

# Atypical Wounds

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# What are the causes of these wounds?



# Just when you thought it couldn't get any worse?



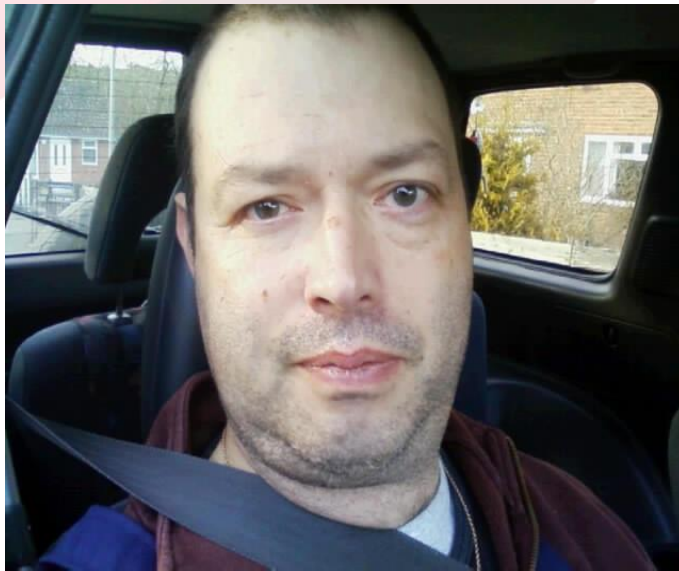


# Reasons for concern in these patients ?



# Nick

- **40 year old with spastic diplegia secondary to Cerebral Palsy**
- **Immensely independent shoe shop owner from Hereford**



- **Recurrent foot ulcers necessitating amputation of toes**
- **Pain "makes me want to cry every morning"**

# First presentation - 2004

- **Referral from orthopaedic surgeon**
- **1 year history of a painful and tender non-healing wound on the dorsum of the right foot**
- **No Diabetes**
- **No venous or arterial disease (duplex normal)**
- **No evidence of osteomyelitis (bloods & x-ray)**
- **No evidence of malignancy (biopsy)**
- **Treated for recurrent infections: antibiotics, local antimicrobials and then debrided under EMLA**
- **Healed after 7 months**
- **Prevention: dependent oedema control & foot care**



**Re-presented in 2008**

**Persistent ulceration**

**Extreme pain**

**Normal blood glucose**

**Treated with potassium permanganate and oral antibiotics.**



# 2008 - 2009



Month 0

**Toes fused  
Painful**



Month 4

**Recurrent Infection  
Wound enlarging  
Pain**



Month 15

**Recurrent infection  
Persistent wound  
Pain**



# Investigations (2008-9)

## Normal:

- FBC
- Urea and electrolytes
- Liver function tests
- Glucose
- Thyroid function
- Bone profile
- Serum globulins
- Electrophoresis
- Autoantibody screen
- X-ray foot
- ABPI
- Arterial & Venous duplex scan

- Inflammatory markers  
CRP: 45mg/L  
ESR: 60mm/hr
- Biopsy tissue microbial cultures:
  - *Pseudomonas aeruginosa*
  - *Stenotrophomonas maltophilia*

**April 2009 - because of persistent pain and recurrent infection he was seen by a vascular surgeon for a forefoot amputation**

