Original paper

Heart arrest due to systemic lupus erythematosus relapse: case report

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Received 9 December 2021; accepted 1 February 2022

Summary

We report a case of a 36-year-old man who was admitted to the Vilnius University Hospital Santaros clinics emergency department suffering from acute chest pain and experienced cardiac arrest. Changes in the electrocardiogram, elevated troponin level, and clinical symptoms lead to ST-elevation myocardial infarction (MI) diagnosis. The patient was diagnosed with systemic lupus erythematosus (SLE). He was in a remission state of SLE and was not taking drugs for one year before MI happened. SLE may be associated with antiphospholipid syndrome, which can cause cardiovascular complications such as MI. Although it is a usual practice to stop treatment in remission, in some cases, clinicians should consider continuing SLE treatment to avoid cardiovascular complications. In this case, the exacerbation of SLE and myocardial infarction occurred almost simultaneously with no previous thrombotic or cardiovascular events.

Seminars in Cardiovascular Medicine 2022; 28:1–4

Keywords: myocardial infarction, systemic lupus erythematosus, antiphospholipid syndrome

Background

Systemic lupus erythematosus (SLE) is a disease characterized by inflammation of autoimmune origin, thrombus formation, and related complications, including cardiovascular disorders [1,2]. Patients with SLE are 10 times more likely to develop ischaemic heart disease [1]. In 2018, the total amount of registered cases of SLE in Lithuania was 732. SLE is rare in Lithuania and its pathogenetic relation to ischaemic heart disease is not fully understood [3]. In this report, we present a case of myocardial infarction (MI), which may have occurred due to the exacerbation of SLE.

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Case presentation

In September of 2020 36-year-old man was admitted to Vilnius University Hospital Santaros clinic because of chest pain with the suspicion of ST-elevation MI. Ventricular fibrillation in electrocardiogram (ECG) was observed on arrival. The patient was resuscitated for 2 minutes and defibrillated once. After recovery of the patient's rhythm, ST-segment elevation was noticed on the ECG (Fig. 1). A week before the event, the patient had a fever up to 38.0°C.

For SLE, the patient was first consulted by a rheumatologist in 2008 who prescribed treatment with steroids and hydroxychloroquine. The patient discontinued steroid medication being in clinical and laboratory remission of the disease in 2015 and hydroxychloroquine in 2019. At the time of the event, he was not taking any medications.

Both ECG and elevated troponin levels showed changes characteristic of MI. Based on the diagnosis, percutaneous coronary intervention and

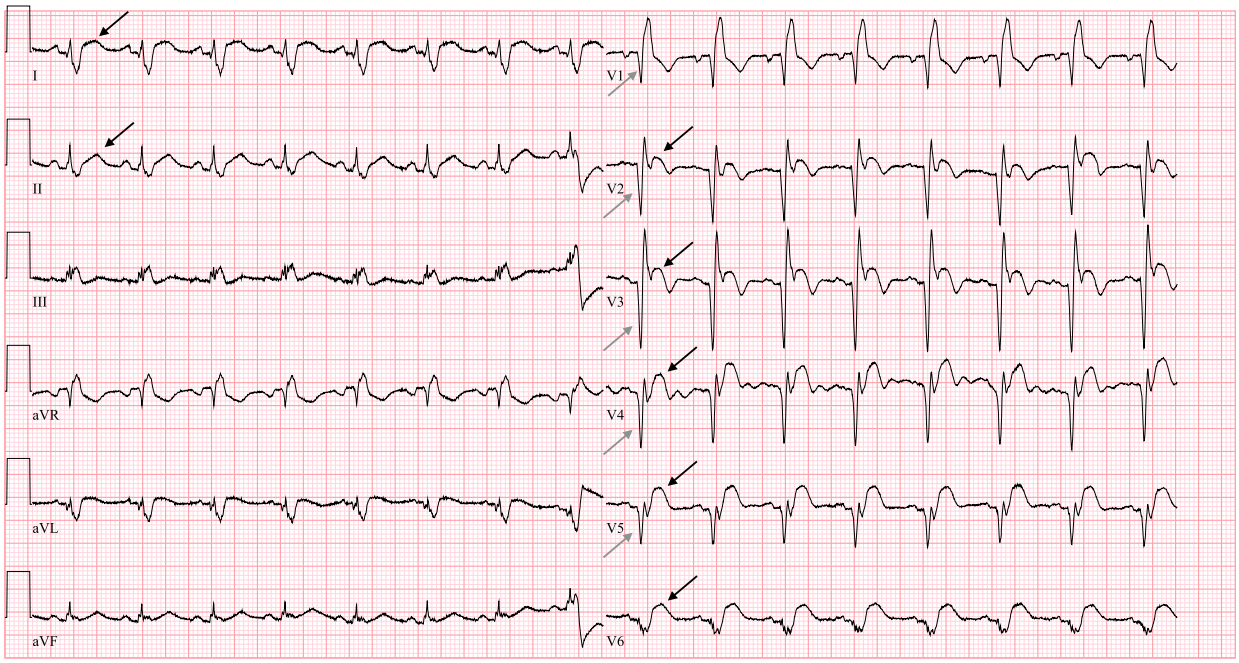


Figure 1. Electrocardiogram: ST elevation in I, II, V2-V6, pathological Q in V1-V5

stenting of the occluded left anterior descending artery (LAD) were performed.

