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The Fetid Diabetic Foot- Common Micro Organisms Isolated & It's Sensitivity

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Abstract

In Diabetes mellitus, fetid foot infection is more common. On review of 30 patients of fetid Diabetic foot treated in Ahalia Diabetic Hospital, Palghat, during the period from April 2012 to December 2013. Out of which in 15 patients pseudomonas & in 5 patients Proteus were isolated. Both Pseudomonas & Proteus were sensitive only to Imepenem groups of Antibiotics.

In other 15 patients, MRSA- methicillin resistant Staphylococcus Aureus & E.Coli were isolated & they were sensitive to Augmentin, Amikacin, Gentamycin, & Piperacillin-Tazobactam.

Keywords: Diabetes mellitus; Pseudomonas; Proteus; Diabetic Foot.

Introduction

Foot is a complex structure with many layers of muscles, ligaments, joints, arches, fat, thick plantar fascia, vascular arches, and neurological system, all these maintains weight bearing, gravity, normal walk, stability, & gait.

Problems in Diabetic Foot

1. Callosities, ulceration
2. Abscess & cellulites of foot
3. Osteomyelitis of different bones of foot like metatarsals, cuneiforms, calcaneum.
4. Diabetic Gangrene
5. Arthritis of the joints.

Meggitt-Wagners Classification of Diabetic Foot-

Grade 0- foot symptoms like pain only

Grade 1- Superficial Ulcer

Grade 2-Deep ulcer

Grade 3-Ulcer with Bone involvement

Grade 4- fore foot gangrene

Grade 5-Full foot gangrene

Pathogenesis of Diabetic Foot/Gangrene

1. High glucose level in tissues is a good culture media for bacteria. So infection is common.
2. Diabetic microangiopathy- causes blockade of micro circulation leading to hypoxia.
3. Diabetic Neuropathy- due to sensory neuropathy, minor injuries are not noticed & so infection occurs. Due to motor neuropathy, dysfunction of muscles, arches of foot & joints occurs. Also loss of reflexes of foot occurs causing more prone for trauma & abscess. Due to Autonomic Neuropathy, skin will be dry, causing defective skin Barrier & so more prone for infection.
4. Diabetic Atherosclerosis itself reduces the blood supply & causes gangrene. Blockage occurs at plantar, tibial & dorsalis pedis vessels.
5. Increased glycosylated haemoglobin in blood causes defective oxygen dissociation leading to more hypoxia. At tissue level there will be increased glycosylated tissue proteins, which prevents proper oxygen utilization & so aggravates hypoxia.

Materials and Methods

This study was conducted on 30 Diabetic patients

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with fetid foot in Ahalia Diabetic Hospital, Palghat, Kerala. All the patients presented with foul smelling infection of foot with pain, swelling & Abscess or ulcers of grade 1 or 2 of Meggitt - Wagner's. Classification. Since because *the kerala state is well educated, Osteomyelitis & gangrene of foot is not so common.*

For all 30 patients, fasting & Postprandial Blood sugars, Glycosylated haemoglobin estimation, Renal function test, urine micro albumin, Lipid profile, Thyroid function test, Biothesiometry for peripheral neuropathy, hand arterial Doppler, ECG, X-ray Foot & Pus Culture & sensitivity all done.

In all Patients no Neuro Vascular Deficit

In Pus C&S, the common organisms isolated in 15 patients are Pseudomonas, 5 patients with proteus, all 20 patients were treated with Imepenem groups of drugs which were sensitive.

In remaining 10 patients, MRSA & E.Coli were isolated and treated with Augmentin, Amikacin or Gentamycin.

Discussion

All 30 patients had stage 1 & stage 2 diabetic foot involving superficial & deep tissues with foul smelling fetid foot. No involvement of Bone or Neuro vascular structures. This Fetid foot is due to poor control of blood sugar and foot care. All the patients are treated with wound debridement & regular dressings, appropriate antibiotics, good diabetic control by Insulin.

Conclusion

By this study, the Fetid Diabetic foot without involving bone & Neuro vascular structures can be treated nicely with appropriate antibiotics by Pus culture & sensitivity and wound debridement with daily dressings, also insulin to control blood sugar.

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