THE MANAGEMENT OF A PATIENT WITH NECROTISING FASCIITIS EMPLOYING TOPICAL NEGATIVE PRESSURE

'Necrotising fasciitis can occur in any age group, but has been discovered to be more common in men with an underlying condition'

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This article reports on literature focusing on necrotising fasciitis, and the treatment and management of this life-threatening disorder. The risks of extensive tissue necrosis, systematic sepsis and potential organ failure makes the management, diagnosis and prompt assessment by the multidisciplinary team crucial in terms of patient survival.

In the accompanying case study, the focus is on the management of one patient (admitted as an emergency) with an abscess in his right groin. He was also found to have sepsis from necrotising fasciitis that resulted in him being admitted to an intensive care unit, post-operatively. As part of his treatment, the Avance® NPWT (Mölnlycke Health Care) system was used, along with traditional transparent film dressing and Avance film with Safetac® technology (Molnlycke Health Care). This article examines the patient's journey until he was transferred to the plastics unit for reconstructive surgery.

BACKGROUND

Evidence of necrotising fasciitis dates back to the 5th century BC, where the disease was initially defined by Hippocrates (Carter and Banwell, 2004). In the 16th century, Ambroise Paré described a gangrene-like condition that resembled today's 'flesh-eating disease' (Wilson, 1952). During the American Civil War in the 19th century, an army surgeon, Joseph Jones, described 2,642 cases of hospital gangrene around the genitalia and perineum. This was studied by Jean-Alfred Fournier in 1883, and subsequently became known as Fournier's gangrene (Carter and Banwell, 2004), which had a reported mortality rate of 50%.

In 1952, Wilson proposed the term 'necrotising fasciitis' to replace terms such as gangrenous erysipelas, hospital gangrene, acute cutaneous cellulitis, streptococcal gangrene, synergistic necrotising and Meleney cellulitis (Wilson, 1952). Necrotising fasciitis is commonly recognised by the public as 'flesh-eating bacteria' and 'killer bug' (Schroeder and Steinke, 2005).

EPIDEMIOLOGY

Due to the complexity of categorisation and terminology in necrotising fasciitis, it is difficult to collect the incidence of this disease. Skin and soft tissue infections (SSTIs) are best classified according to anatomical site of infections, depth of infection, microbial source of infection and severity (minor superficial to invasive, fulminant and fatal infections) (Sarani et al, 2009). The incidence of necrotising fasciitis in the UK is estimated at 500 new cases each year (Hasham et al, 2005) and 1,000 cases a year in the USA (Sarani et al, 2009). The mortality rate is estimated to range from 6% to 76% and 26% in more recent studies (Sarani et al, 2009).

Necrotising fasciitis can occur in any age group, but has been discovered to be more common in men with an underlying condition (Hasham et al, 2005). Old age has been identified as an important factor in susceptibility to necrotising fasciitis, and the estimated age at which the prevalence increases varies from 60 years (Benbow, 2008) to 65 years (Carter and Banwell, 2004). Timmons (2004) suggested that the incidence is more common in slightly younger patients — from the age of 50 years.

AETIOLOGY

Necrotising fasciitis is a rare, but potentially fatal, soft tissue infection involving the skin, subcutaneous tissue and muscle (Morgan, 2010). This condition is the most aggressive form of soft tissue infection and involves the superficial and deep fascial

layers of the extremities, abdomen, or perineum (Ozaley et al, 2006). It is normally accompanied by the systemic inflammatory response syndrome (SIRS) and needs prolonged intensive care treatment (Levine and Manders, 2005).

In some patients, there are definite clinical signs (hypotension, crepitus, skin necrosis, bullae, gas on radiographs), but these are not always present. Necrotising fasciitis is characterised by widespread necrosis of the subcutaneous tissue, and deep fasciae and muscle, and can be rapid in its destructive, clinical course (Tang et al, 2001).

Necrotising fasciitis involves a mixture of aerobic and anaerobic organisms, including *Bacteriodes, Clostridium, Peptostretococcus, Enterobacteriaciae, Proteus, Pseudomonas,* and Klebsiella, but group A *haemolytic Streptococcus* and *Staphylococcus aureus*, alone or in combination, are the initiating infecting bacteria (Sarani et al, 2009) (*Table 1).*

Prompt diagnosis and immediate aggressive surgical debridement of all compromised tissue are critical to reducing morbidity and mortality in these rapidly progressive infections (Ozaley et al, 2006).

