Review Article

Acute Wound Complications After Total Knee Arthroplasty: Prevention and Management

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Abstract

Normal wound healing with avoidance of early wound complications is critical to the success of total knee arthroplasty. The severity of acute complications includes less morbid problems, such as quickly resolved drainage and small superficial eschars, to persistent drainage and full-thickness tissue necrosis, which may require advanced soft-tissue coverage. To achieve proper healing, surgeons must respond to persistent drainage by addressing modifiable patient risk factors, using meticulous surgical technique, and implementing an algorithmic approach to treatment.

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The incidence of wound complications after total knee arthroplasty (TKA) that requires further surgery is low, with 0.33% of >17,000 TKAs in a Mayo Clinic Total Joint Registry study needing surgical intervention within 30 days after the TKA.[1] Compared with the tissue encapsulation about the hip, the soft-tissue envelope surrounding the knee tolerates far less compromise before progressing to a complicated wound. For TKA patients who required acute returns to surgery to manage wound complications, the rate of deep infection at 2-year follow-up was 6.0%, and the rate for subsequent major surgery (ie, component resection, muscle flap coverage, amputation) was 5.3%.[1] In a related case-control study, Galat et al[2] found that acute returns to surgery within 30 days to specifically evacuate hematomas following TKA correlated with a 12.3% probability of needing additional major surgery within 2 years and a 10.5% probability of a deep infection. A history of a bleeding disorder was significantly associated with hematoma formation requiring surgery ($P = 0.046$). Comparatively, uncomplicated wounds following TKA required additional major surgery or had a deep infection at a rate of 0.6% and 0.8%, respectively, within 2 years.[1] These results highlight the importance of obtaining primary wound healing after TKA.

Persistent drainage is an important sign that a surgical wound may become problematic. Postoperative incisional drainage occurs in 1% to 10% of patients undergoing primary TKA.[3-5] After the skin heals, however, drainage should diminish or cease. Persistent wound drainage after TKA is defined as continued drainage from the surgical incision for >72 hours; this standard allows for earlier intervention and may therefore limit adverse consequences.[6] Drainage that continues beyond 1 week, whether light persistent drainage or massive acute effluence, is particularly concerning and typically requires surgical intervention. A disciplined approach is essential to discriminate among arthroplasties with slow healing but noninfected wounds, superficial site infections, and deep

Table 1

Preoperative Medical Condition or Risk Factor and Optimization Strategy in Patients Undergoing Total Knee Arthroplasty

Condition or Risk Factor	Optimization
Diabetes mellitus	Tight glycemic control
Rheumatoid arthritis	Medication review: hold 1 to 2 wk preoperative; restart 2 wk postoperative
Smoking	Cessation at least 6–8 wk before total knee arthroplasty
Obesity	Weight loss from calorie reduction and/or bariatric surgery
Malnutrition	Nutritional screening, education, and/or supplementation

infections. It is critical to recognize modifiable and nonmodifiable risk factors, as well as technique variables that may obstruct proper wound healing. To prevent complications and lessen the potential for infection, surgeons must address patient preoperative medical optimization, use meticulous surgical technique, and recognize a problem wound early to implement proper and expeditious wound management.[7]

Biology of Wound Healing

A healed wound serves as a protective barrier to a TKA prosthesis from retrograde bacterial contamination from the skin. Knowledge of the three phases of normal healing (ie, inflammation, proliferation, maturation) is critical during the clinical assessment of acute postoperative drainage. The inflammation phase begins at the initial incision and continues through days 4 to 6.[8] At the time of incision, exposure of collagen begins the inflammatory process by activating the clotting cascade. The initial fibrin clot serves to attract cells and concentrate cytokines and growth factors. Neutrophils migrate via chemotaxis from mediators such as interleukin-1, tumor necrosis factor-α, transforming growth factor-β, and platelet factor 4.[8]

After 48 to 96 hours from incision, macrophages accumulate and facilitate angiogenesis and fibroplasia necessary for transition into the proliferation phase.[8] The proliferation phase occurs from day 4 to 14 and involves epithelialization, angiogenesis, granulation tissue formation, and collagen deposition. Some skin edge hyperemia is expected at the proliferation phase and should be differentiated from surgical site infection and reactive skin problems, such as a psoriatic skin plaque. The maturation phase, and associated remodeling, occurs from day 8 through 1 year and mainly consists of collagen synthesis and organization. At 1 week postoperatively, a healing incision exhibits 3% of its final strength; at 3 weeks, 30%; and at 3 months, 80%. Wound strength never achieves that of normal tissue (even after 1 year) because of less organized collagen.[8,9] Disease and malnutrition have a substantial effect on wound strength because of adverse effects on matrix deposition.

