Hardware Management in Postoperative Infections

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A postoperative infection, as defined by The Centers for Disease Control and Prevention (CDC), must be diagnosed within 30 days of the surgery, or 1 year if an implant is in place and the infection is closely related to the incision site. Surgical site infections account for 38% of all nosocomial infections (1), but are relatively low for ambulatory surgery (0.31% at 14 days and 0.48% at 30 days) (2).

The risks of having a postoperative infection correlate with microbial, patient, and surgical characteristics. The microbial characteristics include the virulence of the organisms and the burden of inoculation. It is estimated that 10^5 organisms are required to cause an infection in a surgical wound with proper antibiotic prophylaxis. The patient characteristics take into account their comorbidities and their immune system. The surgical characteristics include presence of implanted foreign material, endogenous, and exogenous contamination during the procedure (3). It is important to know that gram-positive cocci from skin flora remain the leading cause of surgical site infections. Even with surgical scrubbing of the skin, there is risk of contamination, since 20% of the skin flora is present in the skin appendages (4).

The exogenous contamination can be due to operating room (OR) personnel, surgical instruments, deficient ventilation systems, and other factors such as OR traffic. The degree of microbial contamination in the air is directly proportional to the number of personnel present in the OR (3,5). There are several modifiable risk factors that should be noted, such as surgical time and technique. A surgery lasting longer than 90 minutes was identified as an independent risk factor for postoperative infections (6). An atraumatic surgical technique will limit damaged or non-viable tissues that serve as a substrate for bacterial colonization. Also, acellular tissues are at increased risk of colonization, (i.e., cartilage or bone stripped of its periosteum). Some strains of *Staphylococcus aureus* have collagen receptors that directly bind to ligands on articular cartilage (7).

Postoperative infections are always a stressful complication of surgery, but the presence of hardware in the surgical site makes it even more challenging because of the biofilm formation. A biofilm is a structured association of bacteria embedded in a self-produced polymer matrix consisting of protein, polysaccharide, and extracellular DNA. Biofilms typically grow on foreign material, like implants or hardware. They are associated with prolonged resistant infections, due to their resistance to antibiotics and immune defense. This microenvironment has nutrientpoor areas with metabolically inactive cells, responsible for tolerance to antibiotics. The biofilm growth is associated with mutations and resistance (8). This complicating factor is one of the definitive reasons why the decision to remove or keep the hardware in place is of concern. The presence of colonized biomaterials at the infection site is also of concern. Significantly higher levels of antibiotics are necessary to eliminate surface adherent bacteria, even if they are not a biofilm producer .

Staphylococcus aureus is the dominant organism associated with infected metal implants. A retrospective study by Torbert et al (10) evaluated the bacterial speciation and antibiotic resistance in deep infections after operative fixation of fractures and identified that 56% of the infections had *S aureus* present, which was methicillin resistant in 58% of the cases in their center. The second most common group of organisms was gram-negative rods, present in 32% of the cases, with 4% being drug resistant.

The decision of implant type should be part of the surgical planning, as some materials, surfaces, and configurations are more prone to bacterial adherence. A study by Chang and Merritt evaluated the adherence of Staphylococcus epidermitis on various types of materials. They concluded that adherence was more concentrated on stainless steel followed by titanium, with polymethylmethacrylate to be the least adherent. The surface roughness of the material has also been proven to increase bacterial attachment and biofilm formation, which is less likely with a smooth surface (12, 13). The surface configuration has also been evaluated and porous materials have a higher infection rate than more dense materials. It has been shown that bacteria tend to adhere to crevasses and irregularities that conform to their size, rather than grooves larger or smaller than their dimension (14).

