Standard Review Article

Geriatric trauma: there is more to it than just the implant!

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Abstract Geriatric trauma continues to rise, corresponding with the continuing growth of the older population. These fractures continue to expand, demonstrated by the incidence of hip fractures having grown to 1.5 million adults worldwide per year. This patient population and their associated fracture patterns present unique challenges to the surgeon, as well as having a profound economic impact on the health care system. Pharmacologic treatment has focused on prevention, with aging adults having impaired fracture healing in addition to diminished bone mineral density. Intraoperatively, novel ideas to assess fracture reduction to facilitate decreased fracture collapse have recently been explored. Postoperatively, pharmacologic avenues have focused on future fracture prevention, while shared care models between geriatrics and orthopaedics have shown promise regarding decreasing mortality and length of stay. As geriatric trauma continues to grow, it is imperative that we look to optimize all phases of care, from preoperative to postoperative.

Keywords: geriatric trauma, fracture healing

1. Preoperative: Is Fracture Healing Different in the Elderly, and How Can It Be Optimized?

The older population in the United States has been steadily increasing, with those aged 65 years and older expected to comprise 17% of the population by 2030.[1] While the population of geriatric patients continues to grow, so too does the economic impact of osteoporosis and their corresponding fragility fractures. Conditions such as osteoporosis increase with age as does a decline in healing potential, which may result in increased rates of delayed healing or nonunion.[2,3]

Fracture healing proceeds through multiple phases characterized by anabolic and catabolic phases.[4] The healing potential is markedly affected by aging. Numerous studies have demonstrated delayed fracture healing in older animals, with associated reduction in cartilage and bone formation, delayed cartilage resorption, and slower mineralization within the callus.[5–7] In the study by Lopas et al,[5] osteochondral stem cell proliferation was diminished and associated with a significant decrease in bone and cartilage content with the fracture callus of aged mice compared with young.

Aging additionally affects chondrocytes and osteoblasts located in the periosteum during fracture healing.[8–10] Senescence and greater oxidative damage were associated with periosteal-derived progenitor cells from old humans compared with young.[11] It has been shown in mice that chondrogenic potential of stem cells in the periosteum is decreased in addition to chondrogenic differentiation from periosteal cells being delayed.[9,12] Furthermore, the expression of type II collagen and delayed cartilage matrix deposition at early time points of fracture healing is delayed in older animals.[12] Similarly, osteoblast differentiation and osteocalcin expression is delayed from periosteal cells at the fracture site in older mice.[12]

Endochondral ossification is also affected by age. In the study by Lu et al,[12] characteristic histological findings that mark endochondral ossification, hypertrophic chondrocytes, type X collagen expression, and vascular invasion were all delayed in older mice compared with young. In addition, the conversion of cartilage to bone within callus was also found to be delayed in older mice.

Age-related changes diffusely influence the stages of fracture healing. However, these changes do not entirely encompass the poorer healing outcomes and increased morbidity reported in older patients. Pharmacological therapy has currently been

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targeted at reducing the risk of fractures, rather than influencing the various stages of fracture healing. Medications to treat osteoporosis have been categorized as antiresorptive or anabolic.

Antiresorptive medications have included bisphosphonates, hormonal therapies, and denosumab. Bisphosphonates bind to the mineral matrix of the bone and inhibit osteoclast resorption of the bone, leading to a decrease in bone turnover and a net gain in bone mass.[13–15] They are available in multiple formulations, with some iterations available as IV injections. Bisphosphonates are excreted by the kidneys; thus, toxicities can occur in patients with renal impairment.[15] Complications include the rare incidence of osteonecrosis of the jaw, in addition to the relatively uncommon but difficult to treat complication of low-trauma atypical femur fractures.[15,16]

Denosumab is an additional agent, whose mechanism involves inhibiting RANKL to decrease bone resorption. It is a first-line therapy for patients at high risk of fracture who are unable to use oral therapy.[17] In the FREEDOM trial, 7,868 postmenopausal women were evaluated, and the results demonstrated that 60 mg of denosumab every 5 months for 36 months significantly reduced the risk of hip, nonvertebral, and vertebral fractures in comparison with placebo.[18] Adverse reactions have included hypersensitivity, serious infections, dermatological reactions, hypercholesterolemia, and hypocalcemia. Rare cases of osteonecrosis of the jaw and atypical femur fractures have also been reported.[17]

