Case Report

Bone Mineral Density During Total Contact Cast Immobilization for a Patient With Neuropathic (Charcot) Arthropathy

Background and Purpose. Diabetes mellitus (DM)-related neuropathic arthropathy of the foot is a destructive bone and joint process. The effect of cast immobilization and non–weight bearing on bone loss has not been well studied. The purpose of this case report is to describe the changes in bone mineral density (BMD) of the calcaneus in the feet of a patient with acute neuropathic arthropathy during total contact cast immobilization. Case Description. The patient was a 34-year-old woman with type 1 DM, renal failure requiring dialysis, and a 7-week duration of neuropathic arthropathy of the midfoot. Intervention included total contact casting and minimal to no weight bearing for 10 weeks, with transition to therapeutic footwear. Ultrasound-derived estimates of BMD were taken of both involved and uninvolved calcanei. Outcome. Bone mineral density decreased for the involved foot (from 0.25 g/cm^2 to 0.20 g/cm^2) and increased for the uninvolved foot (from 0.27 g/cm^2 to 0.31 g/cm^2) during casting. Discussion. The low initial BMD and further loss during casting suggest the need for transitional bracing and a well-monitored return to full activity to minimize the risk of recurrence and progression of foot deformity. [Hastings MK, Sinacore DR, Fielder FA, Johnson JE. Bone mineral density during total contact cast immobilization for a patient with neuropathic (Charcot) arthropathy. *Phys Ther.* 2005;85:249–256.]

Key Words: *Arthropathy; Diabetes mellitus; Foot diseases; Fractures, neurogenic.*

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Neuropathic arthropathy (Charcot arthropathy) is a complication of diabetes mellitus (DM) that results in fractures, dislocations, subluxations, and the potential for progressive deformity of the foot and ankle.[1] A long-term concern is that the resulting foot deformity increases the risk of amputation either by increasing the potential for ulceration that can lead to amputation or by progressive bone and joint deterioration, leaving the foot unstable for weight bearing.[2] Although the cause of neuropathic arthropathy of the foot remains poorly understood, it may be affected by low bone mineral density (BMD) of the foot or ankle and by the presence and severity of peripheral neuropathy (autonomic and somatic).[2–4]

An injury to the foot may initiate an unregulated and prolonged inflammatory response in patients with DM and peripheral neuropathy. A warm, swollen, and reddened foot marks the acute neuropathic arthropathy process. The acute inflammatory response to injury stimulates osteoclast activity[5,6] and is thought to be exacerbated by the presence of autonomic neuropathy, which is regularly seen in patients with neuropathic arthropathy.[7] Autonomic neuropathy results in the inability to constrict blood vessels appropriately in response to even minor trauma.[8–10] Prolonged and unregulated hyperemia in the foot may lead to excessive osteoclastic bone resorption and may result in a decrease in BMD of the foot, which further increases the risk of bone and joint destruction.

Sensory neuropathy allows even minor injuries to go unnoticed; thus, individuals may not decrease their activity levels to allow healing of the injury. Therapeutic intervention often is not sought in a reasonable time frame, which allows the fracture process to persist.[1,2]

An injury to the foot initiates an unregulated and prolonged inflammatory response in patients with diabetes mellitus and peripheral neuropathy.

Individuals with DM and an acute onset of neuropathic arthropathy may have a loss of BMD in the affected foot.[11] The intervention most often prescribed to assist in healing the foot is cast immobilization without weight bearing for 12 weeks.[3,12] This intervention, although effective in resolving the acute inflammation, may result in additional bone loss through disuse.[13] The purpose of this case report is to describe serial measures of BMD of the calcaneus, skin temperature, and edema in a patient with DM, acute neuropathic arthropathy, and renal failure (which required dialysis) during cast immobilization to resolve the inflammatory phase of the arthropathy. From the descriptive data, we speculate that treatment decisions may be influenced regarding: (1) the duration of casting; (2) the need for transitional footwear, including the use of bracing for partial immobilization; (3) the type and progression of rehabilitation required for individuals with diabetes-related complications; and (4) the potential need for supplemental pharmacologic therapy to limit bone loss in order to prevent future foot deformity.

