

Management and morbidity of cellulitis of the leg

Neil H Cox BSc FRCP Graham B Colver MB FRCP¹ William D Paterson DM FRCP

J R Soc Med 1998;91:634-637

SUMMARY

Ascending cellulitis of the leg is a common emergency. An audit was conducted in two district general hospitals to determine how it is managed and the long-term morbidity, and to formulate a treatment strategy. Case notes were reviewed for 92 patients admitted to hospital under adult specialties.

Mean duration of inpatient therapy was 10 days. A likely portal of entry was identified in 51/92 cases, of which the commonest were minor injuries and tinea pedis. Pathogens were rarely identified, group G streptococci being the single most frequent organism. Benzylpenicillin was administered in only 43 cases. Long-term morbidity, identified in 8 of 70 patients with over six months' follow-up, included persistent oedema (6) and leg ulceration (2); an additional 19 patients had either suffered previous episodes or experienced a further episode subsequently.

Ascending cellulitis of the leg has substantial short-term and long-term morbidity. Important but often neglected therapeutic suggestions are the inclusion of benzylpenicillin in all cases without a contraindication, assessment and treatment of tinea pedis, use of support hosiery, and serological testing for streptococci to confirm the diagnosis in retrospect. The high frequency of recurrent episodes suggests that longer courses of penicillin, or penicillin prophylaxis, might be useful.

INTRODUCTION

Rapidly ascending cellulitis of the leg is a common medical emergency characterized by swelling, oedema, fever and malaise. It is a truly multidisciplinary disorder since patients may present to casualty, general medical, elderly care, dermatology, orthopaedic, vascular or general surgical departments. The most frequent causal organisms for this pattern of cellulitis are streptococci^{1,2}, although some cases may be due to staphylococcal or mixed infection, especially if the cellulitis is localized or follows a penetrating injury. Bacteriological diagnosis is difficult since most patients have no wound site from which cultures can be obtained¹⁻⁸. Prompt investigations may be required to exclude differential diagnoses such as deep venous thrombosis, infected venous eczema or lymphoedema, tibial compartment syndrome, and myositis or fasciitis. Although the potential severity of streptococcal infections such as necrotizing fasciitis or streptococcal toxic shock syndrome are well known², and the short-term therapeutic problems of streptococcal erysipelas/cellulitis are well recognized, the potential long-term morbidity of lower limb cellulitis is poorly documented.

PATIENTS AND METHODS

All case records identifiable from our 'PAS' hospital inpatient databases with a possible diagnosis of ascending cellulitis of the leg were reviewed in our two hospitals over three years (Carlisle) and six months (Chesterfield). This disorder has no specific ICD9 code and required medical review of records by two of us (NHC, GBC) to exclude cases with localized cellulitis, bursitis or abscesses or other incorrect diagnoses. Data extracted from the valid cases included demographic details, evidence of predisposing cause, records of short-term morbidity (pattern of spread and associated systemic features such as pyrexia or malaise), any investigations to prove the diagnosis, initial antibiotic treatment, duration of admission, and treatment after discharge. Any previous or subsequent episodes were documented, and any other evidence of long-term morbidity was recorded for those patients with subsequent hospital casenote records at least six months after their admission.

RESULTS

Demographic and admission data

92 patients were evaluated (38 men, 54 women) with age range 24-93 years (median 67). Duration of hospital admission was 1-47 days (mean 10). In 50/92 pyrexia or malaise was specifically recorded at the time of admission; 31 were documented to be afebrile but the cellulitis had

Department of Dermatology, Cumberland Infirmary, Carlisle CA2 7HY, UK;

¹Department of Dermatology, Chesterfield & North Derbyshire Royal Hospital, Chesterfield S44 5BL, UK

Correspondence to: Dr N H Cox

not resolved with antibiotic treatment before hospital referral.

Investigations and treatment

A likely portal of entry was identified in 51/92 patients (Table 1), the most important treatable factor being tinea pedis which was probable in 12 cases but only dermatologically confirmed in 6. In most cases, spread of cellulitis was from the toes or dorsum of foot upwards to around the knee, but 15/92 were recorded to have calf or lower leg swelling without obvious swelling of the foot. Bacteriology swabs were sent in 18 cases, of which 3 were negative (all with previous antibiotic therapy), 3 grew *Staphylococcus aureus* alone (1 patient had a foot sinus, 1 had probable infected eczema as a cause of cellulitis, 1 had longstanding ulceration and several previous episodes of non-staphylococcal cellulitis), 4 grew streptococci alone (all group G), and 4 were mixed cultures of streptococci with *S. aureus* (2), *Proteus* spp (1) and coliforms (1). One swab from a leg ulcer grew cultured *Pseudomonas aeruginosa*, and one result was not recorded.

