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A structured evaluation of the symptomatic medial Oxford unicompartmental knee arthroplasty (UKA)

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- Unicompartmental knee arthroplasty (UKA) has several advantages over total knee arthroplasty; however, in many reports, the risk of revision remains higher after UKA.
- Many reasons for failure of UKA exist.
- Successful treatment starts with accurate assessment of the symptomatic UKA as a specific mode of failure requires a specific solution.
- A structured and comprehensive evaluation aids assessment of the symptomatic UKA.
- This review provides an overview of the causes for a symptomatic medial UKA, its risk factors, diagnostic modalities that can be used, and briefly discusses treatment options.

Keywords: arthroplasty; UKA; unicompartmental

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Introduction

Suggested advantages of unicompartmental knee arthroplasty (UKA) over total knee arthroplasty (TKA) are more natural knee kinematics, greater range of motion, lower complication and mortality rates, higher satisfaction, faster recovery, and better patient-reported outcomes.[1] However, revision rate has often been reported to be higher after UKA (7% at 10-year for the medial Oxford UKA[2]). The five most common reasons for revision of a medial Oxford UKA are: lateral disease progression (1.4%), aseptic loosening (1.3%), bearing dislocation (0.58%), pain (0.57%), and infection (0.47%).[3] Proper patient selection is key for success; ideal candidates present with painful isolated bone-on-bone anteromedial osteoarthritis with a correctable deformity and intact ligaments.[3]

We explored the current literature to provide an overview of the causes for a symptomatic UKA, its risk factors and diagnostic modalities that can be used, and we briefly discuss treatment options.

Methods

The review protocol was registered on PROSPERO (CRD42019123249). On 28 January 2019, we searched the PubMed, Embase, and Cochrane libraries using the keywords: medial AND unicompartmental AND knee arthroplasty including synonyms, restricted to English papers published after 2000 (Appendix 1). This cut-off was used because of recent UKA innovations.

We identified 2,067 papers (Fig. 1). Titles and abstracts were screened to identify potentially relevant papers describing: prevalence, definition, cause/risk factors, diagnostic modalities, and corresponding treatment options for symptomatic, failed, and/or revision UKA. We excluded papers concerning lateral, patellofemoral, and bi/tricompartmental UKA. We focused on the Oxford (Zimmer-Biomet, Warsaw, IN, USA) medial UKA (cemented/uncemented congruent mobile bearing UKA) as this prosthesis accounts for the largest market share. Despite the focus on this specific implant, we did not limit ourselves and included papers that were relevant, but described a different UKA brand, and indicated this where applicable.

The descriptive nature of this review did not allow for any statistical analysis or data pooling; therefore, no quality appraisal was deployed. Title and abstract screening identified 216 potentially relevant papers. All full texts were read by two reviewers and 102 papers were deemed useful. Per cause of symptomatic UKA, we described – where relevant – the definition, its probability, history and symptoms, physical examination, laboratory investigations, imaging and other possible diagnostic modalities, and briefly discuss treatment. Causes were ordered based on probability.