Case Description

Patient Description

The patient was a 34-year-old woman with a 21-year history of type 1 DM. She had a body mass of 75.5 kg (after dialysis), a height of 170 cm, and a body mass

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Dr Hastings, Dr Sinacore, and Dr Johnson provided concept/idea/project design. All authors provided writing and consultation (including review of manuscript before submission). Dr Hastings, Dr Sinacore, and Ms Fielder provided data collection, and Dr Hastings and Dr Sinacore provided data analysis. Dr Hastings and Dr Sinacore provided project management. Dr Sinacore provided fund procurement. Ms Fielder provided the patient. Dr Sinacore and Ms Fielder provided facilities/equipment. The authors thank Bob Koger, BS, for assistance in data collection and Dwight A Towler, MD, PhD, from the Division of Bone and Mineral Diseases at Washington University School of Medicine for the use of the bone sonometer.

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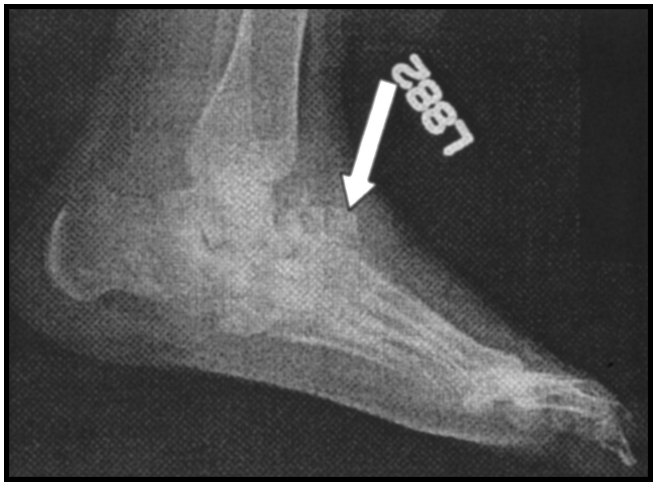

Figure 1.
Radiograph with arrow demonstrating fractured and displaced navicular bone. The fractures of the second and third metatarsals are not apparent in this view.

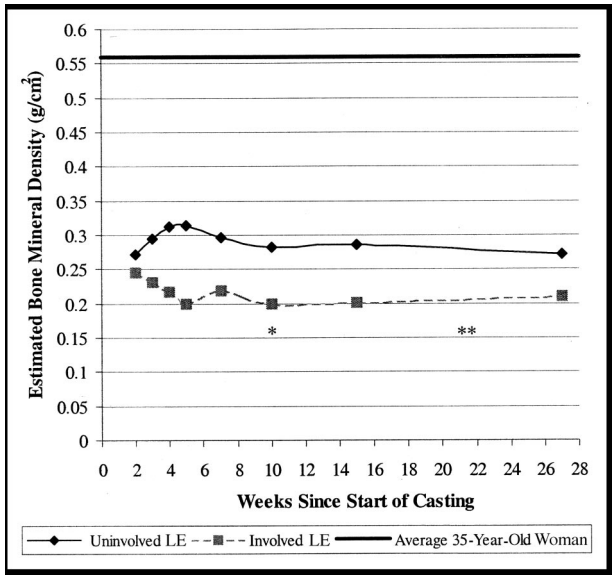

Figure 2.
Estimated bone mineral density for the uninvolved and involved lower extremities (LEs) during cast immobilization. Asterisk denotes the end of casting and transition into Charcot restraint orthotic walker. Double asterisk denotes the transition into Gillette calf lacer with bilateral metal uprights.

index of 26 kg/m^2. She was evaluated by an orthopedic surgeon (JEJ) 7 weeks after the sudden onset of edema and redness in her left foot. She recalled no trauma to her foot and had been hospitalized by her primary care physician to rule out a foot infection 3 weeks prior to her visit with the orthopedic surgeon. The evaluation by the orthopedic surgeon included standard-view radiographs that revealed fractures of the left navicular bone and the second and third proximal metatarsals of the left foot (Fig. 1). Her medical history included factors that may have influenced her fracture healing and intervention, including amenorrhea for the previous 1½ years and 1 year on renal dialysis (approximately 9 hours per week). She had other chronic DM complications, including retinal damage with partial vision in her right eye and glaucoma affecting the left eye, resulting in complete vision loss. She had no history of foot ulcers, cardiac complications, or peripheral vascular disease.

Tests and Measures

The patient consented to participate in this case report before the examination and signed a consent form approved by the Washington University School of Medicine Human Studies Committee. Testing was not completed at the initial visit or at the first follow-up visit 1 week later due to administrative error. The patient was tested at weeks 2 to 5, 7, 8, 10, 15, and 27 from the start of the intervention. Not all tests were completed at every visit; testing frequency is described for each measure. All measurements were obtained in the morning prior to her dialysis.

