EFORT open reviews

The draining surgical wound post total hip and knee arthroplasty: what are my options? A narrative review

Richard Peter Almeida
Lipalo Mokete
Nkhodiseni Sikhauli
Allan Roy Sekeitto
Jurek Pietrzak

- Total hip arthroplasty (THA) and total knee arthroplasty (TKA) are successful orthopaedic procedures with an ever-increasing demand annually worldwide, and persistent wound drainage (PWD) is a well-known complication following these procedures. Despite many definitions for PWD having been proposed, a validated description remains elusive.

- PWD is a risk factor for periprosthetic joint infection (PJI). PJI is a devastating complication of THA and TKA, and a leading cause of revision surgery with dramatic morbidity and mortality and a significant burden on health socioeconomics.

- Prevention of PJI has become an essential focus in THA and TKA. Understanding the pathophysiology, risk factors and subsequent management of PWD may aid in decreasing the rate of PJI.

- Risk factors of PWD can be divided into modifiable and non-modifiable patient risk factors, pharmacological and surgical risk factors. No gold standard treatment protocol to address PWD exists; however, non-operative options progressing to surgical interventions have been described.

- The aim of this study was to review the current literature regarding PWD and consolidate the risk factors and management strategies available.

Introduction

Primary total joint arthroplasty (TJA), including total hip arthroplasty (THA) and total knee arthroplasty (TKA), are highly successful, reproducible surgical procedures. The demand for TJA is increasing globally, with projections showing sustained increases beyond 2030.[1,2] Associated complications will subsequently increase in conjunction with this demand.[2] Persistent wound drainage (PWD) is a post-operative wound complication following TJA. It is reported to occur in between 0.2% to 21% of all cases of primary TJA; however, there is lack of agreement on the definition of PWD.[3] PWD has been reported as a risk factor for periprosthetic joint infection (PJI).[4] Patel et al[4] showed that each extra day of PWD carried an additional 42% risk of wound infection in TKA and 29% risk of wound infection in THA. The rate of PJI in wounds that persistently drain post-operatively has been reported in various studies to range from 1.3% to up to 50%, with the wide range possibly attributable to a lack of standardized definition of persistent wound drainage used and the heterogenicity and retrospective nature of available literature.[4–6]

PJIs are associated with significant morbidity and mortality and place a heavy economic burden on healthcare facilities and resources.[3,7] PJI is the most common reason for revision TKA and third most common cause of revision THA. It is the most common reason for revision within two years of TJA.[6] A 3.58 times increased risk of death exists after revision surgery for PJI and five-year mortality is 21%.[8] Much focus is now devoted to the prevention of PJI and the recognition and treatment of PWD should be a logical step in preventing PJI. However, evidence-based clinical guidelines for the diagnosis and treatment of PWD in TJA are still lacking.

Pathophysiology

Surgical wound healing has been divided into different phases needed to complete closure of the wound and

restore the vital barrier to physical, chemical and biological pathogens.[9] Wound healing starts with haemostasis, inflammation, proliferation, maturation and ends in remodelling, with any deviation within these phases resulting in delayed or abnormal healing of a surgical wound.[9]

Disturbance in wound healing may be physiological and non-infectious, resulting in wound drainage for a short duration. Surgical disruption of the superficial capillaries may result in unimportant, transitory serous or serosanguinous wound drainage post-operatively.[10] This surgical disruption may result in drainage within the first 72 hours, which is usually serosanguinous and involves the superficial tissue layers.[11] Drainage continuing after 72 hours may arise from fat necrosis sustained during surgery, dissolving haematoma from poor haemostasis, or fluid from a deep capsular defect, and must be considered potentially infectious and demands intervention.[11]

In PWD, the natural barrier of the skin is bypassed, providing a retrograde pathway for pathogens to enter the wound and ultimately contaminate the joint.[1,12,13] The majority of wound drainage resolves spontaneously with physiological healing.[4] When normal healing does not occur, PWD may forewarn of a developing, underlying infectious process and should not be ignored.[10,14–17] Whether delayed wound healing results in PWD or vice versa, where exactly does the draining fluid originate from within the wound and to what extent a retrograde pathway is made available for pathogens to enter the joint are all difficult to clarify, yet remain important considerations.[3]

Numerous definitions have been proposed for PWD, but a single validated definition has yet to be fully adopted.[3,10] It has been suggested that wound drainage from two to nine days post-operatively is persistent. A wound is said to be actively draining if an area of the wound dressing of more than 2×2 cm is wet beyond 72 hours post-operatively.[3,11,18] Other definitions include drainage for more than 48 hours soaking through the dressings; continued drainage beyond day four post-operatively; drainage beyond two days post-operatively for non-infected cases and 5.5 days post-operatively for infected cases.[10]

The lack of consensus regarding the definition of PWD was highlighted by an online survey of the Netherlands Orthopaedic Association, which reported that 59.1% of surgeons allowed three to seven days of PWD before starting non-surgical management while 44.1% intervened surgically only after 10 days of PWD after index TJA.[19] According to the proceedings of international consensus on orthopaedic infections, the suggested definition of persistent wound drainage is 'any continued fluid extrusion from the operative site occurring beyond 72 hours from index surgery'.[10]

Risk factors

The risk factors for PWD can be considered as patient-specific, pharmacological and surgical.

