Review Article

Diabetes Mellitus: Musculoskeletal Manifestations and Perioperative Considerations for the Orthopaedic Surgeon

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Abstract

Diabetes mellitus is a disease of uncontrolled hyperglycemia. Despite a more sophisticated understanding of the pathophysiology of diabetes mellitus and despite pharmacologic advancements that enable better glycemic control, the prevalence of this disease and its devastating sequelae continue to rise. The adverse effects of diabetes on the nervous, vascular, and immune systems render the musculoskeletal system vulnerable to considerable damage. Foot involvement has traditionally been thought of as the most severe and frequently encountered orthopaedic consequence. However, the upper extremity, spine, and muscles are also commonly affected. Orthopaedic surgeons are more involved than ever in the care of patients with diabetes mellitus, and they play a vital role in the multidisciplinary approach used to treat these patients. As a result, surgeons must have a comprehensive understanding of the musculoskeletal manifestations and perioperative considerations of diabetes in order to most effectively care for patients with diabetes mellitus.

Diabetes mellitus (DM) is a condition marked by high blood glucose levels. Type 1 diabetes is caused by the autoimmune destruction of the insulin-producing β cells of the pancreas. Persons with type 1 disease are insulin-deficient and prone to developing ketosis.[1] Type 1 diabetes is sometimes referred to as juvenile-onset diabetes, even though it can present at any age. Type 2 diabetes accounts for 90% to 95% of all cases; persons with this type produce insulin, but their bodies are unable to properly use it due to end-organ insulin resistance. There are likely many causes of type 2 diabetes, but the specific etiologies remain unknown.[1] Type 2 is traditionally referred to as adult-onset diabetes; however, because of the obesity epidemic, it is now seen in younger patients as well, including adolescents.[1]

Diabetes is a systemic disease that affects the nervous, vascular, immune, integumentary, and musculoskeletal systems. To optimize the care of the patient with diabetes and reduce the risk of complications, a team-oriented approach to treatment is required, with the orthopaedic surgeon playing a significant role.

Epidemiology

In 2012, approximately 22.3 million Americans had diabetes, and they incurred an estimated $306 billion in direct medical costs.[2] The annual medical expenditures of persons with diabetes are approximately 2.3 times

Table 1

The Prevalence of Various Musculoskeletal Conditions in Patients With and Without Diabetes Mellitus[7-9]

Musculoskeletal Disorder	Percent Incidence in Patients With Diabetes	Percent Incidence in Patients Without Diabetes
Adhesive capsulitis	11–30	2–10
Dupuytren contracture	16–42	13
Carpal tunnel syndrome	11–20	0.05
Flexor tenosynovitis	11	<1
Diffuse idiopathic skeletal hyperostosis	13–49	1.6–13
Limited joint mobility	8–50	0–26

higher than those of persons who do not have this condition ($13,741 and $5,853, respectively). Americans with diabetes are responsible for an additional $69 billion in non-healthcare costs due to diabetes-related disability and premature mortality. Approximately 48.3 million Americans are projected to have diabetes by 2050, which will generate an even greater burden on the healthcare system and society in general.[3]

In the United States, diabetes is the seventh leading cause of death and the leading cause of kidney failure and blindness in working-age adults.[4] It is also a major risk factor for heart disease and stroke.

Persons with DM have an estimated 15% lifetime risk of developing a foot ulcer, and the 10-year cumulative incidence of lower extremity amputation is estimated to be 5% in persons diagnosed before age 30 years (younger-onset diabetes) and 7% in patients diagnosed at age 30 years or older (older-onset diabetes).[5] Of those patients, an estimated 28% to 51% will require a second amputation within 5 years of the first one.[5] Management of these ulcers is expensive, with 2 years of treatment costing approximately $28,000.[6] Patients with DM are also significantly more likely than patients without DM to develop adhesive capsulitis, Dupuytren contracture, carpal tunnel syndrome, flexor tenosynovitis, cheiroarthropathy (a cutaneous condition characterized by thickened skin and limited joint mobility of the hands and fingers that leads to flexion contracture), and diffuse idiopathic skeletal hyperostosis (DISH)[7-9] (Table 1).

