Diagnostic Algorithm for Residual Pain After Total Knee Arthroplasty

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Abstract

Although total knee arthroplasty is a successful and cost-effective procedure, patient dissatisfaction remains as high as 50%. Postoperative residual knee pain after total knee arthroplasty, with or without crepitation, is a major factor that contributes to patient dissatisfaction. The most common location for residual pain after total knee arthroplasty is anteriorly. Because residual pain has been associated with an un-resurfaced patella, this review includes only registry data and total knee arthroplasty with patella replacement. Some suggest that the pathogenesis of residual knee pain may be related to mechanical stimuli that activate free nerve endings around the patellofemoral joint. Various etiologies have been implicated in residual pain, including (1) low-grade infection, (2) midflexion instability, and (3) component malalignment with patellar maltracking. Less common causes include (4) crepitation and patellar clunk syndrome; (5) patellofemoral symptoms, including overstuffing and avascular necrosis of the patella; (6) early aseptic loosening; (7) hypersensitivity to metal or cement; (8) complex regional pain syndrome; and (9) pseudoaneurysm. Because all of these conditions can lead to residual pain, identifying the etiology can be a difficult diagnostic challenge. Often, patients with persistent pain and normal findings on radiographs and laboratory workup may benefit from a diagnostic injection or further imaging. However, up to 10% to 15% of patients with residual pain may have unexplained pain. This literature review summarizes the findings on the causes of residual pain and presents a diagnostic algorithm to facilitate an accurate diagnosis for residual pain after total knee arthroplasty. [Orthopedics. 2016; 39(2):e246-e252.]

Total knee arthroplasty (TKA) is a highly successful and cost-effective procedure to relieve pain, correct deformity, and improve function in patients with end-stage arthritis of the knee. However, patient dissatisfaction remains as high as 50%. Several factors contribute to dissatisfaction, including unmet expectations, functional limitations, and most commonly, residual pain. Residual pain after TKA is well documented. The incidence of anterior knee pain varies, but has been reported to be as high as 20%. One study reported that 10% of patients had persistent anterior knee pain at up to 10 years of follow-up. During later follow-up, an additional 10% had anterior knee pain. Finding a precise cause of anterior knee pain is a diagnostic challenge.

This review does not include referred pain as a result of other causes (ie, ipsilateral hip or spine). When such pain is present, a diagnostic injection of bupivacaine into the knee can relieve pain if the source of pain is local. A systemic, methodical approach to investigate the etiology of local pain is required.

This review summarizes the literature on various causes of anterior knee pain, with or without crepitation, that may lead to patient dissatisfaction after TKA. It also discusses the current incidence and
etiology and provides a systematic guide to the accurate diagnosis of the cause of residual knee pain after TKA (Figure).

**PATHOGENESIS AND ETIOLOGY OF KNEE PAIN AFTER TKA**

A literature review identified several potential causes of postoperative pain, 1 of which is related to an un-resurfaced patella. This review includes only registry data on revised knees and literature on TKA with patella replacement. After TKA, the knee is often symptomatic for 1 to 2 years. In a significant number of patients, complete fibroblastic repair and devascularization can take 6 months to 1 year. The incidence of anterior knee pain should be evaluated between 1 and 2 years of follow-up.

Many believe that the pathogenesis of residual knee pain after TKA involves thermal, chemical, or mechanical stimuli that activate free nerve endings in the peripheral nervous system. Around the patellofemoral joint there are many structures with a large number of free nerve endings, including the quadriceps, retinacula, and synovium. Once stimulated, these free nerve endings transmit signals to the central nervous system via either myelinated A fibers or unmyelinated C fibers. The A fibers are largely responsible for sharp, acute pain, whereas the C fibers are much slower and transmit a dull, chronic ache.

Various etiologies have been implicated in the stimulation of free nerve endings and subsequently result in residual pain after TKA, including: (1) low-grade infection, (2) midflexion instability, and (3) component malalignment with patellar maltracking. Less common causes include (4) crepitation and patellar clunk syndrome; (5) patellofemoral symptoms, including overstuffing and avascular necrosis; (6) early aseptic loosening; (7) hypersensitivity to metal or cement; (8) complex regional pain syndrome; and (9) pseudoaneurysm.

