Complicated infection caused by *Streptococcus suis* serotype 14 transmitted from a wild boar

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**Introduction:** *Streptococcus suis* is a zoonotic pathogen transmitted to humans from infected pigs. Nearly all human cases of *S. suis* are caused by serotype 2 organisms, and meningitis is the best-documented type of human infection. On rare occasions, *S. suis* can be transmitted to humans from wild boars.

**Case presentation:** Here we report a case where *S. suis* of serotype 14 was transmitted from a wild boar to a previously healthy 63-year-old man, causing meningitis, spondylodiscitis, a psoas abscess and a prolonged post-infectious inflammatory condition. The infection was treated with a long course of β-lactam antibiotics, but signs of inflammation were relieved only after the addition of corticosteroids. The isolate was found to harbour the virulence-associated gene sly.

**Conclusion:** *S. suis* of serotypes other than type 2 can be transmitted to humans from wild boars and the disease may become complicated. Increasing numbers of wild boars in some European countries calls for increased vigilance to this type of infection.

**Keywords:** meningitis; serotype; *Streptococcus suis*; wild boar; zoonosis.

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**Introduction**

*Streptococcus suis* is part of the normal flora of pigs, but it is also an important pathogen causing pneumonia, meningitis and endocarditis in these animals. *S. suis* can infect humans who come into contact with pigs or unprocessed pork meat, causing severe infections, most often meningitis (Wertheim et al., 2009). Invasive *S. suis* infections have also been described in hunters after butchering of wild boars (Bonmarchand et al., 1985; Halaby et al., 2000; Grebe et al., 1997; Rosenkranz et al., 2003; Wertheim et al., 2009). The vast majority of human *S. suis* infections are caused by serotype 2 isolates (Lun et al., 2007; Wertheim et al., 2009), including the known cases transmitted from wild boars (Grebe et al., 1997; Halaby et al., 2000; Rosenkranz et al., 2003). The few cases in which other serotypes have been isolated from human infections include a case infection with a serotype 1 isolate (Vilaichone et al., 2002), meningitis with a serotype 4 isolate (Arends & Zanen, 1988), spontaneous peritonitis and septic arthritis with serotype 5 isolates (Gustavsson & Rasmussen, 2014; Kerdzin et al., 2011), fatal bacteraemia with a serotype 16 isolate (Kerdzin et al., 2011; Rosenkranz et al., 2003), spontaneous peritonitis with a serotype 21 isolate (Callejo et al., 2014), sepsis with a serotype 24 isolate (Kerdzin et al., 2011) and cases of bacteremia, meningitis, septic arthritis and endocarditis caused by serotype 14 isolates (Gottschalk et al., 1989; Kerdzin et al., 2009; Poggenborg et al., 2008; Takeuchi et al., 2012; Watkins et al., 2001). Here, we report a case of meningitis complicated by spondylodiscitis and an abscess caused by *S. suis* serotype 14 appearing after slaughter of a wild boar.

**Case report**

The patient was a smoking, previously healthy 63-year-old man who had cut his finger during slaughtering of a wild boar 2 days before admission. One day before admission, he had experienced chills, general muscular pains and had slight diarrhoea. On admission, the patient was disoriented but afebrile, normotensive and without obvious stiffness of the neck. Laboratory investigations revealed an elevated C-reactive protein (CRP; 304 mg l⁻¹), leukocytosis (18 × 10⁹ l⁻¹), thrombocytopenia (68 × 10⁹ l⁻¹) and elevated serum creatinine (148 μM). A lumbar puncture was attempted but failed, and a computed tomography (CT) of the brain was unremarkable. On the suspicion of bacterial meningitis, treatment with betamethasone,cefotaxime and ampicillin was initiated. His general condition improved rapidly, but the patient reported neck pain. Magnetic resonance imaging (MRI) revealed signs of acute spondylodiscitis at the C3–C4 level (Fig. 1a). On day 2, the laboratory reported the presence of *S. suis* in all four blood culture flasks. The identification was based on matrix-assisted laser desorption/ionization time-of-flight mass...
spectrometry, which gave a score of >2.3 for S. suis. Treatment with ampicillin was continued, and a transthoracic echocardiography was performed without signs of any valvular vegetations. On day 5, the patient was transferred to the Malmö Department of Infectious Diseases at the University Hospital of Skåne, Sweden. A lumbar puncture was performed and analysis revealed $72 \times 10^6$ mononuclear cells $l^{-1}$, $1.3 \times 10^6$ polymonuclear cells $l^{-1}$ and slightly elevated levels of lactate and protein. Cultures of cerebrospinal fluid were negative, but 16S rRNA gene PCR and sequencing revealed the presence of DNA from S. suis. On day 7, a swelling of the right sternoclavicular joint and elevation of CRP from 52 to 208 mg l$^{-1}$ was noted. A renewed CT revealed a possible ileopsoas abscess; attempts to drain this abscess were unsuccessful. A small volume of fluid was aspirated from the sternoclavicular joint, but 16S rRNA gene PCR on this material was negative. Transoesophageal echocardiography (TEE) did not reveal any vegetations. On the eleventh day the treatment was shifted to piperacillin with tazobactam and CRP gradually decreased from 300 to around 100. The patient was sent home after 23 days of hospitalization and continued with amoxicillin 750 mg two times daily.

