Management of Acute Diabetic Ankle Fractures

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Ankle fractures are common skeletal injuries and are one of the most managed joint injuries in orthopedic surgery. Surgical fixation is well-established as the treatment of choice for displaced fractures with an anatomic reduction of the mortise decreasing instability and lessening the development of post-traumatic arthrosis. Even in the diabetic patient, acute, displaced fractures are routinely managed with surgical intervention [1]. Although the use of nonoperative care for some fractures have demonstrated good outcomes, non-surgical treatment is usually reserved for patients presenting with non-displaced fractures, those whose medical co-morbidities that preclude any surgical intervention, patients who refuse surgery or most often as an intermediate step to allow the soft tissue envelope to improve. This then allow surgeons to proceed with surgical intervention of the ankle. After an anatomic reduction and stable fixation, and excluding any skin or wound complications, the postoperative care of the nondiabetic often results in reproducible and good outcomes.

So why then is there a different concern in managing the diabetic patient? One large problem is that because ankle fractures are so routinely treated, there is often a certain disregard for the seriousness and potential complications that can occur in the diabetic population. Due to their hyperglycemia, the preoperative diabetic can already have impaired healing of the wound and bone, a decrease in their immune response, vascular insufficiency, terrible skin problems, and peripheral neuropathy. Although these are all significant factors, it is their peripheral neuropathy, of sensory, motor and autonomic dysfunction, which may result in patients failing to recognize that they have sustained an injury and have them seek medical attention in a timely manner. This may result in a late presentation of the fracture, a deformity and contracture of the ankle, and potentially ulceration and compromise of the soft tissue envelope, all of which may lead to the development of Charcot arthropathy, even after undergoing surgery [1, 2].

How then, do we manage the acute diabetic ankle fracture? Do we withhold certain treatments because they will be too expensive? Or do we withhold treatments, due to expectations that they will have poorer outcomes than the nondiabetic patient? This comes with the understanding that withholding treatment can produce avoidable complications, result in significant disabilities, and create chronic conditions that can lead to socio-economic burdens to the patient, their families, and to payer systems. Given the advancements in techniques, implants, and the

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management of diabetes, this chapter will hopefully provide a rational approach for physicians tasked with managing acute ankle fractures in the diabetic patient.

1   Epidemiology

Current estimates report that there are more than 34 million people in the United States diagnosed with diabetes mellitus, with greater than eight million who are undiagnosed [3–5]. This represents about 13% of adults, affects more than 25% of people greater than 65 years of age and is the seventh leading cause of death in the United States [4, 5]. Worldwide diabetes has increased from 108 million in 1980 to 463 million in 2019 [6–8]. This equates to one of every 11 people and current projections indicate that this number will exceed 700 million people (1 in 10) by the year 2045 [6–8]. In addition, two studies have reported that the lifetime risk of developing diabetes in the United States [9] and India [10] for males was 32.5% (55.5% India) and 38.5% for females (64.6% India), with the results of both studies reporting that the diagnosis of diabetes produced a dramatic decrease in life-years for both males and females.

Although complications are often related to poor glucose control, hypertension, and dyslipidemia, only 36% to 57% of patients achieve adequate glycemic or blood pressure levels, while only 13.2% of all patients achieve all three target levels [11]. Approximately 89% have one additional comorbidity and 15% have four or more [12]. Contributing to problems, patients often smoke and have sedentary lifestyles that often lead them to being overweight or obese [12]. Additionally, 10% of patients in the United States present with some degree of neuropathy at initial diagnosis, 40% will develop neuropathy within the first decade following that diagnosis and > 50% of patients over age sixty have some degree of neuropathy [13, 14]. The combination of neuropathy and at least one other comorbidity produces higher rates of complications (47% vs. 14%) compared to diabetics without neuropathy or another comorbidity [15]. All of which has

resulted in an increase in medical expenditures for the care of these patients. This has been borne out with studies demonstrating that in the United States $302 billion (USD) is spent annually managing diabetes with an additional $102 billion (USD) lost in revenue due to a reduced labor force, early mortality and lower productivity. The result is an economic burden of $1240 (USD) for each American with an annual medical expenditure of $13,240 (USD) for the medical care of each diabetic patient [12, 16, 17].

Specifically addressing adult ankle fractures, the incidence has been reported to be 100.8/100,000/per year. The ratio of men to women is 47:53, with bi- and trimalleolar fractures increasing in incidence, more so in women, as patients get older. In the United States, it has been estimated that approximately 260,000 Americans per year sustain an ankle fracture, with about 25% undergoing surgical management [18]. Within this population nearly 6%, or almost 16,000 patients per year, are diabetics who sustain an ankle fracture [19]. If the 25% needing surgery is extrapolated into the diabetic population, one would expect that annually approximately 4000 diabetics sustain an ankle fracture requiring surgery. Thus, less than 2% of all patients who sustain an ankle fracture in the United States are diabetics who are managed surgically for their injury.

2   Pathophysiology

Diabetes mellitus, and its resulting hyperglycemia, can lead to neuropathy, retinopathy, nephropathy, and cardiovascular damage [20, 21]. Chronic hyperglycemia results in increased blood viscosity, it impairs the ability of the red blood cell to deliver oxygen, it affects nitric oxide, which functions as an antioxidant and neurotransmitter, and it leads to microvascular compromise. The last of which can lead to coronary artery disease, stroke, peripheral artery disease, and produce nerve ischemia [22, 23]. In addition, hyperglycemia also decreases the ability of immune cells, specifically fibroblasts, from migrating and attaching to wounds, resulting in

healing stagnation that may last for up to 8 weeks [24].

Chronic hyperglycemia also affects bone physiology with the hallmarks being a decrease in bone turnover, a functionally weaker bone and an increased fracture risk [25]. Accumulation of advanced glycation end products (AGEs) disrupts cellular biology and the bony microarchitecture producing inflammation. This results in its binding to the receptor for AGEs (RAGE) [25, 26]. RAGE increases osteoclastic activity, i.e., bone loss, leading to osteoporosis and demineralization. In addition, AGEs also interfere with osteoblastic development, collagen, and osteocalcin production and increases osteoblast apoptosis [27]. The result is a decrease in osteon formation and the ability of the bone to remodel. Hyperglycemia also impairs proliferation and migration of the osteocytes primarily by increasing adipogenesis of mesenchymal stem cells to preferentially differentiate into adipocytes rather than osteocytes [28].

