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A rare case of Meleney's Ulcer after partial chemical matricectomy

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ABSTRACT

Background. Meleney's ulcer is a rare, but potentially deadly infection that often occurs in post-surgical sites. This type of ulcer has not previously been reported in the toenail after phenol matricectomy.

Patient Case. A female patient underwent partial phenolization of the medial nail matrix of the hallux, but after 2 months had a recurrent spicula that caused Meleney's ulcers.

Results. The ulcers remained after treatment with antibiotics, and further surgery was required to fully clear the infection.

Conclusion. This case and review of Meleney's ulcer highlights the deceptively benign initial presentation of necrotizing fasciitis at the hallux after partial chemical matricectomy surgery using a phenol-based approach.

Un caso poco frecuente de úlcera de Meleney tras matricectomía química parcial

RESUMEN

Antecedentes. La úlcera de Meleney es una infección poco frecuente, pero potencialmente grave que ocurre a menudo en el postoperatorio. Este tipo de úlcera no ha sido anteriormente descrita en el pie tras la realización de una matricectomía parcial con fenol.

Caso Clínico. Una paciente fue intervenida mediante fenolización parcial de la matriz ungueal medial del dedo gordo del pie, y transcurridos 2 meses tuvo una recidiva de la deformidad ungueal que causó las úlceras de Meleney.

Resultados. Las úlceras se mantuvieron sin cicatrizar aún pautando un régimen de tratamiento con antibióticos, por lo que se requirió de una nueva intervención quirúrgica para eliminar completamente la infección.

Conclusión. Este caso presenta una úlcera de Meleney destacando una presentación inicial aparentemente benigna de la fascitis necrotizante en el hallux tras la realización de una matricectomía química parcial mediante abordaje quirúrgico utilizando fenol.

INTRODUCTION

Meleney's ulcer is a rare, but potentially deadly infection that often occurs in post-surgical sites. A type of necrotizing fasciitis, Meleney's ulcer is also referred to as "progressive bacterial synergistic gangrene" due to the synergistic effects of multiple bacterial infections believed to contribute to its severity[1]. This progressive gangrene was first described by New York surgeons Dr. Brewer and Dr. Meleney in 1926[2], and the contributing microbial effectors further characterized in 1931[1]. The eponymic term, Meleney's ulcer, is also often replaced with the term Progressive Necrotizing Infection.

This type of wound begins as a small superficial ulcer post-surgery, followed by infection of subcutaneous tissue leading to small vessel thrombosis and eventual necrosis. The infection is polymicrobial, hence the synergistic identification often used to describe the infection. Both microaerophilic and aerobic microorganisms (microaerophilic, non-hemolytic *Streptococcus* and a hemolytic *Staphylococcus aureus*) flourish in the wound site, typically at the advancing edge of the ulcer[3]. The lesion is slow growing and often presents within a few weeks of a surgical procedure. Meleney's ulcers can present on any part of the body, but are quite often found on the trunk in response to some form of abdominal surgery.

Although our understanding of the pathophysiology of necrotizing fasciitis continues to improve, physicians confronted with this clinical entity continue to be faced a condition which unfortunately can present with delayed or even missed diagnoses leading to tragic consequences. In this article, we present one of our recent cases to highlight the deceptively benign initial presentation of necrotizing fasciitis at the hallux after partial chemical matricectomy surgery using phenol.

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CASE REPORT

Affiliation. The patient was a 45 year old Caucasian female with unknown past medical history.

Present illness. The patient underwent partial chemical matricectomy 2 months prior with a normal post-operative examination. The patient claimed that one week prior to the current examination, she experienced redness, and felt pain at the site of phenolization. She noticed an itchy, scaly red patch of skin at the medial border of the hallux with ulcers that were increasing in size, which motivated her to seek consultation.

Clinical examination. Erythema was present and the site was tender to palpation. The pain described suggested a soft-tissue infection. Two ulcers were present at the medial border of the right hallux and one ulcer was found under the nail plate of the medial fold of the right hallux (figure 1). Exudative discharge was also observed at the site of pain. The patient had no fever, and no popliteal, or inguinal lymph node involvement.

