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Diagnosis and Management of Extra-articular Causes of Pain After Total Knee Arthroplasty

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Abstract

Postoperative pain, which has been attributed to poor outcomes after total knee arthroplasty (TKA), remains problematic for many patients. Although the source of TKA pain can often be delineated, establishing a precise diagnosis can be challenging. It is often classified as intra-articular or extra-articular pain, depending on etiology. After intra-articular causes, such as instability, aseptic loosening, infection, or osteolysis, have been ruled out, extra-articular sources of pain should be considered. Physical examination of the other joints may reveal sources of localized knee pain, including diseases of the spine, hip, foot, and ankle. Additional extra-articular pathologies that have potential to instigate pain after TKA include vascular pathologies, tendinitis, bursitis, and iliotibial band friction syndrome. Patients with medical comorbidities, such as metabolic bone disease and psychological illness, may also experience prolonged postoperative pain. By better understanding the diagnosis and treatment options for extra-articular causes of pain after TKA, orthopaedic surgeons may better treat patients with this potentially debilitating complication.

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By 2030, the demand for total knee arthroplasty (TKA) in the United States is expected to quadruple from current levels to nearly 3.5 million procedures annually.[1] Although the safety and efficacy of TKA is well known, postoperative pain remains problematic and has been attributed to most poor outcomes after TKA.[2] Recent reports suggest that 11% to 25% of patients treated with TKA are dissatisfied with their postoperative outcomes, largely because of pain.[3] A study by Puolakka et al[4] found that 36% of TKA patients reported daily pain. Hawker et al[5] reported similar findings in patients treated with TKA at 2 to 7 years postoperatively.

There is a lack of consensus regarding the effect of age and sex on pain after TKA. Although certain studies have cited female sex and younger age as important predictors of postoperative pain, other research, including the 2003 National Institutes of Health consensus statement, reported a lack of correlation between age, sex, and

Table 1
Potential Sources of Postoperative Pain After Total Knee Arthroplasty

Intra-articular	Extra-articular
Infection	Hip disease
Crystalline disease	Foot and ankle disease
Instability	Neurologic/spine disease
Component malalignment	Tendinitis/bursitis
Limb malalignment	Iliotibial band syndrome
Aseptic loosening	Psychological factors
Soft-tissue irritation	Heterotopic ossification
Component wear	Connective tissue disorders
Osteolysis	Material hypersensitivity
Extensor mechanism pathology	Unexplained pain
Recurrent hemarthrosis	

pain after TKA.[6-14] Younger patients are known to have greater expectations for improved performance after TKA; however, the effects of these expectations and higher postoperative activity levels and the lower expectations of older patients remain unclear.[2]

Regardless of a patient's risk factors, the diagnosis of an aseptic, painful TKA may be confounded by a myriad of potential intra- and extra-articular causes (**Table 1**). If preoperative symptoms are not relieved by TKA, extra-articular etiologies should be investigated. Ruling out infection should be the first step in diagnosing the source of postoperative pain after TKA, and the clinical practice guidelines on this topic from the American Academy of Orthopaedic Surgeons can help direct the evaluation.[15] Obtaining a thorough history may aid in distinguishing between intra- and extra-articular pain sources, because a lack of pain relief immediately after TKA could suggest that the initial pain was not attributable to knee pathology. Additional symptoms, including delayed onset of postoperative pain, a sharp catching pain during movement, joint instability, or an abnormal radiographic finding, may indicate intra-articular causes. An intra-articular injection of a local anesthetic also can be used to rule out an intra-articular source of pain.

Hip Disease

Pain can be referred from the hip to the knee via the obturator and femoral nerves originating from L2-L4.[16] Immunohistochemistry staining has been used to demonstrate the location of referred pain from the hip joint, and fluoroscopically guided diagnostic hip injections have been used to map patterns of referred pain to the knee.[16,17] Mechanical knee pain can result from increased knee torque resulting from decreased hip rotation or altered gait from various diseases affecting the hip joint. Patients with severe hip osteoarthritis who are awaiting hip arthroplasty may have pain that is localized to the anterior and posterior aspects of the knee. Several studies have demonstrated the association between various

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forms of hip disease, including osteoarthritis, infection, hip fracture, and posterior hip dislocation, with ipsilateral knee pain.[18-21] Other sources of hip disease, such as osteonecrosis, dysplasia, and implant loosening after total hip arthroplasty, can present as knee pain. Stress fractures, including femoral neck, pubic, and subtrochanteric stress fractures, are rare complications after TKA in patients with poor bone quality but can be secondary causes of pain.[22-26] Generally, all patients who present with knee pain should have a physical examination of the ipsilateral hip to screen for pathology. If a painful or decreased range of hip motion is noted after TKA, radiographic studies are recommended.