Patient-specific risk factors

Patient factors associated with PWD include age, obesity, malnutrition, diabetes, anaemia, inflammatory arthritis, smoking, *Staphylococcus aureus* colonization, and malnutrition.[2,6,11] Shahi et al[6] retrospectively reviewed 4873 TJAs and reported an incidence of PWD of 6.2% with a subsequent rate of PJI of 15.9%. Diabetes inferred a 21 times greater risk of PWD. The possibility of PWD was increased by 17.3 times in morbid obesity, 14.2 times in rheumatoid arthritis, 4.3 times in chronic alcohol use and 2.8 times in hypothyroidism.[6]

Obesity is a modifiable risk factor for complications related to TJA, and an independent risk factor for PWD.[2,6] This may be related to fat necrosis that occurs due to larger surgical incisions as well as increased surgical time.[4,6] Therefore counselling patients about weight loss is advisable pre-operatively.[11]

Malnutrition negatively affects the immune system and wound healing. Reduced serum measurements of albumin < 35 g/L, total lymphocyte count of < 1500/mm³, or transferrin level <2 g/L have been associated with wound complications.[11] Surgery is known to increase metabolic demand, making borderline deficiencies pre-operatively more significant, and therefore these deficits should be restored.[9] Protein malnutrition, identified with the surrogate measurement of albumin, is a significant risk factor as there is increased protein turnover during the wound healing process.[9] Vitamin C, vitamin A, zinc and magnesium have been identified as key factors for wound healing, and supplementation of these has been suggested to improve wound healing in deficient patients.[9]

Diabetes mellitus (DM) is a systemic disease, with multiple systems and mechanisms implicated in the pathogenesis of poor wound healing. Hyperglycaemia as a result of poorly controlled DM results in structural and functional alteration of proteins and enzymes.[9] The macro and microvascular complications of DM also impair blood flow and subsequent oxygen delivery at the tissue level.[9] The altered proteins and enzymes, poor circulation as well as the poor immune system associated with DM all affect wound healing and contribute to increased risk of PWD.[6,9]

Thyroid hormone is associated with fibroblast proliferation needed in the process of wound healing, therefore, suppression of thyroid hormone results in the disturbance of collagen synthesis in wound healing.[20] This is supported by Shahi et al,[6] indicating hypothyroidism as a risk factor for PWD.

Anaemia is a risk factor for PWD, but the exact relationship between anaemia and PWD is poorly understood.

However, it can be deduced that there would be a higher rate of peri-operative allogenic blood transfusions in anaemic patients. Blood transfusions have been shown to be associated with increased superficial wound complications possibly due to the associated immunomodulation effect.[2,21]

Rheumatoid arthritis is associated with impaired immune function and it has been suggested that both the underlying disease process and the medications used in the management are responsible for poor wound healing and PWD.[2,6] Steroids used in the management of inflammatory disorders lead to poor wound healing, due to the anti-inflammatory effects, inhibition of epithelialization and reduced collagen production.[9]

Smoking results in poor wound healing due to the negative effects of nicotine, carbon monoxide and hydrogen cyanide.[9] The effects of nicotine cause vasoconstriction and local tissue hypoxia.[9,11] Carbon monoxide binds to haemoglobin and produces methaemoglobin, thereby reducing the oxygen delivery of haemoglobin, and hydrogen cyanide inhibits oxidative metabolism.[9] Due to these effects, the cells needed during wound healing are dysfunctional at low oxygen levels, and collagen deposition is reduced.[9] Therefore cessation of smoking is advised, and although uniform guidelines do not exist, cessation of at least 4–8 weeks before and four weeks post-operatively has been recommended.[9,11]

Bacterial colonization, particularly with *Staphylococcus aureus* is a risk factor for surgical site infection.[22] Bacteria growth within a wound bed affects the various stages of wound healing, and can alter haemostasis, needed in the initial stage of wound healing.[9] Whether PWD is a cause or consequence of infection is debatable as it has been suggested that wounds that are draining may be draining because they already have some level of infection.[23,24]

HIV infection and the associated immunocompromised state has been associated with post-operative wound complications, and emphasis has been placed on pre-operative optimization by improving cell cluster of differentiation counts (CD4 > 200) and ensuring viral load suppression to avoid those complications.[25,26] Increased surgical site complications including PWD have been reported with hepatitis C infections.[27,28] It has been hypothesized that small vessel vasculitis together with liver, kidney, haematological and immune system impairments affect wound healing and wound infection.[27]

Chronic alcohol use has been identified as a risk factor for PWD.[6] Whether this is related to the reported associated risk factors of malnutrition or liver disease[25] that can result from chronic alcohol use needs further evaluation.

Chronic obstructive lung disease has been reported to result in an increased risk of surgical site complications,[29] with Gu et al[30] reporting that patients with COPD are 2.9 times more likely to develop wound dehiscence. Whether this is directly related to COPD, related comorbidities or the association with current or previous smoking is yet to be determined.

Pharmacological risk factors

The initial stage in wound healing starts with haemostasis, therefore any disruption to this stage disrupts and prolongs wound healing.[9] The use of anti-coagulation postoperatively may disrupt haemostasis and potentially result in PWD. Disrupted haemostasis may result in the formation of a haematoma, providing a rich medium for bacterial growth.[22] Anti-coagulation therapies used include warfarin, enoxaparin (low molecular weight heparin), fondaparinux, rivaroxaban and aspirin to mitigate the risk of venous thromboembolic events (VTE).[4,31] Each of the agents have different mechanisms of actions, dosages and routes of administration, with negative and positive attributes regarding their uses that need to be considered in VTE prophylaxis. Peri-operative VTE can be catastrophic but so too can deep and superficial wound complications, therefore risk stratification is needed to balance anticoagulation peri-operatively, and patients requiring therapeutic anti-coagulation need to be counselled about the risk regarding wound complications and infection.[32]

