TREATMENT OF ANKLE FRACTURES IN PATIENTS WITH DIABETES

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Abstract: Patients with diabetes mellitus have a higher risk of complications after sustaining an ankle fracture, including fracture displacement, superficial and deep infection, hardware failure, and neuropathic arthropathy. With the increased incidence of diabetes among the aged, the increased incidence of complications due to diabetes mellitus and its sequelae are important to keep in mind when treating ankle fractures.

Ankle fractures are one of the most common orthopedic injuries. More than 260,000 ankle fractures are diagnosed annually in the United States, and these numbers are increasing as the population ages. Approximately one-quarter of all ankle fractures are treated surgically. With the increased incidence of diabetes among the aged, the increased incidence of complications due to diabetes mellitus and its sequelae are important to keep in mind when treating ankle fractures.

Diabetes Mellitus

Diabetes mellitus is a growing problem in public health today. According to the Centers for Disease Control, 25.8 million people in the United States had this disease in 2010, representing 8.3% of the population. Of these people, 18.8 million carry a known diagnosis and 7 million are undiagnosed. In the elderly, 26.9% have the disease, amounting to 10.9 million people. One point nine million new cases of diabetes are diagnosed every year. The prevalence of diabetes between 1990 and 2001 has increased 61% and shows no signs of slowing. In fact, it has been estimated that between 2000 and 2050, the prevalence will increase by 165%, with higher rates of increase among the elderly and minorities. The world population of diabetics is expected to swell to 366 million by 2030. The risk of developing diabetes through the course of a person’s lifetime in the United States in 2000 was estimated to be 32.5% for men and 38.5% for women. This risk is higher for minorities, especially Hispanics. Diagnosis of type 2 diabetes has been associated with decreased life expectancy of 14.3 life-years in women and 11.6 life-years in men. In addition to decreased life expectancy, a decrease of quality-adjusted life-years of 22.0 for women and 18.6 for men has been predicted.

Diabetes mellitus refers to a group of metabolic disorders resulting in systemic hyperglycemia. The 2 main categories of the disease are type 1 diabetes, caused by autoimmune destruction of insulin producing β-cells in the pancreas, and type 2 diabetes, caused by a combination of increased peripheral insulin resistance and decreased insulin production by the β-cells in the pancreas. The diagnosis of diabetes mellitus is contingent on 1 of 4 criteria:

1. Fasting plasma glucose ≥ 126 mg/dL.
2. Elevated random glucose levels of ≥ 200 mg/dL, with associated symptoms of hyperglycemia, including polyuria, polydipsia, and unexplained weight loss.
3. A positive glucose challenge test, resulting in a blood sugar of ≥ 200 mg/dL.
4. An elevated hemoglobin A1c ≥ 6.5%.

The end result of diabetes is systemic hyperglycemia. This hyperglycemia leads to the glycosylation of proteins throughout the body and intracellular formation of sorbitol and other alcohols. This hyperglycemic state also disrupts the regulation of reactive oxygen species (such as peroxides and oxygen ions), which leads to large amounts of these reactive species.
oxygen radicals being formed. These reactive oxygen species damage cellular proteins, membrane lipids, and nuclear material, leading to increased levels of apoptosis. Decreased nitrous oxide production leads to vasoconstriction, nerve ischemia and decreased production of neurotrophic factors. All of these effects lead to the common sequelae of poorly controlled diabetes: peripheral neuropathy, vasculopathy, nephropathy, immune dysfunction, and retinopathy.6

Peripheral neuropathy is a common complication of uncontrolled diabetes. Approximately 10% of diabetics have some element of neuropathy on presentation and 40% will be diagnosed with neuropathy within 1 year. More than half of all diabetics older than 60 years have some element of neuropathy. Peripheral neuropathy is diagnosed using the 10-g (5.07) nylon Semmes-Weinstein monofilament test, which has a sensitivity of up to 91% and a specificity of up to 86%.7,9 A 128-Hz tuning fork may also be used to test the sensitivity of the extremities to vibration, and may be more sensitive for early neuropathy.10

Vasculopathy is another common complication of uncontrolled diabetes. Both the large and small arteries are affected, as well as the microcirculation.11 This leads to difficulties with oxygen transport and localized tissue ischemia. The impaired wound healing seen in uncontrolled diabetics is due to difficulties in collagen production and cross-linking, as well as deficiencies of fibroblast function in an ischemic environment.12 Diabetics also have more aggressive and diffuse atherosclerosis than the average person, and after 20 years of having active diabetes, >45% of patients have some element of atherosclerosis.