Pathophysiology

Nervous System

Hyperglycemia-induced metabolic abnormalities contribute to the development of neuropathy in patients with diabetes.[10] The motor, sensory, and autonomic divisions of the nervous system are all adversely affected.

Diabetic neuropathy most commonly manifests as a symmetric peripheral polyneuropathy, with findings such as diminished sensation in a stocking-glove distribution. Isolated involvement of individual peripheral and cranial nerves is less common but has been described in the literature.[8] Carpal tunnel syndrome is one such example. Autonomic neuropathy causes gastrointestinal, genitourinary, and cardiovascular symptoms, as well as sexual dysfunction.

Vascular System

Diabetes is associated with both microvascular and macrovascular disease. Microvascular disease contributes to the development of neuropathy, nephropathy, and retinopathy. Macrovascular disease can lead to coronary artery disease, cerebrovascular disease, stroke, peripheral arterial disease (PAD), and lower extremity infection and amputation. Uncontrolled hyperglycemia contributes to vasoconstriction, a hypercoagulable state, and arterial luminal stenosis.[11]

PAD is more often bilateral and more rapidly progressive in persons with diabetes than in those without the disease. Additionally, diabetes is associated most strongly with the femoropopliteal and below-knee tibial manifestations of PAD.[12,13] Other risk factors (eg, smoking) typically cause more proximal disease in the aortoiliofemoral vessels.[12]

Immune System

Diabetes impairs the immune system in many ways. Altered polymorphonuclear leukocyte function is one such impairment. This state creates a favorable environment for bacterial growth and compromises fibroblast function and collagen synthesis, factors that interfere with wound healing and increase the incidence of postoperative wound infections.[14]

Musculoskeletal System

High blood glucose concentrations are directly responsible for increased collagen cross-linking via advanced glycosylation end products, which decreases the solubility and digestibility of collagen.[15] This ultimately increases the stiffness of both collagen and the structures built on a collagenous framework (eg, tendons), thereby making them more vulnerable to contractures.[15]

The Diabetic Foot

The diabetic foot is susceptible to ulceration, infection, neuroarthropathy, and fracture. Evaluation must begin with a thorough history and physical examination.

A reduced or lost ankle jerk reflex is suggestive of neuropathy and often is one of the earliest signs of diminished protective sensation. Peripheral neuropathy can also be identified with the use of a 128-Hz tuning fork and the 10-g Semmes-Weinstein 5.07 monofilament. Although this monofilament is the accepted medical standard as the minimum physiologic threshold for defining normal foot sensation, some have found that the loss of protective sensation is detected earlier with the 4-g monofilament than with the 10-g monofilament.[16]

The vascular examination must begin with inspection of the foot and palpation of peripheral pulses. Visible signs of PAD include dependent rubor; pallor on elevation; dystrophic toenails; lack of hair growth; and cool, dry, fissured skin.[12] Palpation of the femoral, popliteal, and pedal pulses is mandatory; the absence of the dorsalis pedis and posterior tibial pulses is highly suggestive of vascular disease.

In detecting PAD, noninvasive ankle brachial index (ABI) measurements are more reproducible and accurate than is palpation of pulses.[17] The ABI is defined as the ratio of the systolic blood pressure in the ankle divided by the systolic blood pressure in the arm. A normal ABI is 0.91 to 1.3. A value <0.91 is indicative of obstruction. The lower the ABI, the greater the severity of disease. Of note, an ABI >1.3 suggests the presence of poorly compressible vessels as the result of calcification, thereby rendering the ABI inaccurate.

