Postoperative infection – removal of screws and plates?

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Infection is an omnipresent risk of every surgical procedure. Having hardware poses an extra risk because implanted material is avascular, and therefore not protected by the host´s immune system. Antibiotics can only reach infected area by diffusion from the surrounding tissues. The absence of vascularized tissue and the presence of metal hardware near the wound create the environment for postoperative infection. Unfilled dead space, such as the space that develops around hardware and bones when significant soft tissue damage occurs, increases the chance for infection (1).

Susceptibility to infection

The three key features of susceptibility to infection are firstly the personality of the injury (open/closed, degree of soft tissue injury, energy of fracture, degree of vascular injury or contamination, and patient aspects such as age, diabetes, steroids, smoking, drugs or alcohol abuse, and compliance), secondly, the quality of the operation and facilities (surgical technique, postoperative care and cleanliness), and thirdly, the nature of the device (biocompatibility of material used, implant surface properties, implant design, number of possible dead spaces and adjacent beside moving tissues as tendons) (2).

Biofilm

Implanted biomaterials are surrounded by an immunoincompetent fibroinflammatory zone. Gristina et al (3) postulated that bacteria adhere to the implant, colonize its surface and form a biofilm, acquiring additional pathogenic potential. In biofilm bacteria enter into a slow or stationary phase, which makes them much more resistant to most antimicrobial agents. Once the biofilm has established, immune system and antibiotics cannot eradicate bacteria until the implant is removed. Because of biofilm production, prompt treatment of early infections with sufficient antimicrobial treatment is preferable to treating delayed or late infections, where biofilm already exists protecting the bacteria within it. Early diagnosis is therefore very important with a combination of clinical, histopathological and microbiological studies (2).

Treatment

Treatment of postoperative wound infection includes a prolonged course of antibiotics, debridement, and most importantly, removal of the infected fixation material. Nevertheless, fractures present a dilemma when infection occurs in the acute postoperative period (<6 weeks), as vast majority of fractures will not have achieved osseous union in this time period. Either fracture stabilization and healing are optimized through retention of hardware, or the hardware is removed to give the patient the best chance to clear the infectious process. It is important to understand that hardware removal may result in substantial morbidity, and is not always a favourable option if a functional outcome is to be obtained. Thus, physicians are faced with a challenging situation where the hardware must be maintained even though it is infected (4,5).

Retention or removal of hardware?

There is surprisingly scant literature to help to guide the decision regarding the retention or removal of hardware in the presence of an acute postoperative infection following internal fixation of a fracture. Only a few studies have addressed this topic (6), but even those have not specifically examined the outcomes for patients in whom an infection has developed in an acute postoperative (<6 weeks) setting - when the

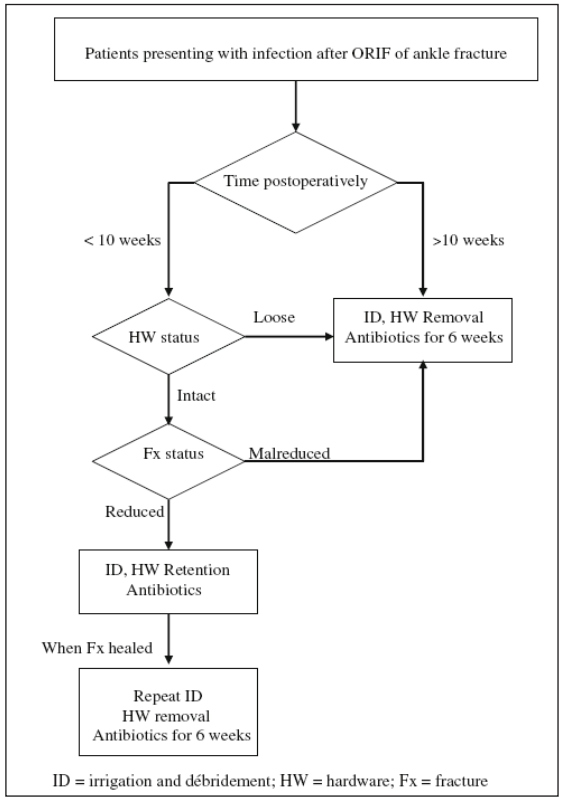

Figure 1. Treatment protocol for infections after ORIF of ankle fractures.

decision to retain or remove hardware prior to fracture union is most critical. Rightmire et al (7) retrospectively identified 69 patients with acute and late postoperative (<16 weeks) infection and their findings included a success rate of 68% of osseous union with original hardware in place.