Foot and Ankle Disease

Malalignment of the hindfoot resulting in excess valgus alignment of the heel secondary to posterior tibial tendon insufficiency or pes planus increases stress forces on the knee.[27] This hindfoot valgus leads to increased lateral loading of the knee because of changes in ground reactive forces and an increased valgus thrust.[28] Bhave et al[29] reported that patients with planovalgus malalignment had substantial postoperative pain relief with shoe modifications. In addition to a physical examination, screening for malalignment of the hindfoot and ankle also can be performed during radiographic measurement of the weight-bearing axis. Numerous studies have noted the efficacy of including the hindfoot distal to the ankle and subtalar joint in an assessment of the ground mechanical axis deviation.[27,28,30,31] A comprehensive radiographic evaluation of the rearfoot, ankle, and leg taken in multiple planes and angles may be appropriate before TKA in select patients and can be useful in ruling out foot and ankle-related etiologies in patients with pain after TKA.

Vascular Disease

Vascular diseases, such as lower extremity peripheral artery disease (PAD) or thrombosis, also have potential to contribute to symptoms of leg pain and are common in older patients. Although often useful as indicators for PAD, neither the presence of known risk factors (such as hyperlipidemia, cardiac disease, and diabetes) nor symptoms of claudication are definitive predictors of PAD.[32,33] In a study of 50 consecutive patients referred to an orthopaedic surgeon for leg pain, Bernstein et al[34] noted that 20% had undiagnosed PAD. Assessing for PAD via pulse volume recording or ankle-brachial pulse indexing (an index of <0.9 may indicate PAD) is neither invasive nor costly, especially given the frequency of PAD in patients without a history of trauma or claudication who present with leg pain.[34]

Indirect injury to arteries via compression can occur during TKA as external forces applied to diseased vessels during tourniquet application can lead to vascular damage, plaque disruption, and subsequent thrombus formation. Thrombosis is one of the most common causes of vascular insufficiency after TKA and can be confirmed with Doppler imaging secondary to a positive Homan sign.[35] If an extra-articular source of pain after TKA is suspected, the presence of vascular diseases should be investigated.

Lumbar Spine and Neurologic Disorders

Lumbar spinal stenosis (LSS) has an incidence as high as 8% and is a common cause of leg pain in older adults.[36] Nevertheless, patients with LSS who undergo TKA have been shown to have comparable outcomes to control patients with regard to revision rates and radiographic outcomes despite their lower preoperative functional scores.[37] LSS is often idiopathic and presents as intermittent neurogenic claudication caused by the compression of the cauda equina, which results from enlargement of the bony and soft tissues associated with the spinal canal. In a study of 100 patients, Amundsen et al[38] reported additional common symptoms of back pain, leg pain, weakness, and voiding disturbances. Double nerve root involvement is typical, and the L5 nerve root is the most commonly affected (prevalence of 91%), followed by S1 (63%), L1-4 (28%), and S2-5 (5%).

In patients with suspected LSS, a thorough physical examination should be conducted to rule out any other pathology that may be the source of referred pain to the legs. Sensory examination should include vibration, proprioception, and pin-prick testing in the appropriate nerve distribution regions. Loss of tendon reflexes, sciatic notch tenderness, and reduced lumbar lordosis may also be present in patients with suspected LSS. Motor weakness typically presents in the L5 nerve distribution. Amundsen et al[38] reported sensory changes in 51% of patients with LSS, reflex changes in 47%, lumbar tenderness in 40%, reduced spinal mobility in 36%, a positive straight-leg–raising test in 24%, weakness in 23%, and perianal numbness in 6%. If a clinical diagnosis of LSS is indicated, imaging studies should be used for confirmation. Upright plain radiographs are useful for excluding other spine pathologies, and CT and MRI can confirm nerve compression.[38]

Other neurologic disorders, such as complex regional pain syndrome (CRPS) and cutaneous neuroma, also have the potential to contribute to the incidence of pain after TKA. Previously referred to as reflex sympathetic dystrophy, CRPS symptoms include cutaneous hypersensitivity, abnormal skin color, edema, motor function impairment, and abnormal sudomotor activity.[39,40] The occurrence of CRPS after TKA is rare; a diagnosis of CRPS was made in only 5 of 662 patients (0.8%) in a recent series.[41] As a testament to the elusiveness of a CRPS diagnosis, the mean diagnostic time in this patient group was 5 months postoperatively.

