Časopis Udruženja kardiologa Srbije

SRCE i krvni sudovi

Heart and Blood Vessels

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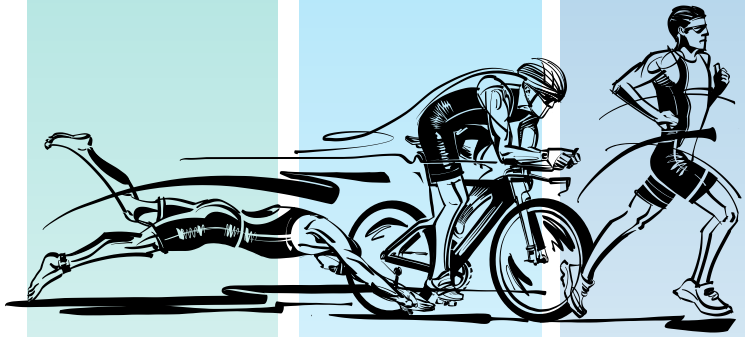

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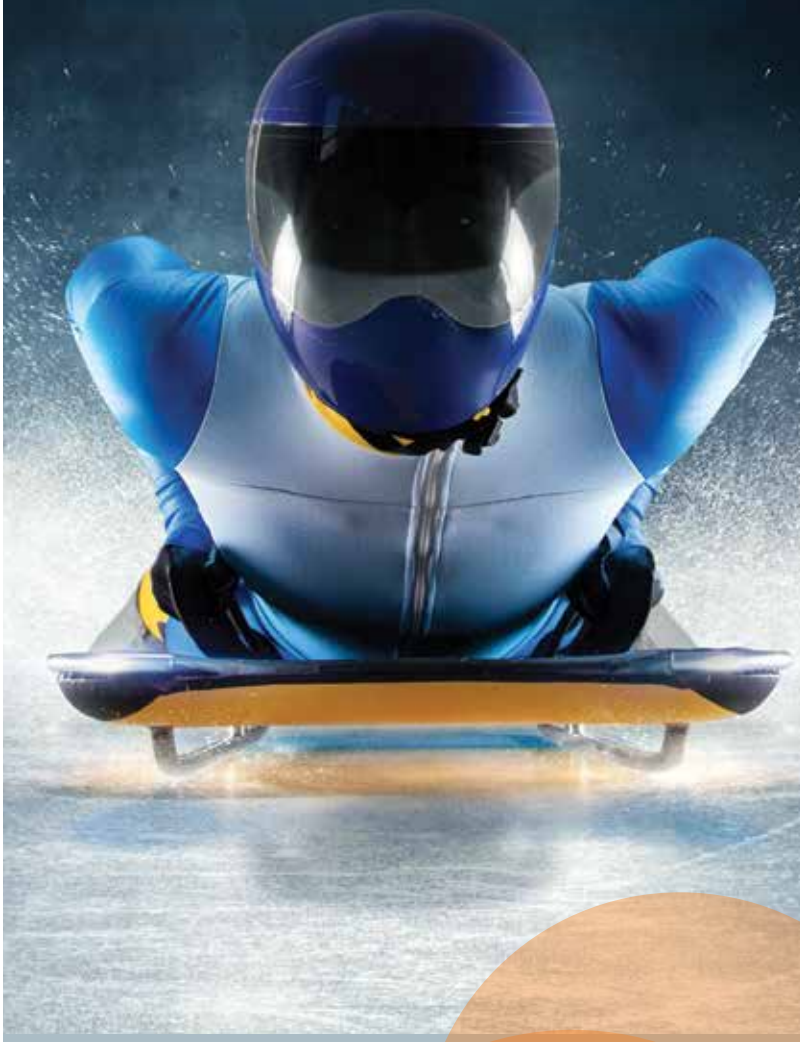

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Review article

Spontaneous coronary artery dissection: What we know so far?

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Abstract Spontaneous coronary artery dissection (SCAD) is one of the causes of acute coronary syndrome (ACS). Although it is difficult to differentiate SCAD from other causes of ACS based on the clinical presentation, some patient characteristics can aid the clinician in considering SCAD as a diagnosis. Namely, SCAD is far more common in women, especially in the peripartum period, and patients often do not have typical risk factors associated with atherosclerosis-related myocardial infarction. Furthermore, there is an association with FMD, migraine, and inherited connective tissue disorders. The primary pathophysiologic disturbance is the separation of layers of the coronary artery, either by hemorrhage from the vasa vasorum and formation of an intramural hematoma or by blood entering the vessel wall through an endothelial disruption thus creating a false lumen. The most common presentations are myocardial infarction, cardiogenic shock, and ventricular arrhythmias, while the most common presenting symptom is chest pain. The gold standard in diagnostics is coronary angiography. Angiography findings can be classified by the Yip-Saw classification into three categories, and the procedure should be approached with special attention due to the fragility of coronary arteries in these patients. Further imaging techniques that can be of use are OCT, IVUS, CTCA.

Initial management is directed toward treating the myocardial infarction and its complications. A significant portion of SCAD can be managed conservatively, and the PCI is used to maintain a grade 3 TIMI flow with minimal intervention. In case there are clinical or anatomical high-risk features present, the patient can be referred to CABG if it is available.

Other than managing the myocardial infarction, medical management plays a role in managing chronic chest pain, preventing recurrence of SCAD, treating extracoronary vascular disorders, and improving patients' quality of life. Patients with left ventricular dysfunction and patients after PCI are managed according to the current guidelines. Dual antiplatelet therapy is prescribed to all patients following PCI, in the acute phase of SCAD, and for up to 1 year to patients who are treated medically. After the first year, it is needed to tailor the treatment to each patient individually.

Aiming to preserve and improve the quality of patients' lives, they should receive regular follow-up and timely diagnosis and treatment for possible issues, which will, along with cardiovascular rehabilitation, aid in reaching this goal.

Kew words Spontaneous coronary artery dissection, acute coronary syndrome

Introduction

Spontaneous coronary artery dissection (SCAD) is one of the acute coronary syndrome causes. Once a rare phenomenon, SCAD is now a recognizable entity thanks to the use of coronary angiography and highly-sensitive troponins in the early diagnosis of acute coronary syndrome (ACS) and greater awareness of this disease. Although randomized controlled studies are still nonexistent, thanks to national SCAD registers from the last five years, a significant advance has been made in diagnosing and treating these patients[1]. This review paper will provide a revision of all the latest findings concerning the appropriate diagnostics, as well as the current therapeutic modalities.

Frequency and cause of SCAD

Although specific symptoms and signs that would help differentiate spontaneous dissection (SCAD) from other

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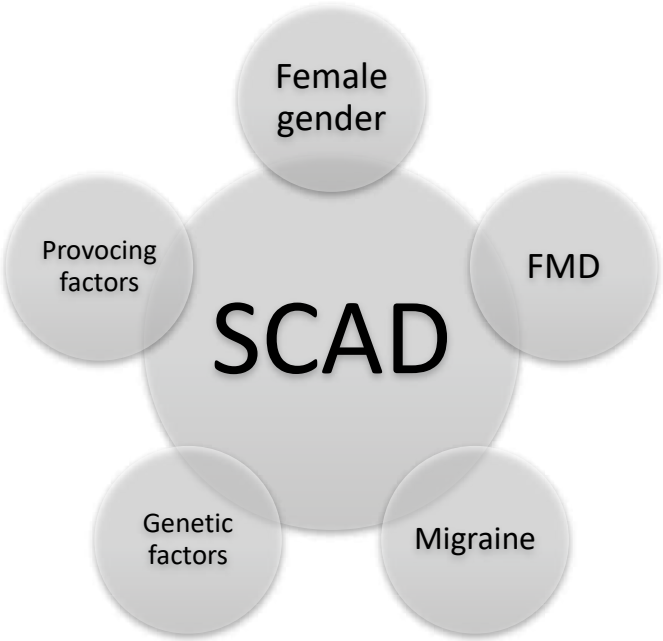

Figure 1. Causes of spontaneous dissection, including associated conditions. Provoking factors include emotional stress, certain medications, physical activity and Valsalva maneuver. SCAD (Spontaneous Dissection of Coronary Arteries); FMD (Fibromuscular Dysplasia).

common causes of acute coronary syndrome (ACS) do not exist, specific patient characteristics can direct us toward this diagnosis. A significantly lower prevalence of risk factors characteristic of the development of myocardial infarction associated with atherosclerosis was observed[2,3]. Hypertension is present in approximately 30% of SCAD patients, while the presence of diabetes is rare[4].