**Persistent Pain  
despite:  
Buprenorphine  
patches  
Tramadol  
Paracetamol**



**Right foot**

**He then developed ulceration of the other foot**

**April 2009**



**Left foot**



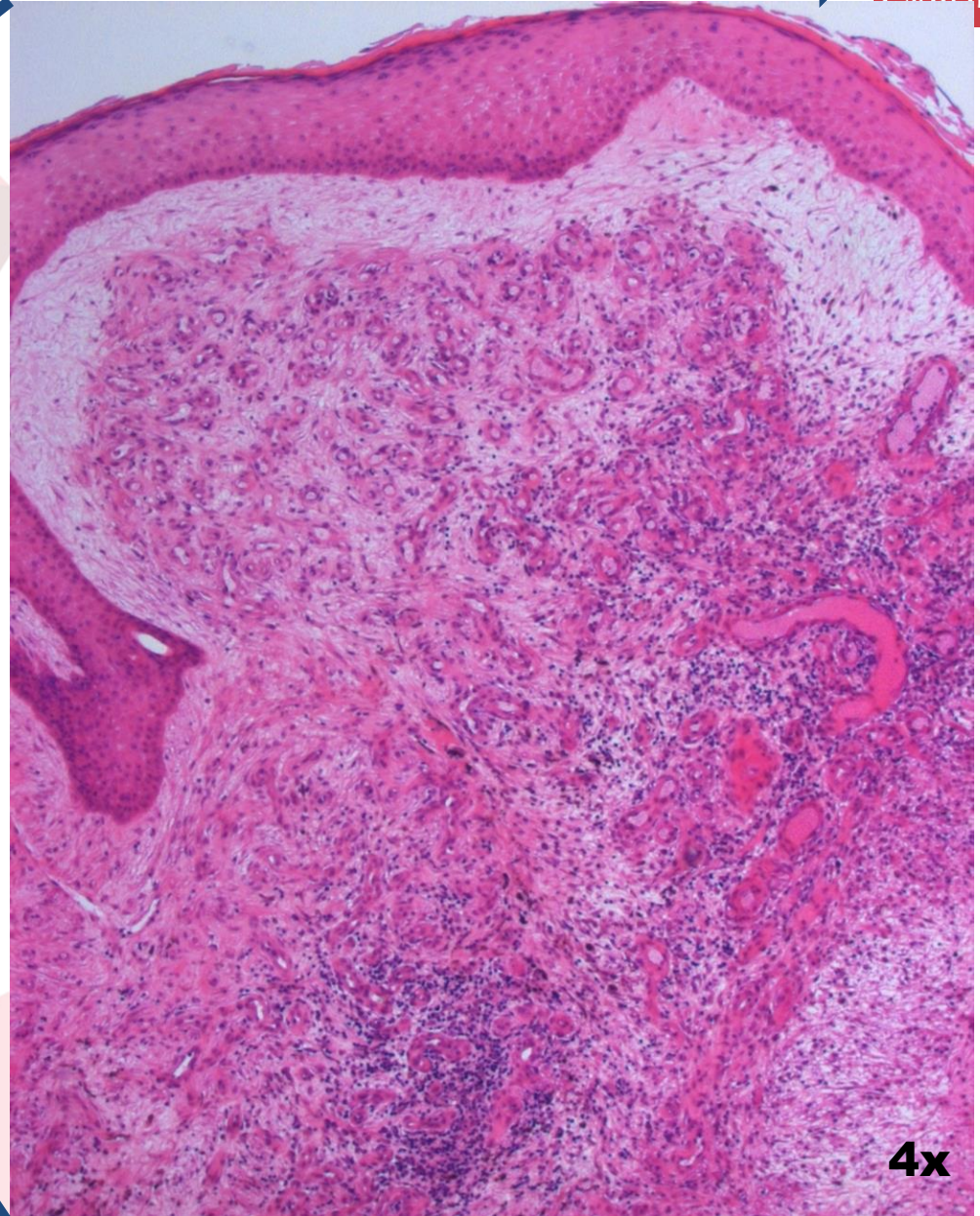
**Right foot**



Ulcer edge



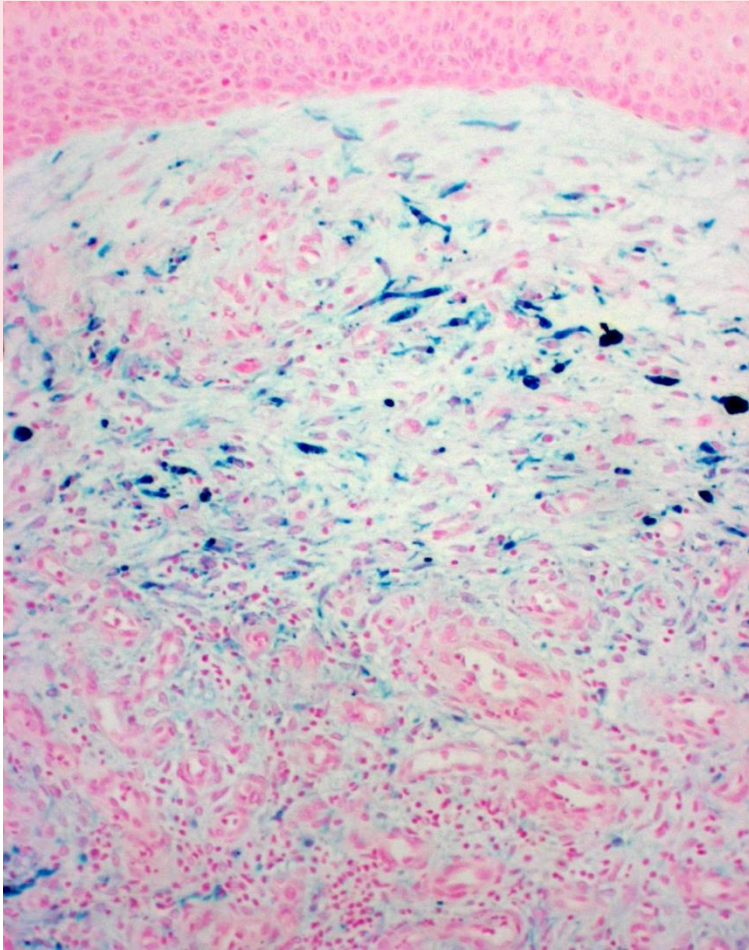
Right foot



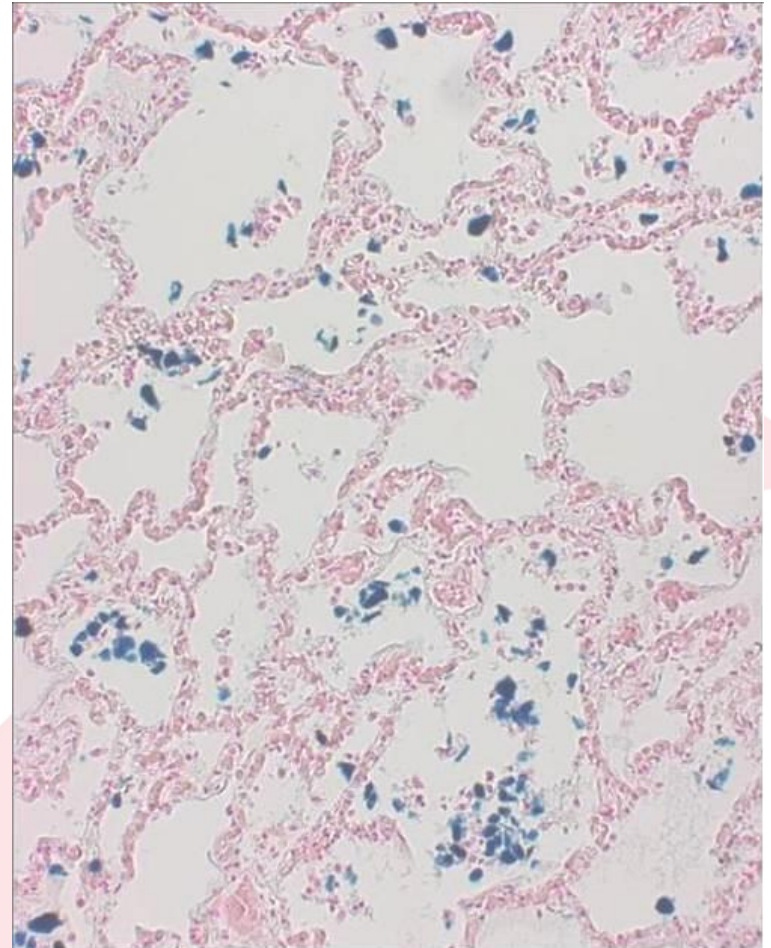
4x



# Perl Stain – Iron



**Patient**

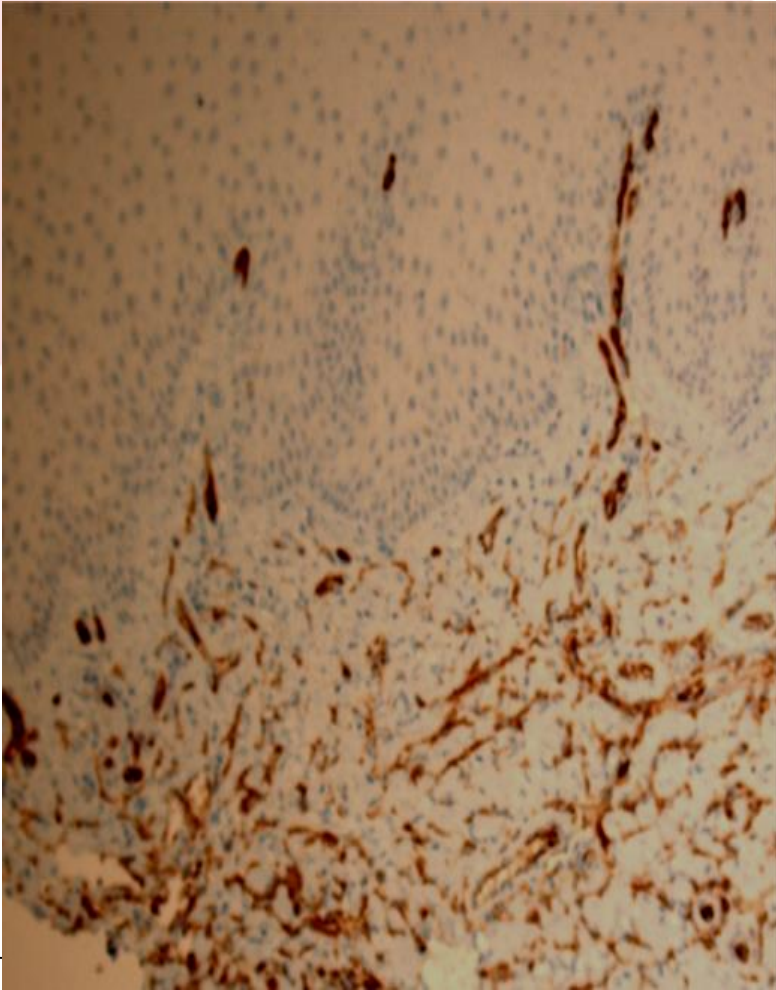


**Positive control**

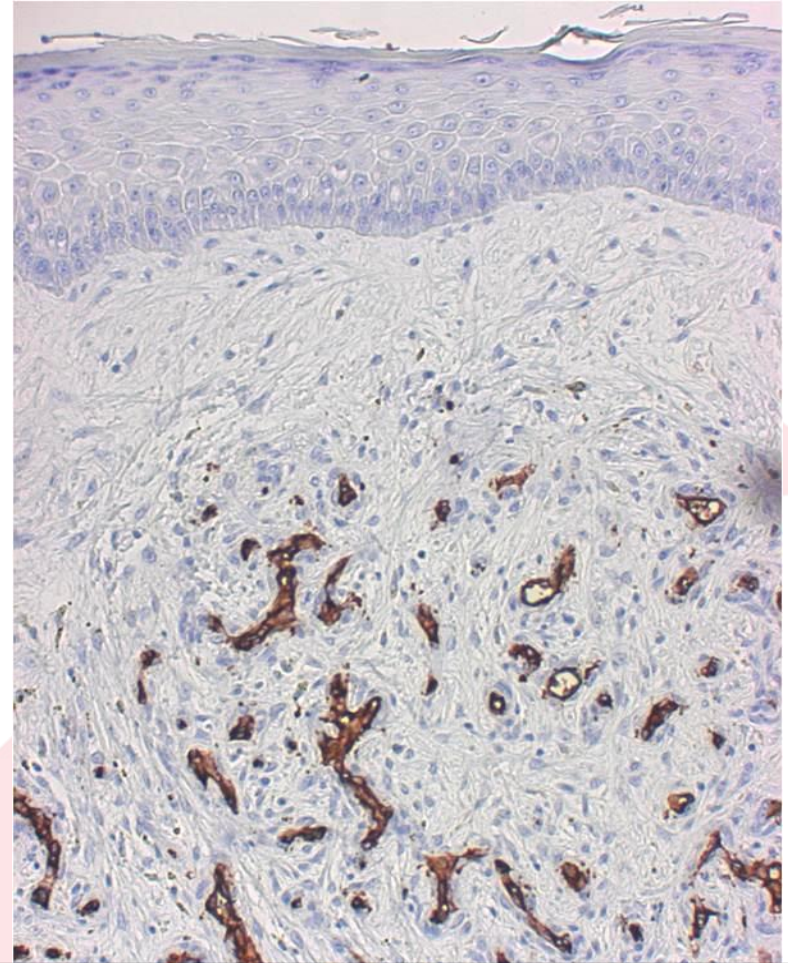