Hormonal therapies have included selective estrogen receptor modulators, estrogen–progestin therapy, testosterone therapy, and calcitonin. Raloxifene, characterized as an estrogen agonist/antagonist, has been recommended as a first-line therapy for patients requiring a reduced risk of spine fractures only. It has also been considered in women at increased risk for vertebral fractures in addition to developing breast cancer, as it has selective antagonistic effects on breast tissue.[15] Estrogen–progestin therapy was found in a randomized controlled trial of 16,608 postmenopausal women to significantly reduce fractures, though with increased risks of cardiovascular events, stroke, venous thromboembolism, and invasive breast cancer.[19] Calcitonin has been shown to reduce the risk of vertebral fractures by 33% but lacks data showing its effects on nonvertebral fractures.[15]

Teriparatide falls in the calls of parathyroid hormone analogs and is the first anabolic treatment approved for osteoporosis.[20] In the study by Saag et al,[21] teriparatide increased bone mineral density in the spine and hip resulting in significantly fewer vertebral fractures in comparison with alendronate but had no significant difference in the incidence of nonvertebral fractures. Abaloparatide is the second recombinant human parathryoid hormone to reach the market and has been found to reduce the incidence of new vertebral fractures by 86% over an 18-month period and reduce the risk of nonvertebral fractures by 43%.[22]

Still, emerging therapies and investigational drugs are on the horizon. As the population continues to age, fragility fractures remain a large economic and medical burden on health care systems. Older patients present particular challenges to fracture healing, both innately in their diminished capacity to heal as well as challenges with fixation due to low bone mineral density.

2. Intraoperative: Are We Getting This Right? How to Assess Reduction

Assessing radiographic reduction and implant placement is integral to the prevention of deformity. Intraoperative fluoroscopy is universally used to assess fracture reduction and fixation. However, systematic and structured education on the use of intraoperative fluoroscopy is lacking, as reflected in a survey of 98 surgeons where only 27% of trainees received fluoroscopic training.[23]

To evaluate fluoroscopic views during fracture fixation, it is imperative to understand proximal femoral relationships in an intact femur and how they vary depending on the location of the fluoroscopic beam, the leg position, and normal anatomical variance. An anteroposterior (AP) image of an uninjured proximal femur typically demonstrates a standard relationship with the tip of the trochanter in line with the center of the femoral head and a smooth unbroken curve between the inferior aspect of the femoral neck and the proximal medial femoral shaft. The femoral neck–shaft angle is typically 129.7 ± 6.2 degrees, and there is often no overlap between the greater trochanter and femoral neck.[24] As the proximal femur transitions from an internally rotated position to more external rotation, the femoral neck–shaft angle (NSA) increases and the distance between the trochanter and femoral head decreases. Similarly, hip flexion and extension can significantly alter the fluoroscopic appearance of the neck–shaft angle. In the study by Bhasyam et al,[25] hip flexion–extension and rotation had a synergistic effect on the measurement of the NSA, and measurement error was minimized when hip flexion–extension was within 10 degrees of neutral. This becomes particularly important when patients are positioned lateral, as the affected hip tends to drift in to flexion and internal rotation, which can give the illusion of increased varus.

Two separate lateral radiographs are essential to assess proper implant placement and reduction quality. An appropriate lateral radiograph for implant placement should have collinearity between the femoral head, femoral neck, and femoral shaft.[23] This is not to be confused with the more externally rotated view of the proximal femur used to assess reduction of the anteromedial cortical buttress, which has been shown to correlate with the amount of fracture collapse.[26] When using a fracture table, the C-arm is brought in from the contralateral side, and the proximal femur imaged orthogonal to the femoral neck. When lateral, to obtain a similar view, the C-arm must be tilted with the image intensifier toward the head.

