

Acute myocarditis manifested by signs of ST – elevation acute coronary syndrome

H. P. Verezhnikova*^{1,2,A-E}, A. H. Hul^{1,B-E}, N. O. Lifantieva^{1,2,B,C}, O. Yo. Zharinov^{1,2,E,F}

¹Shupyk National Medical Academy of Postgraduate Education, Kyiv, Ukraine, ²Heart Institute, Ministry of Health of Ukraine, Kyiv

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

Key words:

clinical case, myocarditis, ST-segment elevation, acute coronary syndrome.

Zaporozhye medical journal 2019; 21 (3), 406–410

DOI: 10.14739/2310-1210.2019.3.169216

*E-mail: anna.verezhnikova@gmail.com

Ключевые слова:

клинический случай, элевация сегмента ST, острый коронарный синдром, миокардит.

Запорізький медичний журнал. – 2019. – Т. 21, № 3(114). – С. 406–410

Ключевые слова:

клинический случай, элевация сегмента ST, острый коронарный синдром, миокардит.

Запорожский медицинский журнал. – 2019. – Т. 21, № 3(114). – С. 406–410

Purpose – to share the experience of differential diagnosis and monitoring of patient with acute myocarditis that caused acute chest pain and persistent ST-segment elevation on electrocardiogram.

Materials and methods. Diagnosis and treatment of patient with acute chest pain and persistent ST-segment elevation was performed according to the protocols of the Health Ministry of Ukraine for the diagnosis and treatment of cardiovascular diseases.

Results. The article describes a case of the acute myocarditis in 33-year-old female with acute chest pain combined with ST-segment elevation on electrocardiogram and a rise in serum cardiac enzymes. The main data of anamnesis, objective, laboratory and instrumental examination of patient, 6-month follow-up results are presented.

Conclusions. The success of therapy and prognosis depends on timely diagnosis of the disease. A definitive diagnosis should be established taking into consideration clinical manifestations, anamnesis, ECG, laboratory tests, coronary angiography and dynamic patient monitoring results.

Гострий міокардит, що маніфестував ознаками гострого коронарного синдрому зі стійкою елевацією сегмента ST

Г. П. Вережнікова, А. Г. Гуль, Н. О. Ліфантьєва, О. Й. Жарінов

Мета роботи – описати досвід диференціальної діагностики та нагляду за пацієнтом із гострим міокардитом, що став причиною гострого болю за грудиною та стійкої елевації сегмента ST на електрокардіограмі.

Матеріали та методи. Діагностику, лікування пацієнта з гострим больовим синдромом і стійкою елевацією сегмента ST здійснили відповідно до протоколів Міністерства охорони здоров'я України з діагностики та лікування захворювань серцево-судинної системи.

Результати. Описано випадок міокардиту в жінки віком 33 роки з гострим болем за грудиною в поєднанні з елевацією ST-сегмента на електрокардіограмі та збільшенням серцевих ферментів у сироватці крові. Наведено основні дані анамнезу, об'єктивного, лабораторного, інструментального обстеження, а також результати спостереження через 6 місяців.

Висновки. Успіх терапії залежить від своєчасної діагностики захворювання. У пацієнтів із гострим больовим синдромом за грудиною та елевацією ST-сегмента на ЕКГ діагноз треба встановлювати, враховуючи клінічні прояви, дані анамнезу, електрокардіографії, лабораторних досліджень, коронароангіографії, результати динамічного спостереження.

Острый миокардит, манифестировавший признаками острого коронарного синдрома со стойкой элевацией сегмента ST

Г. П. Вережнікова, А. Г. Гуль, Н. А. Лифантьєва, О. И. Жаринов

Цель работы – поделиться опытом дифференциальной диагностики и последующего наблюдения за пациентом с острым миокардитом, который стал причиной острой боли за грудиной и стойкой элевации сегмента ST на электрокардиограмме.

Материалы и методы. Диагностику, лечение пациента с острым болевым синдромом и стойкой элевацией сегмента ST проводили в соответствии с протоколами Министерства здравоохранения Украины по диагностике и лечению заболеваний сердечно-сосудистой системы.