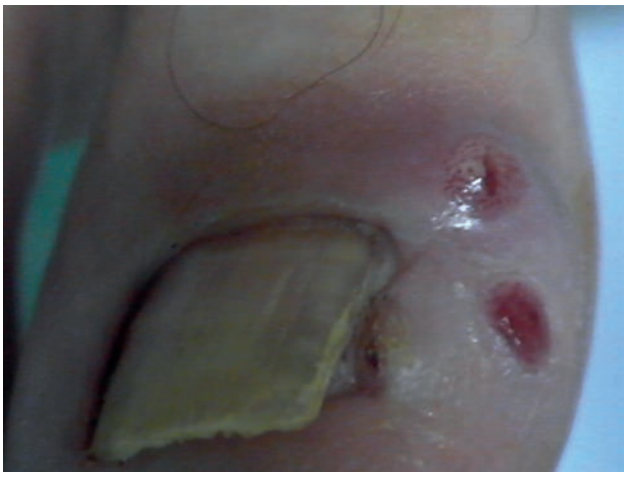

Figure 1 Two ulcers were present at the medial border of the right hallux and one ulcer was found under the nail plate of the medial fold of the right hallux

Complementary examinations. Blood counts, blood chemistry and liver lipid profile were all within normal limits. Cultures of the secretion from the ulcers were positive for *S. aureus* and *Proteus mirabillis*. The probe to bone test was positive (figure 2) and an X-ray was taken to rule out osteomyelitis.

TREATMENT

The patient received oral antibiotics (ciprofloxacin 500 mg/12 hours in combination with clindamycin 150 mg/6 hours) for 10 days based on the antimicrobial susceptibility observed in the disk diffusion test. In the first 24 hours, the patient's symptoms improved; and after 10 days, the injury was resolved. However after 20 days, although the wounds appeared to be resolved, the medial matrix of the right hallux was still erythematous and the zone was indurated, slightly swollen, and tender to palpation. No exudation was found. The patient then underwent a partial medial matricectomy of the nail matrix of the hallux using the Winograd technique and remained without recurrence for nine months. These initial ulcers were deemed to be Meleney's ulcers.

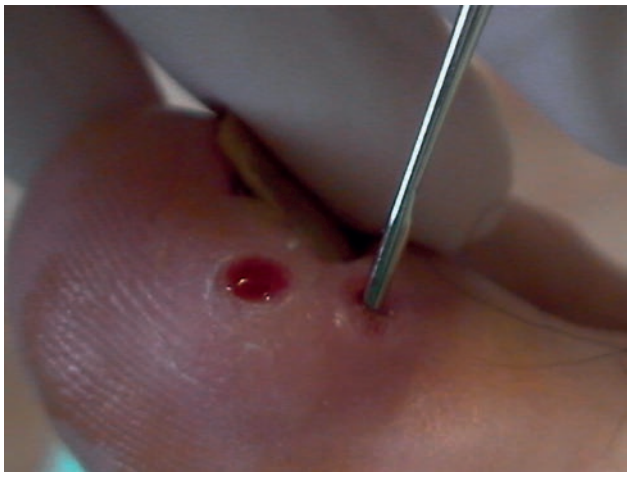

Figure 2 The probe to bone test

DISCUSSION

Forms of necrotizing fasciitis. Different forms of necrotizing fasciitis can have a rate of spread measured in hours up to weeks. Histologically, necrotizing fasciitis is characterized by angiothrombotic microbial invasion of the fascia. As the process progresses, occlusion of perforating nutrient vessels to the skin causes ischemia. An intermediate stage includes cutaneous blisters, later developing large hemorrhagic bullae, frank cutaneous gangrene, and sensory motor deficits[4].

Necrotizing fasciitis exists in two forms. Type 1 is a polymicrobial infection caused by the synergistic effect of anaerobic and aerobic bacteria. This type most commonly affects people with a compromised vascular supply, or results after surgical procedures. The Type 2 form is caused by *Streptococcus pyogenes*, also known as Group A (beta-hemolytic) *Streptococcus* (GAS) either alone or in combination with *S. aureus*. This form is more often described in immunocompromised patients[5].

Meleney's ulcer falls under the Type 1 form of necrotizing fasciitis. This progressive bacterial synergistic gangrene is a slow progressive superficially necrotizing fasciitis of the skin and subcutaneous fat. It has an additional feature over typical Type 1 necrotizing fasciitis involving burrowing necrotic tracts through tissue planes that emerge at distant sites in the skin[6]. Total skin thickness is affected, but not deep fascia[7]. This "undermining burrowing ulcer" is extensively destructive to the cutaneous skin layer and creates multiple ulcers separated by bridges of skin[6].

