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Case Report

Calf Pressure Ulcers along with Typical Pressure Ulcers - A Complication of Hemodynamic Shock

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Received: June 24, 2021; **Published:** July 28, 2021

Abstract

Pressure ulcers are localized areas of damage to the skin and underlying tissues where decubitus provokes high interstitial pressures causing capillaries to collapse. According to longtime accepted data the capillary closure pressure is 30 - 32 mmHg. Since the body-interface pressure is 10 - 20 mmHg on the calves it appeared inconceivable that pressure ulcers would arise on the calves. Observations from the bedside oppose this concept. The case of an elderly person who developed pressure ulcers following septic shock on each calf along with a presacral pressure ulcer lends support to a different, broadened understanding of the pathophysiology of pressure injury.

Keywords: *Pressure Injury; Pressure Ulcer; Skin Ulcer; Skin Failure*

Introduction

Decubitus provokes high interstitial pressures which distort and collapse the capillaries where soft tissues are compressed between external surface and a bony prominence [1-4]. When lasting for several hours the resulting tissue hypoxia progresses to tissue necrosis. Traditionally it is agreed that the capillary closing pressure measures 30 - 32 mmHg [5]. The tissue pressures with the patient supine are at the sacrum, buttocks, heels and occiput are in the range of 50 - 60 mmHg [5]: these areas are predisposed to pressure injury [1,2]. At a difference, the interface pressure between the calf and support surface is 10 - 20 mmHg, not enough to collapse the capillaries. Hence, according to the classical concept, pressure ulcers could not occur on the calves.

Calf ulcers are common in practice, occurring in a variety of circumstances: as a complication of venous insufficiency, vasculitis, metabolic disorders, neoplasia, infection, the effect of medications. The configuration of these ulcers differs from the appearance of pressure ulcers. Also different are the features of the skin adjacent to the ulcer [6]. In distinction to the latter, several patients referred to our institution had calf ulcers which appeared to be genuine pressure ulcers. Yet, perusing the literature under the key wards “calf, calves” AND “pressure ulcer, pressure sore, decubitus ulcer, bed sore, pressure injury” we could not find reference to calf pressure ulcers, except two recent references of ours [7,8].

Case History

A 81-year-old man was admitted to our word for treatment of pressure ulcers, which had evolved during a recent hospitalization for severe sepsis, shock, and acute renal failure. Having received empirical antibiotic treatment at home before referral to hospital, the blood and urine cultures were unrewarding. Under wide spectrum antibiotics and supportive care in the intensive care unit he was slowly recovering. Pressure injuries were revealed after 5 days after his admission to hospital. His medical history included ischemic heart disease, diabetes mellitus, arterial hypertension, moderate severity aortic valve stenosis, chronic atrial fibrillation, and neurocognitive decline (minimental score 20/30). Admitted to our ward for aftercare the patient was alert, eating with appetite, his blood pressure and other vital signs were within the normal range. A systolic grade 3/6 ejection murmur was perceived over the aortic focus radiating to the left clavicle. The tibialis posterior and dorsalis pedis pulses were palpable. There was a stage 3 typical presacral pressure ulcer 7 cm in diameter, and stage one stage 3 skin ulcer on each calf (Figure 1).

