WMAI
Wound Management Association of Ireland
Quarterly Newsletter For Members
Promoting Best Practise in Wound Care
Issue 21 – Sept 2016

Dear Members and Colleagues,

Welcome to the Autumn 2016 edition of the WMAI newsletter. In this edition the focus is on the management of infection in wound care.

We are anticipating the update to the national wound care guidelines and a large amount of work has gone into these and we are quite excited about their imminent publication.

These guidelines will include an update to the original guidelines on areas including leg ulcers, diabetic foot ulcers, pressure ulcers, paediatrics, palliative wounds as well as the general principles underlying good wound care.

All geographical areas have run educational events over the autumn. These have been well attended and with good points for discussion raised. Please keep in touch with your regional secretary and the website for updates on the next event in your area.

Don't forget Stop pressure ulcer day on the 17th November, most areas will have events on for this day and our next newsletter will have a focus on pressure ulcers. Please send us photographs and reports of what you did locally to mark this day.

We are going to restart journal watch with this newsletter, so when you are doing your reading, please let us know which journals articles or case studies you found interesting or thought provoking and why. We will then share this with the members and hopefully we can get some interesting debate amongst wound care practitioners.

Do remember that WMAI is only as strong as we are when we work together. Team work is key in our association as in our practice. Innovation can only occur when we work together. Lets pull together to make Stop. pressure ulcer day a huge success in Ireland. We look forward to hearing form you.

Niamh McLain, Chairperson WMAI

STOP PRESSURE ULCER DAY 2016

Stop Pressure Ulcer Day takes place this year on November 17th, and aims to help bring pressure ulcers to a wider audience.

Send us your photos and details of events taking place throughout the regions

In this Issue

Infection in the Diabetic Foot - A ticking time bomb that can easily go BOOM!

I don't know how many of you play candy crush, I do guiltily admit to playing it on occasion. On some of the level there are candy bombs which if you don't keep a close eye on can quite easily catch you out and go boom finishing your game, I think that this can be quite a similar problem in the diabetic foot. It can be very easy to concentrate on all the issues needed to manage the diabetic foot and then quite often just miss a very subtle sign that can lead to the bomb of infection going off. I noted this morning that one of the master classes at the FDUK meeting in November this year is about the infection time bomb and what to do when it goes off!

It is quite easy to miss the subtle signs of infection in the diabetic foot and this is widely reported in the literature, I know that when I read papers on this issue I often think – I would never do that or that the problem is so overt that it couldn't be missed, but like the candy bombs in candy crush if we just focus on something else boom! There is an overt infection. I am going to highlight a case which recently presented to our diabetic foot clinic where the time element is key.

A patient well known to our service was recently discharged from hospital following a 6 night stay for the management of skin and soft tissue infection secondary to diabetic foot ulceration. As part of his discharge he was given an extended 2 week course of PO antibiotics. MRI scans showed no osteomyelitis (bone infection) and the diabetic foot team continued with previous management strategies. We address our management strategy across 4 main areas Infection control, Offloading, Vascular and Debridement with Wound management and Diabetes control as a secondary aim as suggested by the IWGDF guidelines

Infection management was with systemic antibiotics as per infectious disease consultants and the wound showed no sign of local infection / critical colonisation.

In this case the patient was offloaded with a rocker bottom boot (aircast) and was recruited for a trial of a new boot (pulseflow)

Vascular supply was considered adequate with abis of 1.45 and 1.38 and toe pressures of 154/95. Despite obvious calcification commonly seen in patients with diabetes pulses were strong and palpable.

Debridement was completed twice weekly by the podiatrists in the diabetic foot team.

On this occasion the patient had called the podiatrists and left messages concerned that he had noticed some new pain following on from a party over the weekend. When I called him back and discussed this with him he reported no change in the wound reporting the dressing still in place with no strike through, no change in blood sugar level, no change in wellness overall. As we continued to discuss he reported that the pain was in a different part of the foot and was similar to a pain he had felt before which was diagnosed as plantar fasciitis. He also reported no evidence of swelling or redness. He then also admitted to returning to work (he is a singer in a bar) and a party where he had had a few drinks and had been dancing! We decided to restart conservative treatment for plantar fasciitis and have him attend the clinic in the morning early. Upon arrival to the clinic at 7am he reported no change and that the pain had eased since restarting the exercises last night. He told me that he felt a bit of a fraud for coming in. On examination there was an increase in depth of the wound over his 5th Metatarsal head but no other signs of infection. Given the history I decided that it was quite likely that there was either a new focus of infection here or a restart of the previous infection. Given this information I ordered inflammatory marker blood tests. I advised him to go for blood tests and come back to the clinic and we would see him again then to discuss the options.

When he returned from the phlebotomy department 30 minutes later and I reviewed the wound – it was larger with purulent discharge, oedema and erythema! It was decided to await the result from the blood tests before making a decision on what the best course of action would be. Over the course of the next 3 hours he continued to deteriorate and by lunchtime there was evidence

of rigoring and he was febrile too. The blood tests taken at 8 am showed that he was neutropenic with wcc of 18.8 with ESR of 51 and CRP of 137.98. He was started on intravenous antibiotics and admitted. Over the course of the next 36 hours his CRP continued to climb peaking at 175 even with optimal antibiotic management. It is interesting to note that blood tests taken 4 days previously were all within normal limits.

He had an extended stay of 10 days on this occasion and is currently healed within a total contact cast.