The results of this fatty tissue formation, combined with hemoglobin A1c levels ≥6.5%, decreases callus formation, tensile strength, bone stiffness, and fracture healing [29], resulting in poorer radiographic restoration. Studies have reported union times to increase to 163%, compared to nondiabetic patients, which is further increased to 187% when the fractures are displaced [30]. This abnormal bony pathology also increases the chances of sustaining a more severe ankle fracture, along with increasing mortality rates, postoperative complications, lengths of hospital stays, and costs, than in the nondiabetic patient [30, 31]. It is, perhaps, for all of these reasons that the use of nonoperative care is more often considered for management of the diabetic patient who presents with an ankle fracture.

3 Preoperative Evaluations

Unless the patient presents with an open fracture or an irreducible dislocation, there is no need for emergency surgery. It is important that one understands that both *medical and surgical* treatment will be needed to manage these patients, rather than placing them conveniently onto the surgical schedule.

3.1 History

The management begins with a thorough history, specifically asking about the mechanisms and the timing of the injury. Up to 74% of diabetic patients have scores less than the threshold for osteopenia and 39% below the threshold for osteoporosis [32]. Therefore, a low (ground level fall) mechanism of energy resulting in a complex fracture pattern may indicate poor bone quality. Additionally, how long they have been a diabetic and questions about when the injury occurred are also important. Because neuropathy is present in 10% of diabetics, 40% within the first decade following that diagnosis and > 50% of patients over age 60 [13, 14], neuropathy can be inferred for any patient continuing to ambulate on the injured extremity and presenting more than 24 h after the injury occurred.

The history should also include questions about the presence of comorbidities since these have also been shown to increase the rates of complications. With approximately 89% of diabetics presenting with one additional comorbidity and 15% have four or more [12], this means that all medical and vascular evaluations should be performed prior to any surgical intervention. Additional questions should also include whether ambulatory aids were used prior to their injury, whether they smoke, their use of insulin or other medications, and whether they have a history of previous ulcers, amputations, or infections.

3.2 Physical Examination

The examination should begin by inspecting the soft tissue envelope of the limb. Any wounds or lacerations should be evaluated for an open fracture. Look and palpate for changes in skin color, temperature changes, or any bony prominences, all of which may be an indication of impending skin necrosis. Additionally, fracture blisters or the presence of any tense compartments may

indicate that the extremity is not ready for operative fixation.

The neurologic examination begins with an observation of the extremity. Motor dysfunction, indicating intrinsic atrophy, is often manifested as clawing of the toes while autonomic dysfunction is suspected in patients presenting with dry, cracking, hyperemic skin (Fig. 1a, b). Of greatest concern is the loss of protective sensation due to neuropathy. Loss of vibratory sensation, pinprick, sense of position, or absence of deep tendon reflexes at the ankle (difficult to perform in the presence of a fracture) may indicate neuropathy but have only a fair agreement amongst evaluators [33]. Although the gold standard for identifying peripheral neuropathy is a nerve conduction study, the accepted method for detecting the loss of protective sensation is the use of a 5.07 (10-g) Semmes-Weinstein monofilament. This simple exam has a sensitivity and specificity of 91% and 86%, respectively [34], which increases with a minimum testing of four plantar sites (great toe, first, third, and fifth metatarsal heads) [33]. Detecting peripheral neuropathy is important since it contributes to poor protective sensation, physiologically inappropriate weight-bearing, noncompliance, and postoperative infections by a factor of four [35].

The last part of the physical exam should include a vascular evaluation. This is important since more than 40% of diabetics present with peripheral arterial disease [36]. The popliteal trifurcation is most affected however, vessel calcification in the ankle and the foot are also suggestive of vascular compromise (Fig. 2a, b). Visual signs suggestive of peripheral artery disease include dependent rubor, pallor with elevation of the extremity, dystrophic toenails, and hair loss [37]. The evaluation should continue with an attempt to palpate pulses and comparing it with the contralateral extremity. If pulses are still absent or diminished, after reducing the dislocation or improving the fracture alignment, the aid of Doppler ultrasound can be used to identify the vessels. Further evaluations can be made with the use of the ankle-brachial (ABI) index, which is often described as a more sensitive, noninvasive test for evaluating the patient's vascular status. A value of 0.91 to 1.3 is considered normal. In the diabetic, an ABI ≥ 1.1 is suggestive of arterial calcinosis and an ABI > 1.3 indicates poor compressibility of the vessel [37, 38]. However, in patients with acute ankle fractures an ABI may be difficult to perform, so for these patients one should pursue additional testing.

Fig. 1 (**a**) Picture of hyperemic skin in a 68-year-old male presenting with significant autonomic neuropathy and a fracture of the right ankle. Note the thick, stiff, dry, scaly and inflexible appearance of the skin that can crack easily and become an entry for infection. (**b**) Mortise view of the patient's ankle demonstrating a non-displaced fracture of the fibula (arrow) with no talar shift, treated successfully nonoperatively

Fig. 2 (**a**) Mortise view of left ankle demonstrating a displaced left bimalleolar ankle fracture (and a healing stress fracture of the fibula, arrow) with calcification of the vessels. (**b**) Lateral view demonstrating that the calcification of the vessels extends into the foot

Currently, three noninvasive vascular tests are available. These consist of measuring transcutaneous oxygen pressures (T_cPO_2), toe pressures (TP), and the toe-brachial index (TBI). The T_cPO_2 measures oxygen level beneath the skin. Pressures >70 mmHg are normal, < 40 mmHg the minimum indicate impaired wound healing [36]. The TP test places small blood pressure cuffs around each toe and measures the systolic pressure of each toe. A toe pressure of ≥68 mmHg is predictive of good wound healing [37, 38]. The TBI is calculated by dividing the toe pressure by the highest obtained ankle pressure. Currently a value >0.7 has been reported as the cutoff for a normal value [39]. The problem with the TBI is that in the presence of a fracture the patient may not tolerate a cuff placed around the ankle. If there is any question about the patient's vascular supply, they should be referred to a vascular surgeon for further workup. Currently, the authors' preferred method of vascular evaluation is to obtain toe pressures on all patients.