Reducing pertrochanteric fractures remains challenging, even in less complex intertrochanteric fractures. For closed reduction of intertrochanteric fractures, patients are commonly placed supine on a fracture table with the affected limb placed in traction and internally rotated ~15 degrees to obtain a reduction. In the study by Ramanoudjame et al,[27] internal rotation of the distal segment greater than 15 degrees was present in 40% of patients, which is comparable with malrotation found after femoral shaft intramedullary fixation. Notably, this rate of malrotation was present with placing the foot in internal rotation sufficiently to center the patella. The use of the contralateral uninjured limb as a template can be helpful, with a centered patella and ipsilateral proximal femur view serving as a reproducible template. The effects of malrotation, especially in the geriatric population, remain difficult to assess. In the study by Johnson et al, external rotation malunions are more poorly tolerated than internal rotation deformities.[28]

Critical assessment of radiographic reduction requires evaluation of the anteromedial cortical buttress. As the calcar femorale is often disrupted and comminuted in pertrochanteric fractures, the anteromedial cortical buttress provides a final structure restraint to fracture fragment telescoping. On the AP view, an anatomic or positive anteromedial cortical buttress leads to reduced controlled collapse of the fracture. Perhaps most importantly, on the lateral view, the anteromedial cortical buttress should be anatomic or remain extramedullary, while a lateral intramedullary reduction leads to increased collapse of the fracture, regardless of a positive or anatomic reduction on the AP. An anatomic or extramedullary reduction, on the lateral, can be difficult to obtain, possibly due to the restriction imparted by the strong iliofemoral ligament, which can prevent anterior displacement of the femoral neck. A jocher elevator can be used to cantilever the fracture out of an intussuscepted position, a technique described by Carr et al.[28] An additional technique that can be used is placement of a Schanz pin from anterior to posterior along the medial and inferior neck. This can allow for elevating the anteromedial cortical buttress in to an anatomic and/or positive reduction.

3. Postoperative: Preventative Management to Reduce Subsequent Fractures

About 1 in 2 women and 1 in 4 men will experience and osteoporotic fracture at some point in their lifetime.[29] Our goal as orthopaedic surgeons was to recognize the patients with low bone density (osteopenia) and osteoporosis after presenting with an initial fragility fracture (the sentinel event) and get involved in the preventative management to reduce subsequent fractures. Sadly, despite the awareness of fragility fractures and the availability of effective treatments to reduce subsequent fracture risk, only 9%–23% of patients with osteoporotic-related fractures receive adequate workup or pharmacologic treatment.[30,31] This defines the treatment gap which has changed little over the past 20 years.

Treatment works! There are many examples of fracture liaison programs, both local and national designed to address this problem. The American Orthopaedic Association's (AOA) Own the Bone (OTB) has helped start programs across the county since 2008 to address the sentinel event. Kaiser Permanente demonstrated enormous cost savings with the implementation of a bone health program, reducing subsequent fragility fractures 3–7-fold while saving their system roughly 50 million dollars over 5 years.[32] There are 6 measures: nutrition counseling, physical activity counseling, lifestyle counseling, pharmacology, testing, and communication, all consistent with recommendations from OTB, the National Osteoporosis Foundation (NOF), the Centers for Medicare and Medicaid Services, the Joint Commission, the World Health Organization, and the AMA.

Nutrition recommendations vary but should include calcium and vitamin D, realizing that less than 50%–80% of the adult population meet the requirements. Recommendations from the NOF include 1000–1200 mg/day of calcium, incorporating dietary supplements when the patients' diet is insufficient.[33] Similarly, a total of 800–1000 IU/day of vitamin D is recommended through diet and supplements as needed.[33] Diets in the older population are often poor in overall calorie intake suggesting protein supplementation may be indicated, especially in the postoperative period.

Physical activity should focus on weight-bearing and muscle strengthening exercises to improve bone strength/density, balance, posture, and endurance. Falls are a significant problem and balance training exercises most importantly will help to reduce falls leading to fewer fractures. Equally important in reducing falls is counseling assessing risk factors that may increase the likelihood for falls. This may also include evaluation of medications, home safety evaluations (such as throw rugs, stairs, lighting, vision evaluation), and more controversial correction of vitamin D deficiency.[34]

Lifestyle modification falls into 2 categories. One is considered modifiable and includes avoiding excessive alcohol intake, ensuring adequate diet, remaining physically active, and avoiding tobacco products/smoking. The second category is not truly modifiable and centers on medications, cognition (resulting in increased falls), and health disorders/diseases that cause or contribute to poor bone and fractures (Table 1).[35] The chronic use of steroids and heparin is worth mentioning because these tend to be medications we see and use as orthopaedic surgeons.

