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Diabetic Foot Infections

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1 Introduction/Epidemiology

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia, impaired insulin production (type 1), defective insulin utilization (type 2), or a combination thereof [1]. Type 1 diabetes involves the selective destruction of insulin-producing pancreatic β-cells while type 2 involves downregulation of peripheral insulin receptors and decreased insulin utilization. Of the two, type 1 diabetes represents roughly 10% of the world's cases, while its counterpart represents 90% of cases [2]. The incidence of diabetes increases year by year and was the seventh leading cause of death worldwide in 2010 [3]. It is estimated that the worldwide prevalence of diabetes will rise from 415 million in 2015 to 642 million in 2040 [3].

There are many clinical consequences of diabetes including autonomic dysfunction, retinopathy, and nephropathy. However, diabetic foot ulcers (DFUs) are one of the most common and serious complications of diabetes, affecting nearly 15% of all diabetic patients. Of patients with diabetic foot ulceration, 20% will have inadequate blood flow, 50% will have neuropathy, and approximately 80% will have both conditions [3]. In addition, the rate of lower extremity amputation is 15-fold greater in diabetics compared to nondiabetics [1]. It is therefore integral that specialists of different disciplines work hand in hand to tackle all aspects of this debilitating disease.

2 Pathophysiology

Hyperglycemia is at the center of the physiologically negative effects of diabetes. In the human body, the polyol pathway is responsible for the metabolism of excess glucose into sorbitol and, eventually, fructose. Glucose is first degraded by aldose reductase into sorbitol, followed by the conversion of sorbitol into fructose by sorbitol dehydrogenase. With hyperglycemia, large amounts of sorbitol and fructose are created by this pathway, resulting in oxidative stress, endothelial dysfunction, inhibition of nitric oxide production, and formation of nonenzymatic advanced glycation end products (AGEs) [4, 5]. In addition, fructose is a ten times more potent glycation agent than glucose [6].

AGEs are produced when glucose (or fructose) binds with cellular proteins, nucleic acids, and lipids, resulting in the formation of a product known as a Schiff base. This product then rearranges itself into a different form known as an

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Amadori product; it is the Amadori product that is the direct precursor to AGEs. Once formed, AGEs interact with cellular surface receptors (RAGEs) to convert those molecules into pro-oxidant, procoagulant, and pro-inflammatory agents [1, 4, 7]. AGEs also produce a biochemical alteration of joint and muscular tissue by increasing collagen cross-links. This leads to mechanical alteration of the tissues with a resultant loss of elasticity and tensile strength. [8].

Within the vascular endothelium, AGE accumulation leads to oxidative damage, basement membrane thickening, and a propensity to develop atherosclerotic plaques. AGEs also reduce the bioavailability and activity of endothelium-derived nitric oxide, decreasing vessel's vasodilatory potential [7]. Oxidative stress by AGEs is further compounded by the depletion of NADPH in the polyol pathway, decreasing the NADPH needed for production of key antioxidants such as glutathione. The end result of these physiologic changes is micro- and macrovascular compromise leading to retinopathy, nephropathy, and neuropathy [4, 9].

The pathogenesis of the diabetic foot ulcer is a multifactorial combination of vascular disease, neuropathy, and autonomic dysfunction. In regard to vascular disease, diabetic patients can develop calcifications of the endothelial tunica media leading in a loss of vessel elasticity. This calcification, known as Mönckeberg's sclerosis, is secondary to the differentiation of vascular smooth muscle cells into chondrocyte-like cells, capable of expressing and releasing proteins regulating calcification [10]. As a result of this calcification, it is easier for atherosclerotic plaques to develop along the intimal lining, damaging the vessels in the process and putting the patient at risk for ischemia [11].

Diabetic peripheral neuropathy has three main components and how it impacts the diabetic patient.

2.1 Loss of Protective Sensation

The neuropathic manifestations of diabetes include the loss of protective sensation, proprioception, temperature recognition, decreased sweating, and decreased muscle tone (specifically the intrinsic muscles of the foot). Nerve damage stems from the accumulation of reactive oxygen species secondary to the polyol pathway, as well as a loss of nerve blood flow from nutrient arteries known as the vasa nervorum [6]. With a loss of protective sensation, the diabetic foot is more apt to mechanical and thermal injury. Often, patients do not recognize cutaneous damage to their feet until they start noticing other manifestations, such as drainage and malodor coming from the wound.

2.2 Autonomic Diabetic Peripheral Neuropathy

Damage to the autonomic nervous system causes the opening of cutaneous arteriovenous shunts and malfunction of the precapillary sphincter, resulting in decreased blood flow and dry skin [12].

2.3 Motor Diabetic Neuropathy

Intrinsic pedal musculature also loses its tone and mechanical strength, resulting in extrinsic muscles from the leg gaining mechanical advantage. Glycosylation of muscle and tendon structures also leads to stiffness and a loss of joint range of motion, specifically the gastroc-soleus aponeurosis [8]. The sum of these changes leads to the formation of biomechanical pathology (i.e., hammer toes, equinus), abnormal pressure distribution, and cutaneous ulceration [4].

Diabetic patients have a decreased ability to combat infection. Hyperglycemia has been shown to inhibit the chemotactic, phagocytic, and antimicrobial activities of neutrophils and promote the nonenzymatic glycosylation (and eventual damage) of immunoglobins [13, 14]. Studies have also shown a decrease in the proliferative function of CD4 lymphocytes in diabetic patients [13]. As a result, diabetics are not only more susceptible to infection but also have a harder time mounting an adequate immune response.

Case 1 (Fig. 1)
This is a diabetic with an ischemic third digit with a deep-space infection. Wound is foul smelling, fluctuant with drainage. The patient

