Colchester Hospital University NHS Foundation Trust
North East Essex Clinical Commissioning Group

# Guidelines for the management of Diabetic Foot Infection

Written by Podiatry supported by NEEDS and Microbiology.
Approved by North East Essex Medicines Management Committee
October 2015

Review Date October 2017

# Contents

## Summary of Recommendations

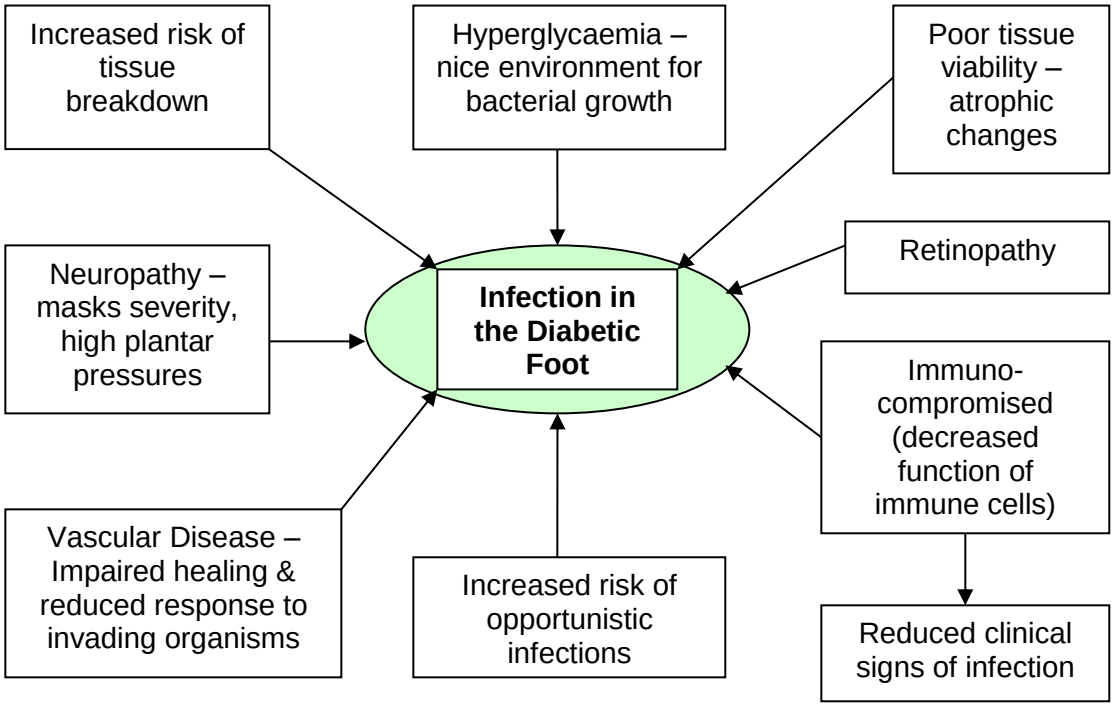
- All patients presenting with diabetic foot infection should be referred to a member of the multidisciplinary team for expert assessment within 24 hours.
- Where clinical infection is suspected microbiological sampling is advocated particularly in those with chronic infections or who have recently been treated with antibiotics.
- Diagnosis of an infection should be based on clinical findings. Cultures are meant to identify organisms and to assist in treatment of an infection rather than be used to diagnose infection.
- When clinical signs of infection are present the patient should be issued immediately with an empiric antibiotic regimen on the basis of the severity of the infection and the likely pathogen/s.
- Non-infected diabetic foot ulceration do not require antibiotics, however appropriate wound care and management by a member of the multidisciplinary foot care team should be instigated.
- Where oral antibiotic issue is advocated this should initially be for 2 weeks, with a clinical review set within 1 week to assess clinical response and assess any microbiological findings.
- Antibiotic therapy should continue until, but not beyond, resolution of clinical findings of infection.
- If antibiotic courses are prolonged over 2 weeks duration renal and liver function should be monitored regularly and adjustments made accordingly.

# Introduction

People with diabetes are at high risk of infection due to multiple factors (see figure 1). Impaired leukocyte chemotaxis and phagocytosis is compounded by high glucose levels and poor tissue perfusion. The decreased ability to fight off infection combined with tissue hypoxia is thought to create an ideal environment for a necrotizing infection (Murray and Boulton, 1995; Gilchrist, 1997).