Blood for antistreptolysin O (ASO) titre was sent in 6 cases before (2) or after (4) advice from dermatologists. One result was not recorded, 2 were raised, and 3 (all sent within 9 days of onset) were normal. One of the patients with a raised ASO had a negative result when a further sample was sent 3 days after a second episode of cellulitis. One additional patient had ASO measured after relapse of cellulitis three weeks after discharge, at which stage the titre was four times the upper limit of normal.

Intravenous antibiotics were used in 52/92 patients initially, and a further 6 patients who started on oral antibiotics required intravenous therapy subsequently. Flucloxacillin alone or in combination was the most frequently used antibiotic (Table 2), whilst benzylpenicillin was used in only 43. The duration of antibiotic treatment recommended after discharge from hospital was nil to 6 months (mean 14 days, median 7 days). Only 11 patients

were advised to continue antibiotics for 4 weeks or more.

Morbidity

9 patients had experienced previous episodes of ascending cellulitis (1–3 occasions), if we include 2 probable cases diagnosed as deep vein thrombosis but with negative venograms. 2 of these probably had chronic lymphoedema (one dermatologically diagnosed) and one had chronic tinea pedis. Of 70 patients in whom at least six months had elapsed since the index episode, 4 had experienced rapid relapse requiring further antibiotic treatment in hospital (at least one of whom had chronic tinea pedis). A further 6 had relapses within three years, including 3 with leg ulcers and one with tinea pedis. Thus, at least 19 patients had either preceding or subsequent episodes of cellulitis within a short period. Additionally, at least 6 had chronic oedema after the episode, and 2 elderly patients developed persistent leg ulceration.

DISCUSSION

Our audit demonstrates that patients with cellulitis of the leg severe enough to warrant hospital admission have substantial short-term morbidity, evident from a mean admission duration of 10 days and the recorded frequency of pyrexia and malaise. This is undoubtedly due in part to the co-morbid disorders in the age-group mainly affected, but severity of symptoms was not related to age. An important point is that 31 were specifically noted to be afebrile at the time of admission, after oral antibiotics, but still had severe local symptoms or sufficient disability to require inpatient treatment. This dichotomy between resolution of pyrexia and continuing inflammation may be due to one or more of a persistent immunological reaction to infection, damage to lymphatics, or the ability of streptococci to produce exotoxins and superantigens^{3,9–11}.

The role of streptococci in our cases is in accord with other reports, but this organism tends to be neglected partly because of the difficulty in obtaining cultures from intact skin. Perhaps for this reason, *S. aureus* seems to account for a greater proportion of positive cultures^{7,8} than the proportion of cases in which it is perceived to be the major pathogen. Group G and other non-group-A streptococci were the most prominent organisms in our series; the importance of group G infections and their comparable severity to group A infections has been documented in other reports^{12,13}. The ASO titre can be a useful investigation to confirm the diagnosis in retrospect, especially if long-term prophylactic penicillin is a consideration, but may be negative during the first 10 days

Table 1 Portals of entry and preceding causes for cellulitis

Portal/cause	Number
Local injury (most minor, some non-penetrating)	13
Tinea pedis/toeweb maceration	12
Eczema of foot/leg	8
Diabetic foot/peripheral vascular disease	6
Leg ulceration	5
Severe lymphoedema	2
Blisters of foot	2
Others (chilblain, haematoma, chronic sinus)	3

Table 2 Initial antibiotic treatment

	Alone	With flucloxacillin	With benzylpenicillin	With erythromycin	With ampicillin/amoxycillin	With metronidazole	With gentamicin
Flucloxacillin	14	xxxxxx	31	2	8	1	0
Benzylpenicillin	7	(31)	xxxxxx	3	0	1	1
Erythromycin	6	(2)	(3)	xxxxxx	0	0	0
Cephalosporin	7	1	0	0	0	0	0
Augmentin	4	0	0	0	0	0	0
Ampi/amoxycillin	2	(8)	0	0	xxxxxx	1	0
Ciprofloxacin	1	0	0	0	0	0	0
Metronidazole	1	(1)	0	0	(1)	xxxxxx	0
Fusidic acid	1	0	0	0	0	0	0

Figures in parentheses appear in a previous line of the table

after infection. A rise in antibody may be inhibited by prompt penicillin therapy¹⁴ and the test was seldom requested by clinicians in the present study. The very varied choices of antibiotic as initial therapy probably reflects the experiences of different specialties, but demonstrates clearly that the importance of streptococci is underestimated. Whilst we accept that numerous organisms may cause cellulitis, and that there are differences which may relate to body site, to pre-existing injury or soft-tissue defects (such as leg ulcers), and to background factors (such as diabetes), we agree with Bisno and Stevens² that penicillin should be included in any regimen in the absence of contraindications, at least until bacteriology results are available.