Peripheral neuropathy was assessed on the first visit using Semmes-Weinstein monofilaments. The method of sensory testing followed a previously described technique.[14] In a study by Diamond et al[15] using this method to assess sensation in 31 subjects, the kappa values for interrater and intrarater reliability ranged from .72 to .83. Sympathetic autonomic function was assessed by comparing blood pressure measurements taken with the patient positioned supine with measurements taken immediately after she stood. A decrease in systolic blood pressure greater or equal to 30 mm Hg is considered abnormal and indicative of autonomic dysfunction.[16]

Bone mineral density of the calcaneus was measured using the Sahara Clinical Bone Sonometer.* Bone mineral density was measured at all test occasions except week 8 (Fig. 2). The right foot was measured and then the left foot; then each foot was measured again, and the average was calculated for each foot. The reliability and precision of the quantitative ultrasound estimates of BMD have been described previously.[17–19] The coefficient of variation (CV) from 8 measurements over a 4-week period on a cadaver foot phantom was 2.2% for estimated BMD.[17] The intraclass correlation coefficient (ICC [3,1]) calculated from test-retest measurements at a 1-week interval on 20 subjects without known pathology or impairments was .97 for estimated BMD.[17] The manufacturer of the Sahara Clinical Bone Sonometer reported the average test-retest measurement difference for estimated BMD to be 0.014 g/cm^2.[20] We assessed the test-retest variability of estimated BMD for the foot of a cadaver over 4 weeks for our sonometer, and it was 0.002 g/cm^2. The test-retest standard errors of the

* Hologic Inc, 35 Crosby Dr, Bedford, MA 01730.

measure (SEMs) for 20 subjects (13 female, 7 male) within 10 days were 0.002 g/cm^2 for the right foot and 0.003 g/cm^2 for the left foot. A T score is calculated by the sonometer. The T score is the number of standard deviation units the patient's BMD varies from the mean of a reference population of 20- to 29-year-old women without known pathology or impairments (ie, the age range of maximum BMD accrual).[20]

Skin temperature was measured using a hand-held infrared dermal thermometer (Dermatemp Model DT 1001[†]) and was used as an indicator of inflammation. The methods used for temperature measurement were similar to those reported by Armstrong and Lavery.[21] Temperature was measured approximately 15 minutes after the footwear and cast were removed. The average room temperature was 22.8°C (SD=1.1°C) during the testing occasions. Skin temperature was measured at 8 locations on each foot: dorsum of the foot; plantar surfaces of the first, third, and fifth metatarsal heads; medial midfoot; lateral midfoot; heel; and site of the Charcot arthropathy (navicular bone). The 8 sites were assessed consecutively, with measurements taken 3 times, and the average was calculated for each site. Only the data from the navicular bone (site of one fracture) will be reported because it was representative of the temperature changes that occurred over time in the other 7 sites. In order to address normal temperature fluctuations, the uninvolved foot was used as the control and temperature difference was calculated by subtracting the temperature of the uninvolved foot from the temperature of the involved foot. The thermometer measures in increments of 0.1°C and is accurate to within ±0.1°C.[22] We have found the measurement of skin temperature at a specific site to be stable. Our average CV among 3 trials on a single test occasion at one site was 0.5%. Skin temperature was measured at all test occasions (Fig. 3).

Edema, also an indicator of inflammation, in the foot and ankle was measured using a water displacement method with a foot volumeter (model 7484[‡]). Water was poured into the volumeter. The patient was sitting in a chair, and her foot was placed into the volumeter with the knee flexed to 90 degrees, and the ankle was maintained as close as possible to 0 degrees of dorsiflexion. The amount of water displaced (in milliliters) for each foot was recorded. Volume was measured twice for each foot, and the average was calculated for each foot. In the hand, the volumeter has been found to be accurate to within 1% of the measured volume when used according to the manufacturer's directions.[23] Petersen et al[24] reported ICCs of .99 for interrater reliability and .98 to .99 for intrarater reliability for foot water volumetry. The water temperature was not measured but was considered cool (tap water). Water temperature has been reported to not alter volume measurement.[25] Volume measurements were completed during the initial 8 weeks of casting. Clinical observation, verified by minimal change in volume measurements near the end of casting, indicated little value in continuing with this measure of inflammation after the eighth week of casting.

