The diagnosis of necrotizing fasciitis

Chin-Ho Wong[a] and Yi-Shi Wang[b]

Purpose of review

A delay in the diagnosis and appropriate treatment of necrotizing fasciitis has clearly been demonstrated to increase mortality. However, paucity of specific cutaneous signs makes early recognition extremely difficult. This review highlights recent developments in the approaches to the diagnosis of necrotizing fasciitis.

Recent findings

A clinical staging of necrotizing fasciitis is proposed to better define the progression of the disease. Several clinical subtypes of necrotizing fasciitis have been described recently with hyperacute and sub-acute variants. Imaging techniques, such as magnetic resonance imaging and frozen section biopsies, have been reported to be of value in the early recognition of necrotizing fasciitis. However availability and cost limit the routine use of these tests. Several diagnostic adjuncts that have been developed recently to help in early recognition will be discussed. These include the fasciitis LRINEC (laboratory risk indicator for necrotizing fasciitis) score and transcutaneous tissue oxygen saturation monitoring. Some may have the potential for widespread application in the assessment of severe soft tissue infections.

Summary

Delayed recognition, with consequent massive soft tissue loss and sepsis, remains a deadly pitfall in the management of necrotizing fasciitis. With a better understanding of the clinical manifestations and the potential use and limitations of various diagnostic adjuncts available for the assessment of equivocal cases of soft tissue infections, it is hoped that a clear and logical approach to the diagnosis of necrotizing fasciitis may be developed.

Keywords

diagnosis, frozen section, group A streptococcus, imaging, LRINEC score, necrotizing fasciitis, necrotizing soft tissue infections, tissue oxygen saturation, review

Curr Opin Infect Dis 18:101–106. © 2005 Lippincott Williams & Wilkins.

[a]Department of Plastic Surgery, Singapore General Hospital and [b]Division of Dermatology, Department of Internal Medicine, Changi General Hospital, Singapore

Correspondence to Dr Chin-Ho Wong, Department of Plastic Surgery, Singapore General Hospital, Outram Road, Singapore 169608
Tel: +65 6321 4686; fax: +65 6220 9340; e-mail: wchinho@hotmail.com

Current Opinion in Infectious Diseases 2005, 18:101–106

Abbreviations

MRI magnetic resonance imaging
LRINEC laboratory risk indicator for necrotizing fasciitis

© 2005 Lippincott Williams & Wilkins
0951-7375

Introduction

Necrotizing fasciitis is perhaps the most severe form of soft tissue infection primarily involving the superficial fascia. This disease has bewildered physicians for centuries. Hippocrates in the fifth century BC gave the first description of this dreaded disease [1]. The first report of this disease in the United States was by a Confederate Army surgeon, Joseph Jones in 1871 and he named this entity 'hospital gangrene' [2]. Meleney [3] in 1924 reported an outbreak of hospital gangrene in Beijing and coined the term hemolytic streptococcal gangrene. The term necrotizing fasciitis was first introduced by Wilson [4] in 1952 and is the preferred term today describing the most consistent and key feature of this disease, fascia necrosis.

While the understanding of the pathophysiology of necrotizing fasciitis continues to improve, the mortality of this disease remains alarmingly high with reported mortality rates ranging from 6 to 76% [5]. Delayed diagnosis and consequently delayed operative debridement have been shown in multiple studies to increase mortality [5,6•,7–16]. This is understandable: the greater the delay, the greater the tissue loss and sepsis with consequent increased mortality. One of the main reasons for the continued high mortality of patients afflicted by necrotizing fasciitis today is a failure to recognize and diagnose the condition early because of the paucity of specific cutaneous signs early in its evolution [5–12]. It is therefore imperative that the treating physician has a high index of suspicion and is aware of the armamentarium of diagnostic adjuncts at his disposal when confronted with such clinical uncertainties. This article discusses the clinical presentation of necrotizing fasciitis and highlights some recent advances in diagnostic adjuncts that potentially may be helpful in the diagnosis of necrotizing fasciitis.

