CONDUCTION DISTURBANCES IN A WOMAN WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Jovana Cvetković¹, Tomislav Kostić^{2,3}, Jovan Nedović¹, Sonja Stojanović^{1,3}, Bojana Stamenković^{1,3}, Dragana Stanojević², Viktor Stoičkov^{1,3}

Cardiovascular involvement represents the leading cause of mortality in Systemic lupus erythematosus (SLE) patients. Its most common manifestations include pericarditis, valvular affections, conduction disorders, and arterial hypertension. Conduction disturbances are rare complication in the course of systemic lupus erythematosus. We describe the case of a woman with systemic lupus erythematosus and lupus cardiomyopathy who was admitted to our hospital due to dizziness showing on the electrocardiogram Mobitz type II atrioventricular block and left anterior bundle branch block. This problem was resolved with a pacemaker implantation. *Acta Medica Medianae* 2017;56(3):38-41.

Key words: SystemiclLupus erythematosus, Mobitz type II atrioventricular block, pacemaker

Institute for Treatment and Rehabilitation Niška Banja, Niška Banja, Serbia¹ Clinic of Cardiology, Clinical Center Niš, Niš, Serbia² University of Niš, Faculty of Medicine, Niš, Serbia³

Contact: Jovana Cvetković Institute for Treatment and Rehabilitation, Niška Banja, Serbia E-mail: miljevicjovana@yahoo.com

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by the production of autoantibodies, immune complex deposition, and heterogeneous clinical manifestations. Cardiac involvement with systemic lupus erythematosus usually occurs in patients with an established diagnosis and typically involves the pericardium, myocardium, valves, and coronary vessels with conduction abnormalities being rare (1). Cardiovascular involvement is present in up to 50% cases (2) and it is associated with increased morbidity and mortality of SLE patients.

Case report

A female patient, 48 years old, was admitted to the Clinic for cardiovascular diseases, Clinical Center Niš, due to dizziness on 22 April, 2016. The patient was referred from a rheumatologist, who performed a detailed ambulatory examination and found bradycardia. Beside dizziness, the patient had pain in the hands and in the right shoulder. Physical examination showed discrete hyperemia of cheekbones, absence of active arthritis, the Lazarević's sign was negative, and auscultatory irregular and slow cardiac rhytms were found. On the ECG, the heart rate was 40/minute, with Mobitz type II atrioventricular block and left anterior bundle branch block (Figure 1). After hospital admission, we performed echocardiography which showed that left ventricle ejection fraction was 62%, without significant abnormalities, except for a mild mitral regurgitation due to sclerotic mitral leaflets.

Detailed medical history was taken. The first complaints patient had in 2002. She had a pain in the right hip and right leg, with pain and swelling of the elbows, shoulders, ankles and in the small joints of the hands and feet. Further, she was complaining of the morning stiffness lasting up to 15 minutes. In 2003, the diagnosis of rheumatoid arthritis was made and therapy with methotrexate



Figure 1. : ECG showing Mobitz type II atrioventricular block



Figure 2. : Coronarography showing normal epicardial coronary arteries

was started, but was stopped in 2004 due to an increase in transaminases. Soon after methotrexate exclusion from the therapy, the patient started to have a high fever (39.8°C), swelling of the peripheral joints, stiffness of the jaw and of entire body, difficulty swallowing, facial flushing, photosensitivity, erythematous changes on the fingertips, and ulcers around the edges of the nail plates. In 2004, she noticed lymphadenopathy in the neck and in the inquinal area. Because of these symptoms she was hospitalized in 2005 at the Clinic for Rheumatology in the Institute for treatment and rehabilitation "Niška Banja", where clinical and laboratory examinations with additional serology (anti-dsDNA, Le cells, antinucleolar antibodies - ANA, low complement C3) were performed and diagnosis of the overlap syndrome (rheumatoid arthritis plus systemic lupus erythematosus - SLE) was made with the introduction