SCAD can present in both genders, with around 90% of patients being women aged 47-53[3,4,5,6], while approximately 10% of patients are men, as indicated in available literature[4,7,8]. It is a very rare occurrence in young patients (<25 years old) and older ones (>80 years old). It is a more common occurrence in patients suffering from hereditary connective tissue disorders or during pregnancy[9]. SCAD is responsible for around 23-68% of acute coronary syndromes during pregnancies, which is reflected as a common occurrence during the peripartum period[10,11]. Cohort studies have shown that SCAD might also appear in primiparous, multiparous, and pregnant women, in the postpartum and even postmenopausal period[2,4]. It remains to be determined whether the amount of estrogen and progesterone or their fluctuations have a role in the development of this entity[12]. It is assumed that estrogen's influence manifests itself through metalloproteinase activation, while progesterone's high level leads to weakening of the blood vessel wall[13].

Other provoking factors that could lead to SCAD development include strenuous physical activity in men and emotional distress in women[4,14,15]. The development of SCAD after using cocaine and after emesis was shown in various case reports[15,16,17].

Considering SCAD association with fibromuscular dysplasia (up to 50%), as well as migraine, the presence of these diseases in the patient's history can lead physicians toward SCAD[18]. Both clinical entities are more commonly seen in women. In addition, patients that suffer from connective tissue disorders (Marfan syndrome, Ehlers-Danlos, Loeys-Dietz), as well as Alport syndrome or adult polycystic kidney disease, or have a positive family history of these diseases, and experience signs and symptoms that resemble ACS, need to undergo a workup for SCAD. Gene sequencing showed that 3,5% of patients suffering from SCAD also suffer from one of the above-mentioned diseases[19].

Pathophysiology

The central pathophysiological event of spontaneous coronary artery dissection is the formation of a false lumen in the media that leads to separation of the coronary artery wall layers. As the name suggests, this disease is not associated with atherosclerosis, trauma, or iatrogenic injury but instead occurs spontaneously[1,18,21,22]. What divided SCAD into two of the largest groups is the presence of endothelial disruption and the formation of an intimal flap. Thus, the two hypotheses that explain the pathophysiology of this disease are proposed. The first is the so-called "inside-out" hypothesis, which suggests that blood enters the subintimal space through the endothelial disruption, thus creating the false lumen. However, back in 2002, with the help of intravascular ultrasound, an intramural hematoma without signs of endothelial disruption was noticed[23], leading to the creation of the second "outside-in" hypothesis that suggests that a hematoma can occur de novo, most likely as a consequence of microvasculature dysfunctions in the coronary artery wall[24,25,26]. After the initial development, ischemia and consequent necrosis due to luminal compression caused by axial propagation or the propagation of the intramural hematoma occur[1].

Clinical presentation

The clinical presentation of patients with SCAD is most similar to other causes of ACS. In over 90% of patients with SCAD, it manifests as a myocardial infarction (STEMI and NSTEMI)[4,8,27)], but its presentation can also be a ventricular arrhythmia (3-5%)[8,27-30] as well as cardiogenic shock (2%)[27]. By far, the most common symptom is chest pain, with the possibility of propagation to the neck, arms, or back, followed by dyspnea and sweating. Although certain factors can act as provocateurs for the appearance of symptoms (emotional stress, physical exertion), further differential-diagnostic measures must be taken with caution because these provoking factors are common for other clinical entities with a similar presentation (emotional stress – Takotsubo cardiomyopathy; physical exertion – atherosclerotic plaque rupture)[31,32].

Angiography

As patients with SCAD almost always show signs of ACS, the gold standard for diagnosing these patients is

Table 1. Characteristics of SCAD types[3]				
	Type I	**Type II**	**Type III**	**Type IV**
Angiographic appearance	Pathognomonic appearance of an arterial dissection with contrast entering the false lumen through the intimal flap; visible intimal flap	Narrowing of the lumen due to an intramural hematoma in abscence of an intimal tear; lesions usually long (>20mm) and characterized by sudden decrease in lumen size	Compression of the lumen by an intramural hematoma, but the lesions are usually <20mm, thus can resemble an atherosclerotic plaque	Complete oclussion of the coronary artery wall; may resemble thromboembolic oclussion
Frequency	29%	67%	4%	

Table 2. Differential diagnosis of SCAD	
Differential diagnosis	Comment
Atherosclerosis	May angiographically resemble SCAD types 1, 2 and 3. The presence of a thrombus distally to the stenosis, presence of calcifications or a lipid plaque lean toward the diagnosis of atherosclerosis.
Vasospasm	May resemble SCAD type 2; however, injecting intracoronary nitrates may confirm the diagnosis.
Embolization	Sudden oclussion may resemble SCAD type 4. Caution is needed during the differential diagnosis. The presence of an arteficial valve, proximal atherosclerotic plaque, coronary ectasis, shunt, hypercoagulable states lean toward the diagnosis of embolization.
Takotsubo cardiomyopathy	Apical akinesia is characteristic. Careful evaluation of terminal parts of LAD on angiography is needed.
Iatrogenic dissection	Rarely seen as an isolated phenomenon, usually traumatic in origin. It is well known that patients with SCAD have an increased tendency toward iatrogenic dissections (28).

coronary angiography. The dissection most commonly occurs in the left anterior descending artery (LAD), more specifically in the mid and distal segments. Multi-vessel SCAD appears in 5-13% of cases[1]. Angiographic findings can be classified into three groups according to the Yip-Saw classification (Table 1). However, certain classification deficiencies are appreciable as it encompasses only the most common angiographical characteristics. For that reason, some authors suggest a fourth group that would account for all the changes that do not fulfill the criteria for the first three, but lead to coronary artery occlusions[33].

A greater coronary artery tortuosity found during coronarography was observed in patients suffering from SCAD (78% as opposed to 17% with controlled subjects), and a clear coronary tortuosity can be used as a marker of recurrent SCAD[34]. Caution is needed when performing coronary angiography on patients with SCAD as their coronary artery walls are considered to be more fragile. Because of that, deep catheter cannulation should be avoided, and a careful injection of intravenous contrast is advised to avoid the risk of further propagation of the dissection.

Although the angiographic findings of SCAD are characteristic, there are certain challenging clinical scenarios in the sense of differential diagnosis that should be noted. Some of them are represented in Table 2.[35]

Post-angiography diagnostics (OCT, IVUS, CTCA)

These diagnostic techniques can be used when the diagnosis is not determined via an angiography[36,37]. Although superior, and with a better resolution, these diagnostic techniques carry the risk of certain complications, such as extensions of the current dissection, iatrogenic dissection, and false lumen cannulation — thus should be performed with additional caution. According to the current data, these complications can occur in 8% of patients with SCAD[38]. For this reason, routine use of these techniques is not recommended. However, if SCAD is suspected but its presence cannot be determined through an angiography, the use of these techniques is justified.

IVUS is superior compared to OCT. It enables deeper penetration and a complete assessment of the degree of blood vessel involvement[35]. A pathognomonic sign of SCAD is the so-called triple-band of the intimal-medial membrane. The limitation of this method is the inability to differentiate SCAD and lipid-rich atheromas due to its lower resolution (which is not the case with OCT)[35].

Despite the shallow penetration, OCT can confirm a SCAD diagnosis due to its excellent resolution and visualization of the real and false lumen, dissection flap, and intramural hematoma. During the intervention, closure of the flap is necessary to ensure favorable long-term results. An OCT is the only technique that can show whether, after stent placement and/or tracing, *restitutio ad integrum* of the coronary wall is present after spontaneous dissection[40].

