

# Endocarditis of native mitral valve which was presented like refractory septic shock

GORDANA CAVRIĆ, MD, PHD<sup>1</sup>, INGRID PRKAČIN, MD, ASSOC. PROF.<sup>1</sup>, KHALED NASSABAIN, MD<sup>2</sup>, MAJA VUČKOVIĆ, MD<sup>1</sup>, DUNJA KURBEGOVIĆ, MD<sup>1</sup>, DUBRAVKA BARTOLEK HAMP, MD, ASSIST. PROF.<sup>3</sup>

<sup>1</sup>Department of Internal Medicine, University Hospital Merkur, Zagreb, Croatia

<sup>2</sup>Centre Hospitalier de l'Ouest Vosgien, Pole mère-enfant, Neufchâteau, France

<sup>3</sup>Department of Anaesthesiology, Resuscitation and Intensive Care, Dubrovnik General Hospital, Dubrovnik, Croatia

Corresponding author:

Gordana Cavrić,

Zajčeva 19, University Hospital Merkur, Zagreb, Croatia,

tel:+38512253232, fax:+38512431393,

e-mail: gordana.cavric1@gmail.com

## ABSTRACT

We showed the case of a sixty-five-year-old woman who developed refractory septic shock as a result of endocarditis of the native mitral valve with maintained neat systolic function of the left ventricle and moderate mitral regurgitation. Streptococcus sanguinis was isolated from hemoculture. The patient did not have clearly known predisposing factors for the development of endocarditis, nor had elevated temperature. She died 32 hours after being admitted to the hospital.

*Key words: endocarditis, native mitral valve, refractory septic shock*

## INTRODUCTION

There is an increasing in number of patients with endocarditis in the world and in Croatia (1, 2). Sometimes, endocarditis may have the clinical picture of septic shock.

## CASE REPORT

A sixty-five-year-old female patient presented to the emergency department because of general weakness, dyspnea and pressure in her chest. Soon after her arrival, clinical signs of severe sepsis of undetermined origin were developed and she was transferred to the medical intensive care unit (MICU). The patient was allegedly born with hip dysplasia, she has walking difficulties but she was not immobile and the last couple of years she has been receiving a treatment for hypothyroidism. Two weeks ago, she complains of general weakness, the first symptom was pain in

her knees, then in her hips and then in her back. She had no fever or chills, and for one week she has been intermittently experiencing a feeling of mild pressure in her chest associated with dyspnea. She spent the last two weeks lying in her bed because of weakness. A diminished appetite led to a weight loss of 10 kg, and to mild constipation. She has no urinary complaints but in the last couple of days she has oliguria. She is not a smoker, nor an alcoholic. The medicaments she used were a combination of tramadol and paracetamol and levothyroxine.

Clinically, she was conscious but had difficulty communicating because of her general condition. She was moderately obese, her temperature was 35.7 °C, skin pale, her blood pressure was 100/50 mmHg, pulse was 99/min, and had a respiratory frequency of 30/min. The APACHE II score at her arrive was 24, the GCS was at 5. Her mouth was dry, her neck veins were not distended, the cardiopulmonary auscultation reveals no respiratory abnormalities except a rare crepitation in the right inferior region, the heart activity was rhythmic, the heart sounds were muffled, with a systolic murmur of 2/6 intensity at the precordium, PM over the apex beat. The pulsations of the dorsalis pedis artery were bilaterally attenuated but the proximal arteries were well palpable. External haemorrhoids with partial thrombosis were visible, not bleeding currently. Rectal examination revealed that little internal haemorrhoids were palpable, and the stool was brown in color. The ECG showed a regular sinus heart rhythm at 98/min. The chest X-ray reveals a wide heart transversal diameter, reduced airiness of right chest parenchyma and accentuated vascular markings in the parahilum region bilaterally, markedly at the right side.

Upon her admission at the MICU, the plasma volume expansion was continued, blood culture and urine culture were ordered. Treatment consisting of vancomycin and meropenem antibiotics was started. An abdominal ultrasound and Doppler of leg veins were carried out. Deep venous thrombosis of the legs was excluded and the abdominal ultrasound did not reveal any significant pathology.

The ultrasound examination of the heart shows a normal heart volume, preserved systolic function, no regional contractility defects were found, no hypertrophy of the right side was visible (left ventricle diastolic diameter 5.1 cm, intraventricular septum 0.9 cm, posterior wall of left ventricle 1.1 cm and ejection fraction (EF) at 65 %). The valves were changed fibrosclerotically. A relatively large calcification was seen beside the posterior mitral leaflet. The two leaflets were enlarged with irregular outlines with a marked prolapse of the posterior leaflet and moderate mitral regurgitation. A moderate tricuspid regurgitation was also found with a pressure in the pulmonary artery of about 45 mmHg.

During her stay clinical shock developed, noradrenaline was rapidly administered and dobutamine and adrenaline were added, consequently, partial stabilization of her condition was initiated with a gradual reduction of vasoactive drugs. However, the condition of the patient deteriorated once again due the development of global respiratory insufficiency and she was intubated and attached to mechanical ventilation. The microbiological laboratory reported that Gram-positive cocci were seen growing. The patient died about 32 hours after her admission to the MICU under the clinical picture of refractory septic shock with multiple organ failure. Post-mortem

information is obtained that *Streptococcus sanguinis* is isolated in her blood culture. Autopsy confirms the diagnosis of endocarditis.