# Immunohistochemistry

## CD34 – blood vessels



**Kaposi's sarcoma**



**Patient**

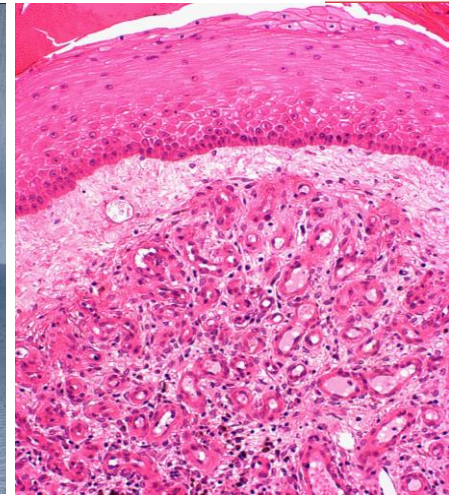


# Diagnosis?

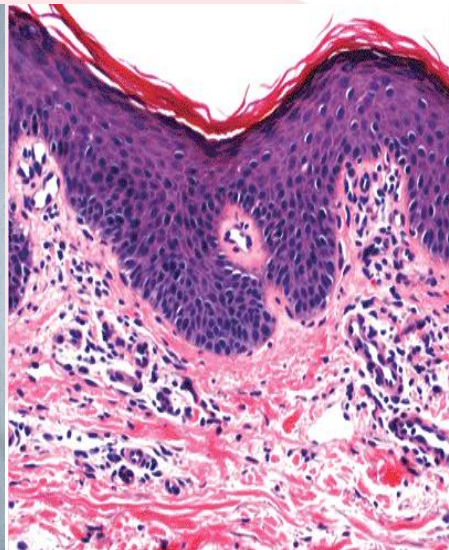


# Acroangiiodermatitis of Mali

- **Syn. Pseudo Kaposi's sarcoma**
- **original paper 18 cases associated with chronic venous leg ulcers**
- **Violaceous papules and plaques that coalesce and ulcerate**
- **Pathology: lobular capillary proliferation and spongiosis**
- **Ages 30-40 years**
- **Mostly males**



