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.

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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

belonged to serotype 2, while the remaining 1 (MNCM07) was serotype 14. MLST analysis resolved the 20 isolates into 8 STs (Table). By using eBURST (http://eburst.mlst.net), we assigned 4 isolates (MNCM01, MNCM06, MNCM07, and MNCM16) from 1 case of endocarditis and 3 cases of meningitis to the ST1 complex. The remaining isolates were assigned to the ST27 complex with a less-stringent group definition (Table), although ST101 (MNCM21) and ST104 (MNCM50) shared only 2 alleles with ST27 and were incorporated into this complex by a chaining effect. Regarding the clinical cases from which the ST27 complex isolates were recovered, the patients had meningitis, endocarditis, septicemia, septic shock, diarrhea, and respiratory involvement. The 2 ST complexes both contained isolates from deceased patients (Table).

All the isolates assigned to the ST1 complex were positive for the suilysin gene *sly*, the extracellular factor gene *epf* or its variant, and the muramidase-released protein gene *mrp* or its variant. With the exception of MNCM21 and MNCM50, which had only *sly*, all isolates classified into the ST27 complex were negative for *sly* and *epf* but positive for *mrp* or its variant. These results showed the congruence between STs and the virulence-associated gene profiles and further support the usefulness of MLST for epidemiologic studies of *S. suis*.

Of the 3 major clonal complexes identified so far in *S. suis* (ST1, ST27, and ST87), the ST1 complex particularly attracts considerable public attention as a clonal group that may have the potential for a higher degree of virulence than the others (*7*), and most (96%) of the human isolates investigated so far, including ST7 isolates, which caused the largest outbreak in China, belong to the ST1 complex (*7–9*). In this study, although no ST7 isolate was found, 4 isolates were assigned to the ST1 complex. This further confirmed the gravity of the ST1 complex not only for swine industries but also for public health.

In contrast to the ST1 complex, only 4 human clinical isolates have so far been reported to belong to the ST27 complex. Three of the 4 are isolates from Canada that belong to ST25

Table. Epidemiologic data of *Streptococcus suis* isolates from patients in Thailand, 1998–2002*

Isolate no.†	Year of isolation	Site of isolation	Virulence-associated genes‡§	Serotype	Diseases and symptoms	ST (ST complex)
MNCM01	2000	Blood	*cps2J*+/*sly*+/*epf*+/*mrp*+	2¶	Endocarditis	1 (1)
MNCM06	2000	Blood, CSF	*cps2J*+/*sly*+/*epf*+/*mrp*+	2	Neck stiffness, deafness (meningitis)	1 (1)
MNCM16	2000	CSF	*cps2J*+/*sly*+/*epf*+/*mrp*+	2	Neck stiffness (meningitis)	1 (1)
MNCM07	2000	Blood, CSF	*cps1J*+/*sly*+/*epf**/*mrp*^{S}+	14	Neck stiffness (meningitis), death	11 (1)
MNCM04	2000	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Neck stiffness, deafness (meningitis)	25 (27)#
MNCM10	2000	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septicemia	25 (27)#
MNCM24	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2¶	Endocarditis	25 (27)#
MNCM26	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Endocarditis, deafness (meningitis)	25 (27)#
MNCM51	2002	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septicemia, diarrhea, death	25 (27)#
MNCM55	2002	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septic shock, death	25 (27)#
LPH4	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septicemia, diarrhea	25 (27)#
LPH12	2002	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septic shock, death	25 (27)#
MNCM43	2002	Blood	*cps2J*+/*sly*–/*epf*–/*mrp*+	2	Endocarditis	28 (27)
MNCM21	1998	CSF	*cps2J*+/*sly*+/*epf*–/*mrp*–	2	Meningitis	101 (27)#
MNCM25	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Neck stiffness (meningitis), diarrhea, death	102 (27)#
MNCM54	2002	Blood	*cps2J*+/*sly*–/*epf*–/–*mrp***+	2	Neck stiffness (meningitis), diarrhea	102 (27)#
MNCM33	2002	Blood, CSF	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Neck stiffness (meningitis)	103 (27)#
LPH3	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Meningitis	103 (27)#
LPH5	2001	Blood	*cps2J*+/*sly*–/*epf*–/*mrp***+	2	Septicemia	103 (27)#
MNCM50	2002	Blood	*cps2J*+/*sly*+/*epf*–/*mrp*–	2	Pulmonary edema, death	104 (27)#