I think the learning points here are multiple. Firstly I still feel that the pain which caused him to contact us was plantar fasciitis which had restarted following on from an extended period of bed rest and increased activity. Not all pain is related to the wound Secondly patients with neuropathic feet often are not able to mount a normal inflammatory response to critical colonisation or local infection so that by the time the infection is systemic they are often significantly unwell or septic. Thirdly if we look back to the initial work with patients with neuropathy secondary to Hansen's disease, in these cases there are reports of them losing digits overnight to animal bites totally unaware. No matter how much we educate patients with diabetes about the loss of feeling from neuropathy they still struggle with the concept of what this often means. We as clinicians often feel like a broken record repeating the same information about checking your feet at night but how often do we look at the bottom of our feet. I suggest only when they hurt!

To conclude I think that it is important to remember that the Diabetic foot is so multifactorial that no 1 clinician can take the whole responsibility for the management of it because with the best will in the world we will miss something. This is why we have multidisciplinary teams and rapid access to hospital clinics all in the aim of stopping these bombs from going off!

Bibliography:

Edmonds M, Dispelling the myths. Diabetic foot journal editorial

The shoes that love made – Dr Paul Brand.

Pauline Wilson **(TITLE AND PLACE OF WORK HERE PLEASE)**

WMAI (West) regional meeting and AGM in association with Healthcare 21

A regional meeting of the Western branch of WMAI will take place on Thursday 17th November 2016 in the Glenlo Abbey Hotel, Bushypark in Galway. Free to WMAI members. Registration is from 18:15. Please register your interest to Tina or Andrea at westernwmai@gmail.com.

STOP IAD CAMPAIGN

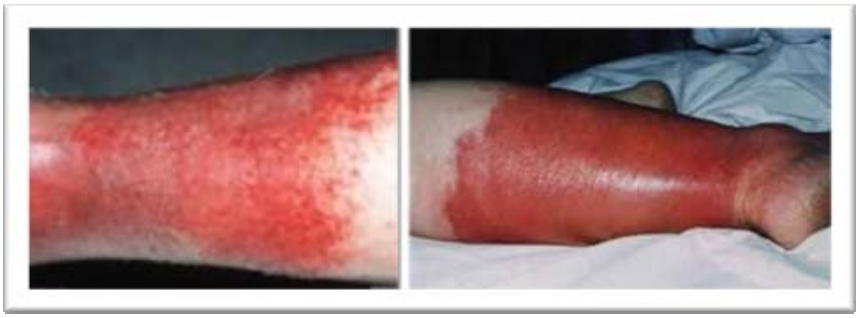
When your patients are incontinent their skin is exposed to urine and/or faeces. Urine decreases skin hardness and makes the skin more vulnerable to friction. It also increases the pH and promotes pathogenic growth. This can lead to IAD (Incontinence Associated Dermatitis). Incontinence-associated dermatitis (IAD), a clinical manifestation of moisture-associated skin damage, is a common problem in patients with faecal and/or urinary incontinence. The lesions are characterized by erosion of the epidermis and a macerated appearance of the skin (Gray et al. 2007a). Incontinence and skin breakdown related to incontinence have a considerable effect on patients' physical and psychological well-being . It is a daily challenge for healthcare professionals in hospitals, nursing homes and homecare to maintain a healthy skin in patients with incontinence. Furthermore recent evidence indicates an association between IAD, its most important etiological factors, and Pus.

Learn more about IAD, Moisture Associated Skin Damage, and Incontinence Associated Dermatitis from www.epuap.org/stop-iad-campaign/

Vascular Eczema Versus Cellulitis

This is something I come across often, confusion between vascular eczema and cellulitis. In truth I find a lot of patients with vascular eczema being treated for cellulitis, usually on to a third or fourth course of antibiotic with no result. It can be difficult to tell them apart in some cases, look at the sign below they both present very similar, however the major difference in not in the signs but in the symptoms. Patients with cellulitis will feel unwell and have a pyrexia. Also if your patient is on another course of antibiotic with out improvement it is worth considering vascular eczema.
Niamh McLain, **(TITLE AND PLACE OF WORK HERE PLEASE)**

Vascular Eczema	Cellulitis
× Swelling × Redness × Pain or itching × Scaling In severe cases: × Oozing × Open areas (cracking or larger ulcers) × Infection	× Swelling × Redness × Pain or Tender × Hot to touch × Feel generally unwell × Nausea × Shivering × Chills × Pyrexia × Raised WCC, ESR, CRP.



Treatment	
• Hydrocortisone cream • Paste wraps Compression (need referral to specialist to out rule arterial disease.	× Systemic Antibiotics

Leg Ulcers: Education, Clinical Learning and Quality Improvement Programme

Would you like to participate in a newly developed programme that will assist and challenge you to improve your practice in the treatment and management of leg ulcers? The Wound Management Association of Ireland (Southern Branch) is pleased to announce they are presenting a new programme which will adopt a different approach, giving you the opportunity to identify your learning needs in order to improve your practice. Mandatory attendance at all four sessions is required. You will be provided with pre-course learning material and a learning record to track your progress throughout the programme.

Wednesday 16th November 2016:
Leg Ulcers: Patient, Limb and Wound Assessment

Wednesday 18th January 2017
Leg Ulcers: Therapeutic Interventions

Wednesday 8th February 2017
Leg Ulcers: Assessment and Practice

Practical workshops will include appropriate assessment, investigations and bandaging techniques

Venue: Bru Columbanus, Wilton, Cork
Time: 6-9pm

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