Pathophysiology and clinical presentation of necrotizing fasciitis

Understanding the pathophysiology of necrotizing fasciitis is important in discerning the clinical presentation of

this disease. The primary site of pathology is in the superficial fascia. Bacteria proliferate within the superficial fascia and elaborate enzymes and toxins enable the organisms to spread through the fascia. The precise mechanism of spread has not been fully elucidated but some investigators have attributed it to expression of bacterial enzymes such as hyaluronidase, which degrades the fascia [17]. The key pathological process resulting from this uncontrolled proliferation of bacteria is angiothrombotic microbial invasion and liquefactive necrosis of the superficial fascia. Histologically, necrosis of the superficial fascia, polymorphonuclear leukocyte infiltration of the deep dermis and fascia, thrombosis and suppuration of the veins and arteries coursing through the fascia, and microorganism proliferation within the destroyed fascia are seen. As this process progresses, occlusion of perforating nutrient vessels to the skin causes progressive skin ischemia [18]. This is the underlying event that is responsible for the cutaneous manifestations of necrotizing fasciitis as the disease evolves. Initially a horizontal phase predominates with rapid spread through the fascia with extensive undermining of the apparently normal looking skin. As the condition evolves, ischemic necrosis of the skin ensues with gangrene of the subcutaneous fat, dermis and epidermis, manifesting progressively as bullae formation, ulceration and skin necrosis [6•].

The clinical presentation of necrotizing fasciitis has been investigated by several authors [5,6•,11,16,19,20]. Table 1 describes our proposed clinical staging of necrotizing fasciitis based on progressive skin changes as the disease evolves. Skin ischemia is the underlying process that explains these progressive changes seen clinically. Commonly skin changes are heterogenous and the skin area with the most advanced skin changes should be taken as the clinical stage. It is generally accepted that early on in the evolution of necrotizing fasciitis (stage 1 necrotizing fasciitis), the disease is clinically indistinguishable from severe soft tissue infection such as cellulitis and erysipelas presenting with only pain, tenderness and warm skin [5,6•,11,16,19,20]. In necrotizing fasciitis, margins of tissue involvement are often poorly defined with tenderness extending beyond the apparent area of involvement [6•,19,20]. Lymphangitis is rarely seen in necrotizing fasciitis [11]. Blister or bulla formation is an important diagnostic clue [6•,20]. When present, it signals the onset of critical skin ischemia (stage 2 necrotizing fasciitis). Blisters are caused by ischemia-induced necrolysis as the vessels coursing through the fascia to supply the skin are progressively thrombosed by the invading organisms. Blistering or bullae formation is rarely seen in erysipelas or cellulitis and should raise the suspicion of necrotizing soft tissue infection [21,22]. The late stage (stage 3 necrotizing fasciitis) signals the onset of tissue necrosis and is characterized by the so call 'hard signs' of necrotizing soft tissue infection such as hemorrhagic bullae, skin anesthesia and frank skin gangrene [5,6•]. Clinical staging is important to better define disease progression and to heighten awareness during serial evaluation of soft tissue infections.

Table 1. Clinical features of necrotizing fasciitis as the disease progress through clinical stages

Stage 1 (Early)	Stage 2 (Intermediate)	Stage 3 (Late)
Tenderness to palpation (extending beyond the apparent area of skin involvement)	Blister or bullae formation (serous fluid)	Hemorrhagic bullae
Erythema	Skin fluctuance	Skin anesthesia
Swelling	Skin induration	Crepitus
Warm to palpation		Skin necrosis with dusky discoloration progressing to frank gangrene