OUTCOMES RESEARCH IN DIABETICS WITH ANKLE FRACTURES

Three recent studies have reported ankle fracture outcomes in the literature. In 2005, Ganesh et al13 analyzed the national inpatient sample for a 12-year period (1988-2000). They looked at 160,598 ankle fractures treated operatively during this time period and found that patients who had diabetes mellitus had significant increases (P<.001) in in-hospital mortality, postoperative complications, length of stay, rate of nonroutine discharge, and total hospital charge as compared to patients without diabetes mellitus.13

In 2006, Egol et al14 prospectively followed 232 patients with ankle fractures for 1 year. They measured baseline characteristics such as common demographics, comorbidities, and American Society of Anesthesiologists (ASA), Short Musculoskeletal Function Assessment, and American Orthopaedic Foot & Ankle Society (AOFAS) ankle/hindfoot scores. At 1 year, complete follow-up was available for 195 patients. Of these patients, 88% had no or mild ankle pain, 90% had no limitations or limitations only in recreational activities, and 90% had >90% recovery of function according to their AOFAS score. They found that predictors of function recovery included being younger than 40 years (P=.004), male sex (P=.02), ASA 1-2 (P=.03), and the absence of diabetes mellitus (P=.02).14

Most recently, Wukich et al15 did a retrospective, case-controlled study comparing 105 patients with ankle fractures: 46 with diabetes mellitus with noted end-organ damage such as peripheral artery disease, nephropathy, or peripheral neuropathy, and 59 with diabetes mellitus and no end-organ damage. They followed the patients for an average of 21 months. Their analysis showed that patients with complicated diabetes mellitus had an increased risk of any complication (odds ratio, 3.8), non-infectious complications (odds ratio, 3.4), and revision surgery (odds ratio, 5.0). They noted that most complications were unrecognized by the patients themselves.15

Common complications noted in many studies include higher rates of postoperative infections, difficulty with wound healing, and development of Charcot arthropathy. Soo Hoo et al16 looked at all patients who had surgery for an ankle fracture according to the California discharge database between 1995 and 2005. A total of 57,193 patients fit into this group and were evaluated for their rates of short-term complications, such as mortality and infection, and intermediate-term complications, such as revision or arthrodesis. Overall, rates of short-term complications, such as pulmonary embolus (0.34%), mortality (1.07%), amputation (0.16%), and infection (1.44%) were low. Similarly, rates of intermediate complications such as revision (0.82%) and arthrodesis/ankle replacement within 5 years (0.96%) were also noted to be relatively low. However, the presence of complicated diabetes (odds ratio, 2.3) and peripheral vascular disease (odds ratio, 1.65) were shown to be indicators of significantly increased risk of both short- and intermediate-term complications.16

INFECTION

Infection has been a problem for diabetics treated either conservatively or surgically. Flynn et al17 retrospectively compared the treatment of closed ankle fractures in 73 nondiabetic patients and 25 patients with diabetes mellitus. They found that the risk of infection was 4 times as great in the diabetic group (32%) as compared to the patients who were not diabetic (8%). Diabetic patients who were treated surgically had twice the infection rate of nondiabetics. Even diabetic patients who were treated in a cast had a 66% infection rate. Diabetic patients with peripheral vascular disease, peripheral neuropathy, swelling and/or ecchymosis, or who were poorly compliant had increased risks of infection.17

In 2010, Wukich et al18 performed a retrospective review of 1000 patients who had had orthopedic and ankle surgery, looking at the association
of age, sex, and history of diabetes mellitus or rheumatoid arthritis, as well as other comorbidities with the incidence of postoperative infections. They found an overall infection rate of 4.8%, with the diabetic patients having >4 times the infection rate of the nondiabetics (13.2% vs 2.8%, respectively).

More than half of the infections (52%) occurred in the diabetic population, which was only 19% of the study population. Patients with complicated diabetes were noted to have 10 times the risk of infection, and even patients with uncomplicated diabetes had 6 times the risk of infection.\textsuperscript{18}

Several studies have looked retrospectively at complication rates in patients with diabetes mellitus treated surgically for their ankle fracture. Costigan et al\textsuperscript{19} evaluated 84 patients with 14 complications, including 10 infections and 4 cases of Charcot arthropathy. Patients with absent pedal pulses or peripheral neuroarthropathy were found to have significantly elevated risk of complications.

Blotter et al\textsuperscript{20} looked at 21 patients who had 13 complications, including 7 infections, 3 losses of fixation, 1 case of reflex sympathetic dystrophy, 1 wound dehiscence, and 1 plantar ulcer. Two of the infections resulted in transarticular amputations. The group of diabetic patients were found to have a complication risk 2.76 times greater than a nondiabetic matched control group. McCormack and Leith\textsuperscript{21} studied 26 patients who had a 42.3% complication rate, including 1 malunion, 1 wound complication requiring flap coverage, and 2 deep infections. This study also had 2 lower-extremity amputations with eventual mortalities.