3.3 Laboratory Evaluations/ Radiologic Evaluations

As discussed, uncontrolled hyperglycemia results in pathophysiologic dysfunctions and can produce an increased rate of complications not seen in the nondiabetic population or in well-controlled diabetics [20, 21, 35, 40–43]. Therefore, in addition to standard preoperative laboratory studies, all patients should also have their glycated hemoglobin A_{1c} (HgA$_{1c}$) levels evaluated. Levels >6.5 have been shown to increase the risk of complications, produce longer hospital stays, and result in poor radiologic outcomes [44]. Those with HgA$_{1c}$ values >8 have a 2.5 times greater risk of developing an infection and poor surgical outcomes [45, 46]. However, for every 1% reduction in HgA$_{1c}$, there is approximately a 25–30% reduction in the rate of complications [47]. Patients should not be excluded from surgery, due to an elevated HgA$_{1c}$, but this information may help manage their diabetes during their postoperative care. In addition, during routine laboratory evaluations, the authors

also evaluate preoperative albumin levels. Studies have shown that low levels of serum albumin may denote malnutrition, which may be an indicator for developing postoperative complications [48, 49]. As with HgA_{1c} values, evaluating these values preoperatively will help during the postoperative management of these patients. The radiologic examination should begin with standard anteroposterior (AP), lateral and mortise views of the ankle. X-rays of the foot can also be performed to determine if there are any injuries to the foot. Advanced imaging studies, consisting of a computed tomographic (CT) scans and magnetic resonance imaging (MRI) images can also be performed to improve accuracy in diagnosing the patient's injuries. A CT scan helps to identify occult fractures and evaluate the incisura for syndesmotic injuries. An MRI scan can help diagnosis bony injuries not identified on plain films and help in diagnosing ligamentous, tendon and chondral injuries.

4   Fracture Management

Whether managed operatively or nonoperatively, the goals of treatment are to achieve a stable and congruent joint, restore function, and to prevent complications. Surgical goals should include obtaining an anatomic reduction of the mortise, providing stable fixation to maintain the reduction until adequate healing has occurred, avoiding the production of pressure areas to the ankle, and

avoiding complications that can lead to loss of limb or death. An additional goal, not often noted in nondiabetic patients, is to prevent instability or an early loss of the reduction that leads to the development of a Charcot joint. Regardless of whether the patient is treated operatively or nonoperatively, a discussion should also include whether prolonged immobilization and nonweight bearing of the patient will be needed as well as whether supplementary (nonoperative) forms of treatment will be necessary to obtain adequate healing. These can include the use of calcium, vitamin D and protein supplements, ambulatory aids, wheelchairs, and appliances such as a Charcot Restraint Orthotic Walker (CROW) boot. The decision driving treatment should be based on the injury pattern and the patient's physiology. Unfortunately, there is no clear algorithm to guide the treatment, based on fracture displacement, for this population. The timing of surgery is important to consider for patient management. Within 8–12 hours after the injury there is interstitial edema that may last 7–21 days. Here, the use of an external fixator, using fine wire/circular fixation or standard transarticular external fixation, will allow one to improve the alignment of the fracture, delay definitive fixation until there is improvement of the soft tissue envelope and allow for medical optimization of the patient before undergoing definitive fixation. Looking for wrinkling of the skin can indicate that tissues can now undergo fixation safely.
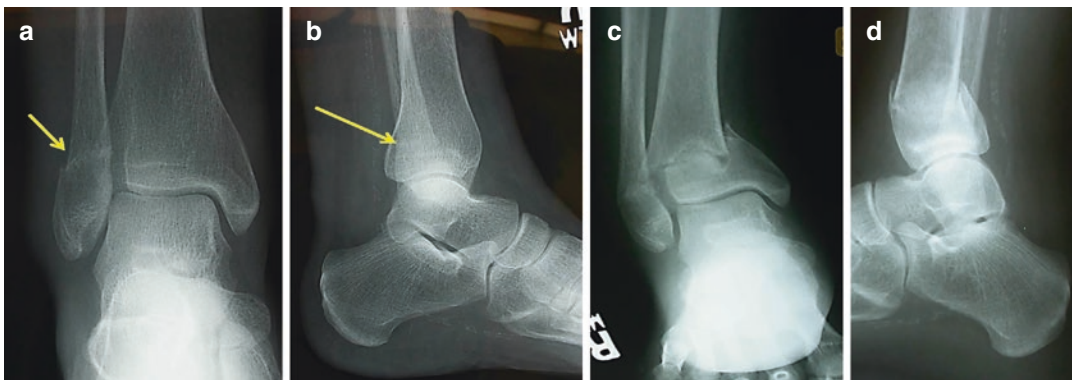

Fig. 3 (**a**, **b**) An AP and lateral views of a 52-year-old female, renal transplant, insulin dependent diabetes, who presented with a stress fracture (arrow) of the fibula demonstrating no displacement of the fracture. (**c**, **d**) Patient was treated conservatively in a walking boot and returned 1 month later. AP and lateral radiographs demonstrate displacement of both the fibula as well as a tibia fracture. The patient underwent surgical management

4.1 Nonoperative Treatment

The nonsurgical management can be controversial because of the concern for displacement however, these patients can be treated successfully. Nonsurgical care is offered to patients presenting with nondisplaced fractures, with a good rule of thumb being to double or triple the treatment offered to nondiabetic patients. Therefore, the nonoperative treatment consists of placing patients into a short leg, non-weight bearing cast for 10–12 weeks. Weekly or biweekly radiographs along with inspection of the soft tissue envelope to ensure that there has been no displacement of the mortise, and no problems to the soft tissue envelope have developed (Fig. 3a–d). After the casting period, patients are placed into a period of protective weight bearing, using a brace or boot, for an additional 2–3 months.

Very few studies discuss the nonsurgical management of diabetic ankle fractures. Most contain small numbers of patients and are often discussed as one of the arms of treatment, *in-lieu* of surgical management [18, 31, 40–43]. The complications reported in these studies have included malunions, due to a loss in the initial reduction; nonunions; the development of Charcot neuroarthropathy; infections; and the development of ulcers. Risk factors for developing a complication include seeing patients infrequently, early weight bearing or noncompliance, having a long duration of diabetes, the presence of neuropathy, insulin dependence, and patients with a history of Charcot neuroarthropathy. All these factors have been shown to contribute to a significantly increased mortality rate, postoperative complications, lengths of hospital stay, rate of nonroutine hospital discharge, and costs, when compared to nondiabetic patients with ankle fractures [19]. Risk factors not associated with complications include age, gender, and type of fracture [18, 31].