**Acroangiiodermatitis**



**Chronic Venous Insufficiency**

Mali JW et al. Arch Dermatol;1965;92:515-518



# August 2009

## Out-patient compression therapy



**Compression**



**Left foot**



**Right foot**

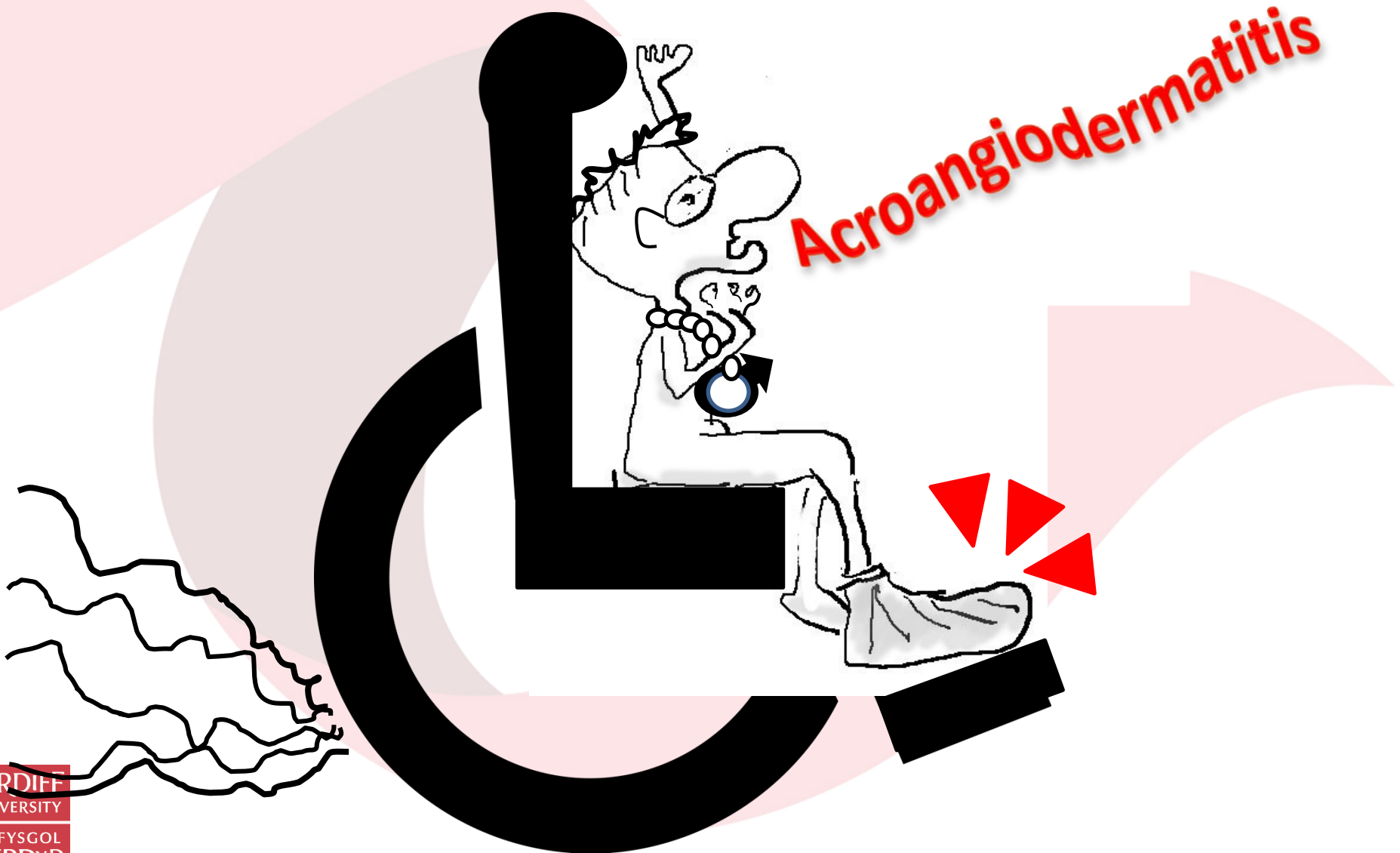


# Nick and his business



**happily re-united**

# The take home message...



# My Girlfriend..... HEIDI

If NOT Expected  
Outcome  
Repeat the process

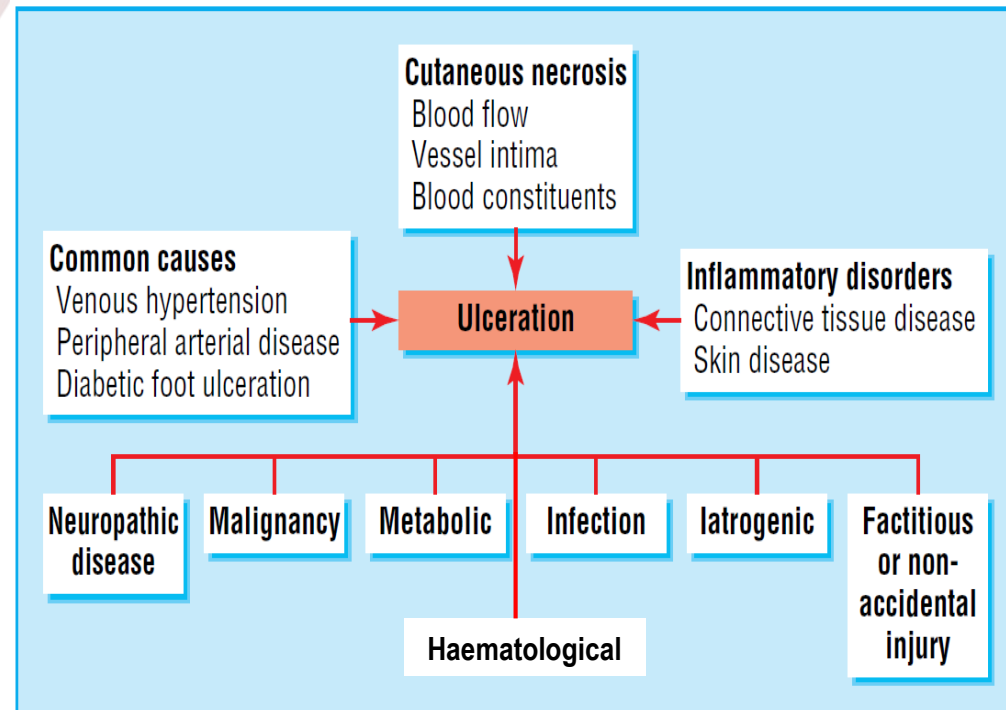
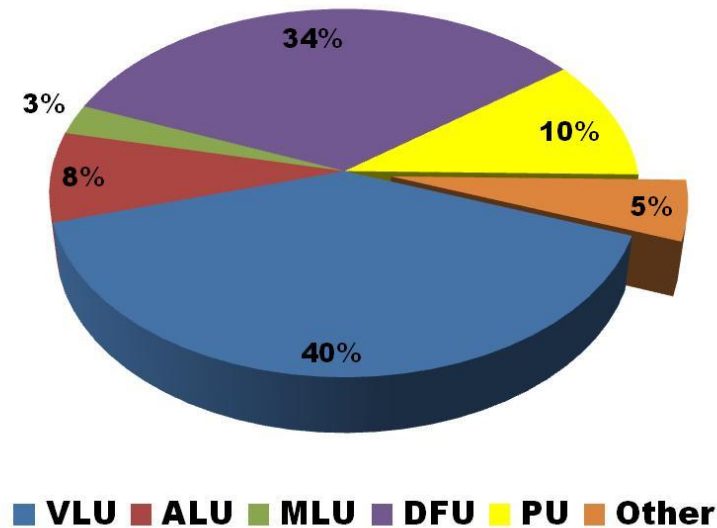
Expected Outcome





# Ulcers: Differential Diagnosis

**Chronic wound types**



# Targeting the patient - setting targets

Overall Management

Symptoms

Healing

# Minimise Appearance



# What is most important problem ?



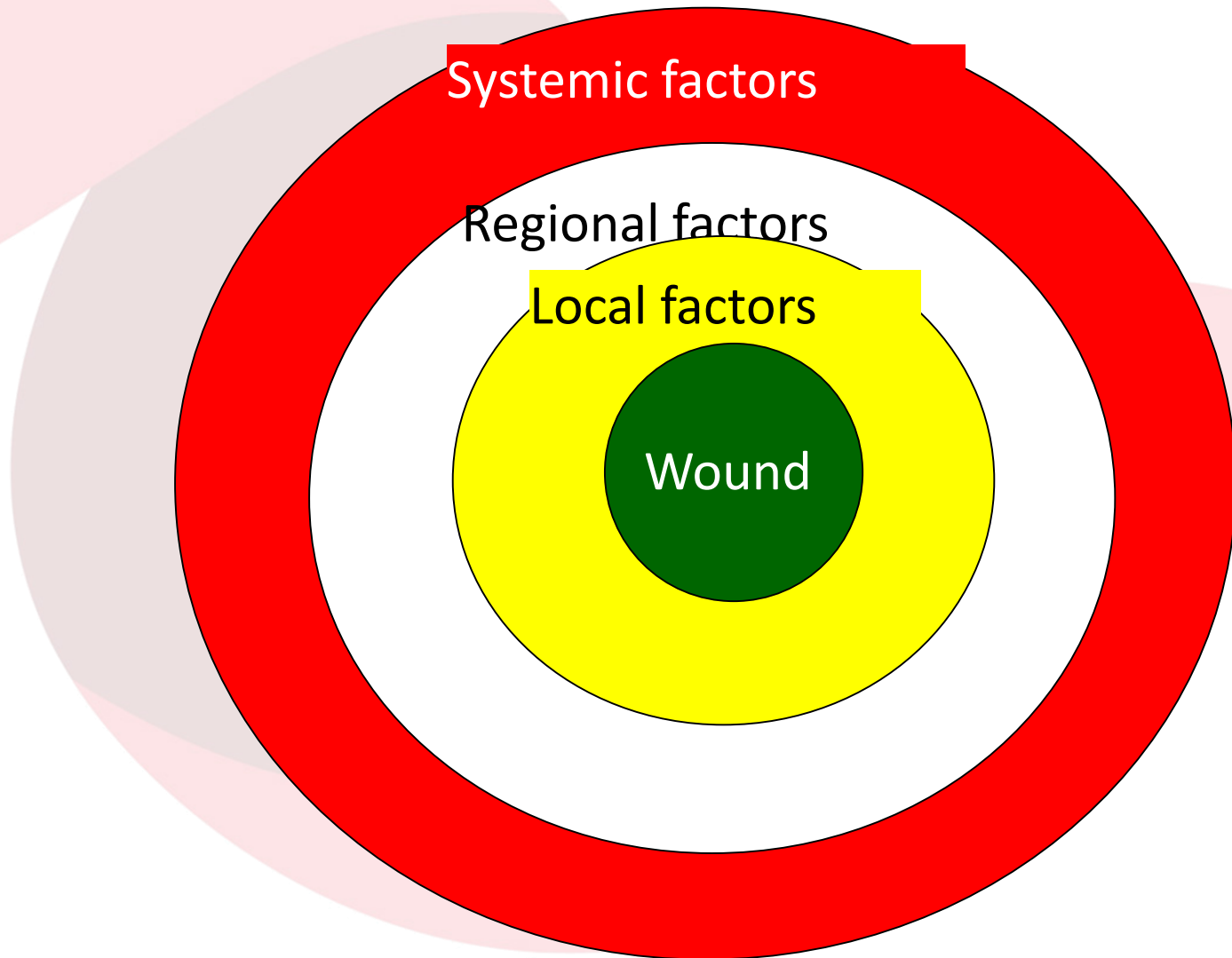
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# Measures of Success?