Foot infections in diabetes patients rank among the most common infectious complications that require hospitalisation and are a frequent cause of lower extremity amputation (Boulton, 2005). For example in a 2 year study by Lavery and co-workers for the 151 patients who developed diabetic foot infection there was a 56 fold increase in risk of hospitalisation and 155 fold increase in risk of amputation (Lavery et al 2006).

***Figure 1- Combined effects leading to the increased risk of infection in the diabetic foot***




# Diagnosing Infection in the Diabetic Foot

The international working group on the diabetic foot and the infectious disease society of America (IDSA) have outlined clinical criteria for diagnosing diabetic foot infections and classifying severity (see figure 2). This classification system has been validated (Lavery et al, 2007) and found to have a significant correlation between the defined severity of the infection and risk of amputation, anatomical level of amputation and hospitalisation. However, it is recognised that clinical signs and symptoms of infection in the diabetic patient may be subdued due to pre-existing peripheral arterial disease, peripheral neuropathy and impaired host response to infection, making diagnosis complicated. In this situation, some evidence supports the correlation of additional or secondary findings, for example, non-purulent secretions, friable or discoloured granulation tissue, undermining of the wound edges, or a foul odour, with evidence of infection (Gardner, 2009). As recommended by NICE guidance NG19 (2015) specialist assessment of diabetic foot ulceration and infection should always be sought by the multidisciplinary diabetic foot team within 24 hours of presentation.

It has been repeatedly reported that patients with deep foot infections which are potentially limb threatening often do not have fever, leukocytosis increase in the white blood cell count or markedly elevated acute phase serum markers and so the absence of these findings does not necessarily exclude a potentially serious infection. Worsened glycemic control is often the only systemic evidence of a serious infection in this setting (Armstrong et al, 1996; Eneroth et al,1999).

***Figure 2 – Classification of Diabetic foot Infection (IDSA)***

| ***Classification*** | ***Definition*** |
|---|---|
| Grade 1 (Uninfected) | No symptoms or signs of infection |
| Grade 2 (Mild) | Infection involving the skin and subcutaneous tissue only, with no involvement of deeper tissues and no systemic signs and symptoms. Exclude other causes of an inflammatory response (e.g. gout, trauma, acute Charcot neuro- osteoarthropathy, fracture, thrombosis, venostasis). At least two of the following manifestations are present:<br>• Localised swelling or induration<br>• Erythema >0.5–2 cm around the ulcer<br>• Local tenderness or pain<br>• Local warmth<br>• Purulent discharge |
| Grade 3 (Moderate) | • Infection involving structures deeper than skin and subcutaneous tissues (e.g. abscess, osteomyelitis, septic arthritis, or necrotizing fasciitis)<br>• Erythema (cellulitis) extending >2 cm around an ulcer in addition to one of the following: oedema, tenderness, heat, purulent discharge<br>• No signs of a systemic inflammatory response as shown in the grade 4 infection |
| Grade 4 (Severe) | Any foot infection with signs of a systemic inflammatory response syndrome (SIRS), manifested by two or more of the following:<br>• Temperature <36 ◦C or >38 ◦C<br>• Heart rate >90 beats/min<br>• Respiratory rate >20 beats/min or $PaCO_2$ <32 mmHg<br>• White blood cell count <4000 or >12 000 cells/µL or ≥10% immature (band) forms |

(Lipsky et al 2012)

# Microbiology of the Diabetic Foot

Isolation of antibiotic-resistant organisms, particularly MRSA but also extended-spectrum β-lactamase (ESBL)–producing gram-negative bacilli and highly resistant *Pseudomonas aeruginosa*, is an increasing problem with diabetic foot infection (Dang et al, 2003; Edmonds, 2005, Richard et al, 2008). Antibiotics used indiscriminately and without need results in an increased probability of developing resistance (Jensenius et al, 1995) and hence the use of antibiotics without the clinical suspicion of the presence of infection is not appropriate. However, the high morbidity and mortality associated with infected diabetic ulcers suggest that when clinical signs of infection are suspected then the patient should be issued immediately an empiric antibiotic regimen on the basis of the severity of the infection and the likely causative pathogens.