The high frequency of recurrent episodes, relapses soon after treatment, and long-term morbidity of this disorder is not well recognized. Lymphatic obstruction and lymphoedema are well documented predisposing factors for recurrent cellulitis of the leg^{2,3,11,15,16}. Venous disease and leg ulcers similarly act as risk factors. Tinea pedis, although seldom diagnosed in hospital departments other than dermatology, is a well documented and usually curable cause of recurrent leg cellulitis^{11,17,18}. Surprisingly, prophylaxis against recurrent episodes in sporadic cases of lower leg cellulitis has received scant formal study^{19,20}; we frequently use long-term or even lifelong low-dose penicillin in such cases with good results. We accept that our results are related only to hospital inpatients and therefore overestimate the overall severity of the condition; however, we did not specifically trace patients or their primary care records, and our data regarding the long-term morbidity are therefore incomplete. In particular, the number of cases identified with oedema or leg ulceration more than six months after the cellulitis is an underestimate since it relates only to the patients with review in hospital; this important measure of the potential long-term morbidity of the disorder is now being studied in more detail. Overall, almost a third of patients had either recurrent episodes or other continuing morbidity.

Our results underline the importance of streptococci, lymphoedema, and tinea pedis in the aetiology of leg cellulitis. Whilst it is important to consider staphylococci and other organisms, especially in cases with preceding wounds, severe cellulitis, or other medical disorders, treatment must include an anti-streptococcal agent. Recurrence is frequent and may be an indication for more prolonged penicillin treatment; whether extension of the treatment period in all patients would prevent relapses remains to be tested. Treatment of tinea pedis, and support hosiery to reduce chronic oedema, are often ignored aspects of treatment that can reduce the likelihood of recurrent episodes and long-term morbidity.

REFERENCES

- 1 Leppard BJ, Seal DV, Colman G, Hallas G. The value of bacteriology and serology in the diagnosis of cellulitis and erysipelas. *Br J Dermatol* 1985;112:559-67
- 2 Bisno AL, Stevens DL. Streptococcal infections of skin and soft tissues. *N Engl J Med* 1996;334:240-5
- 3 Sachs MK. Cutaneous cellulitis. *Arch Dermatol* 1991;127:493-6
- 4 Bernard P, Bedane C, Mounier M, et al. Streptococcal cause of erysipelas and cellulitis in adults. A microbiologic study using a direct immunofluorescence technique. *Arch Dermatol* 1989;125:779-82
- 5 Hook EW, Hooton TM, Horton CA, et al. Microbiologic evaluation of cutaneous cellulitis in adults. *Arch Intern Med* 1986;146:295-7
- 6 Newell PM, Norden CW. Value of needle aspiration in bacteriologic diagnosis of cellulitis in adults. *J Clin Microbiol* 1988;26:401-4
- 7 Lebre C, Girard-Pipau F, Roujeau J-C, Revuz J. Value of fine-needle aspiration in infectious cellulitis. *Arch Dermatol* 1996;132:842-3
- 8 Epperley TD. The value of needle aspiration in the management of cellulitis. *J Fam Pract* 1986;23:337-40
- 9 Duvanel T, Auckenthaler R, Rohner P, et al. Quantitative cultures of biopsy specimens from cutaneous cellulitis. *Arch Intern Med* 1989;149:293-6
- 10 Leung DY, Travers JB, Norris DA. The role of superantigens in skin disease. *J Invest Dermatol* 1996;105(suppl 1):37S-42S
- 11 Pierce RP, Daugird AJ. Recurrent leg cellulitis: pathogenesis, prevention and treatment. *J Am Board Fam Pract* 1992;5:85-7
- 12 Nohlgard C, Bjorklind A, Hammar H. Group G streptococcal infections on a dermatological ward. *Acta Dermatovenereol* 1992;72:128-30
- 13 Baddour LM, Bisno AL. Non-group A beta-hemolytic streptococcal cellulitis: association with venous and lymphatic compromise. *Am J Med* 1985;79:155-9
- 14 Anderson HC, Kunkel HG, McCarty M. Quantitative antistreptokinase studies in patients infected with group A hemolytic streptococci: a comparison with antistreptolysin and gamma globulin levels with special reference to the occurrence of rheumatic fever. *J Clin Invest* 1948;27:425
- 15 Drinker CK, Field ME, Ward HK, et al. Increased susceptibility to local infection following blockage of lymphatic drainage. *Am J Physiol* 1983;112:74-81
- 16 Edwards EA. Recurrent febrile episodes and lymphoedema. *JAMA* 1963;184:858-62
- 17 Traub EF, Tolmach JA. An erysipelas-like eruption complicating dermatophytosis. *JAMA* 1937;108:2187-9
- 18 Brodell JD, Brodell RT. Recurrent lymphangitic cellulitis syndrome. *Contemp Orthop* 1992;25:461-8
- 19 Binnick AN, Klein RB, Baughman RD. Recurrent erysipelas caused by group B streptococcus organisms. *Arch Dermatol* 1980;116:798-9
- 20 Pauszek ME. Prophylaxis for recurrent cellulitis complicating venous and lymphatic insufficiency. *Indiana Med* 1991;84:252-3