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Short Communication

Infective Endocarditis Complicated by Intraventricular Abscesses, Pericarditis, and Mycotic Aneurysm Due to an Emerging Strain of Serotype VI *Streptococcus agalactiae*

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SUMMARY: An increasing number of invasive infections due to *Streptococcus agalactiae* in non-pregnant adults have been reported. We report a case of infective endocarditis complicated by intraventricular abscesses, pericarditis, and mycotic aneurysm due to *S. agalactiae* belonging to ST681 with a capsular serotype VI in a woman with diabetes. The patient also had a myocardial infarction and was treated with percutaneous coronary intervention, pericardiocentesis, and 6 weeks of antibiotic treatment. Invasive infections due to serotype VI *S. agalactiae* are common in Asian countries such as Taiwan and Japan, so continuous monitoring of invasive *S. agalactiae* strains is warranted.

Streptococcus agalactiae, also known as group B *Streptococcus* (GBS), is a Gram-positive coccus that frequently colonizes the human genital and gastrointestinal tracts. Colonization in the genital tract of pregnant women is of particular importance because it can lead to serious infections in neonates. Recently, an increasing number of GBS cases causing invasive disease in non-pregnant adults have been reported. We herein report a case of infective endocarditis complicated by intraventricular abscess, pericarditis, and mycotic aneurysms due to GBS belonging to ST681, an emerging strain with capsular serotype VI.

A 52-year-old woman was admitted to our hospital with generalized pain and muscle weakness over the prior week. Informed consent to publish this paper was obtained from the patient. Her medical history included hypertension, diabetes, hyperlipidemia, and alcoholic liver cirrhosis—all of which were untreated. Her vital signs were as follows: blood pressure, 94/40 mmHg; pulse, irregular and ranging between 30 and 170 beats per minute; temperature, 37.3°C; and respiratory rate, 16 breaths per minute. On physical examination, multiple dental caries and Levine grade 2 systolic murmur at the left apex were noted.

Laboratory findings on admission were as follows: white blood cell count, 36 × 10^9/L; C-reactive protein, 147 mg/L; creatinine, 123.8 μmol/L; blood urea nitrogen, 12.9 mmol/L; aspartate aminotransferase, 61 U/L; alanine aminotransferase, 24 U/L; alkaline phosphatase, 1,176 U/L; γ-glutamyl transpeptidase, 759 U/L; total bilirubin, 53 μmol/L; glucose, 24.1 mmol/L; and HbA1c, 11%. Electrocardiography (ECG) showed generalized ST-T wave changes, atrial fibrillation, atrial extrasystoles, and complete atrioventricular (AV) block. The transthoracic echocardiography revealed a large mass in the right atrium and a massive pericardial effusion. Coronary angiography revealed severe stenosis in the proximal left anterior descending (LAD) and total occlusion in the distal LAD. Balloon dilatation was performed in the proximal LAD.

We performed pericardiocentesis and started empiric intravenous meropenem and vancomycin. The pericardial fluid and 2 sets of blood culture were positive for *S. agalactiae*. The penicillin G minimum inhibitory concentration was ≤ 0.06 μg/mL using the broth microdilution method according to the Clinical Laboratory Standards Institute guidelines. We then switched to continuous infusion of penicillin G (24 million units every 24 hours) with gentamicin (60 mg every 8 hours). On day 10, a contrast-enhanced abdominal computed tomography (CT) scan was performed, on which low-density areas indicative of abscesses in the ventricular septum and free wall were incidentally identified (Fig. 1). We considered surgical treatment; however, the patient and her family refused surgery. On day 11, diffusion magnetic resonance imaging of the brain showed multiple high-intensity areas, suggesting multiple cerebral infarctions. According to the modified Duke criteria, we diagnosed definite infective endocarditis. Antimicrobial therapy was continued for a total of 6 weeks. The complete AV block resolved by day 10 and the low-density areas diminished on the CT scan by day 157; however, at this time, a new aneurysm was detected in the descending aorta. The patient was finally discharged from the hospital on day 189. There was no recurrence over