The literature has stressed the systemic manifestations of necrotizing soft tissue infection with high fever, hypotension, prostration and multiorgan failure [3,5,11,16,20]. The effects are classically caused by superantigens produced by group A streptococcus, when known as streptococcal toxic shock syndrome [23]. In a good review, Green *et al.* [11] listed fever and signs and symptoms of systemic toxicity as diagnostic features of necrotizing fasciitis. This is certainly true in many cases and patients with these systemic features associated with soft tissue infections should certainly raise the suspicion of necrotizing fasciitis. However, we are coming to appreciate that often patients can appear systemically quite well, at least initially [6•]. In a review of 89 consecutive patients, Wong *et al.* [6•] found that only 53% were febrile and 18% were hypotensive at presentation. This is particularly so in immunocompromised patients such as diabetics. It should be remembered that these patients may have a blunted immunological response to infection and may appear systemically well initially despite the presence of severe necrotizing infection. The widespread use of broad-spectrum antimicrobials at the primary care level has also been speculated to be responsible for the apparent lack of systemic manifestation of such severe soft tissue infection. The use of antibiotics reduces the systemic bacterial load and the incidence of organ failure [6•]. This, however, has very little effect on the primary site pathology, where the liquefactive necrosis blocks tissue penetration of antimicrobials. Because of this lack of systemic manifestations, many physicians consider these patients 'too well' to entertain the diagnosis of necrotizing fasciitis, resulting in significant delay in operative intervention.

Hyperacute and sub-acute variants of necrotizing fasciitis are increasingly reported in the literature. The hyperacute variant presents with an extremely fulminant course with extensive undermining of surrounding tissue, severe

septicemia and multiorgan failure within 24 h of the inciting event [24,25]. Vibrio species, including *Vibrio vulnificus*, *Vibrio parahaemolyticus* and *Photobacterium damsela* (*Vibrio damsela*), are notable causative agents of hyperacute necrotizing fasciitis. Vibrio species are comma-shaped Gram-negative rods that multiply well in warm coastal waters (>20 °C) such as in Asia (Singapore, Hong Kong, Southern China, Thailand and Taiwan), South America and Mexico. Reports of such infections have also emerged from cooler regions such as New England, Belgium and Scandinavia. Patients are often over 50 years of age and have underlying comorbidities particularly chronic liver disease and diabetes mellitus [24]. Because of the rapidity of the process, there is minimal time for specific cutaneous signs to develop and the skin looks deceptively normal. The true extent of the infection is appreciated only at operation. Shock and multi-organ derangement is a feature and is a crucial diagnostic clue of hyperacute necrotizing fasciitis. Early diagnosis, extensive debridement and, in extreme cases, willingness to amputate at a very early stage may be the only intervention that can save the lives of patients. Otherwise the disease has an almost 100% mortality [25].

Sub-acute necrotizing fasciitis in contrast runs a more indolent course. Patients often complain of areas of festering soft tissue infection with minimal pain and discomfort. This can fester on for weeks to months. Sudden deterioration is, however, commonly seen and if not treated by early aggressive surgical debridement is associated with a high mortality [26•]. This departure of clinical presentation from the classical necrotizing fasciitis makes clinical recognition of the sub-acute form very difficult. Many have considered the clinical signs too mild to entertain the diagnosis of necrotizing fasciitis. Progression is certain, however, and a delay in diagnosis results in greater soft tissue loss and increases mortality. A high index of suspicion is therefore important when evaluating cases of suspected necrotizing fasciitis. In this context early involvement of an experienced surgical team may be invaluable when patients are being treated in a medical unit.

Diagnosis of necrotizing fasciitis

Necrotizing fasciitis is a clinical diagnosis with corroborating operative findings [5,6•,16,20]. The operative findings in necrotizing fasciitis include the presence of grayish necrotic fascia, demonstration of a lack of resistance of normally adherent superficial fascia to blunt dissection, lack of bleeding of the fascia during dissection and the presence of foul smelling 'dishwater' pus. Tissue specimens for culture and histology are crucial and should be performed for all patients without exception. Culture-guided appropriate antimicrobial selection and histology give a confirmation of diagnosis. These specimens should be generous and taken from the margins of involvement of the fasciitis (of apparently normal fascia) to ensure a good yield.