Результаты. Описан случай миокардита у женщины в возрасте 33 года с острой болью за грудиной в сочетании с элевацией ST-сегмента на электрокардиограмме и увеличением сердечных ферментов в сыворотке крови. Представлены основные данные анамнеза, объективного, лабораторного и инструментального обследования пациента, а также результаты наблюдения через 6 месяцев.

Выводы. Успех терапии зависит от своевременной диагностики заболевания. У пациентов с острым болевым синдромом за грудиной и элевацией ST-сегмента на ЭКГ диагноз должен устанавливаться с учетом клинических проявлений, анамнеза, электрокардиографии, лабораторных исследований, данных коронароангиографии, результатов динамического наблюдения.

Introduction

The acute chest pain combined with ST-segment elevation on electrocardiogram (ECG) and a rise in serum cardiac enzymes are the basic criteria for ST-elevation acute coronary syndrome (STE-ACS) [1]. However, in some cases, the ECG diagnosis of ACS remains questionable, because urgent coronary angiography doesn't show any stenosis [9]. A number of different cardiac and non-cardiac conditions, that may simulate ACS, have been described in the literature [4,6,8,10,11]. Pericarditis and myocarditis have the greatest occurrence among them [2,5]. ECG changes, such as ST-segment elevation or depression and abnormal T-wave morphologies, may mimic myocardial infarction or ischemia [3,6]. This may be confusing to physicians with ACS and myocarditis diagnosis.

Furthermore, cardiac enzymes elevation, abnormality of myocardial wall motion and contractile function deterioration are often found in myocarditis patients. These findings may be the cause of misdiagnosis and incorrect therapeutic decisions regarding the need for thrombolysis and/or percutaneous coronary intervention [2,10]. In this article, we report a case of a 33-year-old female, who was admitted to our hospital with acute chest pain and an episode of loss of consciousness with high suspicion of STE-ACS based on clinical symptoms, electrocardiogram and bedside echocardiography. The patient underwent immediate coronary angiography, which revealed normal coronary arteries. Myocarditis was finally diagnosed in the patient with ST-segment changes mimicking a myocardial infarction.

Purpose

To share the experience of differential diagnosis and monitoring patient with acute myocarditis which manifested by acute chest pain accompanied by persistent ST-segment elevation on electrocardiogram.

Materials and methods

Diagnosis and treatment of the patient with acute chest pain and persistent ST segment elevation was performed according to the protocols of the Ministry of Health of Ukraine for the diagnosis and treatment of cardiovascular diseases. Laboratory tests were performed using the hematology analyzers Sysmax XS – 500i and Sysmax XT – 1800, biochemical analyzers Beckman coulter AU 480, immunochemical analyzer Beckman coulter Access 2 and the original reagents. ECG was conducted on INNOMED HS80G-L1 machine. Philips CX-50 Diagnostic Ultrasound System was used for echocardiography and abdominal ultrasound. X-ray system "FLEXAVISION" (Chimanzu Corporation) was used for a chest X-ray. Coronarography was accomplished on "Acision Artis DBC" x-ray angiography system with flat detectors. MRI was fulfilled on the VANTAGETITAN 3T firm Toshiba Medical Systems Corporation (Japan) production.

Clinical Case

A 33-year-old female was admitted in our hospital with acute chest pain and ST-segment elevation on the ECG. The physical examination showed that her temperature

was 36.5 °C, respiratory rate 18 bpm, pulse 100 bpm and blood pressure was 100/70 mm Hg in both arms. The first and second heart sounds were muffled without any audible murmurs. Her skin and mucous membranes were clean and remanded normal during entire examination. The initial ECG showed (*Fig. 1*) low voltage in standard leads, sinus tachycardia 118 bpm and ST-segment elevation 1 mm in leads I, II, aVL, V4-V6, suggestive of myocardial ischemia. The bedside echocardiography showed global hypokinesia, left ventricular systolic dysfunction with ejection fraction (EF) 40 % and pericardial effusion about 0.4–0.6 cm.

