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## Case report

# Severe infective endocarditis with systemic embolism due to community associated methicillin-resistant *Staphylococcus aureus* ST630



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## ABSTRACT

Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) are increasingly causing infective endocarditis over the past decade. Here we report a healthy man who developed a severe acute infective endocarditis with systemic embolism caused by CA-MRSA. The strain was recovered from repeated blood cultures and was characterized using molecular detection and genotyping. The *S. aureus* isolate was typed as ST630 SCCmecV with *spa*-type t4549, *agr*I/IV and was PVL-negative. This is the only case report, to our knowledge, of CA-MRSA infective endocarditis in China. This case highlights the emergence and geographical spread of life-threatening CA-MRSA infection within China.

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## Introduction

Since the first genuine community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection was reported in Australia in the early 1990s, the emergence of CA-MRSA infection has increased in recent years all over the world. CA-MRSA is primarily associated with healthy individual skin and soft tissue infections. Additionally, invasive diseases, including bacteremia, infective endocarditis (IE), osteomyelitis, and hemorrhagic necrotizing pneumonia,

also have been reported. Infective endocarditis can seriously damage heart valves and cause other serious complications, associated with significant morbidity and mortality, and it is lethal if not promptly treated with appropriate antibiotics, regardless of whether surgery is performed.<sup>1</sup> However, despite improved diagnostic techniques and advances in treatment options, neither its incidence nor mortality has decreased in the past years.<sup>2</sup> *S. aureus* is a leading cause of left-sided infective endocarditis in developing countries, nevertheless, only sporadic cases of endocarditis caused by CA-MRSA among healthy individuals have been reported.<sup>3</sup> Here we describe the

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first case of infective endocarditis complicated with systemic embolism due to a novel CA-MRSA ST630 in China, which was successfully treated with a combination of antimicrobials and surgical therapy.

## Case presentation

A 49-year-old previously healthy man was admitted to the hospital with a 10-day history of chills, fever, and dyspnea in August 2011. He had no history of surgery or intravenous drug use, and had no notable medical record. At the time of hospital admission, physical examination revealed a pulse rate of 62 beats per min, respiratory rate of 20 breaths per min, and blood pressure of 125/83 mmHg. A systolic murmur was best heard at the apex. He was febrile (40.7°C) and he had a minor abrasion on left foot. The rest of his physical examination was unremarkable. Pertinent laboratory investigations revealed white blood cell count of 23,100/mm<sup>3</sup>, with 94.5% neutrophils, 3.2% lymphocytes, and 2.3% mononuclear cells, platelets of 40,000/mm<sup>3</sup> and C-reactive protein of 58.6 mg/L. The patient was hospitalized for a presumptive diagnosis of septicemia. Empirical antibiotics were started with vancomycin and levofloxacin (0.5 g q8 h) therapy.

During the next two days, echocardiography revealed large vegetation on the anterior mitral valve leaflet (3 cm × 3 cm) with moderate mitral valve regurgitation (Fig. 1A). Spiral computed tomography (CT) showed renal and splenic infarction (Fig. 1B). CT scans of the brain demonstrated multiple low-density bilateral lesions of the temporal lobes, right parietal lobe and occipital lobe, suggestive of cerebral embolism. Preliminary blood cultures grew *S. aureus* susceptible to ciprofloxacin, rifampicin, linezolid, vancomycin, tetracycline, sulfamethoxazole, levofloxacin and fosfomycin but resistant to penicillin, oxacillin, clindamycin, ceftazidime, ceftiofur, cefuroxime, and erythromycin, as determined on the basis of CLSI disc diffusion standards. A diagnosis of acute infective endocarditis with systemic embolism caused by CA-MRSA was thus considered. Because of his impaired renal function and bacterial susceptibility profile, the patient was treated with intravenous linezolid (600 mg q12 h) and fosfomycin (8.0 g q12 h).

