Diabetes Mellitus and Peripheral Vascular Disease

Diagnosis and Management

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KEYWORDS

• Diabetes mellitus • Peripheral artery disease • PAD • Peripheral vascular disease

KEY POINTS

- Diabetes and peripheral artery disease (PAD) are both coronary heart disease equivalents and the pathologic cardiovascular processes of one may impact and augment that of the other.
- Be alert for atypical presentations of PAD, as classic symptoms, such as claudication and physical examination findings, may be altered by concurrent presence of diabetes.
- Many noninvasive and imaging studies are available; however, computed tomography angiography is the new standard in assessing PAD and planning for surgical or endovascular intervention.
- Medical management of both diseases includes goal HbA1c lower than 7.0%, blood pressure lower than 130/80, low density lipoprotein lower than 70 mg/dL, and treatment with aspirin and possibly clopidogrel.
- Surgical bypass to revascularize the lower extremities generally requires fewer reinterventions than endovascular therapies, although limb salvage rates appear similar, and both modalities should attempt to restore straight-line pulsatile flow to the foot.

INTRODUCTION: NATURE OF THE PROBLEM

Taken together, diabetes mellitus (DM) and peripheral artery disease (PAD) represent 2 of the fastest growing and most challenging medical-surgical diseases facing the world today. As coronary heart disease risk equivalents, they put patients at significant risk for cardiovascular events and mortality in addition to their other common morbidities, including limb loss. Epidemiologically, they have a quickly growing prevalence in not just developed nations but also developing nations. Acting on peripheral vasculature together, they often present difficult treatment choices from both medical and surgical standpoints. This review provides a brief overview of these disease entities,

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as well as options for their treatment both medically and surgically. Particular attention is paid to the lower extremities of patients with both diseases, as the diagnosis and management in these situations can provide a unique set of challenges.

Epidemiology

The prevalence of DM has risen in the United States at an alarming rate, particularly type 2. Recent Centers for Disease Control and Prevention estimates of the diagnosed prevalence puts 7.8% of the American population with DM, nearly double the prevalence of 4.9% in 1990.[1] This correlates with rising obesity in Americans, now present in 20% of adults. In terms of absolute numbers, 24 million American adults are diabetic in addition to 57 million prediabetic individuals. A rising incidence is noted in the developing world, including China, which has a 1% prevalence of DM but is projected to rise to 8% to 10%.[2]

With regard to PAD, there is an estimated worldwide prevalence of almost 10%, with 15% to 20% of people older than 70 years affected.[3,4] Critical limb ischemia, PAD's most severe manifestation, has an annual incidence of approximately 500 to 1000 per million.[5]

Examining these 2 together, DM doubles the likelihood of developing PAD, and a 1% increase in HbA1c is associated with a 26% increase in risk of developing PAD.[6,7] Furthermore, PAD patients with diabetes are 5–10 times more likely to progress to major amputation compared to PAD patients without diabetes.[8] Despite these morbid statistics, the true prevalence of PAD in diabetic patients has been somewhat difficult to determine, as the reported symptoms and objective findings are often not typical of each of the diseases alone. Many patients may be asymptomatic and others may have an atypical pain pattern due to peripheral neuropathy. The PARTNERS study by general practitioners in diabetic patients older than 50 years used ankle-brachial indexes (ABIs) to diagnose PAD in 29% of patients.[2] Likewise, 50% of patients with diabetic foot ulcers have PAD.[5]

Although exact statistics tend to vary across different studies and populations, none would dispute the association between DM and PAD. Full treatment of one disease will require awareness and optimization of the other.

Pathologic Basis

Diabetes mellitus

The pathophysiology of DM is a highly complex system of both innate and environmental factors that is beyond the scope of this discussion. Briefly, however, DM is classically broken down into type 1 and type 2. Type 1 DM is generally thought of as a chronic autoimmune disease.[9] Indeed, the common pathologic finding is lymphocytic infiltration of the pancreatic islets, with destruction of insulin-secreting beta cells. Of individuals with type 1 DM, 90% are known to have autoantibodies against islets cells, insulin, or glutamic acid decarboxylase; however, the mechanism of these antibodies as cause or effect of type 1 DM remains to be elucidated. As far as its pathogenesis, current models describe an environmental factor triggering an autoimmune response in susceptible patients, which over years leads to destruction of pancreatic beta cells and thereby decreased insulin secretion. Once 80% to 90% of these cells are lost, hyperglycemia may develop and patients will present themselves: 20% with fulminant diabetic ketoacidosis.[2] Over the following months, complete destruction of beta cells renders these patients insulin-dependent.

Type 2 DM, in contrast, is a more heterogeneous and more common entity characterized by insulin resistance. Its association with obesity is well described and accounts for the currently increasing prevalence in young patients in whom obesity