Although the pathophysiologic mechanisms of CRPS remain unclear, the condition is thought to be initiated by sensitization of nociceptive nerve fibers by trauma.[42,43] Regardless of etiology, the diagnosis of CRPS often consists of excluding other painful conditions. A variety of tests, including infrared thermography, quantitative sensory testing, sudomotor testing, and bone scintigraphy, can be used to rule out other painful conditions and differentiate CRPS. In TKA patients with suspected CRPS, orthopaedic disorders with CRPS-like symptoms (such as rheumatic arthritis, tendon infection, or migratory osteoporosis) should be ruled out first. Treatment of CRPS typically centers on therapeutic interventions. Sympathetic blockade by an anesthesiologist, electrotherapy, occupational therapy, physical therapy, and proper counseling are often necessary in managing patients with CRPS.

Cutaneous and subcutaneous neuromas are another rare neurosensory disorder that can cause pain after TKA and are often diagnosed by injecting lidocaine into the suspected neuroma site. Pain relief is often a confirmation of a cutaneous neuroma, which can be subsequently treated with a variety of nonsurgical interventions such as local anesthetics or spinal cord stimulation. If nonsurgical measures prove unsuccessful, cutaneous nerve resection may be warranted.[41,44] In a series of patients who received a diagnosis of cutaneous neuroma after TKA, Katz and Hungerford[41] found that 87% reported substantial improvement in visual analog scale pain scores after sympathetic blockade or sympathectomy by an anesthesiologist.

Tendinitis, Bursitis, and Iliotibial Band Syndrome

Posterolateral knee pain after TKA can arise from a variety of sources. Pandher et al[45] reported a case of biceps tendinitis as the etiology of an acutely painful knee after TKA. Inflammation of the popliteus tendon also can present with posterolateral knee pain secondary to irritation from a laterally displaced femoral component, retained posterolateral osteophyte or cementophyte, or posterolateral tibial tray overhang. Alternatively, quadriceps and patellar tendinitis will present with anterior knee pain and typically result from a traumatic or overuse injury.

Semimembranosus tendinitis has been reported as a source of pain after TKA.[46] It typically presents as pain in the posteromedial knee that radiates into the posterior thigh or calf region and worsens in deep knee flexion. Physical examination findings typically consist of tenderness to palpation at the semimembranosus tibial insertion and increased pain during passive deep flexion. MRI is useful in confirming the diagnosis.

Surrounding the knee are multiple periarticular bursae (prepatellar, infrapatellar, pes anserine, and semimembranosus) that can become acutely inflamed and lead to knee pain (**Figure 1**). The occurrence of a popliteal cyst is commonly associated with knee arthritis or meniscal injury and is usually secondary to inflammation of the semimembranosus bursa. Rauschning[47] showed evidence of a defect in the posteromedial capsule that communicated with the semimembranosus bursa, displacing the semimembranosus and gastrocnemius tendons in 46% of the cadavers with no previous history of knee surgery or disease. Inflammation of the semimembranosus bursa via an intra-articular communication typically presents as a classic Baker cyst (**Figure 2**). Huang et al[48] reported a rare case of foreign body–mediated chronic pes anserinus bursitis induced by polyethylene particles that caused pain, swelling, and drainage.

Iliotibial band traction/friction syndrome should be considered in patients with a painful range of motion (specifically between 20° to 80°) and with pain localized to the lateral femoral epicondylar region.[49,50] Luyckx et al[49] reported a 7.2% incidence of iliotibial band traction/friction syndrome in a cruciate-substituting TKA design. The increased incidence was attributed to excessive lateral femoral condyle translation and internal rotation of the tibia, leading to excessive tension on the iliotibial band. This syndrome can be elicited using the Ober, Thomas, and Noble tests, and it typically presents as an overuse injury in conjunction with increased iliotibial band tightness.[51] An Ober test is conducted with the patient lying on his or her side. After flexion of the knee to 90° and extension and abduction of the affected hip by the examiner, support of the knee is released.