The Infectious Disease Society of America suggests that SSTIs fall into three groups: superficial; uncomplicated (impetigo, ersipealas and cellulitis) and necrotising infections; infections associated with bites and animal contact; and surgical site infections and infections in an immunocompromised host (Tang et al, 2001).

A recent clinical classification for necrotising soft tissue infections (NSTI) has four types:

- Type one: polymicrobial with aerobic and anaerobic bacteria
- Type two: group A streptococcus (GAS) — can occur in any age group;
- Type three: gram-negative monomicrobial infections — this includes marine organisms, e.g *Vibrio spp* and *Aeromonas hydrophilia*, which occurs following seawater contamination of wounds, injuries involving fish fins or stings and raw seaweed consumption
- Type four is fungal (Wong and Tan, 2004; Benbow, 2008; Sarani et al, 2009).

PATHOPHYSIOLOGY

The pathogenesis of necrotising fasciitis is still not fully understood, but the rapid and destructive clinical course of this condition is believed to result from multibacteria symbiosis. Once GAS have entered the body, either by longstanding chronic or acute wounds, the bacteria release toxins. These endo exotoxins cause a histamine reaction that causes fluid to leak out of the local capillaries into the extravascular space (Fink and DeLuca, 2002).

When the toxins are released into the systemic circulation, they produce the systemic inflammatory response syndrome (SIRS), which can progress into septic shock that can result in death (Levine and Manders, 2005; Morgan, 2010; Sartelli et al, 2011). The reduction in the blood supply, in turn, results in secondary tissue ischaemia, clotting and thrombosis of perforated vessels of the skin, thereby affecting the local tissue which may become ischaemic and die (Elliott et al, 1996; Fink and DeLuca, 2002; Timmons, 2004). The skin may have erythematous lesions, be hot to the touch and, over time, change to dusky blue in colour.

On palpation, there may be crepitus and bullae can be observed on the skin surface, which are filled with foul-smelling dish water pus. If untreated, the lesion may spread and systemic toxin reaction may follow once the toxins have entered the blood stream (Headley, 2003). Toxins produced from β-haemolytic streptoccocal infection may lead to single and multiple organ failure and other complications, including intravascular coagulopathy (Timmons, 2004).

As the spread and extent of infection do not correspond with overlying skin changes, an inexperienced surgeon might not clearly determine the extent of the infection taking place under the skin surface and subcutaneous space (Roje et al, 2011).

PRESENTATION

The clinical manifestation of necrotising fasciitis may present as an

References

Bashford C, Yin T, Pack J (2002) Necrotizing Fasciitis: a model nursing care plan. *Medsurg Nurs* 11(1): 37–42

Benbow M (2008) Necrotizing fasciitis. *J Comm Nurs* 22(7): 4–8

Blair MJ, Chi H, Wang Ws, et al (2009) Necrotizing fasciitis in southeast Taiwan: Clinical features, microbiology and prognosis. *Infect Dis* 13(2): 255–60

Brandt MM, Corpron CA, Wahl WL (2008) Necrotizing soft tissue infections: A surgical disease. *Am J Surg* 66: 967–70

Carter PS , Banwell PE (2004) Necrotizing Fasciitis; A new management algorithm based on clinical classification. *Int Wound J* 1(3): 189–98

Cutting KF (2008) Impact of adhesives surgical tape and wound dressings on the skin, with particular reference to skin stripping. *J Wound Care* 17(4): 157–62

Endorf FW, Cancio LC, Klein MB (2009) Necrotizing soft tissue infections: Clinical Guidelines. *J Burn Care Res* 30(5): 769–75

Elliott DC, Kufera JA, Myers RA (1996) Necrotizing soft tissue infections. Risk factors for morality and strategies for management. *Ann Surg* 224: 672–83

Fink A, DeLuca G (2002) Necrotizing fasciitis: pathophysiology and treatment. *Medsurg Nurs* 11(1) 33–36