of combined therapy with Azathioprine and Chloroquine. The patient was re-hospitalized in July 2013 when the symptoms of lumbar herniated disc predominated. Due to low SLE activity, the therapy with Azathiprine was stopped, but she continued to take Resochine and Prednisone. The next re-hospitalization was in July 2015, again due to pain in the lumbosacral spine, both legs with difficulties walking and due to elastic oedema in the lower legs accompanied with a burning sensation in both ankles, small joints of the hands, as well as the occurrence of red spots and bruises on the whole body. NMR showed the presence of lumbosacral spinal disc herniations at the L2-L3 and L4-L5 levels and a neurosurgeon was included in the follow-up. In the February 2016, a thoracic disc prolapse at the level T9-T10 was found. The patient continued with prescribed therapy together with bisphosphonates once monthly due to osteoporosis. On 18 April, 2016 the patient had dizziness and was examined by a cardiologist who found bradycardia with heart rate 45/min and excluded nebivolol from the therapy which patient occasionally took due to palpitations, and she was again addmited to the hospital.

After hospital admission, physical examination, ECG, and echocardiography, the cardiologist decided to implant a permanent pace-maker. First, coronarography was performed to exclude possible



Figure 3.: Procedure of pace-maker implantation (electrode and battery placement)

coronary artery disease, which showed epicardial coronary vessels without significant stenosis (Figure 1). After that a coronarography pace-maker was implanted, there were no complica-tions during the implantation procedure (Figure 2). The patient was discharged from the clinic with a prescribed antibiotic and advice to continue treatment of the systemic disease of connective tissue.

Discussion

Patients with systemic lupus erythematosus rarely show conduction defects (3). A complete congenital heart block is one of the characteristic signs of the neonatal lupus syndrome and has been related to maternal transfers of abnormal (IqG) antibodies (4). However, the appearance of the conduction disturbances, including a complete AV heart block in adults with SLE, is an extremely rare manifestation of this disease. Conduction abnormalities may not be caused by SLE per se. Sometimes this complete AV heart block has been associated with special abnormal antibodies (anti-Ro) (5) or the anti-RNP nuclear antibodies. Anti-Ro/SSAs, including the anti-Ro/SSA-52kD and anti- Ro/SSA-60kD subtypes, are frequently found in patients with connective tissue diseases, particularly in those with Sjögren syndrome (30-95%) and SLE (30-50%), but also in 0.5-2.7% of the healthy population. Although it is believed that adult heart does not represent an immunological target for anti-Ro/SSA, an increasing body of data indicate that these autoantibodies may also affect

the adult conduction system, although to a lesser extent than the fetal heart (6). The inflammatory process in SLE is another possible pathogenic mechanism that could have a role in the AV conduction disturbances and their evolution. Salomone et al. (7) found AV node damage in the endomyocardial biopsy of systemic lupus erythematosus patients and the blood vessels located in the cardiac conducting tissue of patients suffering arrhythmias were also impaired. Atherosclerosis has been found to develop prematurely in patients with SLE. Namely, the presence of conduction disturbances often reflects ischaemic heart disease in SLE. It is thought that antiphospholipid antibodies can bind to oxLDL and β 2-glycoprotein, resulting in the accelerated formation of atheromatous plaques. The incidence of myocardial infarction and carotid artery stenosis in female SLE patients aged 35-44 years is shown to be 50 times higher than in control group in the Framingham Offspring Study. It is therefore vital to screen for and minimize cardiovascular risk factors by treating hypertension and hyperlipidemia, reducing corticosteroids to the lowest dose possible for disease control, and encouraging cessation of smoking (8, 9).

Our patient did not have a coronary heart disease and SLE was in remission. Therefore, the pathogenesis of conduction disturbance development in SLE is multicausal and complex. In patients with SLE and complaints of dizziness, palpitations, fatigue, chest pressure or pain, shortness of breath and syncope, ECG should be performed as soon as possible and a cardiologist should be consulted.