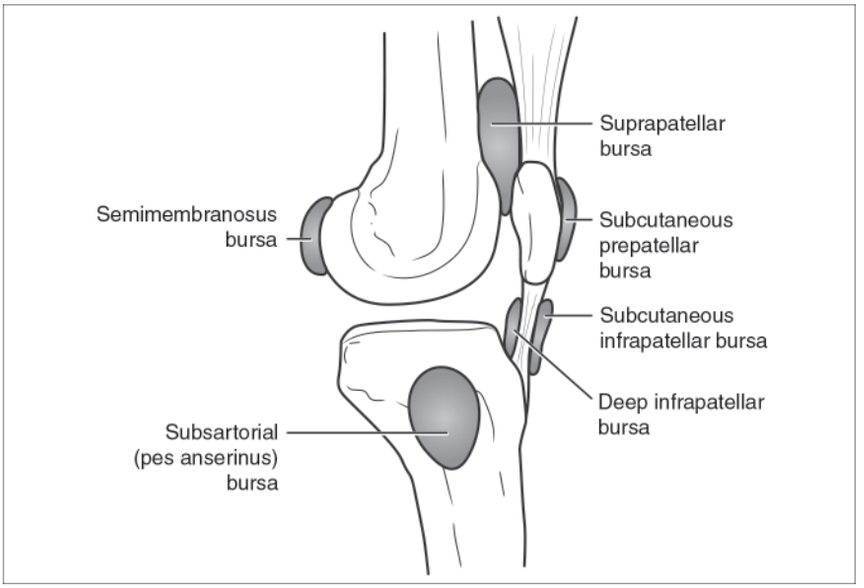

Figure 1 Illustration of some of the prominent knee bursae.

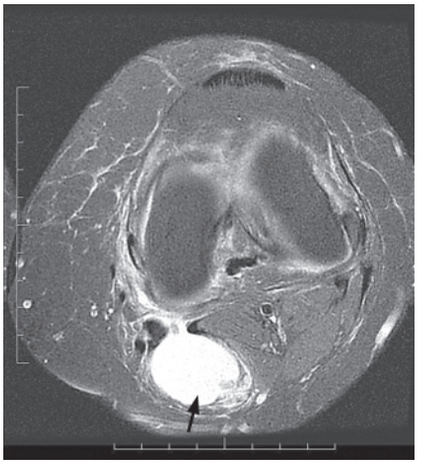

Figure 2 MRI demonstrating inflammation of the semimembranosus bursa and subsequent Baker cyst formation (arrow).

Failure of the hip to adduct indicates a positive test. The Thomas test is conducted with the patient supine. After the examiner places his or her hand under the lumbar spine to prevent hyperlordosis, the unaffected hip and knee are fully flexed. The patient holds the knee. A positive sign is indicated by the affected leg raising off the table. Both legs can then be fully flexed, and the affected leg is slowly lowered back into extension. A lack of full extension may indicate iliopsoas tightness, and any external rotation may suggest iliotibial band tightness. With the patient supine, the Noble test is conducted by flexing the affected hip and knee to 90° and placing pressure on the lateral femoral epicondyle with the thumb. With continuing pressure, the leg is passively and slowly extended. A positive test is indicated by the presence of pain at 30° of flexion.

Many cases of iliotibial band traction/friction syndrome resolve with nonsurgical treatments, including anti-inflammatory medications or local steroid injections. If pain persists, surgical release of the iliotibial band may be necessary.

Heterotopic Ossification

Oxidative stress from hypoxic environments often induces production of mast cells and fibroblast growth factor, which in turn undergo metaplastic transformation and leads to fibrocartilage formation with subsequent endochondral ossification and bone formation.[52] Heterotopic ossification can be asymptomatic or associated with a loss of motion in addition to catching and snapping that is localized to the patellofemoral joint. The incidence of heterotopic ossification varies widely, with an increased incidence in heavier patients, men, and patients with larger preoperative deformities.[53] Patients at elevated risk for heterotopic ossification include those with previous heterotropic ossification, bilateral hypertrophic osteoarthritis, and posttraumatic osteoarthritis with hypertrophic osteophytosis.[54,55]

Radiographs and laboratory testing are rarely helpful in detecting early stages of heterotopic ossification because calcium deposition in the bone matrix and developmental alkaline phosphatase elevation do not occur until several weeks after the initial onset of heterotopic ossification. Bone scanning may be useful for a definitive diagnosis and is known to detect heterotopic ossification earlier than radiographs or laboratory testing.[56] Either a NSAID (such as indomethacin), diphosphonate (such as ethane-1-hydroxy-1,1-diphosphate), or local radiation therapy is recommended for the treatment or prophylaxis of heterotopic ossification.[57]