When using the international normalized ratio (INR) to monitor the response to warfarin, an INR of more than 1.5 is associated with increased risk of developing wound complications.[33] Shahi et al[6] found that the rate of PWD reduced from 6.3% to 3.1% when changing from the use of warfarin to aspirin for post-operative VTE prophylaxis. The time taken to a dry wound is longer in patients on low molecular weight heparin (LMWH) than those on aspirin and mechanical compression or warfarin.[11] Jones et al[34] showed that the use of LMWH and the use of aspirin resulted in a 4.92 and 3.64 times greater increase in wound discharge respectively when compared to the use of no pharmacological thromboprophylaxis. Lum et al[31] proposed that prolonged wound drainage due to anti-coagulation had a positive correlation with increased length of stay (LOS) in hospital. This was supported by Patel et al,[4] therefore using LOS as a surrogate for wound drainage assists in comparing anti-coagulation agents.[31] In order of shortest to longest LOS, the use of aspirin was 2.6 days, warfarin was 3.7 days, Fondaparinux was 3.77 days, rivaroxaban was 4.1 days and enoxaparin was five days.[31] There have been numerous studies reporting the effects of various anti-coagulation therapies, aiming to identify the ideal therapy providing adequate prophylactic effect against VTE while limiting post-operative surgical site complications.[35] Various guidelines have been proposed, and although aspirin seems to be favoured for VTE prophylaxis when considering possible wound complications, the debate continues for the most optimal prophylactic regimen.[35,36]

Surgical risk factors

Surgical risk factors include previous surgery to the area, surgical approach, pre-operative skin preparation, tourniquet use, total surgical time, blood loss, surgical and anaesthetic technique.[4,11,22]

Previous surgery alters the native anatomy and blood supply to the area, with risk of wound complications following subsequent surgeries. The presence of previous skin incisions should be taken into consideration when planning for future skin incisions.[22] It has been advised that around the knee joint, the most lateral vertical incision should be used and skin bridges between new and old incisions of 2.5 cm to 5 cm should be avoided.[11]

Skin preparation prior to surgery is of paramount importance in prevention of infective complications. Currently there is no evidence assessing the relationship between skin preparation and PWD specifically. Many options have been proposed, and the ideal agent is still under discussion.[2] Chlorhexidine-alcohol solution has been shown to be more protective than povidone-iodine in reducing infective complications in multiple studies.[22,37] However Carroll et al[2] found skin preparation with chlorhexidine and alcohol carried a five-fold increase in the risk of superficial wound complications compared with iodine and alcohol. The difference may be explained by the variation in concentrations of the constituents of the skin preparation and may need further investigation.

Surgical techniques with meticulous handling and dissection of soft tissues, accurate closure of the relevant layers, and adequate haemostasis prevent post-operative haematoma which can lead to PWD.[22] The combination of electrocautery devices and pharmacological interventions, such as intravenous and local application of tranexamic acid, have been advocated in achieving haemostasis.[11] Haemostasis is also important in decreasing intra-operative blood loss and the need for blood transfusion which is related to post-operative wound complications.[2]

Surgical approach choice affects PWD. In THA, an increased risk of PWD and superficial wound dehiscence exists with the direct anterior approach (DAA).[38–40] Both the skin quality around the anterior hip and the location of the surgical incision are contributory. The DAA surgical skin incision may be in, or overlapping, the inguinal and waist creases.[39,40] This moist environment may precipitate the incision being exposed to infectious organisms.[39,40] Wound healing may be inhibited by the shear forces generated by hip movement forcibly separating the skin edges.[38] Diabetic and obese patients are most at risk of post-operative wound complications after DAA. In TKA, the subvastus surgical approach has been shown to be protective of PWD.[41]

Wood et al[42] reported that the time taken for wound drainage to stop correlated strongly with the length of the *surgical incision*. Woolson et al,[43] however, reported that the risk of wound complications associated with the length of the wound was negligible provided it was less than 10 cm.

Prolonged tourniquet time has been correlated with an increase in superficial wound complications.[2] This may be attributable to local tissue hypoxia and inflammation compromising post-operative wound healing, as well as decreasing the local tissue concentration of prophylactic antibiotics during surgery. In TKA, shorter tourniquet inflation times and local infiltration of peri-articular anaesthesia significantly decrease subsequent wound drainage.[41] Inhibition of angiogenesis at the surgical incision edges due to relative tissue hypoxia with tourniquet use inhibits the migration of macrophages and fibroblasts necessary for an adequate cellular response. Conversely, release of a tourniquet after prolonged tourniquet use results in a reactive hyperaemia, excessive bleeding and as much as 10% increase in leg size, which places wound edges under undue tensile forces.[41,44] Local infiltration improves pain and facilitates early mobilization which stimulates and enhances soft tissue oxygenation.[41,44]

Duration of surgery in THA is positively correlated with an increase in both minor and major complications within 30 days of surgery. Operating time in THA between 120 and 179 minutes and longer than 180 minutes increased the risk of minor complications by 1.4 and 2.1 times.[45] Although it has been documented that prolonged surgical time predisposes patients to wound complications, we are not aware of any published studies that specifically evaluate the relationship between PWD and surgical time.

Management

In general, management of PWD should include non-surgical and surgical strategies. Jaberi et al[14] reported that PWD longer than 5–7 days was unlikely to respond to non-surgical treatment. Importantly, successful surgical treatment of PWD was associated with expeditious surgical intervention. Surgical debridement at five days was more likely to result in no infective complications at one year than delayed surgery after 10 days. Weiss et al[13] reported that only a quarter of patients had positive cultures when surgical debridement was carried out at 12 days post-operatively.