# Targeting the wound



# Systemic Factors



Factitious Wound



Vasculitis



# Regional Factors



# Easy to Heal ?



Not so Easy now!!



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# Local Factors



# Wound Factors



# Common causes Arterial Ulcer





# Venous Disease



# Diagnosis ?



Venous  
Disease/Lymphoedema/Obesity/Immobility/Infection/ Pain  
/ Renal Failure/ Cardiac Failure/ Malnutrition/ Depression/  
etc !!!!!





# Rheumatoid Arthritis

## Disease/consequences/drugs



WELSH WOUND  
INNOVATION  
ARLOESDD  
GLWYFAU CYMRU



# Diagnosis ?



# Progress over Time





# Leucocytoclastic Vasculitis



WELSH WOUND  
INNOVATION  
ARLOESDD  
GLWYFAU CYMRU



# Inflammatory causes of wounds



Pyoderma gangrenosum

Vasculitis



# Venous ulcer or PG? How do you know?





# It can be treated- Dressings/devices/drugs/biological agents



# Diagnosis ?



# Necrobiosis Lipoidica Diabeticorum





# Necrobiosis Lipoidica Diabeticorum (NLD)

- NLD is an uncommon inflammatory skin condition
- NLD usually affects people with Diabetes Mellitus – 0.3% type 1 and type 2
- One third of individuals will have some form of dermatological condition

# NLD

- More common in female population
- Average age of onset 30 yrs old
- Can be present in non DM, precursor to the disease – monitoring

# NLD clinical presentation

- Red papules that slowly enlarge into erythematous non-scaling plaques with waxy indurated yellow-brownish telangiectatic centres and raised edges
- Plaques can precede ulceration by months/years
- Prevalent on the lower limbs also found on fingers, dorsum of hands, face, scalp, abdomen, interscapular region and penis



# NLD clinical presentation



# NLD clinical presentation

- Self-limiting
- Can resolve spontaneously
- Recurrence and flare ups are frequent
- Reports of it occurring after tattoo and tattoo removal

# NLD complications

- Malignancy
- Secondary infection
- Unsightly scarring



# NLD differential diagnosis

- Granuloma annulare is a chronic asymptomatic dermatosis found on dorsum of the hands, feet and elbow and it can be difficult to distinguish from NLD
- Patients with sarcoidosis have similar presenting lesions

# NLD treatment

- Early treatment – low fat diet (unsuccessful)
- There are no recognized standard treatment, evidence currently available is insufficient to give definitive recommendations as regards the systemic treatment
- Systemic, intra lesional and topical application of corticosteroids
- NSAID

# NLD treatment

- Immune suppressant therapy – Infliximab, tacrolimus, cyclosporin
- Asprin and pentoxifylline to modify blood flow
- Avoid trauma
- Glycaemic control



# Bedtime reading

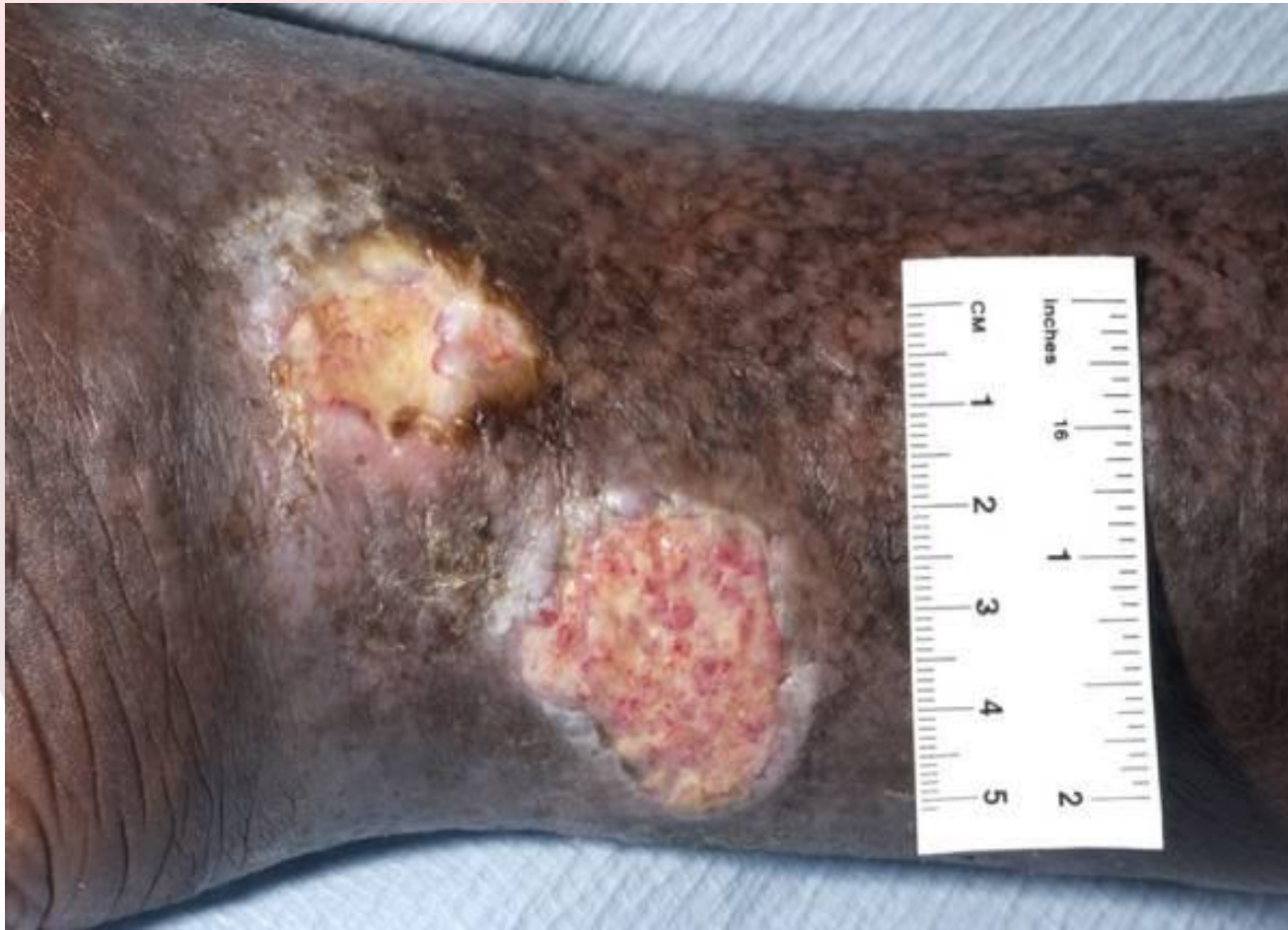
- Back to basics; Understanding NLD
- Wounds UK, volume 15, number 1, pages 40-46
- **<http://www.wwic.wales/publications-and-posters/published-articles>**

# Diagnosis ?



# Martorell's Ulcer

## What else could it be?





# Diagnosis ?



# Ehlers Danlos Type 4



# Hydroxyurea ulcer





# Nicorandil Ulcer



# Nicorandil as a Cause for Non Healing Wounds

June 2007



Sept 2007



# Angiosarcoma





# Marjolin's Ulcer





# Squamous Cell Carcinoma



# Malignant Melanoma



# Diagnosis ?







# Factitious Ulcer



# Cutaneous Calcification





# Diagnosis ?





Diagnosis ?