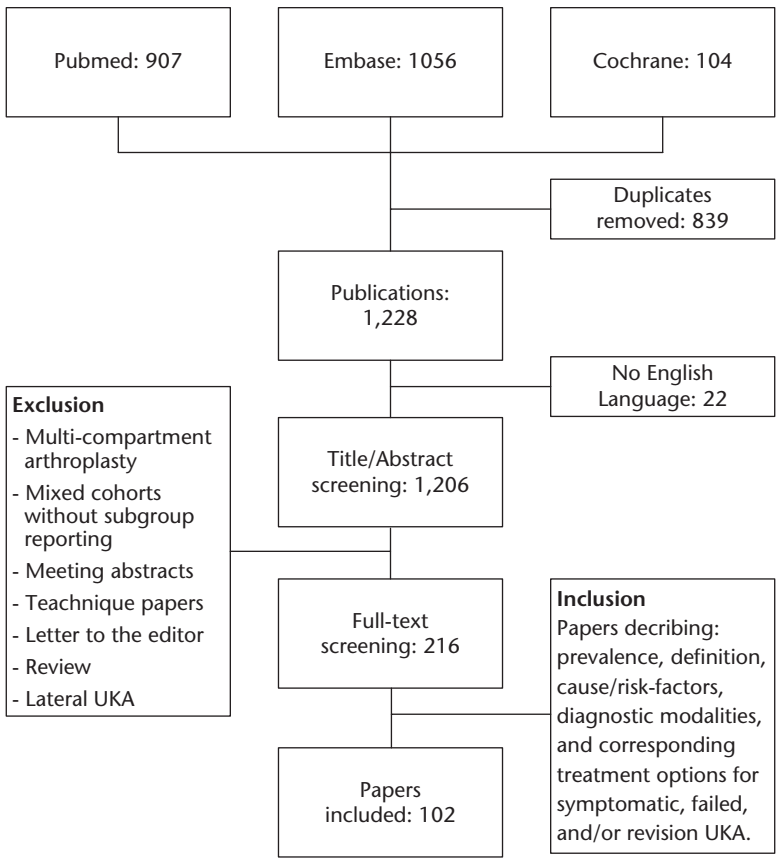

Fig. 1 Flowchart demonstrating number of papers identified per library and article selection using predefined criteria at each stage.
Note. UKA, unicompartmental knee arthroplasty.

Results

Progression of osteoarthritis

Lateral progression of osteoarthritis is the most common reason for revision after UKA, with an overall incidence rate of 1.4% (123 revisions/8,658 cases that were pooled in a meta-analysis with mean follow-up of 4–12 years).[2] Despite this low rate, it should be noted that about 1/4 of all UKA revisions are for lateral osteoarthritis progression.[2,4] Patellofemoral disease progression is a rare (incidence rate: 0.02%) reason for revision.

Correct balancing and femoral–tibial alignment during UKA implantation is considered most important in preventing osteoarthritis progression.[5] A study that assessed the rate of progression in the preserved compartments included 114 medial UKAs (iBalance, Arthrex, Naples, FL, USA; fixed-bearing UKA).[6] They demonstrated that about half of medial UKAs had approximately one Kellgren Lawrence grade worsening in the lateral compartment over five years. Interestingly, women had more progression, while age, body mass index (BMI), and alignment had no effect.[6] It remains unknown whether radiographic progression is associated with symptoms. A different study did find a relationship of mechanical alignment with lateral disease progression.[7] The authors included 156 medial fixed-bearing UKAs (Lotus Mark 1, GUEPAR Group, Plerin, France) and demonstrated that valgus overcorrection lead to lateral joint space narrowing.[7] Burnett et al demonstrated (n = 467) an increased revision risk in valgus overcorrection, explained by enhanced osteoarthritis progression.[8]

On the other hand, a radiographic study demonstrated an improved congruency of the lateral compartment and increased joint space width (± 0.9 mm) after medial UKA without overcorrection. They conclude that a UKA positively affects congruency and potentially limits lateral osteoarthritis progression.[9]

Two biomechanical studies by Heyse et al used cadaveric models with an Accuris (fixed-bearing, Smith & Nephew, Memphis, TN, USA) UKA[5] and an Oxford UKA[10] to measure alignment, medial collateral ligament (MCL) strain, and lateral compartment force, and to compare varying bearing thickness. They demonstrated that overstuffing leads to significantly more valgus and MCL strain; however, peak contact stress in the lateral compartment did not consistently increase. Based on these findings, overstuffing should be avoided as it induces high MCL strains and changes kinematics; although the effect on

lateral compartment stress and ultimately disease progression remains unclear.[5,10] However, MCL attenuation over time might lead to valgus and subsequent osteoarthritis progression.

A meta-analysis by Ro et al demonstrated a higher revision rate for lateral disease progression in Western compared to Asian patients, which they explain by way of a higher rate of constitutional varus in Asian patients.[11]

Retained cement is a rare, but preventable, cause of cartilage damage in the lateral compartment as demonstrated in a case report.[12]