The American Diabetes Association recommends a screening ABI in patients with diabetes who are aged >50 years.[12] Provided the baseline ABI is normal, it should be repeated every 5 years. In persons aged <50 years who have diabetes and who have risk factors for PAD (eg, smoking, hypertension, hyperlipidemia, duration of diabetes >10 years), a screening ABI should be considered. ABI measurement should be done in any person with diabetes and with symptoms of PAD (eg, vascular claudication).

In the patient with a confirmed diagnosis of PAD, vascular surgery evaluation is important because a more objective determination of disease location and severity is helpful in clinical decision-making. Vascular laboratory studies should be obtained, including segmental pressures and pulse volume recordings.[12] Further evaluation with anatomic studies (ie, duplex sonography, magnetic resonance angiography, contrast angiography) also may be necessary, particularly in patients in whom revascularization is being considered.

Ulceration is the most common cause of soft-tissue infection in patients with diabetes, and it is multifactorial in etiology.[1] Boyko et al[18] prospectively determined the relative risk of a multitude of risk factors. Sensory and autonomic neuropathy, greater body mass, poor vision, reduced skin oxygenation and foot perfusion, and Charcot and hammer/claw toe foot deformities were found to place patients at higher risk for ulceration. Conversely, the duration and type of diabetes, race, smoking status, diabetes education, joint mobility, and hallux blood pressure were found to have no independent correlation with the risk of foot ulceration.

Wound healing in diabetic patients is complicated and challenging, and many classification systems have been developed to aid in clinical decision-making. The Wagner classification and the Brodsky depth-ischemia classification are two of the most commonly used[19] (Table 2). Unlike the Wagner and Brodsky classifications, the University of Texas Wound Classification incorporates wound depth (grades 0 to III) with a stage that correlates with the absence of infection or ischemia (A), the presence of infection and absence of ischemia (B), the presence of ischemia and absence of infection (C), or the presence of infection and ischemia (D).[19] This staging component distinguishes the University of Texas Wound Classification, which has been shown to have better prognostic value than the Wagner classification.[20]

Table 2 describes the generalized, classification-based treatment modalities for diabetic ulcers, including the use of a total contact cast (TCC). This intervention is often used to treat Wagner grade 1 and 2 neuropathic ulcerations (Figure 1). However, use of a TCC requires frequent follow-up and weekly or near-weekly cast changes. Complications of the TCC include the development of new pretibial, malleolar, and foot ulcers. Guyton[21] reviewed complications in a consecutive series of 398 casts and reported a complication rate of 5.52% per cast. Of the 70 patients in that study, 30% suffered a complication during their course of treatment.

The TCC can be applied with an open or a closed toe box. At our institution, TCCs are applied with an open toe box. Although some believe that this technique places the toes at risk for dorsal ulceration as they contact the edge of the cast, we have had success with this approach. With careful observation and frequent cast changes, which we recommend regardless of casting technique, iatrogenic ulceration of the dorsum of the toes can be largely avoided.

The meticulous technique and surveillance required with TCCs led to the development of alternative methods of managing diabetic ulcers of the plantar foot. Prefabricated pneumatic walking braces (PPWBs) are one such innovation. These boots have pressure-adjustable air bladders lined with a soft nylon material that is designed to prevent abrasions and ulcerations. Baumhauer et al[22] compared plantar foot pressure metrics in TCCs, PPWBs, and standard shoes and determined that the PPWB can be effective in the management of diabetic plantar foot ulcerations.