Prevention

Pre-operative, medical optimization is vital to allay the risk of post-operative wound complications.[11] Please refer to Table 1 for optimization of risk factors in TJA.

Non-surgical management

Non-surgical management includes immobilization with bed rest combined with braces and cessation of physical

Table 1. Summary of risk factors associated with wound complications in arthroplasty surgery

	Risk parameters	Suggestion	
Pre-operative risk factors - modifiable			
Obesity	BMI > 40 Kg/m^2	Nutritional optimization	2,4,11,22,46,47
Hypoalbuminaemia	Albumin < 35 g/L	Nutritional optimization	3,11,22,48,49
Smoking		4–8 weeks cessation	11,47,50–52
Anaemia	Hb < 13 g/Dl men Hb < 12 g/Dl women	Identify cause of anaemia and provide supplementation if needed Avoid unnecessary peri-operative blood transfusions	2,3,11,21,53
Staphylococcus aureus colonization	Nasal nare colonization	Decolonization with nasal Mupirocin	22,54
Poor dentition		Maintain favourable oral hygiene	55
Urinary tract infection	Symptomatic urinary tract infections	Treat symptomatic urinary tract infections	56,57
Pre-operative risk factors – non-modifiable			
Inflammatory arthropathy	Use of steroids and other immunosuppressive agents	Reduce steroids and other immunosuppressive agents	2,11,47,58
Diabetes mellitus	HBA1C > 7–8%	Medical optimization of treatment	3,11,22,47,59
COPD		Pulmonary assessment and optimization	29,30
Chronic anti-coagulation therapy	INR > 1.5	De-escalate pharmacological anti-coagulation depending on initial indication. Mechanical thromboprophylaxis with aspirin has least wound complication risk	2–4,22,32–34,60
Hepatitis C	Asymptomatic and symptomatic chronic infection	Medical optimization and counselling	27,28
HIV	< 200 CD4, viral load not suppressed	Medical optimization of treatment	26
Previous surgery to area		Adhere to correct surgical principles, adjust surgical incision or approach	11,25
Intra-operative risk factors			
Operating time	Prolonged operating time > 180 min	Optimize surgical time without compromising technique	22,45,61,62
Surgical approach	Higher risk with direct anterior approach to hip in obese patients, and with previous surgery	Tailor surgical approach to patient and patient's risk factors	11,39
Coagulation technique	Poor haemostasis	Meticulous haemostasis using surgical technique, electrocautery and local/systemic haemostatic agents	11,63
Antibiotic administration	Within 60 min of surgical time	IV and local prophylactic antibiotic administration	22,64
Tourniquet time	Prolonged tourniquet time more than 100 min	Reduce tourniquet time	2,41,44,65
Theatre etiquette		Sterility control, laminar flow, reduced traffic, body exhaust suits, temperature control	22
Skin preparation		Iodine or chlorhexidine in 70% alcohol	2,22,37

therapy, appropriate wound care, pressure bandages and cessation of pharmacological VTE prophylaxis.[3,11]

Limiting motion at the surgical site, including provisionally halting physical therapy while monitoring wound drainage for 24 to 48 hours, has been suggested.[23] Continuous passive motion should be stopped. Reich and Ezzet[23] and Shahi et al[6] suggested protocols whereby physical therapy is temporarily put on hold and knee immobilizers used.

The ideal dressing should protect the wound from infiltration of pathogens as well as be absorbent to deal with excess exudate. Initial management of PWD may start with absorbent dressings and pressure bandages.[19] A compressive dressing is all that may be needed for some wounds.[11] Pressure dressing together with other non-operative measures were used successfully in managing PWD by Shahi et al.[6] The use of silver-impregnated dressings has been proposed as their anti-microbial action has shown some benefit.[19]

Negative pressure wound therapy (NPWT) has been reported to decrease wound complications such as haematoma, seroma, dehiscence and infection.[66,67] NPWT reduces local tissue oedema, prevents deformation of the incision bed, stabilizes the wound environment, modulates inflammation, promotes angiogenesis and expedites the time to wound healing.[66] Redfern et al[68] reported a 45% reduction in post-operative haematoma and a 71% decrease in surgical site infections with the use of prophylactic NPWT. Wounds draining after the second or third day may benefit from NPWT, with an expected dry wound within 24 hours of application.[11] Hansen et al[12] found the use of NPWT for PWD resulted in the resolution of PWD in 76% of the patients it was used for. Although NPWT has been shown to be effective in managing PWD, prophylactic use of NPWT for all wounds may be limited by additional costs, resource constraints and an increased risk of severe blistering.[66,69] NPWT has many reported benefits, but there is no absolute indication for the use of NPWT,

and the use of NPWT should be directed by patient risk factors and clinical condition.[67,70]

Pharmacological anti-coagulation therapy has previously been discussed under the heading of risk factors. Anti-coagulation status needs to be reassessed with PWD, balancing the risks versus benefits when prescribing VTE prophylaxis, and short-term cessation should be considered depending on the agent prescribed and reason for anti-coagulation.[11,22,34] When pharmacological anti-coagulation is temporarily discontinued then mechanical VTE prophylaxis should be initiated or continued;[11] however, the evidence does not currently support the sole use of mechanical VTE prophylaxis in TJA.[36] Reich and Ezzet[23] suspended pharmacological anti-coagulation until the wound was assessed to be stable, similar to the protocol of Shahi et al.[6] Although temporary discontinuation of anti-coagulation therapy has been suggested, it is plausible to say the effects on PWD will depend on the type of anti-coagulation initially used, as each agent has different mechanisms of action with varying half-lives, and this needs further investigation (Fig. 1).

