HOSPITAL PHYSICIAN®

INTERNAL MEDICINE BOARD REVIEW MANUAL

Prevention and Management of Common Diabetic Foot Problems; Case Studies in Hypernatremia

Series Editor and Contributor:
Richard J. Simons, MD, FACP

Contributors:
Thomas A. O'Bryan, MD
Gregory M. Caputo, MD
Paul J. Juliano, MD
Jan S. Ulbrecht, MD
Peter R. Cavanagh, PhD
Thomas G. Cody, MD

Table of Contents

Cover Illustration by mb cunney

next page ↓

Series Editor and Contributor Affiliations

Chapter 1—Prevention and Management of Common Diabetic Foot Problems

Series Editor:
Richard J. Simons, MD, FACP
Professor of Medicine, Acting Vice-Dean for Educational Affairs, Staff Physician, Department of Medicine, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

Contributors:
Thomas A. O'Bryan, MD
Assistant Professor of Medicine, Department of Medicine, Staff Physician, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

Gregory M. Caputo, MD
Professor of Medicine, Division of General Internal Medicine, Staff Physician, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

Paul J. Juliano, MD
Associate Professor of Orthopaedic Surgery, Division of Orthopaedics, Staff Physician, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

Jan S. Ulbrecht, MD
Associate Professor of Biobehavioral Health and Medicine, Department of Biobehavioral Health, Pennsylvania State University College of Health and Human Development, University Park, PA

Peter R. Cavanagh, PhD
Distinguished Professor of Biobehavioral Health, Medicine, and Orthopaedics and Rehabilitation, Department of Kinesiology, College of Health and Human Development, Director of Research, Center for Locomotion Studies, Diabetes Foot Clinics, Pennsylvania State University, University Park, PA, Milton S. Hershey Medical Center, Hershey, PA

Chapter 2—Case Studies in Hypernatremia

Series Editor and Contributor:
Richard J. Simons, MD, FACP
Professor of Medicine, Acting Vice-Dean for Educational Affairs, Staff Physician, Department of Medicine, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

Contributor:
Thomas G. Cody, MD
Resident, Internal Medicine and Pediatrics, Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA

next page

Chapter 1—Prevention and Management of Common Diabetic Foot Problems

Thomas A. O'Bryan, MD, Gregory M. Caputo, MD, Paul J. Juliano, MD, Jan S. Ulbrecht, MD, and Peter R. Cavanagh, PhD

I. INTRODUCTION

Lower extremity complications in patients with diabetes are a substantial cause of morbidity, mortality, and healthcare expenditures. Of lower extremity amputations among people with diabetes, it is estimated that at least 50% of the more than 80,000 annual amputations in the United States are preventable.[1] Early identification of the patient at risk is the essential first step in prevention. **Foot ulcer, a common problem seen in internal medicine practice, precedes most amputations.**[2] Yet, foot ulcers are often preventable by regular inspection, proper foot care, and appropriate footwear. It is essential that primary care clinicians be knowledgeable in prevention and initial management of foot problems in the diabetic patient (termed the diabetic foot). In this article, 4 case patients are presented to highlight management of diabetic foot conditions, including neuropathic ulcers, foot infections, peripheral vascular disease, and Charcot foot.

II. SCREENING AND PREVENTION FOR DIABETIC FOOT PROBLEMS

SCREENING GUIDELINES

The American Diabetes Association (ADA) recommends that every diabetic patient receive monofilament testing of the feet to assess for loss of protective sensation (LOPS) and that they have their foot pulses checked yearly.[3] Monofilament testing is performed by pressing the monofilament against the skin as shown in **Figure 1**. Callused portions of the foot should be avoided because sensation is diminished in these areas. If the monofilament is felt at each place, the patient is said to have intact protective sensation. Conversely, failing to feel the monofilament at just one site is considered diagnostic of LOPS. When the monofilament testing and pulses (ie, dorsalis pedis and posterior tibial) are both normal, the patient should be reassured that the risk for complications during the next year is low. If LOPS is present, the patient is considered to be at high risk for foot complications. Patients with LOPS should be seen every 6 to 8 weeks for preventive care. Nails and calluses should be débrided if necessary. At each visit, foot self-care education should be reinforced, and early identification of foot problems can be accomplished.

PREVENTIVE CARE

The program for prevention should include foot self-care education for the patient, periodic professional foot care, and proper footwear. These interventions can be provided by various trained professionals, including podiatrists, physicians, nurses, diabetes educators, physical therapists, or a combination of these professionals. Individual instruction and group sessions are encouraged. A basic understanding of the pathogenesis of foot complications usually leads to better self care than the mechanistic learning of a table of "do's and don'ts." However taught, critical daily foot care practices are important for each high-risk patient (ie, patients with LOPS or ischemia). **Appendix 1** lists basic patient instructions for daily diabetic foot care.

