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Introduction

Articular cartilage defects can be debilitating for patients and difficult for an orthopedic surgeon to treat. They often present in a young athletic population after injury but can also occur following chronic mechanical stress causing degeneration or alongside metabolic disorders of the subchondral bone [1]. Because articular cartilage has low regenerative potential, invasive procedure must often be performed to attempt to recreate the articular surface. If left untreated, focal chondral defects can often progress to osteoarthritis. However, many chondral defects are asymptomatic and incidentally found using advanced imaging techniques [1]. Deciding when to intervene and how to approach each individual scenario is what makes these cases challenging.

Surgeons should follow a patient-centered approach to treating cartilage defects as it is important to consider all factors involved, including the defect characteristics, imaging findings, and patient profile and goals. All of these various factors impact the appropriate management strategy that can range from non-operative treatments

such as physical therapy and intra-articular injections to operative treatments such as debridement chondroplasty, microfracture, collagen scaffold-augmented microfracture, autologous chondrocyte implantation, osteochondral autograft transplant, and osteochondral allograft transplantation. Additionally, concomitant pathology such as meniscal deficiency or malalignment can predispose patients to failure or recurrence and must be addressed either concomitantly or in a staged fashion. Each therapeutic option can be successful when appropriately used. It is imperative to approach each case from all angles to determine the best option for that specific patient.

Clinical Evaluation and Chondral Defect Diagnosis

Clinical History

A thorough clinical history is critical to providing a patient-centered approach to treatment of articular cartilage lesions. Among the factors important to understand in the patient's history are duration of symptoms (acute or chronic), mechanism of injury (direct trauma, twisting, or insidious), symptom severity, symptom quality (sharp, focal, dull, or diffuse), and associated symptoms (clicking, locking, swelling, or instability). Additionally, paying attention to exacerbating

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57 factors, functionality, and patient habits can
58 provide a better understanding of the patient's
59 experience.

60 Patients with symptomatic cartilage lesions
61 will typically have pain that is worse with load
62 bearing and isolated to the compartment contain-
63 ing the chondral defect. Some patients will experi-
64 ence effusions associated with activity, but
65 symptoms do not always correlate with severity
66 of cartilage damage. There is currently no evi-
67 dence to support the treatment of asymptomatic
68 chondral defects, so clinical correlation with
69 arthroscopic or radiologic findings is critical in
70 the management of these patients.

71 Patient goals and performance demands are
72 extremely useful in determining appropriate
73 patient-centered management. Return to sport or
74 work versus return to normal daily activities can
75 play a pivotal role in deciding between operative
76 or non-operative management. The authors
77 highly recommend extensive communication
78 between the patient and provider about the goals
79 of therapy to provide mutual understanding and
80 an appropriate management plan.

81 **Physical Exam**

82 Physical examination of the knee in a patient
83 with a suspected cartilage defect should confirm
84 the symptomatic presentation. Thorough exami-
85 nation should begin with observation of gait and
86 any apparent gross muscular deficiencies fol-
87 lowed by a complete assessment for pathology
88 and specific muscle imbalances. In particular,
89 malalignment should be assessed as it can place
90 increased forces through a specific compartment
91 and contribute to pathology. Malalignment may
92 need to be addressed surgically to redistribute
93 forces in order to increase chances of a successful
94 outcome and prevent recurrence. Lachman, pivot
95 shift, anterior drawer, posterior drawer, and varus
96 and valgus stress testing should be performed
97 because ligamentous injury and instability can
98 often accompany cartilage damage. Assessing
99 the knee for effusion and range of motion may

help identify limitations that point to the severity 100
of intra-articular pathology. Evaluation of the 101
meniscus should also be performed to identify 102
possible concomitant pathology. 103

Diagnostic Imaging 104

Imaging techniques are critical in the diagnosis 105
and management of cartilage damage. 106
Radiographs should be used to assess for osteoar- 107
thritis as severe osteoarthritis can be a contraindi- 108
cation for many cartilage restoration procedures. 109
This may indicate the need for management via 110
arthroplasty assuming non-operative manage- 111
ment has been exhausted. The tibiofemoral joint 112
should be evaluated using weight-bearing antero- 113
posterior and flexion posteroanterior radiographs, 114
whereas the patellofemoral joint is better evalu- 115
ated with Merchant and lateral views. Weight- 116
bearing full-length extremity radiographs are 117
necessary to evaluate possible malalignment that 118
may require surgical correction via an off-loading 119
osteotomy. 120

Radiographs have low sensitivity for the diag- 121
nosis of focal chondral defects which makes 122
magnetic resonance imaging (MRI) critical in the 123
diagnosis of this pathology. In addition to evalu- 124
ating the articular cartilage, MRI allows for iden- 125
tification of meniscus or ligamentous pathology 126
in addition to subchondral bone involvement, 127
osteochondritis dissecans, avascular necrosis, 128
and fracture. The size and characterization of 129
focal chondral defects can be evaluated with two- 130
dimensional fat suppression and three- 131
dimensional fast spin echo sequences, while the 132
quality of the cartilage itself can be evaluated 133
with gadolinium enhancement. Despite the utility 134
of these advanced imaging techniques, the find- 135
ings must be correlated with clinical symptoms, 136
and diagnostic arthroscopy remains the gold 137
standard for evaluation of intra-articular pathol- 138
ogy and relating it to patient-specific complaints, 139
symptoms, and signs present on physical 140
examination. 141