Table 2

Management of Diabetic Foot Ulcers Based on the Brodsky Depth-Ischemia Classification and the Wagner Classification[19,a]

Classification	Definition	Treatment
Depth grade		
0 (Wagner grade 0)	Intact skin, but foot at risk	Patient education, accommodative footwear, regular examination
I (Wagner grade 1)	Superficial ulceration, not infected	External pressure relief: total contact cast, prefabricated pneumatic brace, walking brace, accommodative footwear
II (Wagner grade 2)	Deep ulceration with exposed tendon or joints (± superficial infection)	Surgical débridement, wound care, pressure relief if ulcer converts to Wagner grade 1, antibiotics (if infected)
III (Wagner grade 3)	Deep ulceration with exposed bone, deep infection (osteomyelitis), or abscess	Surgical débridement, antibiotics, pressure relief if ulcer converts to Wagner grade 1. Ray or partial foot amputation may be required in refractory cases.
Ischemia stage		
A	No ischemia	Observation
B	Ischemia without gangrene	Vascular evaluation, possible revascularization
C (Wagner grade 4)	Partial gangrene (forefoot)	Vascular evaluation and reconstruction, local versus larger amputation
D (Wagner grade 5)	Complete gangrene	Amputation, possible vascular reconstruction

[a] In the Brodsky depth-ischemia classification, each foot is assigned both a numeric grade and a lettered stage. The number describes the physical properties of the wound, and the letter indicates the overall vascular status of the foot. The depth-ischemia classification elucidates the complex relationship between ulcer development, wound healing potential, and the vascular system, while also providing an approach to treatment. Wagner grade is based on the presence of ulceration (grades 0 through 3) or deficient vascularity (grades 4 and 5).

Figure 1

A B

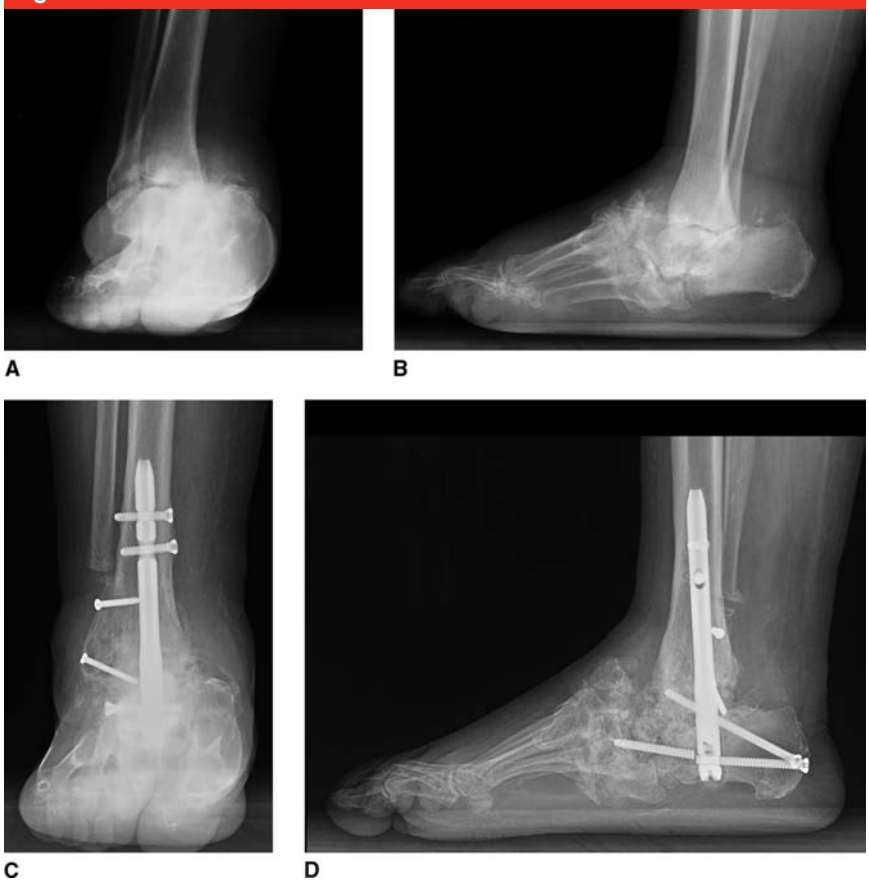
A, Photograph of the left foot of a 56-year-old man with type 2 diabetes and recurrent plantar ulceration. The patient had previously undergone a 5th ray amputation for osteomyelitis. **B,** Photograph of the foot in a total contact cast following local débridement and wound care.