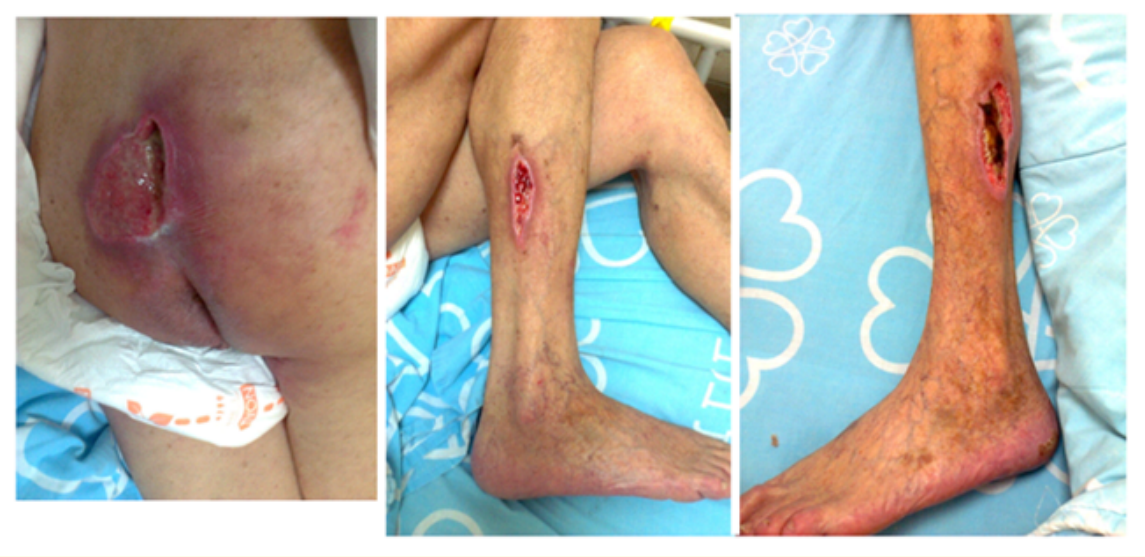

Figure 1: *Stage 3 presacral and calf pressure ulcers.*

The skin ulcer on the right calf, 6 cm long, 2.5 cm wide and 3 mm deep had granulation tissue at the base and a broad rim of new epithelium at the periphery. A skin ulcer on the left calf of similar size was partly covered by escare, re remainder covered by granulation tissue and enclosed by a rim of epithelium. There was a moderate melanic pigmentation of the left calf, no edema, no venectasia of venous varices. The blood cell count and routine chemistry tests were normal with the exception for a mild normocytic anemia. The patient by oral route 35 Kcal/kg/day and protein 1.2 g/kg/day. The ulcers were treated topically with saline gauze dressings and the patient continued receiving his usual medications of insulin glargine and glulisine, tb. apixaban 5 mg b.i.d, tb. amiodarone 200 mg, tb. bisoprolol fumarate 2.5 mg b.i.d., and tb. Nexium 20 mg.

Discussion and Conclusion

The calf ulcers in the proposito differ from venous, ischemic, neuropathic and stasis ulcers as well as from vasculitic, metabolic, infectious, neoplastic skin ulcers, pyoderma gangrenosum and calciphylaxis [1,2,5]. Except for the elongated shape, contrasting to the round

shape of pressure ulcers at other sites, the calf ulcers were like typical pressure ulcers [1,2]. In our prior experience of six patients with calf pressure ulcers, all having the elongated shape, similarities with 'typical pressure ulcers' were highlighted. Alike typical pressure ulcers their borders were regular and well demarcated, the adjacent skin appeared normal, the calf pressure ulcers occurred simultaneously with typical pressure ulcers during shock or other critical state [7,8].

It is suggested that underreporting of calf pressure ulcers might be due to the conflict between bedside observation and the ischemic concept of pressure injury. Recently, the pathophysiology of pressure injuries was revised [9]. The role of systemic factors has been appraised in modifying tissue tolerance to deformation and ischemia. Hypotension, blood flow redistribution, proinflammatory cytokines, acidosis and electrolyte disturbances might impact the tissues' tolerance to ischemia. It has been recognized that there is no fix threshold of tissue pressure above which injury develops, rather different levels of tissue pressure become critical depending on systemic and local influences [10].

Indeed, most pressure ulcers emerge during hospitalization for acute severe illness [2]. Skin failure may be part and parcel of multiorgan failure, characterized by rapid onset and rapid tissue breakdown due to hypoperfusion [11,12]. The concept of acute skin failure as a part of the multiorgan system failure has been proposed along with end-stage skin failure as part of the dying process. Skin failure fits into the emerging concept of unavoidable pressure ulceration [12]. There is a spectrum of pressure injuries between two extremes: a) contained injury due to prolonged immobility, no systemic organ failure, b) generalized skin failure revealed by numerous, rapidly evolving pressure ulcers in the context of shock and multisystem failure, c) in the absence of generalized skin failure, pressure ulcers occurring on typical sites along with pressure ulcers on sites exposed to less interface pressure.

In conclusion, the present case adds to the emerging evidence that calf ulcers may be genuine pressure ulcers as part of systemic pressure injury.

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Volume 4 Issue 8 August 2021
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