Mofidi et al studied the effects of significant preoperative radiographic patellofemoral and lateral compartment osteoarthritis on function, pain, and revision in 134 patients who underwent Mako UKA (fixed-bearing, Surgical Corporation, Fort Lauderdale, FL, USA).[13] They found significant improvement in knee function. However, they did find a higher rate of persistent pain in the patellofemoral and lateral compartment osteoarthritis groups as compared to those patients with non-osteoarthritic preserved compartments. They also found a higher rate of revision in patients with significant patellofemoral osteoarthritis.[13] This impact of patellofemoral osteoarthritis is contradicted by others; Beard et al assessed the influence of patellofemoral osteoarthritic changes on postoperative function after medial UKA in 824 knees and found that only lateral facet bone loss was associated with worse function.[14] They concluded that damage to the patellofemoral joint to the extent of full-thickness cartilage loss is not a contraindication for UKA,[14] which is supported by others.[15-18] This might be explained by improved congruence of the patellofemoral joint after UKA.[19]

Kendrick et al assessed the impact of a focal cartilage defect on the medial side of the lateral femoral condyle – likely caused by tibial spine impingement due to tibiofemoral subluxation – on function after UKA in 769 knees and found no difference in (change of) knee function based on the degree of focal cartilage loss.[20] Hence, focal cartilage loss at the medial side of the lateral femoral condyle is not a contraindication for a medial UKA, and should therefore also not be a reason for revision.

Magnetic resonance imaging (MRI) is reliable for the assessment of cartilage, meniscus, tendon, and ligaments after a zirconium UKA (Accuris);[21] However, its correlation to symptoms and clinical relevance requires additional study.

Revision to a TKA seems to be the contemporary option for lateral and patellofemoral disease progression. However, isolated lateral compartment osteoarthritis progression could be treated using lateral UKA, creating a bicompartmental UKA.[22] Pandit et al described a series including 27 knees in 25 patients that received a lateral UKA – 8.1 years – after medial UKA, which significantly improved knee function.[22]

Based on the above studies, serial radiographs are useful to establish disease progression. We believe that further elucidating the aetiology of lateral disease progression merits additional study. In addition, better understanding of radiographic correlation of osteoarthritis progression to symptoms is needed. The clinical relevance of other imaging modalities (e.g. MRI) for assessment of the preserved cartilage also merits further study.

Aseptic loosening

Aseptic loosening is one of the major reasons for revision after UKA with an estimated incidence rate of 1.3% (108 revisions/8,658 pooled cases).[2] Several possible explanations for aseptic loosening exist, including: micromotion preventing osseointegration, malalignment causing eccentric loading, inadequate cortical coverage, and polyethylene wear.[23,24]

Radiolucent lines in arthroplasty are often considered indicative of loosening. However, narrow (< 2 mm), well-defined, non-progressive radiolucency that is bordered by parallel radiodense lines is not associated with symptoms or future UKA loosening.[25,26] Hence, these lines are termed physiological. Gulati et al demonstrated that 62% out of 161 UKAs had such radiolucent lines around the tibial component without an association with poor outcome. In addition, they found no factors (activity, BMI, alignment) associated with development of physiological radiolucent lines.[25] Interestingly, Pandit et al demonstrated a much lower rate of physiological radiolucent lines after uncemented UKA (7%) versus cemented (75%) UKA in an RCT.[22] A subsequent prospective study corroborated this and demonstrated an incidence of 1.5% in 196 uncemented UKAs.[27] It is important to obtain fluoroscopically aligned parallel radiographs to improve assessment of these lines.[28,29] For cemented components; a prospective study by Clarius et al demonstrated better cement penetration and fewer radiolucent lines in UKAs after pulse versus syringe lavage.[30]

The single-peg Oxford Phase-3 UKA femoral component has been modified to a twin-peg design with the aim of improving stability. Two biomechanical studies using cadaveric knees demonstrated no difference in subsidence or micromotion, but improved load to failure for the twin-peg versus the single-peg design.[31,32] Peg design might play a role in aseptic loosening, but clinical studies are lacking.