*ST, sequence type; CSF, cerebrospinal fluid.
†Isolates with MNCM number and LPH number were isolated from patients at Maharaj Nakorn Chiang Mai Hospital and Lamphun Hospital, Thailand, respectively.
‡Virulence-associated gene profiling was done as described previously (*10*). *cps1J* and *cps2J*, serotype 1 (and 14) and 2 (and 1/2) specific genes, respectively, involved in the capsular biosynthesis; *sly*, suilysin gene; *epf*, extracellular factor gene; *mrp*, muraminidase-released protein gene; +, positive; –, negative.
§*epf**, an *epf* variant that produces an ≈3,000-bp fragment by PCR with primers described previously (*10*); *mrp*** and *mrpS*, *mrp* variants that produce ≈1,800-bp and ≈750-bp fragments, respectively, by PCR with primers described previously (*10*).
¶Coagglutination reaction using anti-serotype 2 serum was weak.
#ST25, ST101, ST102, ST103, and ST104 belong to the ST27 complex, only with a less-stringent approach that defines an ST complex by sharing of alleles at ≥5 of the 7 loci.

(*7*). The remaining 1 is from Japan and assigned to ST28 (*8*). Unlike in previous reports, 80% of the human clinical isolates (16 isolates) characterized in this study were assigned to the ST27 complex. Although previous studies suggested that members of the ST27 complex may have lower potential to cause invasive diseases in swine (*7*), all the isolates were isolated from blood or cerebrospinal fluid of the patients, suggesting a high degree of invasiveness (Table). Because it is unknown whether the ST27 complex is also dominant among isolates from diseased pigs in Thailand, future surveillance will be necessary to know the situation in pigs. However, our data indicate that the ST27 complex is another clonal group that should be assessed for its importance for human infection. Because *mrp*, *epf*, and *sly* are not appropriate as virulence markers for the ST27 complex members, development of novel virulence markers will be needed for efficient discrimination of *S. suis* strains virulent for humans.

This study made use of the *Streptococcus suis* Multilocus Sequence Typing website (http://ssuis.mlst.net); this site is hosted at Imperial College and development is funded by the Wellcome Trust. The study was supported by a grant-in-aid from the Zoonoses Control Project of the Ministry of Agriculture, Forestry and Fisheries of Japan and the Endowment Fund for Medical Research, Faculty of Medicine, Chiang Mai University.

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Streptococcus suis Meningitis, United States

To the Editor: *Streptococcus suis,* commensal and opportunistic pathogens of swine, and prevalent zoonotic agents worldwide, are α-hemolytic gram-positive cocci with 35 different serotypes (*1*). In humans, *S. suis* infection has been associated with bacterial meningitis, septic shock, arthritis, pneumonia, endocarditis, endophthalmitis, and spontaneous bacterial peritonitis (*2*,*3*). Most at risk are those who handle or eat undercooked pork, e.g., farm workers, butchers, and slaughterhouse workers (*4*). Most cases have been reported in Europe or Southeast Asia (*2*,*3*). Meningitis, first recognized in 1968 in Denmark (*1*), is the most common clinical manifestation of human infection with *S. suis*. A case of *S. suis* meningitis in a pig farmer was reported in the United States (*5*). Here, we describe another case in a 60-year-old man from San Francisco who had consumed raw pork while traveling in the Philippines.

In June 2003, this man became ill with fever, diaphoresis, headache, nausea, and anorexia. He had just returned from a 7-month vacation in the Philippines. Three days after symptoms onset, his physician prescribed doxycycline. Symptoms continued and he was admitted to a local hospital 5 days later with a fever of 38.9°C, nuchal rigidity, headache, and general malaise.

The patient described no recent contact with sick persons; past medical