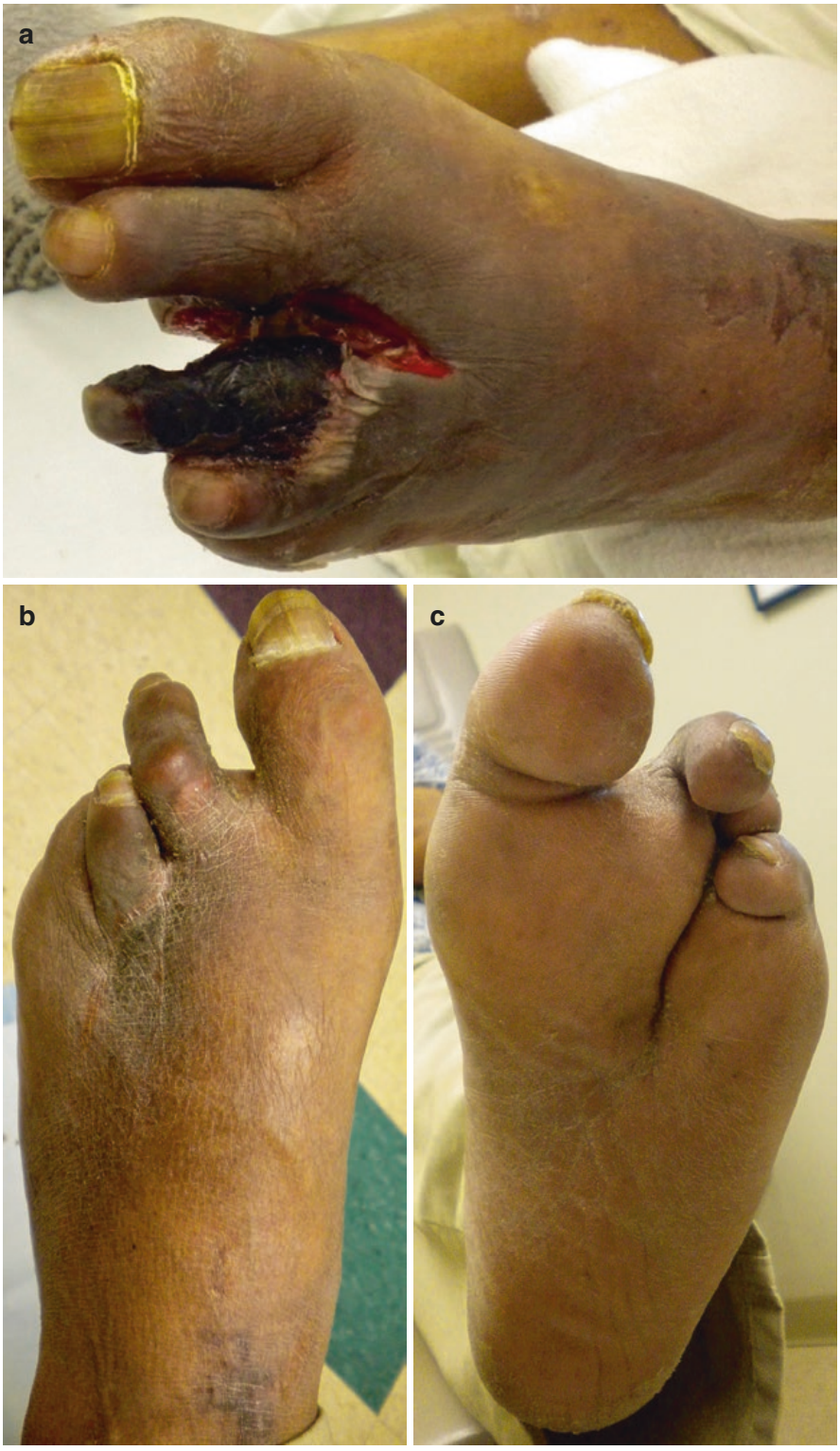

Fig. 1 (**a**) Preoperative. (**b**, **c**) After deep-bone and soft-tissue resection and the use of negative-pressure therapy and hyperbaric oxygen treatment, the wounds healed well

presented with an elevated white blood cell count, inflammatory markers, and a clinical presentation of an urgent need to go to surgery for an aggressive incision and drainage, bone and soft-tissue debridement, and a bone biopsy with irrigation. There was an aggressive bone and deep soft-tissue resection, followed by the use of negative-pressure therapy and hyperbaric oxygen treatment. The wounds are well healed and the patient now wears custom-made diabetic shoes and is completely independent and functional.

3 Risk Factors

Several risk factors have been identified to reduce the risk associated with ulcers, infection, and amputation. The most important of these are a history of previous ulceration, neuropathy, foot deformity, and peripheral vascular disease [15]. A study of 1300 type 2 diabetics recognized the above risk factors, as well as an elevated hemoglobin A1C (>7), as the best predictors of risk for amputation [15]. These risk factors have been utilized to develop risk classification systems to aid providers in categorizing patients. One of the most widely used systems is the International Working Group on the Diabetic Foot [16]. They categorize patients as follows:

Group 0—no neuropathy
Group 1—neuropathy with no deformity or PVD
Group 2—neuropathy with deformity or PVD
Group 3—history of ulceration or amputation

In a prospective study of 225 diabetic patients, stratification using this system was found to be predictive of amputation and ulceration, with only patients classified in groups 2 and 3 undergoing an amputation. This study underscores that those patients with these specific risk factors are at greatest risk. Diabetic patients that develop any or all of these attributes require close monitoring [17].

Unfortunately, shortcomings by physicians have been identified in several studies regarding these risk factors being identified or monitored. A survey of over 1400 clinicians regarding their adherence to the recommendations of routine foot care by the American Diabetes Association showed only a 50% compliance rate with semiannual neurologic and foot exams [18]. Additionally, a retrospective review of a major California health maintenance organization identified 14,539 diabetic patients, only 6% of which had a documented diabetic foot exam within the last 12 calendar months [19].

Case 2 (Fig. 2)
This is a young diabetic male who was previously treated at an outside institution and presented with earlier great toe amputation and an attempt to salvage the dorsal soft tissues. The patient now presents with a severe diabetic foot infection with an elevated white count, inflammatory markers, and clinical signs of severe infection. Following aggressive debridement and resection of all necrotic issue and after the use of negative-pressure therapy, the wound bed is prepared for a split-thickness skin graft for coverage of the wound. A split-thickness skin graft harvested from the thigh is applied to the dorm of the foot.

4 Workup/Diagnosis

In addition to a thorough history, the physical exam performed by the provider/surgeon is the most vital step in identifying diabetic foot infections. The goal of the exam should be to determine the extent and severity of infection, identifying underlying factors that predispose to and promote infection, and assessing the microbial etiology. Initial examination begins with assessment of the patient's vital signs, temperature, heart rate, respiration rate, and blood pressure. The core measurements can instantly provide feedback to the provider of the severity of the patient. One should perform a brief general physical exam to eliminate other possible sources of infection or systemic distress. Lower extremity assessment should be next, and should cover the five major systems consisting of dermatological, musculoskeletal, orthopedic, neurological, and vascular. Identification of the cause and source of the infection, likely from a wound, is critical. Full assessment of the wound should include measurements, depth, tracking, tunneling, exposure of bone, purulence, fluctuant, or crepitus. Debridement may need to be performed in this initial stage in order to obtain an accurate culture. Global or isolated foot deformities contributing to the cause of the infection should be identified at this stage. Extent of swelling, edema, cellulitis, lymphangitis, and palpable lymph nodes should all be noted. Consultation of other medical or surgical services should be determined by your physical exam. Several studies have reported improved outcomes with a multidisciplinary approach to diabetic foot infections. This includes involvement of specialists in wound care, infectious