Decrease in bone marrow density is hypothesized to play a role in implant migration and loosening.[33-36] However, studies that assess change in tibial bone mineral density after UKA demonstrate conflicting results.[35] Richmond et al demonstrated preservation of bone marrow density two years after UKA (n = 50, 26 of which were Oxford UKAs);[35] whereas Scott et al demonstrated decrease in greyscale ratio (as a proxy for bone mineral density) over

time for 173 Phase-3 Oxford UKAs.[34] Both studies did not assess the relationship of change in bone mineral density with loosening, which would have been interesting. A different study by Lee et al demonstrated that trabecular bone density is lowest posteriorly, under the tibial component, emphasizing the need for cortical support.[24]

Wong et al studied the diagnostic value of bone scintigraphy for assessment of loosening. Eleven out of 39 symptomatic UKAs demonstrated loosening intraoperatively; none of the scintigraphy features was associated with loosening.[37] MRI might be useful for assessment of the component–bone interface (in the Accuris & Stryker UKA, Mahwah, NJ, USA) but further study to establish its correlation to loosening is needed.[38,39]

Based on the above studies, radiolucent lines could be monitored with serial fluoroscopically aligned radiographs to assess whether they are progressive. If radiolucent lines are unchanged at two years, one can consider these non-progressive and not a source of symptoms. A deep flexion compared to an extension view can demonstrate a loose femoral component in some cases. Infection should always be considered in case of a loose implant. We do not recommend bone scintigraphy. The usefulness of MRI, but also other imaging modalities such as SPECT (single-photon emission computed tomography) needs to be assessed in future studies.

Bearing dislocation

Bearing dislocation is one of the more common reasons for revision after UKA with an estimated incidence of about 0.58% (50 revisions/8,658 pooled cases),[2] with reported rates up to 5.3%.[11,40]

Mechanisms leading to dislocation include deep flexion, injury, rolling over during sleep;[41] however, most patients do not recall an event.[42,43] Several types of bearing dislocation have been reported, including: anterior (most common),[40] posterior,[40,42] into the condylar notch or subluxation onto the lateral wall of the tibial tray,[41,43] subluxation by upward tilting of the bearing on the medial side,[44] a few cases with a bearing fracture with one piece anteriorly and one piece posteriorly displaced,[40,45] and 180° spin.[46] Spontaneous relocation of a posterior dislocation has been reported.[47] Anterior and posterior dislocations are mostly caused by flexion-extension gap mismatch, malalignment, impingement, ligament (MCL) attenuation, wear, or a combination.[40,48] A dislocation into the notch or jumping of the bearing onto the lateral wall is caused by excessive force on the medial side of the bearing – possibly combined with overhang of the bearing over the medial tibial tray edge – resulting in upward and lateral movement of the bearing.[41,43] Tilting of the bearing is found to be caused by too lateral placement of the femoral component, forcing the bearing to laterally impinging against the lateral wall of the tibial tray.[44] This can be avoided by correctly positioning the components: the tibial vertical cut should be just medial to the apex of the medial spine; the femoral intramedullary rod should be placed in the medial border of the notch (not central) with the femoral drill guide aiming at the middle third of the femoral condyle and checking for lateral wall-to-bearing clearance with trial components.[44,49] Risk factors for dislocation include a decrease in posterior tibial slope,[48] and Asian versus Western population.[11] Dislocations can lead to: metallosis, polyethylene wear, and component loosening.[42] Prompt diagnosis and treatment is warranted. Radiographs readily demonstrate the dislocated bearing including its direction. Symptoms include: pain, instability, inability to bear weight, locking, and clicking noise. Physical examination can show restricted range of motion, instability, a palpable bearing, and effusion; however, a normal exam does not rule out dislocation.[42,43,50]

It is imperative to recognize the type of bearing dislocation in order to understand its cause and select treatment. Radiographs, mechanism of injury, and 'reading' the explanted bearing during surgery (i.e. looking for damage to the bearing) can help determine its aetiopathogenesis.

Treatment options include: closed reduction,[41] open bearing exchange with concomitant assessment and treatment of impingement, conversion to a TKA in case of malalignment, flexion-extension gap mismatch or ligament attenuation/injury.[40] One could even consider (case report) leaving a dislocated bearing in place and placing a new one in a posteriorly dislocated bearing that cannot be retrieved.[51]

Infection

Infection is one of the more common reasons for revision of a UKA, but with an incidence rate of 0.47% (41 revisions/8,658 pooled cases) it is still relatively uncommon.[2] Large cohort studies present infection rates of 0.35–0.6%.[52,53]

We identified only one study, by Labruyère et al, that specifically investigated infection after UKA. This study included nine patients with chronic infection who underwent one-stage revision to TKA with antibiotic therapy started intraoperatively (based on cultures from preoperative joint aspiration) and continued for 12 weeks. Patients had follow-up until two years: no patient experienced recurrence or required revision.[54] Based on these findings, Labruyere et al recommend to identify the causative organism first, and to treat a chronic infection with a total synovectomy and a one-step UKA-to-TKA conversion combined with 12 weeks of antibiotics.[54]