Preoperative Medical Optimization

Knowledge of modifiable risk factors (as well as potential preoperative interventions) that affect wound healing is imperative[10] (Table 1). Diabetes mellitus, rheumatoid arthritis, smoking, obesity, and malnutrition all negatively affect wound healing and are associated with increased soft-tissue complications after TKA.[10] Current evidence also implicates anemia as a contributing factor for complications of wound healing.[11,12]

In addition to impaired blood and oxygen delivery, delayed collagen synthesis and decreased wound strength are also related to hyperglycemia associated with diabetes mellitus.[13,14] Galat et al[1] found a significant association between a history of diabetes mellitus and the development of early wound complications requiring surgical intervention, with an odds ratio of 5.0 (95% confidence interval, 1.4 to 17.3; $P = 0.01$). Although limited research has been carried out on precise perioperative blood glucose values to prevent wound complications,[1,13,14] early preoperative medical assessment should be arranged to develop a perioperative diabetes management strategy and to identify and optimize other comorbidities.[15]

Rheumatoid arthritis is managed with complex drug regimens including NSAIDs, methotrexate, corticosteroids, and biologics, all of which may affect wound healing and the potential for infection. In addition, the pathology of rheumatoid arthritis is associated with a 2 to 3 times greater risk of a surgical site infection than that of osteoarthritis.[10] Newer disease-modifying antirheumatic drugs may require a longer period of discontinuance before surgery, and consultation with the patient's rheumatologist is recommended to balance medical necessity with optimal wound healing.

Smoking is another modifiable risk factor that is associated with increased short-term complications after TKA because nicotine and the byproducts of smoking cause vasoconstriction via diminished oxygen transport and metabolism at the cellular level.[7,16] In a recent study of 78,191 patients, Duchman et al[16] found that current smokers are at an increased risk of wound complications after TKA and total hip arthroplasty (THA; 1.8%) compared with former smokers and nonsmokers (1.3% and 1.1%, respectively; $P < 0.001$). Compared with former smokers and nonsmokers, current smokers had approximately twice the rate of deep infection and increased rates of superficial wound infection and wound dehiscence. The authors' multivariate analysis found current smokers to be at increased risk of wound complications (odds ratio, 1.47; 95% confidence interval, 1.21 to 1.78; $P = 0.001$) compared with nonsmokers. Although no concrete evidence exists regarding smoking cessation before TKA, Møller et al[17] evaluated the effects of smoking intervention, including counseling and nicotine replacement, 6 to 8 weeks before TKA or THA compared with no intervention and demonstrated a significantly decreased overall complication rate in the smoking cessation group (18% versus 52%; $P = 0.0003$).

Counseling obese patients about weight loss, including bariatric surgery, before TKA is advisable,[18] and it must be emphasized to the patient that complication rates are high regardless of the timing of bariatric surgery, whether before or after TKA.[19] Light et al[20] studied the metabolic changes to the skin in obese patients after bariatric surgery and found poorly organized collagen, elastin degradation, and scar formation intermixed within normal tissue. Accordingly, the risk of postoperative complications after TKA is higher in obese patients.[21,22] In one study, obese patients had a 6.7 times greater risk of infection than nonobese patients undergoing TKA.[22]

Proper wound healing and immune function is dependent on optimized nutrition, and malnutrition should be managed on an individual basis before elective orthopaedic procedures.[23,24] A serum albumin level <3.5 g/dL, total lymphocyte count <1,500/mm^3, or transferrin level <200 mg/dL is associated with increased incidence of wound complications.[3,25] Malnutrition in several forms, including the paradoxical malnutrition sometimes seen in obese patients, has been linked to prolonged wound drainage and prosthetic joint infections,[25] and thus, patients in whom laboratory values fall below these lower limits should consult a nutritionist preoperatively.[24]

Prophylactic Soft-Tissue Assessment

Preoperative surgical consultation with an expert in flap coverage and microvascular techniques for soft-tissue management should be considered if difficulties with closure or wound healing are anticipated,[26] especially in patients with previous incisions, severe varus or rotational deformity, and prior trauma with contracted and immobile skin. Prior soft-tissue flaps about the knee should also prompt evaluation by a surgeon with microvascular expertise to obtain detail on the tissue quality and the vascular pedicle. A vascular surgeon should be consulted as well when concern for general circulatory compromise exists, because poor venous return may result in wound edge ischemia and necrosis from venous engorgement.

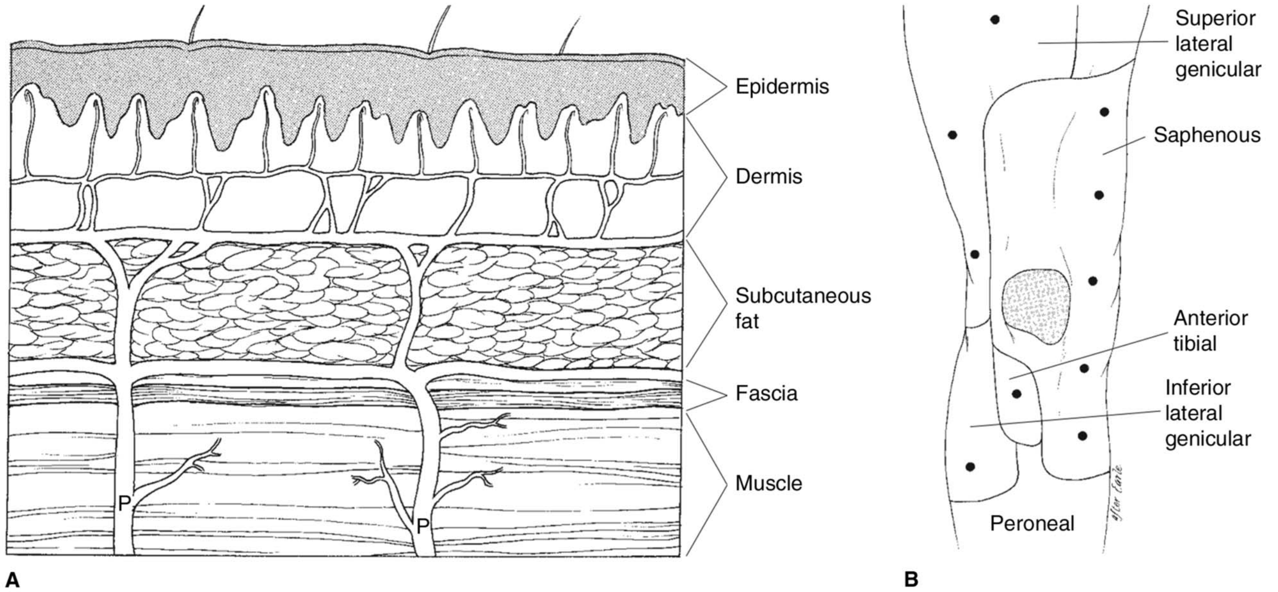
Prophylactic or revision soft-tissue procedures are indicated for knees at high risk for wound problems from multiple prior incisions or poorly placed flaps.[26] In many patients, a planned medial gastrocnemius flap with skin grafting at the time of joint arthroplasty may be required. Alternatively, the preoperative use of soft-tissue expanders may address skin that is severely adherent.[27,28] The techniques of soft-tissue expansion, as well as additional prophylactic and salvage soft-tissue transfers about the knee, are beyond the scope of this article.[26,28]