142 **Diagnostic Arthroscopy**

143 Diagnostic arthroscopy and intra-articular
 144 debridement is the gold standard for diagnosis of
 145 chondral defects and is often the best initial step
 146 in the management. In some patients, this proce-
 147 dure may be therapeutic allowing for delayed
 148 treatment of the cartilage defect and other comor-
 149 bidities. In other patients, arthroscopy allows for
 150 a thorough intra-articular evaluation of the liga-
 151 ments, meniscus, and articular surface providing
 152 index information for definitive treatment recom-
 153 mendations. During arthroscopy, chondral defect
 154 size can be measured and graded based on depth
 155 and appearance according to the Outerbridge or
 156 International Cartilage Repair Society (ICRS)
 157 criteria (Table 10.1, Fig. 10.1), to best determine
 158 the appropriate management. The dimensions of
 159 the chondral defect should be measured accu-
 160 rately as size plays an important role in determin-
 161 ing which treatment options are indicated and
 162 most likely to be successful [2]. However, defect
 163 size coupled with knowledge of prior treatments,
 164 patient goals and expectations, and the status of
 165 the subchondral bone will also play pivotal roles
 166 in the decision-making related to definitive
 167 treatment.

Table 10.1 Chondral defect grading criteria

Outerbridge criteria	ICRS criteria	
Grade 0: Normal cartilage	Grade 0: Normal cartilage	t1.1
Grade 1: Mild cartilage softening or swelling	Grade 1: Superficial lesions, soft indentation, or superficial fissures	t1.2
Grade 2: Fraying or fissuring extending less than 50% of cartilage depth	Grade 2: Lesions extending less than 50% of cartilage depth	t1.3
Grade 3: Partial thickness loss with focal ulceration greater than 50% of cartilage thickness	Grade 3a: Lesions extending greater than 50% of cartilage depth	t1.4
Grade 4: Full-thickness chondral defect with exposed subchondral bone	Grade 3b: Lesions extending greater than 50% of cartilage depth down to calcified layer	t1.5
	Grade 3c: Lesions extending greater than 50% of cartilage depth down to subchondral bone	t1.6
	Grade 3d: Lesions extending greater than 50% of cartilage depth with blisters	t1.7
	Grade 4: Full-thickness chondral defect extending into subchondral bone	t1.8

ICRS International Cartilage Repair Society t1.9

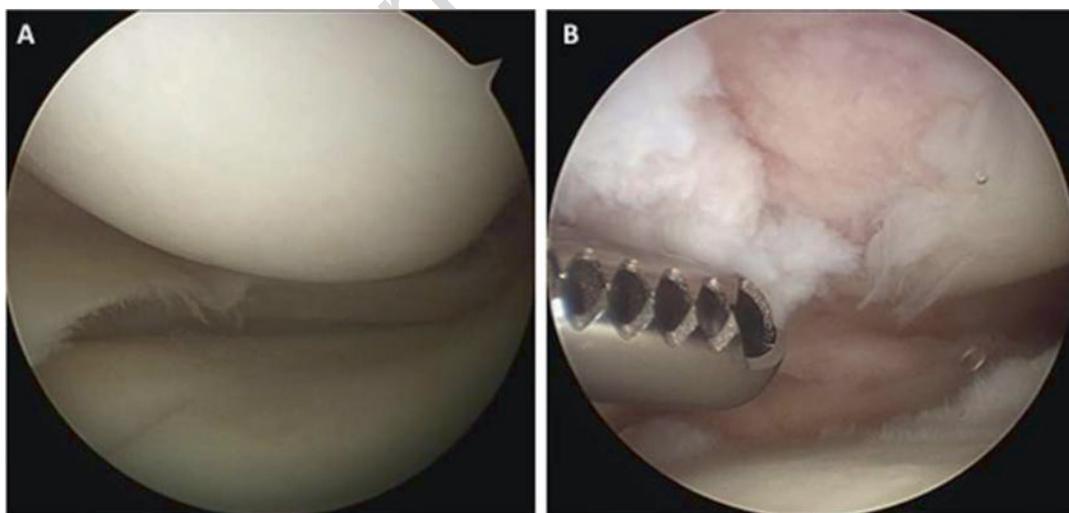


Fig. 10.1 Focal chondral defect. Intraoperative arthroscopic images during left knee arthroscopy demon-

strating (a) normal cartilage of the medial femoral condyle and a (b) grade IV focal chondral defect of the patella with exposed subchondral bone

168 **Factors Contributing to Complexity** 169 **of Chondral Defect Management**

170 The complexity of cartilage repair and restoration
171 surgery is multifaceted and extends far beyond
172 the technical difficulties of performing proce-
173 dures such as microfracture, microfracture with
174 collagen scaffold augmentation, autologous
175 chondrocyte implantation (ACI), or osteochon-
176 dral grafting. The factors contributing to com-
177 plexity are wide ranging including patient
178 demographics, chondral defect characteristics,
179 and concomitant pathology (Figs. 10.2 and 10.3).
180 In order to provide patients with the greatest
181 chance of a successful outcome, it is necessary to
182 incorporate all of these factors into the decision-
183 making process.