In the case of a fracture, stability is another parameter of infection susceptibility. Even though the presence of implants increase the risk of infection with biofilm formation, animal studies have shown that contaminated fractures without internal fixation develop clinical infection more frequently than fixated contaminated fractures. Worlock et al performed an animal study with applied tibial fracture in rabbits and inoculated them with *S aureus*. Half of the subjects were fixated with a stable dynamic compression plate and the remaining half with a loose rod. Their study demonstrated that 71% of the unstable group developed osteomyelitis versus 35% infection in the stable group. These findings suggest that fixation of a fracture is less susceptible to the development of infection due to structural stability of bone fragments and stable soft tissue architecture, which may promote quicker revascularization (16).

To evaluate and diagnose a postoperative infection with implanted hardware, we can relate to the same parameters that are described in the Infectious Diseases Society of America (IDSA) guidelines for prosthetic joint infections (17). A thorough history and physical examination are paramount. It is important to consider the type of surgery, type of material implanted, and if there was presence of intra-operative complications. Additionally, consideration should be given with regard to whether the patient has a history of allergic reactions or intolerance to some materials or medications, or healing issues after surgery. If there are signs and symptoms of infection, the onset of the symptoms are important, because a determination of acute or chronic infection must be diagnosed. Clinical evaluation of the open wound, sinus tract, purulence, or other signs of infection should be noted, and wound cultures should be taken immediately. A sedimentation rate and a C-reactive protein level should be obtained, and if they are both elevated, suspicion of infection is high. A plain radiograph should be taken to evaluate the integrity of the hardware as well as the presence of periosteal reaction, lucency of the hardware, or bone erosions. In the case of a suspected infected total ankle joint, diagnostic arthrocentesis should be done. As in any infection workup, blood cultures are also mandatory if systemic symptoms are present.

Once it is determined that an infection is present with intact hardware, a decision must be made to explant or retain the hardware. Viol et al (18) reviewed the literature from 1960 to 2009 and determined important factors in the management of exposed hardware. They concluded that clinical signs of infection in conjunction with positive cultures define a true infection. They also concluded that the severity of the infection clinically along with positive cultures might necessitate explantation of the hardware. It is also common to retain exposed hardware with positive cultures, but soft tissue coverage is necessary in conjunction with proper antibiotics. However, the rate of failure is greater. It is important to note that a period of infection and hardware exposure of less than 2 weeks leads to higher rates of hardware salvage. A prolonged hardware exposure of more than 1 month is defined as a chronic infection, which is an indication to remove the implants.

The next factor to assess is the stability of the hardware. If the hardware is fractured or unstable, it should immediately be removed. This thorough review also evaluated the location of the hardware. The authors indicated that it is routinely preserved in spinal surgery due to no other alternatives to maintain stability. Lower extremity infection has been traditionally treated with fixation removal and application of external fixation to maintain stability, however, retention is becoming consistently more frequent. If the hardware is preserved, soft tissue coverage is necessary after proper incision and drainage procedures.

A retrospective study by Patel et al (19) evaluated the factors associated with failure in hardware salvage. They included all patients with attempted hardware salvage via free tissue transfer. The factors identified with failed hardware salvage were multiple comorbidities, longer duration before hardware coverage, and increased time of intravenous antibiotics along with positive initial wound cultures and chronic osteomyelitis on initial pathology. The IDSA recommendations (17) are available for infected total joints including hips, knees and ankles. They recommend consideration for debridement in the operating room with retention of the prosthesis if it has been placed within 30 days or if the symptoms have been present for less than 3 weeks. If the prosthetic is stable without a sinus tract, and if susceptible to oral antimicrobial agents, retention is possible. Their recommendations for antibiotherapy can be utilized as a guide not only for total joint implants, but also for other types of hardware. For a staphylococcus infection, the recommendations include 2-6 weeks of pathogen specific parenteral antibiotherapy in combination with rifampin (300-450 mg twice a day). The rifampin is added because of its property to penetrate the biofilm. This regimen needs to be followed by 3 months of oral antibiotic therapy combined with rifampin for total ankle joints. If it is a non-staphylococcal infection, they recommend 4-6 weeks of pathogen specific parenteral or highly bioavailable oral therapy.