Intraoperative Factors

The blood supply to the anterior skin about the knee arises from deep perforators predominantly found along the medial side of the joint. These perforating vessels traverse through the anterior thigh muscles and between the intermuscular septa. Arterioles then arborize in the plane directly superficial to the deep fascia of the subcutaneous layer, requiring skin flap dissection to be deep to this layer to preserve the perforating arteriolar network[29,30] (Figure 1). The blood supply to the patella is separated from the skin by the patellar bursa, arising from terminal branches of the peripatellar anastomoses, with arterial contributions from the supreme geniculate artery, the medial and lateral superior geniculate arteries, the anterior tibial recurrent artery, and a branch of the profunda femoris artery[30,31] (Figure 2).

The area should be meticulously evaluated for evidence of prior incisions. More medial incisions interrupt the blood supply closer to the source, potentially compromising wound healing along the lateral skin edge. Therefore, in patients with multiple old scars, it is safer to use the most lateral, vertical incision, even if this necessitates a lateral arthrotomy (such as in a severe, fixed valgus deformity).[7,32] Transverse incisions should be crossed at 90°.[33] A short oblique incision may be incorporated into a new vertical incision provided the prior incision is near the midline. Ideal spacing between multiple vertical incisions includes a 7-cm skin bridge, because parallel incisions that are close together may compromise the epidermal blood

Figure 1



Illustrations showing the microvascular anatomy of the skin of the thigh (**A**) and the areas supplied by the deep vessels (**B**). The solid circles in panel B indicate the approximate position of deep perforating vessels and the vascular anatomy of the skin about the knee. P = deep perforators. (Reproduced from Younger AS, Duncan CP, Masri BA: Surgical exposures in revision total knee arthroplasty. *J Am Acad Orthop Surg* 1998;6[1]:55-64.)

Figure 2

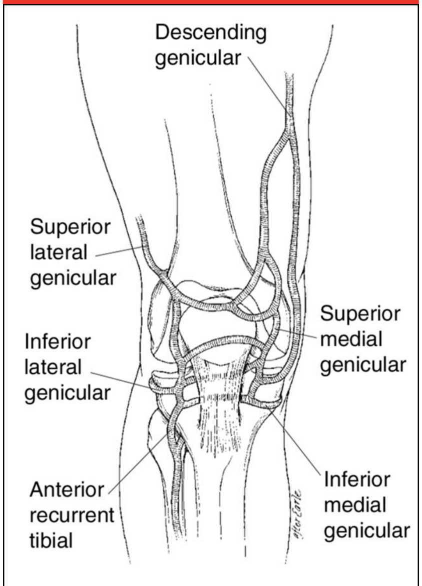


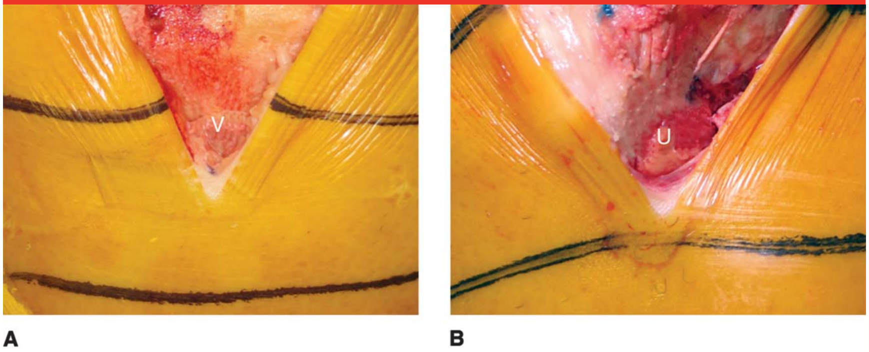
Illustration showing the vascular anatomy of the patella. (Reproduced from Younger AS, Duncan CP, Masri BA: Surgical exposures in revision total knee arthroplasty. *J Am Acad Orthop Surg* 1998;6[1]:55-64.)