184 **Demographics**

185 The patient presenting with a focal chondral
186 defect has many inherent factors worth consider-
187 ing when determining a treatment plan including
188 age, duration of symptoms, body mass index
189 (BMI), occupation, goals of treatment, and smok-
190 ing status [3]. Among various cartilage restora-
191 tion procedures, including osteochondral
192 allografts and autologous chondrocyte implanta-
193 tion, younger age, particularly less than 30 years
194 old, has been associated with better outcomes
195 and lower rates of failure than older patients [4–
196 6]. Additionally, one study reported that patients
197 with a BMI >35 were four times more likely to
198 have unsuccessful outcomes after osteochondral
199 allograft transplantation [7]. In studies investigat-
200 ing the outcomes of ACI and matrix-induced ACI
201 (MACI), longer duration of symptoms has been
202 found to be negatively correlated with successful
203 outcomes [8, 9]. Factors such as these are impor-
204 tant to consider because they can help predict
205 which patients will benefit from various forms of
206 management.

207 Patient occupation or hobbies along with their
208 goals of treatment are critical to determining the
209 appropriate management. Some patients may be
210 looking to avoid surgical management in which
211 case physical therapy, nonsteroidal

anti-inflammatory medications, and intra-articu- 212
lar joint injections with corticosteroids, 213
viscosupplementation, or biologics may be the 214
best course of treatment. Additionally, some 215
patients may be professional athletes or highly 216
active recreational athletes looking to return to 217
sport, whereas others may simply hope to return 218
to their normal daily activities. An athlete's joints 219
undergo significant load-bearing stress during 220
sport and may require a more durable treatment 221
than nonathletes. It is important to consider all 222
available factors to determine the best patient- 223
centered treatment plan. 224

Patients are educated to understand that most 225
treatments might lead to some residual symptoms 226
with higher-level activities. In addition, choosing 227
enduring solutions that can tolerate ballistic 228
activities or collision sports such as isolated oste- 229
otomy or osteotomy with osteochondral allograft 230
transplantation and potentially avoiding a menis- 231
cal allograft when otherwise required are a con- 232
sideration at times in higher-level athletes. 233
Ultimately, the greatest challenge is determining 234
the least amount of surgery to encourage a satis- 235
factory outcome and properly match the patient's 236
expectations. 237

238 **Defect Location**

The location of a focal chondral defect greatly 239
impacts the treatment decision-making process. 240
Femoral condyle lesions are the most common 241
types of chondral defects encountered in the knee 242
[10]. These are followed by lesions seen in the 243
tibial and patellofemoral compartments [10]. 244
Given the load-bearing nature of the tibiofemoral 245
compartment, these lesions may require more 246
durable treatment options such as osteochondral 247
allografts, depending on the lesion's other charac- 248
teristics and the patient-specific factors. Lesions of 249
the patella or trochlea have proven to be a difficult 250
clinical problem due to the complex shape of the 251
patellofemoral articular surface and often concomi- 252
tant joint instability. While recent studies indicate 253
successful outcomes with osteochondral allograft 254
transplants, there is ongoing discussion regarding 255
management of these lesions with osteochondral 256