After stenting, the pain was relieved, but the patient still had a fever up to 38.5°C, increasing inflammatory parameters (C-reactive protein (CRP) 39 → 159.5 mg/l). Fever persisted despite antimicrobial therapy. Consequently, exacerbation of SLE was suspected. The antibody profile showed an active clotting process during MI coming together with SLE and antiphospholipid syndrome (APS) (Table 1). The treatment with hydroxychloroquine 400 mg and prednisolone 10 mg per day was renewed, and three days after, CRP decreased to 11.5 mg/l.

The lipid panel test was done, which showed dyslipidaemia: total cholesterol was 3.49 mmol/l, triglycerides – 1.29 mmol/l, low-density lipoprotein cholesterol – 2.27 mmol/l, high-density lipoprotein cholesterol – 0.63 mmol/l.

Imaging tests were also performed during the hospitalization. The cardiac ultrasound showed ischaemic left ventricular (LV) apical damage and reduced longitudinal contraction of the LV wall (mid-anterior and mid-anteroseptal segments). Cardiac magnetic resonance imaging findings were: decreased ejection fraction (EF) – 34% of LV, post-ischaemic changes in the myocardium, predominant in the theoretical LAD supply area, and thrombus at the apex of LV (Fig. 2). Because of a thrombus in the LV and APS, warfarin was prescribed. As the patient's general condition improved, he was transferred to the rehabilitation unit.

A month after the incident, cardiac ultrasound showed a hypokinetic interventricular septum, a moderately decreased left ventricular inotropy (EF ∼ 40%) and the impaired diastolic function of the LV. According to the blood tests, SLE activity remained high. Hydroxychloroquine and steroids are continued and considered for a prolonged time.

Discussion

SLE is considered an independent cardiovascular risk factor [4]. Heart damage occurs in as many as 50% of people with SLE [5]. This autoimmune disease often affects the myocardium, pericardium, heart valves, and coronary arteries [6]. Those with active SLE also usually have high cholesterol and triglyceride levels and decreased HDL levels [8]. APS, which causes blood clotting abnormalities, is often found in people with SLE. This condition increases the risk of cardiovascular events even more in patients with SLE [7]. For these reasons, patients with the APS should receive low-dose aspirin to prevent thrombosis. If the patient already has a history of thrombotic events, long-term use of anticoagulants, such as warfarin, is indicated [10]. In this particular case, the patient was not taking aspirin because APS had not been diagnosed before.

Although SLE is considered a female disease, it also affects men. For them, SLE is more difficult to diagnose. Males are less likely to have symptoms that suggest suspicion of SLE – malar rash, musculoskeletal involvement. Male SLE can be more severe, often with cardiovascular events [12]. Also, men with SLE have higher lupus anticoagulant

Table 1.
Test results showing exacerbation of systemic lupus erythematosus and antiphospholipid syndrome

Test	Result	Normal value
Anti- double-stranded DNA	209.8	<35
Anti-β-glycoprotein IgG, IgA, IgM	1.218	<0.316
Anti-cardiolipin IgG, IgA, IgM	0.867	<0.297
Extractable nuclear antigen (ENA)	positive	negative
Smith	positive	negative