One consequence of gastrocnemius-soleus complex contracture is limited ankle dorsiflexion, which can result in excess pressure on the plantar forefoot and midfoot. As a result, Achilles tendon lengthening and gastrocnemius recession may be required. The effectiveness of both interventions has been validated.[23,24]

In a randomized controlled trial, Mueller et al[23] compared percutaneous Achilles tendon lengthening and TCC with TCC alone. Higher ulcer healing rates were noted in patients who underwent Achilles tendon lengthening plus TCC than in those treated with TCC alone (100% and 88%, respectively). The risk of ulcer recurrence was 75% lower at 7 months and 52% lower at 2 years in the tendon lengthening group. A gastrocnemius-soleus recession can be used as an alternative treatment and has also been shown to effectively manage diabetic midfoot ulcers.[24]

Charcot neuroarthropathy is a progressive, noninfectious disease of the foot seen in persons with sensory neuropathy. Although diabetes is the most common cause of Charcot neuroarthropathy, its precise pathogenesis remains unknown.[25] Two theories regarding its pathophysiology have been proposed.[25] The

Figure 2



AP (**A**) and lateral (**B**) radiographs of the ankle of a 66-year-old woman with type 2 diabetes and Charcot neuroarthropathy of the ankle and hindfoot. The patient failed nonsurgical treatment and required fusion to recreate a plantigrade foot. Weight-bearing AP (**C**) and lateral (**D**) radiographs of the same patient following tibiotalocalcaneal fusion with an intramedullary device.

neurotraumatic hypothesis proposes that the loss of neuroprotection leads to repetitive microtrauma. The neurotrophic hypothesis proposes that sympathetic neuropathy catalyzes osteoclast bone resorption and fragmentation.[25,26]

The destruction of the bones and joints of the foot in the setting of Charcot neuroarthropathy leads to deformity (Figure 2, A and B) and, if left untreated, can cause ulceration and subsequent infection. Distinguishing infection from a Charcot foot is challenging, particularly in the presence of a foot wound. Imaging modalities are useful diagnostic adjuncts, but they must be interpreted cautiously and only in the context of the overall clinical picture. Plain radiographs reportedly have a sensitivity of 62% to 75% in identifying osteomyelitis, whereas three-phase technetium scanning followed by indium In-111-labeled (^{111}In) leukocyte scintigraphy has been found to have a sensitivity of up to 100% and specificity of approximately 83%.[27,28] MRI is useful in identifying abscesses, but it cannot discriminate between infectious edema and abscess caused by neuroarthropathy.

The probe-to-bone test is a validated evaluation tool that can help distinguish osteomyelitis and Charcot neuroarthropathy.[29,30] A sterile, blunt probe is placed into an ulceration. The test is considered positive when the probe contacts bone. The probe-to-bone test has a high negative predictive value and a greater diagnostic utility for detecting osteomyelitis in the diabetic foot compared with clinical signs of infection, radiography, and ulcer specimen culture.[29,30]

When cellulitis or a more superficial infection is suspected, a C-reactive protein measurement may be useful. The C-reactive protein level often is elevated with infection, but it may be normal in patients with Charcot foot.[31] Leukocytosis may be absent in patients with diabetic foot infection; thus, a white blood cell count may not be beneficial in distinguishing a Charcot foot from infection.[32]

An understanding of the Eichenholtz classification for Charcot neuroarthropathy is crucial in selecting the appropriate treatment.[33] This temporal classification system describes the clinical and radiographic changes of bone and joint destruction and subsequent healing and remodeling that occur with Charcot neuroarthropathy. Table 3 presents the Eichenholtz classification, with a modification to include a stage 0, which was not initially described by Eichenholtz.[34] Regardless of the intervention chosen, the goal is to establish a plantigrade foot that allows weight bearing in a shoe or brace (Figure 2, C and D). Unfortunately, sometimes the best reconstructive option is amputation, particularly in the setting of failed arthrodesis or recurrent ulceration or infection.[35]

Pharmacologic management of Charcot neuroarthropathy with diphosphonates and calcitonin has received recent attention; these medications inhibit osteoclast activity.[25] However, at this time, pharmacologic intervention is not routinely used to manage Charcot neuroarthropathy.