Diagnosis. Unfortunately, some of the early signs of Meleney's ulcer are vague and common, so its diagnosis is often overlooked in the beginning stages when treatment would be optimal. This delayed recognition and, in turn, the delayed treatment increases its associated mortality rates. The diagnostic criteria for Meleney's ulcer includes: a slowly progressive superficial necrotizing process; evidence of a variety of microaerophilic, anaerobic, facultative, or amoebic organisms; hypoxic wound environment; and microvascular thrombosis in a full thickness ulcer. Histology depicts this lesion with microvascular thrombosis in the dermis followed by liquefaction, and the overlying epidermis can become devascularized and necrotic. The macroscopic picture represents a full skin thickness ulcer with a rolled necrotic margin, bordered by a zone of painful erythema, denoting the sub-epidermal spread of the infection.

Treatment. When Meleney first described this condition in 1926, the treatment of choice was zinc peroxide and surgical debridement. As new antimicrobials were introduced, these provided other options to attempt to clear the infection6. Antimicrobials can be an important tool for managing necrotizing fasciitis; however, although they reduce the systemic bacterial load, they often have little effect on the primary site of disease. Thus, antibiotic treatments may temporarily mask the severity of the true underlying disease. In fact, a hallmark of necrotizing fasciitis is that infection always progresses in the face of antibiotic therapy. Thus, surgical tissue debridement is necessary for treatment of the affected tissue[4]. Unfortunately, debridement tends to open up new channels in the normal surrounding tissue where microorganisms can spread and maintain infection. Often, subsequent surgeries are required to fully remove the affected tissue, followed by reconstructive surgery to ameliorate the damaged area. Additional treatment measures are often included with the surgical approach to avoid future surgical interventions.

One such additional treatment option is hyperbaric oxygen therapy. The combination of increased pressure and high oxygen concentrations allows for large amounts of oxygen to be dissolved into the blood and tissues, allowing for the revitalization of tissues with poor circulation. In one study, the addition of hyperbaric oxygen therapy to the surgical and antimicrobial treatment of necrotizing fasciitis significantly reduced mortality and the number of surgical debridement procedures needed, leading to the conclusion that this additional approach should be routinely used in the treatment of necrotizing fasciitis[8].

Specifically, for Meleney's ulcer, hyperbaric oxygen therapy is beneficial in the acute phase by inhibiting the growth of anaerobic or microaerophilic organisms, enhancing neutrophil function that has been compromised by hypoxia, and increasing the efficacy of certain antibiotics (in particular, those that require oxygen dependent intracellular transport). After the necrotic spread has been terminated, hyperbaric oxygen therapy may further promote healing by stimulating angiogenesis and granulation tissue formation, as in other medical conditions for which this therapeutic approach is used.

CONCLUSIONS

Unfortunately, many initial signs of Meleney's ulcer are initially overlooked, leading to a higher than necessary mortality rate caused by this infection. In the post-surgical patient, signs of sepsis, wound dehiscence and discharge at the operative site may suggest Type 1 necrotizing fasciitis. Here, we report an example of Meleney's ulcer following phenol-matricectomy, a surgery that has not been reported to be fraught with this complication, thus emphasizing its evasiveness of diagnosis. To better diagnose this post-surgical complication, tissue samples should be isolated for gram stain and anaerobic/aerobic culturing to guide the true diagnosis and most appropriate therapy[5]. Further, alternative therapies should be integrated in conjunction to debridement, including potential antimicrobials and hyperbaric oxygen therapy.

The margin of the Meleney's ulcer is advanced by the synergistic effect of two organisms growing in a hypoxic environment. Some reports have shown that the wound can also be further colonized by other organisms, such as *Amoeba Proteus*. It has been indicated that the importance of *Entamoeba histolytica*, an anaerobic parasitic protozoan, could be a prime contributor to this infection based on retrospective studies that the clinical presentation of Meleney's postoperative progressive synergistic gangrene and postoperative amoebic skin gangrene were indistinguishable[9]. Regardless of the true initial cause, there are effective treatment options available for this infection if it is recognized in time. Thus, healthcare providers must be aware of this condition and should be proactive in its diagnosis and treatment.

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