Antibiotic treatment has been described to treat PWD, although there are fears that indolent infection may be masked and subsequent laboratory investigations may be compromised.[23] A prospective observational study from Geneva did not find a protective effect of pre-emptive antibiotic therapy regarding future surgical site infections in the case of wound discharge or dehiscence.[71] If antibiotic therapy is chosen there should be a strong indication and prior to administration of any antibiotics, aspiration of the wound is suggested to confirm established infection and direct the therapy.[23] Culturing samples of wound drainage pre-operatively is not indicated as the yield is habitually only normal skin flora.[23] The current consensus discourages the indiscriminate use of antibiotics due to the lack of adequate evidence and risk of increasing antibiotic resistance.[3,24,72]

Surgical site aspiration was used successfully by Reich and Ezzet.[23] The aspiration was diagnostic to rule out infection as well as therapeutic in decompressing any

Fig. 1 An example of a surgical wound post total hip arthroplasty complicated by persistent wound drainage as a result of over anti-coagulation therapy

haematomas. If the aspiration was diagnostic for infection, the non-surgical approach was abandoned and treatment escalated to surgical debridement.[23] Reich and Ezzet[23] successfully treated 24 of 25 patients with PWD using a standardized protocol utilizing surgical site aspiration together with other mentioned non-surgical approaches including closure of open areas of wounds, pausing anti-coagulation therapy, limiting activity and selective prescription of antimicrobial therapy. Limited experience with this treatment strategy makes it difficult to recommend; however, Shahi et al[6] reported successfully managing 65% of patients with PWD with similar approaches using local wound care, pausing anti-coagulation, and reducing movement to the surgical area. Therefore, a combination of these various non-surgical interventions can be recommended with further well-designed prospective studies needed to determine the best possible treatment protocol.

Surgical management

The 2013 International Consensus Meeting on musculoskeletal infections[15] strongly advised strict monitoring of continued wound drainage persisting longer than 72 hours. Surgical management should be considered when PWD continues for more than 5–7 days after initial surgery despite non-surgical management.[15,19] Early surgical exploration and debridement within 5–7 days post-operatively has been shown to resolve PWD in 76% of cases, and it has been noted that delaying debridement may result in PJI.[14]

Surgical treatment for PWD is neither insignificant nor minor surgery and may potentiate the risk of future morbidity and PJI.[11] If a wound has been deemed problematic as described previously, a minimum of superficial exploration with debridement and haematoma evacuation should be performed.[11]

Joint aspiration is recommended prior to the skin incision of surgical debridement to exclude deep infection.[11] Multiple intra-operative tissue samples during surgical debridement should be obtained and cultured for up to 14 days, and empiric antibiotic treatment adjusted according to culture results.[3,11,24] If the joint capsule appears to be compromised intra-operatively, and deep infection is suspected, surgical treatment can be escalated to debridement, antibiotics and implant retention, commonly referred to as a DAIR procedure.[1] As previously suggested under the discussion of non-operative protocols, once deep infection is confirmed the diagnosis and management should shift to that of an acute periprosthetic joint infection. The objective of a DAIR procedure is to reduce the infective microbial load around the prosthesis and wound, including breaking down biofilm.[1,11] Surgical management involves open deep debridement of the joint and thorough wound irrigation and wherever possible modular bearings should be exchanged.[3,11,22,24] The bearings are removed to provide better access to all

prosthetic surfaces, and not exchanging the polyethylene liners has been reported to increase the risk of failure.[24,73]

Antibiotic choice and duration of treatment post-operatively is controversial and will depend on the susceptibilities and virulence of pathogens isolated, route of administration and the need for repeat procedures and host factors.[73] Empiric antibiotics are started after the procedure and de-escalated where appropriate as soon as microbiology results are available.[24] Discussion between the orthopaedic surgeon, microbiologist and infectious disease specialist is suggested to determine the most optimal treatment while still respecting antibiotic stewardship.[73]

Success of the DAIR procedure is defined as retention of the implants without the need for subsequent DAIR procedures or long-term suppressive antibiotic therapy.[74,75] Risk factors for an unsuccessful DAIR procedure include raised inflammatory markers, infection with *Staphylococcus aureus*, retention of polyethylene components, and arthroscopic debridement.[75] Longer duration of symptoms is also a predictor for failure, and therefore the sooner the procedure is carried out the better the outcomes can be expected.[75] Studies have shown the risk of higher failure rates of two-stage revisions if a DAIR procedure has failed,[75] therefore once there is any wound complication suspected following TJA every effort needs to be made to address the identified problem in a timely and efficient manner.

Conclusion

The goal in managing PWD is to minimize the time to achieve a dry, healed wound. Emphasis should be placed on prevention of PWD by identifying and addressing previously discussed risk factors pre-operatively to optimize the patient's condition. Once PWD is identified there should be no time delay in utilizing both non-surgical and surgical treatment options to ultimately prevent the consequence of PJI and the need for revision surgery. However, there is still variation in clinical practice because of the lack of consensus regarding the definition of PWD as well as the lack of evidence-based guidelines in the management of PWD. Future prospective and adequately powered studies evaluating management protocols addressing all aspects of PWD are needed.

AUTHOR INFORMATION

Arthroplasty Unit, Division of Orthopaedic Surgery, Charlotte Maxeke Johannesburg Academic Hospital, University of the Witwatersrand, Johannesburg, South Africa.