In patients with a clinically infected wound, properly obtained wound cultures provide highly useful information for guiding antibiotic therapy, particularly in those with chronic infections or who have recently been treated with antibiotics (Nelson et al, 2006). Tissue samples obtained by biopsy or curettage after the wound has been cleansed and debrided is the most accurate and therefore advocated method of microbiological sampling (Lipsky et al, 2012). Although obtaining swab specimens is more convenient and require less training/skill, they provide less accurate results, particularly if the wound has not been properly debrided. However, if swabbing is the method available at the time of presentation then the accuracy of the results will depend on how the sample is obtained and the general principles in figure 3 should be followed (Gardner et al, 2006).

The majority of mild infections that have not previously been treated are caused by aerobic gram positive cocci, with S*taphylococcus aureus* being the most common isolate. Therefore it is important for empiric treatment to have good aerobic gram positive cover (Lipsky et al, 1990). However, mild infections that have been previously treated are more likely to be complicated by the addition of gram negative bacteria and hence often warrant a broader spectrum of cover initially. It is noted in the literature that obligate anaerobic organisms are isolated from many chronic, previously treated, or severe infections (Wheat et al, 1986; Ng et al 2008), but they are not usually major pathogens in most mild to moderate infections (Armstrong et al, 1995). Treatment with oral antibiotic agents (preferably ones with high bioavailability) is often appropriate for mild to moderate infections in patients without gastrointestinal absorption problems and for whom an oral agent with the appropriate spectrum is available (Lipsky et al, 2012).

Moderate and severe infections, particularly those that have been previously treated or complicated by the presence of peripheral arterial disease are likely to be polymicrobial in nature and hence it is considered safest to promptly commence therapy with a broad-spectrum regimen of adequate strength and bioavailability to penetrate deep structures at the periphery (Lipsky et al, 2012). The antibiotic agent(s) should have activity against gram-positive cocci, as well as common gram-negative and obligate anaerobic organisms. To ensure adequate tissue concentrations for extensive moderate infections and severe infections it is advocated that it is safest to start with parenteral therapy, which can usually be switched to oral treatment within a few days when the patient is systemically well and culture results are available to guide the selection.

## *Figure 3 - Swab Technique*

Diagnosis of an infection should be based on clinical findings. Cultures are meant to identify organisms and to assist in treatment of an infection rather than be used to diagnose infection as only wound surface organisms are sampled (as opposed to organisms within the tissue) (Tammellin et al, 1998). No particular method of wound swabbing can be considered definitive, however the literature recommends the following principles:

| ***Principle*** | ***Rationale*** |
|---|---|
| DO NOT use antiseptic solutions prior to taking wound swab (Kiernan 1998; Cuzzell 1993). | Organisms will be killed, and a false negative result may occur (Cuzzell 1993). |
| DO NOT use local anaesthetic prior to taking a swab. | Local anaesthetics can demonstrate antibacterial effects (Gilchrist 1996). |
| DO NOT culture pooled exudate or wound dressings (Cuzzell 1993) | Risk of non-wound contaminants is high. |
| DO remove excessive debris and all dressing residue without unduly disturbing the wound surface using a gentle stream of normal saline (Lawrence 1998; Donovan 1998; Cooper and Lawrence 1996). | Surface organisms are often different to those causing the wound infection and skin cells and other harmless contaminants may be present on the wound surface (Cuzzell1993). |
| DO wait for 1-2 minutes before taking swab (Lawrence 1999). If wound is fairly dry, moisten swab with sterile normal saline, if wound is moist the swab can be used dry. | Allows organisms to rise to the surface of the wound. Maximisation of uptake of exudate by swab. |
| DO take wound swabs from an area of viable tissue where the clinical signs of infection are present i.e. do not swab eschar or yellow, fibrous slough (Kiernan 1998; Cuzzell1993). | Infection causing organisms are most likely to be found in viable tissue. |
| DO use a zig-zag motion to swab wound surface and rotate swab during swabbing (Donovan1998; Lawrence 1999). Whole wound surface should be swabbed. If wound is very large, swabbing a number of small areas is acceptable (Gilchrist 2000). | Will allow for most complete sampling of wound organisms. |
| DO avoid surrounding skin (Kiernan1998; Cuzzell 1993). | Will avoid introducing superficial skin organisms into the culture. |
| DO transport swab to pathology laboratory as quickly as possible. If a delay of more than 24 hours is expected between taking a wound swab and arrival at the laboratory it is advised to store the swab in a refrigerator at 4°C (Cooper and Lawrence 1996) | Assists in preservation of common wound bacteria. |