We feel that – despite the limited evidence of periprosthetic joint infection (PJI) in UKA – diagnosis and treatment should follow general PJI guidelines from the Musculoskeletal Infection Society.[55]

(Peri)prosthetic fracture

A periprosthetic fracture is a relatively rare complication after UKA, with an estimated incidence rate of about 0.16% (14 revisions/8,658 pooled cases), with reports up to 1%.[2,56] The fracture often occurs during surgery and the tibia is more commonly affected than the femur.[57] This complication – when caused intraoperatively – is considered a technical error, and several explanations exist, including: a deep vertical cut into the posterior proximal tibia,[58,59] insufficient keel slot preparation,[2] blow-out during keel slot preparation when hitting the posterior cortex with the groove cutter, use of a heavy hammer,[57] multiple pin holes (> 2) for the tibial cutting jig,[60] valgus inclination of the tibial component,[58] and low bone mineral density.[61]

A finite element analysis demonstrated increased risk of medial tibial plateau fracture with increasing valgus and with extended sagittal bone cut in the posterior cortex.[61] Intraoperative and postoperative tibia fractures can be diagnosed on radiographs and treatment has been described using various options: a buttress plate with implant retention, revision using TKA with augments and/or stems, and non-operative treatment in a cast.[62-66]

Only a few case reports describe periprosthetic femoral condyle fractures: one which occurred during surgery, and two which occurred during falls after one and three years.[67-69] The first case (hoffa-type coronal fracture) occurred during surgery, but was only noticed directly thereafter and was successfully treated in a non-weight-bearing cast for six weeks.[69] The other two cases concerned AO-B2 type displaced medial condyle fractures diagnosed on radiographs and successfully treated surgically with closed reduction and percutaneous cannulated screw fixation.[67,68]

A rare complication – nowadays – is fracture of the metallic or bearing component. Most of these pertain to older designs.[70–71,72]

Instability

Instability is a relatively rare complication after UKA with an estimated incidence rate of about 0.12% (10 revisions/8,658 pooled cases), with reports up to 0.7%.[2,73] However, one should recognize overlap in aetiopathogenesis with bearing dislocation.[74]

There is no clear definition about what constitutes instability. Symptoms include 'a feeling of instability' and 'giving way'. Physical examination – stress testing of the collateral and cruciate ligaments – should be performed and can support the diagnosis of ligamentous insufficiency. In addition, muscle strength should be assessed as weakness can lead to subjective rather than true instability. Patients should be asked about a history of trauma, acute versus chronic symptoms, onset of symptoms, and worsening of instability. Instability can be caused by ligament injury during surgery, or traumatic ligament rupture or avulsion. Stress testing of collateral and cruciate ligaments under fluoroscopy can be carried out to evaluate the degree and direction of instability, although studies reporting on its diagnostic accuracy are lacking.

Ten cases of instability leading to revision of a UKA have been described in four papers.[52,73-75] The timing of revision (described for 6/10 cases) ranged from one to nine years.[52,73,75] Instability was not further defined. The delayed nature suggests trauma, possibly of an already intraoperatively attenuated ligament. The type of revision (hinged TKA) has only been described for one case.[52]

The importance of the ACL (anterior cruciate ligament) for successful outcome of UKA has been extensively studied.[76-79] Biomechanical studies have demonstrated that anterior tibial translation and force in the ACL do not differ between a native (ACL-intact) knee and a (ACL-intact) knee with a UKA.[77,78] These studies also demonstrated that an ACL-deficient knee with a UKA has greater anterior tibial translation than one with an intact ACL.[77,78] However, fluoroscopic gait analysis demonstrated no difference in kinematics.[79] In addition, survivorship analysis of ACL-deficient versus ACL-intact UKAs did not show a difference.[76] The impact of delayed ACL rupture after UKA implantation is less clear.