On day 10, his clinical status worsened with episodes of tachypnea, pink frothy sputum and oxygen saturation (SpO<sub>2</sub>) decreased rapidly to 83% with ventilator support. Furthermore, renal function deteriorated, oliguria and right lower extremity tissue necrosis appeared. Embolization of the right common iliac artery and right internal and external iliac arteries was seen on echocardiography (Fig. 1C). He subsequently developed a coma with a Glasgow Coma Scale (GCS) score of 6. His clinical condition deteriorated such that he was transferred to the ICU, and replacement of the mitral valve was accomplished with a 29-mm Carbomedics mechanical valve. Considering the presence of coma and fever (39.8°C) postoperatively, brain CT was obtained and showed multiple low-density lesions in temporal lobes, right parietal lobe, and occipital lobe, suggestive of cerebral embolism (Fig. 1D). Levofloxacin (0.75 g qd) was added to his antibiotic regimen. After 10 days of intravenous antibiotics, the patient regained consciousness (GCS score of 9) and made a good clinical recovery.

On day 35, he was transferred to a secondary hospital, and linezolid therapy was continued for eight weeks. He recovered uneventfully and was well at the last follow-up in November 2013.

## Microbiological investigations

The isolate recovered from the vegetation was first identified by the VITEK 2 system and then identified with MicroFlex LT instrument (Bruker Daltonics). Matrix-assisted laser desorption/ionization time of flight mass spectrometry (MALDI-TOF-MS) based fingerprint analysis of extracted proteins yielded a pattern similar to that of confirmed *S. aureus* isolates, Flexcontrol 3.0 software and Biotyper 2.0 database (Bruker Daltonics) identified the isolate as *S. aureus* with a maximum score value of 2.166 (data not shown). In addition to MALDI-TOF-MS analysis, 16S rRNA sequencing was performed in order to identify the origin of the bacteria in the vegetation. The sequence of the PCR product was compared with sequences of closely related species in GenBank by using BLAST. Sequencing of the 16S rRNA gene of the isolates showed that there was 100% identity with the 16S rRNA gene sequence of the isolate of *S. aureus* (GenBank accession no. JN102565), confirming that the isolate was *S. aureus*.

To further investigate the genetic basis of the strain, multilocus sequence typing (MLST), a method that uses seven housekeeping genes (*arcC*, *aroE*, *glpF*, *gmk*, *pta*, *tpi* and *yqiL*) for genetic identification, and result was assigned by comparison with the *S. aureus* MLST database (<http://www.mlst.net/>). The staphylococcal chromosomal cassette (SCC) *mec* type (I–V) was also determined. The *spa* type was analyzed by sequencing of the PCR product of the *spa* gene, and the *spa* type was assigned using an online *spa* database (<http://www.spaserver.ridom.de/>). Detection of the accessory gene regulator (*agr*) allele group was according to PCR and sequencing. Likewise, the antimicrobial drug resistance genes (*mecA*, *msrA*, *msrB*, *ermA*, *ermB*, *ermC* and *blaZ*) were determined. The presence of gene encoding PVL (*lukF/lukS*) and other virulence related genes (*sea*, *seb*, *sec*, *sed*, *see*, *seg*, *seh*, *sei*, *sej*, *sem*, *sen*, *seo*, *sek*, *sel*, *sep*, *seq*, *hla*, *hlb*, *hld*, *hlg*, *hlg2*, *eta*, *etb*, *lukE*, *lukM*, *bsaA* and *edin*) were investigated by PCR. The presence of adhesion genes (*cna*, *clfA*, *clfB*, *fnbA*, *efb* and *icaA*) were also determined by PCR and sequencing.

The CA-MRSA isolate was typed as sequence type (ST) 630 SCC*mecV* with *spa*-type t4549, *agrI/IV* and was PVL-negative. We confirmed the presence of *mecA*, *ermC* and *blaZ* genes by PCR and sequencing. The genome of the MRSA isolate encoded three hemolysin genes (*hlyA*, *hlyB* and *hlyC*) and five adhesion genes (*clfA*, *clfB*, *fnbA*, *efb* and *icaA*) (Fig. 1E).

## Discussion

IE is a rare entity of CA-MRSA presentation, especially in non-intravenous drug users (IVDU), and CA-MRSA endocarditis cases are mostly restricted to IVDUs, especially among HIV-infected patients with the USA300 strain. To date, 11 previously reported cases of infective endocarditis caused by CA-MRSA, excluding cases in IDVU patients were identified. The clinical features of these cases (including the one

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