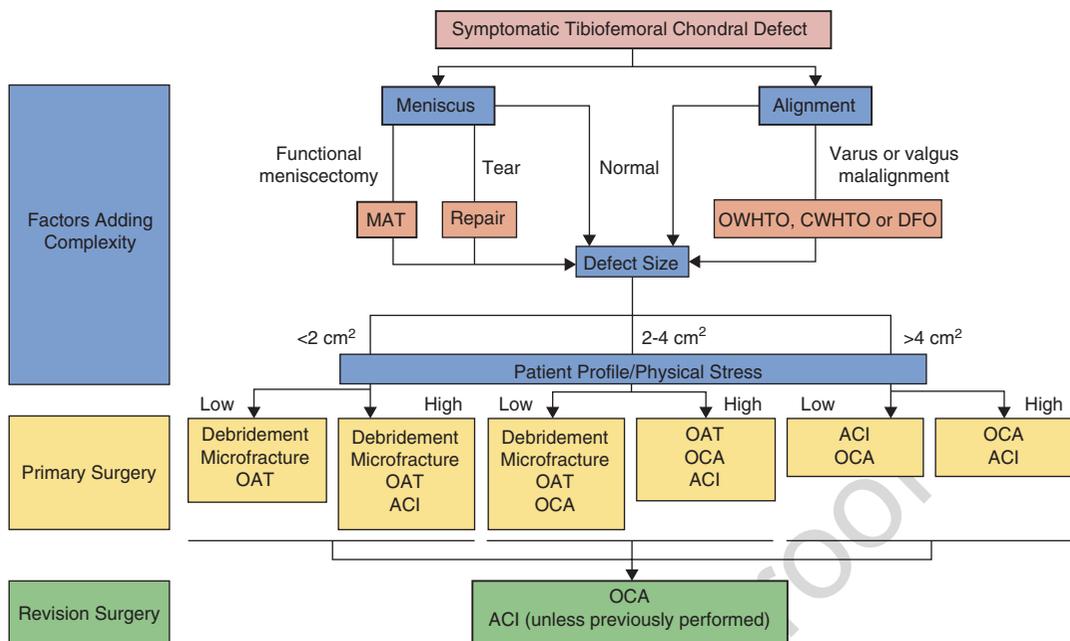


Fig. 10.2 Management of symptomatic tibiofemoral focal chondral defects. Blue represents factors that add complexity to surgical management including meniscal status, coronal plane alignment, patient profile, and, most importantly, defect size. Orange represents procedures that can be performed concomitantly or in a staged fashion to address these factors. Yellow represents primary

surgical options based on all factors considered. Green represents options for surgical revision if necessary. OWHTO, opening wedge high tibial osteotomy; CWHTO, closing wedge high tibial osteotomy; DFO, distal femoral osteotomy; OAT, osteochondral autograft transplantation; ACI, autologous chondrocyte implantation; and OCA, osteochondral allograft

257 allografts due to the difficulty matching the shape
 258 of the articular surface [11, 12]. This leads many
 259 surgeons to prefer surface allograft transplantation
 260 (i.e., ProChondrix, AlloSource, Denver CO;
 261 Cartiform, Arthrex, Naples, FL; DeNovo NT,
 262 Zimmer/Biomet, Warsaw, IN) or cell-based thera-
 263 pies such as ACI or MACI for management of
 264 these lesions. As the literature documenting our
 265 real-world experience improves, knowledge of the
 266 best treatment modality for each lesion location
 267 will likely be elucidated.

268 Defect Size

269 Defect size factors into treatment decision-
 270 making because the efficacy of various treat-
 271 ments for chondral defects changes depending
 272 on the size of the lesion. Small lesions (<math>< 2 \text{ cm}^2</math>)
 273 can be managed successfully with an initial
 274 debridement with the possible addition of
 275 microfracture, which allows the defect to be

257 filled with fibrocartilage. Since fibrocartilage is
 258 not as durable as innate articular cartilage,
 259 microfracture is less successful when treating
 260 larger defects [13, 14]. Depending on other
 261 patient factors such as athletic participation,
 262 osteochondral autograft may be an appropriate
 263 treatment for small defects as well. Medium-
 264 sized defects (2–4 cm^2) may have variable out-
 265 comes with microfracture treatment and may be
 266 better treated with an osteochondral allograft,
 267 osteochondral autograft, surface allograft, or
 268 even ACI/MACI because they are more durable
 269 solutions. Treatment for the largest defects is
 270 limited to osteochondral allograft transplant or
 271 ACI/MACI due to durability and defect-filling
 272 capabilities (Fig. 10.4) [6, 10]. Osteochondral
 273 autograft or mosaicplasty is often not an ideal
 274 option in these larger defects due to donor site
 275 morbidity [15]. As a result, accurate defect mea-
 276 surement complemented by advanced imaging
 277 and diagnostic arthroscopy is critical for appro-
 278 priate surgical planning.

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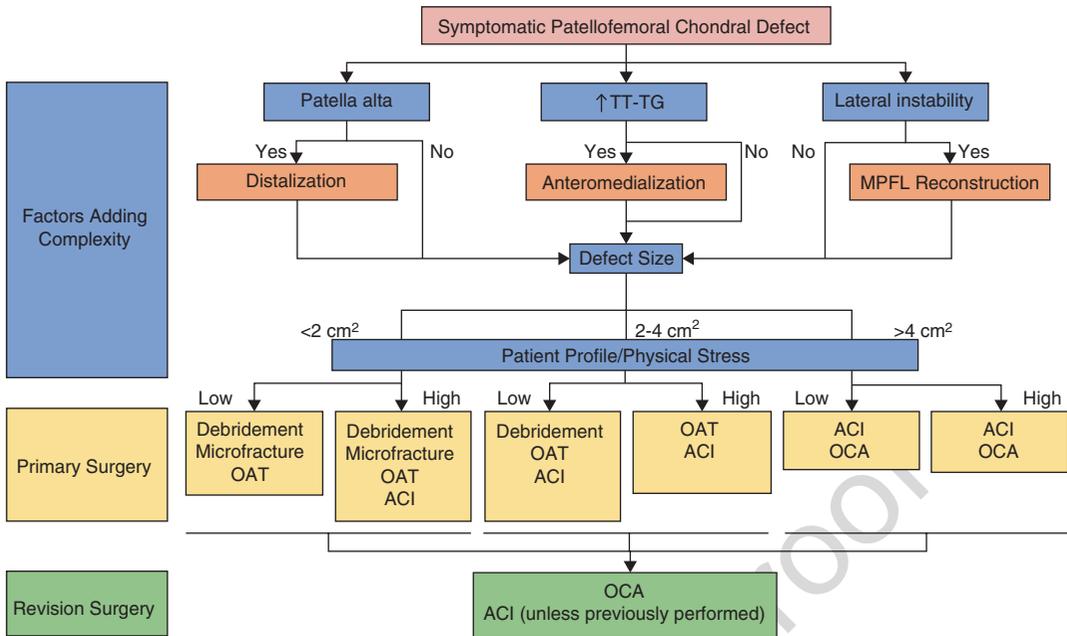