Dual-energy X-ray absorptiometry (DEXA) is the most commonly used test to evaluate bone density and should be performed in both men and women older than 50 years that sustained a fragility fracture. The DEXA give a T score which compares your bone mass to that of a healthy young adult. The T score represents the number of standard deviations above or below the average bone density for a young healthy adult. A T score below −2.5 is considered one definition of osteoporosis, and a score between −1.5 and −2.5 is considered osteopenia. Other tests less commonly used and more controversial but still valid include quantitative computer tomography (qCT), quantitative ultrasound (qUS), and micro CT (more helpful to look at the porosity and microstructure of the bone relating to the strength).[36] Laboratory testing also plays a role to help rule out secondary causes of osteoporosis but is beyond the scope of this article.

Communication with your patient and within your medical system (primary care specialists or advanced practice providers) is key to the successful treatment of their bone health and prevention of future fragility fractures, and development of a fracture liaison service within your community is a must. As orthopaedic surgeons, we can be the champions in our community to develop these programs to address this treatment gap. Our role, at a minimum, should include starting patients on calcium and vitamin D, arranging for a DEXA scan when indicated, and provide a consult to either a fracture liaison service or internist/advanced practice provider/specialist to manage bone health. Addressing the underlying problem will not only prevent further fractures, but save lives.

4. Multidisciplinary Orthogeriatric Care

Hip fractures are devastating injuries to older patients, with worldwide rates expected to increase to approximately 21 million per year by 2050.[37] With 1-year mortality rates estimated to range from 20% to 30%, outcomes remain poor.[38,39] Integrated care and collaboration among the geriatrics and orthopaedics services has been proposed as a means to improve patient care. Various models have been proposed, including routine geriatric consultation, establishing a geriatric ward for hip fractures, and most recently, a shared care model. In a systemic review and meta-analysis by Grigoryan et al,[40] orthogeriatric collaboration was associated with a significant reduction of in-hospital mortality and long-term mortality, with length of stay particularly reduced in the shared care model.

Geriatrician involvement in the care of hip fractures is not a novel pathway. In Great Britain in the 1960s, geriatric consultations were introduced to improve care for the elderly with hip fractures.[41] Over the ensuing years, surgeons and geriatricians have increased their collaboration in the pursuit of optimal patient care, resulting in a variety of treatment models of care. In the study by Folbert et al,[42] a shared care model was used