**Low-Grade Infection**

Pain associated with effusion after surgery in patients without systemic symptoms presents a diagnostic challenge, especially in patients who have been treated with antibiotics. Because of the catastrophic nature of septic failure, infection should be ruled out promptly in patients with symptomatic TKA.

Acute infections occur in 1% to 2% of cases of TKA and are relatively easily diagnosed because of the sudden onset of severe pain. However, recently, there has been increasing awareness of low-grade infections, which are more difficult to diagnose and are likely more common than previously thought. Infections with low-virulence bacteria often cause a moderately painful or stiff knee and may or may not present with radiographic findings of infection. Blood testing is usually the first step in diagnosing a symptomatic TKA.

Blood test results showing an erythrocyte sedimentation rate of greater than 30 mm/h and a C-reactive protein level of greater than 10 mg/L are highly suggestive of inflammation and require further evaluation, including knee aspiration. The joint aspirate should be sent for Gram stain, microbiologic culture, C-reactive protein level, fluid cell count, and differential. Synovial leukocyte count of 1100 to 4000 cells/μL and synovial neutrophil percentage of 64% to 69% or greater are highly indicative of infection. If no organism is recovered in a suspected low-grade infection, repeat culture and/or biopsy of the synovium may be helpful. Other diagnostic tests, such as bone scan, gallium scan, computed tomography (CT) scan, or magnetic resonance imaging (MRI) scan, have limited value. Metal artifact reduction sequence MRI with metal suppression has been used to assess soft tissue reactions and synovitis. Positive findings on labeled leukocyte imaging are also highly suggestive of infection.

Once the diagnosis of periprosthetic infection is established, treatment depends on the virulence of the bacteria, the sensitivity of the organisms, and the symptoms and overall health of the patient. Treatment choices include irrigation, with or without liner exchange, and single- or 2-stage revision, based on the surgeon’s preference.

**Midflexion Instability**

Patients who have pain with mild effusion and a normal erythrocyte sedimentation rate and C-reactive protein level should be evaluated for instability. Historically, midflexion instability has been underdiagnosed, but recently, it has become more widely recognized. Conceptually, midflexion instability occurs when the knee is balanced and stable in both extension and flexion but subtle instability occurs during the arc of motion between full extension and full flexion. Pain is often the first sign of instability and is the result of abnormal stress on the soft tissue envelope. Midflexion instability also can lead to synovium impingement synovitis and recurrent hemarthrosis.

The diagnosis of instability can often be determined from the patient’s history, radiographic findings, identification of the mechanism leading to instability, and the findings of physical examination. On physical examination, midflexion instability is best shown by a modified Lachman’s or anterior drawer test. With the ipsilateral foot stabilized, the knee is flexed to 70º to 90º and anteroposterior force is applied. Laxity greater than 1 cm indicates midflexion instability.

Treatment of instability is revision surgery to correct imbalances of ligamentous flexion and extension.

**Component Malalignment With Patellar Maltracking**

Appropriate rotation of the femoral components is the key to proper patellar tracking. Proper femoral rotation is achieved either by using anatomic landmarks (transepicondylar axis, posterior condylar axis, or anteroposterior axis) with the measured resection technique or by using the parallel to the tibial cut technique with the gap balancing meth-
With either method, the goal is to create rectangular, equal, and symmetric flexion and extension gaps.

Femoral and tibial component malrotation is a major factor contributing to anterior knee pain. There is a clear association between tibial component internal rotation and combined component internal rotation with anterior knee pain after TKA. Tibial component malrotation can occur when the posterolateral tibial plateau is inadequately exposed, placing the tibial component in internal rotation relative to the correct axis of the knee. In up to 16% of cases of TKA, a rotational mismatch occurs between the tibial and femoral components, mostly from the tibial component. Component malalignment is the leading cause of patellar maltracking, further evaluation is required. Rotational malalignment is best evaluated with CT or MRI scan. Once the diagnosis of rotational malalignment is made, treatment is revision TKA with special attention to rotational alignment.