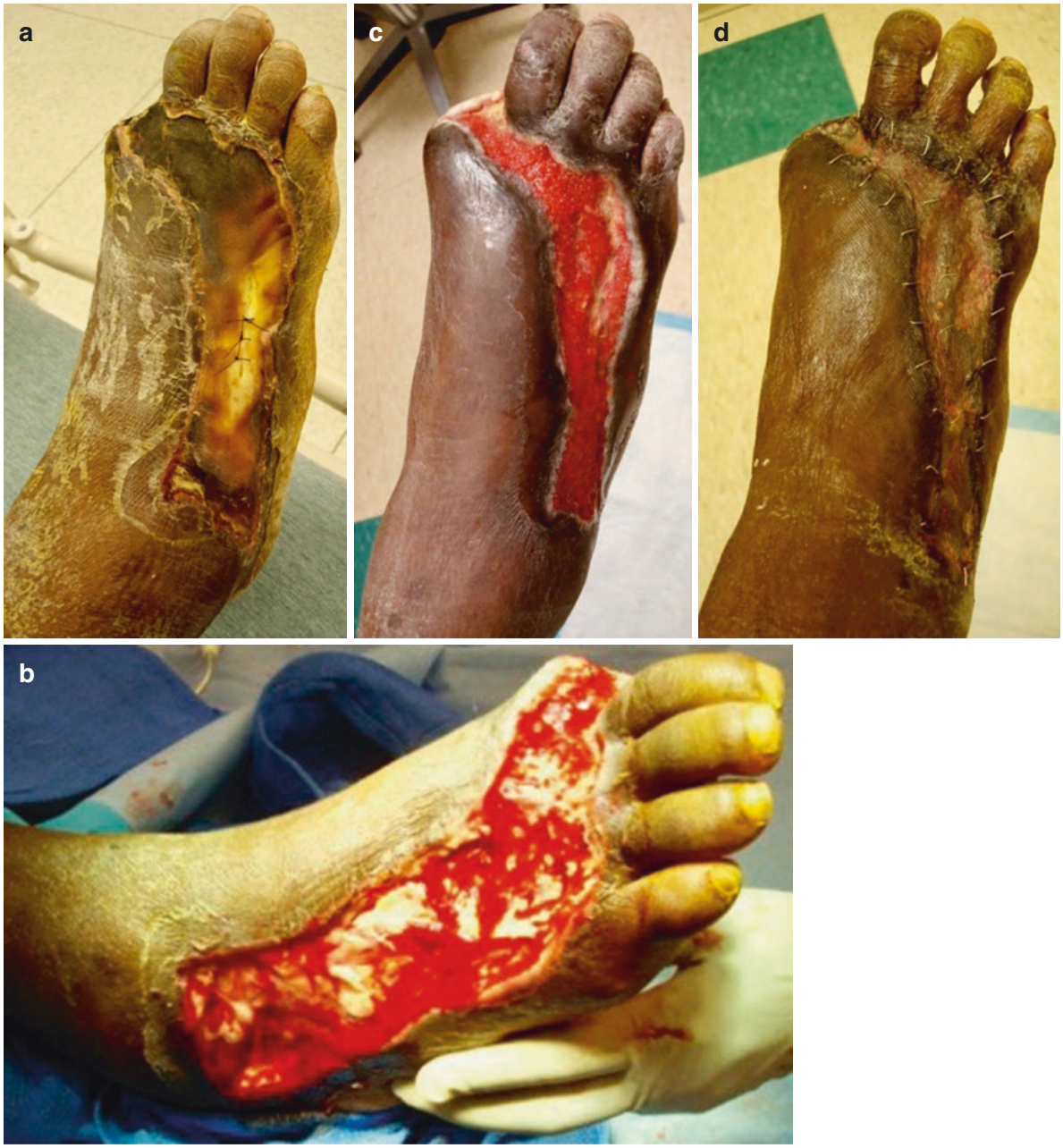

Fig. 2 (**a**) Preoperative. (**b**) Following aggressive debridement and resection of all necrotic issue. (**c**) Following negative-pressure therapy, the wound is prepared for a split-thickness graft. (**d**) Following split-thickness skin graft

diseases, endocrinology, and surgery [20–22]. It has been the author's experience that the diabetic patient appears to respond best when a foot and ankle specialist is involved. The foot and ankle specialist is often on the "front lines" in treating these patients. The quicker and more aggressively this patient population is treated the more likely limb salvage is successful. Additionally, there is typically an underling cause of the initiating wound from failed biomechanics. Once the infection is stabilized a qualified foot and ankle surgeon should attempt to balance the foot and ankle through soft-tissue and/or bony reconstruction to eliminate future problems when appropriate.

Laboratory testing and advanced imaging are the next critical steps after physical exam by the provider. When treating the diabetic patient with a limb-threatening infection, laboratory values can provide information in determining the patient's

medical status globally, as well as the severity of the infection. It is common practice for the diabetic infected patient to undergo CBC, CMP, ESR, CRP, renal and hepatic testing, pan culturing, X-rays, and noninvasive vascular studies. Although these values can guide treatment, they should not be relied on solely. As already established, this patient population is immunocompromised and lab values can be grossly skewed or underestimated [13, 14]. Hepatic and renal function testing not only can guide or aid in antibiotic selection, but can also give the provider a gauge of the patient's immunocompromised status. ESR and CRP, while nonspecific, are used as indicators of systemic inflammation. Specifically, they can be indicators of bone infection when elevated, and when trended over an extended period can be indicators of therapeutic success. It is also common practice for these patients to receive lactic acid and procalcitonin lab monitoring in cases of severe sepsis [23]. But these values have yet to be universally utilized by foot and ankle surgeons since they are not always readily available. Recent literature has shown that procalcitonin can be an effective biomarker for diabetic foot infection and its therapeutic response [24].

Culturing of the diabetic foot infection should involve deep tissue, and depending on the situation bone as well. A meta-analysis showed that superficial swabs have low predictive value of 49% sensitivity and 62% specificity. Additionally, after deeper tissue cultures were performed, antibiotic therapy was changed 56% of the time [25]. Cultures should be taken prior to the administration of antibiotic therapy, and in the most sterile setting whenever possible. In cases of severe septicemia and extreme limb salvage situations provider should not delay appropriate therapy to obtain a higher yield culture. Blood cultures should also routinely be taken in the moderate-to-severe diabetic foot infections as these patients are prone to bacteremia and septicemia. Bone cultures should be taken through uninfected tissue whenever possible, and the provider should consider multiple specimens as the situation dictates. Bone should also be sent to pathology for evaluation.

Noninvasive vascular studies are grossly underutilized in the treatment of diabetic foot infection patients. As previously described in this chapter, the pathophysiology of diabetes leaves these individuals prone to vascular disease. Diabetics with PAD have a threefold increased risk for amputation [26]. It is estimated that 20–30% of diabetic patients have PAD, and 40% of those that present with infections [27]. Accurate and rapid identification of this can ultimately determine the outcome for these patients. Adequate perfusion to the area of infection is paramount for antibiotic delivery, and tissue oxygenation for healing and recovery [28]. Even patients with palpable pulse baseline levels should be established in the setting of limb-threatening infection. ABIs have been shown to underestimate PAD in up to 40% of patients, due to calcification of vessels [29]. Several studies have showed that an absolute toe pressure >30 mmHg is favorable for wound healing although toe pressures >45 to 55 mmHg may be required for healing in patients with diabetes. Because the digital vessels are spared from calcifications, toe pressures are useful to define perfusion at the level of the foot, especially in patients with incompressible vessels [30–32].

Advanced imaging may also be warranted, although surgical intervention of emergent limb-threatening infections should not be delayed. When physical exam eludes to possible deep infection, or in cases where infection is caused by a foreign body/puncture wound, MRI can be of high yield to the surgeon to identify deep or tracking abscesses. In cases of osteomyelitis, MRI can be of beneficial use given the lag of X-ray bone changes that are typically indicative of concern. Additionally, bone scans can be of limited use in patients when trying to discern osteomyelitis from Charcot neuroarthropathy. Despite the advances in imaging in regard to these predicaments, surgical soft-tissue and bone debridement, biopsy, and culture remain the gold standard as shown by Senneville et al. [33] as the most successful outcome predictor.

