



A Short Note on Clinical Study of Diabetic Foot Infections

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Commentary

In persons with diabetes, foot ulcers and other foot disorders are a leading source of morbidity and mortality. Diabetes foot infections (DFIs) are the largest cause of hospitalisation for diabetic patients globally, accounting for 20% of hospital admissions in developing countries like India. DFI is a multifactorial process in which three variables, namely neuropathy, peripheral vascular disease, and susceptibility to infection, lead to tissue destruction anytime there is a direct lesion to the foot at risk. DFIs are frequently polymicrobial infections produced by aerobic gramme positive cocci such as *Staphylococcus aureus*, gramme negative bacilli (*Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*), and anaerobes (*Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*) [1]. Appropriate antibiotic selection is required for proper management of these illnesses. The infections and susceptibility pattern seen in the community where the hospital is located are used to guide empirical treatment.

The most typically prescribed medications for bacterial infections are beta-lactam antibiotics. The most important danger to the therapy of such infections, particularly carbapenem resistance, is the rapid rise of antibiotic resistance to these families of medicines among the prevalent bacteria. The fact that these isolates are frequently multidrug resistant adds to the difficulty of the situation [2].

The NDM metallo-beta-lactamase (MBL) producing genes have recently emerged in numerous enterobacterial species as well as non-fermenters such as *P. aeruginosa* and *Acinetobacter baumannii* in many areas of the world, including India. In fact, the main reservoirs of blaNDM-like Enterobacteriaceae are India and Pakistan [3]. There is a scarcity of resources info on MBL-producing organisms from diabetic foot infections that possess the blaNDM-like gene. In order to guide empiric therapy for diabetic foot infections in our hospital, we investigated the microbial profile and susceptibility pattern of diabetic foot infections in patients with Type 2 Diabetes Mellitus, as well as the occurrence of the blaNDM-like carbapenemase gene among carbapenem-resistant gramme negative pathogens. According to their medical records a deep tissue sample was obtained. Collected from surgical wounds and sent for bacteriology. Cultures of bacteria and fungi.

The wound was cleaned thoroughly with normal saline after surgical debridement of the slough and necrotic tissue over the wound in the operating room; a deep tissue specimen of approximately 0.5 cm was removed from the wound bed. The sample was collected in a sterile container soaked in normal saline and immediately delivered to our microbiology laboratory for further analysis.

In the biosafety cabinet, a portion of the sterile deep tissue specimen was crushed or ground with a sterile mortar and pestle. For fungal culture, the crushed material was stained with gramme staining and streaked on 5 percent sheep blood agar (SBA), MacConkey agar (MA), and Seaboard's Dextrose agar (SDA). After inoculation, the SBA was stored in a candle jar and placed in an incubator at 37°C with the MA. The VITEK 2 Compact automated culture system was used to identify bacterial isolates and yeast-like fungi, as well as conduct susceptibility

tests (bioMérieux, France). The EDTA disc synergy test was performed using a commercial I+ IE (imipenem+ imipenem/EDTA) disc from Himedia in Mumbai, India. Metallo-beta-lactamase production was investigated in all carbapenem-resistant Enterobacteriaceae. The modified Hodge test (MHT) was developed [4]. Vitek was used to detect carbapenem resistance in all Enterobacteriaceae isolates, and an EDTA disc synergy test was used to detect metallo-lactamase synthesis in all gramme negative isolates, according to CLSI standards. Ceftazidime and Ceftazidime/clavulanic acid disc synergy tests were used to validate ESBL generation and AmpC -lactamases were detected.

The isolates were screened for presumptive AmpC production by utilising the Kirby Bauer disc diffusion method to determine their susceptibility to cefoxitin (30 g) and interpreting the results according to CLSI standards. 12 Screen positive isolates were those with an inhibition zone diameter of less than 18 mm [5].

On MHA plate, an *E. coli* ATCC 25922 lawn culture was produced. Several colonies of the test organism were injected on a sterile disc of 6 mm moistened with 20 L of sterile saline. On the infected plate, a cefoxitin disc was placed adjacent to this disc (nearly touching). At 37°C, the plates were incubated overnight. The cefoxitin inhibition zone in the vicinity of the disc flattens or indents. Alkaline lysis was used to extract total DNA from the various bacterial isolates, and PCR was used to detect the blaNDM-like gene, as described by Poirel. From January to June 2014, a prospective study was conducted on 261 diabetic patients with foot ulcers over a six-month period. The research took place in a tertiary care facility in Mumbai, India.

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Conflicts of Interest

The author has no known conflicts of interest associated with this paper.

References

1. Lipsky BA, Berendt AR, Embil J, De Lalla F (2004) Diagnosing and treating diabetic foot infections. *Diabetes Metab Res Rev* 20(S1): S56-S64.
2. Tardáguila-García A, Sanz-Corbalán I, García-Alamino JM, Ahluwalia R, Uccioli L, et al. (2021) Medical versus surgical treatment for the management of diabetic foot osteomyelitis: a systematic review. *J Clin Med* 10(6): 1237.

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3. Lipsky BA (1999) A current approach to diabetic foot infections. *Curr Infect Dis Rep* 1(3): 253-260.
4. Spichler A, Hurwitz BL, Armstrong DG, Lipsky BA (2015) Microbiology of diabetic foot infections: from Louis Pasteur to 'crime scene investigation'. *BMC Med* 13(1): 1-13.
5. Gariani K, Pham TT, Kressmann B, Jornayvaz FR, Gastaldi G, et al. (2021) Three Weeks Versus Six Weeks of Antibiotic Therapy for Diabetic Foot Osteomyelitis: A Prospective, Randomized, Noninferiority Pilot Trial. *Clin Infect Dis* 73(7): e1539-e1545.