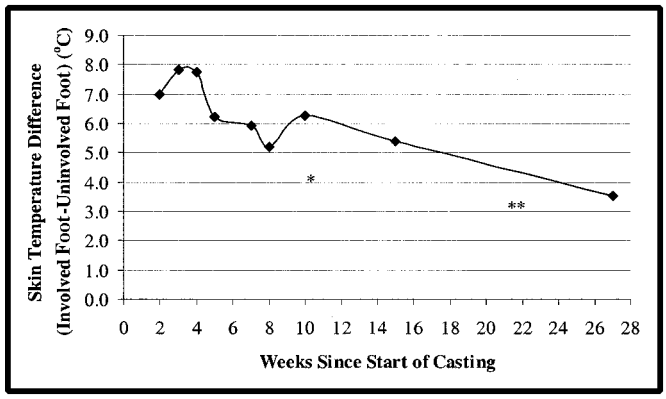

Figure 3.
Skin temperature difference at the navicular bone (temperature of involved foot − temperature of uninvolved foot) during intervention. Asterisk denotes the end of casting and transition into Charcot restraint orthotic walker. Double asterisk denotes the transition into Gillette calf lacer with bilateral metal uprights.

Intervention

The patient's foot was placed in a total contact cast (TCC) as previously described,[26] except the distal end of the toe box was left open and a standard rocker cast shoe was used rather than a walking heel. The patient was instructed to limit weight-bearing activities on her left foot, placing her foot down only for balance, and to use crutches, without weight bearing when walking. No attempt was made to determine the amount of weight-bearing activity that occurred on the involved foot, and force and pressure were not measured inside the cast. The cast was changed weekly until the swelling stabilized (5 weeks). Stabilization of swelling was determined by reports from the patient that she had no excessive movement within the cast during the previous week and from a reduction in the volume change compared with the previous 2 cast changes. The cast was changed twice during the remaining 5 weeks of immobilization.

The patient transitioned into a Charcot restraint orthotic walker (CROW)[27] at 10 weeks and into a Gillette calf lacer with bilateral metal uprights attached to a shoe with a total contact insert at 22 weeks[28] (Fig. 4). She remained in the Gillette calf lacer for 36 weeks. Overall, the patient was partially immobilized and her footwear provided continued partial pressure off-loading more than 1 year. Skin temperature and BMD measurements

[†] Exergen Corp, 15 Water St, Watertown, MA 02472.
[‡] Sammons Preston Rolyan, 4 Sammons Ct, Bolingbrook, IL 60440.

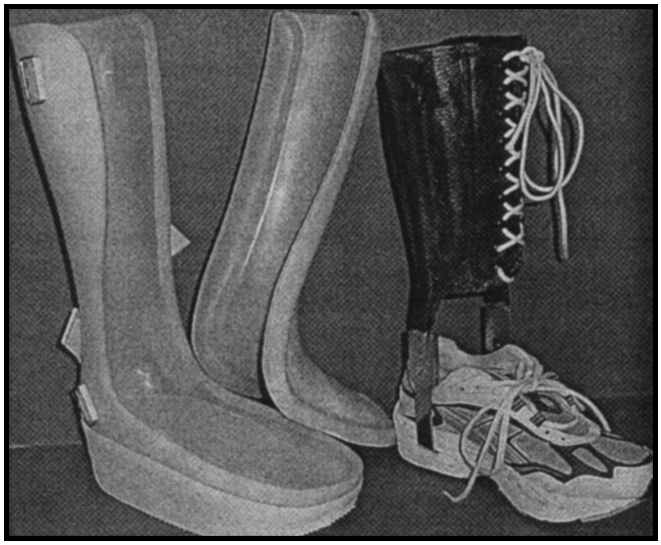

Figure 4.
Charcot restraint orthotic walker (left) and Gillette calf lacer with bilateral metal uprights attached to a shoe with a total contact insert in the shoe (right).

were taken approximately 1 month after each footwear transition.

Outcomes

The patient was unable to feel the 6.10 (75-g) monofilament on the plantar surface of either foot or on the distal two thirds of either leg, indicating an absence of sensation in both feet. The patient's sympathetic autonomic function was impaired because her systolic blood pressure dropped 40 mm Hg from the supine to the standing position.