Case 3 (Fig. 3)

This is a diabetic foot infection that began from a long-standing diabetic foot ulcer from the plantar aspect of the first metatarsal. The infection became deep and tracked proximally both dorsally and

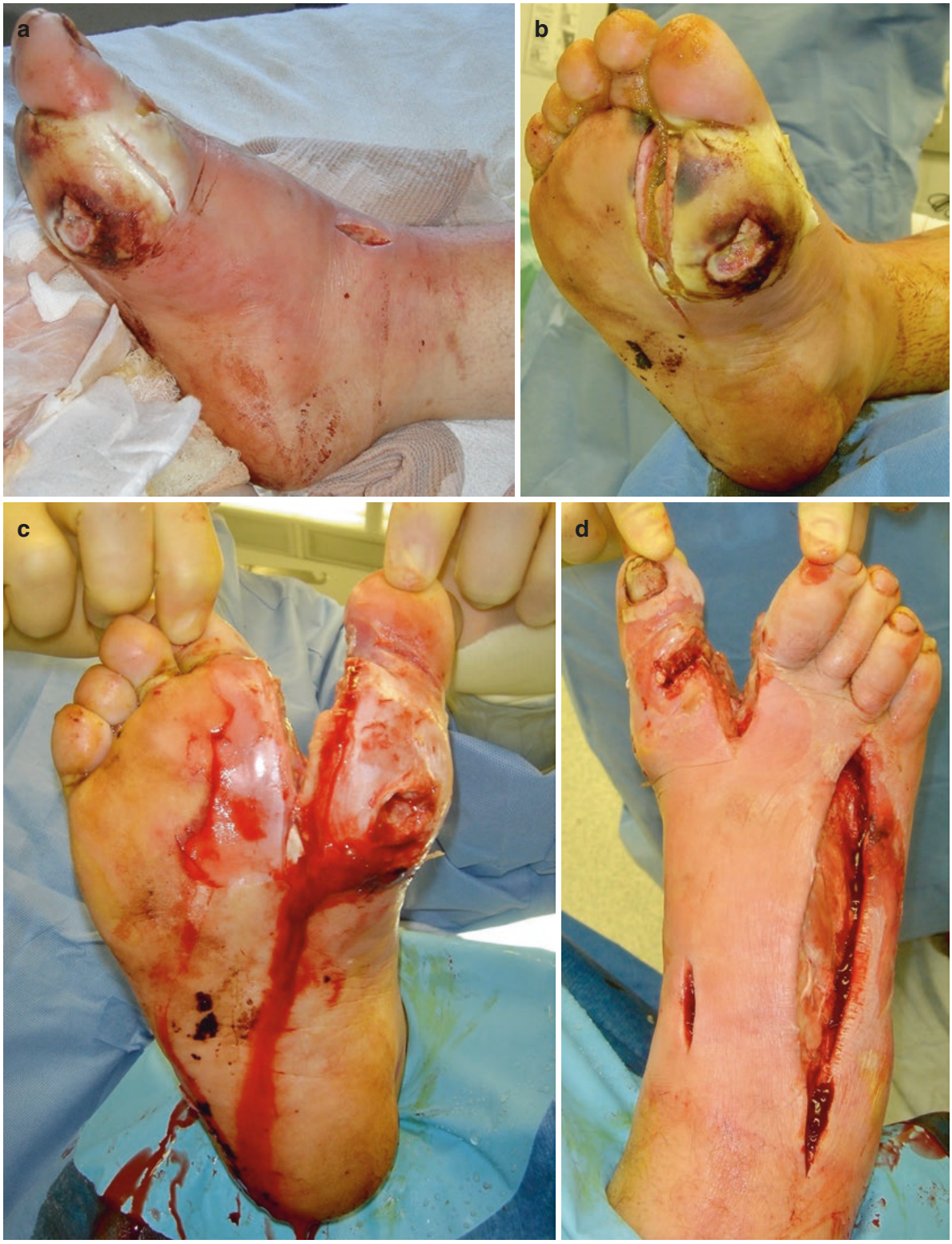

Fig. 3 (**a**, **b**) A diabetic foot infection that began from a long-standing diabetic foot ulcer from the plantar aspect of the first metatarsal. The infection became deep and tracked proximally both dorsally and plantarly along the tendon sheaths creating a severe emergent diabetic foot infection. (**c**, **d**) Following multiple bone and soft-tissue debridements along with negative-pressure wound care, the wounds appear to be much healthier, with improved tissue and color along with a reduction with edema. (**e**) The diabetic foot infection utilizing negative-pressure therapy. (**f**) The diabetic foot infection is healing and maintaining a stable, plantigrade foot

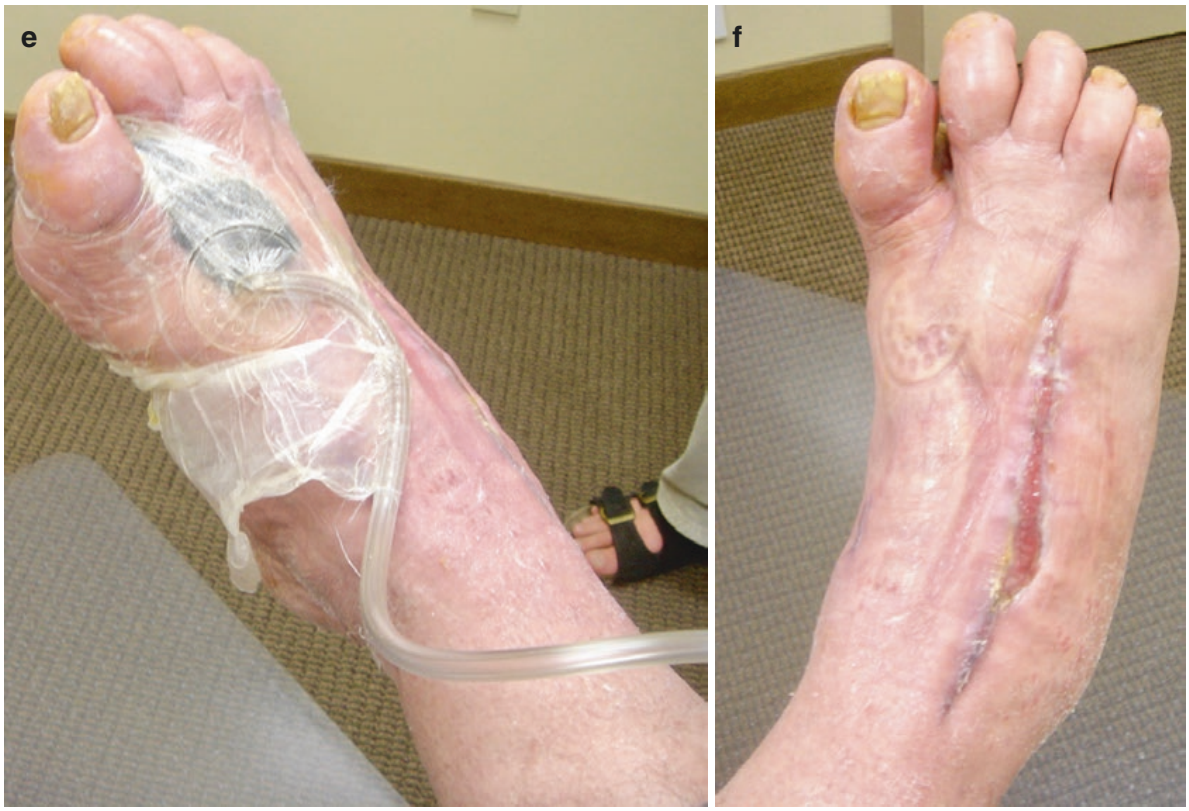

Fig. 3 (continued)

plantarly along the tendon sheaths creating a severe emergent diabetic foot infection. Following multiple bone and soft-tissue debridements along with negative-pressure wound care, the wounds appear to be much healthier, with improved tissue and color along with a reduction with edema. There is maintenance of a stable, plantigrade foot with wounds that are successfully healing.