Histological criteria for diagnosis necrotizing fasciitis as described by Stamenkovic and Lew [18] reliably identified even early cases of necrotizing fasciitis. The histologic criteria for diagnosis were necrosis of the superficial fascia, polymorphonuclear infiltration of the dermis and fascia, fibrinous thrombi of arteries and veins coursing through the fascia, angiitis with fibrinoid necrosis of arterial and venous walls, presence of microorganisms within the destroyed fascia and dermis and an absence of muscle involvement. Histology is important particularly in cases for which the operative findings are equivocal for early necrotizing fasciitis, as it determines the need for an early second look and repeat debridement.

Diagnostic adjuncts for necrotizing fasciitis

Many studies have been done to determine if imaging can reliably differentiate between cellulitis and necrotizing fasciitis. Computed tomography scan, ultrasound and magnetic resonance imaging (MRI) for the imaging of necrotizing soft tissue infection have been intensely studied [27–38]. Features reported to be indicative of necrotizing fasciitis on the computed tomography scan include deep fascial thickening, enhancement, fluid and gas in the soft tissue planes in and around the superficial fascia [27–29]. On ultrasound, suggestive features are thickening and distortion of the deep fascia and fluid collections along the deep fascia [30–33]. There is some controversy on MRI. Some authors have described features that they believe are distinct for necrotizing fasciitis. These include deep fascial thickening, deep fascial fluid collections and hyperintense T2W signal within the muscles [34–36]. Fascial enhancement has been described as a feature by some authors [34,35], whilst other authors report lack of fascial enhancement as a reliable indicator [36]. Other authors have stated that these features are non-specific and may lead to over or under-diagnosis [37,38]. This is because the sensitivity of MRI often exceeds its specificity, resulting in overestimation of extent of deep fascial involvement. However, a negative deep fascial involvement on MRI effectively excludes necrotizing fasciitis. While imaging is an invaluable diagnostic adjunct, it may not be readily available and is certainly not cheap. Certainly, in patients with a suspicion of necrotizing fasciitis, operative debridement should not be delayed while waiting for an MRI scan.

In a small retrospective study, Stamenkovic and Lew [18] reported the utility of frozen section biopsy for early diagnosis of necrotizing fasciitis and found improved survival in patients in whom the condition was detected early by frozen section biopsy compared with patients in whom the diagnosis was made clinically. These authors

recommend excision of approximately a 1 cm^3 piece of tissue specimen from a suspected area under local anesthesia for immediate examination. Positive cases are subjected to immediate surgical debridement. The use of frozen section biopsy was also supported by the work of Majeski and Majeski [12]. In that paper, 43 patients with suspected necrotizing fasciitis were subjected to frozen section examination. Twelve (28%) were later confirmed to have necrotizing fasciitis. While this approach is effective in detecting early necrotizing fasciitis and has indeed been demonstrated to reduce mortality [12,18], adopting the use of frozen sections would require strong support from pathologists familiar with the interpretation of soft tissue infections. The physicians must also be willing to accept a high negative biopsy rate and some morbidity from the tissue sampling. Arguably, considering these associated problems and limitations of frozen section biopsies, it may well be easier to explore all suspected patients in the operating theater. One possibility is to perform the 'finger test' as described by Andreasen *et al.* [39], by which a 2 cm incision down to the deep fascia is made under local anesthesia. Probing of the level of the superficial fascia is then performed. The lack of bleeding, foul smelling dishwater pus and minimal tissue resistance to finger dissection constitute a positive finger test, which is diagnostic of necrotizing fasciitis. The surgeon can then proceed to perform a formal wound debridement if the finger test is positive.

