COMMENTARY

Invited Commentary: The Incidence of Deep Infection Following Lower Leg Circular Frame with Minimum of 1-year Follow-up from Frame Removal

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The authors are to be congratulated on an important addition to the literature. In particular, for the identification of the increased risk of deep infection for periarticular (8.33%) fractures as compared to diaphyseal (1.28%). Unlike the well-documented difference in frequency of pin-site problems, the disparity in deep infection rates has received little attention in the literature. My personal experience reflects that of the authors and supports their legitimate observation.

It is critical to understand the possible causes so that strategies to minimise risk can develop. The authors have noted several potential factors. First is the higher incidence of pin-site infections around periarticular vs diaphyseal wires. These more frequent pin infections would, reasonably, pose a greater risk of deep infection. Whilst all pins can allow for adherence and migration of bacteria along their path, the progression to the status of pin infection and even loosening is typically local to the pin-site unless other factors amplify the problem.

The authors report that the presence of internal fixation and bone void fillers can act as such potentiating factors. Hardware and bone fillers in proximity to contaminated wires can allow for deep bacterial infection and biofilm development. The presence of bone fillers seems especially problematic, but the strength of the association could not be inferred as the total case numbers were not declared. In contrast, the footprint of screws used for joint reconstruction is typically quite small and the area of surrounding bone in which vascularity is compromised by the fixation very limited. In addition, 96 cases with hardware did not develop infection indicating that although hardware may contribute to an increased risk, it alone is not causative.

I believe the major risk factor not mentioned is that of avascular bone fragments which are often present in both open and closed comminuted periarticular fractures. These fragments are revascularized in the healing process and incorporated into the healing mass of callus. However, periarticular wires can provide a pathway for bacteria to access these vulnerable fragments prior to revascularization thereby leading to deep infection. The previous authors have not focused on this which is, I believe, a key concern.

A strategy to potentially limit infection risk in comminuted periarticular fractures is to delay introduction of periarticular wires until revascularization of the comminuted joint pieces occurs and there is sufficient healing of the articular block to allow for removal of the internal fixation. This typically occurs at around 8 weeks after fracture. Therefore, the strategy is anatomic joint reconstruction using small screw fixation combined with an initial fixator that spans the joint without periarticular wires. A staged removal of the screws, placement of periarticular wires, and removal of the joint spanning ring to then occur at approximately 8 weeks. The fixator is removed later as is usual after fracture healing. This strategy is my practice for over 5 years and exclusively for comminuted periarticular fractures. It has been effective. This is anecdotal but serves to introduce the concept for further investigation and validation as a method to address the concerns raised by the authors.

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