## Antibiotic Selection

There is an absence of well controlled clinical trials for antibiotics in diabetic foot infection. This is largely thought to be because study design is particularly challenging. There is great patient to patient variability in

terms of vascular supply, existing co-morbidities, extent and duration of infection and there are a lack of standardised definitions for infections, improvement and cure, especially when surgical intervention is included in the protocol (Jeffcoate et al, 2008), These factors make it difficult to provide clear-cut recommendations regarding antibiotic therapy in the case of diabetic foot infection and hence there are no national guidance specific to this condition on which to rely. This is compounded by the fact that critical reviews of published clinical trials of antibiotic regimens for diabetic foot infections have concluded that there are no standardised treatment recommendations and optimal therapy should rely on local knowledge of the likely pathogens and the spectrum of antibiotics that can provide coverage (Roberts and Simon 2012; Crouzet et al 2011 and Lipsky et al 2004).

Following IDSA guidelines antibiotic therapy should continue until, but not beyond, resolution of clinical findings of infection. It is suggested that an initial oral antibiotic course for mild to moderate infection of 2 weeks is appropriate and seeking urgent admission for IV antibiotic therapy and assessment of the need for surgical drainage and debridement in those presenting with severe infections (Lipsky et al, 2012).

Due to these issues local agreement has been sought to provide clear guidance for staff with regards to first and second line antibiotic choices when presented with Diabetic Foot Infection (Figure 4).

## MRSA

If swab result is returned MRSA positive immediately change antibiotics to doxycycline 200mg stat followed by 100mg bd (presuming reported as tetracycline sensitive). Consult with microbiologist if renal or liver impairment present or tetracycline resistant.

All new cases of MRSA, should be referred to the ACE MRSA decolonisation team.

Colchester Hospital University NHS Foundation Trust
North East Essex Clinical Commissioning Group

# *Figure 4 – Antibiotic Guidance*

PATIENT PRESENTS WITH A DIABETIC FOOT INFECTION

| SEVERITY OF INFECTION | 1st LINE TREATMENT | 2nd LINE TREATMENT |
|---|---|---|
| **Mild**<br>Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs).<br><br>*Common organisms – Staphylococcus aureus and beta haemolytic streptococci if antibiotic naïve* | **If antibiotic naïve:**<br><br>Flucloxacillin 1g qds<br><br>OR if penicillin intolerant:<br><br>clarithromycin 500mg bd | **If unable to tolerate 1st Line, previous antibiotics within the last 3 months or unresponsive to 1st line:**<br><br>Co-amoxiclav 625mg tds<br><br>OR if penicillin intolerant:<br>doxycycline 200mg STAT then 100mg od |
| **Moderate**<br>Local infection with erythema > 2 cm, or involving structures deeper than skin and subcutaneous tissues (e.g. abscess, osteomyelitis), but without systemic inflammatory response signs.<br><br>*Common organisms –Staphylococcus and Streptococcus species plus gram negative species and anaerobes*<br><br>***Consult with Microbiologist in presence of MRSA or unusual pathogens*** | Co-amoxiclav 625mg tds AND amoxicillin 500mg tds<br><br>OR if penicillin intolerant:<br><br>Cefalexin 1g tds<br>(If true penicillin allergy ciprofloxacin 750mg bd AND rifampicin 300mg bd)<br>Add metronidazole 400mg tds if suspect anaerobes present.<br>*If abscess present or wound requires surgical debridement admit and start IV cover as below* | **If unable to tolerate 1st Line or unresponsive to 1st line:**<br>Co-amoxiclav 625mg tds AND flucloxacillin 1g qds<br>**OR**<br>Ciprofloxacin 750mg bd AND rifampicin 300mg bd<br>**OR** doxycycline 200mg STAT then 100mg bd<br><br>Add metronidazole 400mg tds if anaerobes present<br><br>Clindamycin 450mg qds - if staphylococcal osteomyelitis suspected.<br>*Consider admission for IV therapy or IM Teicoplanin 400mg OD if deteriorating* |
| **Severe**<br>Patient presents with systemic signs of infection<br><br>- Clinical signs, nausea, vomiting, lethargy<br>- Raised blood glucose<br>- Temperature (<36 or >38°C)<br>- Raised heart rate (>90 beats/min)<br>- Increased respiratory rate (>20 breaths/min)<br>- White blood cell count <4000 or >12 000 cells/μL or ≥10% immature (band) forms | **Admit via EAU for IV antibiotics**<br>If patient refuses admission treat as per moderate Infection and discuss with Consultant or Microbiologist | IV Tazocin 4.5 g tds +/- IV metronidazole 500mg tds<br><br>OR if penicillin intolerant:<br>IV meropenem 0.5g -1g tds |