4.2 Operative Treatment

4.2.1 Preoperative Care and Planning

Prior to surgery, a discussion with the patient and their family should include the need for preoperative medical evaluations, whether a preoperative vascular evaluation should be performed, whether any adjunctive fixation will be needed to augment and hold the reduction until adequate healing has occurred and whether placement into a rehab or skilled-nursing facility will be needed
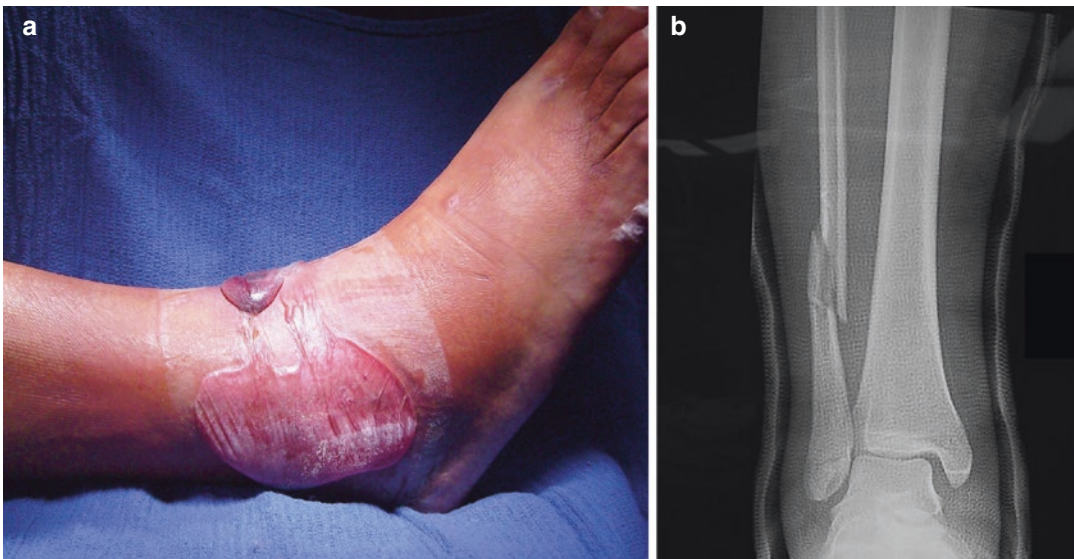
Fig. 4 (**a**) A 49-year-old male missed a step at home twisting his right ankle when he fell. The patient was transferred from an outlying hospital and on presentation demonstrated near circumferential fracture blisters about the ankle. (**b**) The mortise radiograph demonstrates the displaced fracture of the ankle. The patient was initially placed into an external fixator until the soft tissue improved allowing definitive fixation

until the patient and their family can be safely cared for at home.

The indication for surgical management is an unstable ankle fracture. However, before fixation is performed it is important to stabilize the soft tissue envelope. This includes a prompt reduction and splinting of the extremity, especially if fracture blisters have occurred. Immobilization can be achieved using a well-padded, nonremovable splint, or with the use of an external fixator if the reduction cannot be maintained by using the splint alone. The patient is instructed to keep the leg elevated as much as possible and is evaluated at weekly intervals. The ability to wrinkle the skin and a reepithelialization of the skin, after fracture blisters have resolved, indicates that the soft tissues have stabilized and are ready for surgical management (Fig. 4a, b). This may take anywhere between 10 to 21 days and during this period the preoperative evaluations previously discussed should be undertaken as part of the preoperative planning.

A large part of the preoperative planning should be to ensure that all the equipment and implants needed for surgery will be present. This equipment includes small, large and periarticular bone clamps, extra-long drill bits, extra-long screws, with lengths reaching 90–110 mm in length and in sizes ranging from 2.7-mm to 4.5-mm, Steinman pins, and extra-long k-wires. In addition, having a locking mini-fragment set, along with extra-long, small, and large fragment locking plates, should also be readily accessible. Simple rules of thumb are to extend the plates beyond the zone of injury and to use the strongest device that can be tolerated by the soft tissue envelope. The authors prefer the use of a 3.5-mm or 4.5-mm locking plates, or even the use of a plate designed for use in tibial plating, of at least ten holes in length, for fixation of the fibula while avoiding the use of semi-tubular or easily deformable (malleable) plates. Lamina spreaders or distractors should also be on hand if distraction of the fractures to achieve length, especially in the fibula, is anticipated. Lastly, an external fixator should also be ready for use if the ankle construct will need external augmentation.

4.2.2 Operative Management

There are five approaches that can be used to manage the diabetic ankle fracture: *standard*, *trans-syndesmotic*, circular (thin wire) external fixation, *trans-articular,* and a hybrid or *combination* of these techniques. Standard fixation, with expected good outcomes, can be considered for any patient presenting with an HgA_{1c} less than 7.0, a body-mass index (BMI) less than 30, able to sense a 5.07 or smaller Semmes-Weinstein monofilament, good vibratory sensation with a 128-hertz tuning fork, the presence of palpable pulses, nonosteoporotic bone, and those without any manifestations of autonomic dysfunction. Postoperatively, these patients can be managed like nondiabetic patients.

For patients who do not meet these criteria, three methods of fixation are available. These three techniques are different than standard methods of ankle fixation but have been developed to maintain an anatomic mortise and decrease the risk that failure of fixation will occur prior to adequate healing, leading to the development of a Charcot joint. In addition to prolonged immobilization and non-weight bearing, the operative principles for these three techniques include the use of long, rigid, locking plate fixation with long bicortical or quadricortical screw placement, using adjunctive fixation, considering the addition of a bone graft, and contemplating the use a bone stimulator (Table 1). Because of the patient's abnormal bony metabolism, the authors' current treatment of choice is to try (if insurance approval can be obtained) to add a bone stimulator to all patients when using one of these three alternative techniques. Small, prospective studies have described benefit of using a bone stimulator among diabetics undergoing foot and ankle surgery [50, 51]. Additional adjunctive fixation also includes bone graft or bone cement, inserting Steinman pins and leaving them in position until adequate healing has occurred, using calcium, vitamin D, and protein supplementation, and the use of a Strayer or Vulpius procedure to lengthen the Achilles tendon.

The *trans-syndesmotic fixation technique* uses the tibia to help stabilize the fibular fixation. This

Table 1 Authors prefer-
ences for the operative
management and postoper-
ative care of the acute dia-
betic ankle fracture

Rigid Fixation

   Longer and Thicker Plates (Minimum 10 holes)

   Locking Plate Technology

   More and Longer Screws

      Multiple tetracortical screws used for trans-syndesmotic fixation through plate

      Bicortical screws for medial/posteriomalleolar fixation, 4.0mm or larger

   Possible use of an Intramedullary nail

Adjunctive Fixation

   External Fixation

   K-wires across ankle joint

   Steinman Pins antegrade across ankle or retrograde through ankle/subtalar joints

   Combinations of these techniques

   Cement (Calcium Sulfate/Phosphate or polymethyl methacrylate)