References

- 1. Doria A, Iaccarino L, Sarzi-Puttini P, Atzeni F, Turriel M, Petri M. Cardiac involvement in systemic lupus erythematosus. Lupus 2005; 14(9): 683-6. [CrossRef] [PubMed]
- Moder KG, Miller TD, Tazellar HD. Cardiac involvement in systemic lupus erythematosus, Mayo Clinic Proceedings 1999; 74(3): 275–84. [CrossRef] [PubMed]
- 3. Bharati S, de la Fuente DJ, Kallen RJ, Freij Y, Lev M. Conduction system in systemic lupus erythematosus with atrioventricular block. Am J Cardiol 1975; 35(2): 299–304. [CrossRef] [PubMed]
- Scott JS, Maddison PJ, Taylor PV, Esscher E, Scott O, Skinner RP. Connective-tissue disease, antibodies to ribonucleoprotein and congenital heart block. New Engl J Med 1983; 309(4): 209–12. [CrossRef] [PubMed]
- Logar D, Kveder T, Rozman B, Dobovisek J. Possible association between anti-Ro antibodies and myocarditis or cardiac conduction defects in adults

with systemic lupus erythematosus. Ann Rheum Dis 1990; 49(8): 627–9. [CrossRef] [PubMed]

- Lazzerini PE, Capecchi PL, LaghiPasini F. Anti-Ro/SS Aantibodies and cardiac arrhythmias in the adult:facts and hypotheses.Scand J Immunol 2010; 72(3): 213–22. [CrossRef] [PubMed]
- Salomone E, Tamburino C, Bruno G, Di Paola R, Silvestri F. The role of endomyocardial biopsy in the diagnosis of cardiac involvement in systemic lupus erythematosus. Heart Vessels 1989; 5(1): 52–3.
 [CrossRef] [PubMed]
- 8. Lim S, Gordon S. Systemic Lupus Erythematosus Epidemiology. In: Systemic Lupus Erythematosus, Ed. Gordon C, Isenberg D. Oxford University Press, 2016, pp 73-80.
- Wu GC, Liu HR, Leng RX, Li XP, Li XM, Pan HF, Ye DQ. Subclinical atherosclerosis in patients with systemic lupus erythematosus: A systemic review and meta-analysis. Autoimmun Rev 2016; 15(1): 22-37. [CrossRef] [PubMed]

This work is licensed under a Creative Commons Attribution 4.0 International (CC BY 4.0) Licence

Prikaz bolesnika

UDK: 616-002.52:616.127-008 doi:10.5633/amm.2017.0306

POREMEĆAJI SPROVOĐENJA KOD BOLESNICE SA SISTEMSKIM ERITEMSKIM LUPUSOM

Jovana Cvetković¹, Tomislav Kostić^{2,3}, Jovan Nedović¹, Sonja Stojanović^{1,3}, Bojana Stamenković^{1,3}, Dragana Stanojević², Viktor Stoičkov^{1,3}

Institut za lečenje i rehabilitacijuⁿNiška Banja^{*}, Niška Banja, Srbija¹ Klinika za kardiologiju, Klinički centar Niš, Niš, Srbija² Univerzitet u Nišu, Medicinski fakultet Niš, Niš, Srbija 3

Kontakt: Jovana Cvetković Institut za lečenje i rehabilitaciju"Niška Banja", Niška Banja, Srbija E-mail: miljevicjovana@yahoo.com

Zahvaćenost kardiovaskularnog sistema predstavlja vodeći uzrok smrti kod ljudi obolelih od sistemskog eritemskog lupusa (SLE). Najčešće kardiovaskularne manifestacije su perikarditis, valvularna oboljenja, poremećaji u sprovođenju i arterijska hipertenzija. Poremećaji u sprovođenju se kao komplikacije javljaju retko kod obolelih od SLE. Prikazali smo bolesnicu sa sistemskim eritemskim lupusom i lupusnom kardiomiopatijom koja je hospitalizovana zbog tegoba po tipu vrtoglavice sa slikom atrioventrikularnog bloka tipa Mobitz II i prednje levog hemibloka na elektrokardiogramu. Ovaj problem je rešen ugradnjom pejsmejkera. *Acta Medica Medianae 2017;56(3):38-41.*

Ključne reči: sistemski eritemski lupus, atrioventrikularni blok Mobitz tip 2, pejsmejker