**Notes:**

- Courses should be for 14 days, however review after 7 days should be provided and change to targeted antibiotic therapy based on microbiology and clinical findings as appropriate
- Osteomyelitis requires a minimum 6 weeks uninterrupted antibiotics and longer in calcaneal osteomyelitis
- Dose adjustment in presence of renal or hepatic impairment maybe required
- In presence of unusual pathogens or MRSA positive consult with Microbiologist. All new cases of MRSA should be refered to ACE MRSA decolonisation team.

## Special Considerations

| *Antibiotic* | *Comments* | *Precautions* | |
|---|---|---|---|
| | | **Hepatic Impairment** | **Renal Impairment** |
| Amoxicillin | Risk of *Clostridium difficile* in the elderly and previously hospitalised due to its broad spectrum of activity, discontinue immediately in the presence of bloody or severe diarrhoea, start metronidazole and seek microbiological advice. Amoxicillin can reduce the excretion of methotrexate (increased risk of toxicity). | Rarely causes cholestatic jaundice | If eGFR 10-30ml/min/1.73m$^2$ reduce to 500mg bd<br><br>If eGFR under 10ml/min/1.73m$^2$ reduce to 250mg tds |
| Cefalexin | Avoid if history of serious hypersensitivity reaction to penicillins. Risk of *Clostridium difficile* in the elderly and previously hospitalised due to its broad spectrum of activity, discontinue immediately in the presence of bloody or severe diarrhoea, start metronidazole and seek microbiological advice. The effects of coumarins may be enhanced by cephalosporins. | Nil | If eGFR 40-50ml/min/1.73m$^2$ max 1g tds<br><br>If eGFR 10-40ml/min/1.73m$^2$ max 500mg tds<br><br>If eGFR less than 10ml/min/1.73m$^2$ max 250mg tds |
| Ciprofloxacin | Risk of *Clostridium difficile* in the elderly and previously hospitalised due to its broad spectrum of activity, discontinue immediately in the presence of bloody or severe diarrhoea, start metronidazole and seek microbiological advice. Do NOT use in pregnant or breastfeeding women. Use with caution in patients with epilepsy. Milk, indigestion remedies, iron and zinc should not be taken 2 hours before and after taking ciprofloxacin. Multiple significant interactions – refer to BNF appendix 1. | Nil | If eGFR 30-60ml/min/1.73m$^2$ reduce dose to: 500mg bd<br><br>If eGFR under 30ml/min/1.73m$^2$ 500mg od |
| Clarithromycin | Avoid use in pregnant or breastfeeding women. Do not prescribe modified release formulations. Multiple significant interactions – refer to BNF appendix 1. | Avoid in severe liver impairment | If eGFR under 30ml/min/1.73m$^2$, reduce dose to 250mg bd. Maximum duration 14 days. |
| Clindamycin | Risk of *Clostridium difficile* in the elderly and previously hospitalised due to its broad spectrum of activity, discontinue immediately in the presence of bloody or severe diarrhoea, start metronidazole and seek microbiological advice. Monitor liver and renal function where treatment exceeds 10 days. | Nil | Nil |