**Charcot Arthropathy**

Neuropathic joints were first described in 1868 by Jean Martin Charcot. Initially, he was describing the neuroarthropathy associated with high-grade syphilis.\textsuperscript{22} However, this description has come to describe the neuroarthropathy associated with uncontrolled diabetes mellitus, first described by Jordan\textsuperscript{23} in 1936. Eichenholz\textsuperscript{24} was the first to describe the progression of Charcot arthropathy. Stage 1, known as the destruction stage, is characterized by an acute inflammatory process, with edema, hyperemia, and erythema. Stage 2, the coalition stage, is characterized by coalescence of new bone at fracture sites and regression of the edema, hyperemia, and erythema. Stage 3, the resolution stage, is the continuation of the consolidation of the new bone formed in stage 2.\textsuperscript{24} Schon and Marks\textsuperscript{22} have recommended that a stage 0 be added, to represent at-risk patients with risk factors for Charcot arthropathy, such as diabetes mellitus and peripheral neuropathy, who have no radiographic evidence of neuroarthropathy. Brodsky\textsuperscript{25} has proposed an anatomic classification for neuropathic joints: type 1 (60%-70%) involves the midfoot, type 2 (20%) involves the hindfoot, type 3A involves the tibiotalar joint, and type 3b involves the os calcis.

The reason that neuroarthropathic joints occur among diabetic patients is still an area for debate. Johnson\textsuperscript{26} proposed the neurotraumatic theory in 1967. His thought was that the decrease in protective sensation in neuropathic patients allowed accumulation of microtrauma, resulting in joint destruction and fracture. However, detractors of this theory have stated that patients with Charcot joints still feel pain in the lower extremities once fractured and acute fractures can result in neuropathic changes. In 1981, Brower and Allman\textsuperscript{27} proposed the neurovascular theory. This theory supposed that a sympathetic reflex caused increased blood flow to the bone, resulting in bony resorption by osteoclasts. However, detractors of this theory point out that patients who undergo surgical sympathectomy do not get Charcot joints.

**Treatment Supplementation Options**

Unstable ankle fractures in diabetics should be treated surgically, through traditional open reduction and internal fixation. However, recommendations have been made in 3 papers describing methods of supplemental fixation.

Schon and Marks\textsuperscript{22} recommended avoiding the use of bone-holding clamps and using either manual traction or a femoral distractor to assist with the reduction. Bony fragments should be reduced with Kirschner wires. Because of the poor bone quality of the diabetic patient, as well as the concomitant healing and vascular problems, every attempt should be made to maintain the periosteal blood supply to the fracture fragments and to not further fracture them through rough handling. They stated that it may be necessary to place screws into the tibia from the fibular fixation for greater stability, as the bony strength of the fibula may not be sufficient to hold screws well. On the medial side, it may be necessary to use a 2- or 3-hole plate in a neutralization or buttress mode to supplement other fixation in the medial malleolus. Syndesmotic injuries should be fixed with 1 or 2 tetracortical screws for increased stiffness and stability.\textsuperscript{22}

Jani et al\textsuperscript{28} looked at a series of 15 patients (16 ankles) with noted diabetic neuropathy and unstable ankle fractures who were treated operatively and then followed for an average of 69 weeks. They were treated with traditional open reduction and internal fixation, which was supplemented with retrograde trans calcaneal-talar-tibial fixation with Steinmann pins or large screws. Postoperatively, they were made non-weight bearing for 12 weeks in a short-leg total contact cast. This was removed, along with the Steinmann pins or large screws, at 12 to 16 weeks, and the patient was then placed in a short-leg cast or a boot and made partial weight bearing for an additional 12 weeks. After completing this course of partial weight bearing, they were allowed to bear weight as tolerated, but wore an ankle-foot orthosis or total contact inserts. Twelve of the ankles were able to bear weight at follow-up and could walk without difficulty. There were 4 deep infections in the other ankles, 2 requiring...
irradiation and debridement and 2 requiring transtibial amputation.28

Perry et al29 looked at a series of 6 patients with noted diabetic neuropathy who presented for treatment of the failed traditional fixation of their Weber B and C ankle fractures. Five of the patients had neuropathy secondary to diabetes mellitus and 1 secondary to alcoholism. Five of the 6 fractures were due to trauma and 1 presented without an antecedent trauma. All were treated with a laterally placed 4.5 dynamic compression plate on the fibula with multiple 4.5-mm tetracortical syndesmotic screws. The patients remained nonweight bearing in a short-leg cast for a minimum of 3 months. At follow-up, the patients were satisfied with their well-aligned, functional limbs. There were no amputations within this group, resulting in a 100% salvage rate.29