An area of recent development is the analysis of changes of the biochemical profile of patients with necrotizing fasciitis induced by severe sepsis. The clinical profile of nascent cases of necrotizing fasciitis is indistinguishable from other soft tissue infection such as cellulitis. The possible use of the biochemical profile derangement to detect cases of necrotizing fasciitis was first proposed by Wall *et al.* [40,41]. Subsequently, on the basis of the hypothesis that the biochemical profile may manifest a diagnostic clue, Wong *et al.* [42••] compared laboratory parameters of patients with necrotizing fasciitis and patients with other severe soft tissue infections such as cellulitis. The laboratory tests they analyzed were routinely performed for the assessment of severe soft tissue infections (complete blood count, electrolytes, erythrocyte sedimentation rate and C-reactive protein). A numerical score based on the relative significance of the laboratory parameters in distinguishing necrotizing fasciitis from other soft tissue infections, called the LRINEC (laboratory risk indicator for necrotizing fasciitis) score, was devised (Table 2). The score is calculated by totaling up each of the six predictive factors found to be significant in that study. At a cutoff of a LRINEC score of 6 or greater, the model has a positive predictive value of 92.0% (95% CI 84.3–96.0) and negative predictive value of 96.0% (95% CI 92.6–97.9). A score of eight or more is strongly predictive of necrotizing fasciitis (positive predictive value 93.4%, 95% CI 85.5–97.2) [42]. The significance of this study is that all parameters needed for calculation of the LRINEC score are readily available as they are commonly performed for the evaluation of severe soft tissue infections. This score, however, needs to be validated prospectively before its routine application can be advocated [43].

Table 2. The LRINEC (laboratory risk indicator for necrotizing fasciitis) score

Variable	Score
C-reactive protein (mg/l)	
<150	0
150 or more	4
Total white cell count (per mm^3)	
<15	0
15–25	1
>25	2
Hemoglobin (g/dl)	
>13.5	0
11–13.5	1
<11	2
Sodium (mmol/l)	
135 or more	0
<135	2
Creatinine (μmol/l)	
141 or less	0
>41	2
Glucose (mmol/l)	
10 or less	0
>10	1

To convert the values of glucose to milligrams per deciliter, multiply by 18.015. To convert the values of creatinine to milligrams per deciliter, multiply by 0.01131. Adapted with permission [42••].

Wang and Hung [44••] investigated the use of tissue oxygen monitoring with near-infrared spectroscopy for the diagnosis of necrotizing fasciitis. In this prospective observational study 234 patients were included and 19 eventually confirmed to have necrotizing fasciitis. The authors reported, at a cut-off tissue oxygen saturation of less than 70%, this test to have a sensitivity of 100% and a specificity of 97%. Patient selection for the study, however, critically compromised the clinical utility of their findings. All patients with chronic venous stasis, peripheral vascular disease, shock and systemic hypoxia were excluded from the study. This is understandable because patients with these conditions would have impaired tissue perfusion and oxygen saturation and thus give a false positive result. However, most patients who developed necrotizing fasciitis have underlying predisposing conditions that make them susceptible. This is a pity, as this is the group of patients in whom early diagnosis would profoundly affect outcome [45]. Still, in the select group of patients (namely healthy patients without comorbidity) tissue oxygen saturation monitoring may potentially be a valuable diagnostic adjunct.

Conclusion

Necrotizing fasciitis remains one of the most devastating soft tissue infections in modern medicine. Many adjuncts have been described to help in the early recognition of the disease. What is needed in the management of necrotizing fasciitis is a clear and focused approach to the problem. Early surgical debridement decreases mortality and the aim is to diagnose the condition early, ideally within 24 h of admission. Depending on resources available, the managing team should exclude the diagnosis of necrotizing fasciitis with utmost urgency. If MRI is needed, it should be performed within the next few hours. Certainly decision should not be delayed beyond 24 h while waiting for imaging to be available. Future developments of diagnostic adjuncts that can help in the identification of patients with necrotizing fasciitis should be focused on cheap and easily performed 'bedside' tests that are readily available. The LRINEC score and transcutaneous tissue oxygen monitoring are examples of such adjuncts that were recently described but these have yet to show improved diagnostic yields or decreased mortality. Ultimately, when doubts remain, often the best thing to do is to perform an early operative exploration.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1 Descamps V, Aitken J, Lee MG. Hippocrates on necrotizing fasciitis. Lancet 1994; 344:556.

2 Jones J. Investigation on the nature, causes and treatment of hospital gangrene as it prevails in the confederate armies 1861–1865. In: Hasting Hamilton F, editor. Surgical memoirs of the war of rebellion. New York: Sanitary Commission; 1871.