5 Treatment and Surgical Management

Treating the infected diabetic foot presents its challenges to providers. The IDSA Practice Guidelines for the Diagnosis and Treatment of Diabetic Foot Infections have been evaluated as a useful tool in grading, and accurately treating these patients [21, 22]. The system gives the provider a tool in predicting the likely causative organism, and guidance in selecting appropriate empiric therapy or whether to pursue hospitalization. Under these guidelines infections are graded as mild, moderate, or severe.

Mild infections are classified as showing two cardinal signs of infection and a host response. There is generally cellulitis localized to the area and not extending greater than 2 cm in any plane. Pus may be present. There is no ascending cellulitis or lymphangitis; vital signs are within normal limits. WBC count and blood glucose levels should be within the patient's baseline. These patients can be treated with oral antibiotic therapy directed toward gram-positive organisms. It has been shown that the majority of these infections are caused by Staph aureus/Group B Strep, and broader spectrum therapy is no longer warranted in these lower grade infections [21]. In patients with a history of CA-MRSA, hospitalization, or residence in long-term care facility, more aggressive oral therapy may be warranted based on patients' history and clinical indications.

Moderate infections are classified as showing greater than two cardinal signs of infection, and with cellulitis extending greater than 2 cm. There is extension of the infection beneath the superficial fascia into muscle or bone. The patient is

systemically well, and vitally stable, but with an elevated WBC count and elevated blood glucose abnormal to their respective baseline. The transition to severe infection has the same clinical indicators yet these patients are septic. They are vitally and/or metabolically unstable. Patients identified to have severe arterial insufficiency also fall into this category. These infections, contrary to mild infections, tend to be polymicrobial. Additionally, these infections are of greater risk for limb and life loss due to the infection. Staph aureus and Group B Strep continue to be the predominant organisms. Antibiotic coverage against other organisms is continually up for debate, as increasing evidence has shown that these organisms are not "infectious" [21]. Additionally the IDSA Guidelines also give providers an algorithm to help decision-making processes for surgical intervention.

A. When to Consider a Trial of Nonsurgical Treatment
   1. No persisting sepsis (after 48–72 h if on treatment)
   2. Patient can receive and tolerate appropriate antibiotic therapy
   3. Degree of bony destruction has not caused irretrievable compromise to mechanics of foot (bearing in mind potential for bony reconstitution)
   4. Patient prefers to avoid surgery
   5. Patient comorbidities confer high risk to surgery
   6. No contraindications to prolonged antibiotic therapy (e.g., high risk for *C. difficile* infection)
   7. Surgery not otherwise required to deal with adjacent soft-tissue infection or necrosis
B. When to Consider Surgical Intervention/ Bone Resection
   1. Persistent sepsis syndrome with no other explanation
   2. Inability to deliver or patient to tolerate appropriate antibiotic therapy
   3. Progressive bony deterioration despite appropriate therapy
   4. Degree of bony destruction irretrievably compromises mechanics of foot
   5. Patient prefers to avoid prolonged antibiotics or to hasten wound healing
   6. To achieve a manageable soft-tissue wound or primary closure
   7. Prolonged antibiotic therapy is relatively contraindicated or is not likely to be effective (e.g., presence of renal failure)

Excisional and surgical debridement is pivotal and one of the most powerful modalities in the treatment of moderate-to-severe foot infections [34]. The removal of nonviable, contaminated, or infected material decreases the overall bioburden. This tissue is no longer "biologic," and is a harbinger to bacteria. With its removal, restoring a completely biologic environment reactivates the area increasing the capacity for healing [34, 35]. In cases of severe or necrotizing infections, rapid and aggressive debridement directly impacted salvage outcomes [36]. Sudarsky et al. [37] showed that patients who underwent surgical debridement more than 12 h after presentation had a higher amputation and mortality rate than those debrided sooner. The utility of early surgical debridement was illustrated in a retrospective review of 112 diabetic patients with severe foot infections. Those patients who underwent surgical intervention at the time of presentation had a significantly lower rate of above-ankle amputation than those who received debridement after 3 days of intravenous antimicrobial therapy prior to surgery. Irrigation has also been shown to decrease the overall bacterial load. While much debate has revolved around specific methods and products, low-pressure lavage with large volumes has been widely accepted [35].

Other modalities are available for surgeon usage including ultrasonic debridement devices and pulse lavage systems, and these should be used according to surgeon judgment. Negative-pressure therapy is another modality widely used in the diabetic foot infection setting. Negative-pressure therapy aids in exudate management, decreases the bacterial bioburden thru serial debridements with vac changes, and stimulates angiogenesis to the area. Newer systems even include timed irrigation of the wound sites to further decrease the bacterial load. In a randomized trial evaluating vacuum-assisted wound closure including 342 patients with diabetic foot

ulcers, complete ulcer closure was achieved more often among those who used vacuum-assisted closure than those who did not (43% vs. 29%, respectively) [38]. This should be considered on a case-by-case basis.

Many surgical debridements of diabetic foot infections require multiple-staged procedures. It is during these follow-up procedures that one should consider adjunctive procedures to correct the structural or biomechanical abnormality that contributed to the development of the infection. As previously noted, these patients' tissues undergo glycosylation and lose their elasticity [8]. Therefore, the surgeon should consider soft-tissue contractures, as well as skeletal structural abnormalities. Without addressing these issues, the patient will be left in a compromised position and odds of successful limb salvage in jeopardy. Specifically, a gastroc or TAL has been shown to reduce forefoot pressures by 27%, thus reducing the risk of further ulceration [39]. Also falling into this category are those patients with Charcot deformity. Although this topic is too broad to cover in the scope of this chapter, these deformities should also be addressed whether surgically or with bracing to assure long-term success.

Case 4 (Fig. 4)
This patient presents with a severe diabetic foot infection with an ischemic third toe, ascending cellulitis to the ankle and lower leg. This is a medical emergency as the patient is septic and the infection is progressing proximally. An aggressive incision and drainage of the foot were performed, halting the infectious process, and resection of the third toe was done. A split-thickness skin graft, harvested from the right thigh, was applied to the former infected site following multiple debridements associated with adjunctive care to prepare the wound bed for skin grafting. This patient had continued local wound care until the all wounds were completely remodeled.

6 Osteomyelitis

Osteomyelitis is of great concern as these patients are at higher risk for limb loss. Certain clinical findings can support the diagnosis of osteomyelitis. In two systematic reviews that evaluated the diagnostic accuracy of exam findings in the setting of diabetic foot ulcers, the following factors increase the likelihood of osteomyelitis: grossly visible bone or ability to probe to bone, ulcer size larger than 2 cm^2, ulcer duration longer than 1–2 weeks, and erythrocyte sedimentation rate (ESR) >70 mm/h [40, 41]. If the radiograph is indeterminate or normal and the diagnosis remains uncertain, such patients should undergo magnetic resonance imaging (MRI), which is highly sensitive and specific for osteomyelitis and superior to radiographs, three-phase bone scans, and white blood cell scans [40–43]. Biopsies and cultures of the bone in question remain the gold standard at guiding empirical therapy, and possible surgical debridement. In one retrospective study of diabetic patients with osteomyelitis of the toe or metatarsal head, remission (absence of signs of infection and no need for surgery after 1 year) was more likely in the 22 patients treated with regimens guided by bone biopsy data compared with the 28 treated based on swab culture data (82% vs. 50%) [33].