The Upper Extremity

The reported prevalence of adhesive capsulitis is higher in persons with diabetes than in those without it.[36]

Table 3

The Modified Eichenholtz Classification of Charcot Neuroarthropathy and the Corresponding Treatment Modalities for Each Stage[33,34]

Stage	Clinical and Radiographic Findings	Treatment
0	Normal radiographs, swelling and erythema, instability	Serial radiographs to monitor progression, protected weight bearing, patient education
I (fragmentation or dissolution)	Swelling, warmth, and erythema; fractures and/or dislocations; osteopenia; periarticular fragmentation	Protected weight bearing with total contact casting or prefabricated pneumatic brace. Serial follow-up and radiographs until erythema, warmth, and swelling resolve.
II (coalescence)	Reduced warmth and swelling, resorption of small bone fragments, early fusion and sclerosis	Total contact casting, prefabricated pneumatic brace, Charcot restraint orthotic walker, or clamshell ankle-foot orthosis
III (reconstruction)	Consolidation of deformity with joint arthrosis, subchondral sclerosis, dissipation of inflammation, stable on examination	Plantigrade foot: custom inlay shoes. Nonplantigrade foot or ulceration: débridement, exostectomy, correction, or fusion with internal fixation. Osteomyelitis: débridement, staged reconstruction versus amputation.

Among persons with diabetes, Yian et al[37] found patients with type 1 diabetes to have a higher rate of adhesive capsulitis compared with patients with type 2 diabetes. Compared with the general population, persons with diabetes also have a worse response to nonsurgical treatment, including NSAIDs, physical therapy, and corticosteroid injections.[36] Surgical intervention can be performed via an open or arthroscopic approach; techniques include excision of fibrotic tissues and adhesions, as well as manipulation.[36]

The incidence of hand abnormalities is also higher in patients with diabetes, particularly those with long-standing disease. Diabetic cheiroarthropathy (ie, stiff hand syndrome) is marked by limited joint mobility, and it predominantly afflicts persons with poorly controlled type 1 diabetes.[8] It is visualized as thickened, tight, waxy skin on the dorsum of the hands. Flexion contractures of the metacarpophalangeal and interphalangeal joints may also be present. Symptoms progress insidiously and include paresthesias and pain. Management consists of glycemic control and hand therapy. Diabetic cheiroarthropathy is commonly seen concurrently with Dupuytren contracture.

Flexor tenosynovitis, Dupuytren contracture, carpal tunnel syndrome, and reflex sympathetic dystrophy are other musculoskeletal manifestations of diabetes (Table 1). The management of these conditions is similar to that in patients who do not have diabetes.

The Axial Skeleton

DISH is another condition that is more frequently seen in patients with diabetes, especially those with type 2 disease.[9] DISH is characterized by new bone formation, most frequently involving the thoracolumbar vertebrae, as well as calcification of the spinal ligaments. Treatment is identical to that of patients without diabetes and consists of analgesics, NSAIDs, and physical therapy. The degree of glycemic control does not correlate with the incidence or progression of symptoms, and the relationship between DISH and DM, insulin, and insulin-like growth factor-1 requires further investigation.[38]