| Antibiotic | Comments | Precautions | |
|---|---|---|---|
| | | Hepatic Impairment | Renal Impairment |
| Co-amoxiclav | Risk of *Clostridium difficile* in the elderly and previously hospitalised due to its broad spectrum of activity, discontinue immediately in the presence of bloody or severe diarrhoea, start metronidazole and seek microbiological advice. Co-amoxiclav can reduce the excretion of methotrexate (increased risk of toxicity). | Cholestatic jaundice can occur. Use with caution and monitor liver function if treatment should exceed 14 days. | Reduction of dose if eGFR 10-30ml/min/1.73m$^2$ to 625mg bd<br><br>If eGFR <10ml/min/1.73m$^2$ 625mg od |
| Doxycycline | Should NOT be given to pregnant or breast feeding women. Indigestion remedies, iron, and zinc reduce the absorption – patients should be advised to leave a 2 hour gap before and after taking doxycycline. Patients should be advised to protect skin from sunlight. The effects of coumarins and phenindione may be enhanced by doxycycline. Avoid concomitant use with retinoids. | Avoid or use with caution | Use with caution (avoid excessive doses) |
| Flucloxacillin | High rates of gastric disturbance at high doses consider change of antibiotic if patient unable to tolerate adequate dose. Should be taken on an empty stomach at least 1 hour before food or 2 hours after food. | Use with caution. Courses over 2 weeks increase risk of Cholestatic jaundice monitor liver function | Consider a dose reduction if eGFR under 10ml/min/1.73m$^2$ |
| Metronidazole | Reduce dose in pregnant or breastfeeding women. Disulfiram-like reaction with alcohol. Take with or after food.The effects of coumarins may be enhanced by metronidazole. There is a risk of toxicity with some cytotoxics – see BNF appendix 1. | In severe liver impairment reduce dose to one-third (1/3) and give once daily | Nil |
| Rifampicin | Risk of liver disorder, patients should be told how to recognize signs of liver disorder (persistent nausea, vomiting, malaise or jaundice), if symptoms occur discontinue treatment immediately. Should be taken 30-60 minutes before food. A reddish coloration of the urine, sweat, sputum and tears occurs and the patient should be forewarned of this. Soft contact lenses have been permanently stained. Multiple significant interactions – refer to BNF appendix 1. | Reduce dose and monitor liver function. | Use with caution if daily dose over 600mg.<br><br>Consider a dose reduction if eGFR under 10ml/min/1.73m$^2$ |

BNF online, Electronic Medicines Compendium, Renal Drug Handbook 3rd edition.

# Referral Information

Referral to Community Podiatry or MDT foot clinic through NEEDs referral form (see appendix A)

Telephone – 0845 2413313 or 01473 344930 (option 2)
Fax – 01473 225391

Community Podiatry - Monday to Friday, operates from 13 locations across North East Essex

Multidisciplinary Diabetic foot clinic – Monday and Friday mornings, Colchester General Hospital Outpatients Department

Colchester Hospital University NHS Foundation Trust
North East Essex Clinical Commissioning Group

***Appendix A - Application for Podiatry or MDT Foot Clinic Assessment***

**DO NOT USE THIS FORM TO REFER FOR**
**Renal Ophthalmology Retinal Screening Children <16yrs**
For advice on where to refer please call 0845 241 3313
**IF URGENT OR SUSPECTED TYPE 1 PHONE THE ON CALL NUMBER 07590 928225**

**Has this referral been discussed with DSN If urgent, please call DSN directly**
Yes ☐ No ☐ Please note the DSN will contact you prior to an appointment being made

NHS Number: | | | | | | | | | | |

Referral Date: | | | | | | |

**Patient Details** Patient Consent: **Y / N**

| | |
|---|---|
| Name: | Address:<br>Postcode: |
| Tel: Mobile: | |
| DOB: | Gender : M ☐ F ☐ Ethnicity: |

| | |
|---|---|
| **Registered : GP Practice Details**<br>Name: & Address | **Referrers Details**<br>**Name:**<br>**Designation:**<br>**Contact Number:** |

**Reason for Referral – Comments**

Type 1 ☐ Type 2 ☐ Newly Diagnosed ☐ Urgent ☐

| | |
|---|---|
| Risk Stratifications discussed with DSN /Hard to Reach ☐ | Patient Education Desmond/X-Pert (T2) ☐ DAFNE (T1) ☐ |
| Insulin initiation/GLP-1 start ☐ | Admission prevention ☐ |
| Poor Glycaemic control on insulin ☐ | Recurrent Hypoglycaemia ☐ |
| Telephone advice for patient ☐ | Complex/Co-morbidities ☐ |
| Diabetes Podiatry **(please also complete part b *)** ☐ | Dietetics ☐ |
| Orthotics ☐ | MDT Foot **(please also complete part b *)** ☐ |
| Diabetes in Pregnancy ☐ | Diabetes - Planning Pregnancy ☐ |