CHARCOT TREATMENT OPTIONS
Several methods have been described for the treatment of Charcot arthropathy. Initial treatment usually starts conservatively. Patients are put into a total contact cast or a Charcot restraint orthotic walker boot for immobilization while they are in the initial destructive stage of their neuroarthropathy. After they show signs of moving into the coalition phase, with commensurate decrease in edema, erythema, and hyperemia, they are switched to a removable bivalved ankle-foot orthosis. After the consolidation phase has finished and they have moved into the resolution phase, they are placed into custom-molded, dual-density accommodative insoles to decrease the contact pressure on bony prominences. Remaining areas of high pressure due to collapse of the foot architecture can be addressed by an exostectomy of the bony prominences. However, aggressive excision should be avoided to preserve joint stability.29

Arthrodesis is the salvage treatment of Charcot neuroarthropathy. The primary indication for arthrodesis is to retain a foot or leg threatened by damage to its soft tissue envelope. Formation of a stable arthrodesis can be challenging, as the pathophysiology of diabetes mellitus and that of the neuropathic arthropathy itself do not lend themselves to the formation of good bone. Papa et al30 looked at 29 patients with Charcot arthropathy, of which 21 cases were of the ankle. These were all managed with open reduction and arthrodesis: 25 through rigid internal fixation and 4 through the use of external fixation. All patients were nonweight bearing for 4 to 14 months in a total contact cast. After an average 42-month follow-up, 20 patients went on to fusion and 7 went on to a stable pseudarthrosis, for a successful salvage in 93% of patients. Unfortunately, this study noted many complications among its participants, including 1 incidental mortality, 1 traumatic amputation for fixation failure, 3 revisions of fixation, 2 malunions, 8 skin problems (sloughs/ulceration), and 3 superficial infections.31

Pinzur and Noonan32 looked at 9 patients with Charcot arthropathy of the ankle who underwent ankle arthrodesis with a retrograde femoral nail. At 10.5 weeks, fusion was noted on radiographs. All patients were ambulatory at an average 32-week follow-up.32 Dalla Paola et al33 looked at 18 patients with Charcot neuroarthropathy who were treated with retrograde transcalcaneal nailing. Subsequent to their surgery, they were made nonweight bearing for 3 months, followed by partial weight bearing for an additional 3 months. At an average 14-month follow-up, 14 patients had a stable fusion and the 4 remaining patients had fibrous unions, thus resulting in a 100% limb salvage rate.33

Fabrin et al34 described using a compressive external fixator as the means for achieving arthrodesis. They looked at 11 patients (12 ankles) with unstable or malaligned Charcot ankles with noted plantar ulceration. All were treated with arthrodesis, 7 tibiotalar and 5 tibiocalcaneal. Postoperatively, they were kept in compression via the external fixator for 5 weeks, then converted to a total contact cast for 6 weeks. They were allowed to bear weight at 12 weeks in a brace. At follow-up, 1 ankle had gone on to a transtibial amputation secondary to a loosened pin. The other 11 feet were able to independently walk with a brace, for a 92% limb salvage rate.34
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Our Preferred Treatment

Since diabetic patients with bimalleolar, trimalleolar, or ankle subluxation are at high risk for further displacement, we usually fix these fractures even if the initial reduction is acceptable (Figure 1). Because of the prolonged healing time due to poor vascularity and the high incidence of neuropathy, we keep the patients nonweight bearing for up to 12 weeks and protect them in a cast brace or ankle-foot orthosis for an additional 4 to 8 weeks. Patients are seen in the office initially at weekly intervals and then biweekly to monitor the wound for breakdown and the fracture position radiographically. We maintain the restricted weight bearing based on the postoperative time interval, even if the fracture appears to be healing satisfactorily.

Because diabetic patients usually have poor bone quality, we tend to use heavier plates and plates with locking screws (Figure 2). Finally, although not proven in the literature, we tend toward multiple screws and plates with locking screws. He remained nonweight bearing for 12 weeks and then began protected weight bearing for 6 more weeks (C, D).

Figure 2: A 71-year-old man with a long history of type 2 diabetes, well-controlled on insulin, without significant peripheral neuropathy and with good bone quality and no other significant comorbidities sustained a Weber B type ankle fracture-dislocation (A, B). Fracture fixation was performed using a heavy small fragment plate. The syndesmosis was stable, so we did not use transfibial (syndesmotic) screws. However, the patient was kept nonweight bearing for a full 12 weeks postoperatively, and then partial weight bearing in a brace for an additional 4 weeks (C, D).

Figure 3: A 70-year-old man with long-standing type 2 diabetes, treated with insulin, who had developed peripheral neuropathy sustained a bimalleolar ankle fracture dislocation with comminution of the fibula and disruption of the syndesmosis (A, B). He underwent open reduction of the medial malleolus and plating of the fibular fracture with multiple transfibial screws. He remained nonweight bearing for 12 weeks and then began protected weight bearing for 6 more weeks (C, D).

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