Correspondence should be sent to: Dr. Richard Peter Almeida, Arthroplasty Unit, Division of Orthopaedic Surgery, Charlotte Maxeke Johannesburg Academic Hospital, University of the Witwatersrand, Johannesburg, South Africa.
Email: rich.almeida11@gmail.com

SOCIAL MEDIA

http://linkedin.com/in/richard-almeida-a70987121

REFERENCES

1. Löwik CAM, Wagenaar FC, van der Weegen W, et al; LEAK study group. LEAK study: design of a nationwide randomised controlled trial to find the best way to treat wound leakage after primary hip and knee arthroplasty. *BMJ Open* 2017;7:e018673.

2. Carroll K, Dowsey M, Choong P, Peel T. Risk factors for superficial wound complications in hip and knee arthroplasty. *Clin Microbiol Infect* 2014;20:130–135.

3. Wagenaar FBM, Löwik CAM, Zahar A, Jutte PC, Gehrke T, Parvizi J. Persistent wound drainage after total joint arthroplasty: a narrative review. *J Arthroplasty* 2019;34:175–182.

4. 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-A:33–38.

5. Eveillard M, Mertl P, Canarelli B, et al. [Risk of deep infection in first-intention total hip replacement: evaluation concerning a continuous series of 790 cases]. *Presse Med* 2001;30:1868–1875.

6. Shahi A, Boe R, Bullock M, et al. The risk factors and an evidence-based protocol for the management of persistent wound drainage after total hip and knee arthroplasty. *Arthroplast Today* 2019;5:329–333.

7. Carli AV, Negus JJ, Haddad FS. Periprosthetic femoral fractures and trying to avoid them: what is the contribution of femoral component design to the increased risk of periprosthetic femoral fracture? *J Bone Joint Surg [Br]* 2017;99-B:50–59.

8. Natsuhara KM, Shelton TJ, Meehan JP, Lum ZC. Mortality during total hip periprosthetic joint infection. *J Arthroplasty* 2019;34:S337–S342.

9. Janis JE, Harrison B. Wound healing: part I. basic science. *Plast Reconstr Surg* 2016;138:9S–17S.

10. Al-Houraibi RK, Aalirezaie A, Adib F, et al. General assembly, prevention, wound management: proceedings of international consensus on orthopedic infections. *J Arthroplasty* 2019;34:S157–S168.

11. Scuderi GR. Avoiding postoperative wound complications in total joint arthroplasty. *J Arthroplasty* 2018;33:3109–3112.

12. **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;471:3230–3236.

13. **Weiss AP, Krackow KA.** Persistent wound drainage after primary total knee arthroplasty. *J Arthroplasty* 1993;8:285–289.

14. **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;466:1368–1371.

15. **Parvizi J, Gehrke T, Chen AF.** Proceedings of the international consensus on periprosthetic joint infection. *J Bone Joint Surg [Br]* 2013;95-B:1450–1452.

16. **Zmistowski B, Karam JA, Durinka JB, Casper DS, Parvizi J.** Periprosthetic joint infection increases the risk of one-year mortality. *J Bone Joint Surg [Am]*. 2013;95-A:2177-2184.

17. **Saleh K, Olson M, Resig S, et al.** Predictors of wound infection in hip and knee joint replacement: results from a 20 year surveillance program. *J Orthop Res* 2002;20:506–515.

18. **Ghanem E, Heppert V, Spangehl M, et al.** Wound management. *J Orthop Res* 2014;32:S108–S119.

19. **Wagenaar F-C, Löwik CAM, Stevens M, et al.** Managing persistent wound leakage after total knee and hip arthroplasty: results of a nationwide survey among Dutch orthopaedic surgeons. *J Bone Jt Infect* 2017;2:202–207.

20. **Ekmektzoglou KA, Zografos GC.** A concomitant review of the effects of diabetes mellitus and hypothyroidism in wound healing. *World J Gastroenterol* 2006;12:2721–2729.

21. **Weber EWG, Slappendel R, Prins MH, van der Schaaf DB, Durieux ME, Strümper D.** Perioperative blood transfusions and delayed wound healing after hip replacement surgery: effects on duration of hospitalization. *Anesth Analg* 2005;100:1416–1421.

22. **Illingworth KD, Mihalko WM, Parvizi J, Sculco T, McArthur B, El Bitar Y, et al.** How to minimize infection and thereby maximize patient outcomes in total joint arthroplasty: a multicenter approach. *J Bone Joint Surg [Am]* 2013;95-A:1–13.

23. **Reich MS, Ezzet KA.** A nonsurgical protocol for management of postarthroplasty wound drainage. *Arthroplast Today* 2017;4:71–73.

24. **Barros LH, Barbosa TA, Esteves J, Abreu M, Soares D.** Early debridement, antibiotics and implant retention (DAIR) in patients with suspected acute infection after hip or knee arthroplasty: safe, effective and without negative functional impact. *J Bone Jt Infect* 2019;4:300–305.

25. **Jones RE, Russell RD, Huo MH.** Wound healing in total joint replacement. *J Bone Joint Surg [Br]* 2013;95-B:144–147.

26. **Chang CH, Tsai SW, Chen CF, et al.** Optimal timing for elective total hip replacement in HIV-positive patients. *Orthop Traumatol Surg Res* 2018;104:671–674.

27. **Issa K, Boylan MR, Naziri Q, Perfetti DC, Maheshwari AV, Mont MA.** The impact of hepatitis C on short-term outcomes of total joint arthroplasty. *J Bone Joint Surg [Am]* 2015;97-A:1952–1957.