The use of CCTA in the initial SCAD diagnosis is still a controversial topic. CCTA can be of use in the visualization of dissection flaps, intramural hematomas, and stenoses, especially in the proximal segments of coronary arteries[18,41]. However, its specificity and sensitivity are still unknown, and the few available case reports provide information about its false-negative results[42-44].

The primary role of this diagnostic technique can be in the follow-up of patients with SCAD, especially those with recurrent chest pain without evidence of

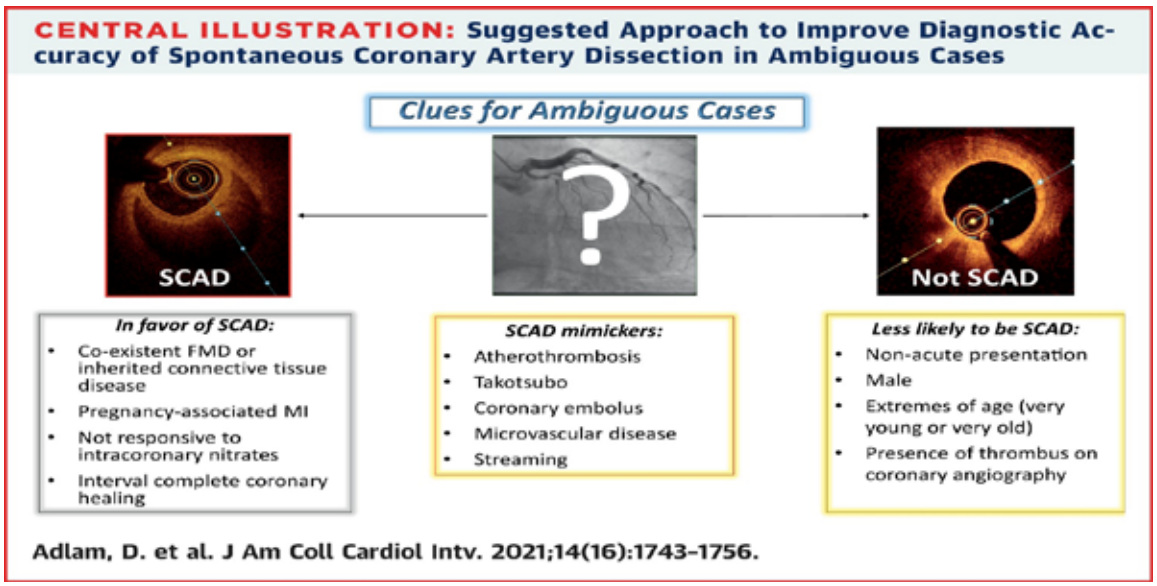

Figure 2. Recommended approach in unclear cases of SCAD. SCAD (Spontaneous Dissection of Coronary Arteries); FMD (Fibromuscular Dysplasia); MI (Myocardial Infarction). From: Adlam D, Tweet MS, Gulati R, Kotecha D, Rao P, Moss AJ, et al. Spontaneous coronary artery dissection. JACC: Cardiovascular Interventions. 2021;14[16]:1743–56.[35]

myocardial ischemia or necrosis. Nevertheless, more research and experience are needed in this direction[44]. In some cases, angiography and intracoronary imaging techniques can yield negative results while the presentation is still leaning toward SCAD. In such cases, some clinical and demographic characteristics can favor SCAD, and follow-up angiography (invasive or CCTA) can help discover the cause of acute coronary syndrome[1].

Therapy
Myocardial infarction treatment

Initial treatment of patients suffering from SCAD focuses on preserving or restoration of coronary blood flow and myocardial function. Thrombolytic therapy should be avoided, considering the possibility of dissection propagation or hematoma. Any decisions concerning the patient's treatment are made in regards to the clinical findings, such as the patient's hemodynamic stability along with the coronary angiography results which can show the extensiveness of the ischemic myocardium, as well as the degree of the distal flow in the affected blood vessel. High risk clinical characteristics are persistent chest pain with proof of ischemic changes, hemodynamic instability, shock, or clinically significant ventricular arrhythmias, whereas high risk anatomical features are more affected blood vessels with more significant proximal dissections or affected left coronary or LAD artery[1,18,45]. Due to a significant occurrence of SCAD in pregnant women, it is important to mention that any risk to the fetus is negligible with appropriate preventive measures; thus, the standard treatment of myocardial infractions is also recommended for pregnant women[46].
Due to pathophysiological differences in the mechanisms of SCAD and myocardial infractions due to atherosclerotic plaque rupture, along with the fact that all medically treated SCAD cases show an angiographically proven flow restitution with decreasing stenosis levels[7,47,48], the majority of patients suffering from SCAD can be conservatively treated, especially hemodynamically stable ones[1,18].

Percutaneous coronary intervention with SCAD

PCI outcomes in SCAD patients are less predictable and associated with a higher risk of complications compared to patients with atherosclerotic myocardial infarction[1,7,18,27], as well as a higher risk of iatrogenic dissections, sudden blood vessel occlusions, or hematoma propagation[49]. Therefore, when the SCAD diagnosis is confirmed, further instrumentalization is not necessary in case of minimal ischemia or distally affected coronary artery. The aim is to sustain TIMI flow grade 3 with minimal intervention, while revascularization is reserved for patients with STEMI, ongoing ischemia, proximal occlusions, unstable heart rate or hemodynamic status, heart failure, as well as those that progress to occlusion after the initial conservative treatment[18,24,50].
When high-risk features are present, it is necessary to consider whether the patient is a candidate for revascularization through PCI or coronary artery bypass grafting (CABG). It is important to emphasize that stent placement with a PCI procedure carries the risk of subsequent stent malposition due to resorption of the intramural hematoma over time[51].

Surgical myocardial revascularization in SCAD

This type of intervention is typically applicable to patients where the PCI procedure was unsuccessful or it was estimated that the risk would be too severe (for example, left coronary artery dissection with ongoing

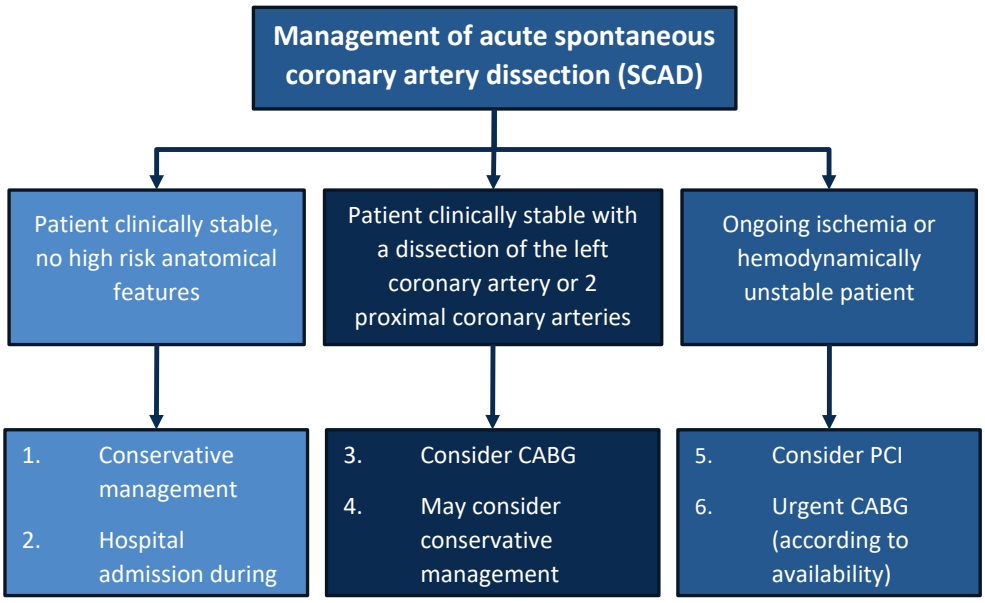

Figure 3. Management algorithm for SCAD. SCAD (Spontaneous Dissection of Coronary Arteries); CABG (Coronary Artery Bypass Grafting); PCI (Percutaneous Coronary Intervention) From: Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, et al. Spontaneous coronary artery dissection. Journal of the American College of Cardiology. 2020;76[8]:961–84.[26]

infarction). The procedure could be technically demanding due to the fragility of the tissue affected by the dissection, especially in patients with hereditary connective tissue disorders. Although this procedure's success rate is high, long-term maintenance of graft patency is not common[7] due to the recanalization of the coronary artery and consequent competitive flow that leads to graft occlusion. For this reason, using venous grafts is recommended for this type of intervention, with the aim of preserving arterial grafts for possible future uses[18].
Patients with severe forms of the disease, including cardiogenic shock and acute myocardial infarction complications due to SCAD, can benefit from measures of advanced mechanical circulatory support with intra-aortic balloon pumps or devices for extracorporeal membrane oxygenation until the patient becomes stable or receives a heart transplant[52,53].