Perioperative Considerations in Patients With Diabetes

Surgical outcomes in persons with diabetes are inferior to those of persons without diabetes. As such, a comprehensive patient evaluation must precede any surgical intervention.[39] Although the orthopaedic surgeon is unlikely to perform this assessment, understanding the perioperative considerations will help the surgeon anticipate and address surgical and systemic complications as they arise (Table 4). Perioperative considerations include wound dehiscence, wound infection (superficial and deep), fracture nonunion, loss of fracture reduction, hardware and implant failure, myocardial infarction, stroke, urinary tract infection, ileus, pulmonary embolus, hemorrhage, increased transfusion requirement, increased length of hospitalization, and death.[40-42]

Approximately 25% of patients are unaware that they have diabetes.[1] Therefore, a glycosylated hemoglobin (HbA_{1c}) screening may be beneficial prior to orthopaedic procedures, particularly in patients with known hyperglycemia in the absence of a formal diagnosis of diabetes.[43] The HbA_{1c} level reflects the average blood glucose over the previous 3 months, and a level ≥6.5% is diagnostic for DM.[1] Knowledge of this value can help facilitate diagnosis, minimize the adverse perioperative outcomes

Table 4

Perioperative Considerations by System for the Patient With Diabetes[39]

System	Consideration
Endocrine	Check hemoglobin A_{1c}
	Evaluate daily glycemic control
	Consider admission 1 d before surgery to optimize metabolic control
	If possible, the patient should be first on the surgical schedule
	Check blood glucose before any anesthesia is administered
Cardiovascular	Recent myocardial infarction (6% rate of reinfarction or death if surgery is performed within 3 mo of a myocardial infarction)
	Check electrocardiogram
	Blood pressure: Preoperative goal of <140/90 mm Hg
Renal	Consider screening for microalbuminuria (elevated albumin excretion rate) and proteinuria. If positive, consult a diabetes specialist.
	Serum creatinine is not always a reliable indicator of renal function.
Nervous (autonomic neuropathy)	Autonomic neuropathy predisposes patients to perioperative hypotension
	Diagnostic tests: Orthostatic blood pressure measurements (fixed pulse indicates significant autonomic neuropathy)
	Patients with autonomic neuropathy require careful perioperative blood pressure and volume monitoring
Gastrointestinal	Diabetic gastroparesis places patients at increased risk both for aspiration during intubation, even if the patient has received nothing by mouth for 6 h before surgery, and for postoperative ileus.

associated with DM, and create an acceptable plan for diabetes management following surgery.

In the orthopaedic literature, a strong correlation has been identified between poor glycemic control and surgical complications. Preoperative HbA_{1c} levels of >6.7% and postoperative blood glucose concentrations >200 mg/dL have been shown to be risk factors for wound complications in the setting of primary total joint arthroplasty.[44] Elevated HbA_{1c} values are also associated with an increased failure of intrasheath triamcinolone injections for flexor tenosynovitis (HbA_{1c} ≥8%),[45] in addition to an increased risk of wound complications in patients undergoing transmetatarsal amputations (HbA_{1c} >8%).[46] Additionally, Koutsoumbelis et al[47] determined a history of diabetes to be a critical risk factor for postoperative infection following posterior lumbar instrumented arthrodesis.

We recommend that orthopaedic surgeons gain familiarity with The Endocrine Society clinical practice guideline on the management of hyperglycemia in noncritically ill hospitalized patients.[43] This invaluable resource provides succinct, evidence-based recommendations that apply to patients admitted for orthopaedic procedures (Table 5). The impetus behind these guidelines and other evidence-based recommendations pertaining to diabetes is the Diabetes Control and Complications Trial, which was a major clinical study performed between 1983 and 1993.[48] The Trial research group compared the effects of standard versus intensive blood glucose control on the complications of diabetes and showed that keeping blood glucose levels as close as possible to normal slows the onset and progression of diabetes-related damage to the eyes, the kidneys, and the nervous system, even in patients with a history of poor glycemic control.