The stability provided by the implants has been proved to help to reduce the incidence of infection after internal fixation, and to aid in the clearance of established infections, thus making the maintenance of fixation a top priority (8,9). In an experimental study Worlock et al fixed diaphyseal fractures of tibia with a stable compression plate or with an unstable endomedular pin, and inoculated Staphylococcus aureus in the fracture zone. They found out that in the unstable group there were two times more infections than in the stable compression plate group (71% vs 35%) (10).

Berkes et al (5) retrospectively analyzed 123 postoperative wound infections (23 femur, 63 tibia/fibula/ankle/foot, 22 pelvis, 15 upper extremity) that had developed within six weeks after internal fixation of a fracture. Their purpose was to determine the prevalence of osseous union with maintenance of hardware. 87 patients (71%) had fracture union with operative debridement, retention of hardware, and culture-specific antibiotic treatment. The number of infections treated successfully with retention of hardware was 56.5% (13/23) in femur, 68.3% (43/63) in tibia/fibula/ankle/foot, 80% (12/15) in upper extremity, and 86.4% (9/22) in pelvis. Predictors of treatment failure were open fracture and the presence of an intramedullary nail ($p<0.05$). Smoking, infection with Pseudomonas species, and involvement of the femur, tibia, ankle, or foot trended toward an association with failure. They concluded that deep infection after internal fixation of a fracture can be treated with operative debridement, antibiotic suppression, and retention of hardware until fracture union occurs. Results may be improved by patient selection based on certain risk factors and the specific bacteria and implants involved.

Zalavras et al (11) presented a treatment protocol for infections after ORIF of ankle fractures (Figure 1). The protocol is based on the time post surgery and the stability provided by the implant. They reviewed retrospectively 26 patients with infections after ORIF of ankle fracture. Patients presenting up to 10 weeks postoperatively were treated by debridement and either hardware retention (if implants were judged stable) or hardware removal (if implants were loose). All patients presenting more than 10 weeks postoperatively underwent debridement and hardware removal, with an exception of one patient who underwent below knee amputation. Staphylococcus aureus was identified if 17 patients (65%) and was oxacillin-resistant in six (23%). The infection recurred in 5 of 18 patients (28%) who were followed up to 18 months. Three recurrent infections were controlled with repeat debridement. The remaining two patients underwent below knee amputation, resulting in amputation in 3 of 18 patients (17%). Two of three amputation patients had diabetes mellitus and, overall, two of five patients (40%) with diabetes underwent a below-knee amputation.

As there are no evidence-based guidelines to dictate whether hardware should be removed or retained in the presence of an acute infection, the management requires flexibility in the treatment plan while trying to avoid a poor outcome such as infected non-union

or a below-knee amputation. According to the available literature, it is recommendable to maintain the hardware in place until osseous union has occurred.

Future

The immediate future of treating infection in fracture fixation is likely to involve local delivery with biodegradable coated implants that have a sustained release of inhibitory concentrations of antibiotics or antiseptics. This approach produces higher local concentrations that are currently possible with systemic delivery, without the toxic side effects, and without the need for hospitalization during intravenous therapy. Having a biodegradable coating avoids the need to remove the local delivery system. Local delivery also allows delivery directly at the implant surface, as well as to the non-vascularized tissue within the healing fracture that cannot be achieved with systemic delivery (2). The long-term future for treating infections in fracture fixation is unlikely to involve antibiotics since in the long-term bacteria will be multi-resistant. If antibiotics are used, then it will probably be in combination with alternative technologies due to the fast build up of resistant strains of Staphylococcus aureus and Staphylococcus epidermidis (2).

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