**Biomedical results (last 3 months) Please attach list of current medication and medical history**

| | Result | Date | | Result | Date |
|---|---|---|---|---|---|
| **HbA1c** | | | **eGFR** | | |
| **Total Cholesterol** | | | **Blood Pressure** | | |
| **Height** | | | **Weight** | | |
| **BMI** | | | **Retinal Screening** | | |
| **Foot Assessment** | **Risk** | | **Creatinine** | | |

**Fax to 01473 225391 or referral.bookingservice@nhs.net**

| | | |
|---|---|---|
| **Name:……** | **NHS No:……** | **DoB:……** |

# Foot Referrals

## NICE Foot Risk Assessment

| Low | Increased | High | Emergency & Foot Ulcer |
|---|---|---|---|
| • Normal Sensation<br>• Palpable Pulses<br>• No foot deformity<br>• No lesions or history of previous foot problems | **(One of Below)**<br>• Neuropathy<br>• Absent foot pulses<br>• Other risk factors (e.g. Corns, callus, deformity, thickened or deformed toenails) | • Neuropathy and/or Absent foot pulses<br>**Plus**<br>• Deformity<br>• Skin changes (e.g. callus)<br>• Previous Foot Ulcer/amputation<br>• Previous Charcot | • Foot ulceration<br>• Infection<br>• Gangrene<br>• Osteomyelitis<br>• Active/Suspected Charcot |

Please Indicate Patient's Foot Risk Category

Low Risk ☐ Increased Risk ☐ High Risk ☐ Emergency ☐

Foot Pathology/Reason for referral (please give as much information as possible)

……
……
……
……

Significant Medical History (please give as much information as possible)

……
……
……
……

History of Mental Illness –

……..

**Urgent**- Yes/No **Reason if Urgent**……

**Fax to 01473 225391 or referral.bookingservice@nhs.net**

# References

Armstrong DG, Lavery LA, Sariaya M, Ashry H. Leukocytosis is a poor indicator of acute osteomyelitis of the foot in diabetes mellitus. *Journal of Foot Ankle Surgery* 1996; 35:280–3

Armstrong DG, Liswood PJ, Todd WF, William J. Stickel Bronze Award. Prevalence of mixed infections in the diabetic pedal wound. A retrospective review of 112 infections. *Journal of the American Podiatric Medical Association* 1995; 85: 533–537

BNF online. Accessed 7th September 2015

Boulton AJM, Vileikyte L, Ragnarson-Tennvall G, Apelqvist J. The global burden of diabetic foot disease. *The Lancet* 2005; 366 (9498): 1719–1724

Cooper R, Lawrence JC. The isolation and identification of bacteria from wounds. *Journal of Wound Care* 1996; 5: 335-340

Crouzet J, Lavigne JP, Richard JL, Sotto A. Diabetic foot infection: a critical review of recent randomized clinical trials on antibiotic therapy. *International Journal of Infectious Diseases* 2011; 15 (9): e601–e610

Cuzzell, J.Z. The right way to culture a wound. *American Journal of Nursing* 1993; 48-50

Dang CN, Prasad YD, Boulton AJ, Jude EB. Methicillin-resistant Staphylococcus aureus in the diabetic foot clinic: a worsening problem. *Diabetic Medicine* 2003; 20:159–61

Donovan S. Wound infection and wound swabbing. *Professional Nurse* 1998; 13: 757-9

Edmonds M. Infection in the neuroischemic foot. *International Journal of lower Extremity Wounds* 2005; 4:145–53

Electronic Medicines Compendium. www.medicines.org.uk Accessed 7th September 2015.