Estimates of BMD are shown in Figure 2. The BMD was 0.25 g/cm^2 initially for the involved foot, and it decreased to 0.20 g/cm^2—a 50-mg/cm^2 decrease—during the initial 5 weeks of casting. Bone mineral density remained reduced during the final 5 weeks of casting, with a slight trend toward return to baseline values during the 17 weeks following total contact casting. For the uninvolved foot, BMD was 0.27 g/cm^2 initially, and it increased to 0.31 g/cm^2—a 40-mg/cm^2 increase—during the initial 5 weeks of casting. The BMD gradually declined for the uninvolved extremity during the remaining 5 weeks of casting and returned to the baseline value at 27 weeks from the start of intervention. The T scores during the fifth week of casting were −2.75 (ie, 2.75 standard deviations below the reference population mean) for the uninvolved foot and −3.45 (ie, 3.45 standard deviations below the reference population mean) for the involved foot.[29]

The skin temperature difference between the right and left navicular bone areas initially was elevated a maximum of 7.8°C (Fig. 3). The temperature difference between sides decreased to 3.5°C at the affected navicular bone 27 weeks after casting and partial immobilization with the CROW and the Gillette calf lacer.

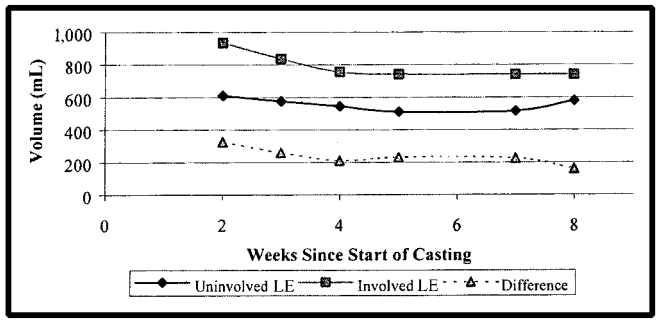

Figure 5.
Absolute foot and ankle volume measurements for the uninvolved and involved lower extremities (LEs) and the difference between sides during cast immobilization.

Edema decreased during the period of cast immobilization. The decrease in volume was most pronounced during the first 4 weeks of casting (volume difference between sides decreased 115 mL from week 2 to week 4 of casting). The volume difference between sides decreased an additional 50 mL from week 4 to week 8 of casting (Fig. 5).

Discussion

The purpose of this case report was to present serial measures of BMD during intervention for neuropathic arthropathy. The measurements of BMD in this individual suggested increased bone loss during the casting period. During cast immobilization without weight bearing on the involved foot, BMD measurements decreased. Previous evidence supports a 1% to 2% decrease in BMD per month in people without known pathology or impairments during weightlessness in space.[13] The decrease in BMD of 20%, a 50-mg/cm^2 decrease, over a 5-week period of time suggests the influence of several synergistic factors contributing to bone loss.

Factors that most likely resulted in a larger-than-expected change in BMD measurements during the first 5 weeks of casting were the persistent inflammation and hyperemic osteolysis due to autonomic neuropathy.[30] Signs of inflammation (increased temperature and edema) persisted on the involved side throughout the intervention period. However, the amount of bone loss due to immobilization and non–weight bearing versus that attributed to autonomic hyperemia cannot be distinguished in this case report. Future studies that include a control group may assist in distinguishing the contribution of inflammation-induced hyperemic osteolysis from bone loss caused by cast immobilization and non–weight bearing. Certainly, additional research is needed to discern the effect of immobilization and non–weight bearing in humans as well as the role of

inflammation contributing to the magnitude of the bone loss.

The normative BMD value for the calcaneus of a 35-year-old woman without known pathology or impairments is 0.56 g/cm² (SD=0.11).[20] The total bone loss in both feet of this individual raises concern. According to the World Health Organization criteria, an individual with a T score of 1 to 2.4 standard deviations below the reference population's mean is considered to be osteopenic, whereas an individual with a T score equal to or greater than 2.5 standard deviations below the mean is considered to be osteoporotic.[29] Both of this patient's feet were classified as osteoporotic.

Osteoporosis is most often thought to be a disease of older individuals. Because of this patient's young age, a clinician might not consider regional osteoporosis to be a major concern. However, a number of risk factors, other than age, may contribute to bone loss. This individual had a 1½-year history of amenorrhea, a long duration of type 1 DM with evidence of microvascular and autonomic complications, and renal failure requiring dialysis that may have contributed to her bone loss.

In several studies, people with type 1 DM were found to have a 6% to 12% decrease in BMD at all sites measured in the femur,[31–34] leg,[33] and foot.[35] However, determining the contribution of type 1 DM alone is difficult because most studies did not exclude participants with multiple complications. Impaired renal function is another complication of DM that affects BMD. Chronic renal failure and renal dialysis have been associated with renal osteodystrophy.[36–38] Peretz et al[38] reported that the average calcaneal BMD in people who received hemodialysis was 17% less than that of control subjects. The synergistic effects of multiple complications related to DM and renal dialysis on BMD need to be studied.