References

Hasham S, Matteucci P, Stanley PR, et al (2005) Necrotizing fasciitis. *BMJ* 330(7495): 830–33

Headley A (2003) Necrotizing soft tissue infections: a primary care review. *Am Fam Physician* 68(2) 323–8

Green JR, Dafoe DC, Raffin TA (1996) Necrotizing fasciitis. *Chest* 110(1): 219

Levine EG, Manders SM (2005) Life-threatening necrotising. *Clin Dermat* 23: 144–47

Magel DC (2008) The role of the nurse managing Necrotizing fasciitis. *AORNJ* 88(6): 977–86

Maynor M (2009) Necrotizing fasciitis 1–20. Available at: *http://emedicine.medscape.com/* (accessed on 14 September, 2012)

Malmso M, Ingemansson R (2011) Green foam < black foam or gauze for NWPT: effects on granulation tissue formation. *J Wound Care* 20(6): 294–99

Morgan MS (2010) Diagnosis and management of necrotizing fasciitis: A multiparametric approach. *J Hospital Infection* 75: 249–57

Ozalay M, Ozkoc G, Akpinar S, Hersekli MA, Tandogan RN (2006) Necrotizing soft tissue infection of a limb: clinical presentation and factors related to mortality. *Foot Ankle Int* 27(8): 598–605

Roje Z, Roje Z, Eterovic D, et al (2008) Influence of adjunvant hyperbaric oxygen on short term complications during surgical reconstruction of upper and lowe extremity war injury: A retrospective cohort study. *CroatMed J* 49: 224–32

Roje Z, Roje Z, Matic D, Libernjak D, Dokuzovic S, Varvodioc J (2011) Necrotizing fasciitis: literature review of contemporary strategies for diagnosing and management with three case reports: torso, abdominal wall, upper and lower limbs. *World J Emerg Surg* 6(46): 3–17

Sarani B, Strong M, Pascual J, Schwab CW (2009) Necrotizing fasciitis: current concepts and review of the literature. *J AM Coll Surg* 208(2): 279–88

Table 1
Classification scheme of skin and soft tissue infections (SSTis)

Classification characteristics	*Most common disease incidence (%)*
Anatomic localisation	*Fournier's gangrene of perineum and scrotum*
Depth of infection	*Necrotising adiposities fascitis, myonecrosis*
Microbial cause	*Type I: polymicrobial/70%–80% of cases* *Type II: Monomicrobial (*Streptococcus, Clostridia spp*) 20% cases* *Type III: Marine-related organisms* *Type VI: Fungal*
Uncomplicated infections	*Superfical: impetigo, ecthyma* *Deeper erysipelas, cellulitis* *Hair follicle-associated: folliculitis, furunculosis* *Abscess: carbuncle,other cutaneous abscesses*
Complicated infections	*Acute wound infections (traumatic,bite related, postoperative* *Chronic wound infections* *(diabetic wounds infection, venous stasis ulcers, pressure ulcers)* *Perineal cellulitis with/without abscess*
Necrotising fascitis *Polymicrobial, type I*	*Fournier's gangrene, necrotising cellulitis with fasclitis and myositis* *Streptococcal gangrene*
Monomicrobial fascitis, type II	*Marine-related organisms* — Vibro vulneriformis *and other* Vibro spp
Myonecrosis *Crepitant myonecrosis*	Clostridial myonecrosis *(trauma gas gangrene and atraumatic gas gangrene-*Clostridium perrigens *and other* Clostridial spp*)* *Synergistic necrotising cellulitis with fasclitis myositis*
Non-crepitant myonecrosis	*Streptococcal gangrene with myonecrosis* – Aeromonas hydrophila *myonecrosis*

Adapted from Sarani et al (2009)

abscess, infected traumatic surgical wound, intravenous drug abuse, pressure ulcers, burns, perforated viscera (particular colon, rectum and anus), recently performed liposuction, infected vascular prostheses and grafts, and invasive cancer (Blair et al, 2009; Green et al, 1996). Establishing early diagnosis of necrotising fasciitis is crucial for a favourable prognosis.