3 Meleney FL. Hemolytic streptococcus gangrene. Arch Surg 1924; 9:317–364.

4 Wilson B. Necrotizing fasciitis. Am Surg 1952; 18:416.

5 McHenry CR, Piotrowski JJ, Petrinic D, Malangoni MA. Determinants of mortality in necrotizing soft tissue infections. Ann Surg 1995; 221:558–563.

6 • Wong CH, Chang HC, Pasupathy S, *et al.* Necrotizing fasciitis: clinical presentation, microbiology and determinants of mortality. J Bone Joint Surg Am 2003; 85A:1454–1460.

This recent retrospective study of value clearly showed that delayed operative debridement increases mortality in necrotizing fasciitis. The paper also describes the clinical and microbiological aspects of the disease.

7 Voros D, Pissiotis C, Georgantas D, *et al.* Role of early and aggressive surgery in the treatment of severe necrotizing soft issue infections. Br J Surg 1993; 80:1190–1191.

8 Rea WJ, Wyrick WJ. Necrotizing fasciitis. Ann Surg 1970; 172:957–964.

9 Masjeski JA, Alexander JW. Early diagnosis, nutritional support and immediate extensive debridement improve survival in necrotizing fasciitis. Am J Surg 1983; 145:781–787.

10 Bilton BD, Zibari GB, McMillan RW, *et al.* Aggressive surgical management of necrotizing fasciitis serves to decrease mortality: A retrospective study. Am J Surg 1998; 64:397–400.

11 Green RJ, Dafoe DC, Raffin TA. Necrotizing fasciitis. Chest 1996; 110:219–229.

12 Majeski J, Majeski E. Necrotizing fasciitis: Improved survival with early recognition by tissue biopsy and aggressive surgical treatment. South Med J 1997; 90:1065–1068.

13 Wang K-C, Shih C-H. Necrotizing fasciitis of the extremities. J Trauma 1992; 32:259–264.

14 Freischlag JA, Ajalat G, Busuttil RW. Treatment of necrotizing soft tissue infections: The need for a new approach. Am J Surg 1985; 149:751–755.

15 Ault MJ, Geiderman J, Sokolov R. Rapid identification of group A streptococcus as the cause of necrotizing fasciitis. Ann Emerg Med 1996; 28:227–230.

16 Elliot DC, Kufera JA, Myers RA. Necrotizing soft tissue infections: Risk factors for mortality and strategies for management. Ann Surg 1996; 224:672–683.

17 Wilkerson R, Paull W, Coville FV. Necrotizing fasciitis: Review of the literature and case report. Clin Orthop 1987; 216:187–192.

18 Stamenkovic I, Lew PD. Early recognition of potentially fatal necrotizing fasciitis: The use of frozen section biopsy. N Engl J Med 1984; 310:1689–1693.

19 Kaiser RE, Cerra FB. Progressive necrotizing surgical infections. A unified approach. J Trauma 1981; 21:349.

20 Patiño JF, Castro D. Necrotizing lesion of the soft tissue: A review. World J Surg 1991; 15:235–239.

21 Seal DV. Necrotizing fasciitis. Curr Opin Infect Dis 2001; 14:127–132.

22 Seal DV, Hay RJ, Middleton KR. Skin and wound infection: investigation and treatment in practice. London: Martin Dunitz; 2000.

23 Bisno AL, Brito MO, Collins CM. Molecular basis of group A streptococcal virulence. Lancet Infect Dis 2003; 3:191–200.

24 Tang WM, Ho PL, Fung KK, *et al.* Necrotizing fasciitis of a limb. J Bone Joint Surg Br 2001; 83B:709–714.

25 Goodell KH, Jordan MR, Graham R, *et al.* Rapidly advancing necrotizing fasciitis caused by Phytobacterium (vibrio) damsela: A hyperaggressive variant. Crit Care Med 2004; 32:278–281.

26 • Wong CH, Tan SH. Subacute necrotising fasciitis. Lancet 2004; 364: 1376.

This case report demonstrates that subacute necrotizing fasciitis presents with atypical and mild symptoms initially.