Six days after discharge, the patient experienced increased pain from his neck and he had become subfebrile. He was readmitted and ampicillin and tazobactam were reintroduced. The CRP was 127 mg l$^{-1}$ and erythrocyte sedimentation rate (ESR) was above 100 mm. Renewed investigations with MRI revealed signal changes in the lumbar region suggestive of spondylitis but no paravertebral abscess was seen (Fig. 1b). CT revealed that the suspected psoas abscess had grown to a cylindrical structure with a diameter of 2.5 cm and a length of 7.5 cm (Fig. 1c). The abscess was punctured, guided by ultrasound, and 10 ml viscous pus was evacuated. Cultures and 16S rRNA gene PCR on this material were both negative. The TEE was repeated but still did not show signs of endocarditis and CT angiography was performed to exclude aortitis. After 14 days of treatment with ampicillin followed by piperacillin-tazobactam, the CRP was still 76 and ESR above 120. At this point, a post-infectious inflammatory condition was suspected, and treatment with prednisolone 40 mg daily was commenced. The CRP and ESR responded swiftly, and the patient was sent home in a clinically improved condition. Treatment with amoxicillin 1.5 g three times daily was continued for 6 months and, during that time, prednisolone was given in de-escalating doses. A follow-up X-ray of the spine did not show signs of sclerosis of the vertebrae affected by the infection. Three months after the cessation of antibiotic and anti-inflammatory therapy, the patient had low inflammatory markers and was deemed to have fully recovered.

**Investigations**

The isolate of S. suis was determined to be serotype 14 by the Statens Serum Institute (Hillerød, Denmark) through agglutination with a latex kit and type-specific serum as well as by microscopic determination of capsule swelling according to Neufeld. The three methods gave concordant results. E-tests (BioMérieux) indicated that the isolate was sensitive to all antibiotics tested. The MIC for cefotaxim was 0.125 mg l$^{-1}$ and for ampicillin was 0.008 mg l$^{-1}$. The isolate was tested for the presence of the virulence-associated gene sly, as well as for mrp and epf, believed to be markers of virulence in serotype 2 isolates, by PCR as described by Kim et al. (2010). A PCR fragment of the predicted size was obtained with primers hybridizing to sly, but no fragment was amplified with primers hybridizing to mrp or epf. A serotype 2 isolate (kindly provided by Susanne Sauer, Statens Serum Institute) and a serotype 5 isolate (Gustavsson & Rasmussen, 2014) were used as positive controls for the epf and mrp primers, respectively.

**Discussion**

In Sweden, more than 70 000 wild boars are shot annually, and the number of animals is steadily increasing. As wild boars in northern Germany are colonized with potentially human pathogenic S. suis isolates (Baums et al., 2007), it is likely that similar isolates are found also among Swedish wild boars. The present case is, to the best of our knowledge, the first describing a non-serotype 2 isolate transmitted from a wild boar to a human and is the third case of S. suis infection reported from Sweden (Christensen & Kronvall 1995; Gustavsson & Rasmussen 2014). The course of the infection described here was complicated, and, despite adequate treatment with antibiotics, an abscess developed. As the infection became so widespread, an intravascular focus was suspected, but repeated TEE and investigation of the aorta failed to detect this. The prolonged course with a high inflammatory activity was suspected to be due to a post-infectious condition rather than a persisting infection. In line with this, the
inflammation declined rapidly with corticosteroid treat-
mant without recurrence of infection. S. suis serotype 14
has been described in at least three cases of fatal infections
(Gottschalk et al., 1989; Takeuchi et al., 2012; Watkins
et al., 2001). Thus, it seems that S. suis of this serotype has
a high pathogenic potential. Nothing is known about the
presence of the putatively virulent S. suis serotype 14 in
wild boars, but the increasing numbers of wild boars in
Sweden and the present case of severe infection calls for
increased vigilance. Protective gloves should be worn when
slaughtering wild boars and note should be taken regarding
infectious symptoms if traumatic cuts are inflicted. Early
antibiotic treatment should be initiated if symptoms occur.

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