It turned out that she had had febrile illness symptoms such as fever, malaise, and fatigue for 4 days. Her child had suffered from an acute respiratory infection about two weeks earlier. She visited a general practitioner and was prescribed with augmentin 625 mg, enisamium iodide 250 mg 2 times daily, aspirin in body temperature elevation. Her condition deteriorated rather than improved and she began to feel chest discomfort, shortness of breathing, severe weakness. She was having episodic acute chest pain and two syncopal episodes for the last two days and was hospitalized after the second one. She had no significant medical or surgical history, no hereditary cardiac disease, history of hypertension or diabetes mellitus. The patient had never smoked cigarettes or abused alcohol.

Emergency coronary angiography revealed no any obstruction of arteries, which was contrary to the diagnosis of myocardial infarction.

Laboratory data on admission showed white blood cells count of $7.9 \times 10^9/l$ and 13.0, 14.1, $10.7 \times 10^9/l$ in the following days, serum C-reactive protein of 47 mg/L, creatinine kinase of 592 IU/L (200 IU/L), lactate dehydrogenase of 242 IU/L, procalcitonin level 0.012ng/ml, troponin I of 3.38 ng/ml (reference value 0.0–0.3 ng/ml) followed by 5.83 ng/ml on day 3 and 4.34 ng/ml on day 5, D-dimer of 0,44 mcg FEU/ml (reference value 0.0–0.5 mcg FEU/ml) and total cholesterol of 5.53 mmol/l. All other laboratory tests were negative. A chest X-ray revealed slightly enlarged size of the heart and mild markings in the lungs, with no clear indications of substantive lesion. The ultrasound examination of the abdominal organs showed no changes.

Patient condition was stabilized by dopamine/dobutamine and furosemide. Simultaneously, angiotensin-converting enzyme (ACE) inhibitor, beta blocker carvedilol and torasemide were prescribed. On day 4, echocardiogram showed end diastolic volume 105 ml, ejection fraction (EF) 35 % with moderately reduced systolic function, an apical floating thrombus was suspected with the size of 1.3–1.4 × 0.5–0.8 cm and a mild pericardial effusion. Rivaroxaban 20 mg per day was prescribed. After 2 weeks of supportive treatment, echocardiogram showed EF 50 %, mild to moderate amount of pericardial effusion and magnetic resonance imaging (MRI) confirmed diffuse myocarditis with pericardial effusion of 0.5–0.7 cm (*Fig. 2*). Signs of acute myocarditis such as patchy areas of subepicardial and mesocardial layers with increased T2 signal intensity indicating regional edema of the left ventricular myocardium, myocardial MR signal intensity ratio of 2.8 (>2.3) compared to that of skeletal muscle were found. Homogeneous, circularly distributed and septated pericardial effusion up to 5–7 mm was revealed.

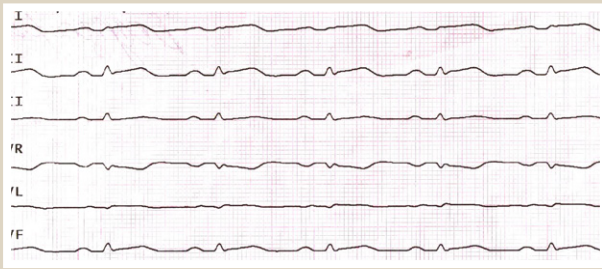


Fig. 1. ECG at admission.

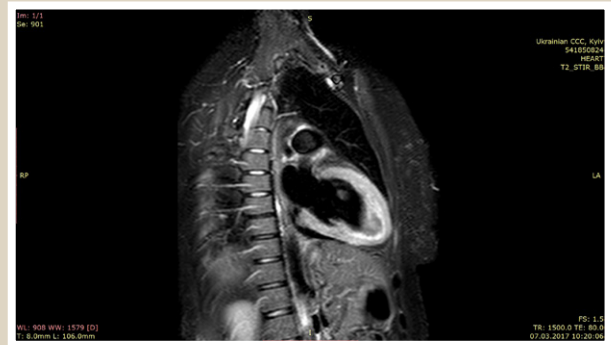
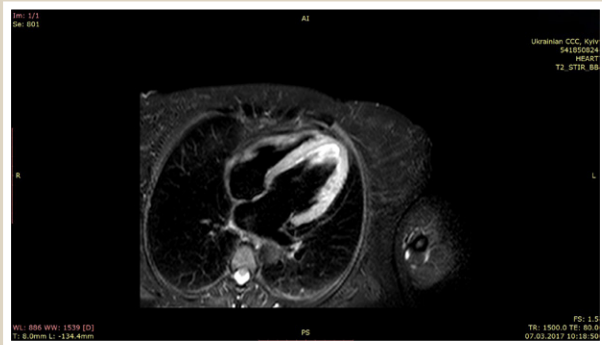