Fibromyalgia and Connective Tissue Disorders

Although the precise source of pain in fibromyalgia remains unknown, it clearly depends on peripheral nociceptive input as well as abnormal central pain processing.[58] Some studies have suggested soft-tissue inflammation as the source of pain in fibromyalgia because pain and weakness of the muscles is one of its main symptoms. Changes

indicating disturbed microcirculation, mitochondrial damage, and a reduction in high-energy phosphates all suggest an energy-deficient state in the resting, painful muscles of patients with fibromyalgia.[59] Although fibromyalgia is characterized by allodynia and chronic pain, it should not be considered a contraindication for TKA because patients with fibromyalgia have shown improvement similar to that of control patients after TKA.[60,61] Nevertheless, these patients have reported lower postoperative satisfaction, largely because of higher levels of persistent postoperative pain.[60,61] The criteria set forth by the American College of Rheumatology are useful in diagnosing fibromyalgia; treatment typically consists of medications or therapy.[62]

Rarer connective tissue disorders, such as Ehlers-Danlos syndrome, also have potential to cause chronic pain in TKA patients. Deficiencies of collagen production in TKA patients with Ehlers-Danlos syndrome has been shown to instigate pain via degeneration and destabilization of the knee joint.[63] In a case series of 10 TKA patients with Ehlers-Danlos syndrome, approximately 30% reported dissatisfaction with TKA because of continued pain and instability.[64] Although rare, a diagnosis of Ehlers-Danlos syndrome should be confirmed through genetic testing and treated with a combination of physical therapy and pain-relieving and anti-inflammatory medications.

Psychological Factors and Pain Catastrophizing

The potential for psychological factors to influence a patient's perceptions of pain after TKA is well known, because pain catastrophizing has been designated a consistent and powerful psychological predictor of poor outcomes after TKA.[65,66] Patients who catastrophize about their pain ruminate more often about their pain, magnify its perceived threat, and experience greater feelings of helplessness when dealing with pain. Patients with higher preoperative pain and anxiety levels typically report extended postoperative TKA pain and diminished function.[11] It is imperative to carefully monitor and, if necessary, refer for further psychological treatment patients who may experience diminished postoperative outcomes because of anxiety, catastrophizing, or other psychological maladies known to intensify pain.

Metal Hypersensitivity

There is a paucity of clinical studies correlating positive hypersensitivity results with clinical TKA outcomes.[67,68] Nevertheless, metal hypersensitivity has been described previously as a type IV immunologic reaction and also may be considered as an unlikely cause for a painful TKA if all other causes have been ruled out.[69] Hypersensitivity reactions typically occur in the periprosthetic region in the months after TKA. Although rarely used, in vitro proliferation testing via lymphocyte transformation testing may be useful in assessing sensitivity to metal implants. Lymphocyte transformation testing consists of measuring lymphocyte proliferation after activation in vitro and comparing the findings with in vivo levels. Skin patch testing for nickel, cobalt, or chromium reaction may also be useful in detecting metal hypersensitivity in TKA patients, although the correlation between a positive test and symptom presentation remains unclear.[3,70] Hypoallergenic implants (specifically those that do not contain nickel) are warranted in a select subset of susceptible patients.

Summary

Determining the cause of pain after TKA can be a challenging task for an orthopaedic surgeon. With a thorough patient history, examination, and appropriate diagnostic approach, most sources of pain can be identified. After sepsis and intra-articular causes have been ruled out, extra-articular sources should be investigated. If the source of pain cannot be identified, revision arthroplasty alone is unlikely to relieve symptoms. By establishing an accurate etiology in a timely manner, unnecessary diagnostic testing will be avoided and the patient can be appropriately treated.