Case 5 (Fig. 5)
This is a patient who came into the emergency room with a limb-threatening infection. The patient was septic and the infection involved the soft tissue and bone of the plantar right foot. Deep tissue and bone cultures and biopsies were performed. The patient was treated with long-term intravenous antibiotics and a multilevel external fixator was applied for stability and to maintain anatomical alignment. The patient underwent serial debridements in order to prepare the wound bed for skin grafting. Amputation of the fifth digit and ray was performed. A split-thickness skin graft was harvested from the thigh and applied to the foot. Once the wounds healed, the external fixator was removed and the patient was placed into an AFO and a pair of accommodative custom-made diabetic shoes to assist with his gait and function and provided continued independence.

Case 6 (Fig. 6)
This is a patient who was seen in the ICU of the hospital with an extremely elevated white blood cell count. The patient was septic and in a diabetic

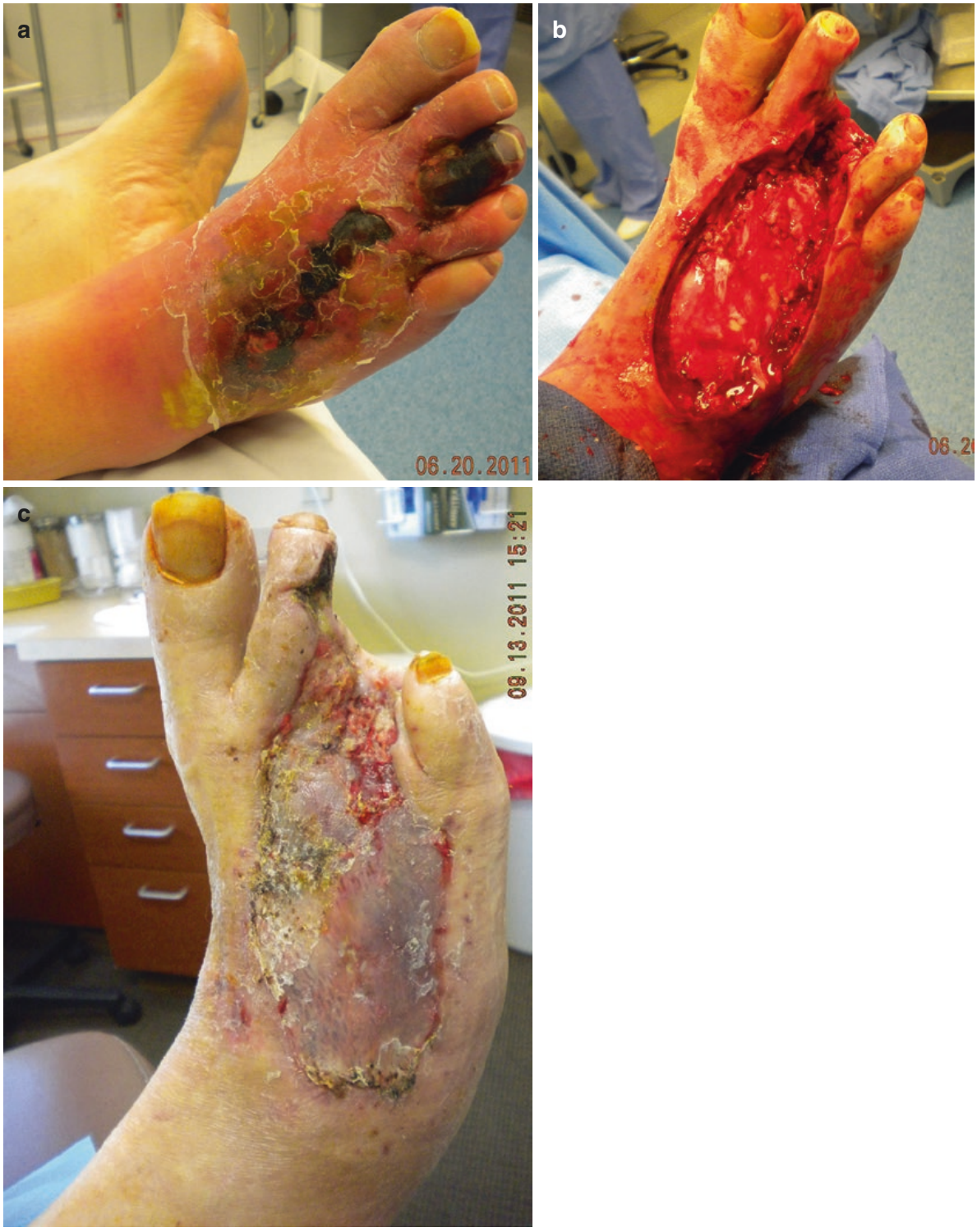

Fig. 4 (**a**) Patient with severe diabetic foot infection. (**b**) Aggressive incision and drainage of the foot. (**c**) Following split-thickness skin graft

coma. He presented with red, hot, swollen ankle joint that was very fluctuant. There was valgus deformity of the ankle that caused a diabetic ulcer to the medial ankle leading to the diabetic ulcer of the medal ankle and a portal to the ankle joint. The talus was dislocated from the tibial talar joint