Roje et al, (2011) state that every physician must know the answer to four main questions:

- What is the clinical course of necrotising soft tissue infection and necrotising fasciitis?
- Which type of organisms are responsible for infection?
- What is the depth of the infection?
- Is necrotising fasciitis a life- or limb-threatening disease?

The answer to the first question ensures early diagnosis of necrotising soft tissue infection and necrotising fasciitis, the second determines the empirical spectrum of antimicrobial therapy and the final two address the timing and extent of surgical intervention.

RISK FACTORS

The major risk factor for the development of necrotising soft tissue infection is diabetes mellitus, which

has an involvement in 56% of all cases (Elliott et al, 1996; Endorf et al, 2009). Underlying conditions include obesity, alcohol abuse, immunodeficiency, chronic renal failure, liver cirrhosis, hypertension, peripheral vascular disease and age (over 60 years) (Benbow, 2008). When all of these conditions occur simultaneously, the mortality rate increases to 65% (Green, 1996; Elliott, 1996).

Roje et al (2011) state that the incidence of necrotising soft tissue infection is increasing due to the number of patients immunocompromised through diabetes, cancer, alcoholism, vascular insufficiencies and organ transplants. The majority of the infections are idiopathic, due to the difficulty of identifying any underlying lesion at the site of necrotising soft tissue infection (Endorf et al, 2009). An example of this is necrotising fasciitis in scrotal or penile Fournier's gangrene.

There are many underlying conditions that may predispose patients to necrotising fasciitis. An awareness of the risk factors is essential in preventing complications and ensuring that an effective patient management regimen is instigated.

TREATMENT

Early clinical suspicion and surgery are the keys to improving the survival rate, particularly in patients with necrotising infections, using a multidisciplinary approach to management. The early diagnosis is crucial in treating necrotising fasciitis and, if the condition