supply.[27,29] Skin bridges <2.5 to 5 cm between existing and new incisions should be avoided. However, in some cases, when considerable time has passed since a prior incision was formed and revascularization is considered adequate, a surgeon may opt for a skin bridge slightly less than the 7-cm ideal. In a midline incision, the distal aspect is the most common area for wound complications and, accordingly, an incision directly medial to the tibial tubercle as it is carried distally optimizes soft-tissue and patellar tendon coverage.[27]

The skin incision should be long enough to prevent excessive tension on the wound edges. Avoiding excessive retraction of the skin edges by forceps and self-retaining retractors will help maintain the subfascial arteriolar supply. Particular attention should be paid to the superior and inferior apices of the skin incision. The apex should form a V (V sign), indicating that the skin is not under excessive tension. Should the V flatten out to become a U (U sign), the skin is under excessive tension, and the incision should be extended to prevent tearing and iatrogenic injury (Figure 3). Using full-thickness skin flaps to avoid undermining the skin and to avoid a lateral retinacular release will help preserve lateral skin oxygenation[34] and reduce the risk of skin necrosis.

Meticulous hemostasis is imperative to prevent postoperative hematoma and persistent drainage. Any vessels exposed during the dissection should be cauterized because a wound hematoma is frequently the initiating event to wound breakdown. Minimizing the infrapatellar fat pad resection likewise diminishes the rate of a subcutaneous hematoma and potential drainage. Hemostasis may be done with any combination of electrocautery devices,[35,36] pharmacologic and intravenous hemostatic agents,[37] and recently, with advances of systemic and local application of tranexamic acid.[38,39] A tension-free closure with correct wound-edge alignment is paramount to prevent skin necrosis and potential drainage. Proper closure of the wound at the distal aspect of the incision near the patellar tendon is especially important because most patients who later develop infection start with early postoperative serous drainage at this site.[33] The routine use of primary wound closure over a drain remains controversial.

Figure 3



Intraoperative photographs showing the V sign (**A**), which indicates proper skin traction, and the U sign (**B**), which indicates that skin is under excessive tension.

Acute Postoperative Drainage

Serosanguinous drainage after TKA is common. Drainage affects 1% to 10% of patients undergoing primary total joint arthroplasty.[3-5] Persistent incisional drainage after TKA is defined as continued drainage from the surgical wound for >72 hours, and substantial drainage (>2 × 2–cm area of gauze) beyond this time period is abnormal.[6] Although drainage requires close monitoring, most cases spontaneously resolve without a need for surgical débridement.[40] Small areas of marginal wound necrosis measuring a few millimeters wide and up to 4 cm long or locally débrided eschar in a delayed manner may be observed. Nonsurgical care may be the appropriate treatment in certain cases; however, profuse and persistent drainage (for >5 to 7 days) is unlikely to stop, and surgical intervention is typically required.[32]

The surgeon must determine the source of early postoperative drainage; the drainage may arise from a superficial or a deep source, and it may involve a benign or infectious process.[6] Immediate postoperative drainage within the first 72 hours is typically serosanguinous and involves the superficial tissue layers. Persistent drainage >72 hours may represent edema, blood products, and fat ischemia but may also constitute fluid from a capsular defect that should be surgically repaired. After 72 hours, drainage is no longer benign and may be considered potentially infectious.[6] In patients with primary wound complications, aggressive treatment of the problem is essential to diminish the risk of a secondary deep periprosthetic infection.

Management

Nonsurgical

It is difficult to define absolute parameters when nonsurgical management of a persistently draining wound is appropriate. Postoperative drainage >48 hours has been identified as a contributing factor to periprosthetic infection. Patel et al[40] found that each day of prolonged incisional drainage after TKA increased the risk of wound infection by 29%. Patients with a draining wound on postoperative days 2 or 3 should remain in the hospital for close clinical monitoring and may initially be treated with compressive dry dressings. Such dressings may be sufficient for most wounds, especially when drainage acutely diminishes. Wounds should be frequently inspected until dry.

Physical therapy, specifically knee range of motion, should be temporarily limited for 24 to 48 hours. Continuous passive motion should be avoided, or at least limited, because flexion >40° after TKA is known to reduce transcutaneous oxygen saturation about the incision.[41] Anticoagulation status should be reviewed, and short-term cessation of anticoagulation should be considered. Patients treated with low-molecular-weight heparin for prophylaxis against deep vein thrombosis have shown longer times to achieve a dry surgical wound, compared with patients treated with aspirin and mechanical compression or warfarin.[40] In light of these findings, it is prudent to temporarily stop anticoagulation with low-molecular-weight heparin, or other chemical anticoagulation, but continue mechanical venous thromboembolism prophylaxis.

An emerging area of interest for a variety of indications is the use of incisional negative pressure wound therapy (NPWT), whereby nonadherent, semipermeable dressings are used along with polyurethane foam and tubing systems over closed incisions. Randomized controlled trial evidence exists that incisional NPWT reduces dehiscence and infection in orthopaedic trauma patients and has a positive effect on wound healing and complication rates in arthroplasty patients. Stannard et al[42] published a randomized study of incisional NPWT applied to patients with high-energy fractures, a population known to be at higher risk for wound complications than arthroplasty patients. A total of 263 lower extremity fractures were evaluated, with 122 managed with standard dry dressings and 141 with incisional NPWT for a mean of 2.5 days. Use of NPWT rather than standard dry dressings resulted in a significant reduction in the rate of deep infection (10.0% and 19.0%, respectively; P = 0.049) and dehiscence (8.6% and 16.5%, respectively; P = 0.044). Reddix et al[43] retrospectively studied 235 patients undergoing acetabular fracture stabilization using incisional NPWT for up to 3 days on the closed incision. The incidence of infection and dehiscence reduced approximately sixfold from 6% to 1% and from 3% to 0.5%, respectively, compared with incidences in 66 consecutive patients before the institutional use of incisional NPWT.