Fig. 10.3 Surgical management of symptomatic patellofemoral focal chondral defects. Blue represents factors contributing to case complexity including patella alta, tibial tubercle to trochanteric groove (TT-TG) distance, lateral instability, patient profile, and, most importantly, defect size. Orange represents procedures that can be performed to address these layers of complexity either con-

comitantly or in a staged fashion. Yellow represents primary surgical management options given the factors considered. Green represents options for surgical revision if necessary. TT-TG, tibial tubercle to trochanteric groove distance; MPFL, medial patellofemoral ligament; OAT, osteochondral autograft transplant; ACI, autologous chondrocyte implantation; and OCA, osteochondral allograft



Fig. 10.4 Osteochondral allograft for treatment of large focal chondral defect. (a) Right knee arthroscopic intraoperative images of a large area (>4 cm²) of grade III/IV chondral changes of the medial femoral condyle. (b) The

same cartilage damage after arthrotomy prior to treatment. (c) Large defect of the medial femoral condyle treated with an osteochondral allograft

298 **Bipolar Disease**

299 Bipolar articular cartilage lesions are defined as
 300 lesions of reciprocal cartilage surfaces such as the
 301 medial tibia and medial femoral condyle or the
 302 patella and trochlea. This poses a unique clinical
 303 challenge because inadequate treatment can lead
 304 to accelerated development of osteoarthritis and
 305 definitive treatment options limited to arthroplasty
 306 [16]. The management of bipolar chondral defects
 307 has been investigated with several treatment
 308 options. Gomoll et al. reported significant clinical
 309 improvement and no difference in the outcomes
 310 between patellofemoral unipolar and bipolar chon-
 311 dral defects treated with ACI [17]. Osteochondral
 312 allograft transplantation has been investigated in
 313 both the tibiofemoral and patellofemoral bipolar
 314 lesions as it provides a location-matched recon-
 315 struction of the articular cartilage and subchondral
 316 bone. Success rates for bipolar osteochondral
 317 allograft transplants range from 40 to 53% with
 318 failure rates up to 46% [16]. Bipolar OCA in the
 319 patellofemoral joint has a lower failure rate than in
 320 the tibiofemoral, likely due to the load-bearing
 321 nature of the tibiofemoral joint [16]. Patients with
 322 grafts that survive, however, show significant clinical
 323 improvement. The high failure rates complicate
 324 management of these lesions because the
 325 patient is at elevated risk of not improving and
 326 being subjected to additional surgery.

327 **Meniscal Deficiency**

328 The meniscus and articular cartilage have a sym-
 329 biotic relationship that cannot be ignored when
 330 managing chondral defects (Fig. 10.5). Intra-
 331 articular changes, particularly increased contact
 332 pressures and cartilage degeneration over time,
 333 have been well documented in the literature when
 334 patients are meniscal deficient [18, 19]. If a repair-
 335 able meniscus tear is present at the time of surgery,
 336 the meniscal repair should be performed as
 337 part of a combined procedure. If cartilage proce-
 338 dures are performed in patients who are meniscal
 339 deficient, those increased contact pressures are
 340 applied to the implanted chondrocyte, graft, or
 341 developing fibrocartilage which may complicate
 342 the outcome. It is therefore critical that a thorough

evaluation of the meniscus is performed during 343
 preoperative planning to determine if a meniscal 344
 allograft transplant is necessary in addition to the 345
 cartilage procedure. 346

Malalignment 347

Joint malalignment can occur within either the 348
 tibiofemoral joint in the form of varus or valgus 349
 deformity or the patellofemoral joint with patella 350
 maltracking or upstream version abnormalities. 351
 Varus or valgus deformity creates an unbalanced 352
 distribution of body weight that places increased 353
 stress on the medial or lateral compartment, 354
 respectively. If malalignment is not addressed, 355
 the patient is predisposed to having failure of 356
 their cartilage procedure either due to the 357
 absence of sufficient symptom reduction or due 358
 to catastrophic failure of the cartilage resurfacing 359
 procedure [20]. It can be corrected surgically 360
 to off-load the joint at the time of cartilage treat- 361
 ment with either a distal femoral osteotomy or 362
 high tibial osteotomy (Fig. 10.6). Patellar insta- 363
 bility or maltracking becomes particularly prob- 364
 lematic during knee flexion such as squatting or 365
 climbing stairs of the knee when contact pres- 366
 sures between the patella and trochlea increase. 367
 Different factors effecting patellar loading such 368 [AU2](#)
 as patella alta and lateral positioning of patella 369
 associated with an increased tibial tubercle to 370
 trochanteric groove/posterior cruciate ligament 371
 distance can be treated with tibial tubercle distal- 372
 ization or tibial tubercle anteromedialization. At 373
 times, the patient may also have recurrent lateral 374
 patellar instability, which is managed by medial 375
 patellofemoral ligament (MPFL) reconstruction 376
 and associated surgery as indicated. The senior 377
 author (B.J.C) prefers to treat malalignment as a 378
 combined procedure, but it can also be managed 379
 in a staged fashion. The advantages of realign- 380
 ment alone are that it is very durable and can tol- 381
 erate high-level athletic activities without 382
 concerns for graft compromise. The disadvan- 383
 tage is that it simply may not be “enough” sur- 384
 gery to satisfy a patient’s objectives and each 385
 surgery comes with muscle debilitation and the 386
 risk of excessive scar formation as well as 387
 interfering with “life.” 388

