An Australian case of *Streptococcus suis* toxic shock syndrome associated with occupational exposure to animal carcasses

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Streptococcus suis is known to cause sporadic infections in people who have occupational exposure to pigs and pig meat. A large outbreak occurred in China in 2005, where there was 62% mortality among those who developed toxic shock syndrome. Despite S. suis being common in pigs, this is the first published report of a human case of S. suis toxic shock syndrome in Australia. (MJA 2008; 188: 538-539)

Clinical record

In April 2007, a 41-year-old man developed sudden-onset lower abdominal pain and rigors. Fevers, headache, diarrhoea, vomiting and dizziness developed and continued throughout the day. In the evening, he presented to the emergency department of a suburban non-teaching hospital in Melbourne. He had no pre-existing illness and had worked for 5 months as a pet-food processor, handling carcasses of sheep, cattle and pigs.

He had a temperature of 38.8°C and his blood pressure was 84/40 mmHg. Investigations revealed acute renal failure and slightly raised serum bilirubin levels (Box). He was presumed to have infective gastroenteritis and was given intravenous fluid replacement.

Septic shock was suspected when the patient's blood pressure failed to normalise after intravenous administration of 6 L physiological saline over 12 hours and he developed neutrophilia and disseminated intravascular coagulation (Box). The presence of multiple cuts on his hands and severe tooth decay led to suspicion of staphylococcal or streptococcal sepsis. He was given vancomycin and flucloxacillin and was transferred to the intensive care unit of a teaching hospital for inotropic support.

Subsequently, culture of seven of eight blood samples taken in the first 36 hours grew α -haemolytic streptococci (on horseblood agar). Given the patient's poor dentition, the antibiotic therapy was changed to high-dose benzylpenicillin with gentamicin to treat possible endocarditis.

API 20 Strep identification system (bioMérieux, Lyon, France) gave the profile 4641473, corresponding to *Streptococcus suis* II (99.9%; bioscore, 0.99). Phenotypically, the isolate was very similar to *Streptococcus parasanguinis*, a human viridans streptococcus that is not in the API 20 Strep database. Our isolate was confirmed as *S. suis* by 16S rRNA (ribosomal RNA) sequencing. A 519-base-pair fragment of the 16S rRNA gene was amplified using universal primers (unpublished sequences). The primerbinding positions corresponded to positions 21–42 and 565–583 of the *Escherichia coli* numbering system. A BLAST search on the GenBank database matched several *S. suis* sequences exactly (519/519 bases; 100%) and partially matched many other *S. suis* sequences, including type strains. The next closest match was *Streptococcus bovis* (≤ 94% sequence homology).

On Day 7, liver function tests gave abnormal results (Box). Results of a liver ultrasound examination and a transoesophageal echocardiogram were normal. The patient received intravenous benzylpenicillin and gentamicin for 2 weeks, followed by oral amoxycillin for 1 week. The patient made a full recovery, with liver function returning to normal; he subsequently changed employment.

Public health response

We notified the Victorian Government Department of Human Services, which planned to respond if additional cases of *S. suis* toxic shock syndrome occurred. We also referred the case to workplace occupational health and safety investigators because of a concern that an inadequate supply of gloves and other personal protective equipment had limited their use in the workplace. A worksite visit found no evidence to substantiate these concerns. Further, there were no concerns about plant and personal hygiene, washing facilities and staff training in the most recent quarterly audit report.

Haematological and biochemical features of a patient with *Streptococcus suis* toxic shock syndrome

	Day after onset				Reference
	0	1	7	33	range
Haematological analysis					
Haemoglobin (g/L)	124	113	115	142	130–170
White cell count (x 10 ⁹ /L)	4.4	24.6	21.1	10.4	4.0-11.0
Neutrophil count (x 10 ⁹ /L)	4.2	22.7*	13.5	5.5	2.0-8.0
Platelet count (× 10 ⁹ /L)	182	103	259	238	140-400
INR	_	2.0	1.0		0.8–1.3
APTT (s)	_	37	22		24–35
Fibrinogen (g/L)	_	2.8	2.8		2.0-5.0
Biochemical analysis					
Sodium (mmol/L)	140	141	138		135-145
Potassium (mmol/L)	3.2	3.6	4.8		3.5-5.5
Creatinine (µmol/L)	120	120	90		50-110
Urea (mmol/L)	7.9	6.9	4.7		2.5-8.3
Albumin (g/L)	42	28	34	41	35-50
Alkaline phosphatase (U/L)	90	72	157	98	< 120
γ -Glutamyltransferase (U/L)	_	37	113	58	< 50
Alanine aminotransferase (U/L)	17	29	86	21	< 55
Aspartate aminotransferase (U/L)	_	38	59	17	< 50
Bilirubin (µmol/L)	46	38	16	15	< 19
Total protein (g/L)	63	49	65	75	60-82
C-reactive protein (mg/L)	_	173	13	<2	< 8