Skin temperature was elevated for the involved foot compared with the uninvolved foot, consistent with an acute inflammatory response. The maximal skin temperature difference we observed (7.8°C) was similar to the temperatures reported by Armstrong and colleagues[21,39] for subjects with acute neuropathic arthropathy (range=5.1°–14.7°C). The maximum decrease in skin temperature difference for the left navicular bone during the 70 days of casting was 2.6°C, from week 3 to week 7. The decrease in skin temperature was expected because the inflammatory process resolved, and the decrease was similar to that reported by McCrory et al[40] (2.1°C [SD=0.5°C] in 100 days of casting).

A number of factors support keeping an individual in the cast longer than 10 weeks. Armstrong and Lavery[1] suggested a temperature difference between sides of 1° to 2°C as a criterion for transitioning out of the cast into a removable walker boot. Our finding of low BMD also suggests that a longer cast duration might be necessary to stabilize the arthropathy process and heal the fractures. However, the decrease in temperature over time in our patient managed with early transition to bracing was similar to that advocated by Armstrong and Lavery,[21] who managed patients with TCC over the same period of time. This finding may indicate that low BMD does not necessarily warrant a longer duration of casting. Perhaps an earlier transition to bracing may be safe in cases where the patient adheres to wearing the brace during all weight-bearing activity.

Volume displacement measurements also demonstrated a steady decline over the first 4 weeks of casting. The reduction in foot edema provided additional evidence for the gradual resolution of the inflammatory process. Additionally, measuring volume changes in the leg and foot can provide guidance as to the length of subsequent cast changes. Large reductions in volume while wearing a TCC can result in the leg and foot becoming excessively loose within the cast. The leg and foot may rub the inside of the cast, increasing the chance for a friction-induced ulcer, a concern in a limb with impaired sensation. Small reductions in volume between cast changes may be an indication that subsequent casts can be worn for a longer duration with less risk of complications.

The measurement of BMD using quantitative ultrasonometry is not yet a standard clinical measure for physical therapists. However, the clinical implications of finding low BMD in both feet, evidence of renal osteoporosis, and a further decrease in BMD during cast immobilization and non–weight bearing are important for physical therapists to recognize.

The finding of low regional BMD in the foot and the temperature findings over the 7-month follow-up period did not indicate the need for a longer duration of casting to resolve the neuropathic arthropathy process. At no time during the 7-month follow-up did the individual's foot show signs of recurrence of the arthropathy process (increase in temperature, redness, further bony destruction or deformity, or swelling).

The patient's positive outcomes suggest that, with appropriate caution, a gradual progression from complete cast immobilization to partial immobilization with a CROW and a Gillette calf lacer with bilateral metal uprights can adequately protect the foot during the healing process. The ability to safely decrease time in a cast and transition to bracing has many important implications, including improved patient comfort and hygiene and minimizing

the time and cost burdens associated with the regular clinic visits required for cast changes.

Physical therapy is often prescribed for individuals with the complications described in this case report. They can be referred for physical therapy during the initial transition from cast to CROW to assist with a gradual and well-monitored return to full weight bearing and regular activity level or for unrelated reasons, for example, in the hospital after a surgical intervention. Physical therapists need to recognize the high risk of excessive bone loss in this patient population and modify the exercise intervention appropriately. We believe the intervention should be conservative (low impact) and well-monitored to ensure the safe return of the individual to full and unrestricted activity while minimizing the risk of arthropathy occurrence and recurrence and preventing progressive foot deformities.

Rapid and profound loss of bone, as our patient demonstrated, may warrant prompt alerting of the person's primary care physician for further evaluation and the possible need for additional intervention. Some individuals may benefit from supplemental pharmacological agents (eg, bisphosphonate or selective estrogen receptor modulators) intended to limit the resorptive bone loss and prevent regional and systemic osteoporosis to reduce the likelihood of additional low-trauma fractures.

Conclusion

The gradual decrease in skin temperature and edema during intervention supports the use of total contact casting to resolve the acute inflammatory arthropathy. This case also shows that individuals with DM and DM-related complications (neuropathic arthropathy, renal failure requiring dialysis, and amenorrhea) can have serious bone loss, regardless of age, and that BMD may be further reduced by immobilization and non–weight bearing. A careful history and special attention to their medical diagnoses can be important when formulating and progressing intervention plans.

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