Bone Graft/Supplements

   Consider using bone for fracture. Add calcium, Vitamin D, protein supplements

Bone Stimulator

   Recommend for use in these patients due to pathologic bone

Post-operative Care

   Week 1: Well-padded post-operative splint

   Week 2: Apply short leg non-weightbearing cast

   Week 3: Remove sutures

   Week 12: Remove Steinman pins, casting completed

   Month 4-5: Boot or brace, therapy, advance to WBAT*

   Month 6: Unrestricted activity

*WBAT: Weight bearing as tolerated

method consists of getting the fibula out to length, reducing the fracture and applying at least a 10-hole 3.5-mm or larger locking plate onto the fibula. The surgeon then inserts as many quadri-cortical (crossing four cortices), locking screws as possible through the fibula and into the tibia [52]. The advantage of using a locking plate is that it provides angular stability, which increases its load-carrying capacity and allows locking plates to be four times stronger than load-sharing con-structs. This means that for failure of fixation to occur it requires that all points of fixation fail as opposed to the loosening of individual screws, as seen with traditional compression plating tech-niques. To complete the fixation of the ankle, long, 4.0-mm bicortical screws should be used to stabi-lize the medial and posterior malleolar fractures (Fig. 5). This construct improves fixation stiffness without relying solely on the screw's purchase in the fibula. Although there is concern that this tech-nique may alter the biomechanics of the syndes-mosis, this has not been demonstrated clinically.

The *trans-articular (nonfusion)* method of fixation and can be approached in one of two ways. The first is to treat the patient using stan-dard reduction techniques, which is then aug-mented using two or three large, smooth, Steinmann pins, placed ante- or retrograde through the tibia, talus, and calcaneus [53, 54]. Although this is an older, described technique, its use can augment fixation in cases where tibiota-lar instability may occur (Fig. 6a–d). This tech-nique produces some stiffness of ankle and the hindfoot, but the advantage is it does not rely solely on standard fixation techniques to main-tain the reduction. The second approach is the use of a retrograde tibio-talar-calcaneal intramedul-lary nail [55]. Although some calcification or arthrodesis of the ankle or subtalar joints is pos-sible, the difference between this method and an arthrodesis technique is that neither the subtalar nor the ankle joint is exposed and prepared as when performing a formal arthrodesis. This approach works well in patients presenting with
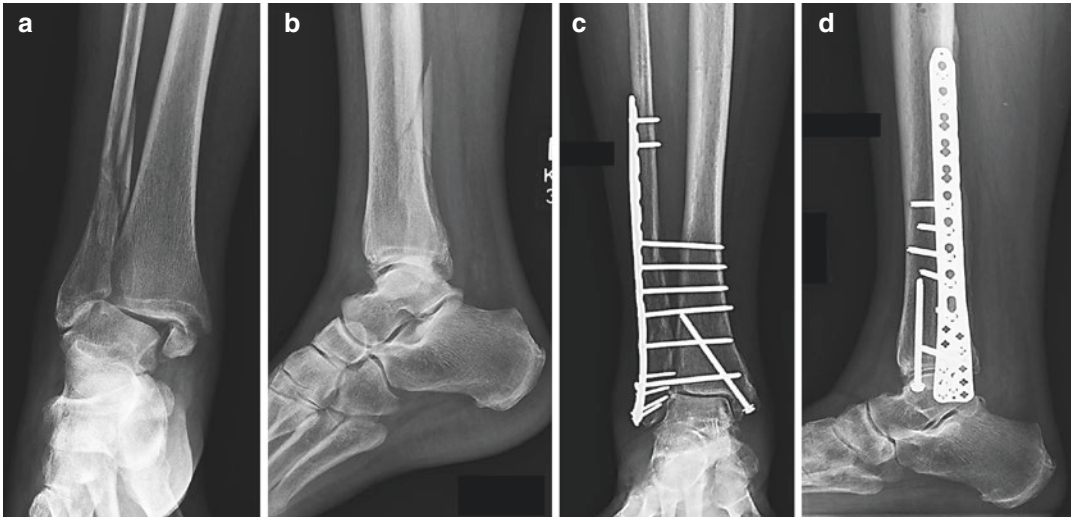
Fig. 5 (**a**, **b**) AP and lateral injury radiographs of the right ankle of a 48-year-old neuropathic, insulin dependent female who sustained a grand level fall at home. Patient's BMI was 28. A closed reduction and a splint were performed in the emergency department. (**c**, **d**)

Postreduction AP and lateral radiographs at 11 months demonstrating a *trans-syndesmotic* technique, using a tibial plate that was used to manage the fracture. Note the bicortical screw used to fix the medial malleolar fracture

pilon fractures but can also be used in certain unstable bi- or trimalleolar ankle fractures, especially in patients with morbid obesity (Fig. 7a–c). Once the fracture is healed a decision regarding nail removal can be made. To complete the discussion of trans-articular methods of fixation, immediate arthrodesis of the ankle has also been described for non-reconstructable fractures [56] but has rarely been performed for an acute diabetic ankle fracture. However, in the setting of poor bone quality, a poorly controlled diabetic with neuropathy, autonomic changes, and poor potential to heal the fracture, an immediate arthrodesis may be considered to improve the outcome of that patient.

The third technique is *a combined technique*. In this approach the trans-syndesmotic technique is augmented using two or three large, smooth Steinman pins, which are placed ante- or retrograde through the tibia, talus, and calcaneus (Fig. 8a–c). This approach provides significant stiffness to the construct and is currently the authors' treatment of choice for the management of acute diabetic ankle fractures that are unable to be managed with standard fixation, especially in the morbidly obese patient. Like other methods described, the resulting stiffness acquired by the ankle with this approach does not seem to be a problem clinically because ambulation progressively restores motion between the tibia and fibula. The authors' preference is to leave the Steinman pins for either the trans-articular or combined technique in place for at least 2 (and possibly) 3 months before they are removed.