28. **Pour AE, Matar WY, Jafari SM, Purtill JJ, Austin MS, Parvizi J.** Total joint arthroplasty in patients with hepatitis C. *J Bone Joint Surg [Am]* 2011;93-A:1448–1454.

29. **Yakubek GA, Curtis GL, Sodhi N, et al.** Chronic obstructive pulmonary disease is associated with short-term complications following total hip arthroplasty. *J Arthroplasty* 2018;33:1926–1929.

30. **Gu A, Wei C, Maybee CM, Sobrio SA, Abdel MP, Sculco PK.** The impact of chronic obstructive pulmonary disease on postoperative outcomes in patients undergoing revision total knee arthroplasty. *J Arthroplasty* 2018;33:2956–2960.

31. **Lum ZC, Monzon RA, Bosque J, Coleman S, Pereira GC, Di Cesare PE.** Effects of fondaparinux on wound drainage after total hip and knee arthroplasty. *J Orthop* 2018;15:388–390.

32. **McDougall CJ, Gray HS, Simpson PM, Whitehouse SL, Crawford RW, Donnelly WJ.** Complications related to therapeutic anticoagulation in total hip arthroplasty. *J Arthroplasty* 2013;28:187–192.

33. **Parvizi J, Ghanem E, Joshi A, Sharkey PF, Hozack WJ, Rothman RH.** Does 'excessive' anticoagulation predispose to periprosthetic infection? *J Arthroplasty* 2007;22:24–28.

34. **Jones CW, Spasojevic S, Goh G, Joseph Z, Wood DJ, Yates PJ.** Wound discharge after pharmacological thromboprophylaxis in lower limb arthroplasty. *J Arthroplasty* 2018;33:224–229.

35. **Agaba P, Kildow BJ, Dhotar H, Seyler TM, Bolognesi M.** Comparison of postoperative complications after total hip arthroplasty among patients receiving aspirin, enoxaparin, warfarin, and factor Xa inhibitors. *J Orthop* 2017;14:537–543.

36. **Pellegrini VD Jr.** Prophylaxis against venous thromboembolism after total hip and knee arthroplasty: a critical analysis review. *JBJS Rev* 2015;3:1–9.

37. **Ayoub F, Quirke M, Conroy R, Hill A.** Chlorhexidine-alcohol versus povidone-iodine for pre-operative skin preparation: a systematic review and meta-analysis. *Int J Surg Open [Internet]* 2015;2015:41–46.

38. **Jahng KH, Bas MA, Rodriguez JA, Cooper HJ.** Risk factors for wound complications after direct anterior approach hip arthroplasty. *J Arthroplasty* 2016;31:2583–2587.

39. **Watts CD, Houdek MT, Wagner ER, Sculco PK, Chalmers BP, Taunton MJ.** High risk of wound complications following direct anterior total hip arthroplasty in obese patients. *J Arthroplasty* 2015;30:2296–2298.

40. **Purcell RL, Parks NL, Gargiulo JM, Hamilton WG.** Severely obese patients have a higher risk of infection after direct anterior approach total hip arthroplasty. *J Arthroplasty* 2016;31:162–165.

41. **Butt U, Ahmad R, Aspros D, Bannister GC.** Factors affecting wound ooze in total knee replacement. *Ann R Coll Surg Engl* 2011;93:54–56.

42. **Wood JJ, Bevis PM, Bannister GC.** Wound oozing after total hip arthroplasty. *Ann R Coll Surg Engl* 2007;89:140–142.

43. **Woolson ST, Mow CS, Syquia JF, Lannin J, Schurman DJ.** Comparison of primary total hip replacements performed with a standard incision or a mini-incision. *J Bone Joint Surg [Am]* 2004;86-A:1353–1358.

44. **Rama KRBS, Apsingi S, Poovali S, Jetti A.** Timing of tourniquet release in knee arthroplasty: meta-analysis of randomized, controlled trials. *J Bone Joint Surg [Am]* 2007;89-A:699–705.

45. **Nowak LL, Schemitsch EH.** Duration of surgery affects the risk of complications following total hip arthroplasty. *J Bone Joint Surg [Br]* 2019;101-B:51–56.

46. **Shohat N, Fleischman A, Tarabichi M, Tan TL, Parvizi J.** Weighing in on body mass index and infection after total joint arthroplasty: is there evidence for a body mass index threshold? *Clin Orthop Relat Res* 2018;476:1964–1969.

47. **Everhart JS, Andridge RR, Scharschmidt TJ, Mayerson JL, Glassman AH, Lemeshow S.** Development and validation of a preoperative surgical site infection risk score for primary or revision knee and hip arthroplasty. *J Bone Joint Surg [Am]* 2016;98-A:1522–1532.

48. **Bohl DD, Shen MR, Kayupov E, Della Valle CJ.** Hypoalbuminemia independently predicts surgical site infection, pneumonia, length of stay, and readmission after total joint arthroplasty. *J Arthroplasty* 2016;31:15–21.

49. Gu A, Malahias MA, Strigelli V, Nocon AA, Sculco TP, Sculco PK. Preoperative malnutrition negatively correlates with postoperative wound complications and infection after total joint arthroplasty: a systematic review and meta-analysis. *J Arthroplasty* 2019;34:1013–1024.

50. Debbi EM, Rajaee SS, Spitzer AI, Paiement GD. Smoking and total hip arthroplasty: increased inpatient complications, costs, and length of stay. *J Arthroplasty* 2019;34:1736–1739.

51. 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-A:1049–1058.

