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The well-aligned yet painful total knee arthroplasty: a timeline-based diagnostic approach to persistent postoperative pain

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ABSTRACT

Persistent pain following technically successful total knee arthroplasty (TKA) affects 10–20% of patients despite satisfactory implant alignment and stability. Conventional diagnostic algorithms predominantly emphasize structural pathology while often under-recognizing extra-articular and centralized pain mechanisms, contributing to delayed diagnosis and unnecessary revision surgery. This narrative review was conducted in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA). PubMed, Embase and the Cochrane Library were searched from database inception to June 2026 for studies addressing persistent pain after primary TKA. Evidence was synthesized across four domains: intra-articular pathology, extra-articular/referred causes, centralized and psychosocial pain mechanisms, and existing diagnostic algorithms. Based on the available literature, a timeline-stratified diagnostic framework was developed. Persistent pain after well-aligned TKA arises from three principal mechanistic categories: intra-articular, extra-articular/referred, and centralized pain. The differential diagnosis evolves with postoperative timing, with structural complications predominating early, instability and patellofemoral disorders emerging during intermediate follow-up, and aseptic loosening alongside centralized pain becoming increasingly relevant later. The proposed algorithm prioritizes systematic exclusion of infection, targeted assessment of mechanical pathology, evaluation of hip, spine and hindfoot disorders, and mandatory screening for central sensitization and neuropathic pain before considering revision surgery. Persistent pain following well-aligned TKA is multifactorial and requires a structured, timeline-based diagnostic strategy that integrates structural, extra-articular and centralized pain mechanisms. This comprehensive approach may facilitate earlier diagnosis, reduce unnecessary investigations and revision procedures, and support more individualized management while providing a practical framework for future prospective validation.

Keywords: Total knee arthroplasty, Persistent postoperative pain, Painful total knee replacement, Diagnostic algorithm, Central sensitization, Revision arthroplasty

INTRODUCTION

Total Knee Arthroplasty (TKA) is a reliable, cost-effective treatment for end-stage knee osteoarthritis, yet dissatisfaction persists in 10% to 20% of patients, with some series reporting rates as high as 50%.[1] Much of this dissatisfaction stems not from component failure or surgical error, but from persistent pain in a knee that is, by

radiographic and clinical criteria, well-aligned and technically satisfactory.[2]

The differential is broad intra-articular causes (patellofemoral mal-tracking, mid-flexion instability, low-grade infection), extra-articular/referred sources (hip, spine, hindfoot) and centralized mechanisms including central sensitization and pain catastrophizing.[3] Without a structured approach, clinicians often default to either exhaustive, costly workups or premature reassurance, both of which delay accurate diagnosis.

Existing diagnostic algorithms predate recent advances in centralized pain phenotyping and dynamic soft-tissue balance concepts and tend to emphasize structural and infective causes while giving comparatively little systematic weight to these newer domains.[1,2] This review synthesizes current evidence on persistent pain after well-aligned TKA and proposes a structured, timeline-stratified diagnostic algorithm integrating intra-articular, extra-articular and centralized pain categories.

METHODS

This narrative review followed the principles outlined in the Scale for the Assessment of Narrative Review Articles (SANRA), addressing justification of the review topic, explicit statement of aims, transparent description of the search process and structured presentation of evidence, rather than the PRISMA framework, which is intended for systematic reviews and meta-analyses with exhaustive, protocol-registered study capture; given the breadth and cross-disciplinary nature of causes underlying persistent pain after TKA, a systematic review methodology was neither appropriate nor feasible. The search was instead organised around four distinct but interconnected evidence streams relevant to the central clinical question, since literature on this topic is distributed across arthroplasty, pain medicine and referred-pain subspecialty sources with limited cross-referencing between them.