27 Wysoki MG, Santora TA, Shah RM, Friedman AC. Necrotizing fasciitis: CT characteristics. Radiology 1997 Jun; 203:859–863.

28 Becker M, Zbaren P, Hermans R, *et al.* Necrotizing fasciitis of the head and neck: role of CT in diagnosis and management. Radiology 1997; 202:471–476.

29 Walshaw CF, Deans H. CT findings in necrotising fasciitis–a report of four cases. Clin Radiol 1996; 51:429–432.

30 Yen ZS, Wang HP, Ma HM, *et al.* Ultrasonographic screening of clinically-suspected necrotizing fasciitis. Acad Emerg Med 2002; 9:1448–1451.

31 Parenti GC, Marri C, Calandra G, *et al.* Necrotizing fasciitis of soft tissues: role of diagnostic imaging and review of the literature. Radiol Med (Torino) 2000; 99:334–339.

32 Chao HC, Kong MS, Lin TY. Diagnosis of necrotizing fasciitis in children. J Ultrasound Med 1999; 18:277–281.

33 Tsai CC, Lai CS, Yu ML, *et al.* Early diagnosis of necrotizing fasciitis by utilization of ultrasonography. Kaohsiung J Med Sci 1996; 12:235–240.

34 Schmid MR, Kossmann T, Duewell S. Differentiation of necrotizing fasciitis and cellulitis using MR imaging. AJR Am J Roentgenol 1998; 170:615–620.

35 Rahmouni A, Chosidow O, Mathieu D, *et al.* MR imaging in acute infectious cellulitis. Radiology 1994; 192:493–496.

36 Brothers TE, Tagge DU, Stutley JE, *et al.* Magnetic resonance imaging differentiates between necrotizing and non-necrotizing fasciitis of the lower extremity. J Am Coll Surg 1998; 187:416–421.

37 Loh NN, Ch'en IY, Cheung LP, Li KC. Deep fascial hyperintensity in soft-tissue abnormalities as revealed by T2-weighted MR imaging. AJR Am J Roentgenol 1997; 168:1301–1304.

38 Arslan A, Pierre-Jerome C, Borthne A. Necrotizing fasciitis: unreliable MRI findings in the preoperative diagnosis. Eur J Radiol 2000; 36:139–143.

39 Andreasen TJ, Green SD, Childers BJ. Massive soft-tissue injury: Diagnosis and management of necrotizing fasciitis and purpura fulminans. Plast Reconstr Surg 2001; 107:1025–1035.

40 Wall DB, de Virgilio C, Black S, Klein SR. Objective criteria may assist in distinguishing necrotizing fasciitis from non-necrotizing soft tissue infection. Am J Surg 2000; 179:17–21.

41 Wall DB, Klein SR, Black S, de Virgilio C. A simple model to help distinguish necrotizing from non-necrotizing soft tissue infection. J Am Coll Surg 2000; 191:227–231.

42 Wong CH, Khin LW, Heng KS, *et al.* The LRINEC (Laboratory Risk Indicator •• for Necrotizing Fasciitis) score: A tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med 2004; 32:1535–1541.

This describes a novel scoring system that is capable of distinguishing necrotizing fasciitis from other soft tissue infections. A prospective validation of this score is warranted as the score was developed from retrospective analysis.

43 Barie PS. The laboratory risk indicator for necrotizing fasciitis (LRINEC) score: useful tool or paralysis by analysis? Crit Care Med 2004; 32:1618–1619.

44 Wang TL, Hung CL. Role of tissue oxygen saturation monitoring in diagnosing •• necrotizing fasciitis of the lower limbs. Ann Emerg Med 2004; 44:222–228.

This interesting study used tissue oxygen saturation monitoring to predict the possibility of necrotizing fasciitis, with impressive results. However, patient selection severely limits the usefulness of this study.

45 Wong CH. Tissue oxygen saturation monitoring in diagnosing necrotizing fasciitis of the lower limb: A valuable tool but only for a selected few. Ann Emerg Med (in press).