Crepitation and Patellar Clunk Syndrome

Crepitation, or a cracking noise with or without pain, has been well documented and is often the result of peripatellar scar formation. The incidence varies, but may be as high as 16.7% for painless crepitation and 1.6% for painful crepitation. Many factors affect the incidence of crepitation, including implant design. Rotating platforms and posterior-stabilized implants have a higher incidence of crepitation than fixed-bearing and cruciate-retaining designs.

Unlike patellar maltracking, which presents during flexion, patellar clunk syndrome is the result of impingement during knee extension. When the knee is extended, a suprapatellar nodule can lodge into the intercondylar notch of the femoral component during flexion and displace on extension, sometimes with an audible “clunk.” The knee with crepitation or patellar clunk syndrome can manifest with or without pain and with or without decreased range of motion, but noise is nearly always present. The diagnosis of crepitation or patellar clunk syndrome is readily made on physical examination by feeling the knee during extension and listening for the clunk. Once the diagnosis of patellar clunk syndrome is made, if all other aspects of the arthroplasty appear normal, treatment is arthroscopic or open debridement.

Patellofemoral Symptoms

Overstuffing of the patellofemoral compartment by either undercutting the patella or oversizing the patellar or femoral components can cause anterior knee pain. Furthermore, medializing of an undersized patella with exposed lateral bone can lead to lateral tilt and lateral facet impingement and can require surgical intervention. Overstuffing can be diagnosed on radiographs by measuring the difference in the deepest part of the trochlear groove to the anterior aspect of the patella between preoperative and follow-up films. A difference of 3 to 5 mm requires further investigation.

Further diagnostic indicators of overstuffing of the patellofemoral joint include parapatellar tenderness and relief after local intra-articular bupivacaine injection. In patients with anterior knee pain with an undercovered patella with overhanging bone or extreme patellar tilt, surgical intervention may be required.

Avascular necrosis is considered when anterior knee pain is associated with local tenderness in the patella and radiographic confirmation of changes in bone density.
When avascular necrosis is suspected, MRI scan is needed to evaluate the extent of necrosis and detect possible early avascular necrosis that is not seen on plain radiographs.

Patellar avascular necrosis is often associated with wide lateral release, especially in knees with extreme preoperative valgus deformity. During lateral release, care is taken to preserve the superior lateral genicular artery. Reducing physical activity and exercise may be required to avoid fracture of the avascular patella. Severe patellar avascular necrosis can result in patellar fragmentation, especially in the setting of aseptic loosening, and removal of the loose prosthesis with patellectomy or patellectomy may be necessary.34,55

Early Aseptic Loosening

Aseptic loosening is recognized as 1 of the most common mechanisms of failure of TKA. Polyethylene wear, osteolysis, and instability can lead to aseptic loosening.25 Before obvious loosening occurs, a loose TKA often causes generalized knee pain that can limit functional ability.56

Diagnosis of aseptic loosening is readily made by a thorough patient history and radiographic evaluation. Obvious implant movement or demarcation at the implant-bone or cement-bone interface suggests aseptic loosening. However, in some cases in which radiographic findings are unremarkable, MRI or CT scan is performed. When the diagnosis of loosening is established, revision of the component is required.

Hypersensitivity to Metals or Cement

Although rare, there have been increasing reports of hypersensitivity to metal, cement, or antibiotics used in TKA.32,57-60 Little is known about the mechanism by which sensitization toward these allergens occurs, but it is generally believed to be independent of exposure.61 Sensitization may cause localized dermatitis, aseptic loosening, and residual pain.57,58

Patients with suspected hypersensitivity should have a dermatologic consultation. Although some have questioned their sensitivity and specificity, patch testing and lymphocyte proliferation testing have been shown to be useful.59,60

The literature provides little information on the treatment of metal or cement hypersensitivity, but the authors believe that it varies based on the specific allergies. Some recommend conversion to other metals or ceramic components for metal sensitivity, and revision to an uncemented TKA may benefit patients with cement sensitivity.32

Complex Regional Pain Syndrome

Complex regional pain syndrome is a sympathetic or parasympathetic disorder that is uncommon after TKA.65 It is associated with disproportionately high levels of pain that can be spontaneous or induced by a stimulus. Symptoms include abnormal regulation of blood flow, sweating, skin edema, trophic changes, and joint stiffness.62 Complex regional pain syndrome can cause disabling pain and stiffness after TKA, and recovery may be prolonged.