PubMed, Embase and the Cochrane Library were searched from database inception to June 2026, restricted to English-language publications involving adult human subjects. Search terms were structured separately for each stream. For the intra-articular causes stream, the terms used were (total knee arthroplasty) AND (patellofemoral OR instability OR malrotation OR malalignment OR infection) AND (residual pain OR persistent pain). For the extra-articular/referred pain stream, the terms used were (total knee arthroplasty) AND (hip OR spine OR lumbar OR hindfoot OR ankle) AND (referred pain OR knee pain). For the centralized/psychosocial pain stream, the terms used were (total knee arthroplasty) AND (central sensitization OR catastrophizing OR chronic pain OR neuropathic) AND (persistent pain OR dissatisfaction). For the diagnostic algorithm/classification stream, the terms used were (total knee arthroplasty) AND (diagnostic algorithm OR painful knee OR failure analysis OR evaluation) AND (approach OR workup). Reference lists of all retrieved articles were hand-searched for additional eligible studies not captured by the primary search terms.

Titles and abstracts were screened for relevance to the four streams, followed by full-text review of potentially eligible articles. Studies were included if they addressed adult patients with persistent or residual pain following primary TKA, reported diagnostic criteria, prevalence or risk factors relevant to a specific pain category or proposed a diagnostic or classification framework; case reports without diagnostic generalizability, revision-specific technical studies unrelated to diagnosis and paediatric studies were excluded.

Evidence extracted from each stream was organised into a literature matrix recording author, year, study design, sample size, pain category/etiology, diagnostic criteria or tool used and principal finding, allowing comparison both within and across streams. This four-stream organisation guided the extraction of evidence into the literature matrix and its subsequent synthesis into a structured diagnostic approach. Given the heterogeneity of diagnostic criteria, outcome measures and study designs across the included literature, no formal quality-effect pooling or meta-analytic synthesis was attempted; the review instead follows a narrative-synthesis approach consistent with current guidance for narrative reviews addressing fragmented, multidisciplinary evidence bases.

Classification framework

A workable diagnostic classification for persistent pain after well-aligned TKA rests on two orthogonal axes: anatomic/mechanistic category and postoperative timeline. Existing frameworks vary in how finely they split the first axis, i.e. some use a simple extra-articular versus intra-articular divide, while others adopt a broader extrinsic/intrinsic distinction that groups tendinopathy, referred hip, spine and ankle pathology and complex regional pain syndrome together under "extrinsic" causes.[4,5] This review instead adopts a three-tier structure intra-articular, extra-articular/referred and centralized/non-mechanical, because collapsing referred and centralized pain into one category obscures two genuinely different diagnostic pathways: referred pain demands investigation of a separate anatomic joint, whereas centralized pain demands a non-structural, mechanistically distinct workup entirely.

Timeline is the second essential axis, since the differential shifts substantially with time from surgery. Early on (under 3 months), pain more often reflects acute infection, inadequate intraoperative soft-tissue balancing, prosthesis malposition or soft-tissue impingement.4 Between 3 and 12 months, flexion and mid-flexion instability, patellofemoral maltracking and evolving low-grade infection move to the fore.[6] Beyond a year, aseptic loosening, wear-associated synovitis and ligamentous attenuation predominate, along with centralized pain, often present from the outset but masked by acute postoperative

pain, becomes easier to isolate once structural causes have been progressively excluded.[4,6]

This timeline dependence is not merely descriptive. A meta-analysis of pain at 3, 6 and 12 months after TKA found that psychological and preoperative factors account for a growing share of pain relative to technical and structural factors as time from surgery increases, direct evidence for stratifying the differential by timeline rather than presenting a single undifferentiated list.[7] The clinical stakes are real: applying a late-stage differential to an early presentation or the reverse, drives both unnecessary investigation and missed diagnoses and is a recognized contributor to the diagnostic delays exceeding 12 months reported in this population.[8]

Intra-articular causes

Component malrotation and malposition represent among the most frequently implicated and most frequently missed, intra-articular causes of persistent pain in a knee that appears well-aligned on standard radiographs. Standard anteroposterior and lateral radiographs assess coronal and sagittal alignment but do not reliably capture rotational malalignment, which requires CT-based assessment; this is a critical diagnostic blind spot, since a knee can be "well-aligned" by routine radiographic criteria while harbouring clinically significant rotational malposition.[9] Internal rotation of the femoral component, in particular, has been consistently associated with patellofemoral maltracking, anterior knee pain and patellar complications and excessive internal rotation of the tibial component has independently been shown to negatively affect clinical outcomes across a systematic review of the topic.[10,11] Even small, individually unremarkable degrees of femoral and tibial malrotation can be cumulative and rotational malalignment has been directly linked to chronic pain or early clinical failure even in the absence of other identifiable structural abnormality.[12]