INR = international normalised ratio. APTT = activated partial thromboplastin time. *Included band forms 18.0×10^9 /L. Bold indicates abnormal results.

We are not aware of any increase in *S. suis* disease in the Australian pig industry around the time our patient presented. In Victoria between 2002 and 2006, most streptococcal isolates submitted to the three main veterinary laboratories were not identified to species level. Therefore, although there were at least 33 confirmed *S. suis* isolates from pig specimens in Victoria during this period, the true burden is unknown (Brenda McCormack, Diagnostic Laboratory Manager, Pig Health Research Unit, Victorian Government Department of Primary Industries; Mary Dep, Scientist, Atwood Veterinary Diagnostic Services; and Dr Mark Williamson, Veterinary Pathologist, Gribbles Pathology, personal communications). Around the time our patient presented, Australia's National Animal Health Information System recorded only four cases of *S. suis*, in piglets in Queensland.²

Discussion

S. suis is a common pathogen in pigs, and in the developed world has been recognised as a cause of sporadic disease in humans who have occupational exposure to pigs and pig meat products.³ *S. suis* infection in humans recently received considerable attention following a large outbreak in China in 2005, where there were 215 cases, with 62% mortality among those who developed toxic shock syndrome.⁴

Sepsis originating from the skin is common in meat workers due to the frequent skin lacerations they sustain, with *Streptococcus pyogenes* and *Staphylococcus aureus* often isolated.⁵ Other causes of sepsis in those working with pig meat products include salmonella, campylobacter, yersinia, leptospira and brucella.⁶

In humans, *S. suis* may cause toxic shock syndrome, meningitis, arthritis, endocarditis and pneumonia.^{3,7} Toxic shock syndrome has a distinct presentation, as seen in our patient. The predominant features are fevers, hypotension, disseminated intravascular coagulation (often with subcutaneous haemorrhage), diarrhoea, vomiting, acute renal failure, abdominal pain and liver dysfunction.³ *S. suis* infection occurs after contact with infected pigs or pork via wounds or inhalation.^{3,7} However, symptomatic or severe infection after exposure appears to be uncommon. In Australia and other high-income countries, pigs are frequently colonised with *S. suis*, and the seroprevalence in pig farmers and meat workers may be as high as 21%.^{7,8}

Measures to prevent *S. suis* infection in meat workers include covering skin lesions; wearing gloves; avoiding eating, drinking and smoking in work areas; and frequent hand washing.⁶ Measures are often less stringent in domestic slaughter of pigs, which facilitated the 2005 Chinese outbreak.⁴ Prohibiting domestic slaughter of pigs and assisting farmers with hygienic handling of dead or sick pigs were the major interventions used to control the Chinese outbreak.⁴

To our knowledge, our patient is the first human case of *S. suis* toxic shock syndrome in Australia. Since submission of this manuscript there have been at least three unpublished recent human cases of *S. suis* infection in other parts of Australia, reported in February 2008 via Ozbug, an email discussion group of the Australasian Society for Infectious Diseases. Phenotypically, *S. suis* resembles the viridans streptococcal species *Streptococcus sanguinis*, *S. parasanguinis and Streptococcus gordonii*, and therefore may be misidentified. This case demonstrates the importance of correct identification to species level, which established the occupational source of infection and prevented it being attributed to poor dental

hygiene. This has implications for compensation, occupational health and safety, and public health.

In conclusion, it is important to consider *S. suis* infection in individuals with occupational exposure to pigs, as mortality may be high without timely treatment. It is also important to inform the microbiology laboratory of exposure to pigs in cases of streptococcal sepsis and meningitis to avoid misidentification.

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Competing interests

None identified.

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