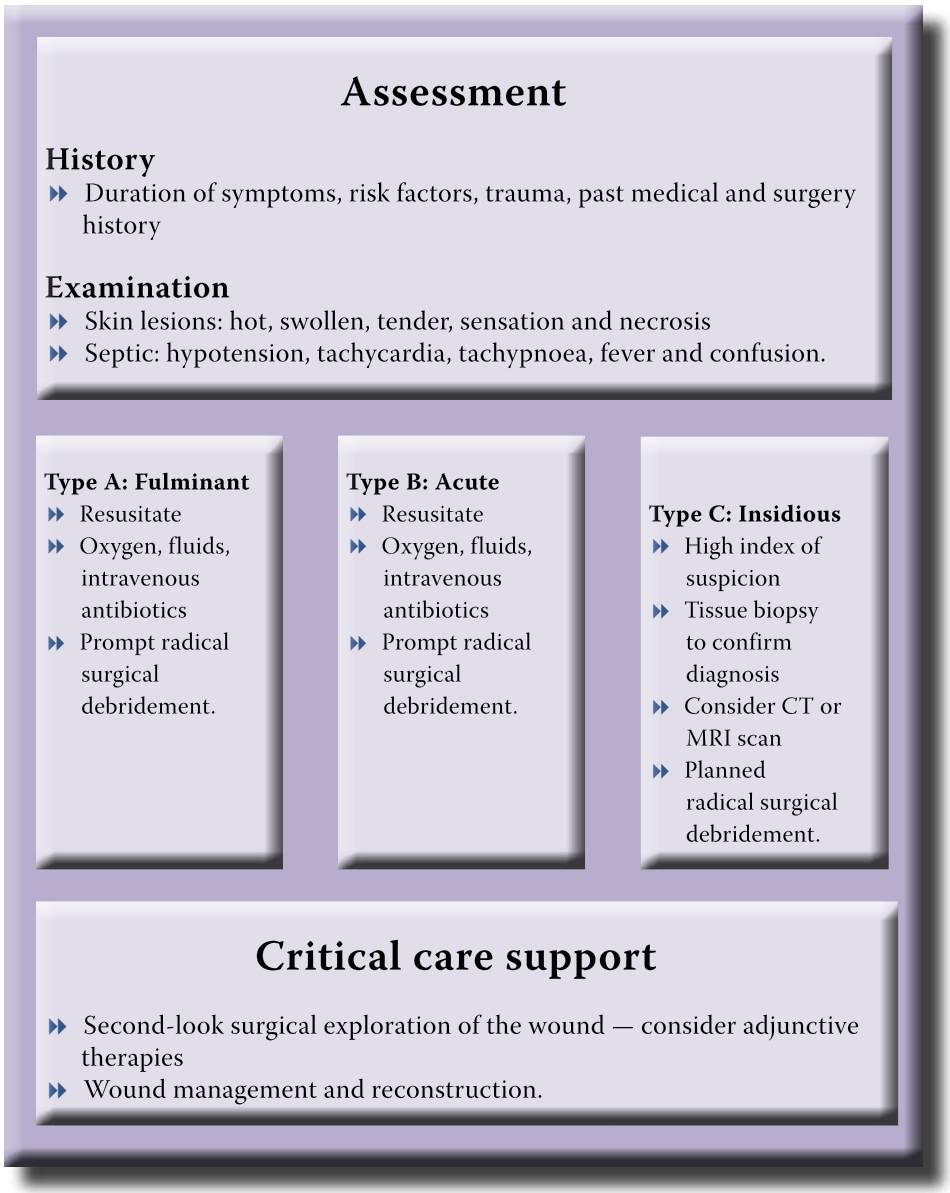

Figure 1: Management algorithm for necrotising infections (Carter and Banwell, 2004)

References

Sartelli M, Viale P, Koike K, et al (2011) WSES Conensus conference : Guidelines for first –line management of intyra-abdominal infections. *World J Emergy Surg* 6: 2

Schroeder JL, Steinke EE (2005) Necrotizing fasciitis: The importance of early diagnosis and debridement. *AORNJ* 82(6): 1031–40

Tang S, Ho PL, Tang VW, Fung KK (2001) Necrotizing fasciitis of a limb. *J Bone Joint Surg* 3(5): 709–14

Timmons J (2004) Necrotizing fasciitis in primary care: Presentation risk factors and treatment. *Br J Comm Nurs* 9(9) S16–24

White R (2008) A multinational survey of assessment of pain when removing dressings. *Wounds UK* 4(1):14–22

Wilson BL (1952) Necrotizing Fascitis. *Am Surg* 18: 416–31

Wong CH, Shih CH (1992) Necrotizing fasciitis of extremities. *J Trauma* 32(2): 179–82

Wong CH, Tan S (2004) Subacute necrotizing fasciitis. Lancet 364(9442): 1376

Table 2
Blood results

Date	*C-reactive protein*	*White blood cell*	*Haemoglobin*	*Sodium*	*Creatinine*	*Glucose*	
13/03/12	*374* *0-10mg/L*			*136* *133-146 mmol/L*	*223* *50-110ummol/L*	*5.9* *4.1-6.0mmols/L*	*33* *35-52g/L*
*16/03/12**				*140*	*70*	*6.8*	*16*
20/03/12		*11.6* *4.0-11.0/L*	*94* *130-180g/L*	*152*	*63*	*6.6*	*18*
25/03/12		*5.5*	*7.9*				

**The swab taken in theatre on 16/03/12 demonstrated :*
Organism 1 *Klebsiella pneumonia — heavy growth*
Organism 1 *Streptococcus milleri (Group) — moderate growth*

is complicated, hospital admission and immediate resuscitation of the patient is required (Carter and Banwell, 2004; Benbow, 2008).

Sarani et al (2009) state that multisystem organ failure can develop within 24 hours of infection and can prove fatal if the condition is not recognised immediately and treated.

Necrotising fasciitis may progress rapidly and signs of toxaemia may develop, resulting in shock and sepsis. The patient will be confused or unconscious, making it difficult to diagnose their condition (Hasham et al, 2005). The destruction of tissue can occur at a rate of one inch per hour (Magel, 2008; Sarani et al, 2009).

Carter and Banwell (2004) have developed a management algorithm for the three types of necrotising fasciitis (*Figure 1*), which assists the course of treatment and management. It is important to have a good understanding of the different categories to ensure the correct course of treatment can be determined.

