The Painful Total Knee Arthroplasty

Mitchell McDowell, DO[a], Andrew Park, MD[b], Tad L. Gerlinger, MD[b,c,*]

KEYWORDS

• Total knee arthroplasty • Revision total knee arthroplasty • Painful total knee • Failed total knee

KEY POINTS

- Pain after total knee arthroplasty can be caused by numerous factors, and a systematic evaluation that includes a thorough history, physical, radiographic and laboratory evaluation, and understanding of the differential diagnosis is essential.
- Intra-articular, peri-articular, and extra-articular pathology may cause pain after total knee arthroplasty.
- Revision total knee arthroplasty performed without a firm diagnosis has a high chance of failure.

INTRODUCTION

Total knee arthroplasty (TKA) has been shown to produce good clinical results in approximately 80% of patients at long-term follow-up,[1–6] but the 20% of patients who continue to experience pain following TKA often present a diagnostic challenge to orthopedic surgeons. The evaluation and treatment of these patients relies on a thorough understanding of the differential diagnosis of a painful TKA, and a systematic approach is instrumental to efficiently and effectively resolve their pain.

DEFINITION AND MECHANISM OF PAIN

Pain can serve as a protective mechanism by inducing a reaction to eliminate a harmful stimulus. Excessive pain after a TKA often diminishes one's quality of life.[7–10] Pain has been defined as "what the patient says it is."[11] This simplistic definition emphasizes that pain is a subjective experience with no reliable objective measures. Thus the patient's self-report is the most reliable indicator of pain and should not be discredited.[12] The International Association for the Study of Pain (IASP) defines pain as an "unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[13] This definition emphasizes that pain is a complex, multifactorial experience that involves multiple organ systems. To better understand this complex problem, 2 basic types of pain have been described: nociceptive and neuropathic pain.

Nociceptive Pain

Nociceptive pain is caused by the ongoing activation of sensory neurons in response to a noxious stimulus, such as injury, disease, or inflammation,[14] and it is indicative of real or potential tissue damage. There is generally a close correlation between pain perception and stimulus intensity. Nociceptive pain may be described as visceral or somatic. Somatic pain arises from tissues such as skin, muscle, joint capsules, and bone and is further categorized as superficial and deep somatic pain. Superficial somatic pain presents with a well-localized, sharp, pricking, or burning

Disclosure Statement: The authors have no relevant financial relationships to disclose. Dr T.L. Gerlinger is a paid consultant for Smith & Nephew.

[a] Department of Orthopaedic Surgery, Kaiser Permanente Riverside Medical Center, 10800 Magnolia Avenue, Riverside, CA, USA; [b] Department of Orthopaedic Surgery, Midwest Orthopedics at Rush, 1611 West Harrison Street Suite 300, Chicago, IL, USA; [c] Uniformed Services University of the Health Sciences, Bethesda, MD 20814, USA

* Corresponding author.

E-mail address: tad.gerlinger@rushortho.com

Orthop Clin N Am 47 (2016) 317–326

http://dx.doi.org/10.1016/j.ocl.2015.09.008

sensation, whereas deep somatic pain presents as a diffuse, dull, or aching sensation. Deep somatic pain has also been described as a cramping sensation that may be referred to or from other sites (ie, referred pain).

Neuropathic Pain

Neuropathic pain is caused by aberrant signal processing in the peripheral or central nervous system and is broadly categorized as peripheral or central in origin. Animal studies suggest that several changes likely contribute to neuropathic pain:

1. Generation of spontaneous ectopic activity,
2. Loss of normal inhibitory mechanisms in the dorsal horn,
3. Altered primary afferent neuron phenotypes, and
4. Sprouting of nerve fibers leading to altered neural connections.[15]

These changes result in abnormal nerve firing and/or abnormal signal amplification.[16] Common causes of neuropathic pain include nerve transection, inflammation, tumors, toxins, metabolic diseases, infections, and primary neurologic diseases. Neuropathic pain is sometimes called "pathologic" pain because it serves no purpose. A chronic pain state may occur when pathophysiologic changes become independent of the inciting event.[17] Neuropathic pain may be continuous or episodic and is perceived as a variety of different sensations (eg, burning, tingling, prickling, shooting, electric shock–like, jabbing, squeezing, deep aching, spasm, or cold). In contrast to nociceptive pain, neuropathic pain is often unresponsive or poorly responsive to nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids.[18] However, neuropathic pain may respond to antiepileptic drugs, antidepressants, or local anesthetics.[19]

Sensitization has a vital role in the etiology of neuropathic pain. With intense, repeated, or prolonged exposure to inflammatory mediators, nociceptors exhibit a lowered threshold for activation and an increased rate of firing.[20] Peripheral sensitization in turn plays a vital role in central sensitization and clinical pain states such as hyperalgesia and allodynia.[16,21,22] Central sensitization refers to a state of spinal neuron hyperexcitability.[23] Dysregulated activation of certain N-methyl-D-aspartate (NMDA) receptors is responsible for this process.[24] Central sensitization is associated with a reduction in central inhibition, spontaneous dorsal horn neuron activity, the recruitment of responses from neurons that normally only respond to low-intensity stimuli (ie, altered neural connections), and expansion of dorsal horn neuron receptive fields.[25] Clinically, these changes may manifest as (1) hyperalgesia, (2) allodynia, (3) prolonged pain after a transient stimulus (persistent pain), and (4) the spread of pain to uninjured tissue (ie, referred pain).[25]

Sensitization is likely responsible for most of the continuing pain and hyperalgesia after an injury or surgery.[26] This may be due to noxious stimuli from injured and inflamed tissue or "abnormal" input from injured nerves. Sensitization may serve an adaptive purpose to encourage protection of the injured area during the healing phase; however, these processes may persist long after healing of the injury and lead to chronic pain. Furthermore, sensitization may be why neuropathic pain often exceeds the provoking stimulus, both spatially and temporally.[23] Finally, central sensitization may explain why chronic pain is more difficult to treat than acute pain.[27]

EVALUATION OF PAINFUL TOTAL KNEE ARTHROPLASTY

The management of a painful TKA requires a multidisciplinary team approach that involves orthopedic surgeons, physical therapists, pain management physicians, and primary medical doctors.[10] A full assessment of both the surgical and nonsurgical factors that can cause pain after TKA is often time-consuming, but timely management of the pain is essential and should be approached irrespective of the origin of the pain and whether or not it may be addressed surgically. Depending on the acuity and necessity of surgical intervention, this evaluation may best be done by a pain management specialist, which in itself may help to alleviate anxiety. As a result, the emphasis may be focused on a nonsurgical resolution of the problem.[10]

The use of appropriate analgesics may help to alleviate pain, reduce the sense of urgency for further intervention, and decrease the desperation often felt by patients and their families. Most patients report low pain scores in the first 3 months after TKA, but some report continued pain or even increasing pain as time passes. This often correlates with the cessation of regular pain medications by patients who feel that these medications are not required for such a long period postoperatively. The pain may lead to a reduction in the range of motion, with subsequent stiffness and increased sensitivity around the joint. Patients should be encouraged to take analgesics if warranted, and these should be prescribed according to the World Health Organization (WHO) analgesic ladder (**Fig. 1**).[28] However, medical management