TABLE 1

Conditions, Diseases, and Medications Related to Osteoporosis and Fractures

Lifestyle factors		
Alcohol abuse	Excessive thinness	Excess vitamin A
Frequent falling	High salt intake	Immobilization
Inadequate physical activity	Low calcium intake	Smoking (active or passive)
Vitamin D insufficiency		
Genetic diseases		
Cystic fibrosis	Ehlers–Danlos	Gaucher's disease
Glycogen storage diseases	Hemochromatosis	Homocystinuria
Hypophosphatasia	Marfan syndrome	Menkes steely hair syndrome
Osteogenesis imperfecta	Parental history of hip fracture	Porphyria
Riley–Day syndrome		
Hypogonadal states		
Androgen insensitivity	Anorexia nervosa	Athletic amenorrhea
Hyperprolactinemia	Panhypopituitarism	Premature menopause (<40 y)
Turner and Klinefelter syndromes		
Endocrine disorders		
Central obesity	Cushing syndrome	Diabetes mellitus (types 1 and 2)
Hyperparathyroidism	Thyrotoxicosis	
Gastrointestinal disorders		
Inflammatory bowel disease	Malabsorption	Pancreatic disease
Primary biliary cirrhosis		
Hematologic disorders		
Hemophilia	Leukemia and lymphomas	Monoclonal gammopathies
Multiple myeloma	Sickle cell disease	Systemic mastocytosis
Thalassemia		
Rheumatologic and autoimmune diseases		
Ankylosing spondylitis	Other rheumatic and autoimmune diseases	
Rheumatoid arthritis	Systemic lupus	
Neurological and musculoskeletal risk factors		
Epilepsy	Multiple sclerosis	Muscular dystrophy
Parkinson disease	Spinal cord injury	Stroke
Miscellaneous conditions and diseases		
AIDS/HIV	Amyloidosis	Chronic metabolic acidosis
Chronic obstructive lung disease	Congestive heart failure	Depression
End-stage renal disease	Hypercalciuria	Idiopathic scoliosis
Posttransplant bone disease	Sarcoidosis	Weight loss
Medications		
Aluminum (in antacids)	Anticoagulants (heparin)	Anticonvulsants
Aromatase inhibitors	Barbiturates	Cancer chemotherapeutic drugs
Depo-medroxyprogesterone (premenopausal contraception)	Glucocorticoids (≥5 mg/day prednisone or equivalent for ≥3 mo)	GnRH (gonadotropin-releasing hormone) agonists
Lithium cyclosporine A and tacrolimus	Methotrexate	Parental nutrition
Proton pump inhibitors	Selective serotonin reuptake inhibitors	Thyroid hormones (in excess)
Tamoxifen® (premenopausal use)	Thiazolidinediones (such as Actos® and Avandia®)	

From: The Surgeon General's Report,[7] modified.

and was found to significantly decrease 1-year mortality in frail older patients to 23.2% in comparison with the 35.1% mortality rate in historical control patients treated with standard care. Risk factors for 1-year mortality included increasing age, male gender, malnutrition, physical limitations, increasing Barthel Index, and medical conditions. Similarly, in the study by Gosch et al,[43] orthogeriatric comanagement significantly decreased 1-year mortality rates, with older age and lower Parker Mobility Scale as predictive factors. With increasing literature demonstrating the benefits of geriatric and orthopaedic collaboration, it is evident that shared care models can have a profound positive impact on this relatively fragile patient population.

What has additionally emerged with increased scrutiny on this patient population is the unfavorable profile of older patients with hip fractures, who appear to be more frail than older patients without a hip fracture.[44–46] A number of measuring instruments have been evaluated for the prognostic scoring of older patients with hip fractures who are at risk of adverse outcomes.[46] Indeed, perioperative risk prediction may represent an essential element in clinical practice dictating clinical decision making. Perhaps the most optimal way to categorize these patients is through the emerging geriatric concept of frailty. This has been defined as a syndrome with multiple reduced physiologic functions that increases an individual's vulnerability for developing increased dependency and/or death.[47] Two main models currently characterize how frailty develops and manifests. In the "phenotype" model by Fried et al,[48] frailty manifests as decline in lean body mass, strength, endurance, balance, walking performance, and low activity. Patients with 3 or more of the 5 features of slowness, weakness, exhaustion, weight loss, and physical activity are deemed frail, while patients with 1 or 2 of the 5 features are classified as "prefrail." The second model, referred to as the Frailty Index (FI) by Rockwood et al,[49] conceptualizes aging as the accumulation of deficits and views frailty as a multidimensional risk state quantified by the number of deficits rather than by the nature of the health problems. FI is calculated by counting the number of deficits presents in an individual divided by the total number of deficits measured. The FI score ranges between 0 and 1, with higher scores indicating greater degree of frailty. In the systematic review by Lin et al,[47] there was strong evidence that frailty in older-old and oldest-old surgical patients predicted postoperative mortality, complications, and prolonged length of stay.

The optimal management of frail patients with fragility fractures includes both orthogeriatric care and proper assessment of their frailty. The implementation of shared care models has been to shown to decrease 30-day and 1-year mortality rates, while also helping to achieve optimal recovery through perioperative management based on best practice evidence.[47] Frailty and bone fragility often coexist in the same patient, and optimal management must include a multidisciplinary team with the ability to address and manage frailty, fall prevention, and bone health from admission through to postacute phase care.

