Diabetic Peripheral Neuropathy

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KEYWORDS

- Neuropathy Peripheral neuropathy Diabetes mellitus
- Lower extremity amputation

Peripheral neuropathy is defined as any disorder of the somatic or autonomic nervous systems. An appreciation of this disease entity becomes essential to orthopedic surgeons when they care for lower extremity disease or injury in the diabetic population. Whereas most types of peripheral neuropathy cause pain or impaired sensation in the extremities, when present in diabetic patients it often leads to the development of foot ulcers and infection, neuropathic (Charcot) arthropathy with subsequent deformity, and impaired immunity in healing lower extremity wounds or injury. Foot ulcers, infections, and deformity are some of the major sources of mortality and morbidity among the diabetic population. At any point in time, 3% to 4% of the diabetic population will have a foot ulcer or infection. Fifteen percent of individuals with diabetes will have a foot ulcer in their lifetime, and foot ulcers precede 85% of lower extremity amputations in diabetic patients.¹⁻⁷ In 2004 there were 71,000 lower extremity amputations in diabetic patients in the United States. Eighty-five percent were preceded by a diabetic foot ulcer.^{1,8–10} Once amputated, the 2-year mortality has been reported to be as high as 36%.^{11–14} These patients are at greater risk for premature death, even if they do not undergo an amputation.^{11,15} An International Consensus on the Diabetic Foot estimated that in 1999, the direct cost of a diabetes-related lower extremity amputation was US\$30,000 to \$60,000. The long-term (3-year) cost has been estimated to be as high as \$60,000 for home care and social services, and the overall cost to the United States economy has been estimated to be 4 billion dollars per year.^{16,17}

DEFINITION

Diabetic peripheral neuropathy is the most destructive type of form of peripheral neuropathy encountered by the orthopedic surgeon. It is defined as "peripheral, somatic or autonomic nerve damage attributable solely to diabetes mellitus."¹⁸ The extent of peripheral neuropathy present in a specific patient is based on electromyography and nerve conduction studies. The threshold of peripheral neuropathy of

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Foot Ankle Clin N Am 16 (2011) 345–349 doi:10.1016/j.fcl.2011.01.002 1083-7515/11/\$ – see front matter © 2011 Elsevier Inc. All rights reserved.

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importance to the orthopedic surgeon is insensitivity to the Semmes-Weinstein 5.07 monofilament (**Fig. 1**). Failure to "feel" the 10 g of pressure applied by this nylon fiber is one of the most important risk factors for the development of diabetes-associated foot morbidity. Electrodiagnostic studies are rarely necessary for the orthopedic surgeon to assist in either diagnosis or treatment. While sensory neuropathy is the easiest to identify, its presence should alert the physician to the presence of similar levels of motor and vasomotor neuropathy.

EPIDEMIOLOGY

The Rochester Diabetic Neuropathy Study suggested that as many as 65% of individuals with type 1 or type 2 diabetes had some evidence for peripheral neuropathy.¹⁹ Clinical screening with the Semmes-Weinstein monofilament has detected an incidence of approximately 1 in 4.^{3,5,20,21} Multiple investigators have demonstrated that the presence of clinical peripheral neuropathy is the most predictive risk factor for the development of diabetic foot ulcers (the precursor to lower extremity amputation), foot infection, and the development of Charcot foot arthropathy.^{1–6,8}

RISK FACTORS FOR THE DEVELOPMENT AND PROGRESSION OF DIABETIC PERIPHERAL NEUROPATHY

The two predictors for the development, progression, and severity of diabetic peripheral neuropathy are duration of diabetes and metabolic control. The presence of endorgan disease such as nephropathy, proliferative retinopathy, and cardiovascular disease may be a common end point of the process that leads to the development of the neuropathy.²¹ The roles of excessive alcohol consumption and smoking are less clear.^{22–24} Genetic factors may also play a role in the susceptibility to the development of diabetic peripheral neuropathy.

PATHOPHYSIOLOGY

Several mechanisms have been proposed for the development of peripheral neuropathy, but none has gained widespread acceptance. There is significant support for both vascular and metabolic origins for the genesis of this common end point.



Fig. 1. The Semmes-Weinstein 5.07 monofilament. Applying the 5.07 monofilament to the pulps of the toes applies 10 g of pressure to the skin. Failure to perceive this amount of pressure is a key risk factor for developing all of the diabetic foot morbidities.

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