Fig. 2. MRI 2 weeks after the onset of symptoms.

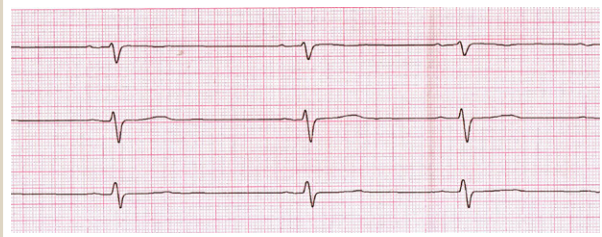
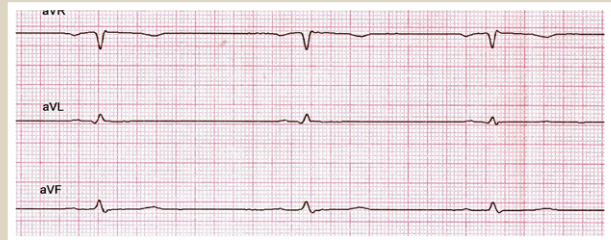
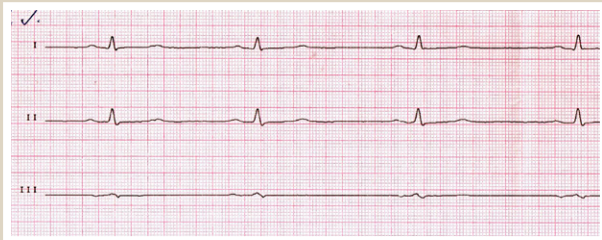


Fig. 3. ECG after 7 months.



Fig. 4. MRI after 7 months.

In a subsequent period, methylprednisolone 12 mg daily for one month was prescribed followed by a lower dose until complete withdrawal. The patient's condition was improved and she was discharged after 2 weeks with beta blockers, ACE inhibitors, spironolactone and furosemide. The patient was examined 10 days after discharge and she felt much better with improved condition. She was recommended to continue her therapy over the next 6 months.

The patient was followed for 7 months after the onset of disease. She had no complaints. Her state of health was satisfactory. We noted a slightly lowered QRS voltage and flat T-waves on the ECG (Fig. 3). Echocardiogram showed EF 61 %. Repeated MRI 7 months after the diagnosis confirmed that myocardial inflammation and effusion was resolved (Fig. 4). MR signs of the inflammatory process activity were not determined. Myocardiofibrosis was

supported by MR data such as patchy areas of delayed contrast enhancement in subepi-/mesocardial layers indicating regional injury. The systolic function of the left ventricle was preserved, the cavity was not dilated. Mild systolic dysfunction of the right ventricle was present.

Discussion

A number of different cardiac and non-cardiac conditions that may simulate ACS have been described in the modern literature. Some groups of diseases and conditions which manifest as acute chest pain, electrocardiographic pattern as STE-ACS or non-STE-ACS but without coronary arteries stenosis on angiography can be identified. This pattern can be present in patients with cardiac inflammatory diseases, such as pericarditis and myocarditis [3,5,12]. A group of patients with vascular pathology, such as pulmonary embolism, aortic dissection, coronary aneurysm and others can have STE-ACS – like presentation. Non-cardiac conditions, such as neurological pathology, noncardiovascular thoracic diseases and especially involving gastrointestinal tract may also manifest in an infarction-like manner [8,10]. A number of other cardiac pathologies, such as cardiomyopathies, including Takotsubo cardiomyopathy, congenital and acquired heart diseases, Prinzmetal angina, and inherited diseases can lead to myocardial infarction-like ECG changes. Brugada syndrome, Osborn syndrome, drug induced conditions, electrolyte disturbances as hyperkalemia and other states with certain symptoms may be misdiagnosed as STE-ACS [4,6,11,12].