Medical management of acute coronary syndrome caused by SCAD

Except for its role in the treatment of acute myocardial infarction, drug therapy has a role in treating chronic chest pain, preventing SCAD recurrence, treating extracoronary vascular disorders, and improving patients' quality of life.
Patients with left ventricular dysfunction are treated according to current recommendations for treating heart failure (beta-blockers and ACE inhibitors or angiotensin receptor blockers)[18,54]; whereas, patients who underwent a stent placement receive therapy in accordance with recommendations for treatment after PCI[55]. It is also necessary to treat hypertension as it is associated with recurrent SCAD[28].
According to the latest recommendations for the treatment of acute myocardial infarction[55,56], anticoagulant and dual antiplatelet therapy are commonly prescribed to patients before being diagnosed with SCAD. However, based on expert consensus, anticoagulant therapy should be ceased upon diagnosing SCAD on angiography[1,18]. The use of thrombolytic therapy for treating acute SCAD is not recommended[1].
Dual antiplatelet therapy is prescribed to all patients following PCI[55,56]. Grounded on expert recommendations, this therapy can be used during the acute phase of SCAD**,** as well as during the following year in patients treated conservatively[18]. Nevertheless, a recent study demonstrated a significantly higher frequency of major adverse cardiovascular events (MACE) in conservatively treated patients that were on dual antiplatelet therapy compared to those prescribed a single antiplatelet medication. As a result, further research and possible reconsideration of this therapeutic modality is necessary[57].
Due to insufficient evidence, the length of antiplatelet therapy after the first year should be adjusted according to individual patient characteristics. For instance, aspirin could be prescribed to patients with FMD to prevent thrombotic and thromboembolic complications. On the other hand, perimenopausal women suffering from menorrhagia could benefit from a shorter duration of antiplatelet therapy in lack of other indications for this treatment[58].
No evidence favors routine use of statins after SCAD, leading to this medication being reserved for patients with hyperlipidemia. As for the antianginal medications, only nitrates, calcium channel blockers, and ranolazine can be considered[18]. To prevent SCAD recurrence, beta-blockers can be used as there are indications of their benefits in this regard[28]. Factors related to recurrent SCAD are hypertension[28], FMD, migraine[3], and coronary artery tortuosity[34]. For the purpose of preventing SCAD recurrence, patients are recommended avoiding

strenuous physical activity, along with activities that involve the prolonged Valsalva maneuver[1,18].
To preserve and improve the patients' quality of life, special attention should be given to the signs and symptoms of migraines, anxiety, depression, and post-traumatic stress disorder, which are common occurrences after SCAD and significantly influence the quality of life[60]. That being said, screening with the goal of early detection of these conditions and treating patients can tremendously affect the patient's recovery. Cardiovascular rehabilitation is also of importance and is recommended for all patients following SCAD events[18].

Conclusion

SCAD is a rare but significant cause of acute myocardial infarction (AMI), which affects women more frequently. It differs pathophysiologically from the acute myocardial infarction associated with atherosclerosis, and the basis of acute coronary artery stenosis is a hematoma rather than a thrombus. It is important to think about this cause of AMI, especially in women under the age of 55, who are without risk factors for atherosclerotic disease. Myocardial revascularization is the treatment of choice in case of proximal coronary artery occlusion, ongoing ischemia, or cardiogenic shock. Drug treatment is preferred over percutaneous intervention. Patients with SCAD-induced AMI who are treated conservatively, primarily with short-term anticoagulation therapy and a single antithrombotic agent as a long-term treatment, had a better prognosis, according to recent observational studies. In over 85% of SCAD patients treated with medication, control angiography after 30 days shows spontaneous healing of the coronary artery.

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Sažetak

Spontana disekcija koronarnih arterija: Šta znamo do sada?

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Spontana disekcija koronarne arterije (SCAD) je jedan od uzroka akutnog koronarnog sindroma (AKS). Iako je teško razlikovati SCAD od drugih uzroka AKS na osnovu kliničke slike, neke karakteristike pacijenata mogu pomoći kliničaru da SCAD razmotri kao dijagnozu. Naime, SCAD je daleko češći kod žena, posebno u peripartalnom periodu, a pacijentkinje često nemaju tipične faktore rizika povezane sa infarktom miokarda izazvanom aterosklerozom. Štaviše, postoji povezanost sa FMD, migrenom i naslednim poremećajima vezivnog tkiva.

Primarni patofiziološki poremećaj je odvajanje slojeva koronarne arterije, bilo krvarenjem iz vasa vasorum i formiranjem intramuralnog hematoma ili ulaskom krvi u zid suda kroz endotelni poremećaj stvarajući lažni lumen.

Najčešći simptomi su infarkt miokarda, kardiogeni šok i ventrikularne aritmije, dok je najčešći simptom bol u grudima. Zlatni standard u dijagnostici je koronarna angiografija. Nalaz angiografije se prema Iip-Sav klasifikaciji može klasifikovati u tri kategorije, a proceduri treba pristupiti sa posebnom pažnjom zbog krhkosti koronarnih arterija kod ovih pacijenata. Dodatne tehnike snimanja koje mogu biti od koristi su OCT, IVUS, CTCA.
Početni tretman je usmeren na lečenje infarkta miokarda i njegovih komplikacija. Značajnim delom SCAD-a može se upravljati konzervativno, a PCI se koristi za održavanje protoka TIMI stepena 3 uz minimalnu intervenciju. U slučaju da postoje kliničke ili anatomske karakteristike visokog rizika, pacijent se može uputiti na CABG ako je dostupan.
Osim lečenja infarkta miokarda, posebnu ulogu ima i lečenje hroničnog bola u grudima, sprečavanju ponavljanja SCAD-a, lečenju ekstrakoronarnih vaskularnih poremećaja i poboljšanju kvaliteta života pacijenata. Pacijenti sa disfunkcijom leve komore i pacijenti posle PCI leče se u skladu sa važećim smernicama. Dualna antitrombocitna terapija se propisuje svim pacijentima nakon PCI, u akutnoj fazi SCAD-a i do 1 godine pacijentima koji se leče medicinski. Nakon prve godine potrebno je prilagoditi tretman svakom pacijentu pojedinačno.
U cilju očuvanja i poboljšanja kvaliteta života pacijenata, pacijenti treba da budu redovno praćeni i blagovremeno dijagnostikovani što će, uz kardiovaskularnu rehabilitaciju, pomoći u postizanju ovog cilja.

Ključne reči: Spontana disekcija koronarnih arterija, akutni koronarni sindrom

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Review article

Initial assessment of chest pain

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Abstract **The aim** of this paper is to show the procedure with the patient presenting with anginal chest pain during every day clinical routine.

