Enterobacter Hormochae in a Diabetic Wound

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Abstract

In the treatment of chronic wounds, the amount to which bacteria obstruct wound healing is a point of contention. Clinicians must evaluate a large number of chronic wounds using identical sequencing and bioinformatics methods in order to select appropriate empiric therapies due to the high diversity and extreme variability of the microbiota between individual chronic wounds, which leads to inconsistency in small cohort studies. The oxidase-negative gram-negative rod Enterobacterhormaechei (E. hormaechei) was initially discovered as a distinct species in 1989. The fungus E. hormaechei can be found in a variety of habitats. It's widely thought to be a pathogen that causes nosocomial infections, however it doesn't normally infect animals other than humans.

Keywords: Diabetic ulcer; Wound; Enterobacterhomochae.

Introduction

Diabetic foot is ulceration in the lower leg associated with neuropathy and/or peripheral vascular disease in a diabetic patient. It is one of the most significant and disabling symptoms of the disease. Diabetes foot ulcers affect 4–10 percent of diabetics, with the elderly being more susceptible.[1–3] Foot ulceration affects about 5% of people with diabetes mellitus, and the overall lifetime risk of patients with diabetes who are prone to developing this problem is 15%.[1–3] The vast majority of diabetic ulcers, 60–80%, will heal, although 10–15% will remain very active, and 5–24% of these people will require limb amputation within 6–18 months of the initial evaluation. Neuropathy-related wounds heal in about 20 weeks, whereas neuroischemic ulcers take longer to heal and are more likely to require amputation.[4] Diabetic individuals account for 40–70% of all non-traumatic lower limb amputations, according to research.[5] Furthermore, diabetic foot ulcers are seen in roughly 85% of all diabetes amputations, according to various research.[5]

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With age and the duration of diabetes, the total risk of foot ulceration and limb amputation rises.[6,7] Because of the poor impact on a patient's quality of life and the associated financial burden on the healthcare system, diabetic foot prevention is critical.[8]

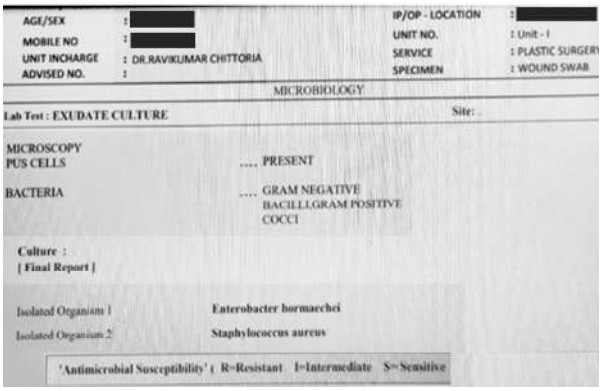
Diabetic foot ulceration is a serious medical condition that necessitates a multidisciplinary treatment plan. This review aims to provide a

comprehensive overview of current diabetic foot ulcer care methods, ranging from prevention to treatment options. In 1989, Enterobacterhormaechei (E. hormaechei) was found as a distinct species of oxidase-negative gram-negative rods. E. hormaechei can be found in a variety of environments. It is generally thought to be the cause of nosocomial infections, yet it seldom causes sickness in animals other than humans.

Materials and Methods

The study was carried out in a tertiary care hospital in South India, after receiving approval from institutional ethical council. The patient is a 40-year-old male with history of accidental abrasion over back of right ankle following which he developed unbearable pain and applied kerosene oil over the wound after which the wound progressed to an ulcer. Exudate and tissue culture showed Enterobacterhormochea (Figure 1).

He was managed with multiple sittings of hydrojet debridement, prolotherapy, insulin therapy heterografting, APRP, LLLT, and RONPWT for wound bed preparation. He received culture based antibiotics and underwent split thickness skin grafting STSG, LLLT, APRP for raw area cover. Then the response was assessed by Vancouver scar scale score. Scar was managed with coconut oil massage, silicon sheet application, compression garments.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UNIT NO. : Unit - I
SERVICE : PLASTIC SURGERY
SPECIMEN : WOUND SWAB

MICROBIOLOGY

Lab Test : EXUDATE CULTURE Site:

MICROSCOPY
PUS CELLS .... PRESENT
BACTERIA .... GRAM NEGATIVE BACILLI,GRAM POSITIVE COCCI

Culture :
[Final Report]

Isolated Organism 1 Enterobacter hormaechei
Isolated Organism 2 Staphylococcus aureus

'Antimicrobial Susceptibility' (R=Resistant I=Intermediate S= Sensitive

Fig. 1: Exudate culture showing Enterobacter Hormochae.