Chronic hemarthrosis

Chronic or recurrent hemarthrosis can result in stiffness, poor function, and infection.[80] It is a rare complication after UKA as well as after TKA, with an incidence rate of 0.07% for UKA[2] and 0.3% in TKA.[80] We only encountered four papers reporting on chronic hemarthrosis in UKA: two case reports, one retrospective review of 31 patients with chronic hemarthrosis after knee arthroplasty (TKA in 29 and UKA in two), and one retrospective study reporting on the results of UKAs in 213 patients, describing three (1.4%) patients with chronic hemarthrosis.[81-84] Chronic hemarthrosis is defined as ≥ 2 bleeding episodes > 2 weeks after surgery.[80] Conservative treatment is often successful and should be considered first, and includes: joint aspiration, compression, rest, ice application, splinting, and discontinuation of antithrombotic therapy. Successful invasive options – in case of failure of conservative management – are angiographic embolization and open synovectomy.[81-84]

Polyethylene wear

Polyethylene wear by itself is a rare reason for revision, with an incidence rate of only 0.06% (five revisions/8,658 pooled cases).[2] However, excessive wear may cause osteolysis and subsequent loosening which is a common cause of revision.[2] We feel that, not necessarily the treatment of wear, but predominantly understanding its

cause and how to minimize wear, is important. Older UKA designs demonstrated high failure rates due to wear (up to 22% of all UKA revisions in registries). This has predominantly been attributed to fixed incongruent bearing designs with limited areas of contact and to poor quality polyethylene.[70,85]

Evidence from retrieval studies of normally functioning Oxford UKA (Phase-1 and 2) bearings demonstrated a mean wear of 0.01 mm/year.[85,86] Such a low rate will not cause in vivo penetration or alter alignment (±1° degree varus per 1 mm wear).[85] However, several factors can accelerate wear: impingement (5x higher wear), pitting of the bearing, polyethylene quality (2x higher wear in Phase-1 vs. Phase-2 UKA).[85,86] Retrieval studies have demonstrated that bearing thickness does not influence wear.[85,86] This is corroborated by the finding that implant survival is not reduced in thin bearings.[87]

Studies using roentgen stereophotogrammetric analysis (RSA) have demonstrated a mean linear wear of 0.02–0.05 mm/year for the Phase-2 and Phase-3 medial UKA at 10–20 years.[88-90] Older implant designs (Phase-1 vs. Phase-2 UKA) demonstrated significantly more wear; however, this might be explained by alteration in femoral bone preparation (milling instead of saw cuts) reducing the risk of anterior impingement rather than the implant design on itself. A randomized controlled trial (RCT) demonstrated no difference in wear as measured by RSA between cemented and uncemented UKA at five years.[88] They did find a significant association of bearing overhang with increased wear.[88]

Knee wear simulator and finite element studies demonstrate increased wear with: joint line deviation,[91] ACL transection,[78,92] and limited tibial slope.[93,94] In addition, many studies hypothesize substantial effect of debris (e.g. cement) on wear.

Based on the findings above, symptomatic wear in terms of polyethylene debris causing aseptic loosening, extreme thinning of the bearing, or bearing fracture can – at least in part – be considered a technical error at implantation. It is critical to avoid impingement, achieve a stable articulation, and avoid debris at implantation. These factors, together with component fixation, should be assessed when replacing a bearing for wear.

Malposition

Malposition on its own is a rare reason for revision, with an incidence rate of only 0.03% (three revisions/8,658 pooled cases).[2] However, malposition is associated with most reasons for UKA revision described above, including: osteoarthritis progression, loosening, bearing dislocation, periprosthetic fracture, instability, and polyethylene wear. This paragraph is a more general treatise on UKA (mal) position.

Optimal implant position of the medial Oxford UKA is 0° (±10°) femoral component varus/valgus measured relative to the tibial axis, and 0° to 15° femoral component flexion measured relative to the femoral anatomical axis, 0° (±5°) tibial component varus/valgus and 7° (±5°) tibial component slope, both measured relative to the tibial axis.[95,96] Holme et al studied the reliability of measuring tibial component orientation using low-dose three-dimensional computerized tomography (3D CT) versus radiographs.[97] They found somewhat – but not significant – better reliability for assessment of tibial component varus/valgus, rotation, and slope on 3D CT. However, all measures demonstrated good agreement, also on radiographs.[97] For assessment of tibial component rotation, Akagi's anteroposterior (AP) line (medial edge of patellar tendon to middle of posterior cruciate ligament (PCL) can be used as rotational reference for the tibial component on CT.[98-100]