## DISCUSSION

By reporting this case we wanted to draw attention to the occurrence of endocarditis in a patient who had no history of serious illness, who was not a cardiovascular patient, nor did she have any cardiac anomalies. By anamnesis and clinical examination it was difficult to determine the initial location of infection which lead to septicemia and endocarditis. The endocarditis in this case was not a "mechanical" cause for shock, however, its infectious component lead to shock development.

The number of infectious endocarditis patients is increasing in the world (1) and in our country (2). Fever is the most common symptom; it is reported in up to 90% of patients. Arthralgia, weakness, loss of appetite and dyspnea (the main symptoms of

our patient) are also commonly common symptoms although they are non-specific and do not fall into the clinical criteria for diagnosis (3). In a study that included 2781 patients with infectious endocarditis, *Streptococcus viridans* (in which *Streptococcus sanguinis* is included) was found in 17% of patients (1, 4). Vancomycin is an adequate initial empirical therapy of infectious endocarditis in most patients (5).

Refractory shock is a condition requiring more than 0.5 µg / kg / min of noradrenaline or adrenaline to maintain the target pressure after adequate fluid replenishment. We can find refractory shock in about 6% of critically ill patients (6).

Taking into consideration that this was a refractive septic shock, we think that in such a case we might think about applying VA ECMO. Some French literature describes the treatment of septic shock in cases of reduced ejection fraction or lowered cardiac index (7), but there are also papers describing its use in cases of preserved ejection fraction. One of them

describes survival of 15% (8). Surely the survival rate is not great, but 15% survival is still a chance in cases of refractive shock where practically every patient is considered a "candidate for dying".

So, in describing this patient we would like to emphasize two things: 1) the appearance of endocarditis without the usual signs; elevated body temperature and without previously known predisposing factors for the development of endocarditis; 2) considering the well-known high mortality in cases of refractory septic shock, after all that can be done from conservative treatment in the 21st century, would one try to take another step and try the VA ECMO?

## ACKNOWLEDGEMENTS

We thank the staff of the department of cardiology. We also thank Maja Mijić, MD, and Mihaela Gunjača, MD, for technical support.

## REFERENCES

1. Sexton DJ, Chu VH. Epidemiology, risk factors, and microbiology of infective endocarditis. From: [https://www.uptodate.com/contents/epidemiology-risk-factors-and-microbiology-of-infective-endocarditis?search=endocarditis&source=search\\_result&selectedTitle=3~150&usage\\_type=default&display\\_rank=3](https://www.uptodate.com/contents/epidemiology-risk-factors-and-microbiology-of-infective-endocarditis?search=endocarditis&source=search_result&selectedTitle=3~150&usage_type=default&display_rank=3)
2. Baršić B. Infective endocarditis chemoprophylaxis. *Medicus* 2016; 25: 177- 179.
3. Sexton DJ, Chu VH. Clinical manifestation and evaluation of adults with suspected native valve endocarditis. From: [https://www.uptodate.com/contents/clinical-manifestations-and-evaluation-of-adults-with-suspected-native-valve-endocarditis?search=endocarditis&source=search\\_result&selectedTitle=1~150&usage\\_type=default&display\\_rank=1](https://www.uptodate.com/contents/clinical-manifestations-and-evaluation-of-adults-with-suspected-native-valve-endocarditis?search=endocarditis&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1)
4. Murdoch DR, Corey GR, Hoen B, Miró JM, Fowler VG Jr, Bayer AS, et al. Clinical presentation, etiology, and outcome of infective endocarditis in the 21st century: the International Collaboration on Endocarditis- Prospective Cohort Study. *Arch Intern Med* 2009; 169: 463- 73.
5. Sexton DJ. Antibicrobial therapy of native valve endocarditis. From: [https://www.uptodate.com/contents/antimicrobial-therapy-of-native-valve-endocarditis?search=endocarditis&source=search\\_result&selectedTitle=2~150&usage\\_type=default&display\\_rank=2](https://www.uptodate.com/contents/antimicrobial-therapy-of-native-valve-endocarditis?search=endocarditis&source=search_result&selectedTitle=2~150&usage_type=default&display_rank=2)
6. Bassi E, Park M, Azevedo LC. i sur. Therapeutic strategies for high-dose vasopressor-dependent shock. *Critical Care Research and Practice*. 2013; 2013:654-708.
7. Bréchet N, Luyt CE, Schmidt M, Leprince P, Trouillet JL, Léger P et al. Venoarterial Extracorporeal membrane oxygenation support for refractory cardiovascular dysfunction during severe bacterial septic shock. *Crit Care Med* 2013; 41:1616- 26.
8. Huang CT, Tsai YJ, Tsai PR, Ko WJ. Extracorporeal membrane oxygenation resuscitation in adult patients with refractory septic shock. *J Thorac Cardiovasc Surg* 2013; 146: 1041- 6.