Streptococcal necrotizing fasciitis in Gloucestershire: 1994

That Gloucestershire’s outbreak of necrotizing fasciitis generated such media excitement at the time probably owed more to the copywriting talent of the journalist, who dubbed its supposed aetiological agent the ‘flesh-eating killer virus’, than to any real threat to public health. Even so, it was the first cluster of necrotizing fasciitis cases in the UK this century, and the public’s concern may not have been entirely misplaced.

Confusingly for us, the onset coincided with the local adoption of mesh repair for inguinal hernia; the first patient, a 66-year-old man, fell ill the day after Lichtenstein herniorrhaphy. The infection was assumed to have been polymicrobial and no general alert was sounded. When, 3 days later, a 64-year-old woman suffered the same postoperative complication after saphenofemoral disconnection carried out by a different surgeon, but in the same community hospital operating theatre, the alarm was raised. The theatre was closed, the patients on the involved and intervening lists were contacted, and the theatre staff were swabbed. Tissues from the second patient yielded a pure growth of Lancefield group A β-haemolytic Streptococcus (β-HS). When Gram stains of the first patient’s tissue revealed positive chained cocci (and his antistreptolysin titre rose later) and a Streptococcus of the same subtype was cultured from the throat and nares of a member of the theatre staff present (though not scrubbed) at both procedures, the sequence of events seemed explained.

However, four further cases of group A streptococcal necrotizing fasciitis followed in quick succession, but all arose spontaneously in patients in the community. Two patients lived near Stroud, as did the postoperative cases, raising the possibility of a community-wide outbreak and, not surprisingly, the media’s interest was excited. However, later study showed that four different subtypes of group A β-HS were involved, arguing against a single virulent bacterial clone.

What other explanations could be advanced? Given the dramatically serious nature of the condition, ascertainment bias is unlikely. Could a vagrant virus have infected, and by DNA manipulation, envenomed the resident streptococcal population, as the ‘killer virus’ epithet suggested? If so, the ‘outbreak’ would surely have been greater. Indeed, why should it have stopped? The answer is almost certainly more humdrum. Examples of necrotizing fasciitis, both that complicating clean surgery and that occurring spontaneously, are well documented; the present cluster is, therefore, most likely to have arisen by chance.

What have we learned? The insidious onset and devastating nature of streptococcal necrotizing fasciitis has been rediscovered. One of the postoperative patients exhibited no early signs or symptoms other than hypotension and profuse diarrhoea. Both operated patients survived but two of the four community-acquired infections were fatal. All four survivors required extensive debridement and prolonged intensive system support.

Although Meleney in 1924 thought haemolytic Streptococcus was responsible for necrotizing fasciitis, finding it in all of 17 cases, his work preceded that of Lancefield and no facilities were available for culturing fastidious organisms. Giuliano and colleagues in 1977, using good aerobic and anaerobic culture techniques, found group A β-HS in three of 16 cases and as a single pathogen in one only. They deduced that polymicrobial synergism was mainly responsible (they isolated 75 different bacterial species). However, we have been forcibly reminded that group A β-HS can, indeed, do it alone and, furthermore, communally.

Nichols and Smith have recently suggested that necrotizing infections occurring within 48 h of surgery are more likely to be due to a single organism, whereas later cases, particularly those arising after bowel surgery or complicating perineal sepsis, are polymicrobial. Thus, until proved otherwise, it may be reasonable to presume that necrotizing fasciitis complicating ‘clean’ surgery is due to pure group A β-HS and to take appropriate action. The theatre should be closed and involved staff suspended from duties until the case is investigated. If group A β-HS is found to be responsible, bacterial clearance of both theatre and staff is required before they
are put back to work. Carriers of group A β-HS of the same subtype must have the organism eliminated before re-entering the theatre environment. However, necrotizing fasciitis of endogenous origin (perianal, postanal, and in the immunologically compromised) confers no risk of cross-infection.

About 10 per cent of the population are throat carriers of group A β-HS; far fewer harbour it in the nose. Surprisingly, when outbreaks of streptococcal infection have been investigated, perineal carriers have been implicated more frequently than nasopharyngeal ones. Therefore, screening for streptococcal carriage after outbreaks should include anal and vaginal swabbing. Routine screening of theatre staff is impractical as neither the frequency of false-negative swabs nor the incidence of streptococcal acquisition is known.

Dissemination of nasopharyngeal streptococci is more likely after respiratory infections, most of which will be viral. The staff member implicated in the authors' operating theatre outbreak had been off work with a cough and a cold, and had just returned without symptoms after a course of antibiotics. Should all personnel with sore throats or respiratory infections be excluded from theatre until confirmed free from Streptococcus? This would have been the only way of avoiding the authors' first two cases, but such a policy is impractical.

Necrotizing fasciitis travels fast and hours matter. Had either of the surgical victims gone home on the day of operation, perhaps neither would have sought medical advice until too late. Should all patients having day-case surgery be informed of the rare possibility of such sepsis and the general, rather than local, effects? The authors have, with misgivings, incorporated this warning in their information leaflets.

The international epidemiology of group A streptococcal infections remains as baffling as the Gloucestershire cluster. Norway, Sweden and the USA have all recognized significant rises in the incidence of invasive streptococcal infections recently, including necrotizing fasciitis. Other countries, including the UK and France, have recorded no change in disease rates. Time will tell if the Scandinavian and American experience is to be mirrored in other nations.

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