If the condition is stable for implant retention, it is paramount for soft tissue coverage of the hardware immediately. There are multiple strategies that can be used for hardware coverage, (i.e., muscle flaps and split-thickness skin grafts) (Figures 1-5). Flap related complications can occur but are often needed in the face of exposed hardware (20). Common risk factors related to flap failure are peripheral arterial disease, history of multiple angioplasties in the extremity, and use of immunosuppressive agents following renal transplant (21).

If the patient is not a candidate for a muscle flap, coverage can be achieved with biological grafts (Figures 6, 7) in conjunction with wound VAC therapy. Primary closure following debridement is typically more successful when possible (Figures 8, 9). Aytac et al (22) described a concept of persisting fistula on 59 patients with postoperative osteomyelitis. After radical debridement, irrigation, and hardware retention, a drain was inserted in contact with the implant before the wound was closed. The drain and

hardware were maintained until wound healing and fracture consolidation for approximately 6-8 weeks. This technique demonstrated 89% of success with bone healing.

Berkes et al (23) performed a retrospective study of 123 infected postoperative fractures one to six weeks after surgery treated with debridement, culture specific antibiotics and maintenance of hardware. They demonstrated 87 (71%) of the patients had successful osseous union with retained hardware, but 26 later needed a removal of hardware due to



Figure 1. Patient 2 weeks after revisional ankle arthrodesis.



Figure 3. Split-thickness skin graft applied to cover the gracilis muscle flap.

infection recurrence. A retrospective study by Rightmire et al (24) showed similar results, with 68% of union with retained hardware, requiring later hardware removal for recurrent infection in 36% of the cases. These studies show that there is an acceptable success rate, but with high infection recurrence necessitating delayed hardware removal. These findings suggest that hardware removal after osseous union may provide the best outcome (Figures 10-12).



Figure 2. Debridement and application of gracilis muscle flap with retained hardware.



Figure 4. Appearance at 8 months postoperative.



Figure 5. Successful union with retained hardware.



Figure 7. Coverage is achieved with a bilayer biological graft.

In conclusion, management of infection with retained hardware demonstrates much controversy in the literature. The key components are diagnosis of the infection early, proper soft tissue coverage and directed antibiotic therapy. Delays in any of these steps have higher rates of failure. A multidisciplinary approach with plastic surgeons and infectious diseases specialists are also paramount in the management of these cases.



Figure 6. Appearance 2 weeks after first metatarsocuneiform joint arthrodesis.



Figure 8. Patient 2 weeks after first metatarsophalangeal fusion.

REFERENCES

- Lewis SS, Mochring RW, Chen LF, Sexton DJ, Anderson DJ. Assessing the relative burden of hospital-acquired infections in a network of community hospitals. Infect Control Hosp Epidemiol 2013;34:1229-30.
- Owens PL, Barrett ML, Raetzman S, Maggard-Gibbons M, Steiner CA. Surgical site infections following ambulatory surgery procedures. JAMA 2014;311:709-16.
- Anderson DJ. Surgical site infections. Infect Dis Clin North Am 2011;25:135-53.
- Tuazon CU. Skin and skin structure infections in the patient at risk: carrier state of Staphylococcus aureus. Am J Med 1984;76:166-71.



Figure 9. After debridement and deep cultures are taken, the wound was primarily closed and the patient was placed on the proper antibiotherapy.



Figure 11. Hardware is removed after bone union and the surgical site is closed and bilayer biological graft applied.