Because there are no specific diagnostic modalities, complex regional pain syndrome is often overlooked as a source of postoperative pain.61 Early treatment is critical, but there is no consensus on the most effective management. The most common intervention includes a series of regional anesthetic and local sympathetic nerve blocks.63

Pseudoaneurysm

Vascular complications after TKA are rare. Pseudoaneurysms are often caused by leaky arteries as a result of traumatic injury of the geniculate or popliteal arteries during osteophyte resection.64,65 Patients typically present with knee swelling, an enlarged pulsatile mass, thrill, hemorrhage, ecchymosis, sensory or motor deficit, and decreased peripheral pulse.66 Before limb complications occur, hemolytic synovitis can compress the joint, ultimately causing moderate to severe knee pain.66

Pseudoaneurysms often require a high level of suspicion in the evaluation of symptomatic TKA. Often, affected patients are taking an anticoagulant. Many methods are available for diagnosis. Less sensitive tests include joint aspiration with bloody synovium and duplex ultrasonography. The most definitive means to confirm the diagnosis is angiography or MRI.67 Once the diagnosis has been confirmed, management varies based on the size and location of the pseudoaneurysm. Treatment options include surgical repair, percutaneous embolization, and endovascular stenting.

DISCUSSION

Residual pain, especially anterior knee pain, is among the most common causes of dissatisfaction after TKA. The incidence of anterior knee pain varies considerably, with some studies reporting a rate of as high as 20%.5,9,10,13-18 Because of the variety of conditions that can affect anterior knee pain, identifying the primary cause of pain can be a diagnostic challenge. The goal of this review is to outline the incidence and etiology of residual pain after TKA as well as to provide a systematic approach to obtain an accurate diagnosis (Figure).

Patient dissatisfaction after TKA can stem from a variety of reasons and is usually related to pain. Little is known about the pathogenesis of anterior knee pain; however, many believe that it is the result of abnormal kinematics or that thermal or chemical stimuli can activate free nerve endings that send signals regarding pain and inflammation to the central nervous system.20,21,67 Although psychological factors can predict residual pain, because of the believed pathogenesis, mechanical causes of pain should be ruled out. Common causes of residual pain after TKA include low-grade infection, midflexion instability, and patellar maltracking as a result of malrotated femoral and tibial
components. Because of the catastrophic nature of septic TKA, infection should be ruled out first with routine blood work and aspiration. 20, 26, 27 Once infection has been ruled out, other mechanical causes of pain should be considered. Midflexion instability and patellar maltracking secondary to malrotated components can be ruled out with physical examination and further imaging, respectively. 27, 35 During physical examination, crepitation and patellar clunk can often be ruled out as well.

In patients who have persistent pain after TKA and normal findings on routine radiographs and laboratory workup, a diagnostic injection or further imaging can be helpful. Less common causes of residual pain require a high level of suspicion and include patellofemoral symptoms, such as overstretching or avascular necrosis of the patella, early aseptic loosening, metal or cement hypersensitivity, complex regional pain syndrome, and pseudoaneurysm. 18, 55, 68 In patients who have persistent pain without any of these causes, uncommon causes, such as nerve damage or psychosocial etiologies, should be considered. 18, 69, 71 As many as 10% to 15% of patients with residual pain may have unexplained pain. The authors believe that this may be the result of disruption of C fibers during anterior exposure of the knee.

Once the diagnosis has been established, treatment options vary from conservative management to revision TKA. Residual pain after TKA is not uncommon and can decrease for up to 5 years postoperatively, and so conservative treatment is advisable in cases without a clear cause of pain. 26 When a definitive diagnosis has been established, most causes of residual pain can be treated surgically; however, surgical treatment is reserved for persistent moderate to severe pain. If no definitive cause of residual pain is found, conservative treatment is recommended.

References
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