Fig. 6 (**a**, **b**) An AP and lateral views demonstrating a displaced ankle fracture in a 60-year-old insulin dependent diabetic female, status post renal transplant who sustained a ground level fall at home. (**c**, **d**) An AP and lateral views demonstrating a *trans-articular fixation* technique consisting of standard fixation augmented with two vertically placed Steinman pins. Pins were pulled at 3 months
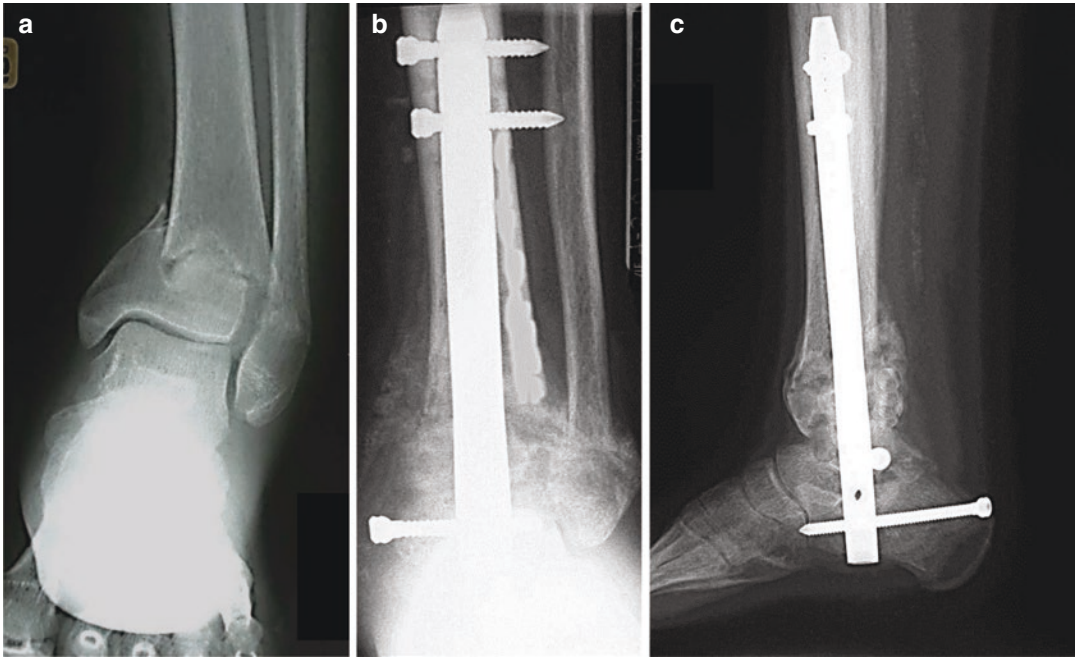
Fig. 7 (**a**) An AP view of the left ankle in a morbidly obese male who tripped walking downstairs demonstrating a displaced extra-articular pilon fracture. (**b**, **c**) An AP and lateral views demonstrating fixation using an intra-medullary hindfoot nail. Note that the preoperative varus positioning has been corrected. No takedown of the ankle or subtalar joints were performed
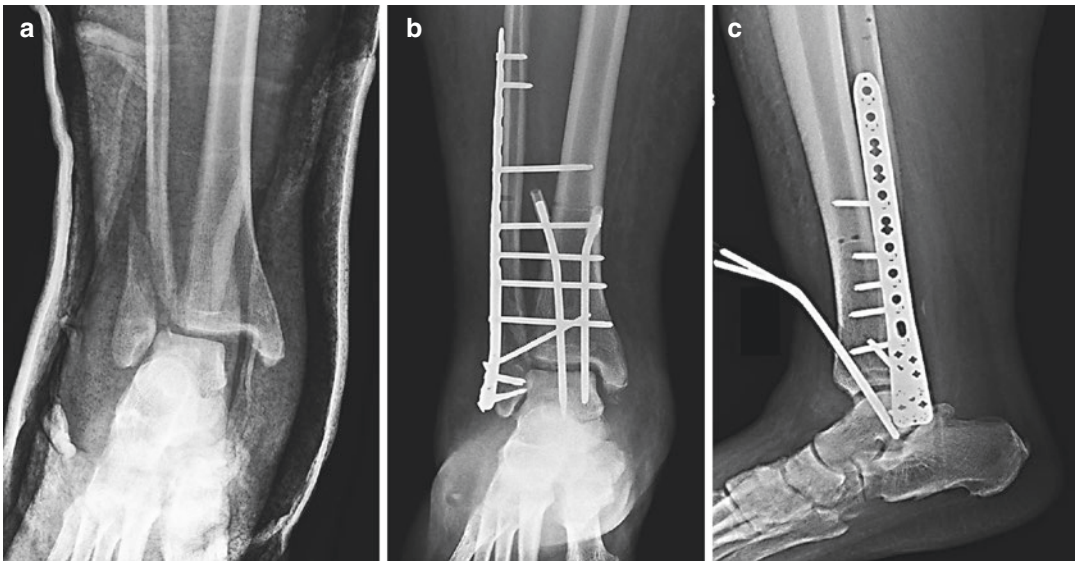

Fig. 8 (**a**) A 68-year-old morbidly obese female fell while getting out of her car, twisting her right ankle. The AP view demonstrates an unstable ankle fracture with a displaced fibula fracture and a lateral talar shift. The patient was neuropathic and was a poorly controlled type II diabetic. (**b**, **c**) Postoperative AP and lateral views demonstrating the use of a *combination* technique in which a tibial plate was used for fixation of the fibula showing the placement of multiple trans-syndesmotic screws augmented with two vertical Steinman pins placed antegrade

5 Complications and Salvage

Complications are more common in diabetics than nondiabetics, occurring in 26% of patients [57, 58] but can range from 3.6% to 43% [19, 31, 35, 40–43, 45, 58–61] and can occur individually or in any combination. Using multivariable regression analysis, African American race, obesity, tobacco use, and neuropathy were found to be significant independent predictors of worse functional outcomes, with an unplanned secondary operation rate over two times that of nondiabetic patients [57]. The four major complications associated with managing these patients consist of *failure of fixation* leading to malunions or nonunions, *skin and wound problems*, *infections,* and the *development of Charcot neuroarthropathy*. It should not be surprising that the rates of complications are higher in poorly controlled diabetics [42, 60]. The question is, after operating on these high-risk patients, can their complication(s) be treated without necessitating an amputation as the only salvageable option?

5.1 Failure of Fixation

In this context, failure of fixation is empirically defined as a loss of the reduction early in the postoperative period (within the first 2–4 weeks), without the development of a Charcot joint (Fig. 9a, b). The most common reasons for this complication are often a combination of the patient's neuropathy, their inability to avoid weight bearing on the extremity, and an inadequate fixation performed at the time of the index procedure. By far the biggest mistake is in trying to manage these patients like a well-controlled or nondiabetic patient. More than 60% of patients are obese or morbidly obese [48] and have little or no upper body strength. Thus, immobility and non-weight bearing becomes a significant issue for these patients. Either because of their central nervous system (CNS) neuropathy or because they are seeking independence, they will often begin full weight bearing within hours or days after their surgery. To decrease failure of fixation, leading to nonunions or malunions (2–5%) [58], patients may need to be placed into a skilled nursing or rehabilitation facility where they can be supervised. If they are going home, they

should be placed into wheelchairs to help them maintain a non-weight bearing attitude. In addition, a discussion should be made with their caregivers about the importance of non-weight bearing, with weekly visits necessary if noncompliance persists, to make sure that displacement or failure of fixation has not occurred.