# Systemic Sclerosis

## BOX 1. Subtypes of systemic sclerosis (adapted from Denton and Khanna, 2017)

### **Limited cutaneous systemic sclerosis (also known as CREST syndrome):**

- Distal skin sclerosis (face and neck, and skin distal to elbows and knees)
- Pulmonary artery hypertension, severe gut disease, and a long history of Raynaud's phenomenon.

### **Diffuse cutaneous systemic sclerosis:**

- Skin sclerosis of proximal limbs or trunk
- Short history of Raynaud's phenomenon
- Increased risk of renal or cardiac involvement
- Severe lung fibrosis common.

### **Sine scleroderma:**

- Raynaud's phenomenon along with autoantibodies and other organ-based or other vascular manifestations of systemic sclerosis, but no skin changes.

# Calcium

- Skeletal muscle and myocardial contraction
- Neurotransmission
- Blood coagulation pathway
- Cell-to-cell communication
- Keratinocyte proliferation, differentiation and adhesion

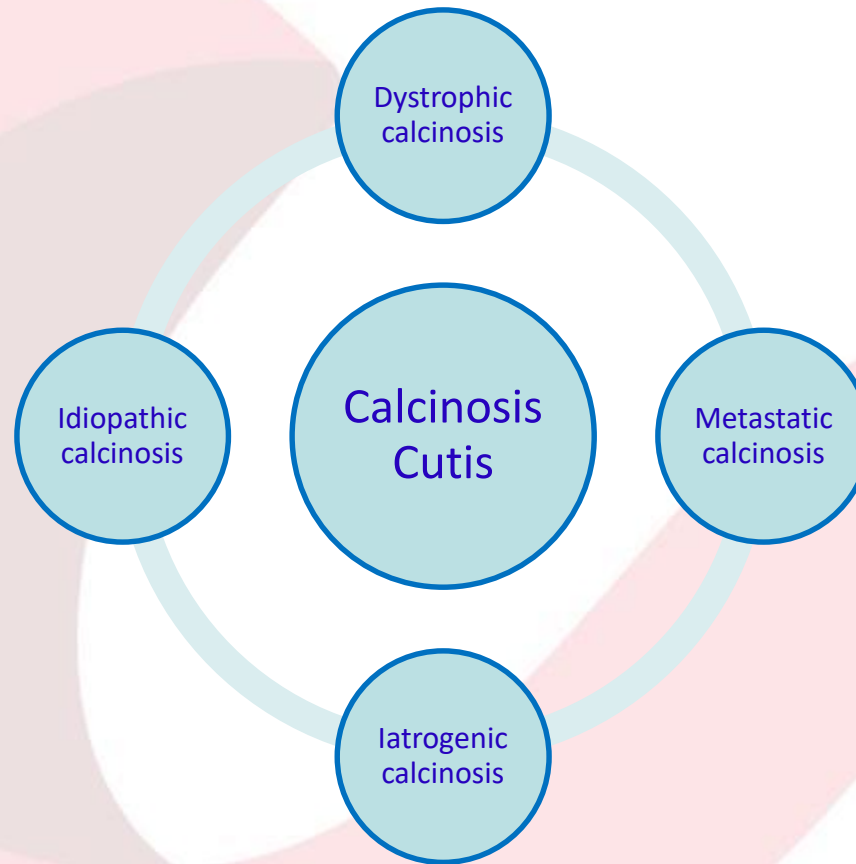


# Calcium regulation & deposition

- Serum calcium is strictly controlled by the parathyroid hormone
- Insoluble calcium can be deposited in the skin and subcutaneous tissues
- Mural calcification – calcium in the walls of the arteries



# Calcinosis cutis



# Dystrophic calcification

- Most common type of calcinosis cutis
- Multiple and local deposition of calcium
- Not found in internal organs
- Can be found in tendons and muscles
- Serum calcium and phosphate levels are normal
- Occurs in local tissue trauma – leg ulceration
- Dead tissue within the wound bed

# Dystrophic calcification

- Can remain unidentified until X-ray is performed
- Associated with connective tissue disorders – systemic sclerosis
- Wound bed contains grains of calcium
- Hard and firmly adhered to the wound bed
- Calcinosis universalis – rare, bands of calcium along the fascial planes, poor prognosis

# Dystrophic calcification





# Calcium deposits removed from a wound



# Metastatic calcification

- Abnormal calcium and phosphate metabolism
- Hypercalcaemia and hyperphosphatemia
- Secondary to Increased secretion of parathyroid hormone due to hyperparathyroidism (benign/malignant tumours)
- Tumours of the bone or bone marrow
- Paget's disease
- Renal failure
- Vitamin D-related disorders

# Idiopathic calcification

- Normal calcium and phosphate levels
- Subcutaneous tissue – scrotum
- Paediatrics

# Idiopathic calcification





# Iatrogenic calcification

- Complication of extravasation of intravenous calcium chloride and calcium gluconate
- Localised elevated level of concentration of calcium in the tissue
- Painful calcified nodules
- Resolve in time following discontinuation of the therapy at the damaged area

# Warfarin-induced skin necrosis



# Warfarin-induced skin necrosis

- Acute onset within 24 hours of taking warfarin
- Parathesia, odema, mild rash
- Develops and resembles clinical presentation of calciphylaxis
- Full-thickness skin necrosis with deep subcutaneous ulceration
- Areas with increased subcutaneous fat – abdomen, buttocks, thighs, breasts
- Biopsy to distinguish from Calciphylaxis

# Calciophylaxis

- Now 5<sup>th</sup> Variant of Calcinosis Cutis
- Can have normal parathyroid and renal function
- Complication of renal transplantation and end-stage renal disease
- Dialysis patients



# Calciophylaxis



# Calciophylaxis

- Common on lower limbs
- Purple-coloured mottling of the skin with blood-filled blisters
- Necrotic violaceous plaques
- Nodules
- Multiple painful non-healing ulcers
- Poor prognosis – inability to reverse the vascular disease, sepsis

