**Case Report**

A 28-year-old woman was admitted to our hospital presenting with fever, headache, abdominal pain and nausea. Small left to right shunt through the VSD was identified several years before (congenital). The patient showed sudden onset of fever, headache, abdominal pain and nausea, and had a history of cephalosporin usage for 15 days.

**Figure 1** - A vegetation attached to the right ventricular wall near the ostium of the ventricular septal defect. RV: right ventricle, LV: left ventricle, RA: right atrium, LA: left atrium, AO: aorta, PA: pulmonary artery, V: vegetation.
Physical examination was temperature 38°C, respirations 20/min, blood pressure 160/90 mmHg, pale conjunctivae, crackles on down left lung fields, S1, S2 normal, regular rhythm, 4/6 halosystolic murmur in the third left intercostal space determined.

Laboratory findings were Hb:10.9 g/dL, Hct 31.7%, WBC 18,300 mm³ with 94.2% polymorphonuclear cells, 3.6% lymphocytes, 2.1% monocytes; platelets 398,000 mm³; aspartate aminotransferase 37 U/L, alanine aminotransferase 60 U/L, lactate dehydrogenase 457 U/L, blood urea nitrogen 9 mg/dL, creatinine 0.78 mg/dL, C reactive protein 1.8 mg/dL, sedimentation rate 30 mm/h; urinalysis: 6-7 red blast cell; compleman 3 (C3): 68.1 mg/dL (90-180 mg/dL), (C4): 20.4 mg/dL (10-40 mg/dL); rheumatoid factor <8.94 U/mL. Methicillin-susceptible Staphylococcus aureus (MSSA) was detected twice from blood cultures.

Chest radiography and abdomen ultrasonography were normal. Fundoscopic examination was normal. Transthoracic echocardiographic examination showed VSD and vegetation attached to the right ventricular wall near the ostium of the VSD (Figure 1). Ampicillin-sulbactam, 2 g every 6 h, combined with gentamicin, 80 mg every 8 h initiated, and used for the first 5 days. Repeated blood cultures 48 hours later were negative. Intravenous ampicillin-sulbactam was administered for a total of four weeks.

**DISCUSSION**

IE was invariably lethal in the pre-antibiotic era. Fifty years ago, the introduction of penicillin followed by other antibiotics revolutionized the treatment and prognosis of IE. More recently, developments in clinical microbiology and the availability of improved imaging techniques, especially transthoracic and transesophageal echocardiography, have led to new, more accurate diagnostic criteria (4).

In industrialized countries, despite improvements in health care and a sharp decrease in the incidence of chronic rheumatic heart disease, IE has not disappeared and has even increased in some populations (intravenous drug users, elderly people with sclerotic cardiac valves and patient who have intravascular prostheses) (5). Congenital heart disease is a lifelong risk factor for IE. It is a major risk factor in children - a less frequent risk factor in adults. This includes all of the cardiac abnormalities associated with turbulent blood flow. Tetralogy of Fallot carries the highest risk for IE, followed by bicuspid aortic valve, coarctation of the aorta and VSD (5). Size of the VSD is not correlated with IE risk; surgical closure of VSD lowers the risk of IE (2). Frontera Izquierdo et al. studied 882 cases of isolated VSD: only five patients (0.5%) developed IE [6]. Otterstad et al. reviewed clinical and haemodynamic findings in 109 consecutive patients in whom an isolated VSD was diagnosed after the age of 15 years (range 15-65 years): 16 (15%) patients had developed IE [7]. Our patient was admitted to our hospital because of fever, headache, abdominal pain and nausea. She had been diagnosed as having VSD. In addition to positive physical examination findings, positive blood cultures for MSSA and the detection of vegetation attached to the right ventricular wall near the ostium of the VSD confirmed the diagnosis of IE. According to Dukes’ criteria for IE diagnosis, one major and four minor clinical criteria were detected in our patient. The major criteria was a typical micro-organism for IE from two separate blood cultures (viridans streptococci, Strep- tococcus bovis, HACEK -Haemophilus spp., Actinobacillus actinomyctemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae- group, or community-acquired Staphylococcus aureus or enterococci in the absence of a primary focus). Staphylococci cause at least 20 to 30% of the cases of IE, and 80 to 90% of these are due to S. aureus (1). MSSA was cultured from the blood in our case. Primary focuses could not be found for MSSA.

Our patient’s minor criteria were VSD (predisposing cardiac condition), ≥38 °C fever, microscopic hematuria (immunologic phenomena: glomerulonephritis) and vegetation attached to the right ventricular wall near the ostium of the VSD.

In general tricuspid valve involvement is mostly seen in VSD (8-10). In our case the vegetation was located in the right ventricular free wall. In the published literature only one similar case accompanying a complicated pulmonary embolism was determined (3). As a result, our patient was discharged without any complication after six weeks’ antibiotic therapy. It must be borne in mind that such rare cases may occur.

**Key words:** infectious endocarditis, vegetation, ventricular septal defect
A 28-year-old woman previously known to have a ventricular septal defect presented with fever, headache, abdominal pain and nausea. Positive blood culture of methicillin-sensitive *Staphylococcus aureus* and the detection of vegetation attached to the right ventricular wall near the ostium of the ventricular septal defect confirmed diagnosis of infective endocarditis. After four weeks’ treatment with proper antibiotics the patient recovered.

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**SUMMARY**

**REFERENCES**