Eneroth M, Larsson J, Apelqvist J. Deep foot infections in patients with diabetes and foot ulcer: an entity with different characteristics, treatments, and prognosis. *Journal of Diabetes Complications* 1999; 13:254–63

Gardner SE, Frantz RA, Saltzman CL, Hillis SL, Park H, Scherubel M. Diagnostic validity of three swab techniques for identifying chronic wound infection. *Wound Repair and Regeneration* 2006; 14:548–57

Gardner SE, Hillis SL, Frantz RA. Clinical signs of infection in diabetic foot ulcers with high microbial load. *Biological Research for Nursing* 2009; 11:119–28

Gilchrist, B. Wound infection, *Journal of Wound Care*. 1996; 5 (8): 386-388

Gilchist B. Infection and culturing. In: Krasner D, Kane D (eds). *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Second Edition.* Wayne, PA: Health Management Publications, Inc., 1997;109-114

Gilchrist, B. Taking a wound swab, *Nursing Times* 2000; 96 (4): 2
Jeffcoate WJ, Lipsky BA, Berendt AR, Cavanagh PR, Bus SA, Peters EJ, van Houtum WH, Valk GD,

Bakker K; International Working Group on the Diabetic Foot. Unresolved issues in the management of ulcers of the foot in diabetes. *Diabetic Medicine*. 2008; 25 (12):1380-1389

Jensenius M, von der Lippe B, Melby K, Steinbakk M. Proper use of antibiotics: What is that? *Tidsskr Nor Laegeforen* 1995;115 (28): 3504-3507

Kiernan, M. Role of swabbing in wound management. *Nurse Prescriber/Community Nurse* 1998; 45-46

Lavery LA, Armstrong DG, Wunderlich RP, Mohler MJ, Wendel CS, Lipsky BA. Risk factors for foot infections in individuals with diabetes. *Diabetes Care* 2006; 29:1288–93

Lavery LA, Armstrong DG, Murdoch DP, Peters EJ, Lipsky BA. Validation of the Infectious Diseases Society of America's diabetic foot infection classification system. *Clinical Infectious Diseases* 2007; 44:562–565

Lawrence, J. Swabs and other sampling techniques. *Journal of Wound Care* 1998; 7 (5): 232-233

Lawrence, J. Swab taking. *Journal of Wound Care* 1999; 8 (5): 251

Lipsky BA, Pecoraro RE, Larson SA, Hanley ME, Ahroni JH. Outpatient management of uncomplicated lower-extremity infections in diabetic patients. *Achieves of Internal Medicine* 1990; 150:790–797

Lipsky BA, Berendt AR, Deery HG, et al. Diagnosis and treatment of diabetic foot infections. *Clinical Infectious Diseases* 2004; 39: 885–910

Lipsky BA, Berendt AR, Cornia PB, Pile JC, Peters EJG, Armstrong DG, Gunner Deery H, Embil JM, Joseph WS, Karchmer AW, Pinzur MS, Eric Senneville E. Infectious Diseases Society of America Clinical Practice Guideline for the Diagnosis and Treatment of Diabetic Foot Infections. *Clinical Infectious Diseases* 2012; 54 (12):132–173

Murray HJ, Boulton AJM. The pathophysiology of diabetic foot ulceration. *Clinics in Podiatric Medicine and Surgery* 1995;12:117

Nelson EA, O'Meara S, Craig D, et al. A series of systematic reviews to inform a decision analysis for sampling and treating infected diabetic foot ulcers. *Health Technology Assessments* 2006; 10:iii–iv, ix–x, 1–221

Ng LS, Kwang LL, Yeow SC, Tan TY. Anaerobic culture of diabetic foot infections: organisms and antimicrobial susceptibilities. *Annals, Academy of Medicine Singapore* 2008; 37: 936–9

NICE Clinical guidelines (NG19) Diabetic foot problems: prevention and management. August 2015.

Renal Drug Handbook. 3rd edition. Caroline Ashley, Aileen Currie.

Richard JL, Sotto A, Jourdan N, et al. Risk factors and healing impact of multidrug-resistant bacteria in diabetic foot ulcers. *Diabetes and Metabolism* 2008; 34: 363–9

Roberts AD and Simon GL. Diabetic Foot Infections: The Role of Microbiology and Antibiotic Treatment. *Seminars in Vascular Surgery* 2012; 25 (2): 75–81

Tammellin A, Lindholm C, Hambraeus A. Chronic ulcers and antibiotic treatment. *Journal of Wound Care* 1998;7 (9): 435-437

Wheat LJ, Allen SD, Henry M, et al. Diabetic foot infections. Bacteriologic analysis. *Achieves of Internal Medicine* 1986; 146:1935–1940