Three finite element analysis studies assessed the impact of tibial component alignment on stress distribution in the tibia. They consistently demonstrated that neutral alignment in the coronal plane is most optimal in terms of strain on the native tibia.[101-103]

Several studies assessed the effect of component orientation on function. Gulati et al included 211 UKAs with a four-year follow-up and divided patients into 2.5° intervals of femoral and tibial varus/valgus, flexion/extension, and tilt. Ninety-eight per cent of femoral components, and 92% of tibial components were within the indicated tolerance ranges.[95] Within these ranges, there were no significant differences in function between groups.[95] Kamenaga et al demonstrated worse function in patients with tibial component valgus, lower placement of the tibial component, and more tibial component external rotation.[104,105] Wahal et al studied the influence of bearing position and excursion on function in 30 patients. They found less bearing excursion, and a more posterior bearing position in patients with worse function.[106] A clinical study comparing single-peg versus twin-peg UKAs demonstrated better femoral component orientation of the twin-peg design.[107]

Malposition can be best assessed on fluoroscopically aligned anteroposterior and lateral radiographs. Long-leg radiographs are useful to assess the tibiofemoral angle, and CT scan can be used to measure tibial component rotation.

Miscellaneous

Other suggested explanations for a symptomatic UKA include: tibial bone strain, MCL strain, tibial cyst,[108] residual osteophytes,[18] snapping pes syndrome (including bursitis),[44] neuroma, and Baker's cyst. In addition, one should consider pathology outside of the knee, such as referred pain from the hip, and radiculopathy.

Pain due to bone strain is commonly localized over the anteromedial aspect of the proximal tibia. The elevated strain can be caused by: implant malposition (coronal malalignment, tibial component overhang, low tibial resection level), increased load, and bone properties.[103] The pain often settles within one year because of bony remodelling.

Kumar et al investigated the outcomes after UKA in patients with and without chondrocalcinosis and found no difference.[109] Another rare reason for symptoms was suggested in a study by Lisowski et al, namely a patient who was allergic to cobalt. The patient was successfully revised to a titanium TKA four years after UKA.[110]

Despite the numerous pathophysiological explanations for symptoms after UKA, there remains a subgroup of patients with unexplained pain. This vexing problem does not justify revision as studies demonstrate persistent poor outcome after revision for unexplained pain.[111]

Conclusion

This review aims to improve understanding of the aetiology of a symptomatic UKA, its risk factors, and useful diagnostic modalities. Helpful diagnostic tools to evaluate the symptomatic UKA are summarized in Table 1.

Table 1. Overview of diagnostic modalities useful for assessment of the symptomatic UKA

Cause of symptomatic UKA	Diagnostic modalities
Lateral and patellofemoral disease progression	Serial anteroposterior radiographs for lateral disease progression. Skyline view for patellofemoral disease progression. Long-leg radiographs for alignment.
Aseptic loosening	Serial fluoroscopically aligned anteroposterior and lateral radiographs. Consider presence of (low-grade) infection.
Bearing dislocation	Anteroposterior and lateral radiographs.
Infection	Physical examination to assess presence of a fistula. Erythrocyte sedimentation rate and serum C-reactive protein. Cultures including sensitivity. Synovial leucocyte and neutrophil count.
(Peri)prosthetic fracture	Anteroposterior and lateral radiographs for diagnosis. CT scan for more precise assessment of the fracture if needed.
Instability	Physical stress testing of the collateral and cruciate ligaments. Anteroposterior and lateral radiographs.
Chronic hemarthrosis	Angiography in recurrent cases.
Polyethylene wear	Serial fluoroscopically aligned anteroposterior and lateral radiographs.
Malposition	Fluoroscopically aligned anteroposterior and lateral radiographs, long-leg radiographs for assessment of tibiofemoral angle, CT scan for assessment of rotation.
Miscellaneous	MRI might be useful for specific indications (e.g. lateral meniscus injury, avascular necrosis).

Note. UKA, unicompartmental knee arthroplasty; CT, computerized tomography; MRI, magnetic resonance imaging.

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SUPPLEMENTAL MATERIAL

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