CHAPTER 37

TOTAL CONTACT CASTING

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Each year, more than half a billion dollars is spent in the United States on amputation secondary to diabetes mellitus.[1] It has been reported that each year 2.5% of diabetics will develop a foot ulcer. Treatment of pedal complications accounts for 20% to 25% of all hospital admissions of diabetic patients. Recent estimates show that the diabetic patient population is responsible for 30,000 to 50,000 major lower extremity amputations per year.[2] The diabetic patient is 15 times more likely to have an amputation than the non-diabetic patient.[3] Once an amputation has been performed, statistics show that the patient has a 50% chance of developing a serious lesion on the contralateral limb within 2 years. It is estimated that foot care and patient education programs can reduce lower extremity amputation rates by 40% to 50%, largely by reducing the frequency and severity of foot ulcers. With the radical changes occurring in the health care system, the challenge of cost-effective management of lower extremity problems in diabetics is increased.[4]

Ischemia, neuropathy and an altered response to infection form the triad of complications, usually initiated by minor trauma, which leads to ulceration. The two most common types of ulcers observed in diabetics are neuropathic and ischemic. Mixed ulcers may occur in a patient with both peripheral neuropathy and peripheral vascular disease.

Neuropathic ulcers are produced from repetitive pressure and shear forces on a foot with decreased or absent foot sensation. Although more commonly seen with diabetes, morphologically similar ulcers can be associated with other neuropathies such as familial dysautonomia leprosy, tabes dorsalis, and various neurological syndromes.[5] Neuropathic ulcers are characteristically located over pressure points on the foot, especially under the first and fifth metatarsal heads and great toes.

Diabetic peripheral neuropathy is a symmetrical polyneuropathy occurring in a stocking and glove distribution, and is the most common cause of foot ulcers. Neuropathy may affect sensory, motor and autonomic nerves.

Pain and temperature are usually the first sensations to be affected by sensory neuropathy.[6] As a result, the diabetic patient will not be aware of the warning signals of early inflammation and continued tissue damage, and ulceration will occur as the patient continues walking on a pressure point. Motor neuropathy leads to denervation of small muscles in the foot followed by the extrinsic muscles. Muscles weaken and waste, resulting in hammering and/or clawing of the digits, pes cavus, and prominent metatarsal heads with anterior displacement of the protective plantar fibro-fatty padding. This leads to increased weight-bearing pressure under the metatarsal heads, particularly under the first metatarsal head, with associated plantar callus formation. This hyperkeratotic tissue has low tensile strength. When external pressures exerted on this callused tissue exceed capillary pressure for prolonged periods of time, microvessel hemorrhaging with subsequent tissue necrosis occurs.[7]

When peripheral autonomic nerves are affected by neuropathy, the vasomotor tone of the blood vessels is impaired, resulting in local vasodilatation, osteoporosis and disruption of nutrient flow to tissues. Autonomic nerve involvement accounts for the dryness, scaling, and fissuring associated with decreased sweating and autosympathectomy. In addition, patients with sympathetic neuropathy can develop Charcot foot, a condition with bone resorption and fracturing, leading to significant foot malformation and unusual bony prominences. These complications result in the formation of plantar hyperkeratotic skin lesions, increased skin temperature, increased prominence of dorsal veins and decreased nutrition to capillary beds.[2] The low tensile strength of these plantar hyperkeratotic lesions, combined with increased loading and shear forces created during weight bearing facilitate the formation of pedal ulcerations.

The measurement of pressure sensation is considered an appropriate way to identify individuals at risk for ulceration because unperceived pressure is a major mechanism of injury in the diabetic foot.[8] The Semmes-Weinstein monofilaments are one of the most objective tests

for measuring cutaneous pressure sensation because they consistently reflect gradual decreases in sensory nerve functioning.