Incisions that drain serous fluid after the second postoperative day are indicated for NPWT. Webb[4] reported that 10% of elective hip and knee surgeries have serous drainage at or beyond postoperative day 2 that is correctable with incisional NPWT. In such situations, the pressure setting is lowered to −50 mm Hg to reduce skin irritation. In most patients, dry wounds can be expected with this treatment within 24 hours after one application.[4] In THA patients with persistent draining wounds, Hansen et al[44] found resolution in 76% of patients treated with incisional NPWT. A significantly lower volume of seroma was confirmed with ultrasonography in THA patients treated with a single-use incisional NPWT for 5 days versus a standard dry dressing, (1.97 mL with NPWT and 5.08 mL with standard dressing, respectively; P = 0.021).[45] Animal models confirm that incisional NPWT accelerates wound healing more quickly than simple dressings at normal or hyperbaric oxygen pressures,[46] likely by diminishing postoperative edema through effects on lymph drainage.

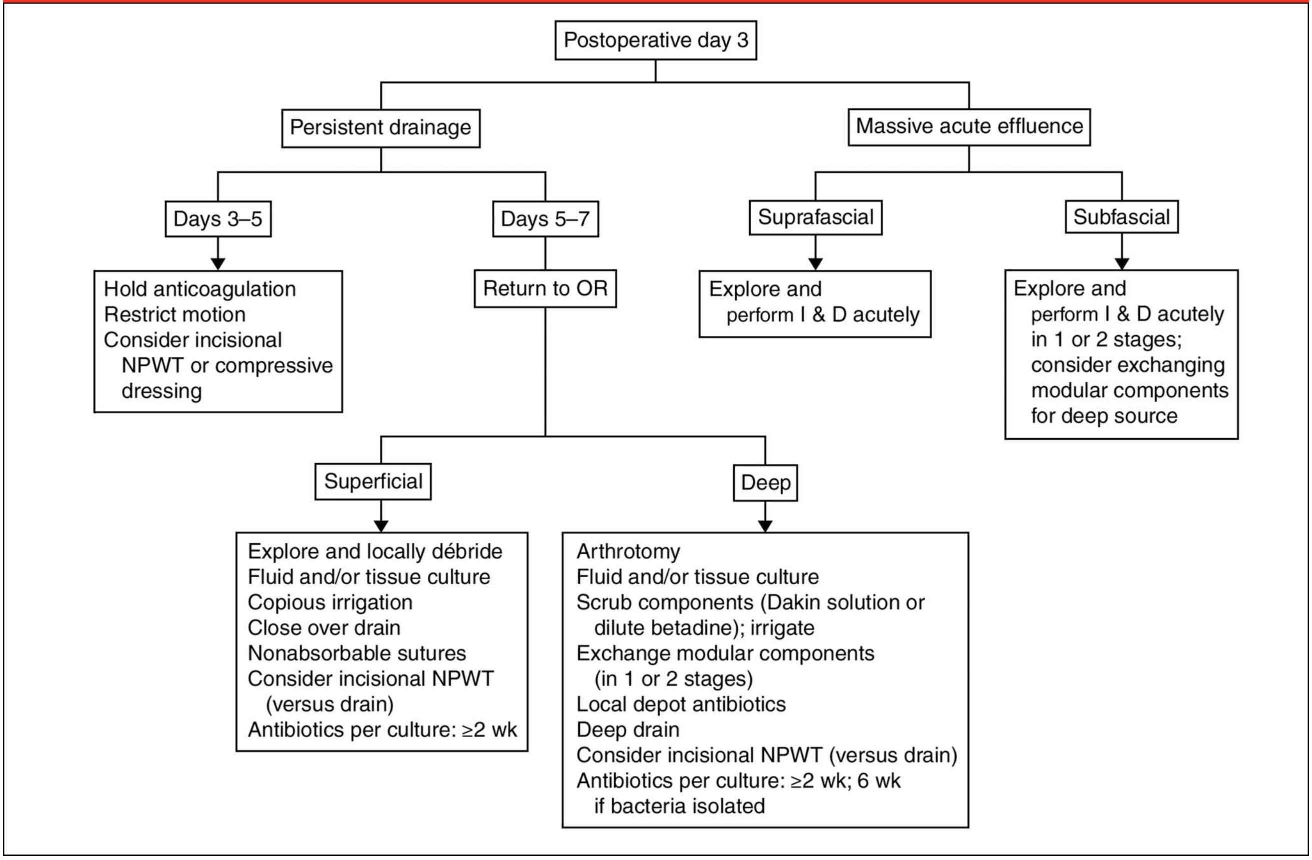
Surgical

The *2013 Proceedings of the International Consensus on Periprosthetic Joint Infection* had a strong consensus that a persistently draining wound for >5 to 7 days should expeditiously undergo revision surgery to prevent a deep periprosthetic joint infection.[6] The workgroup noted that it is reasonable to wait until postoperative day 5 because this interval to a dry wound may be affected by anticoagulation. Beyond this time point, it is important to exclude and prevent a deep infection, and aspiration of the knee joint should be performed either preoperatively or intraoperatively. Joint aspiration helps determine whether drainage arises from a structural defect of the arthrotomy or an acute deep infection. Although cell count data from fluid aspiration in the acute postoperative period may have confounding variables, Bedair et al[47] found that infection may be presumed for white blood cell count values >28,000 cells/mL and 89% polymorphonuclear cells. No evidence currently exists to demonstrate that reflexive administration of antibiotics improves the outcome of a draining wound or reduces the occurrence of a surgical site infection, and the *2013 Proceedings of the International Consensus on Periprosthetic Joint Infection* strongly recommends against such treatment.[6] In their rationale, the workgroup cites the known adverse systemic effects of antibiotics, emerging bacterial antibiotic resistance, and the obfuscation of diagnosing a deep infection.

Although concern exists that early surgical intervention may increase the risk of periprosthetic infection by potentially contaminating a sterile joint, studies to date suggest the opposite; that prompt intervention likely reduces the risk of wound breakdown and deep infection by limiting retrograde skin contamination. Weiss and Krackow[5] found that of 8 of 597 TKA patients who underwent early surgical intervention for prolonged drainage at an average of 12.5 days after index surgery, no patient developed a deep periprosthetic infection with this strategy. Jaberi et al[3] studied 10,325 patients undergoing THA or TKA in which 300 patients postoperatively developed drainage beyond 48 hours (2.9%). Drainage stopped in 217 patients at 2 to 4 days with local wound care with no adverse outcomes, and of the remaining 83 patients, 63 (76%) had successful treatment with a single débridement. Patients who underwent débridement at a mean of 5 days after the onset of drainage were more likely to be infection free at 1 year compared with patients who underwent delayed débridement at a mean of 10 days.[3] Because drainage >5 to 7 days is unlikely to stop, surgical intervention is now advocated.[5,6,32]