# Calciophylaxis



**Table 1. Types of calcification (adapted from Reiter et al, 2011a)**

Type	Mechanism	Metabolic disturbance	Conditions where it may be seen
Dystrophic	Local tissue injury or abnormalities	<ul style="list-style-type: none"> <li>• None: calcium and phosphate metabolism and their serum levels are normal</li> <li>• Internal organs unaffected</li> </ul>	Chronic venous insufficiency, connective tissue diseases, trauma, inflammation, cutaneous neoplasms
Metastatic	Abnormal calcium and/or phosphate metabolism	<ul style="list-style-type: none"> <li>• Hypercalcaemia and/or hyperphosphataemia</li> <li>• Leads to widespread calcification of cutaneous, subcutaneous and deep tissues (blood vessels, kidneys, lungs and gastric mucosa)</li> </ul>	Chronic renal failure, hyperparathyroidism, malignant neoplasms, hypervitaminosis D, milk-alkali syndrome, sarcoidosis, Albright hereditary osteodystrophy
Idiopathic	No underlying cause identified	<ul style="list-style-type: none"> <li>• Calcium and phosphate metabolism and their serum levels are normal</li> <li>• Internal organs unaffected</li> </ul>	Seen in the scrotum, around major joints (tumoral calcinosis), progressive osseous heteroplasia and head or extremities (subepidermal calcified nodules)
Iatrogenic	Extravasation of calcium chloride or calcium gluconate during intravenous therapy	<ul style="list-style-type: none"> <li>• Calcium and phosphate metabolism and their serum levels are normal</li> <li>• Elevated tissue concentration of calcium at the site of extravasation</li> <li>• Internal organs unaffected</li> </ul>	Not associated with any particular disease or disorder
Calciophylaxis	Not fully understood, however, in chronic renal failure associated with secondary hyperparathyroidism, elevated phosphate levels in the blood which combine with calcium	<ul style="list-style-type: none"> <li>• Vascular calcification of the skin, small and medium sized blood vessels, Clots form due to calcification within the blood vessels</li> <li>• Can present with high or normal levels of serum calcium and phosphate with or without Vitamin D replacement</li> </ul>	Most commonly seen in fatty areas of the lower limb, lesions can occur on the trunk, abdomen, buttocks and thighs. Can occur in normal renal function in the presence of hypercoagulability states, i.e. liver disease, diabetes and warfarin



# Calcinosis cutis treatments

- Lack of randomised controlled trials
- Metastatic calcification – control of calcium and phosphate metabolism
- Parathyroidectomy
- Alteration in type of dialysis
- Alternative anticoagulant therapy
- Removal of the calcium deposits – local anaesthetic, surgical debridement
- Recurrence

# Diagnosis ?



# Hidradenitis Suppurativa (HS)

- Acneform disorder with follicular occlusion

*Chronic inflammatory, recurrent, debilitating skin disease of the hair follicle that usually presents after puberty with painful deep-seated, inflamed lesions in the apocrine gland-bearing areas of the body, most commonly the axillae, inguinal and anogenital regions.*

*Zouboulis et al 2015*

# HS

- 1% prevalence in Europe
- Rare in children and associated with hormonal disorders
- Predominance in the female population (hormonal)
- Incidence is higher in African and Afro-Caribbean population
- Starts around puberty



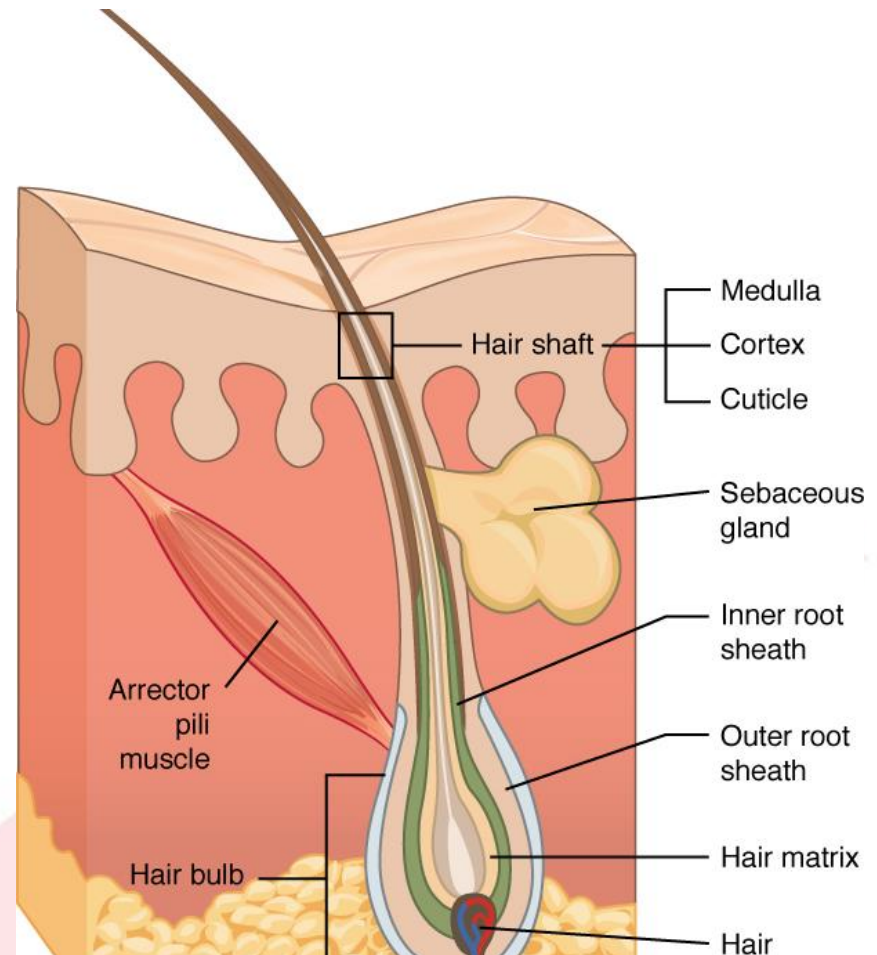
# HS course of the disease

- Most individuals only suffer from mild form of HS
- Eventually disease burns itself out
- Scarred and fibrotic skin
- Recurrence is common
- Average duration of the disease 19 years



# HS pathophysiology

- Uncertain aetiology
- Acute or chronic disease
- Skin disease of the hair follicles especially the follicular pilosebaceous unit
- Not a classical infectious disease, initial sterile process
- No unique bacterial agent
- Lack of lymph node involvement



# HS diagnostic criteria

- Delay in diagnosis (8.5 years) – mistaken for a simple infected lesion

*Have you had outbreaks of boils during the last six months with a minimum of two boils in one of the following five locations; axillae, groin, genitals, under the breasts, and other locations e.g. perianal region, neck and abdomen?*

# HS co-morbidities and risk factors

- Strongly associated with smoking and obesity
- Arthropathy
- Inflammatory bowel disease
- Metabolic syndrome
- Family history (genetic)
- Hormonal influence
- Not related to poor personal hygiene



# HS differential diagnosis

- Localised Staphylococcal infection
- Cutaneous Crohn's disease (associated with intestinal Crohn's disease)
- Abscesses, pilonidal/dermoid cysts
- Sexually transmitted diseases
- Tuberculosis of the skin
- Primary or metastatic tumour
- Squamous cell carcinoma can co-exist with HS

# HS treatment

- Pain relief – NSAID, opioids
- Topical clindamycin (antibiotic) – superficial and localised infection
- Systemic antibiotic therapy +/- clindamycin, rifampicin
- Immunosuppressant therapy – cyclosporin
- Corticosteroids – prednisolone

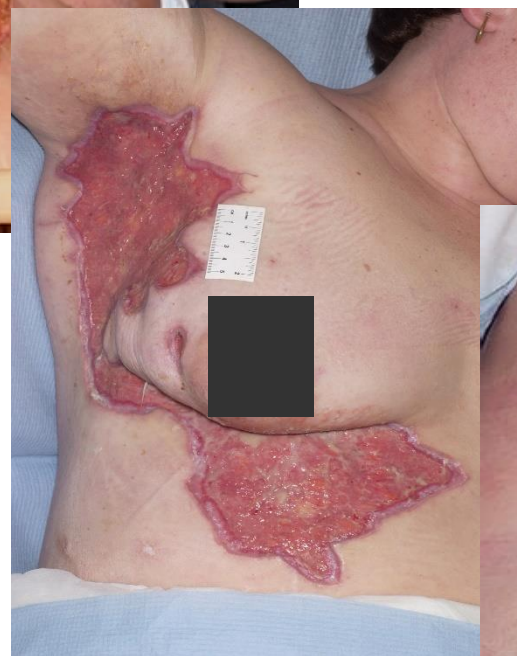
# HS treatment

- Retinoids – Vitamin-A based medications, isotretinoin, more success in treating acne
- Anti-inflammatory – dapsone, cyclosporin
- Hormonal therapy - anti-androgens, females with peaks before menstrual cycle
- Biological therapy – adalimumab (Cochrane supported), infliximab, (inhibit pro-inflammatory cytokines), high cost, adverse effects, recurrence common on discontinuing treatment