Roje et al (2008) state that the algorithm for care is:

- Resuscitate any patient in shock
- Begin broad spectrum antibiotics
- Undertake comprehensive debridement of dead tissue and histology of tissue
- Further debridement should be repeated 24–48 hours.

Radical debridement has to be performed to minimise overall tissue loss. Regular inspection of the wound is essential to ensure all of the dead tissue has been debrided. The progression of the infection is dependent on the timing of surgical intervention and effective debridement of the dead tissue (Roje et al, 2008).

Postoperative management consists of serial dressing changes (24–48 hourly) until the wound is free of concurrent infection or an ongoing infection. It can take between two to five debridement procedures to stabilise the wound (Roje et al, 2011).

Topical negative pressure can be used in the management of necrotising fasciitis following debridement as it promotes wound healing, closes wounds and reduces the size of difficult-to-manage large defects (Hasham et al, 2005). Banwell (2004) states that topical negative pressure reduces the oedema, encourages granulation tissue and minimises the pain associated with dressing changes.

Topical negative pressure also prepares the wound bed for skin grafting as it rapidly cleans and reduces the size of the wound and is effective in the closure of large wounds (Timmons, 2004). The dressings used in topical negative pressure dressing enables the exudate levels to be managed effectively, while maintaining patient dignity and reducing the number of

dressing changes that are required with conventional dressings.

THE AVANCE NPWT SYSTEM

The Avance NPWT system has been developed as a flexible and easy-to-use treatment that helps to promote wound healing, including drainage and removal of infectious material or other fluids, under the influence of constant and/or intermittent negative pressure.

The Avance system is lightweight and portable and incorporates a rechargeable battery so that it can be operated independently of the mains, thereby encouraging patient mobility during treatment.

Avance dressing kits include the choice of either foam- or gauze-based wound dressings. Clinicians also have the option of utilising dressing kits, which include an atraumatic soft silicone wound contact layer (Mepitel with Safetac technology) and can be used between the wound bed and the wound dressing to minimise trauma and pain at dressing changes, prevent ingrowth of tissue into the wound dressing, and protect delicate deep structures.

Avance Foam

The green-coloured Avance Foam is a hydrophobic reticulated polyurethane foam with a large open cell structure. It is intended for use in the Avance NPWT system to distribute the pressure across the wound surface and allow passage of fluids and exudate through to the negative pressure system.

Avance Foam stimulates wound healing by promotion of granulation tissue formation. The foam is available in small, medium and large dressing kits. The green foam has the advantage of allowing bleeding to be monitored more easily through being identified in the wound bed (Malmso and Ingemansson, 2011).

CASE STUDY

A 38-year-old male presented with an abscess on his right thigh from an injection site following a 14-day history of diarrhoea. His past medical history included acute renal failure, and deep vein thrombosis of his right iliac and femoral veins. He was a known drug user and had hepatitis C. He was admitted from the A&E department on 16 March, 2012 and was extremely septic.

An emergency surgical debridement was performed on all the affected tissue, which is the primary treatment modality for necrotising soft tissue infections and necrotising fasciitis. It includes prompt and radical surgical debridement necrectomy and fasciotomy in cases presenting with compartment syndrome (Brandt et al, 2008; Maynor, 2009).

The surgery also minimises the overall tissue loss because it removes the dead tissue and, therefore, preventing the infection spreading into the fascial planes and soft tissue (Wong and Shih, 1992). All deep fascia muscle should be inspected for potential involvement with *Streptococcal myositis* or *Clostridium* infection. All non-viable tissue is excised, leaving a wide margin of unaffected tissue to minimise the risk of occurrence (Fink and DeLuca, 2002; Headley, 2003).

Because of novel reconstruction methods using simple to complex skin grafts, which plastic surgeons can employ to cover tissue defects, the extent of wound debridement should not be needlessly limited (Roje et al, 2011). The wound was washed with hydrogen peroxide and saline. A swab was taken for analysis and the patient began taking antibiotics. The surgeon dressed the wound postoperatively with a proflavine pack of gauze in the right inner thigh wound and a large gamgee and crepe bandage. The blood results also assisted with the diagnosis of necrotising fasciitis.