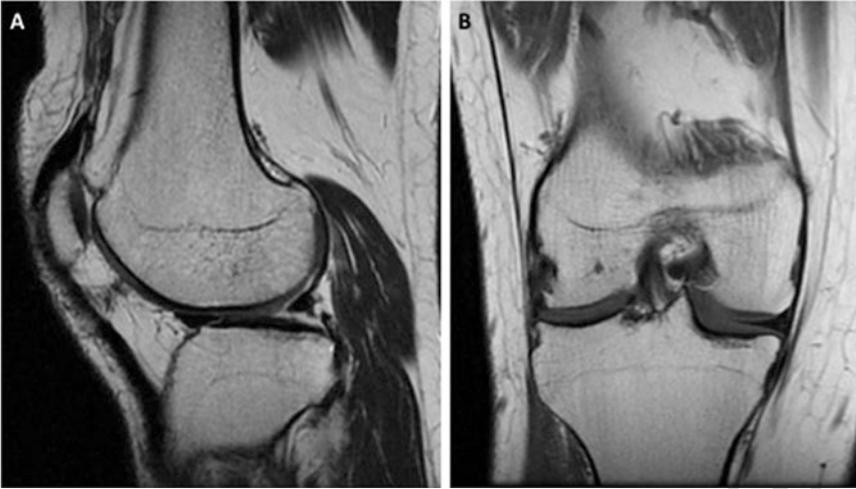


Fig. 10.5 Meniscus deficiency requiring meniscal allograft transplant. (a) T2-weighted sagittal plane MRI of the right knee showing the lateral tibial plateau, lateral femoral condyle, and anterior and posterior horns of the

lateral meniscus. (b) T1-weighted coronal plane MRI of the right knee showing meniscal deficiency in the lateral compartment

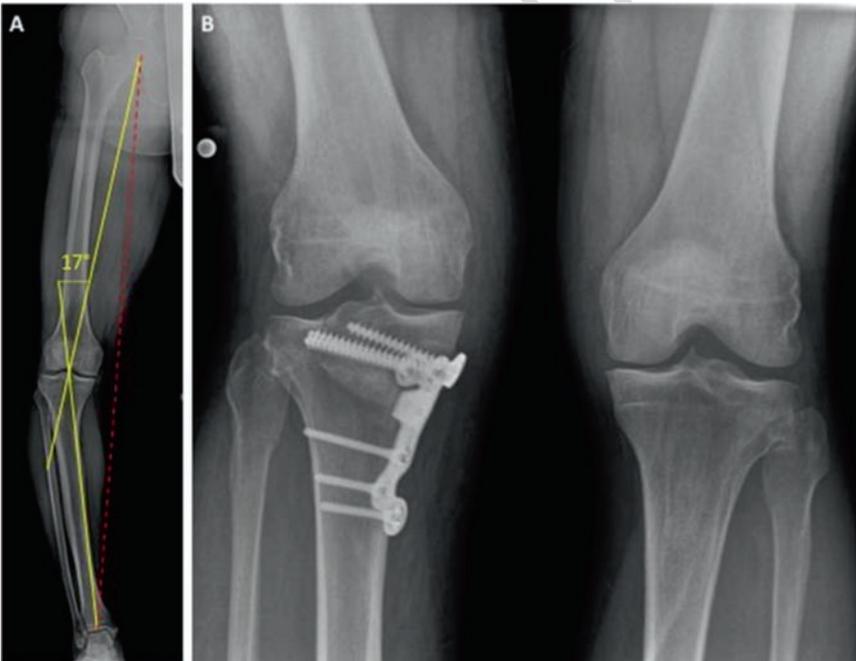


Fig. 10.6 Coronal malalignment corrected by opening wedge high tibial osteotomy. (a) Standing weight-bearing anteroposterior radiograph of the right knee demonstrating varus deformity causing excessive mechanical stress on the medial compartment. Yellow lines indicate the ana-

tomomic axes of the femur and tibia, while the red dashed line indicates the mechanical axis of the right lower extremity. Patient was calculated to have 17° of varus deformity. (b) Postoperative skier's view radiograph showing the varus deformity corrected by opening wedge high tibial osteotomy

