May (19)):1967–1975.