Drainage or hematoma that compromises the skin or causes skin ischemia, substantially increases pain, or fails to diminish must be aggressively managed.[3,5] After a problem wound has been established, interventions at minimum must include a superficial incisional exploration, excising necrotic skin edges, and evacuating any hematoma. If the joint capsule is compromised, treatment must include opening the fascia, performing a thorough irrigation and débridement, with synovectomy and removal of all contaminated tissue. The use of low-pressure pulsatile lavage has a success rate similar to

Figure 4



Treatment algorithm for acute incisional knee drainage. I & D = incision and drainage, NPWT = negative pressure wound therapy, OR = operating room

high-pressure lavage during the open débridement of orthopaedic implants. Thorough irrigation with 6 to 9 L of solution is used, and retained components are scrubbed with either Dakin solution or dilute betadine (0.3%), made by mixing 17 mL of sterile 10% povidone-iodine to 500 mL saline in a basin. New gowns and gloves should be donned, suction tips and tubing changed, and additional sterile drapes used after completing the irrigation. The modular polyethylene component should be exchanged and a meticulous fascial closure performed over a drain.

Although the reports of single-stage débridement with component retention have not indicated overwhelming success, Estes et al[48] developed an approach for managing acute periprosthetic joint infections with a two-stage approach. The first step is a débridement with retention of the prosthesis and placement of antibiotic-impregnated cement beads followed by a second procedure within 1 week, at which time the beads are removed and new modular components are inserted. Patients receive intravenous antibiotics for 6 weeks, followed by oral antibiotics, depending on the susceptibility of the bacterial organism. In a retrospective review of 20 patients, the authors controlled infection in 18 patients with this technique, which compared favorably with prior reports with component retention. With proper patient selection, this two-stage procedure may be a viable option in the management of acute periprosthetic total joint infections; however, further research is needed.

To promote healing in patients with soft-tissue deficiency, NPWT may help prevent additional tissue necrosis by reducing edema and minimizing shear forces. In the setting of more severe soft-tissue defects, coverage with muscle flaps or other advanced soft-tissue reconstructive techniques may be necessary. Should an infection be present, deep cultures should be taken at the time of revision surgery to provide antibiotic guidance (Figure 4).

The success of early aggressive intervention without introducing infection and with no statistically significant morbidity comes from limited data,[1,3,5] and it is likely that some patients would never have developed complications with nonsurgical management. However, because of the strong association between prolonged drainage and deep prosthetic infection, we think that early prophylactic irrigation and débridement is preferable to delayed management or no management to mitigate or prevent the potentially devastating postoperative problems of wound breakdown leading to an established infection.

Summary

Postoperative soft-tissue problems about the knee can severely compromise the generally excellent outcomes of TKA. Modifiable patient factors should be preoperatively addressed to optimize wound healing potential. Soft-tissue problems may still occur, despite appropriate measures to minimize complications. When an acute wound problem occurs, the guiding principle is close monitoring and aggressive prophylactic intervention to resolve the wound complication before a secondary periprosthetic infection ensues. Surgical intervention should take place within 5 to 7 days in the setting of persistent drainage or skin necrosis to prevent (or manage) a deep infection.