5. Atypical Femur Fractures: How to Optimize Treatment

Atypical femur fractures (AFF) account for 3% of all femur fractures and 2% of patients treated with bisphosphonates.[50] Despite being rare, it is paramount that treating surgeons recognize patient and radiographic characteristic that can be associated with the diagnosis. Women of Asian descent have been noted to have a higher likelihood of having an AFF when compared with men and other ethnicities.[51] A majority of these fractures occur in the setting of a low-energy fall and prodromal anterior thigh, or groin pain occurs in 34%–70% of the patients before fracture identification.[52,53] Furthermore, bisphonate use for greater than 3 years has been linked to a diagnosis of AFF.[53]

Successful surgical management of AFF can be challenging. The goal of surgical management is to restore length, alignment, and rotation. While the radiographic features of these fractures are misleadingly simple, routine techniques for nailing may not be suffice and intraoperative complications are not infrequent. Since these are pathologic fractures, additional steps are routinely required to achieve not only reduction but union. These steps include formal open reduction, fracture bed preparation, interfragmentary compression, and possible use of bone graft or biologic augmentation.[50]

5.2. Surgical Strategy

The first step to determine the optimal surgical strategy is to scrutinize the radiographs. The overall coronal and sagittal plane alignment of the contralateral limb should be assessed to determine whether the patient has an excessive femoral bow on either plane. Canal diameter should be measure at the narrowest point and the location and the size of the sclerotic pedestal should be noted.

If the patient does not have significant bow in either plane, a small pedestal, and does not have a small canal, then standard nailing techniques can be used. Preference for a cephalomedullary nail or a reconstruction nail with fixation across the femoral neck is preferred to protect this region. In cases of a narrow canal, small diameter adolescent nails should be considered. However, the surgeon needs to recognize that many of these fractures require a prolonged (6 months or greater) healing time and smaller nails may fatigue prior to healing.[54] This is especially the case in which adequate intrinsic stability was not achieved with near an anatomic reduction during surgery.

Depending on the size and density of focal intramedullary sclerosis on the lateral cortex, the surgeon may have to alter the approach. Careful attention should be placed on the path of the reamers as they are being passed by the sclerosis. If the reamers are being deflected medially, then preferential reaming of the medial cortex will occur. This will result in a translational deformity once the nail is placed. This preferential reaming can potentially be minimized by holding the fracture anatomically reduced during reaming. Unicortical 5-mm Shanz pins can be placed in the lateral cortex or posterior transcortical pins, both proximal and distal to the fracture. The pins can be used to control the distal and proximal segment, in an attempt to preferentially ream the sclerotic region. Alternative percutaneous techniques can also be used such as using bone hooks or spike pushers. If these techniques do not work and preferential reaming is occurring medially, then the area of sclerotic bone should be directly addressed. To address the pedestal, it is recommended to start with minimal invasive technique and work to an open approach, if that is what is required.

If the pedestal is proximal to the isthmus and the patient does not have a significant bow, then a stiff straight reamer or sharp awl can be used to remove the pedestal from the canal portion of the lateral aspect of the femur (Fig. 1A).[50] Of note, it is important to not start and stop the reamer in the region of the sclerotic pedestal because this may result in an iatrogenic fracture and comminution. Furthermore, it is prudent to ream at least 2 mm over the diameter of the selected nail in the region of the pedestal. If the location of the pedestal and/or femoral geometry prevents safe access to the pedestal, then an open approach may be required. This can be done with a formal subvastus lateral approach to gain access to the fracture and pedestal. Once the pedestal is identified, it can be removed with a small diameter burr or alternatively a short rigid reamer (Fig. 1B). Then, the fracture is reduced, reaming is completed, and the nail placed. If the surgeon notes significant resistance during nail placement and the surgeon has overreamed by 2 mm, then alternative options need to be assessed to avoid iatrogenic fractures or distracting across the fracture site. One potential reason for difficulty passing the nail could be anterolateral bowing of the femur.