Approximately 45% of all diabetics will develop peripheral vascular disease within 20 years of diagnosis.[9] There is a higher incidence of arterial disease in diabetic patients than in non-diabetic patients, particularly in the vessels below the knee (tibial and peroneal arteries), usually with sparing of the arteries of the foot. Intermittent claudication of the calf secondary to this may be masked by neuropathy until minor trauma results in ischemic ulceration. Signs and symptoms of vascular disease in the diabetic foot include intermittent claudication, cold feet, night pain in toes when resting, absent to diminished pulses, blanching on elevation, delayed venous filling time, dependent rubor, atrophy of subcutaneous fatty tissues, loss of hair on the foot and toes, thickened nails, and gangrene.[2] Determination of the venous filling time is more important than simple evaluation of capillary filling, which, because of autonomic neuropathy, may remain normal despite ischemia.[4]

HISTORICAL PERSPECTIVE

Historically, walking casts were used in Ceylon in the early 1930s to treat the neuropathic ulcers encountered in Hansen's disease. In the 1950s in India, Dr. Paul Brand used a modified technique, molding the plaster cast to the shape of the leg and foot without the use of padding. Experiences of other clinicians had shown that the padding in the cast would compress over time and the cast would begin to slip and cause new ulcers. Brand noted an impressive regularity and rate of healing, which supported the concept that the ulcer is directly related to mechanical stress. Since the 1960s this method has been employed and popularized at the Gillis W. Long Hansen's Disease Center in Carville, Louisiana, initially in the care of ulcers related to Hansen's disease, but now also for ulcers associated with diabetes and other disorders with insensitive feet.[10]

THE CAST

The total contact cast functions primarily by reducing pressures on the plantar surface. Pressures are redistributed over the entire surface of the foot. This increases the effective plantar surface area, relieving the excessive pressure responsible for the ulcerations. Total contact casting necessitates a shortening of the patient's stride length and decreasing gait velocity, hence diminishing the vertical forces on the foot.[11] The cast effectively creates the most suitable environment required to decrease microtrauma from shearing forces between shoes and the foot. By redistributing forces, the healing of ulcers is expedited, and redistributing the forces ensures long term protection of subcutaneous tissues and skin.[12] Additionally, the total contact cast prevents sagittal plane motion at the ankle joint, thus eliminating propulsion during the gait cycle. With cessation of forefoot propulsion, ulcers located primarily in the submetatarsal region, tend to heal faster.

Apart from limiting motion and redistributing pressures, the cast protects the foot from foreign objects. The custom fit of the cast, with complete enclosure of the toes, acts as a structural barrier against entry of environmental debris such as stones, etc. This is significantly beneficial since the diabetic foot is typically insensate and unable to detect the presence of foreign objects. Immobilization caused by the cast enables localization of any minor infection, and prevents spread to adjacent tissues. Also, the casting material absorbs any exudate to preserve a dry and clean environment conducive to wound healing.

Another adjunct advantage with the cast is the ability to drastically control and reduce edema. Uncontrolled edema increases hydrostatic pressure, resulting in enzymatic tissue necrosis and subsequent ulcerations. Better fluid exchange in the limb is facilitated by the cast through a mechanism similar to "support stockings in venous stasis and lower extremity edema."[12] The drastic reduction in edema often necessitates a cast change within the first 48 hours. Otherwise the cast will slide on the leg, which increases the shear force, potentially leading to further soft tissue damage.

Use of total contact cast is not limited to the treatment of plantar neuropathic ulcerations. It may be successfully used as a treatment modality for neuropathic fractures and for postoperative immobilization. Inappropriate usage of the total contact cast can cause significant damage to an already immunocompromised patient. The major contraindication to the use of the total contact cast is the presence of an active infection. Also, the ulcer must be wider at the surface than it is deep. Failure to adhere to this requirement would

result in epithelialization of the surface before the wound has a chance to granulate, resulting in possible abscess formation. Other contradictions to the use of total contact casting include active dermatitis, fragile skin, claustrophobia, extreme obesity, noncompliance, blindness, excessive lower extremity swelling and Doppler pressures less than 0.4.