According to the literature data, the acute myocarditis is diagnosed in 12–33 % of patients with STE-ACS and normal coronary arteries [2,5,6].

Acute myocarditis is an inflammatory disease of the myocardium with heterogeneous clinical manifestations and progression. It is characterized by myocyte necrosis and inflammatory infiltrate in the myocardium. Most often, myocarditis is caused by common viral infections; less common specific forms of myocarditis may result from other pathogens [3]. The host immune response may play an important role as well as direct cytolysis by viral infection. Myocarditis presents in many different ways, ranging from mild symptoms of chest pain and palpitation to life threatening cardiogenic shock and severe heart failure. Although a viral prodrome with fever, myalgia, and respiratory or gastrointestinal symptoms is usual, reported symptoms are highly variable. This variety of clinical images of myocarditis makes it challenging to diagnose and treat. Among 3055 patients with suspected acute or chronic myocarditis who were screened in the European Study of the Epidemiology and Treatment of Inflammatory Heart Disease, 72 % had dyspnea, 32 % had chest pain, and 18 % had arrhythmias [7].

In our case, patient presented on admission with chest pain, ST-segment elevation and elevated troponin I level similar to the presentation of STE-ACS. Several days before hospitalization, the patient had experienced malaise, fatigue, decreased exercise tolerance, palpitations. She had had episodic precordial chest pain and two syncope episodes over the last two days. She had been prescribed antiviral treatment because she had had sick contact before.

Nevertheless, there were signs that gave rise to suspicion of acute myocardial ischemia and thus STE-ACS could not be excluded.

The injury of myocytes causes disturbance of the electrical activity of the myocardium. Therefore ECG can show different abnormalities, including ST-T wave elevation or depression, atrial and ventricular arrhythmias, SA- and AV-block, intraventricular conduction defects and early repolarization syndrome [12]. T-wave changes are more typical. Concave ST elevation is more common, than convex. ST elevation is usually diffuse and detected in many leads at once without reciprocal changes. However, ECG signs are neither specific nor sensitive. In our case, the patient had sinus tachycardia 118 bpm and ST segment elevation in leads I, II, aVL, V4–V6. She experienced syncope twice, which could have been due to conduction disorders. Unfortunately, there was no evidence of it on the ECG in our patient on admission or in the hospital.

Echocardiography is a necessary tool to exclude non-inflammatory cardiac diseases such as valve disease and to monitor changes in cardiac chamber size, wall thickness, ventricular function and pericardial effusion. Global ventricular dysfunction, regional wall motion abnormalities and diastolic dysfunction with preserved ejection fraction may occur in myocarditis. In this case, the patient had moderately reduced left ventricle function and reduced ejection fraction. The role of newer imaging techniques such as tissue Doppler and strain rate imaging for myocarditis diagnosis remains to be determined.

Cardiovascular MRI provides non-invasive myocardial tissue characterization and can support the diagnosis of myocarditis. Myocardial edema in the acute phase of the lesion is manifested by the myocardium thickening, an increase in the intensity of the magnetic resonance signal from the affected segments, the accumulation of a contrast agent in the lesion sites at the early and delayed phases, volumes of extracellular and intracellular fluid. Pericardial effusion has been reported in 32–57 % of patients with myocarditis. Although it is not specific for myocarditis, its presence is supportive evidence for active inflammation [5]. In our case, MRI revealed signs of acute myocarditis and confirmed our assumptions regarding the diagnosis. Also, dynamic MRI enabled us to verify recovery of the patient from myocarditis.