In cases in which the patient has native anterolateral bowing of the femur, a number of different techniques can be used to help with nail placement. The surgeon can medialize the start site, convert to a smaller nail, externally rotate the nail, and perform an osteotomy. In many cases, externally rotating a smaller nail will help allow for easier passage of the nail due to the fact that the nails inherent bow in the sagittal plane can be used in the coronal plane to better match the patients coronal plane anatomy (Fig. 1C). In these situations, the addition of a provisional plate during nail placement can help maintain reduction and these unicortical plates can be left on to assist with additional stability. Although this is not routinely recommended, especially in the diaphysis, it can be used when additional fixation may be required in the setting of slow fracture healing and an undersized nail. A recent study demonstrated a 95% union rate in patients who had incomplete AFF in the setting of increased anterolateral bowing, using a simple corrective osteotomy and intramedullary nail placement with augmented plate fixation.[55] In the setting when the femoral canal is too narrow for nail placement, then plating osteosynthesis can be used.

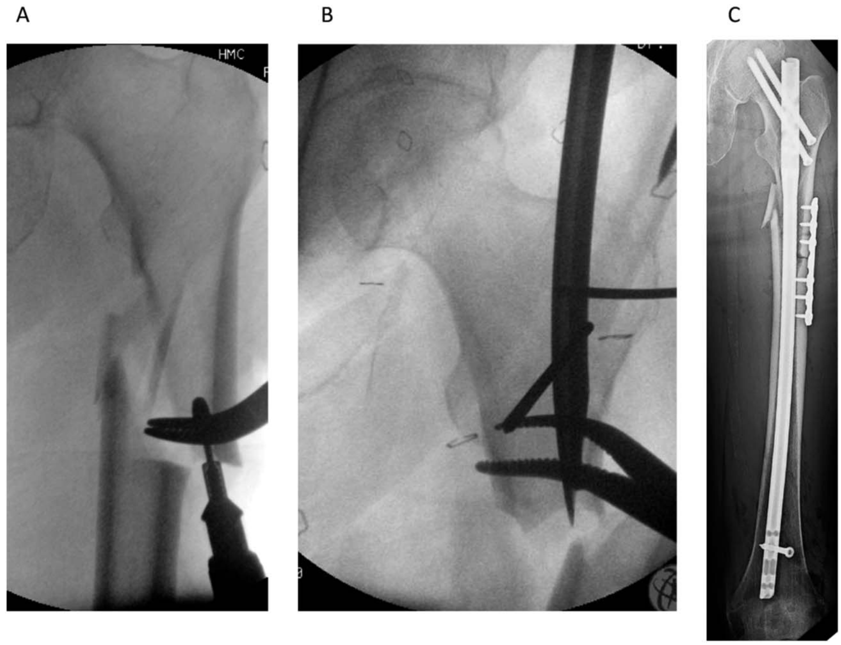

Figure 1. A, Intraoperative image of a revision case, demonstrating the use of a sharp awl to remove the pedestal through the nail start site. B, Intraoperative image demonstrating the use of a burr to remove the pedestal, after an open subvastus lateral approach had been performed. C, Postoperative AP film of a patient who had a narrow canal and increased anterolateral bow. An open approach was performed to remove the pedestal and anatomically reduce the fracture and hold the fracture reduced with a unicortical plate. A reconstruction nail was then introduced and externally rotated while advancing to allow a better fit between the nail and the patients anterolateral bow.

If plate osteosynthesis is going to be used, then interfragmentary compression should be optimized to compress the tensile lateral cortex. Depending on the location of the fracture, then several different options exist for plating. Proximal femoral locking plates and blade plates can be used in the subtrochanteric region. For more distal locations, large fragment-based plates can be used that are designed either for proximal femur fractures or supracondylar distal femur fractures, depending on the location of the fracture. While plating allows for interfragmentary compression and anatomic reduction, weight-bearing should be restricted for a longer period compared with nailing.

No substantial evidence is available to support the use of biologic augmentation for treating AFF. However, evidence supports the cessation of bisphosphate therapy and increasing calcium and vitamin D intake.[56–58] Furthermore, what is clear is that one of the main risk factors for nonunion is a fracture that was fixed in varus. To summarize, to optimize management of AFF the surgeon needs a clear surgical strategy that addresses the unique characteristics of this injury pattern.

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