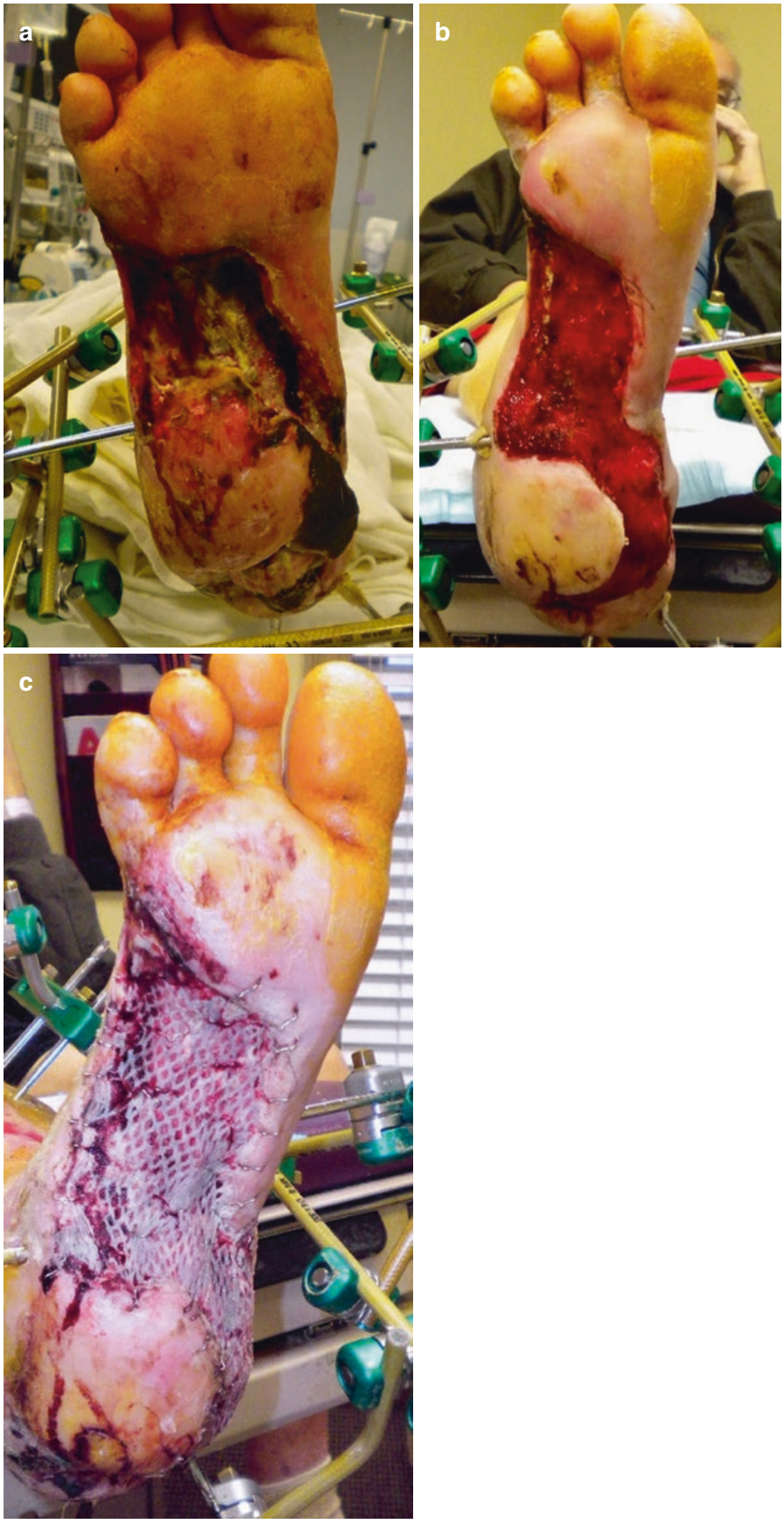

Fig. 5 (**a**) Preoperative for the second debridement following an initial debridement and application of external fixations for gross instability of the mid foot and hind foot. The patient was admitted for sepsis stemming from a Charcot foot and ankle deformity. (**b**) Following multiple serial debridements, amputation of the fifth digit and ray demonstrating good granulation tissue and coverage over the osseous and soft-tissue defects. Note that the external fixation provides excellent stability. (**c**) Following a split-thickness skin graft that was harvested from the thigh and applied to the foot after multiple serial soft-tissue and bone debridements. Once the wounds healed, the external fixator was removed and the patient was placed into an AFO and a pair of accommodative custom-made diabetic shoes to assist with his gait and function and provided continued independence

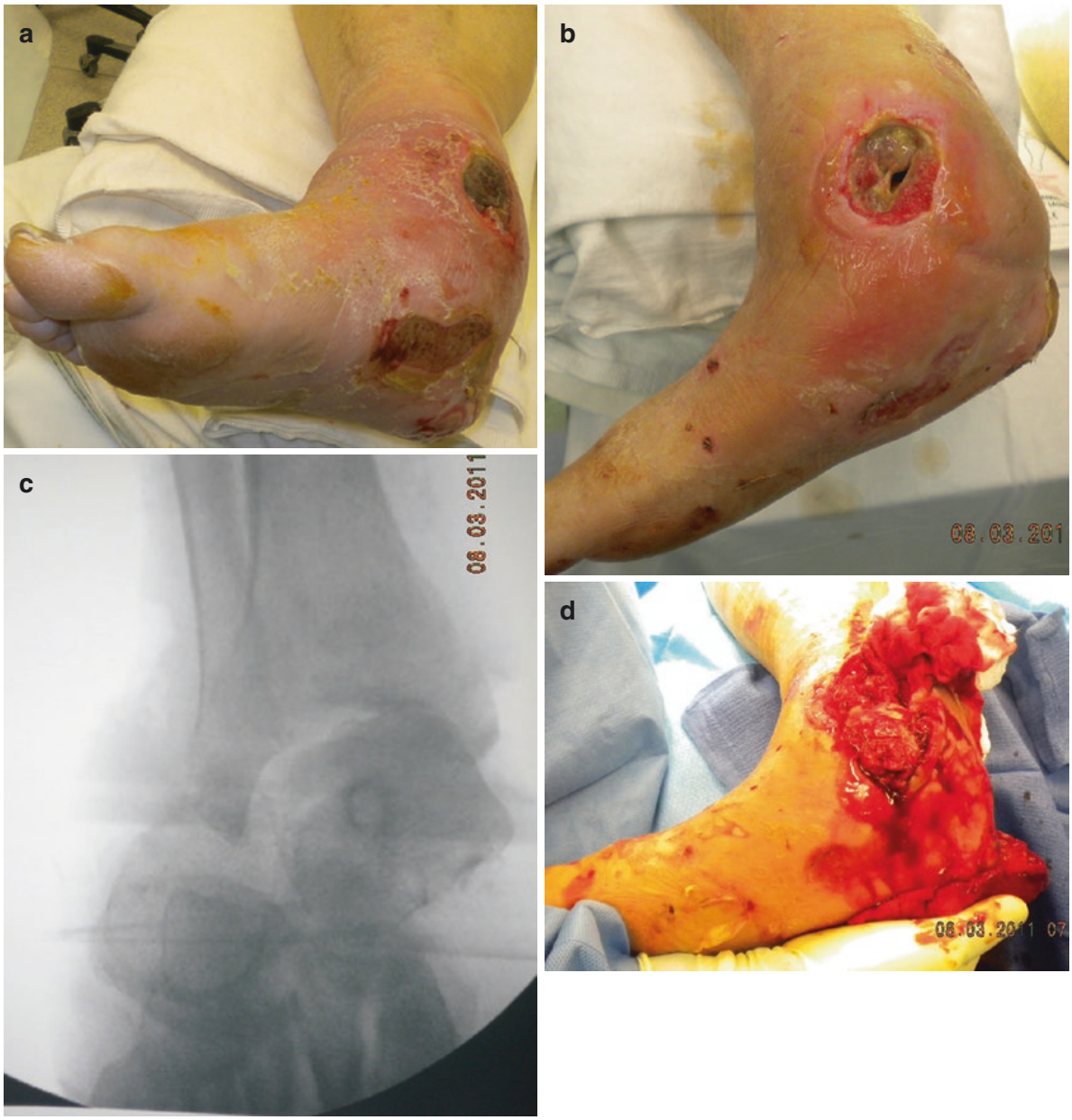

Fig. 6 (**a**, **b**) This is a patient who was seen in the ICU of the hospital with an extremely elevated white blood cell count. The patient was septic and in a diabetic coma. He presented with red, hot, swollen ankle joint that was very fluctuant. Note the valgus deformity of the ankle that caused a diabetic ulcer to the medial ankle and a portal to the ankle joint. (**c**) The talus dislocated from the tibial talar joint as well as the subtalar joint secondary to severe infectious process of the ankle and subtalar joint. (**d**) An incision drainage with an aggressive resection of bone and soft tissue of the right ankle. (**e**) The talus was resected from the ankle joint. (**f**) An antibiotic-impregnated bone cement (polymethyl methacrylate) shaped similarly to the talus to fill the void and the dead space following the talectomy. The antibiotic spacer will provide and elude high doses of local antibiotics in combination with intravenous antibiotics to treat the osteomyelitis of the foot and ankle. (**g**) An external fixator was applied for stability and to maintain anatomic alignment. (**h**, **i**) Following multiple soft-tissue and bony debridements and long-term IV antibiotics, all inflammatory markers were stabilized and negative cultures were maintained. A reconstructive tibial calcaneal arthrodesis was performed providing excellent anatomic alignment, stability, and plantigrade foot and ankle allowing the patient to maintain function and independence