Appropriate Footwear

Inappropriate footwear can cause complications.[4] Most dorsal and lateral ulcers, for example, are caused by poorly fitting shoes. Footwear that does not protect the plantar surface from the effects of repeated stresses at high-pressure sites will facilitate the development of plantar ulcers. Once a patient is identified as being at high risk, every step that the patient takes should be in prescribed footwear. A professional with experience and interest in this area should handle footwear prescription in the high-risk patient. However, it is still helpful for primary care providers to understand the basic features of well-designed footwear even if they choose to refer their patients to another professional

next page

Chapter 2—Case Studies in Hypernatremia

Thomas G. Cody, MD, and Richard J. Simons, MD, FACP

I. INTRODUCTION

Hypernatremia is a very common clinical problem. By convention, *hypernatremia* is defined as a serum sodium level of more than 145 mmol/L.[1] It most frequently occurs in hospitalized or severely debilitated individuals, specifically infants and elderly persons or others who have impaired mental status or the inability to ask for or take fluids independently.[2] The evaluation of hypernatremia is typically straightforward and the cause is often elicited from a history and clinical situation. The most common causes of hypernatremia are related to water losses in excess of salt from the kidneys, gastrointestinal tract, or skin that are not adequately replaced by free water intake (**Table 2**).[3] When the cause is unclear, evaluation of the antidiuretic hormone (ADH)–renal axis function (ADH level and urine osmolality) is the next key step. Once the diagnosis and underlying causes have been determined, a precise understanding of treatment (ie, free water repletion or medication) is critical for management. In this article, 2 case patients are presented to highlight the management of hypernatremia.

II. GENERAL PRINCIPLES

SYMPTOMS OF HYPERNATREMIA

The metabolic derangement of hypernatremia can be recognized by several clinical signs and symptoms. The pathophysiology of these symptoms involves water shifts within the nervous system. As the result of an increase in intravascular sodium, free water diffuses out of brain cells, causing these cells to contract. With movement of water out of the brain cells, the brain contracts, also creating the possibility of focal cerebral vein rupture and subarachnoid hemorrhage as well as permanent neurologic damage or death.[1,4] The earliest symptoms are lethargy, fatigue, and irritability. If progressive, there can be increased muscle tone, twitching (including spasticity and hyperreflexia), and, perhaps, seizures (usually in children). More severe symptoms are not usually present until the sodium level reaches 155 to 158 mmol/L.[3,5] The incidence of mortality is increased at sodium levels that approach 180 mmol/L.[3]

As with other metabolic derangements (eg, hyponatremia), an acute change in sodium concentration is more apt to create clinical symptoms than a slow, chronic increase in concentration.[3] The reason for this difference is that time allows for the formation of a compensatory flow of free water and solutes into the brain parenchyma from the cerebrospinal fluid and extracellular fluids.[1,3] In fact, some individuals can have a sodium approaching 170 to 180 mmol/L with few symptoms of severe hypernatremia.[5] Accordingly, rapid correction via a relatively hypotonic solution can induce fluid shifts with "reverse" (ie, opposite) consequences, such as cerebral parenchymal swelling.

DIFFERENTIAL DIAGNOSIS

When evaluating a patient with hypernatremia, one must have a working differential diagnosis. In all cases, only individuals who cannot drink (for one reason or another) will develop elevated sodium levels.[1] Once this principle is understood, the causes of hypernatremia can be broken down into 2 main groups: excessive loss of free water that is not replaced or insufficient intake of free water. Most patients fall into the first category. As mentioned, the losses can be through skin, renal, or gastrointestinal systems. In spite of massive losses of free water, these individuals usually will not develop hypernatremia as long as they have access to water. The second category of individuals who develop hypernatremia have insufficient intake of free water or *hypodipsia*. These people have a defective thirst or osmoreceptor function.[3] Of note, a small third category termed *iatrogenic hypernatremia* includes patients who receive excessive hypertonic sodium (Table 2).[1,2] In the following cases, hypernatremia resulting from excessive free water losses will be discussed because this is the predominant category of clinical relevance.

ETIOLOGY OF HYPERNATREMIA

Gastrointestinal Losses

The differential category of gastrointestinal losses that can lead to hypernatremia is quite large and cannot be discussed in detail, although some aspects deserve mention. The category includes not only infectious and