Out of all patient presenting with chest pain, only 5.1% are with acute coronary syndrome (ACS), while half of the patients does not have a cardiac cause of the pain. So, we need to distinguish cardiac pain, or we shall make an emphasis on ischemic, from other forms. This pain judged by the patient description can be dubbed Typical anginal pain, or on the other hand what it was called atypical anginal pain, or so called "pain equivalent" e.g. like dyspnea. The pre-test probability (PTP) assessment is done based on: 1. The circumstances of pain onset (like physical exertion or emotional stress). 2. Localization of the pain (retrosternal) 3. Circumstances of pain cessation (rest or nitroglycerin intake). Based on these three characteristics we are judging the type of the pain. So, if we have only one criterion-the pain is **not anginal pain**., 2 criteria is dubbed like so called **atypical anginal pain.** And if we have all three presents, then this pain is called **typical anginal pain**. On the other hand, the numeric value of pre-test probability puts the patient into low, intermediate or high-risk group for coronary artery disease (CAD). So, the patients from the low-risk group can be excluded from further investigation. Intermediate group patients are pushed towards non invasive tests for CAD (physical exercise test, physical or pharmacological imaging tests (stress echocardiography, myocardial perfusion imaging). The patients from the high-risk group, high likelihood of CAD can be directly referred to invasive angiographic imaging. Based on the chest pain and ECG changes it is necessary to differentiate, whether we have a patient with acute coronary syndrome in our office, which would require urgent hospital treatment, or it is a patient with chronic coronary syndrome which would not require urgent treatment. Modern concept of chronic coronary syndrome diagnostic is characterized by diagnostic algorithm which is actually based on the above mentioned pre-test probability for CAD likelihood.

Kew words chest pain, pre-test probability, coronary artery disease

Introduction

Chest pain is a common symptom which patient are presenting with. As mentioned before, out of all patients presenting with chest pain to various services, only 5.1% have acute coronary syndrome, while half of them does not have a cardiac cause of the pain at all (1). So it is necessary to distinguish cardiac-ischemic pain from other types of it.

The characteristics of cardiac ischemic (anginal) pain are: 1. type 2. Localization with propagation characteristics 3. The duration of the pain 4. The type of onset 5. The type of cessation. So, based on those, the pain can be characterized as cardiac (ischemic, or as it was used to be called typical) and chest pain equivalent (as it was used to be called atypical) for CAD.[1]

Clinical presentation of typical anginal pain is represented by its characteristics: 1. Type, like squeezing, pressure, throbbing, heaviness, burning. The patient describes it as "dull" with no relation to movement or position. 2. Localization and propagation of the pain, retrosternal, left sided, larger area affected, propagating to the inner portion of left upper arm, elbow and lower arm, but also to lower jaw, to the back (interscapular), left scapula, sometimes to the both arms, epigastrically. 3. Duration of the pain, from couple of minutes, and up to 20 minutes (in differentiation to other types of pain which last couple of seconds, or on the other hand several hours on end). 4. Pain onset, usually is provoked by physical exertion, mental stress, cold air intake, large meal. The pain has an upstream tendency with physical exertion. 5. Pain cessation, after rest, or after nitroglycerin intake[2].

Clinical presentation of atypical anginal pain or pain equivalent, is usually differentiated in pain type, with its equivalents, like feeling of air loss, squeezing in lower jaw or neck, dyspeptic symptoms (heartburn, burping)[2]. This atypical of CAD presentation is to be expected

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in older people, women, diabetics, demented patients, and patients with chronic kidney failure (3).
The aim of this paper is to show the procedure with the patient presenting with anginal chest pain during every day clinical routine.

Discussion

When we are presented with a patient complaining of chest pain, we need to make some first contact assessments for patients with acute coronary syndrome (need urgent hospitalization and treatment) or chronic coronary syndrome (out hospital diagnostics and treatment CAD):
The character of the chest pain
- Anginal pain
- Non anginal pain

The pain characterized like heaviness, pressure, squeezing, tension, burning, positioned behind the sternum, centrally, provoked by exertion or stress point to HIGH clinical likelihood of anginal chest pain. On the other hand, sharp, migrating, respiratory movement, or rib cage movement related, pointed pain, usually points to LOW clinical likelihood of anginal chest pain (1).
Pain duration:
- Short - up 20 minutes
- Long - over 20 minutes

20 minutes makes a critical demarcation between short and long pain duration, so inf the pain shows the abovementioned characteristics of anginal chest pain, and lasts over 20 minutes, this patient usually requires hospitalization and observation. If the duration is under 20 minutes, and pain subsided to the moment of the exam, this patient rarely requires hospitalization but this patients stay into diagnostics algorithm.
ECG picture
- ST elevation
- ST depression or negative T waves
- Newly diagnosed left bundle branch block
- No ST and T changes

Further diagnostic work-up
- Troponin levels (highly sensitive preferably)
- Echocardiography
- Coronary angiography-invasive

Additional non-invasive work up is not necessary with patients presenting with all the above anginal pain characteristics, followed by ECG changes. This work-up is of the essence with patients presenting with non-ischemic pain characteristics in order to rule in or rule out acute coronary syndrome or other non-ischemic cardiac conditions (aortic dissection, acute pericarditis, valvular heart disease, hypertrophic cardiomyopathy), or on the other hand non cardiac conditions (esophageal spasm, pneumothorax, muscle spasm etc.)[1].

<u>Emergencies which require urgent hospitalization and treatment patients with ACS:</u>
Emergencies with hospital admission and treatment of CAD in patients presenting with typical chest pain over 20 minutes followed by ECG changes. Here we have several possible scenarios:

1. **Typical chest pain over 20 minutes duration accompanied by new ST elevation or new left bundle branch block**
This scenario requires urgent treatment according to STEMI guidelines with urgent pPCI or fibrinolytic therapy if pPCI is not available[4].

2. **Typical chest pain over 20 minutes duration accompanied by new ST depression or T wave changes**
This scenario requires urgent treatment according to NSTEMI/UA guidelines, low risk vs high risk patients, meaning urgent invasive strategy vs medical therapy and then invasive strategy during hospitalization[3].

3. **Typical chest pain over 20 minutes duration not accompanied by ECG changes**
These patients require additional diagnostic work-up, with hsTni levels (1 or 3hr rule in/rule out protocol if feasible, or hospitalization and observation for rule in/out differentiation, regarding local feasibility), echocardiography for other cardiac and non-cardiac causes. We should point-out that this population of patients is not small, meaning that almost 30% of NSTEMI patients do not present with ECG changes during first medical contact[3].

<u>Conditions which require diagnostic and therapy work-up of chronic coronary syndrome</u>
1. **Typical chest pain under 20 minutes duration, accompanied by new ST depression or T wave changes**
hsTNI levels with ACS rule in/rule out. Ruled out patients are referred to further non or invasive CAD risk assessment.
2. **Typical/atypical chest pain or equivalent, under 20 minutes duration not accompanied by ECG changes**
This where pre-test likelihood of CAD comes into place, low likelihood patients are ruled out of further CAD work up, intermediate to high likelihood patients are refereed to further non or invasive CAD work up.
For PTP assessment we need only three chest pain characteristics:
- Pain onset factors (exertion, mental stress)
- Pain localization (retrosternal)
- Pain cessation (rest, or nitroglycerin uptake)

So, we use them to judge about the presumed pain origin:
- One criterion -**Non anginal pain**
- Two criteria – **atypical anginal pain**
- All three criteria-**typical anginal pain**[2]

Diamond i Forrester[5], showed in their original work in 1979. that simple clinical and anamnestic work up can yield very useful and reliable data for CAD likelihood assessment in patients with chest pain. The showed on 4952 patients after invasive angiography that CAD likelihood is dependent on chest pain characteristics. Patients that had chest pain with ischemic characteristics, had CAD proven by invasive angiography in 90% of total number. On the other hand, those percent were 50% and 16% respectively for pain equivalent and non-cardiac group ($p<0,001$). The other part of their research were autopsy findings from 23996 adults who were not diagnosed with CAD during their lives. They showed with high significance that in women under the age of 40, CAD was present only in 0.3% of the patients, while

the number in men of the same age was 1.9%. In men over 60 years of age this number was 12.3%, and 7.5% for women of that age. Combining these two (angiography and autopsy) the made charts with pre test likelihood of CAD by the age, gender, and chest pain characteristics.