Residual soft-tissue imbalance and instability form a second major intra-articular category and one with direct relevance to the static-versus-dynamic alignment distinction discussed in this review series: a knee can meet conventional static alignment targets while remaining functionally unbalanced through the flexion arc. Flexion instability, characterized by subjective giving-way, recurrent effusion and excessive anteroposterior laxity at 900 flexion, is an increasingly recognized cause of otherwise unexplained pain and dissatisfaction, particularly with cruciate-retaining designs.[9,13] Mid-flexion instability, i.e. laxity occurring specifically between 300 and 900 rather than at either extremity of the arc, is a related but distinct entity, with patient-, implant- and technique-related risk factors identified across a dedicated systematic review; because this instability manifests only within the mid-range and not at the extension or 900 checkpoints conventionally examined clinically, it is disproportionately likely to be missed on routine assessment of a "stable-appearing" knee.[14]

Patellofemoral pathology beyond rotational malalignment, including an un-resurfaced or maltracking patella, patellar clunk syndrome and overstuffing of the patellofemoral joint, remains among the most common sources of anterior knee pain after TKA and should be systematically assessed regardless of coronal alignment status.[15] Low-grade periprosthetic joint infection must be actively excluded in any painful TKA workup, since it can present indolently without classic signs of acute infection; the 2018 International Consensus Meeting criteria provide the current evidence-based diagnostic standard and should be applied even when clinical suspicion is low.[16] Aseptic loosening and polyethylene wear-associated synovitis, though more typical of the late postoperative period, complete this differential, loosening is diagnosed through progressive radiolucent lines or migration on serial radiographs, while wear-associated synovitis typically presents with a delayed, often painless effusion and requires aspiration and synovial analysis to distinguish it from indolent infection.

Extra-articular and referred sources

Extra-articular and referred pathology accounts for a clinically significant proportion of persistent pain following TKA and is frequently under-investigated because clinical attention naturally gravitates toward the operated joint itself. In a series of 122 aseptic painful TKAs with symptoms unresolved beyond one year, 32% were ultimately attributed to extra-articular causes, with nerve entrapment referable to the spine accounting for the largest single subgroup, followed by hip osteoarthritis or femoral head avascular necrosis.[17] A decision-tree-based evaluation of unexplained painful TKA similarly found that once intra-articular causes were systematically excluded, extra-articular and referred sources, i.e. hip, spine and peripheral nerve pathology among them accounted for the majority of eventually identified diagnoses.[18] Referred pain from hip osteoarthritis is a particularly important trap, since hip pathology can mimic anterior or medial knee pain closely enough to be misattributed to the arthroplasty itself and a focused hip examination (including internal rotation provocation) should be routine in any painful TKA workup regardless of how confidently the knee itself appears normal.[19]

Lumbar spine pathology, particularly nerve root compression at L3-L4 or L4-L5, can likewise present as anterior or lateral knee pain and should be considered especially when the pain distribution does not correspond cleanly to a single intra-articular structure.[17,19]

Hindfoot and ankle malalignment represents a less routinely considered but biomechanically well-grounded extra-articular contributor. Correction of coronal knee deformity during TKA measurably alters the hip-knee-ankle axis and this change does not occur in isolation: in a prospective series of 108 varus knees, hindfoot alignment shifted significantly by three months postoperatively as the hindfoot adjusted from a compensatory valgus position

toward neutral, with the magnitude of hindfoot change correlating with the degree of knee coronal correction, particularly in younger patients.[20]