References

1. Kurtz S, Ong K, Lau E, Mowat F, Halpern M: Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. *J Bone Joint Surg Am* 2007;89(4):780-785.
2. Fehring TK, Odum S, Griffin WL, Mason JB, Nadaud M: Early failures in total knee arthroplasty. *Clin Orthop Relat Res* 2001;392:315-318.
3. Seil R, Pape D: Causes of failure and etiology of painful primary total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc* 2011;19(9):1418-1432.
4. Puolakka PA, Rorarius MG, Roviola M, Puolakka TJ, Nordhausen K, Lindgren L: Persistent pain following knee arthroplasty. *Eur J Anaesthesiol* 2010;27(5):455-460.
5. Hawker GA, Stewart L, French MR, et al: Understanding the pain experience in hip and knee osteoarthritis: An OARSI/OMERACT initiative. *Osteoarthritis Cartilage* 2008;16(4):415-422.
6. Singh JA, Gabriel S, Lewallen D: The impact of gender, age, and preoperative pain severity on pain after TKA. *Clin Orthop Relat Res* 2008;466(11):2717-2723.

7. Elson DW, Brenkel IJ: Predicting pain after total knee arthroplasty. *J Arthroplasty* 2006;21(7):1047-1053.
8. Fitzgerald JD, Orav EJ, Lee TH, et al: Patient quality of life during the 12 months following joint replacement surgery. *Arthritis Rheum* 2004;51(1):100-109.
9. Lingard EA, Katz JN, Wright EA, Sledge CB; Kinemax Outcomes Group: Predicting the outcome of total knee arthroplasty. *J Bone Joint Surg Am* 2004;86(10):2179-2186.
10. March LM, Cross MJ, Lapsley H, et al: Outcomes after hip or knee replacement surgery for osteoarthritis: A prospective cohort study comparing patients' quality of life before and after surgery with age-related population norms. *Med J Aust* 1999;171(5):235-238.
11. Brander VA, Stulberg SD, Adams AD, et al: Predicting total knee replacement pain: A prospective, observational study. *Clin Orthop Relat Res* 2003;416:27-36.
12. Fortin PR, Clarke AE, Joseph L, et al: Outcomes of total hip and knee replacement: Preoperative functional status predicts outcomes at six months after surgery. *Arthritis Rheum* 1999;42(8):1722-1728.
13. Jones CA, Voaklander DC, Johnston DW, Suarez-Almazor ME: The effect of age on pain, function, and quality of life after total hip and knee arthroplasty. *Arch Intern Med* 2001;161(3):454-460.
14. Roth ML, Tripp DA, Harrison MH, Sullivan M, Carson P: Demographic and psychosocial predictors of acute perioperative pain for total knee arthroplasty. *Pain Res Manag* 2007;12(3):185-194.
15. Parvizi J, Della Valle CJ: AAOS Clinical Practice Guideline: Diagnosis and treatment of periprosthetic joint infections of the hip and knee. *J Am Acad Orthop Surg* 2010;18(12):771-772.
16. Takeshita M, Nakamura J, Ohtori S, et al: Sensory innervation and inflammatory cytokines in hypertrophic synovia associated with pain transmission in osteoarthritis of the hip: A case-control study. *Rheumatology (Oxford)* 2012;51(10):1790-1795.
17. Lesher JM, Dreyfuss P, Hager N, Kaplan M, Furman M: Hip joint pain referral patterns: A descriptive study. *Pain Med* 2008;9(1):22-25.
18. Crawford RW, Gie GA, Ling RS, Murray DW: Diagnostic value of intra-articular anaesthetic in primary osteoarthritis of the hip. *J Bone Joint Surg Br* 1998;80(2):279-281.
19. Emms NW, O'Connor M, Montgomery SC: Hip pathology can masquerade as knee pain in adults. *Age Ageing* 2002;31(1):67-69.
20. Hammer SG: Hip fracture presenting as knee pain in an elderly patient. *Am Fam Physician* 1996;54(3):872.
21. Hetsroni I, Weigl D: Referred knee pain in posterior dislocation of the hip. *Clin Pediatr (Phila)* 2006;45(1):93-95.
22. Tharani R, Nakasone C, Vince KG: Periprosthetic fractures after total knee arthroplasty. *J Arthroplasty* 2005;20(4, suppl 2):27-32.
23. Smith MD, Henke JA: Pubic ramus fatigue fractures after total knee arthroplasty: A case report. *Orthopedics* 1988;11(2):315-317.
24. Torisu T: Fatigue fracture of the pelvis after total knee replacements: Report of a case. *Clin Orthop Relat Res* 1980;149:216-219.
25. Palance Martin D, Albareda J, Seral F: Subcapital stress fracture of the femoral neck after total knee arthroplasty. *Int Orthop* 1994;18(5):308-309.
26. Lesniewski PJ, Testa NN: Stress fracture of the hip as a complication of total knee replacement: Case report. *J Bone Joint Surg Am* 1982;64(2):304-306.
27. Meding JB, Keating EM, Ritter MA, Faris PM, Berend ME, Malinzak RA: The planovalgus foot: A harbinger of failure of posterior cruciate-retaining total knee replacement. *J Bone Joint Surg Am* 2005;87(suppl 2):59-62.
28. Mullaji A, Shetty GM: Persistent hindfoot valgus causes lateral deviation of weightbearing axis after total knee arthroplasty. *Clin Orthop Relat Res* 2011;469(4):1154-1160.
29. Bhave A, Mont M, Tennis S, Nickey M, Starr R, Etienne G: Functional problems and treatment solutions after total hip and knee joint arthroplasty. *J Bone Joint Surg Am* 2005;87(suppl 2):9-21.
30. Chandler JT, Moskal JT: Evaluation of knee and hindfoot alignment before and after total knee arthroplasty: A prospective analysis. *J Arthroplasty* 2004;19(2):211-216.
31. Guichet JM, Javed A, Russell J, Saleh M: Effect of the foot on the mechanical alignment of the lower limbs. *Clin Orthop Relat Res* 2003;415:193-201.
32. Meijer WT, Hoes AW, Rutgers D, Bots ML, Hofman A, Grobbee DE: Peripheral arterial disease in the elderly: The Rotterdam Study. *Arterioscler Thromb Vasc Biol* 1998;18(2):185-192.
33. Criqui MH, Fronek A, Barrett-Connor E, Klauber MR, Gabriel S, Goodman D: The prevalence of peripheral arterial disease in a defined population. *Circulation* 1985;71(3):510-515.
34. Bernstein J, Esterhai JL, Staska M, Reinhardt S, Mitchell ME: The prevalence of occult peripheral arterial disease among patients referred for orthopedic evaluation of leg pain. *Vasc Med* 2008;13(3):235-238.
35. Parvizi J, Pulido L, Slenker N, Macgibeny M, Purtill JJ, Rothman RH: Vascular injuries after total joint arthroplasty. *J Arthroplasty* 2008;23(8):1115-1121.
36. De Villiers PD, Booysen EL: Fibrous spinal stenosis: A report on 850 myelograms with a water-soluble contrast medium. *Clin Orthop Relat Res* 1976;115:140-144.
37. Pivec R, Johnson AJ, Naziri Q, Issa K, Mont MA, Bonutti PM: Lumbar spinal stenosis impairs function following total knee arthroplasty. *J Knee Surg* 2013;26(1):59-64.
38. Amundsen T, Weber H, Lilleås F, Nordal HJ, Abdelnoor M, Magnaes B: Lumbar spinal stenosis: Clinical and radiologic features. *Spine (Phila Pa 1976)* 1995;20(10):1178-1186.
39. Bonica JJ: Causalgia and other reflex sympathetic dystrophies. *Postgrad Med* 1973;53(6):143-148.
40. Vacariu G: Complex regional pain syndrome. *Disabil Rehabil* 2002;24(8):435-442.