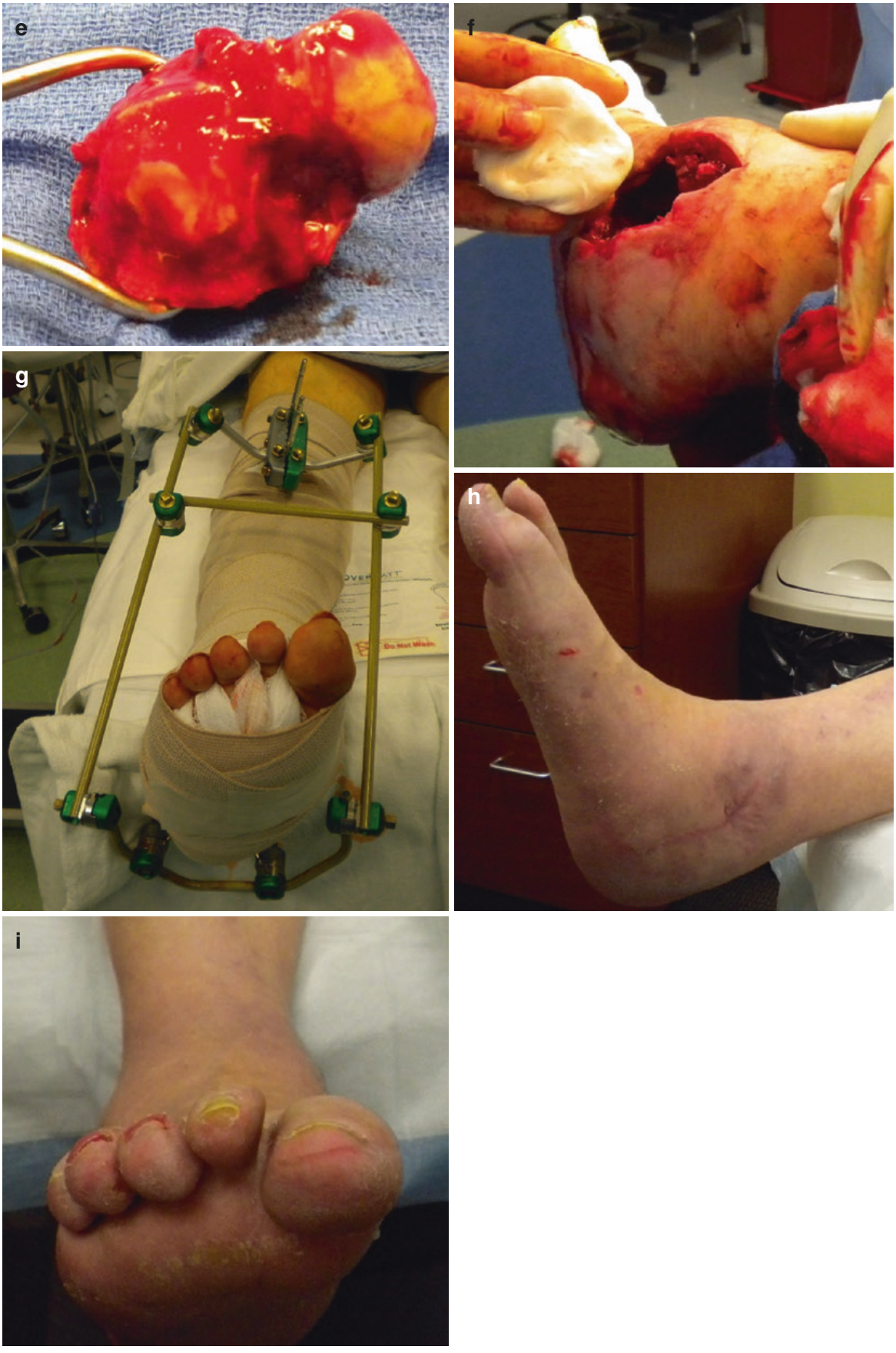

Fig. 6 (continued)

as well as the subtalar joint secondary to severe infectious process of the ankle and subtalar joint. An incision and drainage were performed with aggressive resection of bone and soft tissue of the right ankle. The talus was resected from the ankle joint and an antibiotic-impregnated bone cement (polymethyl methacrylate) was shaped similar to the talus to fill the void and the dead space following the talectomy. The antibiotic spacer will provide and elude high doses of local antibiotics in combination with intravenous antibiotics to treat the osteomyelitis of the foot and ankle. An external fixator was applied for stability and to maintain anatomic alignment. Once all inflammatory markers were stabilized and negative cultures were maintained a reconstructive tibial calcaneal arthrodesis was performed providing excellent anatomic alignment, stability, and plantigrade foot and ankle allowing the patient to maintain function and independence.

7 Postsurgical/Long-Term Care

Maintenance of these patients is of utmost importance. One prospective study found a 70% 5-year recurrence rate among diabetics who primarily healed a foot ulcer [44]. Close monitoring, daily foot checks, and extreme diligence help prevent recurrence and early recognition of potentially hazardous complications. Good local wound care, off-loading, and accommodative shoe gear help reduce the risk of infection and need for possible amputation. A nonhealing ulcer precedes 85% of lower extremity amputations in diabetics [25, 26, 44]. Regular assessment for changes in vascular status should also be monitored. Noninvasive vascular studies should be considered on a yearly basis, or if a wound has not progressed by 50% with 4 weeks of standard local wound care. These patients quite often require custom bracing to achieve proper off-loading or accommodation for amputations. This should be handled by a qualified pedorthotist, and the patient should be checked routinely in case alterations or adjustments are needed.

Studies over the past two decades have established that the majority of diabetic foot ulcers take at least 20 weeks to heal [16, 17, 31]. Given these statistics, it is clear why aggressive wound care is necessary to facilitate closure and reduce the risk of infection and amputation. The longer the wound remains open, the greater the risk. Creation of an environment conducive to healing will remain the foundation of good foot care in diabetic patients.

Conclusions

Limb salvage in a diabetic patient who is suspected of having a deep-space infection should be treated as early and aggressively as possible. This patient population can change abruptly for the worst given the circumstances if not treated appropriately. If in question, the physician should utilize all diagnostic modalities as needed as well as his/her clinical skills to make the diagnosis and error on the side of being aggressive with a surgical intervention. It has been the authors' experience that those patients who have been mistreated/undertreated continue to be at risk for limb and sometimes life-threatening scenarios. In the event that the patient has a component of peripheral vascular disease in the face of an infection, it is necessary for the foot and ankle physician to halt the infection and stabilize the patient and then consult vascular surgery for possible vascular reconstruction. The goal is to save a life and then a limb. If a patient has an infected extremity, the vascular surgeon cannot perform a vascular reconstruction in the event of a limb-threatening infection; therefore it is priority for the foot and ankle surgeon to halt the infection and stabilize the patient. Once the infection is halted and the wounds heal, reconstructive foot and ankle surgery can be performed in order to provide a stabile, plantigrade foot/ankle to allow the patient independence and function.

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