The optimal definitive surgical treatment continues to be refined, and with the advent of new markers to diagnose early deep periprosthetic infection, a more exact distinction between primary aseptic wound healing problems and secondary complications from a deep infection may emerge. The management of acute wound complications will also benefit from data on incisional NPWT in TKA and from advances in soft-tissue reconstruction techniques to cover larger and more complex peri-incisional defects.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 11, 17, 35, 36, 38, 39, 41, 42, and 45 are level I studies. References 16, 19, 21, 22, 34, and 47 are level II studies. References 1-3, 5, 10, 13, 14, 20, 25, 26, 40, and 43 are level III studies. References 7, 28, 30, 44, 46, and 48 are level IV studies. References 4, 6, 8, 9, 12, 15, 18, 23, 34, 27, 29, 31-33, and 37 are level V expert opinion.

References printed in **bold type** are those published within the past 5 years.

1. Galat DD, McGovern SC, Larson DR, Harrington JR, Hanssen AD, Clarke HD: Surgical treatment of early wound complications following primary total knee arthroplasty. *J Bone Joint Surg Am* 2009;91(1):48-54.
2. Galat DD, McGovern SC, Hanssen AD, Larson DR, Harrington JR, Clarke HD: Early return to surgery for evacuation of a postoperative hematoma after primary total knee arthroplasty. *J Bone Joint Surg Am* 2008;90(11):2331-2336.
3. Jaberi FM, Parvizi J, Haytmanek CT, Joshi A, Purtill J: Procrastination of wound drainage and malnutrition affect the outcome of joint arthroplasty. *Clin Orthop Relat Res* 2008;466(6):1368-1371.
4. Webb LX: New techniques in wound management: Vacuum-assisted wound closure. *J Am Acad Orthop Surg* 2002;10(5):303-311.
5. Weiss AP, Krackow KA: Persistent wound drainage after primary total knee arthroplasty. *J Arthroplasty* 1993;8(3):285-289.
6. **Parvizi J, Gehrke T, Chen AF: Proceedings of the International Consensus on Periprosthetic Joint Infection. *Bone Joint J* 2013;95-B(11):1450-1452.**
7. Vince K, Chivas D, Droll KP: Wound complications after total knee arthroplasty. *J Arthroplasty* 2007;22(4 suppl 1):39-44.
8. Broughton G II, Janis JE, Attinger CE: The basic science of wound healing. *Plast Reconstr Surg* 2006;117(7 suppl):12S-34S.
9. Broughton G II, Janis JE, Attinger CE: Wound healing: An overview. *Plast Reconstr Surg* 2006;117(7 suppl):1e-S-32e-S.
10. Moucha CS, Clyburn T, Evans RP, Prokuski L: Modifiable risk factors for surgical site infection. *J Bone Joint Surg Am* 2011;93(4):398-404.
11. Jonsson K, Jensen JA, Goodson WH III, et al: Tissue oxygenation, anemia, and perfusion in relation to wound healing in surgical patients. *Ann Surg* 1991;214(5):605-613.
12. Rohrich RJ: Current concepts in wound healing: Update 2011. *Plast Reconstr Surg* 2011;127(suppl 1):1S-2S.
13. Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A: Glucose control lowers the risk of wound infection in diabetics after open heart operations. *Ann Thorac Surg* 1997;63(2):356-361.
14. England SP, Stern SH, Insall JN, Windsor RE: Total knee arthroplasty in diabetes mellitus. *Clin Orthop Relat Res* 1990;260:130-134.
15. **Handelsman Y, Bloomgarden ZT, Grunberger G, et al: American Association of Clinical Endocrinologists and American College of Endocrinology: Clinical practice guidelines for developing a diabetes mellitus comprehensive care plan. 2015. *Endocr Pract* 2015;21(suppl 1):1-87.**
16. **Duchman KR, Gao Y, Pugely AJ, Martin CT, Noiseux NO, Callaghan JJ: The effect of smoking on short-term complications following total hip and knee arthroplasty. *J Bone Joint Surg Am* 2015;97(13):1049-1058.**
17. Møller AM, Villebro N, Pedersen T, Tønnesen H: Effect of preoperative smoking intervention on postoperative complications: A randomised clinical trial. *Lancet* 2002;359(9301):114-117.
18. **Workgroup of the American Association of Hip and Knee Surgeons Evidence Based Committee: Obesity and total joint arthroplasty: A literature based review. *J Arthroplasty* 2013;28(5):714-721.**
19. Severson EP, Singh JA, Browne JA, Trousdale RT, Sarr MG, Lewallen DG: Total knee arthroplasty in morbidly obese patients treated with bariatric surgery: A comparative study. *J Arthroplasty* 2012;27(9):1696-1700.
20. Light D, Arvanitis GM, Abramson D, Glasberg SB: Effect of weight loss after bariatric surgery on skin and the extracellular matrix. *Plast Reconstr Surg* 2010;125(1):343-351.
21. **McElroy MJ, Pivec R, Issa K, Harwin SF, Mont MA: The effects of obesity and morbid obesity on outcomes in TKA. *J Knee Surg* 2013;26(2):83-88.**
22. Namba RS, Paxton L, Fithian DC, Stone ML: Obesity and perioperative morbidity in total hip and total knee arthroplasty patients. *J Arthroplasty* 2005;20(7 suppl 3):46-50.
23. Smith TK: Prevention of complications in orthopedic surgery secondary to nutritional depletion. *Clin Orthop Relat Res* 1987;222:91-97.
24. **Cross MB, Yi PH, Thomas CF, Garcia J, Della Valle CJ: Evaluation of malnutrition in orthopaedic surgery. *J Am Acad Orthop Surg* 2014;22(3):193-199.**

25. Greene KA, Wilde AH, Stulberg BN: Preoperative nutritional status of total joint patients: Relationship to postoperative wound complications. *J Arthroplasty* 1991; 6(4):321-325.