This has a direct clinical implication for the painful "well-aligned" knee: a knee corrected to a neutral mechanical axis leaves the previously compensating hindfoot to undergo its own realignment process and an incompletely or asymmetrically compensating hindfoot can generate altered loading through the newly aligned knee, presenting as persistent pain despite an unremarkable knee radiograph. Clinical examination of hindfoot posture and, where indicated, weight-bearing hindfoot alignment imaging should therefore be considered a routine component of the extra-articular workup rather than a subspecialty afterthought, particularly in patients who underwent TKA for significant preoperative varus deformity. Vascular and neurogenic causes complete this category. Vascular claudication can mimic activity-related knee pain and is distinguished by its relief with rest regardless of limb position, in contrast to mechanical knee pathology.[19] Peripheral nerve entrapment, including infrapatellar branch neuroma and, less commonly, obturator nerve syndrome, should be considered particularly when pain has a burning, radiating or dysesthetic quality rather than a mechanical pattern. Complex regional pain syndrome type 1 (CRPS-1), while uncommon, represents a distinct and important diagnosis within this category, presenting with disproportionate pain, autonomic changes and hypersensitivity and accounting for a small but clinically significant minority of otherwise unexplained painful TKA cases.

Centralized and non-mechanical pain

Central sensitization, a state of amplified central nervous system pain processing that persists independent of ongoing peripheral tissue pathology, has emerged as a significant and previously underrecognized contributor to persistent pain after TKA. A systematic review and meta-analysis found that markers of central sensitization are associated with inferior postoperative pain and functional outcomes following TKA and more granular work distinguishing central sensitization from neuropathic pain found that patients with both conditions had significantly worse patient-reported outcomes than those with either condition alone or with neither, indicating a cumulative rather than interchangeable relationship between the two mechanisms.[3,21] This distinction matters clinically: central sensitization and neuropathic pain are frequently conflated in practice, yet they represent different underlying mechanisms and may warrant different management approaches. Validated screening tools allow this centralized component to be identified preoperatively rather than inferred only after a structural workup has been exhausted. The Central Sensitization Inventory (CSI), a self-reported questionnaire with an established cutoff score of 40, is the most widely used instrument for this purpose, while the painDETECT Questionnaire identifies a neuropathic pain component using a cutoff score of 19. Incorporating either or both instruments into preoperative assessment allows patients at elevated risk of centralized, non-structural pain to be identified before surgery, informing both preoperative counselling and postoperative expectation management rather than leaving this mechanism to be discovered only after structural causes have been serially excluded.

Psychosocial predictors of dissatisfaction operate alongside and partly overlap with, centralized pain mechanisms. Pain catastrophizing, a cognitive-affective pattern characterized by rumination, magnification and helplessness in response to pain, has been consistently identified as a risk factor for chronic pain after TKA across a dedicated systematic review.[22] Preoperative expectation mismatch represents a related but distinct contributor; patients with central sensitization have been shown to hold significantly higher preoperative expectations of surgical outcome than non-sensitized patients, creating a compounding risk in which the patients most biologically predisposed to persistent postoperative pain also enter surgery with the outcome expectations least likely to be met. Recognizing this overlap is clinically important, since addressing expectation mismatch through preoperative counselling alone, without also addressing the underlying centralized pain processing, is unlikely to fully resolve postoperative dissatisfaction in this subgroup.

Diagnostic algorithm

The algorithm proceeds through five sequential steps. A positive finding at any step redirects toward targeted management for that specific etiology; a negative finding moves the workup forward rather than triggering premature revision surgery. Infection screening is placed first and applies regardless of clinical suspicion, since indolent PJI can mimic every downstream category.

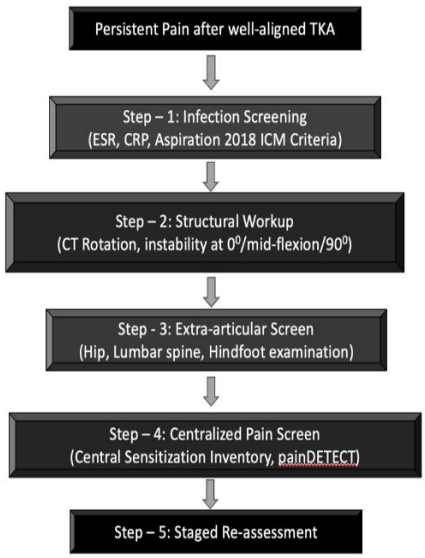


Figure 1: Stepwise diagnostic algorithm for the assessment of persistent pain after technically successful, well-aligned total knee arthroplasty.