Care Pathway

Postoperatively, the patient was transferred to the intensive care unit and underwent two further surgical debridements on 18 March and 20 March, 2012. When the wound was stabilised, the surgeon decided to treat it with topical negative pressure. This was applied on 20 March in theatre by the surgeon and reapplied under supervision of the Avance NPWT company representative on 24 March, 2012.

The author was requested by the surgeon to apply the topical negative pressure

Figure 2: Dressing change demonstrating the extent of tissue damage in the groin.

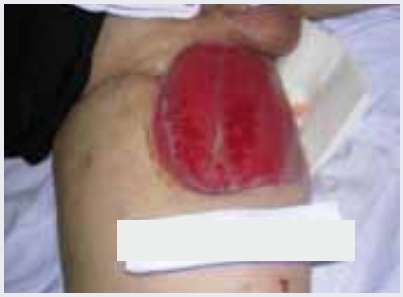

Figure 3: Another example of the full extent of tissue damage.

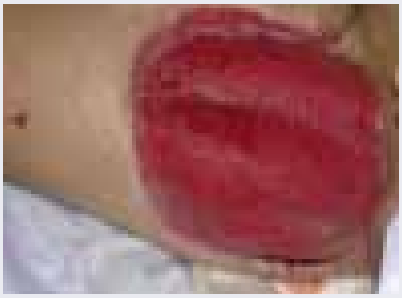

Figure 4: Progress of the wound one month after Figure 3.

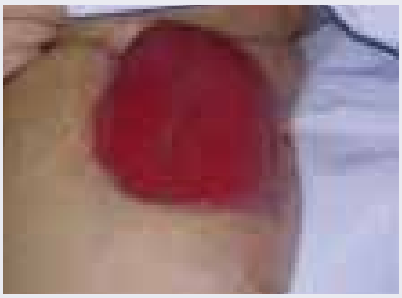

Figure 5: Progress of the wound one month after Figure 3.

Figure 6: Avance film with Safetac in situ.

dressing on the intensive care unit on 27 March, 2012. The patient was being seen by the dietician to help manage protein loss from the wound. Calorific input was increased to allow the patient to make a full recovery and to provide the essential energy and amino acids required for tissue repair (Bashford et al, 2002).

On the first consultation, the nursing staff on the intensive care unit joined the author and the company representative to decide the best strategy in which to apply the topical negative pressure dressing. It was decided that the best way to apply the dressing was with the patient on his back and then tilted on his side to gain an effective seal around his buttocks and top of his thigh. This had to be coordinated with intensive care staff as the initial dressing application took two hours to perform.

Application of the topical negative pressure

On removal of the dressing, the inner wound measured 20cm x 40cm and the tissue was extremely healthy. The wound was assessed using the TIME (tissue, infection, moisture and edge) model.

The tissue appeared to be 80% pink and 20% yellow, but was well-perfused (*Figure 2*). The tissue around the wound was well perfused and demonstrated no evidence of infection. There was no maceration of the wound edges, but the limb was very swollen and had a high amount of oedema present. The edges of the wound were non-advancing and inflammation had subsided.

The Avance topical negative dressing therapy was applied using the following method. Hydrocolloid was cut into narrow strips and then applied around the periwound edges to ensure a good seal. Mepiseal® (Mölnlycke Health Care) was also applied around the groin area, which was a difficult area to gain a good seal that would last until the next dressing change.

The two large green foams were applied to the right groin wound and cavity in the inner thigh. The drape and drainage were applied. The Avance pump was set at 120Hmg of negative pressure and continuous therapy. Staff were instructed that if the topical negative pressure dressing went down or lost its seal for longer than two hours the wound would require redressing with Sorbsan ribbon and Biatain® (Coloplast) or reapplication of the topical negative pressure dressing, which is a standard care pathway if the topical negative therapy stops and a qualified nurse is not available to reapply it.

The topical negative dressing changes were undertaken on Monday, Wednesday and Friday, in conjunction with intensive care study and with the assistance of the company representative. This had to be planned and organised with the intensive therapy unit.

The patient was alert on his third dressing change, but had been given morphine during this procedure. He was initially very shocked at the size of the wound and the extent of the debridement. He was reassured that the plastic surgeon would be involved as early as possible for reconstructive surgery.