Summary

Orthopaedic surgeons have a significant role in the care of many patients with diabetes, and this involvement is likely to grow as the prevalence of DM continues to rise. The destructive musculoskeletal sequelae of DM, which frequently involve the foot and ankle, are attributed at least in part to its adverse effects on the nervous, vascular, and immune systems.

The metabolic and physiologic complexity of patients with diabetes puts them at high risk for any surgical procedure. Therefore, a comprehensive preoperative evaluation before any orthopaedic procedure is necessary to reduce the perioperative risk factors and optimize the potential for a complete and safe outcome. In the setting of poor glycemic control, patients are significantly more vulnerable to perioperative complications, including increased mortality.

Table 5

Summary of Recommendations From The Endocrine Society Clinical Practice Guideline on the Management of Hyperglycemia in Noncritically Ill Hospitalized Patients That Are Relevant to Caring for Orthopaedic Patients[43]

Diagnosis and recognition of hyperglycemia and diabetes in the hospital setting
All patients, independent of a prior diagnosis of diabetes, should have blood glucose (BG) testing on admission.
Patients without a history of diabetes with BG >140 mg/dL should be monitored with bedside testing for at least 24 to 48 h. If the BG remains >140 mg/dL, ongoing testing and appropriate therapeutic intervention is required.
All inpatients with known diabetes or hyperglycemia should be assessed with a hemoglobin A_{1C} level if it has not been performed in the preceding 2–3 mo.
Monitoring BG in the noncritical care setting
The timing of BG measurements should match the patient's nutritional intake and medication regimen. Testing should be done before meals and at bedtime in patients who are eating and every 4–6 h in patients who are receiving nothing by mouth.
Blood glucose targets in the noncritical care setting
A premeal target of <140 mg/dL is recommended and a random BG of <180 mg/dL for the majority of hospitalized patients with noncritical illness.
To prevent hypoglycemia, antidiabetic therapy should be reevaluated when BG values fall below 100 mg/dL.
Pharmacologic therapy
Insulin therapy is the preferred method for achieving glycemic control in the hospitalized patient with hyperglycemia.
Oral hypoglycemic agents should be discontinued and insulin therapy initiated for the majority of patients with type 2 DM at the time of hospital admission.
In those patients treated with insulin before admission, the dose should be modified according to clinical status to reduce the risk for hypoglycemia and hyperglycemia.
During hospitalization, the use of an insulin sliding scale as the sole method for glycemic control in patients with diabetes should be avoided.
Scheduled subcutaneous insulin therapy should consist of basal or intermediate-acting insulin given once or twice a day in combination with rapid- or short-acting insulin before meals in patients who are eating.
In those patients with acceptable preadmission glycemic control, the preadmission antidiabetic regimen should be reinstituted, beginning at least 1 d before discharge to ensure a safe transition.
Perioperative BG control
All patients with type 1 diabetes who undergo any surgical procedure should receive either continuous insulin infusion or subcutaneous basal insulin with bolus insulin as required to prevent hyperglycemia during the perioperative period.
Oral and non-insulin injectable antidiabetic agents should be discontinued before surgery with the initiation of insulin therapy in those who develop hyperglycemia.
The preferred approach for postoperative BG control is subcutaneous insulin therapy, with basal (for patients who are receiving nothing by mouth) or basal bolus (for patients who are eating) insulin therapy.
Recognition and management of hypoglycemia in the hospital setting
When BG drops below 70 mg/dL, immediate therapy is required to restore a euglycemic state.

DM = diabetes mellitus

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 17, 18, 20, 23, and 48 are level I studies. References 3, 6-9, 11, 13, 16, 19, 22, 29-31, 36-38, 45, and 47 are level II studies. References 5, 32, 40-42, 44, and 46 are level III studies. References 21, 24, and 26 are level IV studies. References 1, 2, 4, 10, 12, 14, 15, 25, 27, 35, 39, and 43 are level V expert opinion.

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