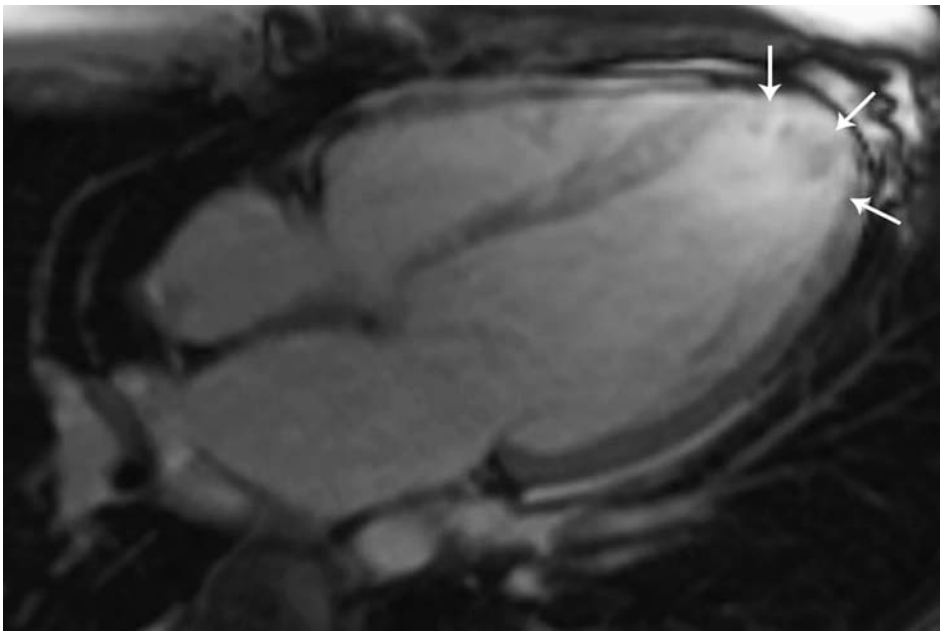


Figure 2. Magnetic resonance imaging of the heart: post-ischaemic scar lesions in the myocardium; thrombus at the apex of the left ventricle

levels and are more likely to develop thrombosis [13]. For these reasons, clinicians should be aware that SLE and its relapse can occur with unexpected complications and without warning signs in the men population.

In addition to the treatment above, it is appropriate to prescribe hydroxychloroquine to patients with SLE [14]. This drug is associated with lower thrombus formation and longer life expectancy. Also, studies show that this drug protects against vascular stiffness and early cardiovascular events [15]. Thus, treatment for SLE is necessary to reduce the risk of cardiovascular events [11].

Conclusion

This clinical case of a man in his forties showed that even in remission, the autoimmune inflammation might be present with thrombus formation and severe blood flow obstruction in vital organs, heart, in particular. The minimal dose of steroids and aspirin in remission might be considered as an appropriate measure to prevent thrombotic events.

References

[1] Giannelou M, Mavragani CP. Cardiovascular disease in systemic lupus erythematosus: a comprehensive update. J Autoimmun 2017;82:1–12.
[2] Fava A, Petri M. Systemic lupus erythematosus diagnosis and clinical management. J Autoimmun 2019;96:1–13.
[3] Lithuanian Ministry of Health, Health Information Centre of Institute of Hygiene. Number of registered cases by diagnosis in Lithuania in 2018. https://stat.hi.lt/default.aspx?report_id=169.
[4] Zeller CB, Appenzeller S. Cardiovascular disease in systemic lupus erythematosus: the role of traditional and lupus related risk factors. Curr Cardiol Rev 2008;4(2):116–22.
[5] Kreps A, Paltoo K, McFarlane I. Cardiac manifestations in systemic lupus erythematosus: a case report and review of the literature. Am J Med Case Rep 2018;6(9):180–3.
[6] Fosam H. Cardiovascular disease risk in patients with systemic lupus erythematosus. Rheumatology Advisor. Published online 2017.
[7] Soltész P, Szekanecz Z, Kiss E, Shoenfeld Y. Cardiac manifestations in antiphospholipid syndrome. Autoimmun Rev 2007;6:379–86.

[8] Esdaile JM, Abrahamowicz M, Grodzicky T, Li Y, Panaritis C, du Berger R et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 2001;44(10):2331–7.
[9] Ünlü O, Zuily S, Erkan D. The clinical significance of antiphospholipid antibodies in systemic lupus erythematosus. Eur J Rheumatol 2016;3(2):75–84.
[10] Del Papa N, Vaso N. Management of antiphospholipid syndrome. Ther Adv Musculoskelet Dis 2010;4(2):221–7.
[11] Skamra C, Ramsey-Goldman R. Management of cardiovascular complications in systemic lupus erythematosus. Int J Clin Rheumtol 2010;5(1):75–100.
[12] Murphy G, Isenberg D. Effect of gender on clinical presentation in systemic lupus erythematosus. Rheumatology (Oxford) 2013;52(12):2108–15.
[13] do Socorro Teixeira Moreira Almeida M, da Costa Arcoverde J, Barros Jacobino MN, Coimbra Neto AR. Male systemic lupus erythematosus, an overlooked diagnosis. Clin Pract 2011;1(4):e103.
[14] Buleu F, Sirbu E, Caraba A, Dragan S. Heart involvement in inflammatory rheumatic diseases: a systematic literature review. Medicina (Kaunas) 2019;55(6):249.
[15] Selzer F, Sutton-Tyrrell K, Fitzgerald S, Tracy R, Kuller L, Manzi S. Vascular stiffness in women with systemic lupus erythematosus. Hypertension 2001;37(4):1075–82.