41. Katz MM, Hungerford DS: Reflex sympathetic dystrophy affecting the knee. *J Bone Joint Surg Br* 1987;69(5):797-803.

42. Agnati LF, Tiengo M, Ferraguti F, et al: Pain, analgesia, and stress: An integrated view. *Clin J Pain* 1991;7(suppl 1):S23-S37.

43. Woolf CJ, Shortland P, Sivilotti LG: Sensitization of high mechanothreshold superficial dorsal horn and flexor motor neurones following chemosensitive primary afferent activation. *Pain* 1994;58(2):141-155.

44. Dellon AL, Mont MA, Krackow KA, Hungerford DS: Partial denervation for persistent neuroma pain after total knee arthroplasty. *Clin Orthop Relat Res* 1995;316:145-150.

45. Pandher DS, Boparai RS, Kapila R: Biceps tendinitis as a cause of acute painful knee after total knee arthroplasty. *J Arthroplasty* 2009;24(8):1292.e15-1292.e18.

46. Hendel D, Weisbort M, Garti A: Semimembranosus tendonitis after total knee arthroplasty: Good outcome after surgery in 6 patients. *Acta Orthop Scand* 2003;74(4):429-430.

47. Rauschning W: Anatomy and function of the communication between knee joint and popliteal bursae. *Ann Rheum Dis* 1980;39(4):354-358.