After this study, another study was published in 1981, CASS (Coronary Artery Surgery Study)[6]. This study was done on 20391 patients, divided in groups by age, sex, and chest pain characteristics, where presence of CAD was proven by angiography. In their words, 93% of men, and 72% of women with "definitive anginal pain" had a CAD on angiography, 66% of men, and 36% of women with "probable anginal pain", and 14% of men and 6% of women with "nonspecific pain" (p<0,001). They concluded that age, sex, and pain characteristics are important determinants in CAD prevalence and severity assessment. Regarding that the data on CAD prevalence in relation to pain, sex, age was similar in both studies, the are combined in one table (Table 1)

Table 1. Pre-test likelihood of CAD in percent in symptomatic patients, related to type of chest pain, sex, age. Combined data from Diamond/Forrester i CASS-Coronary Artery Surgery Study[2]

Age (years)	Non anginal pain		Atypical anginal pain		Typical anginal pain	
	M (%)	Ž(%)	M(%)	Ž(%)	M(%)	Ž(%)
30-39	4	2	34	12	76	26
40-49	13	3	51	22	87	55
50-59	20	7	65	31	93	73
60-69	27	14	72	51	94	86

Patients with low pretest probability for CAD, <20%, with intermediate pretest probability, 20-80%, high pretest probability, >80%[2].

Based on numeric values of pre-test probability, we put the patient in one of three groups, low, intermediate or high-risk group for CAD. The patients from low-risk group could safely be excluded from further CAD diagnostic work-up, patients from intermediate are referred to further non-invasive work-up (exercise test, stress echo, myocardial perfusion imaging). Patient in high-risk group can be directly referred to invasive strategy.

This concept of calculating pre test probability of CAD likelihood based on anamnestic data Is kept in recent ESC guidelines for chronic coronary syndrome[7]. Genders et al[8] updated these data on CAD prevalence from Diamond and Forester. They modeled previous CAD likelihood numbers according to new data, for patients in low-risk geographical areas, where it's good to know that these numbers can vary for different regions7. Pretest probability of CAD table (Table 2), is now changed by adding separate data for patients aged 70 and over. As another add on, a data on dyspnea like chest pain equivalent are added.

Patients with low pre test probability of CAD <5%, intermediate 5-15%, high >15% [7]. Out of all patients with chest pain or equivalent, 57% had pre-test pretest probability of under 15%. Studies showed that annual risk of cardiovascular death or myocardial infarction is under 1%, so it is safe to adapt routine testing of these patients to local resources, diagnostic test availability and clinical judgment (9). Patients with pretest likelihood of under 5% should be excluded from further CAD diagnostic algorithm, with data showing a larger number of false positives in this group (7).

Table 2. Pre test likelihood of CAD in percent in symptomatic patients in relation to chest pain characteristics, gender, age and dyspnea like chest pain equivalent[7]

Age (years)	Non anginal pain		Atypical anginal pain		Non anginal pain		Dyspnea	
	M (%)	Ž(%)	M(%)	Ž(%)	M(%)	Ž(%)	M(%)	Ž(%)
30-39	3	5	4	3	1	1	0	3
40-49	22	10	10	6	3	2	12	3
50-59	32	13	17	6	11	3	20	9
60-69	44	16	26	11	22	6	27	14
70+	52	27	34	19	24	10	32	12

Based on pre-test probability tables, CAD diagnostics in non anginal chest pain should be done only in patients aged 60 and above. In patients with pain equivalents this border is moved from to 40 and above for both sexes, to 50 and above for men, and 70 and above for women. Patients with ischemic chest pain were diagnosed regardless of age and sex, now this limit is set to 40 and above for men and 60 and above for women.

CONCLUSION

Based on chest pain and ECG characteristics, we need to assess whether we have a patient with acute coronary syndrome, which require urgent therapy and hospitalization, or a patient with chronic coronary syndrome which does not require such urgent procedures. This modern concept of chronic coronary syndrome envelops gradual approach of diagnostic algorithm which is based pretest probability of coronary artery disease.

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Sažetak

Inicijalna procena bola u grudima

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CILJ rada *je prikaz postupka sa pacijentima sa bolom u grudima, u svakodnevnom kliničkom radu.*
*Od svih pacijenata sa bolom u grudima koji se javljaju u službama urgentnog prijema, samo 5,1% ima akutni koronarni sindrom (AKS), a polovina pacijenata nema kardiološki uzrok bola. Zato je neophodno razlikovati srčani, ishemijski bol od drugih. Anginozni bol prema opisu pacijenta može biti tipičan i atipičan za koronarnu bolest. Procena pre-test verovatnoće (PTV) vrši se na osnovu: 1. okolnosti nastanka bola (na napor ili psihički stres), 2. lokalizacije bola (retrosternalni), 3. okolnosti prestanka bola (odmor ili upotreba NTG) . Na osnvou ove tri karakteritike određuje se tipičnost bola: Prisustvo 1 kriterijuma-***bol nije anginozni****; Prisustvo 2 kriterijuma-***atipična angina***, *Prisustvo sva 3 kriterijuma-***tipična angina.** *Na osnovu numeričke vrednosti pre-test verovatnoće pacijent se procenjuje sa niskom verovatnoćom, srednjom verovatnoćom ili sa visokom verovatnoćom za postojanje koronarne bolesti. Pacijenti sa niskom verovatnoćom za postojanje Kkoronarne bolesti mogu se bezbedno isključiti iz dalje dijagnostike. Pacijenti sa srednjom verovatnoćom neinvazivnu dijagnostičku obradu za dokazivanje koronarne bolesti: test fizičkim opterećenjem, fizički ili farmakološki imaging testovi (stres ehokardiografija, stres-rest perfuziona scintigrafija miokarda). Pacijenti sa visokom verovatnoćom za postojanje koronarne bolesti mogu se direktno uputiti na invazivnu dijagnostiku (koronarografiju). Na osnovu bola u grudima i EKG promena potrebno je proceniti da li se radi o pacijentu sa akutnim koronarnim sindromom, koji zahtevaju hitnu hospitalizaciju i terapiju, ili o pacijentu sa hroničnim koronarnim sidromom. Savremeni koncept dijagnostike hronične koronarne bolesti podrazumeva etapnu primenu dijagnostičkog algoritma u čijoj je osnovi upravo vednost pre-test verovatnoće za postojanje koronarne bolesti.*

Ključne reči: bol u grudima, pre-test verovatnoća, koronarna bolest

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Case report

Multivessel coronary artery disease – how to reach Heart Team's optimal decision

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Abstract We present a case of a 71-year-old gentleman who suffered an inferoposterior ST elevation myocardial infarction treated with thrombolytic therapy where coronary angiography showed triple vessel disease and coronary artery bypass graft (CABG) was suggested. Based on the results of coronary physiology and non-significant values of FFR, Heart team denied revascularization and recommended medical therapy. Data from 11 randomized studies involving more than 11,000 patients comparing PCI to CABG in patients with multivessel disease (MVD) showed pronounced benefit of surgical revascularization in patients with more complex coronary anatomy (higher SYNTAX score) and diabetes. If there is a complex MVD with left main stenosis, the advantage should be given to CABG, as well as in the case of complex MVD and diabetes, while PCI has an advantage in patients with lower SYNTAX score, the ones with advanced age and comorbidities where PCI would offer faster postprocedural recovery. In retrospective analysis by Basman et al., patients subjected to hybrid revascularization techniques had similar mortality as patients that underwent CABG or multivessel PCI and similar incidence of composite outcomes.

Kew words coronary artery disease, PCI, CABG

Case presentation

We present a case of a 71-year-old gentleman who suffered an inferoposterior ST elevation myocardial infarction that was treated with thrombolytic therapy. Regarding risk factors for atherosclerosis, he used to smoke and received medication for hypertension and dyslipidemia. Coronary angiography was done 18 days after STEMI and a triple vessel disease was seen and coronary artery bypass graft (CABG) was suggested. At admission, he reported occasional chest pain (CCS Class II) and had moderately limited activity (NYHA II). He denies any other illnesses. The echocardiogram showed slightly reduced left ventricular systolic function of with ejection fraction of 45% and akinesia of the basal half of the septum and inferior wall, hypokinesia of the basal half of the posterior wall.

Calculated Synergy Between PCI With Taxus and Cardiac Surgery (SYNTAX) score was 14 and SYNTAX II score predicted equipoise between percutaneous coronary intervention (PCI) and CABG-with 4-year mortality of 6.0% and 7.1% respectively (Figure 1).