52. Bedard NA, DeMik DE, Owens JM, Glass NA, DeBerg J, Callaghan JJ. Tobacco use and risk of wound complications and periprosthetic joint infection: a systematic review and meta-analysis of primary total joint arthroplasty procedures. *J Arthroplasty* 2019;34:385–396.e4.

53. Kotzé A, Carter LA, Scally AJ. Effect of a patient blood management programme on preoperative anaemia, transfusion rate, and outcome after primary hip or knee arthroplasty: a quality improvement cycle. *Br J Anaesth* 2012;108:943–952.

54. Sporer SM, Rogers T, Abella L. Methicillin-resistant and methicillin-sensitive Staphylococcus aureus screening and decolonization to reduce surgical site infection in elective total joint arthroplasty. *J Arthroplasty* 2016;31:144–147.

55. Barrere S, Reina N, Peters OA, Rapp L, Vergnes JN, Maret D. Dental assessment prior to orthopedic surgery: a systematic review. *Orthop Traumatol Surg Res* 2019;105:761–772.

56. Ollivere BJ, Ellahee N, Logan K, Miller-Jones JCA, Allen PW. Asymptomatic urinary tract colonisation predisposes to superficial wound infection in elective orthopaedic surgery. *Int Orthop* 2009;33:847–850.

57. Gou W, Chen J, Jia Y, Wang Y. Preoperative asymptomatic leucocyturia and early prosthetic joint infections in patients undergoing joint arthroplasty. *J Arthroplasty* 2014;29:473–476.

58. Somayaji R, Barnabe C, Martin L. Risk factors for infection following total joint arthroplasty in rheumatoid arthritis. *Open Rheumatol J* 2013;7:119–124.

59. Lopez LF, Reaven PD, Harman SM. Review: the relationship of hemoglobin A1c to postoperative surgical risk with an emphasis on joint replacement surgery. *J Diabetes Complications* 2017;31:1710–1718.

60. Mortazavi SMJ, Hansen P, Zmistowski B, Kane PW, Restrepo C, Parvizi J. Hematoma following primary total hip arthroplasty: a grave complication. *J Arthroplasty* 2013;28:498–503.

61. Bohl DD, Ondeck NT, Darrith B, Hannon CP, Fillingham YA, Della Valle CJ. Impact of operative time on adverse events following primary total joint arthroplasty. *J Arthroplasty* 2018;33:2256–2262.e4.

62. Pugely AJ, Martin CT, Gao Y, Schweizer ML, Callaghan JJ. The incidence of and risk factors for 30-day surgical site infections following primary and revision total joint arthroplasty. *J Arthroplasty* 2015;30:47–50.

63. Remérand F, Cotten M, N'Guessan YF, et al. Tranexamic acid decreases risk of haematomas but not pain after hip arthroplasty. *Orthop Traumatol Surg Res* 2013;99: 667–673.

64. Chandrananth J, Rabinovich A, Karahalios A, Guy S, Tran P. Impact of adherence to local antibiotic prophylaxis guidelines on infection outcome after total hip or knee arthroplasty. *J Hosp Infect* 2016;93:423–427.

65. Olivecrona C, Lapidus LJ, Benson L, Blomfeldt R. Tourniquet time affects postoperative complications after knee arthroplasty. *Int Orthop* 2013;37:827–832.

66. Manoharan V, Grant AL, Harris AC, Hazratwala K, Wilkinson MPR, McEwen PJC. Closed incision negative pressure wound therapy vs conventional dry dressings after primary knee arthroplasty: a randomized controlled study. *J Arthroplasty* 2016;31:2487–2494.

67. Shiroky J, Lillie E, Muaddi H, Sevigny M, Choi WJ, Karanicolas PJ. The impact of negative pressure wound therapy for closed surgical incisions on surgical site infection: a systematic review and meta-analysis. *Surgery* 2020;167:1001–1009.

68. Redfern RE, Cameron-Ruetz C, O'Drobinak SK, Chen JT, Beer KJ. Closed incision negative pressure therapy effects on postoperative infection and surgical site complication after total hip and knee arthroplasty. *J Arthroplasty* 2017;32:3333–3339.

69. Howell RD, Hadley S, Strauss E, Pelham FR. Blister formation with negative pressure dressings after total knee arthroplasty. *Curr Orthop Pract* 2011;22:176–179.

70. Wang C, Zhang Y, Qu H. Negative pressure wound therapy for closed incisions in orthopedic trauma surgery: a meta-analysis. *J Orthop Surg Res* 2019;14:427.

71. Wuarin L, Abbas M, Harbarth S, et al. Changing perioperative prophylaxis during antibiotic therapy and iterative debridement for orthopedic infections? *PLoS One* 2019;14:e0226674.

72. Uçkay I, Agostinho A, Belaieff W, et al. Noninfectious wound complications in clean surgery: epidemiology, risk factors, and association with antibiotic use. *World J Surg* 2011;35:973–980.

73. Qasim SN, Swann A, Ashford R. The DAIR (debridement, antibiotics and implant retention) procedure for infected total knee replacement: a literature review. *SICOT J* 2017;3:2.

74. Kim K, Zhu M, Cavadino A, Munro JT, Young SW. Failed debridement and implant retention does not compromise the success of subsequent staged revision in infected total knee arthroplasty. *J Arthroplasty* 2019;34:1214–1220.e1.

75. Löwik CAM, Jutte PC, Tornero E, et al; Northern Infection Network Joint Arthroplasty (NINJA). Predicting failure in early acute prosthetic joint infection treated with debridement, antibiotics, and implant retention: external validation of the KLIC score. *J Arthroplasty* 2018;33:2582–2587.