Results

Enterobacterhormochae is found in exudates and tissue cultures. Hydrosurgery and fast debridement using LLLT, APRP, and antibiotics based on culture. After two months of monitoring the patient with the aforementioned methods, the wound healing was much improved, and the graft uptake was effective.

Discussion

The most significant risk factors for foot ulcers include diabetes neuropathy, peripheral artery disease, and subsequent foot injuries. Diabetic neuropathy is responsible for over 90% of diabetic foot ulcers.[9] as well as the motor, sensory, and autonomic nerve fibres are all damaged by diabetes. Motor neuropathy causes muscle loss and atrophy.[10,11] Pain, pressure, and heat are no longer interpreted as protective signals due to sensory neuropathy. Vasodilation and decreased sweating are caused by autonomic dysfunction, which adds to skin deterioration and makes it sensitive to microbial infection.[12]

Diabetics have peripheral artery disease early, advances faster, and is often more severe than the general population. It most commonly occurs between the knee and the ankle. It has been shown to be a predictor of the prognosis of foot ulcers as well as an independent risk factor for cardiovascular disease.[13] When small injuries are aggravated by infection, the demand for blood in the feet increases, and a shortage of blood flow can lead to foot ulceration and limb amputation.[14] The majority of foot ulcers are of mixed aetiology (neuroischemic) 15, notably in the elderly.

Patients with peripheral diabetic neuropathy may develop foot ulcers as a result of recurrent minor injuries from either internal (calluses, nails, foot deformities) or external (shoes, burns, foreign substances) sources. This can cause an ulcer, which can lead to amputation of the foot, particularly in people with peripheral vascular disease. Poor glycemic control, cigarette smoking, and diabetic nephropathy, as well as a history of foot ulceration or amputation, are all risk factors for foot ulcers. In certain research, foot ulcers have been connected to diabetes.[14,16] Foot ulcers have also been linked to social factors like low socioeconomic level, limited access to healthcare, and a lack of educational accomplishment.[16,14]

Assessment and Classification

The skin, as well as the vascular, neurological, and musculoskeletal systems, are assessed during a diabetic foot physical examination. A visual assessment of the skin of the legs and feet, particularly the dorsal, plantar, medial, lateral, and posterior surfaces, as well as a careful examination of each toe nail, are all part of the dermatological examination. Another symptom to watch for is peeling skin, as well as maceration or fissuring of the interdigital skin. If autonomic neuropathy and

sudomotor dysfunction are visually identifiable, they are more likely to be diagnosed. Peripheral vascular insufficiency can obstruct ulcer healing, resulting in chronic, infection-prone ulcers.[15]

An ABI of less than 0.9 indicates peripheral vascular disease, which is associated with 50% or more stenosis in one or more main arteries. An ABI of 0.4–0.9 implies claudication related arterial obstruction to some degree. An ABI of less than 0.4 or a systolic ankle pressure of less than 50 mmHg indicate advanced ischemia.

An ABI of more than 1.3 indicates calcified vasculature. By measuring the blood pressure in the toe and computing the toe–brachial index, an accurate pressure can be achieved in such patients. ABIs and segmental pressures should be taken before and after treadmill exercise if ABIs are normal at rest but symptoms strongly imply claudication. This could reveal hemodynamically substantial subclinical stenosis that isn't noticeable at rest but becomes noticeable when exerted.

Diabetic neuropathy can be detected with a brief clinical history and a thorough examination. Burning, tingling, shooting, acute, or agonising symptoms, and muscular spasms, which are diffused uniformly in both appendages ("loading and glove dispersion") and are often more uncomfortable around evening time, are all prevalent side effects of fringe neuropathy. The Neuropathy Symptom Measure (NSS), a validated side effect score with a good predictive value for evaluating diabetic fringe neuropathy, can also be used to assess diabetic fringe neuropathy.

During the physical examination of the foot, the perception of superficial pain (pinprick), temperature feeling (with a two-metal rod), light sensation (with the edge of a cotton-wool twist), and pressure (with the Semmes–Weinstein 5.07 monofilament) are all measured. The doctor should also use a tuning fork and/or a biothesiometer to measure vibration perception. It's also a good idea to examine your proprioception (position sense) and deep tendon reflexes (Achilles tendon, patellar tendon).[4]

According to the American Diabetes Association, a foot that has lost its protective sensibility is termed a "foot at risk" for ulceration. The presence of a foot at risk is confirmed by a positive 5.07/10-g monofilament test, as well as one of the following tests: vibration test (using a 128-Hz tuning fork or a biothesiometer), pinprick feeling, or lower leg reflexes. If the patient has diabetes fringe neuropathy but no ischemia, the ulcer is defined as neuropathic; ischemic if the patient has diabetic fringe neuropathy but no ischemia; and neuroischemic if the neuropathy and ischemia are present at the same time. Other attempts have been made to classify foot ulcers according to severity, size and depth, location, contamination, and ischemia, in addition to this wide classification. One of the most well-known foot ulcer classifications is the Meggitt-Wagner order. Other frameworks for characterising diabetic foot ulcers have also been presented and approved.