389	Complex Cases		
390	Meniscal Deficiency with Femoral		
391	Condyle Defect		
392	As described above, meniscus evaluation is	restoration/repair except for a higher reoperation	435
393	essential when determining an appropriate man-	rate [21]. The clinical outcomes measured by	436
394	agement plan in patients with a femoral condyle	combinations of Lysholm, KOOS, IKDC, Tegner,	437
395	defect due to the symbiotic relationship between	Modified HSS, and SF-36 scores improved sig-	438
396	the meniscus and articular surface. In patients	nificantly, and the overall failure rate was 12%	439
397	with a symptomatic femoral condyle defect who	[21]. Overall, surgical management of femoral	440
398	have had a prior ipsilateral subtotal meniscec-	condyle chondral defects with concomitant MAT	441
399	tomy, a meniscal allograft transplant (MAT) is	provides predicable successful outcomes for	442
400	indicated in addition to the cartilage procedure to	management of this combined pathology.	443
401	reduce the contact pressures on the treated carti-		
402	lage site. Multiple MAT techniques have been	Chondral Defect with Ligamentous	444
403	described including the bridge-in-slot, bone plug,	Injury	445
404	dovetail, and soft-tissue only techniques, but the	Incidental findings of cartilage defects are com-	446
405	senior author (B.J.C.) prefers the bridge-in-slot	mon at the time of planned knee ligament recon-	447
406	technique for both medial and lateral	struction, but they add complexity to the patient's	448
407	MAT. Treatment of the cartilage defect should be	management. When determining the appropriate	449
408	determined by the same algorithm as an isolated	treatment plan, it is critical to determine if the	450
409	cartilage defect, primarily based on defect size	chondral defect is symptomatic. In the setting of	451
410	and expected stress. Small defects (<2 cm ²) can	an acute ligamentous injury, chondral defects are	452
411	be managed with debridement or microfracture	presumed to be asymptomatic and typically	453
412	(with or without adjunct scaffolds and biologics	treated with a simple debridement. However, in a	454
413	such as BioCartilage (Arthrex, Inc., Naples, FL),	chronic ligamentous injury, chondral defects are	455
414	while medium sized (2–4 cm ²) will likely require	more likely to be symptomatic resulting from the	456
415	surface treatment with cartilage allografts	inherent joint instability. As the time between	457
416	(Cartiform, ProChondrix, and DeNovo NT),	ligamentous injury and surgical management	458
417	OATS, or OCA, and large defects (>4 cm ²) are	increases, the frequency and severity of pain and	459
418	likely best treated with OCA or ACI/MACI.	cartilage or meniscus pathology tend to increase	460
419	Combined MAT and cartilage restoration pro-	[22–24]. When managing a chronic ligamentous	461
420	cedures have been well described in the literature	injury, therefore, it is typically preferred to per-	462
421	with excellent, reliable outcomes. When done in	form a combined procedure to also definitively	463
422	combination, MAT is performed first to prevent	address the chondral defect according to the typi-	464
423	iatrogenic damage to a newly restored cartilage	cal algorithm.	465
424	surface. The senior author (B.J.C.) prefers an		
425	open arthroscopic technique when performing	Chondral Defect with Malalignment	466
426	MAT, whereas the cartilage restoration procedure	Within the tibiofemoral joint, varus and valgus	467
427	is then performed using the appropriate tech-	deformity in the knee place increased mechanical	468
428	nique for the indicated treatment (i.e., arthroscopic	stress on the medial and lateral compartments,	469
429	for MFx or ACI versus open for OCA). A	respectively. Varus deformity can be corrected	470
430	systematic review evaluating six studies with a	with opening wedge high tibial osteotomy	471
431	total of 110 patients at mean follow-up of	(OWHTO) to off-load the medial compartment,	472
432	36 months who underwent combined MAT and	while valgus deformity can be corrected with	473
433	cartilage restoration/repair surgery found out-	closing wedge high tibial osteotomy (CWHTO),	474
434	comes similar to those for isolated cartilage	distal femur osteotomy (DFO), or proximal lat-	475
		eral opening tibial varus osteotomy [25] to	476

477 off-load the lateral compartment. The
 478 patellofemoral joint can be off-loaded with a
 479 Fulkerson modified Maquet (anterior) or
 480 Fulkerson (anteromedial) tibial tubercle osteot-
 481 omy. Patients with uncorrected malalignment
 482 have less successful clinical outcomes after carti-
 483 lage procedure [26]. This has made concomitant
 484 cartilage and realignment procedures increas-
 485 ingly popular, especially in comparison to less
 486 desirable alternatives such as unicompartmental
 487 arthroplasty in the young patient.

488 The results of combined osteotomy and carti-
 489 lage surgery have been shown to reliably provide
 490 symptomatic relief and improved functional sta-
 491 tus. A recent systematic review of 18 studies by
 492 Kahlenberg et al. compiled a total of 827 patients
 493 who underwent combined HTO and cartilage
 494 repair or restoration surgery with at least 2-year
 495 follow-up. They reported clinical improvement
 496 and a complication rate of 10.3%. The rate of
 497 conversion to arthroplasty was 6.3% with a range
 498 of mean time from HTO to conversion of 4.9–
 499 13.0 years [27]. Overall, the recent literature sup-
 500 ports concomitant HTO and cartilage surgery for
 501 this pathology with reliably successful
 502 outcomes.

