

Editorial

Fracture nonunions – We can do better

Saqib Rehman, MD MBA^{1a}

¹ Department of Orthopaedic Surgery & Sports Medicine, Lewis Katz School of Medicine at Temple University, Philadelphia, USA

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No Abstract



Dr. Saqib Rehman, MD MBA is the Vice Chair and Professor of Orthopaedic Surgery & Sports Medicine Service, and Director of Orthopaedic Trauma at Lewis Katz School of Medicine at Temple University, Philadelphia, PA, USA

“The impact of tibia shaft fracture nonunion on physical health was comparable with the reported impact of end-stage hip arthrosis and worse than that of congestive heart failure.”¹ I’m not sure we have made substantial progress in the last ten years since Dr. Brinker reported on the devastating effects of tibial nonunion on health-related quality of life back in 2013. How can we do better? I believe it comes down to a better understanding of why the normal healing cascade becomes compromised, earlier detection of fractures that will ultimately go on to nonunion, and better ways to prevent known causes of nonunion, such as infection.²

Fracture nonunions occur due to inappropriate mechanical instability, insufficient biological requirements for healing, or a combination of both. Whereas we understand how to address classic hypertrophic nonunions through im-

proved mechanical stability, we don’t fully understand how to optimize micromotion and macro motion when fixing fractures or how to best translate Perren’s strain theory into practice.³ Through the widespread adoption of AO principles and improvements in implant design, we have been able to provide better stability with more biologically friendly techniques. But we are still trying to figure out the optimal construct for distal femur fractures – are locking plates too rigid and need far-cortical locking? Or should we be doing more nails that allow more motion? And if fixation has been too rigid, then how does doing a nail plus a plate (the latest fad) make things less rigid? Or does that not matter anymore?

When it comes to the biological etiologies of fracture nonunion, agents such as bone morphogenetic protein -2 (BMP-2) held much promise, but 20 years have passed, and we still do not have convincing evidence that these, stem cells, or other related interventions can consistently prevent or treat nonunions. That said, more basic science and clinical research is needed to understand how we can replicate success in rats and mice to success in human populations. There is promise here, but we need better trials to get evidence on what really works.

Earlier recognition of fractures at risk for nonunion through biomarkers and improved imaging (such as better detection of implant or fracture motion) are possibly on the horizon. This could at least allow us to intervene earlier more confidently. Infection prevention continues to be challenging, but we continue to learn from newer and better-designed clinical trials. The only thing worse than a nonunion is an infected nonunion, and I’m hopeful that by better prevention and detection of infection, we can make real progress for our patients.

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^a Corresponding author:

Saqib Rehman, MD MBA

Department of Orthopaedic Surgery & Sports Medicine, Lewis Katz School of Medicine at Temple University, Philadelphia, USA

srehmanmd@yahoo.com



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