The salvage of a failed fixation is via one of the three previously discussed alternative approaches, with the timing dependent on the health of the soft tissue envelope. Continued conservative treatment of the misaligned extremity will often result in malunions, nonunions, the development of contractures, and skin breakdown and/or ulcerations. In some cases, the addition of trans-articular external fixation can improve the overall alignment, but it may not always produce an anatomic reduction of the mortise (Fig. 10a–d). If a revision of the fixation is unable to be performed, then a salvage using an ankle or double hindfoot arthrodesis (ankle and subtalar joint), may be necessary to salvage the extremity.

5.2 Skin and Wound Problems

Wound edge necrosis and dehiscence, without the presence of infection, are always concerns when managing these patients. Even without surgery, there is already a considerable challenge in trying to get things to heal in this population [42, 43, 62]. It has already been noted that hyperglycemia decreases blood flow to both small and large vessels [63], increases blood viscosity, impairs the ability of the red blood cells to flow, and decreases the amount of oxygen reaching the tissues. This hypoxia inhibits fibroblasts from migrating to the wound and causes them to lose their ability to proliferate, which may last for up to 8 weeks [19, 42, 62, 64]. The addition of smoking, hypertension, dyslipidemia, increased body mass index and advanced age, have also been shown to have a negative effect on wound healing [19, 30, 31, 38, 41, 45, 64].

The combination of hyperglycemia, fracture edema, and hypoxia create a poor environment for diabetic wound healing [38, 58, 59, 62], even in patients managed nonoperatively. Early salvage requires frequent (weekly) clinic visits since these
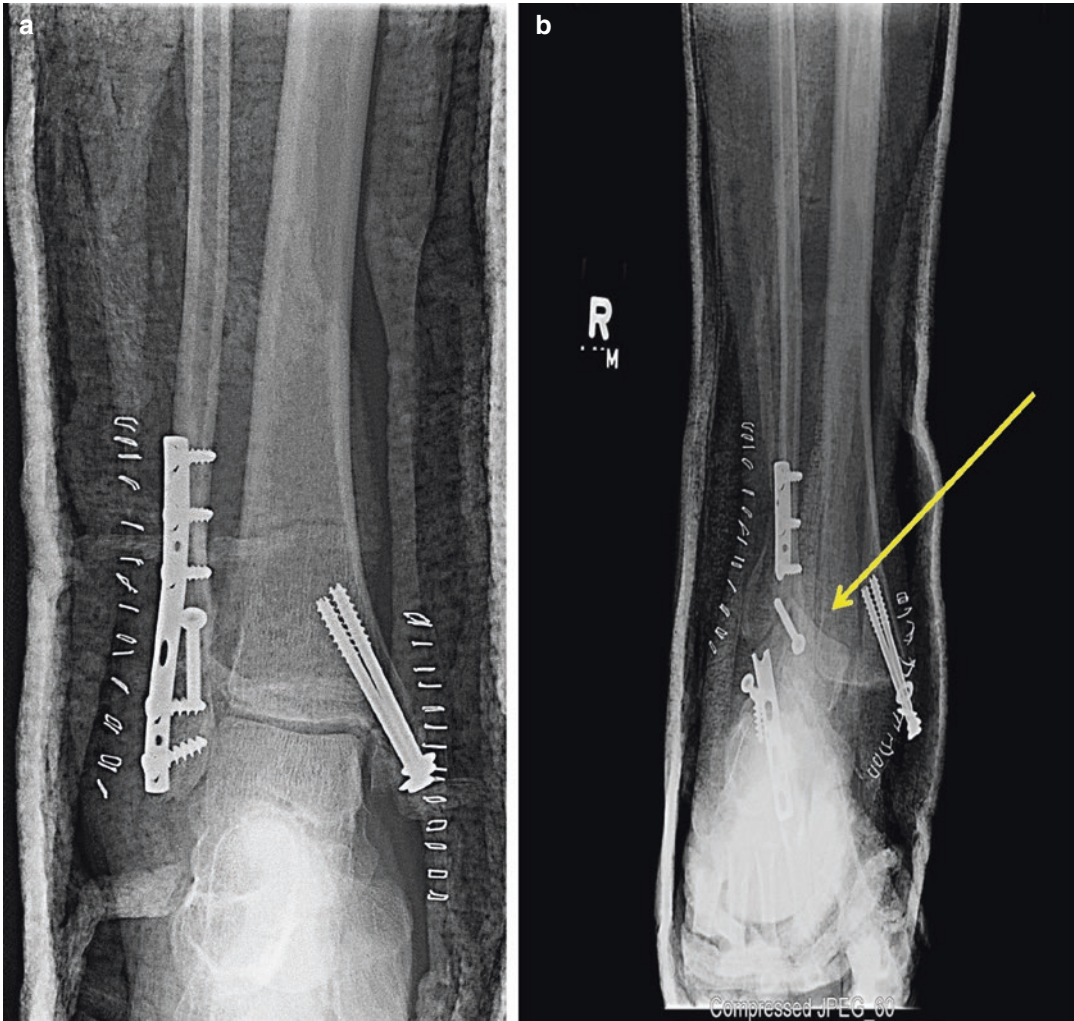
Fig. 9 (**a**) Immediate postoperative AP view of a supination-external rotation injury that was managed using standard fixation technique in an obese, neuropathic 51-year-old male. (**b**) At the first postoperative visit 2 weeks later, the AP radiograph demonstrates failure of fixation with a broken fibular plate and displacement of the fracture. Note the dislocation of the tibiotalar joint (arrow). Salvage was achieved using a hindfoot nail

problems may be identified early during routine cast changes. During these visits, encouraging good control of their diabetes, discussing the need for elevating the extremity, and placing them into wheelchairs may all help with healing, compliance, and edema. In addition, reapplying a well-padded splint, *in-lieu* of the cast, may help avoid pressure to the compromised skin. When skin or wound problems are identified, the authors' preference is for weekly office visits, daily wound care (e.g., wet-to dry-dressings) [65], through a windowed cast, and the empiric use of a broad-spectrum oral antibiotic. If the wound fails to improve, the formal use of irrigation and debridement and

negative pressure wound therapy may be necessary. If after 4–6 weeks of negative pressure therapy, worsening or no improvement is noted, a plastic surgery consultation may be needed.