503 **Meniscus Injury, Chondral Defect,** 504 **and Malalignment**

505 Meniscus injury is known to predispose patients
 506 to the development of cartilage injury [18, 19].
 507 When meniscal deficiency is combined with
 508 malalignment, the increased stress on the medial
 509 or lateral compartment can lead to severe, rapid
 510 cartilage degeneration. Traditionally, meniscal-
 511 deficient patients with chondral defects and con-
 512 comitant malalignment were thought to be
 513 contraindicated for MAT because the malalign-
 514 ment would prove to cause excess stress on the
 515 treated compartment. However, recent literature
 516 reports encouraging results in patients with this
 517 combined pathology undergoing distal femoral
 518 or high tibial osteotomy, MAT, and OCA.

519 Harris et al. reported on a cohort of 18 patients
 520 at mean 6.5-year follow-up who underwent com-
 521 bined distal femoral or high tibial osteotomy,

522 MAT, and OCA. Their patients showed significant
 523 clinical improvement by IKDC, Lysholm, and
 524 KOOS scores. Additionally, while there was a
 525 55.5% reoperation rate, the revision rate and rate
 526 of conversion to arthroplasty were both 5.6%
 527 [28]. Previously, Gomoll et al. reported on a
 528 cohort of seven patients in which they showed
 529 significant clinical improvement and six of seven
 530 patients were able to return to unrestricted activi-
 531 ties [29]. Despite the high reoperation rate, these
 532 results suggest that this triad of meniscal defi-
 533 ciency, malalignment, and femoral condyle chon-
 534 dral defect can be successfully managed without
 535 conversion to arthroplasty.

536 The senior author (B.J.C.) prefers to manage
 537 this triad with a combined procedure. The MAT
 538 is performed first due to the significant varus or
 539 valgus stress required for graft passage, place-
 540 ment, and fixation. Additionally, this prevents the
 541 possibility of iatrogenic injury to the treated
 542 articular surface. The cartilage procedure and
 543 realignment osteotomy can then be performed in
 544 the surgeon's order of preference. If ACI/MACI
 545 is the indicated cartilage treatment, however, it
 546 should be performed last to avoid disruption of
 547 the type I-III collagen or periosteal patch used to
 548 cover the implanted chondrocytes.

549 **Failed Prior Cartilage Restoration**

550 Patients presenting with a recurrence of symp-
 551 toms after a failed prior cartilage repair or resto-
 552 ration procedure present a unique challenge to
 553 the surgeon because the treatment options are
 554 limited. In the management of these patients, it is
 555 essential to investigate the reason for failure
 556 which could be untreated malalignment, strenu-
 557 ous patient activities, or improper rehabilitation
 558 so that appropriate adjustments can be made at
 559 the time of revision. Choice of revision treatment
 560 is dependent on the all of the same factors as the
 561 initial management, in addition to the type of
 562 index procedure performed. Revision treatment
 563 for the femoral condyle for a small defect after
 564 microfracture, for example, can be managed with
 565 OATS, while a large defect would be better man-
 566 aged with OCA. For the patella, revision of

567 defects treated with microfracture can be
 568 managed successfully with ACI/MACI or
 569 OCA. However, failed ACI/MACI of the patello-
 570 femoral joint should be managed with OCA. It is
 571 generally accepted that OCA is the best option
 572 for a salvage procedure when managing focal
 573 chondral defects [30–32]. ACI can also be used
 574 as a revision technique, but it has been shown to
 575 have a 3–5% higher failure rate than when used
 576 as a primary treatment [33, 34].

577 The outcomes of revision cartilage repair,
 578 especially with OCA, are reliably successful long
 579 term. Gracitelli et al. investigated the outcomes
 580 of OCA after failed microfracture surgery com-
 581 pared to OCA as the index procedure and found
 582 no difference in outcomes or failure rates between
 583 the two groups, although those with prior failed
 584 microfracture had a higher reoperation rate [30].
 585 Additionally, a subsequent study by Gracitelli
 586 et al. investigated outcomes of revision OCA
 587 after failed microfracture, OAT, or ACI. They
 588 reported a 16% failure rate at a mean time of
 589 2.6 years, but overall survivorship was 87.8% and
 590 82% at 5-year and 10-year follow-up, respec-
 591 tively. Their cohort showed significant clinical
 592 improvement and 89% satisfaction after their
 593 revision procedure [31]. These results are encour-
 594 aging for patients requiring revision surgery as
 595 conversion to arthroplasty can be delayed or pos-
 596 sibly avoided.

597 Conclusion

598 The orthopedic surgeon has several options avail-
 599 able for the treatment of symptomatic focal chon-
 600 dral defects. Many factors contribute to the
 601 complexity of managing chondral defects and
 602 must be considered. When deciding the appropri-
 603 ate therapeutic method whether operative or non-
 604 operative, it is critical that a thorough assessment
 605 of the patient's medical history, demographics,
 606 goals of treatment, symptoms, defect characteris-
 607 tics, imaging findings, and concomitant pathology
 608 is performed. Concomitant pathology such as
 609 meniscal deficiency, coronal malalignment, liga-
 610 mentous injury, and patellar instability must be
 611 addressed in either as staged or combined proce-

dures to avoid failure or symptom recurrence. 612
 When appropriately used, cartilage repair or res- 613
 toration procedures can provide successful out- 614
 comes even in the most complex cases. 615

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