# HS treatment

- Surgical option if medical therapy is ineffective
- Localised excision and drainage (will not stop disease process)
- Wide surgical excision +/- negative pressure wound therapy





# Bedtime reading!

## PRACTICE DEVELOPMENT

### Back to basics: understanding hidradenitis suppurativa

#### KEY WORDS

- » Dermatology
- » Fistulae
- » Hidradenitis Suppurativa
- » Scarring
- » Sinus tracts

Hidradenitis Suppurativa (HS) is a chronic recurrent debilitating skin disease of the hair follicle. It is a condition that has been overlooked in wound care publications, with most articles found in dermatological journals. However, the condition affects 1% of the population in Europe and produces painful nodules in one or more of the apocrine gland bearing aspects of the skin that can ulcerate and produce pain and a foul odour and can multiply and eventually develop sinus tracts and fistulae. HS is often misdiagnosed as alternative skin ulcerating conditions, leaving the individuals with many years of suffering from the physical symptoms and their psychological consequences. The disease often begins in puberty and burns out by middle age, leaving the individual with unsightly scarring. This article examines the pathophysiology, clinical presentations and comorbidities associated with the disease. The treatment options focus on controlling the comorbidities, moderating life-style behaviours and arresting the disease. The medical and surgical options are discussed along with their limitations.

**H**idradenitis Suppurativa (HS) was first described by the French surgeon Velpeau in 1839. The origin of the term HS comes from the Greek *hidros* meaning sweat and *aden* denoting glands (Ather et al, 2006). Initially it was thought to be due to infection of the sweat glands however it is now recognized as an acneiform disorder which begins with follicular occlusion. Therefore, apocrine involvement is incidental and not essential to the pathogenesis (Ather et al, 2006).

The following Dessau definition of HS was created at the first international conference on HS in Dessau, Germany in 2006: HS is a chronic inflammatory, recurrent, debilitating skin disease of the hair follicle that usually presents after puberty with painful deep-seated, inflamed lesions in the apocrine gland-bearing areas of the body, most commonly the axillae, inguinal and anogenital regions (Zouboulis et al, 2015a). HS is sometimes referred to as apocinitis, acne inversa and pyoderma fistulosum signum (World Union of Wound Healing Societies [WUWHIS], 2016).

The reported prevalence varies across geographical areas due to misclassification and

selection bias, however is thought to be 1% in Europe (Gulliver et al, 2016; WUWHIS, 2016). Prevalence is rare in children and when HS does occur in this population it is often associated with hormonal disorders such as metabolic syndrome, precocious puberty, adrenal hyperplasia and premature adrenarche (Vivier and Kruse, 2017).

There is a predominance of HS in the female population with women being three times more affected. Incidence is higher in African and Afro-Caribbean populations (WUWHIS, 2016).

#### PATHOPHYSIOLOGY

The aetiology of HS is uncertain (Jianbing et al, 2013). It can be an acute or chronic disease and recurrence is common (Jianbing et al, 2013; Persaud et al, 2017). It is a skin disease of the hair follicles, specifically the follicular pilosebaceous unit (Beltrame and Staffolani, 2017; Persaud et al, 2017). The upper part of the hair follicles is occluded by keratinized stratified squamous epithelium that then progresses to dilation and rupture of the follicles with their contents deposited into the surrounding dermal tissue. This

## Back to basics; understanding Hidradenitis Suppurativa

*Wounds UK 2018, vol 14,  
no 1, pages 51 – 55*

<http://www.wwic.wales/uploads/files/documents/Professionals/New%20Articles/hydradenitis%20suppurativa.pdf>

# Diagnosis?



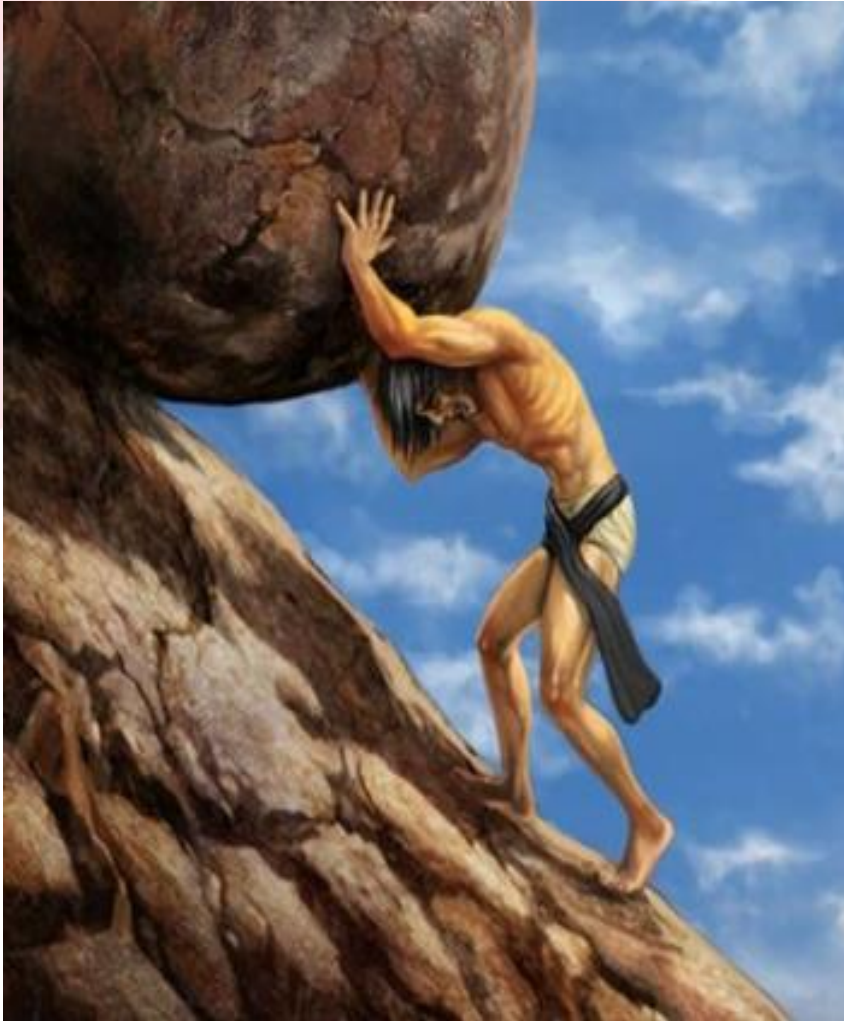


# Venous Ulceration does damage to your tattoos!!!!





# Conclusions



- Many pathologies can cause chronic wounds
- Diagnosis is needed before Treatment
- Consider HEIDI
- Treatment can consist of
  - Dressings
  - Devices
  - Drugs
  - Surgery
  - Biological agents
  - Combinations of above
- Wounds are diverse and challenging
- Need an MDT approach