- Ayliffe GA. Role of the environment of the operating suite in surgical wound infection. Rev Infect Dis 1991;13 Suppl 10:S800-4.
- Ovaska MT, Makinen TJ, Madanat R, Huotari K, Vahlberg T, Hirvensalo E, et al. Risk factors for deep surgical site infection following operative treatment of ankle fractures. J Bone Joint Surg Am 2013;95:348-53.
- Gristina AG, Naylor PT, Myrvik QN. Mechanisms of musculoskeletal sepsis. Orthop Clin North Am 1991;22:363-71.
- Hoiby N, Ciofu O, Johansen HK, Song Z-J, Moser C, Jensen PO, et al. The clinical impact of bacterial biofilms. Int J Oral Sci 2011; 3:55-65.
- Barth E, Myrvik QM, Wagner W, Gristina AG. In vitro and in vivo comparative colonization of Staphylococcus aureus and Staphylococcus epidermidis on orthopaedic implant materials. Biomaterials 1989;10:325-8.
- Torbert JT, Joshi M, Moraff A, Matuszewski PE, Holmes A, Pollak AN, et al. Current bacterial speciation and antibiotic resistance in deep infections after operative fixation of fractures. J Orthop Trauma 2015;29:7-17.



Figure 10. Presence of osseous union at the first metatarsocuneiform joint.



Figure 12. Patient is now completely healed.

- 11 Chang CC, Merritt K. Infection at the site of implanted materials with and without preadhered bacteria. J Orthop Res 1994;12:526-31.
- Scheuerman TR, Camper AK, Hamilton MA. Effects of substratum topography on bacterial adhesion. J Colloid Interface Sci 1998;208:23-33.
- Mitik-Dineva N, Wang J, Mocanasu RC, Stoddart PR, Crawford RJ, Ivanova EP. Impact of nano-topography on bacterial attachment. Biotechnol J 2008;3:536-44.
- Katsikogianni M, Missirlis YF. Concise review of mechanisms of bacterial adhesion to biomaterials and of techniques used in estimating bacteria-material interactions. Eur Cell Mater 2004; 8:37-57.
- Worlock P, Slack R, Harvey L, Mawhinney R. The prevention of infection in open fractures: an experimental study of the effect of fracture stability. Injury 1994;25:31-8.
- Schmidt AH, Swiontkowski MF. Pathophysiology of infections after internal fixation of fractures. J Am Acad Orthop Surg 2000;8: 285-91.

- 17. Osmon DR, Berbari EF, Berendt AR, Lew D, Zimmerli W, Steckelberg JM, et al. Diagnosis and management of prosthetic joint infection: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis 2013;56:e1-25.
- Viol A, Pradka SP, Baumeister SP, Wang D, Moyer KE, Zura RD, et al. Soft-tissue defects and exposed hardware: a review of indications for soft-tissue reconstruction and hardware preservation. Plast Reconstr Surg 2009;123:1256-63.
- Patel KM, Seruya M, Franklin B, Attinger CE, Ducic I. Factors associated with failed hardware salvage in high-risk patients after microsurgical lower extremity reconstruction. Ann Plast Surg 2012;69:399-402.
- Ovaska MT, Madanat R, Tukiainen E, Pulliainen L, Sintonen H, Makinen TJ. Flap reconstruction for soft-tissue defects with exposed hardware following deep infection after internal fixation of ankle fractures. Injury 2014;45:2029-34.

- Oh TS, Lee HS, Hong JP. Diabetic foot reconstruction using free flaps increases 5-year-survival rate. J Plast Reconstr Aesthet Surg 2013;66:243-50.
- Aytaç S, Schnetzke M, Swartman B, Herrmann P, Woelfl C, Heppert V, et al. Posttraumatic and postoperative osteomyelitis: surgical revision strategy with persisting fistula. Arch Orthop Trauma Surg 2014;134:159-65.
- Berkes M, Obremskey WT, Scannell B, Ellington JK, Hymes RA, Bosse M, et al. Maintenance of hardware after early postoperative infection following fracture internal fixation. J Bone Joint Surg Am 2010;92:823-8.
- 24. Rightmire E, Zurakowski D, Vrahas M. Acute infections after fracture repair: management with hardware in place. Clin Orthop Relat Res 2008;466:466-72.