E. hormaechei infection is common in intensive care patients and neonates, who typically contract it from contaminated food. These findings imply that E. hormaechei can infect immunologically deficient hosts. Previous studies have indicated that E. hormaechei can contaminate neonatal formula, and cases have been found in Italy, the Czech Republic, and Holland[11], demonstrating that the risk of animal-to-human transmission is not zero.

Conclusion

EnterobacterHormochae in a Diabetic Wound culture was a rare finding. We demonstrate that hydrosurgery is an effective and rapid debridement method that can be used safely along with LLLT, APRP and culture based antibiotics.

Conflicts of interest: None

Declarations: None

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References

1. Saseedharan S, Sahu M, Chaddha R, et al. Epidemiology of diabetic foot infections in a reference tertiary hospital in India. Braz J Microbiol. 2018;49(2):401-406.
2. Alexiadou, Kleopatra, and John Doupis. "Management of diabetic foot ulcers." Diabetes therapy: research, treatment and education of diabetes and related disorders vol. 3,1 2012.
3. Abbott CA, Carrington AL, Ashe H, North-West Diabetes Foot Care Study et al. The North-West Diabetes Foot Care Study: incidence of, and risk factors for, new diabetic foot ulceration in a community-based patient cohort. Diabet Med. 2002;19:377–384.
4. Centers for Disease Control and Prevention Lower extremity disease among persons aged ≥40 years with and without diabetes—United States, 1999–2002. MMWR Morb Mortal Wkly Rep. 2005;54:1158–1160.

5. Lauterbach S, Kostev K, Kohlmann T. Prevalence of diabetic foot syndrome and its risk factors in the UK. J Wound Care. 2010;19:333–337.
6. Katsilambros N, Dounis E, Makrilakis K, Tentolouris N, Tsapogas P. Atlas of the diabetic foot. 2. Oxford: Wiley-Blackwell; 2010.
7. Moxey PW, Gogalniceanu P, Hinchliffe RJ, et al. Lower extremity amputations—a review of global variability in incidence. Diabet Med. 2011;28:1144–1153.
8. Lavery LA, Armstrong DG, Vela SA, Quebedeaux TL, Fleischli JG. Practical criteria for screening patients at high risk for diabetic foot ulceration. Arch Intern Med. 1998;158:157–162. doi: 10.1001/archinte.158.2.157.
9. Malgrange D, Richard JL, Leymarie F, French Working Group On The Diabetic Foot Screening diabetic patients at risk for foot ulceration. A multi-centre hospital-based study in France. Diabetes Metab. 2003;29:261–268.
10. Prompers L, Huijberts M, Schaper N, et al. Resource utilisation and costs associated with the treatment of diabetic foot ulcers. Prospective data from the Eurodiale Study. Diabetologia. 2008;51:1826–1834.
11. Kumar S, Ashe HA, Parnell LN, et al. The prevalence of foot ulceration and its correlates in type 2 diabetic patients: a population-based study. Diabet Med. 1994;11:480–484.
12. Tesfaye S, Stevens LK, Stephenson JM, et al. Prevalence of diabetic peripheral neuropathy and its relation to glycaemic control and potential risk factors: the Eurodiab IDDM Complications Study. Diabetologia. 1996;39:1377–1384.
13. Brem H, Sheehan P, Boulton AJ. Protocol for treatment of diabetic foot ulcers. Am J Surg. 2004;187:1S–10S.
14. Bowering CK. Diabetic foot ulcers. Pathophysiology, assessment, and therapy. Can Fam Physician. 2001;47:1007–1016.
15. Management of peripheral arterial disease (PAD). TransAtlantic Inter-Society Consensus (TASC). Eur J VascEndovasc Surg. 2000;19(Suppl. A):S1–250.
16. Prompers L, Huijberts M, Apelqvist J, et al. High prevalence of ischaemia, infection and serious comorbidity in patients with diabetic foot disease in Europe. Baseline results from the Eurodiale study. Diabetologia. 2007;50:18–25.