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Case Cluster of Necrotizing Fasciitis and Cellulitis Associated with Vein Sclerotherapy

To the Editor: Varicose vein sclerotherapy is a commonly performed cosmetic surgical procedure in which a sclerosing agent is injected into small varicose veins of the leg by using small gauge needles. It is regarded as a minor, safe procedure, usually performed in an office clinic (*1*). We describe a cluster of infections with group A *Streptococcus* spp. associated with throat carriage in a cosmetic surgeon.

In early December 2006, 3 patients were seen over a 10-day period at Geelong Hospital with infections following varicose vein sclerotherapy. All patients had undergone varicose vein sclerotherapy with polidocanol (Laurath-9; Aethoxysklerol, BSN Medical, Mount Waverley, Victoria, Australia) at a clinic of a single cosmetic surgeon. The index patient (patient A) had toxic shock syndrome and necrotizing fasciitis of the treated legs. The 2 other patients (patients C and D) had multifocal cellulitis directly correlating to the injection sites. The time between sclerotherapy and disease onset was 1–2 days.

A case-patient was defined as a patient who had undergone sclerotherapy at the clinic and subsequently had infection directly related to the site of sclerosant injection. Events were dated from the day on which the index patient had her surgical procedure. We reviewed clinic notes and infection control procedures in conjunction with the Department of Human Services of the State Government of Victoria, Australia. Specimens, where available, were collected for culture from patients by the treating clinicians. A throat swab was taken from the cosmetic surgeon. Specimens were transported and cultured by using standard methods.

During the outbreak period, 44 patients had vein sclerotherapy with 3% polidocanol at the cosmetic surgeon's clinic. In addition to the 3 patients identified on admission to hospital, a fourth patient (patient B) sought treatment from her general practitioner for medical care for a postprocedure infection. All patients had procedures on day 1 or day 7 (Figure); patients A and B were seen consecutively on day 1, and 2 patients were treated between patients C and D on day 7.

Patient A required surgical debridement, intravenous antimicrobial drugs, intensive care, and hyperbaric oxygen therapy. Intraoperative specimens taken from her during debridement cultured group A *Streptococcus* spp. Patients B, C, and D had cellulitis, but no specimens suitable for microbiologic diagnosis of cellulitis were taken for culture. Patient B was treated with oral antimicrobial agents as an outpatient. Patient C was admitted to hospital for intravenous antimicrobial therapy, and patient D showed no improvement on oral antimicrobial therapy as an outpatient and was subsequently admitted to hospital for intravenous antimicrobial agents.

Group A *Streptococcus* spp. was isolated from a throat swab taken on day 16 from the cosmetic surgeon. He reported no upper respiratory tract infection symptoms before the outbreak. He also reported that antiseptic skin preparation was not routinely used during the procedures; nor were gloves used. However, alcohol hand rubs were used between patients. The surgeon had not changed his infection control procedures recently and had not been aware of any infective complications previously. Environmental surface swabs taken on day 14 from 3 different areas (procedural trolley, surgical spotlight, and examination couch) in the clinic during the assessment yielded no pathogenic organisms. The infection control assessment team noted overall cleaning, disinfection, and hand hygiene to be inadequate.

Decolonization of the surgeon was performed by using rifampin 600 mg daily and amoxicillin 500