Despite considerable progress in the diagnosis of myocarditis, it remains a challenge for physicians to conduct differential diagnosis between acute myocarditis and myocardial infarction in certain cases, particularly in the early phase. A differentiated approach to evaluation of data, including medical history, clinical presentation and results of other auxiliary tests are necessary for the accurate diagnosis of myocarditis and proper treatment. According to the current guidelines, percutaneous coronary intervention or immediate thrombolysis are recommended for patients with STE-ACS. The goal of urgent care of patient is to reduce time delay from the onset of ST-segment elevation myocardial infarction symptoms to the first medical contact and the diagnosis up to 10 minutes. And if a patient is triaged for a primary percutaneous coronary intervention strategy, it means that a patient should be transferred directly to the catheterization laboratory. If percutaneous coronary intervention cannot be performed within 120 min from ST-segment elevation

myocardial infarction diagnosis, the decision on thrombolysis should be made by the emergency doctor within 10 minutes after STE-ACS diagnosis [1]. In this case, the patient with viral myocarditis presented with retrosternal pain, elevated cardiac marker levels and ST-T changes on the ECG, similar to the clinical manifestations of acute myocardial infarction. Clinical manifestations, including a fever with temperature fluctuations, severe weakness, syncopal episodes, coronary angiography and the results of auxiliary examinations could aid in the differential diagnosis between acute myocarditis and myocardial infarction. The etiology, pathogenesis, diagnosis and therapy of myocarditis remain controversial. All patients should be followed up with clinical assessment, ECG, echocardiography and cardiovascular MRI, if necessary. In our reported case, patient underwent cardiovascular MRI along with other modalities to confirm recovery from myocarditis.

Conclusions

1. The diagnosis in patient with acute chest pain and persistent ST segment elevation on ECG should be made taking into consideration clinical manifestations, anamnesis, ECG, laboratory tests, results of dynamic patient monitoring with the exclusion of coronary artery disease by angiography.

2. The other additional instrumental methods of examination should be used, if necessary. In the acute period of the disease, imaging methods such as echocardiography are important for the early detection of myocardial contractility. In our case, cardiovascular MRI gave additional and supportive evidence to confirm myocarditis as the correct diagnosis.

Conflicts of interest: authors have no conflict of interest to declare.
Конфлікт інтересів: відсутній.

Надійшла до редакції / Received: 25.06.2018
Після доопрацювання / Revised: 03.07.2018
Прийнято до друку / Accepted: 28.08.2018

Information about authors:

Verezhnikova H. P., MD, PhD, Associate Professor, Department of Functional Diagnosis, Shupyk National Medical Academy of Postgraduate Education; Heart Institute of the Ministry of Health of Ukraine, Kyiv.

Hul A. H., MD, Resident Physician, Department of Functional Diagnosis, Shupyk National Medical Academy of Postgraduate Education, Kyiv, Ukraine.

Lifantieva N. O., MD, PhD, Assistant of the Department of Functional Diagnosis, Shupyk National Medical Academy of Postgraduate Education; Heart Institute of the Ministry of Health of Ukraine, Kyiv.

Zharinov O. Yu., MD, PhD, DSc, Professor, Department of Functional Diagnosis, Shupyk National Medical Academy of Postgraduate Education; Heart Institute of the Ministry of Health of Ukraine, Kyiv.

Відомості про авторів:

Вережнікова Г. П., канд. мед. наук, доцент каф. функціональної діагностики, Національна медична академія післядипломної освіти імені П. Л. Шупика, Інститут серця МОЗ України, м. Київ.
Гуль А. Г., клінічний ординатор, каф. функціональної діагностики, Національна медична академія післядипломної освіти імені П. Л. Шупика, м. Київ, Україна.

Ліфантьєва Н. А., канд. мед. наук, асистент каф. функціональної діагностики, Національна медична академія післядипломної освіти імені П. Л. Шупика; Інститут серця МОЗ України, м. Київ.
Жарінов О. Й., д-р мед. наук, професор, зав. каф. функціональної діагностики, Національна медична академія післядипломної освіти імені П. Л. Шупика; Інститут серця МОЗ України, м. Київ.