26. Casey WJ III, Rebecca AM, Krochmal DJ, et al: Prophylactic flap reconstruction of the knee prior to total knee arthroplasty in high-risk patients. *Ann Plast Surg* 2011;66 (4):381-387.

27. Garbedian S, Sternheim A, Backstein D: Wound healing problems in total knee arthroplasty. *Orthopedics* 2011;34(9): e516-e518.

28. Long WJ, Wilson CH, Scott SM, Cushner FD, Scott WN: 15-year experience with soft tissue expansion in total knee arthroplasty. *J Arthroplasty* 2012;27(3): 362-367.

29. Younger AS, Duncan CP, Masri BA: Surgical exposures in revision total knee arthroplasty. *J Am Acad Orthop Surg* 1998;6(1):55-64.

30. Lazaro LE, Cross MB, Lorich DG: Vascular anatomy of the patella: Implications for total knee arthroplasty surgical approaches. *Knee* 2014;21(3):655-660.

31. Haertsch PA: The blood supply to the skin of the leg: A post-mortem investigation. *Br J Plast Surg* 1981;34(4): 470-477.

32. Dennis DA: Wound complications in total knee arthroplasty. *Instr Course Lect* 1997; 46:165-169.

33. Sanna M, Sanna C, Caputo F, Piu G, Salvi M: Surgical approaches in total knee arthroplasty. *Joints* 2013;1(2): 34-44.

34. Scuderi G, Scharf SC, Meltzer LP, Scott WN: The relationship of lateral releases to patella viability in total knee arthroplasty. *J Arthroplasty* 1987;2(3):209-214.

35. Plymale MF, Capogna BM, Lovy AJ, Adler ML, Hirsh DM, Kim SJ: Unipolar vs bipolar hemostasis in total knee arthroplasty: A prospective randomized trial. *J Arthroplasty* 2012;27(6): 1133-1137.e1.

36. Marulanda GA, Krebs VE, Bierbaum BE, et al: Hemostasis using a bipolar sealer in primary unilateral total knee arthroplasty. *Am J Orthop (Belle Mead NJ)* 2009;38(12): E179-E183.

37. Levine BR, Haughom B, Strong B, Hellman M, Frank RM: Blood management strategies for total knee arthroplasty. *J Am Acad Orthop Surg* 2014;22(6):361-371.

38. Aguilera X, Martinez-Zapata MJ, Bosch A, et al: Efficacy and safety of fibrin glue and tranexamic acid to prevent postoperative blood loss in total knee arthroplasty: A randomized controlled clinical trial. *J Bone Joint Surg Am* 2013;95(22):2001-2007.

39. Wong J, Abrishami A, El Beheiry H, et al: Topical application of tranexamic acid reduces postoperative blood loss in total knee arthroplasty: A randomized, controlled trial. *J Bone Joint Surg Am* 2010; 92(15):2503-2513.

40. Patel VP, Walsh M, Sehgal B, Preston C, DeWal H, Di Cesare PE: Factors associated with prolonged wound drainage after primary total hip and knee arthroplasty. *J Bone Joint Surg Am* 2007;89(1):33-38.

41. Johnson DP: The effect of continuous passive motion on wound-healing and joint mobility after knee arthroplasty. *J Bone Joint Surg Am* 1990;72(3):421-426.

42. Stannard JP, Volgas DA, McGwin G III, et al: Incisional negative pressure wound therapy after high-risk lower extremity fractures. *J Orthop Trauma* 2012;26(1):37-42.

43. Reddix RN Jr, Leng XI, Woodall J, Jackson B, Dedmond B, Webb LX: The effect of incisional negative pressure therapy on wound complications after acetabular fracture surgery. *J Surg Orthop Adv* 2010;19(2):91-97.

44. Hansen E, Durinka JB, Costanzo JA, Austin MS, Deirmengian GK: Negative pressure wound therapy is associated with resolution of incisional drainage in most wounds after hip arthroplasty. *Clin Orthop Relat Res* 2013;471(10):3230-3236.

45. Pachowsky M, Gusinde J, Klein A, et al: Negative pressure wound therapy to prevent seromas and treat surgical incisions after total hip arthroplasty. *Int Orthop* 2012;36(4):719-722.

46. Fabian TS, Kaufman HJ, Lett ED, et al: The evaluation of subatmospheric pressure and hyperbaric oxygen in ischemic full-thickness wound healing. *Am Surg* 2000;66 (12):1136-1143.

47. Bedair H, Ting N, Jacovides C, et al: The Mark Coventry Award: Diagnosis of early postoperative TKA infection using synovial fluid analysis. *Clin Orthop Relat Res* 2011; 469(1):34-40.

48. Estes CS, Beauchamp CP, Clarke HD, Spangehl MJ: A two-stage retention débridement protocol for acute periprosthetic joint infections. *Clin Orthop Relat Res* 2010;468(8):2029-2038.