Outpatient Assessment and Management of the Diabetic Foot

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KEYWORDS

• Diabetes mellitus • Peripheral neuropathy • Charcot arthropathy • Ulceration

KEY POINTS

- Patients with diabetes are at risk for the development of peripheral neuropathy.
- Peripheral neuropathy, when associated with a traumatic event, can lead to Charcot (neuropathic) arthropathy.
- Charcot arthropathy often leads to significant deformity of the ankle and hindfoot.
- Deformity due to neuropathic arthropathy when associated with the insensate foot puts the patient at significant risk for ulcer formation.
- Neuropathic changes in the foot and ankle are best initially managed with immobilization. This immobilization protects the foot from injury, allowing for the process to develop while minimizing further progression.
- For unstable deformities or neuropathic ulcers, surgical correction of these deformities may be required. Urgent referral should be made to the foot and ankle specialist for those individuals with an ulcer or in whom Charcot arthropathy is suspected.

INTRODUCTION

Diabetes is characterized by high blood glucose. Individuals with high blood sugars fall into one of 2 categories. Type I diabetes is characterized by an inability to manufacture insulin due to autoimmune destruction of the insulin-producing pancreatic beta cells.[1] It represents approximately 5% of all diagnosed cases of diabetes. Exogenous insulin is necessary for survival. It typically is first diagnosed in children and young adults. Risk factors included autoimmune, genetic, or environmental causes. Type II diabetes accounts for 95% of diagnosed diabetes in adults. A well-balanced diet along with exercise and certain prescription medications can help control complications. Diabetes is a major cause of heart disease, vision loss, kidney failure, and lower extremity amputation. The final common pathway to limb loss is peripheral neuropathy, peripheral vascular disease, ulceration, and

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infection. Tight glucose control, as measured by A1C levels, can help prevent these complications.[2]

From 1990 through 2010, the number of new cases of diagnosed diabetes nearly tripled. This rise in incidence is attributed to increases in obesity, decreases in physical activity, and an aging US population.[3] The prevalence during this same time period also increased, and many people are unaware of their undiagnosed diabetes. It is thought that if trends continue, as many as 1 in 3 American adults will have diabetes by 2050.[4]

Medical expenses for a person with diabetes are more than twice as high as those without diabetes. In 2007, the estimated cost of diabetes in the United States was $174 billion. This included $116 billion in direct medical care costs and $58 billion in costs due to disability, productivity loss, and premature death.[5]

The ability to lead a functional life hinges on one's mobility. Managing the sequelae of diabetic foot disease (peripheral neuropathy, Charcot arthropathy, and peripheral vascular disease) is thus essential.

The focus of this review is to define the various manifestations of peripheral neuropathy, the pathophysiology of foot ulceration, neuropathic arthropathy (Charcot arthropathy), their assessment, and initial steps in management. Criteria for referral to an orthopedic foot and ankle surgeon are also discussed.

PATHOGENESIS OF INSULIN-DEPENDENT DIABETES MELLITUS

Insulin-dependent diabetes mellitus (IDDM) is most common in individuals of Northern European descent and less common in African American, Native American, and Asian individuals. These differences may be explained by varied genetic susceptibility in racially distinct populations; however, diet and environmental factors likely play a role.[6] Susceptibility is inherited, and the main gene associated with a predisposition to IDDM is the major histocompatibility complex (MHC) on chromosome 6 in the region associated with the genes encoding for HLA recognition molecules. The interaction to a cell bearing an HLA molecule associated with an antigenic peptide and a T lymphocyte bearing a receptor capable of recognizing the HLA peptide complex triggers the activation and proliferation of T lymphocytes. Susceptibility or resistance to IDDM is associated with different HLA-DR and HLA-DQ genotypes, and 95% of patients with IDDM has at least 1 of these HLA-DR antigens.[1]

IDDM is a chronic autoimmune disease that exists in a preclinical phase. The most consistent histologic finding of the pancreas is the lack of insulin-secreting beta cells. Associated with this is a chronic inflammatory infiltrate. Histologic studies have suggested that an 80% reduction in the volume of beta cells is necessary to induce symptomatic IDDM.[7]

The association of microvascular disease and neuropathy with diabetes and the relationship of these conditions to the duration of diabetes suggest that they are linked to hyperglycemia. The Diabetes Control and Complications Trial (DCCT) demonstrated that the incidence and development of retinopathy, nephropathy, and neuropathy could be reduced by intensive treatment.[2]

The retina, kidney, and nerves are freely permeable to glucose. Increases in blood glucose concentrations leads to increased intracellular concentration of both glucose and its metabolic by-products. The mechanism by which hyperglycemia leads to microvascular and neurologic complications includes the increased accumulation of polyols through the aldose reductase pathway and of advanced glycosylation end products.[8]