Centralized pain screening is deliberately positioned as a defined, mandatory step using the CSI and painDETECT, rather than a diagnosis reached only after every structural avenue is exhausted, reflecting the growing evidence that centralized mechanisms are often present from the outset rather than emerging only once other causes are ruled out. If no cause emerges by the end of the algorithm, staged clinical reassessment is preferred over proceeding directly to revision, since unexplained pain has been consistently associated with poor revision outcomes (Figure 1).

Management implications by diagnosis

Once a specific diagnosis is reached through the algorithm, first-line management follows the identified etiology rather than a generic "revision if unimproved" pathway. Confirmed periprosthetic joint infection is managed according to chronicity: acute infection is treated with debridement, antibiotics and implant retention where feasible, while chronic or indolent infection typically requires staged revision with an antibiotic spacer.[16,23] Structural intra-articular causes are managed according to the specific lesion identified: rotational malposition or flexion/mid-flexion instability confirmed on targeted workup generally requires revision arthroplasty, whereas patellofemoral maltracking without significant rotational malalignment may respond to targeted physiotherapy or lateral release before revision is considered.[23,24]

Extra-articular and referred causes are managed at their source rather than through knee-directed intervention: hip pathology and lumbar radicular causes are referred to the appropriate subspecialty and hindfoot malalignment identified as a contributor is managed with orthotic correction or, in persistent cases, dedicated hindfoot realignment rather than further knee-directed treatment.[23] Peripheral nerve-related pain, when confirmed, may respond to targeted nerve decompression or neuroma management in refractory cases where conservative measures have failed.[19]

Centralized pain identified through CSI or painDETECT screening requires a fundamentally different first-line approach: multimodal pain management addressing central sensitization directly (rather than continued structural investigation), combined with realistic expectation-setting, since this subgroup is disproportionately unlikely to benefit from further surgical intervention directed at the knee itself. Across all categories, patients in whom the full algorithm yields no identifiable cause should be managed conservatively and reassessed longitudinally, since revision surgery performed for unexplained pain carries a well-documented risk of failing to resolve symptoms and should be avoided as a diagnosis of last resort.

Limitations and future directions

Several limitations constrain the current evidence base on persistent pain after well-aligned TKA. First, there is no consensus definition of what constitutes clinically significant "persistent" or "unexplained" pain: studies vary in their use of minimal clinically important difference (MCID) and patient acceptable symptom state (PASS) thresholds, timing of assessment and outcome instruments, making direct comparison across studies difficult and limiting the strength of any pooled conclusions. Second, existing diagnostic algorithms, including the one proposed here, have generally been validated only retrospectively against cohorts that ultimately proceeded to revision surgery, introducing selection bias toward patients with identifiable structural pathology; prospective validation across a broader population, including patients managed conservatively, has not yet been performed. A promising future direction lies in more granular characterization of pain itself as a diagnostic signal. Systematic characterization of pain quality, location and aggravating factors in a cohort of unhappy TKA patients identified distinct pain patterns correlating with specific underlying pathologies; for instance, particular pain qualities and localization patterns tracking closely with instability versus patellar-related causes along with subsequent correlation of these pain patterns with SPECT/CT bone tracer uptake findings has begun to validate pain characterization as an objective diagnostic adjunct rather than a purely subjective symptom.[25,26]

Incorporating structured pain-pattern characterization into future diagnostic algorithms, alongside validated centralized-pain screening instruments, represents a natural next step toward a more standardized, objective and prospectively validated diagnostic pathway for this challenging patient population.

CONCLUSION

Persistent pain after radiographically well-aligned TKA remains common and diagnostically challenging, spanning intra-articular, extra-articular/referred, and centralized mechanisms that shift with time from surgery. A structured, timeline-informed algorithm, beginning with infection exclusion and structural workup, extending to extra-articular and hindfoot assessment, incorporating centralized pain screening as a defined step rather than a diagnosis of exclusion, offers a more systematic approach than ad hoc investigation. Revision surgery without a clear diagnosis should be avoided; staged reassessment remains preferable when the cause stays elusive.

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