The patient was transferred from the intensive care unit to a general surgical ward on the 30 March, 2012 and the ward staff on the surgical ward took over the management of this patient's topical negative dressing changes with the assistance of the company representative.

The author and plastic surgeon reviewed the patient on 18 April, 2012 four weeks after his initial surgery. A new photograph was taken (*Figure 3*). The wound had now reduced to 14cm x 13.5cm and the wound bed was extremely clean. There was 100% pink granulation tissue and the cavity in the inner thigh was also healing well.

In order to facilitate his transfer to the nearest plastic surgery unit, the patient required three clear wound swabs to demonstrate he was free from Methicillin-resistant *Staphylococcus aureus* (MRSA). The patient's pain relief had been reduced to Ibuprofen during dressing changes. The patient reported the most painful part of the dressing regimen was the removal of the film dressing.

The patient was worried that his inner thigh was larger than his left thigh. The plastic surgeon, therefore, explained about oedema; that swelling may remain for some time. The plastic surgeon discussed how he would cover the wound and that there would be a dent due to tissue loss in the subcutaneous layer of the skin.

The plastic surgeon requested a new photograph to be taken on the following Tuesday (24 April, 2012) so he could review it when he was in clinic to check the progress of this wound prior to plastic surgery.

The author reviewed the patient on 24 April, 2012 and a new photograph was taken with the patient's permission for teaching and publication (*Figures 4 and 5*). The wound had reduced to 13cm x 13.5cm, while the wound bed was extremely healthy. There was a 6cm cavity in the inner thigh, but there was no infection present and the moisture balance of the wound was optimal with the wound edges well-perfused. The patient required three clear wound swabs before being transferred to the nearest plastic unit if there was a bed.

The author asked the patient if he would evaluate the Avance Film with Safetac technology, to ascertain if it was comfortable and reduced the pain of the traditional transparent film removal. Safetac technology has been developed to employ soft silicone technology, which adheres readily to intact dry skin while adhering to the surface of the moist wound bed.

Safetac technology can be applied without causing damage to newly formed tissue in the wound or stripping in the periwound area, as well as minimising pain at dressing removal (Cutting, 2008). Safetac technology has been demonstrated to reduce trauma to periwound skin and wound-related pain during and after dressing change (White, 2008).

A photograph demonstrates the Avance Film with Safetac technology *in situ* (*Figure 6*). The wound was redressed with half a round of PHMB gauze providing conformability to the wound bed to promote wound healing. The Avance pump was reduced to 80Hmg of negative pressure and continuous therapy due to the fact that the patient experienced pain at 120Hmg. His final swab was taken ready for his transfer as the plastic surgeon was ready to accept him when a bed become available. The author reviewed the patient once more prior to transfer and the wound continued to progress.

The patient feedback on the Avance Film with Safetac technology stated that he felt no pain on removal, rated on a visual analogue scale. He said it was very comfortable but there was, however, some leakage from the dressing and it had to be patched. He was delighted about the ease of removal as he had had to use one tin of adhesive removal spray to removal the traditional film dressing.

CONCLUSION

This article has outlined the most relevant literature available on the management and treatment of necrotising fasciitis. Early diagnosis of this life-threatening infection is crucial, as it can spread rapidly. Close collaboration between the multidisciplinary team, including general surgeons and plastic surgeons, is vital from the onset. Effective surgical debridement, antibiotics, pain relief, good nutrition and effective wound management are all vital to deliver a good patient outcome.

While this is only one case study, it demonstrates the effectiveness of the Avance topical negative system. The management of these complex wounds requires a great deal of collaboration between members of the multidisciplinary team and the company. The Avance Film with Safetac technology delivers a pain-free dressing change towards the end of the patient care pathway.

Holistic patient care should also focus on patients and families to both enable significant health gain, and also to allow patients to recover physically and emotionally following this life-threatening condition. The current health economy prompts clinicians to deliver cost-effective care within the resources available to them, while delivering quality patient outcomes. WUK

DECLARATION

This article was produced with the support of Mölnlycke Health Care.

'Safetac technology can be applied without causing damage to newly formed tissue.'