Given the low SYNTAX score and previous MI, we opted for coronary physiology – an invasive examination of the significance of narrowing by measuring the fractional flow reserve (FFR), coronary flow reserves (CFR) and index of microcirculatory resistance (IMR) by using coronary wire for pressure and temperature measurement

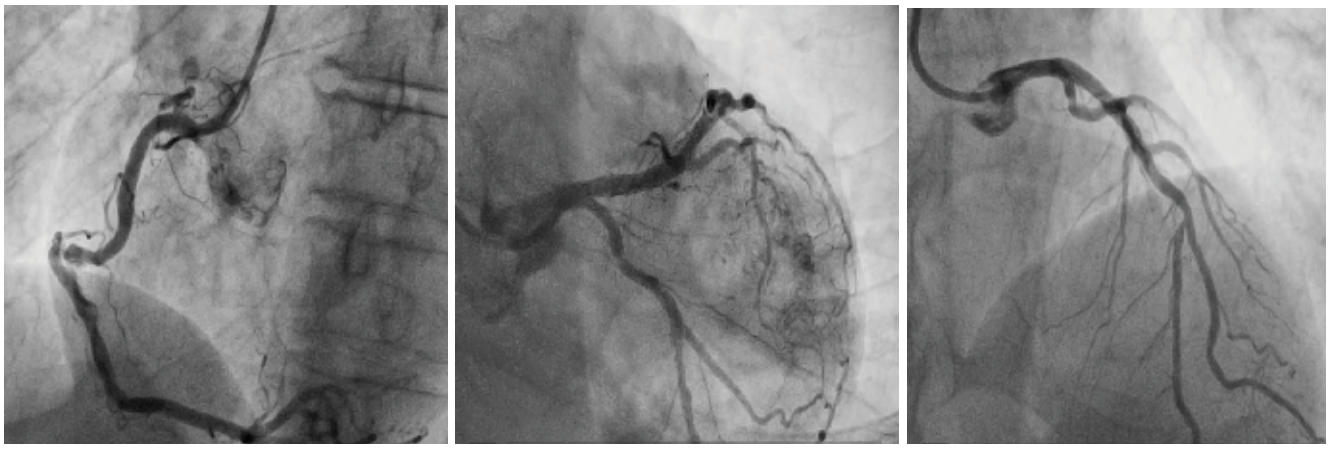

Figure 1. Coronary angiography which was used to suggest CABG

Figure 2. Measured coronary physiology parameters in RCA, Cx and LAD

Pressure Wire X (Abbott Vascular, Plymouth, MN, US) and continuous adenosine infusion. The acquired values were as follows: right coronary artery (RCA) FFR 0.84, CFR 1.3, IMR 24.8; circumflex artery (Cx) - FFR 0.95, CFR 2.6, IMR 18.3 and left anterior descending (LAD) FFR 0.90, CFR 1.9, IMR 10.9 (Figure 2).

Based on the results of coronary physiology and non-significant values of FFR with low CFR and borderline IMR values that indicated presence of microvascular dysfunction without significant narrowing of the epicardial coronary arteries, Heart team denied any form of revascularization and recommended maximum medical therapy.

Discussion

Multivessel coronary artery disease (MVD) and adequate method of revascularization has been a matter of debate in the cardiology community for more than 20 years[1]. The concept of "Heart Team" (HT) was established for multidisciplinary approach to individual patient in order to reach an evidence-based decision regarding the appropriate treatment. In case of a patient with coronary artery disease (CAD), the role of HT is to adequately evaluate anatomical complexity of coronary disease, the possibility of complete revascularization, the assessment of surgical risk of procedure and possible complications of any method of revascularization. The aim of the assessment is to determine the risk – benefit ratio in terms of procedural risks (risk of death, myocardial infarction, heart failure) and periprocedural complications (cerebrovascular event, kidney failure, complications at puncture site, need for transfusion, new onset arrhythmia or wound infection) versus possible benefits in terms of prolongation of life, absence of myocardial infarction and improvement in quality of life.

The superiority of surgical treatment in case of a patient with MVD has been repeatedly examined in relation to PCI with the development of advanced techniques in interventional cardiology[3]. In daily practice, despite the development in interventional techniques – new generations of drug eluting stents (DES), intravascular imaging (IVUS, OCT), use of coronary physiology (CFR, FFR) and advanced antithrombotic therapies in patients with MVD, the advantage is almost always given to surgical treatment. Current recommendations from European Society of Cardiologists and the European Association of Cardiothoracic Surgeons give absolute advantage to surgical treatment in patients with anatomically complex MVD (high SYNTAX score), while in the intermediate complexity of coronary disease (SYNTAX<22) outcomes, except for myocardial infarction, are similar. If the patient suffers from diabetes, the things are slightly different. Aggregated data from 11 randomized studies involving more than 11,000 patients and comparing PCI to CABG in patients with MVD showed lower five-year mortality rates in those treated with surgical revascularization compared to those treated with PCI (11.5% after PCI to 8.9% after CABG; HR 1. 28, 95% CI 1. 09-1·49;p=0. 0019), including diabetes patients (15. 5% *vs.* 10. 0%; 1. Forty-eight, one. 19-1. 84; p=0. 0004), but not in patients who did not suffer from diabetes (8.7% *vs* 8.0%; 1.08, 0.86–1.36; p=0.49). Additionally, the benefit of surgical revascularization was more pronounced in patients with more complex coronary anatomy (higher SYNTAX score)[4].

A special attention should be devoted to a patient with significant stenosis of left main stem. Previously, available data from randomized studies and meta-analyses showed similar results regarding death, myocardial infarction and repeated revascularization if patients underwent CABG or PCI with DES stent[2,5]. The aforementioned meta-analysis demonstrated similar five-year mortality (10.7% after PCI *vs* 10.5% after CABG; p=0·52)[4]. In patients with complex coronary anatomy and LM stenosis, despite fewer patients with these characteristics in randomized studies, surgical treatment would probably be the best treatment option[2,4].

Previous research in this field has been designed before significant advances in interventional cardiology like development of coronary physiology and imaging techniques, which means that, in previously published randomized trials, not many patients had these techniques applied during PCI. Therefore, when choosing revascularization techniques in MVD, complete revascularization of all hemodynamically significant lesions should be sought, either based on anatomical or functional significance obtained using non-invasive or invasive tests to prove it (CFR and FFR)[6].

An interesting alternative is a hybrid approach - combining the surgical revascularization with LIMA (left internal mammary artery) graft and PCI of other lesions in pa-

tients with MVD. In retrospective analysis by Basman et al., after propensity matching, patients subjected to hybrid revascularization techniques had similar mortality as patients that underwent CABG or multivessel PCI (5.0% vs. 4.0% vs. 9.0%) and similar incidence of composite outcomes - death, repeated revascularization and myocardial infarction (HCR 21.0% vs CABG 15.0%, P = .36; HCR 21.0% vs PCI 25.0%, P = .60). Despite higher preprocedural SYNTAX score, hybrid revascularization achieved a lower residual score after revascularization than multivessel PCI (7).

Finally, approach to a patient with MVD should be individualized primarily based on the anatomy of coronary artery lesions. If there is a complex MVD with LM stenosis, the advantage should be given to CABG, as well as in the case of multivessel complex MVD and diabetes. In the absence of these characteristics, the severity of CAD should be analyzed while paying respect to patient's preference. The advantage to surgery should be given in patients with very complex lesions (long lesions, chronic total occlusions, calcifications) where percutaneous complete revascularization would be difficult to achieve, while PCI has an advantage in patients of advanced age, with high risk of cerebrovascular complications, bleeding and infection of the surgical wound, as well as in those where functional capacity is reduced where PCI would offer faster postprocedural recovery.

