

A case of right-side infective endocarditis with ventricular septal defect

Un caso di endocardite del cuore destro con difetto del setto ventricolare

Özge Turhan, Rabin Saba, Aytul Belgi¹, Dilara Inan, Hicran Karaoglan, Ata Nevzat Yalcin

Department of Infectious Diseases and Clinical Microbiology;

¹Department of Cardiology, Medicine Faculty, Akdeniz University, Antalya, Turkey

INTRODUCTION

Infective endocarditis (IE) denotes infection of the endocardial surface of the heart and implies the physical presence of micro-organisms in the lesion. Although the heart valves are most commonly affected, the disease may also occur within septal defects or on the mural endocardium (1).

Congenital heart disease (especially patent ductus arteriosus, ventricular septal defect (VSD), coarctation of the aorta, bicuspid aortic valve, tetralogy of Fallot and rarely, pulmonary stenosis) is responsible in 6 to 24% of the cases (1). For VSD, the incidence rate of IE was 14.5 per 10,000 person-years (2). The classic vegetation of IE is usually located along the line closure of a valve leaflet on the atrial surface for atrioventricular valves or the ventricular surface for semilunar valves (1). Rarely was the lesion right ventricular free wall (3). Here we present a case of IE in a patient with VSD showing vegetation attached to the right ventricular wall.

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.

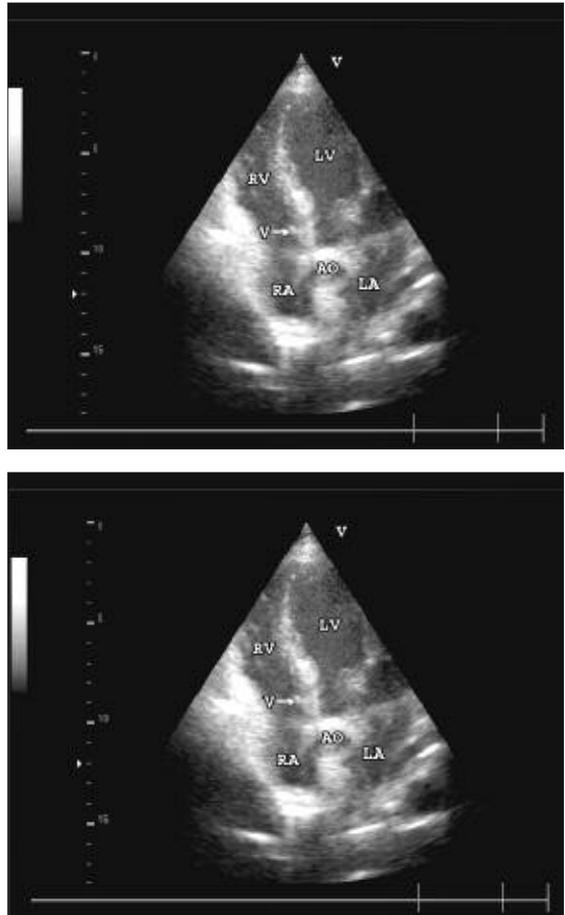


Figure 1 - A vegetation attached to the right ventricular wall near the ostium of the ventricular septal defect. RV: right ventricle, LV: left ventricle, RA: right atrium, LA: left atrium, AO: aorta, PA: pulmonary artery, V: vegetation.

Physical examination was temperature 38°C, respirations 20/min, blood pressure 160/90 mmHg, pale conjunctivae, crackles on down left lung fields, S₁S₂ 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, *Streptococcus bovis*, HACEK -Haemophilus spp., *Actinobacillus actinomycetemcomitans*, *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

SUMMARY

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 vegeta-

tion 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.

RIASSUNTO

Gli autori descrivono un caso clinico di endocardite infettiva osservato in una donna di 28 anni, con difetto del setto ventricolare, giunta alla loro osservazione con una sintomatologia caratterizzata da febbre, cefalea, dolore addominale e nausea. La diagnosi fu resa possibile grazie all'isolamento, da

emocultura, di Staphylococcus aureus, meticillino-sensibile, e dall'osservazione di vegetazioni localizzate sulla parete ventricolare destra, prossima all'ostio del setto ventricolare. La guarigione è stata ottenuta a seguito di quattro settimane di terapia antibiotica.

REFERENCES

- [1] Bayer A.S., Scheld W.M. Endocarditis and intravascular infections. In: Mandell GL, Bennett JE, Dolin R. Principles and practice of infectious diseases, fifth edition, Churchill Livingstone, Philadelphia, 2000; 857-902.
- [2] Gersony W.M., Hayes C.J., Driscoll D.J., et al. Bacterial endocarditis in patients with aortic stenosis, pulmonary stenosis, or ventricular septal defect. *Circulation* 87 (Suppl.2), I121-26, 1993.
- [3] Zijlstra F., Fioretti P., Roelandt J.R. Echocardiographic demonstration of free wall vegetative endocarditis complicated by a pulmonary embolism in a patient with ventricular septal defect. *Br. Heart J.* 55, 5, 497-499, 1986.
- [4] Durack D.T., Lukes A.S., Bright D.K. New criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings. Duke Endocarditis Service. *Am. J. Med.* 96, 3, 200-209, 1994.
- [5] Moreillon P. Endocarditis and Endarteritis. In: Armstrong D, Cohen J. Infectious Diseases, 1999, Mosby, London; 2 (50.1).
- [6] Frontera-Izquierdo P., Cabezuelo-Huerta G. Natural and modified history of isolated ventricular septal defect: a 17-year study. *Pediatr Cardiol.* 3, 4, 193-197, 1992.
- [7] Otterstad J.E., Nitter-Hauge S., Myhre E. Isolated ventricular septal defect in adults. Clinical and haemodynamic findings. *Br. Heart J.* 50, 4, 343-348, 1983.
- [8] Spyridopoulos I., Helber U., Mewis C., et al. Tricuspid valve endocarditis due to a jet lesion detected by echocardiography in a 27-year old man with congenital ventricular septal defect. *J. Cardiovasc. Surg. (Torino)*. 37, 5, 517-520, 1996.
- [9] Wu M.H., Chang C.I., Wang J.K., Lue H.C. Characterization of aneurysmal transformation in perimembranous ventricular septal defects: an adhered anterior leaflet of tricuspid valve predisposes to the development of left ventricular-to-right atrial shunt. *Int. J. Cardiol.* 47, 2, 117-125, 1994.
- [10] Itoh N., Shigematsu H., Itoh M., Yamada H. Right-sided infective endocarditis combined with mitral involvement in a patient with ventricular septal defect. *Acta Pathol. Jpn.* 35, 2, 459-471, 1985.