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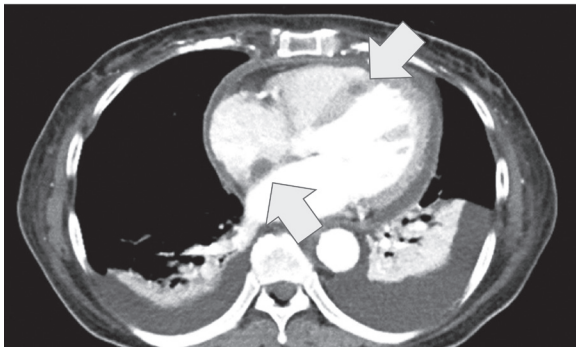

Fig. 1. Enhanced abdominal computed tomography scan showing abscesses in ventricular septum and free wall.

an 8-month follow-up period with careful observation of the aneurysm.

Identification of GBS was carried out by a polymerase chain reaction (PCR) assay and 16S ribosomal RNA analysis. PCR was based on the *dltS* gene, which encodes a histidine kinase specific to GBS. According to the EZBioCloud database (http://www.ezbiocloud.net), the isolate was 99.93% (1,418/1,419 bp) identical to *S. agalactiae* ATCC 13813(T), while concordance with other *Streptococcus* species was less than 97.26%. Multilocus sequence typing (MLST) analysis revealed that this isolate was the ST681 strain according to the MLST database (http://pubmlst.org/sagalactiae). Identification of the capsular type of GBS isolates was performed by multiplex PCR assay according to a previously published procedure (1). This isolate was indicated to be serotype VI.

GBS causes a wide range of illnesses and accounts for 2–3% of all cases of native valve infective endocarditis. We found large vegetation in the right atrium—one of the distinctive features of GBS infective endocarditis—which is sometimes mistaken for an atrial myxoma. Pericarditis, intraventricular abscess, and mycotic aneurysm are rarer forms of GBS infection, with only a few cases being reported to date. There has been only one case report of a patient with GBS complicated by infective endocarditis, pericarditis, and intraventricular abscess; this patient died after 19 days of therapy (2). Our case is the first that was further complicated by mycotic aneurysm. As in our case, mycotic aneurysms can develop during or after the completion of therapy; therefore, careful follow-up is required.

Abscess formation in infective endocarditis can be classified as a perivalvular abscess or an intramyocardial abscess. Perivalvular abscesses are estimated to complicate 30–40% of cases of infective endocarditis, whereas intramyocardial abscesses are rare and can also occur without infective endocarditis. The patient in our case had both myocardial infarction and myocardial abscess; abscesses have been reported to form in the area of myocardial ischemia and occur secondary to myocardial infarction (3). The patient in our case had severe and untreated diabetes mellitus and likely had preexisting coronary artery stenosis, both of which may have contributed to abscess formation in the ischemic area.

GBS can be classified into serotypes (Ia, Ib, and II–IX) based on the structure of the surface capsular polysaccharides, which are the main target of currently developed vaccines. The distribution of serotypes differs according to several factors, such as geographic area and type of disease. Most of the vaccines are designed to cover common serotypes such as Ia, III, and V, which are commonly found in the United States, Canada, and European countries. In Japan, serotype VI has been a common serotype since the 1990s. Morozumi et al. reported that among adult invasive diseases, serotype VI GBS had the highest mortality and accounted for 9.5% of all cases (4). The prevalence of serotype VI has also increased in Taiwan (5). Most serotype VI isolates belong to clonal complex 1; the ST681 strain that was found in our case also belongs to this clonal complex. Only one isolate belonging to ST681 had been found previously in Taiwan; our case is the second. The patient in our case had no travel history and the route of infection is unclear (5). An association between virulence and certain serotypes such as ST17 has been established. Therefore, careful monitoring of the serotypes and genetic backgrounds of invasive GBS isolates is warranted (6).

In conclusion, we successfully treated a case of infective endocarditis due to GBS complicated by pericarditis, intraventricular abscesses, and mycotic aneurysm. The organism was identified as an emerging strain of serotype VI GBS; therefore, continuous monitoring of molecular characteristics of invasive GBS strains is warranted.

Conflict of interest None to declare.

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