Streptococcus sinensis causing infective endocarditis in the Netherlands: our experiences from the UK

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This editorial refers to 'Fatal outcome of first case of Streptococcus sinensis in infective endocarditis in the Netherlands: a case report', by A.M.L.N. van Ommen et al. doi:10.1093/ehjcr/ytz237.

We read with interest the recent publication by van Ommen et al.¹ of the first case of *Streptococcus sinensis* causing infective endocarditis (IE) in the Netherlands.

The article does not describe the patient's recent travel history. In cases of *S. sinensis*, the travel history is important in determining the possible geographical reservoirs for this agent. Cases have largely been reported in South East Asia and specifically Hong Kong, ^{2,3} although more recently cases are being described in Europe. ^{4–6}

We published a case of *S. sinensis* in the UK in a Caucasian male who was a frequent traveller to Vietnam but had undergone dental work in Hong Kong. We hypothesized that the patient's oral cavity, a natural reservoir for the bacterium, became colonized with *S. sinensis* during his frequent visits to the region and that he became bacteraemic at the time of his dental work.

van Ommen et al. report the first known mortality of IE postulated to be the result of the acute phase of *S. sinensis*. The report states the patient had symptoms for 6 months, suggesting a period of chronicity in keeping with the insidious onset of IE caused by *S. sinensis*. The patient died from acute left hemisphere cerebral haemorrhage. Given recent computed tomography scan demonstrating a right hemisphere infarction and the late presentation, the cerebral haemorrhage is likely to have been secondary to a mycotic aneurysm rather than haemorrhagic transformation of a new cerebral infarction.

The introduction of MALDI-TOF mass spectrometry into the diagnostic microbiology laboratory in the last few years has dramatically improved the accurate identification of bacteria, including viridans streptococci such as *S. sinensis.*⁸ Previously this would have required research techniques such as 16SrRNA typing to speciate bacteria.⁹

The actual number of cases of *S. sinensis* causing infections may therefore have been underestimated in the past due to misidentification by commercial kits.

Viridans streptococci, such as *S. sinensis*, are part of the normal human oral bacterial flora and as such are well known causative agents of IE. The mechanism of pathogenesis is partly via their ability to adhere to heart valves via extracellular dextran production. The hypothesis regarding the potential for a human genomemediated susceptibility to *S. sinensis* is solely theoretical. Basic research into micro-organism integration into the human genome is ongoing and currently, there are no studies looking at *S. sinensis* that have identified individuals with a genotype for susceptibility to this infection. Higher bacterial mutational rates lend themselves to increased virulence which has been widely reported in antibiotic resistance. ¹⁰ It is more likely the bacterial genome has adapted to result in increased pathogenicity rather than an acquired human genome susceptibility.

This, in conjunction with our case, would be the second presentation of *S. sinensis* causing IE in a patient without known pre-existing valvular disease. Nevertheless, *S. sinensis* remains an uncommon pathogen for IE but should be suspected in the returning traveller from an endemic area.

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Lead author biography



Dr James Tomlinson MBChB, MRCP (UK), is a cardiology specialty registrar with an interest in infective endocarditis (IE) and echocardiography. He has collaborated with Dr Sally Curtis and Dr Rachael James, consultants in microbiology and cardiology respectively with expert interests in IE, to publish a rare case of Streptococcus sinensis causing IE in the UK.

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Consent: This is an Editorial Comment which refers to a post-mortem case report with written informed consent for publication.

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