5.3 Infection

A major concern in managing these patients is the development of an infection. Both superficial and deep infections can occur with rates ranging from 6% to 43% [41, 57]. Risk factors for the development of an infection include, the presence of peripheral arterial disease, neuropathy, diabetes
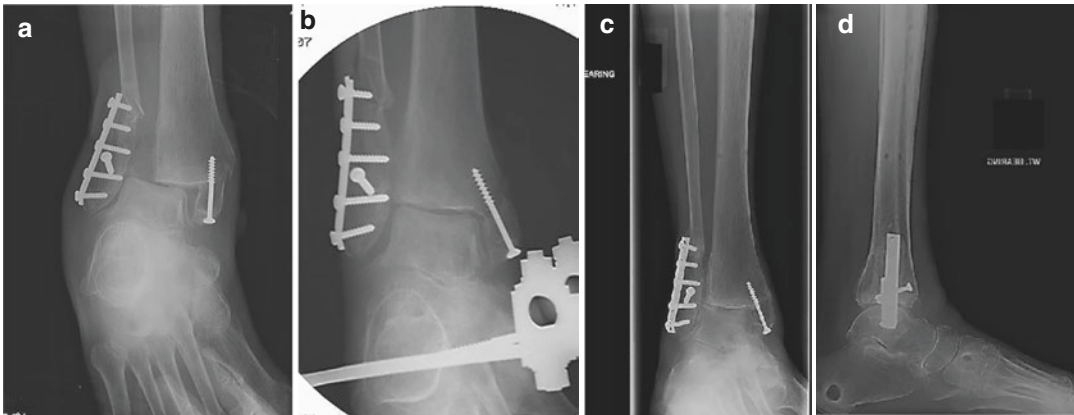
Fig. 10 (**a**) Use of a standard fixation technique demonstrating failure of fixation in an obese, neuropathic female seen on postoperative day 16. (**b**) The patient underwent the placement of a trans-articular external fixator. The mortise view demonstrates an anatomic reduction of the mortise. The fixator was left in position for 3 months. (**c**, **d**) At 3.6 years, AP and lateral radiographs demonstrate an anatomic mortise of the ankle joint. Note that the patient does have some post-traumatic arthritis of the tibiotalar joint but did not develop a Charcot joint

of long duration, poor glucose control (especially a $HgA_{1c} > 8$), the presence of a Charcot joint, the presence of edema and ecchymosis, older patients, obesity, a history of rheumatoid arthritis, a history of a previous ulcer, and in patients presenting with an open fracture [18, 31, 35, 40, 45]. The presence of neuropathy is biggest risk factor. Patients lose their ability to sense an infection, which is why even patients treated nonoperatively have been identified with an infection [18]. Factors that do not increase the risk of infection include tobacco use, gender, type of fracture, American Society of Anesthesiologists (ASA) classification, and whether the surgery was performed as an inpatient or an outpatient [31, 35]. Consistent with current literature, diabetes on admission has a 2–3 times greater risk of infection and a seven times risk of amputation after an ankle fracture [57, 59].

Frequent visits may not decrease this complication from occurring but can offer earlier treatment when they are identified. As with wound complications, the infection is often identified during a routine change of the patient's cast. For superficial infections, windowing the cast, to allow local, daily wound care, providing oral antibiotics, and weekly office visits may be sufficient to manage the problem. In contrast, all deep infections should be managed with irrigation and debridement, a minimum 6-week course of intravenous antibiotics, and removal of all loose implants. Avoid the urge to perform a local swab of the area. Rather, deep cultures or even a bone biopsy may be necessary to identify the organism(s) if osteomyelitis is suspected. Once the infection has been controlled, the use of a local flap or a free tissue transfer may be necessary if the wound is not able to be managed with secondary closures. If after bony debridement significant bone has been removed or the articular surfaces have been lost, then an ankle or double hindfoot arthrodesis may be needed to salvage the extremity. If the extremity is not salvageable then an amputation may be necessary.

5.4 Charcot Neuroarthropathy

The incidence, in diabetic ankle fractures, has been reported to occur between 6% and 47% [31, 43, 45, 61, 64, 66]. It is challenging to manage, especially when it presents after the surgical care of an ankle fracture, because it is often confused with infection. On initial presentation, patients often present with erythema, edema and warmth to palpation. The differential diagnosis can include gout, cellulitis, abscess, and osteomyelitis however, the diagnosis of a Charcot joint should be considered in any compliant patient, who had an anatomic reduction of the mortise and presents with failure of fixation. Careful physical, laboratory, and radiographic examinations will identify whether the patient has developed a Charcot neuroarthropathy or has a postoperative infection (Table 2).

Table 2 Differentiation of charcot and charcot with infection

	Charcot	**Infected Charcot**
Physical Findings		
Skin	Edema, Erythema, Warm	Edema, Erythema, Warm
Leg Elevation	Resolves edema/erythema	Edema/erythema persist
Ulcer	Non-draining	Draining, extends to bone
Temperature	Afebrile	Febrile
Discomfort	Painless	Painful
Lab Findings		
Glucose	No change	Out of Control
WBC**	Normal	Elevated
CRP,ESR+	Normal	Elevated
X-ray Exam	Fracture/Dislocation, periosteal reaction	Same, *Air Present*
99m-TC^ Bone Scan	Positive	Positive
Indium-111 Bone Scan	Negative (usually)	Positive
MRI* Scan	Soft tissue & bony edema, joint destruction	Fluid Collections; Air

White blood cells**
C-reactive protein, E Sed Rate+
Technetium-99m^
Magnetic Resonance Imaging*

The salvage of these patients can be difficult because they often present late with malunions, nonunions, and contractures of the extremities. Reconstruction should be considered when the extremity is in the subacute or chronic stages. Indications for surgery should include failure of conservative care, chronic deformity, instability not amenable to bracing, and evidence of abnormal plantar pressures, despite the use of an orthoses and special shoes. Reconstructions often involve the combination of bony and soft tissue procedures to improve the alignment and obtain a viable extremity. Further discussions on reconstructions can be found in the chapter on the Management of the Charcot Ankle.

In conclusion, if the patient is neuropathic, obese, has peripheral arterial disease and an elevated HgA_{1c}, avoid managing the acute diabetic ankle fracture like those treated in the nondiabetic population. These patients have increased rates of complications and infections and are usually noncompliant due to their neuropathy. Patients should be advised about higher risks of complications as related to ankle surgery. Careful preoperative eval-

uations and postoperative vigilance can improve outcomes. These patients require very rigid fixation, often with adjunctive fixation, with extended periods of immobilization and protective weight bearing. Significant deformities can produce abnormal plantar pressure, irritability with shoewear and malalignment of the extremity. However, good outcomes can be expected with alternative techniques and even some mild residual deformity does not seem to produce much disability.

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