Сведения об авторах:

Вережнікова Г. П., канд. мед. наук, доцент каф. функциональной диагностики, Национальная медицинская академия последипломного образования имени П. Л. Шупика; Институт сердца МЗ Украины, г. Киев.

Гуль А. Г., клинический ординатор, каф. функциональной диагностики, Национальная медицинская академия последипломного образования имени П. Л. Шупика, г. Киев, Украина.

Лифантьева Н. А., канд. мед. наук, ассистент каф. функциональной диагностики, Национальная медицинская академия последипломного образования имени П. Л. Шупика; Институт сердца МЗ Украины, г. Киев.

Жаринов О. И., д-р мед. наук, профессор, зав. каф. функциональной диагностики, Национальная медицинская академия последипломного образования имени П. Л. Шупика, Институт сердца МЗ Украины, г. Киев.

References

- Ibanez, B., James, S., Agewall, S., Antunes, M. J., Bucciarelli-Ducci, C., Bueno, H., et al. (2018) 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology. *European Heart Journal*, 39(2), 119–177.
- Andersson, H. B., Pedersen, F., Engstrom, T., Helqvist, S., Jensen, M. K., Jorgensen, E., et al. (2018) Long-term survival and causes of death in patients with ST-elevation acute coronary syndrome without obstructive coronary artery disease. *European Heart Journal*, 39(2), 102–110. doi: 10.1093/eurheartj/ehx491
- Cooper, L. T. (2009) Myocarditis. *N. Engl. J. Med.*, 360(15), 1526–1538. doi: 10.1056/NEJMra0800028
- Coppola, G., Carità, P., Corrado, E., Borrelli, A., Rotolo, A., Guglielmo, M., et al. (2013) Italian Study Group of Cardiovascular Emergencies of the Italian Society of Cardiology. ST segment elevations: always a marker of acute myocardial infarction? *Indian Heart J.*, 65(4), 412–23. doi: 10.1016/j.ihj.2013.06.013
- Friedrich, M. G., Sechtem, U., Schulz-Menger, J., Holmvang, G., Alakija, P., Cooper, L. T., et al. (2009) International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J. Am. Coll. Cardiol.*, 53(17), 1475–1487. doi: 10.1016/j.jacc.2009.02.007
- Gu, Y. L., Svilaas, T., van der Horst, I. C. C., & Zijlstra, F. (2008) Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. *Netherlands Heart Journal*, 16(10), 325–331. doi: 10.1007/BF03086173
- Hufnagel, G., Pankuweit, S., Richter, A., Schönian, U., & Maisch, B. (2000) The European Study of Epidemiology and Treatment of Cardiac Inflammatory Diseases (ESETCID): first epidemiological results. *Herz*, 25(3), 279–285. doi: 10.1007/s000590050021
- Park, I., Kim, Y., Ahn, S., Sohn, C., Seo, D., & Kim, W. (2015) Subarachnoid hemorrhage mimicking ST-segment elevation myocardial infarction after return of spontaneous circulation. *Clinical and Experimental Emergency Medicine*, 2(4), 260–263. doi: 10.15441/ceem.15.012
- Schweitzer, P. (1990) The electrocardiographic diagnosis of acute myocardial infarction in the thrombolytic era. *Am. Heart J.*, 119, 642–654. doi: 10.1016/S0002-8703(05)80288-1
- Upadhyay, A., Chauhan, S., Jangda, U., Bodar, V., & Al-Chalabi, A. A. (2017) Reversible Inferolateral ST-Segment Elevation Associated with Small Bowel Obstruction. *Case Reports in Medicine*, 2017, 5982910. doi: 10.1155/2017/5982910
- Wang, K., Asinger, R. W., & Marriott, H. J. (2003) ST-segment elevation in conditions other than acute myocardial infarction. *N. Engl. J. Med.*, 349(22), 2128–35. doi: 10.1056/NEJMra022580
- Zhang, T., Miao, W., Wang, S., Wei, M., Su, G., & Li, Z. (2015) Acute myocarditis mimicking ST-elevation myocardial infarction: A case report and review of the literature. *Exp. Ther. Med.*, 10(2), 459–464. doi: 10.3892/etm.2015.2576