CASTING

Application of the cast requires skill and proper technique. Materials needed to fabricate the total contact cast are listed in Table 1. Before the decision is made to use a total contact cast, the ulcer must be evaluated and all necrotic and hyperkeratotic tissue must be debrided. Before application of the cast, a piece of exposed x-ray film and felt pen are used to mark the outline of the ulcer to serve as a reference for measuring the progress of healing. A sterile dressing is applied to the ulcer site and secured with paper tape.

1. The patient lies in a prone position with the knee and ankle bent at 90 degrees. This position prevents edema formation in the foot and also allows relaxation of the gastrocnemius muscle.
2. Cotton padding is placed between the toes to prevent interdigital maceration.
3. A stockinette is pulled over the foot and ankle, enclosing the toes distally. The toe region is covered with foam rubber padding. Folds in the stockinette at the ankle are cut and taped to prevent wrinkles between the plaster and the patient's skin.
4. Oval felt pads are skived and placed over the malleoli and other bony prominences.
5. A felt strip is skived and is placed from the top of the casted area to the toes and over the tibial crest and dorsum of the foot. This prevents the cast from rubbing on the bony prominences and also enables safer removal.
6. Self-adhesive polyurethane foam is applied directly over the ulcerated area.
7. Plaster is then applied to make the inner shell of the cast. It is applied loosely and carefully over the leg to prevent wrinkle formation. Small backfolds are incorporated with each wrap around the leg and foot. This application allows the plaster to be easily molded over bony prominences and into crevices. This inner shell is applied from a level about 2 cm distal to the head of the fibula to the level of the toe sulcus distal to the metatarsal heads. Several layers are applied, allowing the inner layer to dry before subsequent applications. While the plaster is setting, it is constantly being molded (by rubbing) to the exact contour of the foot and leg. The inner shell molds better when a fast setting Gypsona plaster bandage is used.
8. The remaining cast is built with plaster to give strength and support to the inner shell.
9. Splints are placed along the posterior aspect of the leg, on the bottom of the foot and along the sides for additional strength. Extreme care should be taken to prevent wrinkling of the inner shell.
10. A 1/4 inch plywood board, cut smaller than the patient's foot, is applied over the plantar surface of the cast.
11. A rubber-walking heel is placed over the board immediately behind the midfoot. The area between the board and the cast is filled with a wet plaster roll.
12. Plaster or fiberglass incorporates the board and walking heel into the cast and reinforces the cast.

The patient is instructed to limit ambulating for the first 24 hours in order to give the deeper layers sufficient time to dry. The patient must be provided with crutches to aid ambulation. To prevent abnormal curvature of the spine, it is imperative to modify the contralateral shoe sole to equilibrate the height of the cast off the ground.

The first cast is left on for 3 to 7 days, depending on the fit of the cast to the foot and leg. Since the total contact cast removes dependency edema from the leg, the first cast will quickly become loose, and no longer conform to the lower extremity. The next cast is applied in the same manner and left on for up to 6 weeks.

Careful monitoring of the cast and the patient, and progression of ulcer healing are key factors for the success of total contact cast in treating plantar neuropathic ulcers. Total contact casts should be strongly considered as a component of the treatment armamentarium at every multidisciplinary center dedicated to salvage of the diabetic foot.

Table 1

MATERIALS REQUIRED FOR TOTAL CONTACT CAST

Exposed X-ray film that has been sterilized
Felt tip marking pen
Scissors
Cotton cast padding
1/8 inch felt strip 2 inch by 20 inch
1/8 inch felt oval pads
Self adhesive polyurethane foam 1/4 to 1/2 inch thick
2 rolls of 4 inch fast-setting creamy plaster i.e. Gypsona
2 rolls of 5 inch standard fast setting plaster i.e. Specialist
2 sets of 5 inch plaster splints (5 layers thick)
1/4 inch oval plywood
Rubber walking heel
2 rolls of 4 inch fiberglass casting tape

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