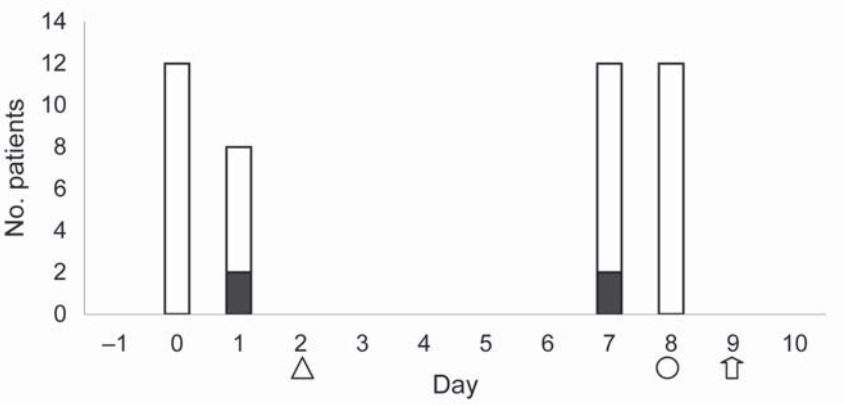

Figure. Days of procedures for infected and noninfected patients and their first manifestations of infection. □, uninfected; ■, infected; Δ, patients A and B seen with infection; O, patient C seen with infection; and ⇧, patient D seen with infection.

mg. every 6 hours for 10 days, during which time the surgeon suspended surgical procedures. Recommendations were made regarding infection prevention practices; these were undertaken by the surgeon.

Although soft tissue infection following sclerotherapy may be underreported, large case series have not noted this complication in the past (*2*,*3*); this finding suggests that any soft tissue infection following sclerotherapy should be investigated. These cases highlight the need for vigilance when considering infection control for minor procedures that take place outside of the support of hospital-based infection control services.

Soft tissue infections as complications following varicose vein sclerotherapy appear to be rare (*1*–*3*). The Australian Aethoxysklerol study reported no cellulitis in 16,804 legs injected with the sclerosing agent, and superficial thrombophlebitis occurred at a rate of 0.08% at 2-year review (*2*). Likewise, a multicenter registry with 22 European phlebology clinics reported no cellulitis or necrotizing fasciitis in 12,173 sessions (*3*).

Similarly, surgical site infections with Group A *Streptococcus* spp. are uncommon. A multicenter survey of 72 centers worldwide reported all β-hemolytic *Streptococcus* spp. (including group A and group G) accounted for <5% of infections (*4*), while surveillance in the 1990s by Centers for Disease Control and Prevention reported <1% of all surgical wound infections was caused by group A *Streptococcus* spp. (*5*). A Canadian study reported invasive group A *Streptococcus* infections following surgery in 1.1 cases per 100,000 admissions (*6*). Outbreaks have been infrequently described (*5*,*7*–*10*), and sources of colonization range from throat to anus and vagina.

Acknowledgments

We thank Rosemary Lester and Michelle Cullen for their input.

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Streptococcus suis in Humans, Thailand

To the Editor: *Streptococcus suis* is an important zoonotic pathogen for swine and humans. Among 33 serotypes, serotype 2 is more frequently isolated from diseased pigs than other serotypes (*1*). However, not all serotype 2 strains are virulent, and degree of virulence varies among strains (*2*). Previous studies have reported several *S. suis* putative virulence factors, including the polysaccharide capsule, the muramidase-released protein, the extracellular factor, and suilysin (*3*–*5*). Some of these factors have been used as virulence-associated markers, and the association of the factors of *S. suis* isolates with virulence or clinical background has been suggested in Europe (*2*,*5*). However, because many virulent isolates lacking these factors have also been isolated from clinical cases in Canada (*6*), they cannot be used as virulence markers in North America.

Recent analysis of *S. suis* isolates by multilocus sequence typing (MLST) suggested the association of some clonal groups with particular clinical manifestations. That is, most invasive isolates belonged to the sequence type (ST) 1 complex, while the ST27 and ST87 complexes were found to include a higher proportion of lung isolates (*7*). Although *S. suis* has been prevalent worldwide, the geographic location of the isolates used so far was mainly Europe, North America, and East Asia (*7*–*9*). Moreover, the clonal association with virulence of *S. suis* has been discussed mainly on the basis of clinical and experimental data in swine (*7*). In this report, to broaden understanding of the population structure of *S. suis* as a zoonotic agent, we characterize 20 *S. suis* isolates (Table) recovered from humans in Thailand in 1998–2002.

Serotyping by coagglutination tests showed that 19 of the 20 isolates