48. Huang TW, Wang CJ, Huang SC: Polyethylene-induced pes anserinus bursitis mimicking an infected total knee arthroplasty: A case report and review of the literature. *J Arthroplasty* 2003;18(3):383-386.

49. Luyckx L, Luyckx T, Bellemans J, Victor J: Iliotibial band traction syndrome in guided motion TKA: A new clinical entity after TKA. *Acta Orthop Belg* 2010;76(4):507-512.

50. Renne JW: The iliotibial band friction syndrome. *J Bone Joint Surg Am* 1975;57(8):1110-1111.

51. Strauss EJ, Kim S, Calcei JG, Park D: Iliotibial band syndrome: Evaluation and management. *J Am Acad Orthop Surg* 2011;19(12):728-736.

52. Freeman TA, Parvizi J, Dela Valle CJ, Steinbeck MJ: Mast cells and hypoxia drive tissue metaplasia and heterotopic ossification in idiopathic arthrofibrosis after total knee arthroplasty. *Fibrogenesis Tissue Repair* 2010;3(17):1.

53. Dalury DF, Jiranek WA: The incidence of heterotopic ossification after total knee arthroplasty. *J Arthroplasty* 2004;19(4):447-452.

54. Farris MK, Chowdhry VK, Lemke S, Kilpatrick M, Lacombe M: Osteosarcoma following single fraction radiation prophylaxis for heterotopic ossification. *Radiat Oncol* 2012;7(140):1.

55. DeLee J, Ferrari A, Charnley J: Ectopic bone formation following low friction arthroplasty of the hip. *Clin Orthop Relat Res* 1976;121:53-59.

56. Freed JH, Hahn H, Menter R, Dillon T: The use of the three-phase bone scan in the early diagnosis of heterotopic ossification (HO) and in the evaluation of Didronel therapy. *Paraplegia* 1982;20(4):208-216.

57. Shehab D, Elgazzar AH, Collier BD: Heterotopic ossification. *J Nucl Med* 2002;43(3):346-353.

58. Staud R: Fibromyalgia pain: Do we know the source? *Curr Opin Rheumatol* 2004;16(2):157-163.

59. Bengtsson A, Henriksson KG: The muscle in fibromyalgia: A review of Swedish studies. *J Rheumatol Suppl* 1989;19:144-149.

60. Bican O, Jacovides C, Pulido L, Saunders C, Parvizi J: Total knee arthroplasty in patients with fibromyalgia. *J Knee Surg* 2011;24(4):265-271.

61. D'Apuzzo MR, Cabanela ME, Trousdale RT, Sierra RJ: Primary total knee arthroplasty in patients with fibromyalgia. *Orthopedics* 2012;35(2):e175-e178.

62. Wolfe F, Smythe HA, Yunus MB, et al: The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Rheum* 1990;33(2):160-172.

63. Weinberg J, Doering C, McFarland EG: Joint surgery in Ehlers-Danlos patients: Results of a survey. *Am J Orthop (Belle Mead NJ)* 1999;28(7):406-409.

64. Rose PS, Johnson CA, Hungerford DS, McFarland EG: Total knee arthroplasty in Ehlers-Danlos syndrome. *J Arthroplasty* 2004;19(2):190-196.

65. Riddle DL, Keefe FJ, Ang D, et al: A phase III randomized three-arm trial of physical therapist delivered pain coping skills training for patients with total knee arthroplasty: The KAST-Pain protocol. *BMC Musculoskelet Disord* 2012;13(149):1.

66. Lingard EA, Riddle DL: Impact of psychological distress on pain and function following knee arthroplasty. *J Bone Joint Surg Am* 2007;89(6):1161-1169.

67. Nilsdotter AK, Toksvig-Larsen S, Roos EM: Knee arthroplasty: Are patients' expectations fulfilled? A prospective study of pain and function in 102 patients with 5-year follow-up. *Acta Orthop* 2009;80(1):55-61.

68. Noble PC, Conditt MA, Cook KF, Mathis KB: Patient expectations affect satisfaction with total knee arthroplasty. *Clin Orthop Relat Res* 2006;452:35-43.

69. Jacobs JJ, Hallab NJ: Loosening and osteolysis associated with metal-on-metal bearings: A local effect of metal hypersensitivity? *J Bone Joint Surg Am* 2006;88(6):1171-1172.

70. Christensen CP, Crawford JJ, Olin MD, Vail TP: Revision of the stiff total knee arthroplasty. *J Arthroplasty* 2002;17(4):409-415.