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Sažetak

Višesudovna koronarna bolest – kako doći do optimalne odluke "Heart Team-a"

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Predstavili smo slučaj muškarca starog 71 godinu koji je lečen trombolitičkom terapijom nakon akutnog infarkta sa ST elevacijom. Koronarografija je pokazala trosudovnu koronarnu bolest I predloženo je hirurško lečenje bajpas graftom (CABG). Urađena je koronarna fiziologija i na osnovu neznačajnih vrednosti frakcione rezerve protoka (FFR) "Heart team" je indikovao maksimalnu medikamentnu terapiju. Podaci iz 11 studija koje su uključile preko 11000 pacijenata pokazali su korist od CABG-a kod pacijenata sa kompleksnom koronarnom anatomijom (visok SYNTAX skor) i dijabetesom, dok perkutana koronarna intervencija (PCI) ima prednost kod pacijenata sa nižim SYNTAX skorom, starijih sa komorbiditetima, gde PCI nudi brži postproceduralni oporavak. U retrospektivnoj analizi Basmana i saradnika pokazano je da hibridni metod revaskularizacije ima sličnu incidenciju smrti i kompozitnih ishoda kao CABG i višesudovna PCI.

Ključne reči: koronarna bolest, CABG, PCI

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Case report

Cerebral T wave on ECG in a patient with stroke

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Abstract Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke. It is important to recognize the cerebral T wave, differentiate it from other disorders presenting with negative T waves, and thus avoid the pitfall of acute coronary syndrome misdiagnosis, and wrong therapy selection (i.e. fibrinolytic, anticoagulant and antithrombotic), which would be very dangerous for a patient with cerebral hemorrhage. The female patient, 84 years old, was hospitalized due to a hip fracture to the orthopedic department of the Health center Zajecar hospital. Her past medical history was remarkable for treated arterial hypertension and paroxysmal atrial fibrillation. She was hemodynamically stable and asymptomatic. As part of the preoperative management, an internist consultation was sought due to newly developed ECG abnormalities: sinus rhythm, HR of 65/min, left bundle branch block (LBBB) and negative T waves of 5mm in the precordial leads. There were echocardiographic wall motion abnormalities of the left ventricle. Hs troponin level was 278 ng/ml and acute coronary syndrome was suspected. The patient was started on ACE inhibitor, beta-blocker, statin and dual antiplatelet therapy. The patient develops right-sided muscle weakness. Brain CT was done and the zone of left low temporoccipital acute ischemia was described. The patient gave the informed consent for coronary angiography this time and after stabilization it was performed. The angiographic findings were normal. Consequently, with optimized medical therapy, she was returned to orthopedics for further orthopedic care.

Key words cerebral T wave, ischemic stroke

Introduction

Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke [1]. It was first described by George Burche in 1954[2]. It is important to recognize the cerebral T wave, differentiate it from other disorders presenting with negative T waves, and thus avoid the pitfall of acute coronary syndrome misdiagnosis, and wrong therapy selection (i.e. fibrinolytic, anticoagulant and antithrombotic), which would be very dangerous for a patient with cerebral hemorrhage.The pathophysiology is incompletely understood[3]. In various articles numerous potential explanations exist. *Sakamoto et al* believe that focal myocytolysis occurs due to excessive stimulation of sympathetic centers in the hypothalamus which leads to the release of catecholamines that can lead to myocardial damage, either by direct toxic effects or by causing constriction of myocardial microcirculation leading to focal ischemia[4]. *Baroldi et al* think that focal myocardial lesions are caused by the release of catecholamines from the intramyocardial nerve endings and not from the circulation[5]. Some of the authors believe that the ECG changes that occur in an acute cerebral event are actually a consequence of injury or stimulation of the islet cortex, which has been proven to have cardiovascular effects on stimulation[6,7]. The belief that cerebral T waves are neuron-induced is supported by the observation that negative T waves can be normalized if brain death occurs.
ECG abnormalities seen in patients with elevated intracranial pressure are: cerebral T wave, QT interval prolongation and bradycardia, indicating brainstem herniation - Cushing's reflex. Far less often abnormalities on the ECG are: increased U wave amplitude, arrhythmias, as well as ST segment elevations or depressions that can mimic pericarditis or myocardial ischemia. ECG changes, in case of increased intracranial pressure, most often occur in large intracranial hemorrhages, namely: in subarachnoid hemorrhage, intraparenchymal hemorrhage. Also it occurs in large ischemic stroke which leads to cerebral edema. Then there are traumas as well as tumors and metastases in the brain as etiologies[8]. In the case of our patient, a negative T wave is the result of temporooccipital ischemia. According to a study by *Goldstein et al* in 150 consecutive patients with acute stroke, cerebral T wave occurs in as many as 29%[9].

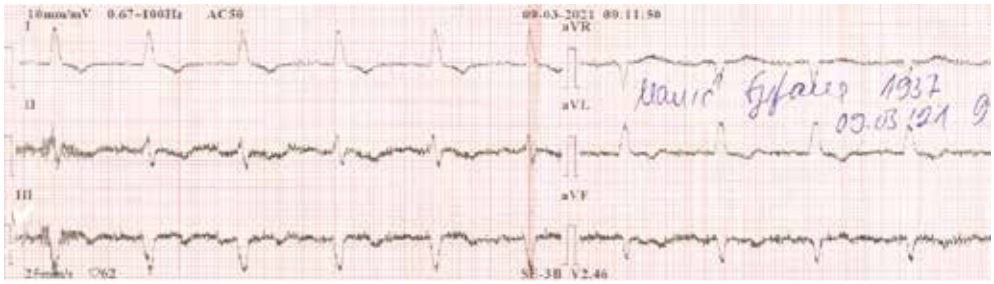

Figure 1a. ECG tracing of the patient during the hospital stay - standard leads

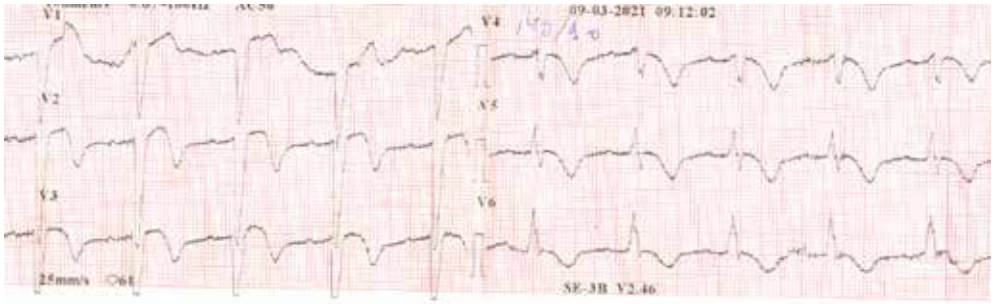

Figure 1b. ECG tracing of the patient during the hospital stay - precordial leads

Case report

Female patient, 84 years old, was admitted to the orthopedics department on March 8, 2021 due to a left hip fracture. Surgical treatment with the partial hip arthroplasty was indicated. At the presentation, in addition to left leg and hip pain, she doesn't have other complaints. Past medical history is remarkable for arterial hypertension and paroxismal atrial fibrillation. She takes an ACE inhibitor for her hypertension.

On physical exam the patient is conscious, oriented, afebrile, eupneic. Preoperatively an internist consultation was requested on March 9, 2021. The patient is without any complaints. Lung and heart examination is unremarkable. TA is 130/80 mmHg. On ECG there was the sinus rhythm, normal axis, HR 75 bpm, LBBB, negative T waves precordially (not described on previous ECG tracing and figured as novel finding - figures 1a, 1b). As part of further assessment, the cardiospecific enzymes and echocardiographic examination were requested.

On March 10, 2021, an ultrasound examination of the heart was performed, where the LV ejection fraction was estimated at 44% with apicoseptal and distal anterior akinesia. Diastolic dysfunction by type of delayed relaxation was observed. Aortic root dimension was 2.9 cm, left ventricle was dilated 5.6 cm diastolic and 4.3 cm systolic dimension. Laboratory results were as follows: hs troponin 278 ng/mL and BNP 179 pg/mL. A working diagnosis of acute coronary syndrome is made and dual antiplatelet therapy, low molecular weight heparin, beta blocker and statin are introduced. Coronary angiography was indicated, but the patient refused invasive diagnostic workup.

On the next day, March 11th 2021. weakness of the right extremities develops, a neurologist is consulted, and a brain CT scan (left low temporooccipital zone of acute ischemia) was performed, and she was transferred to the neurology department, where treatment of ischemic stroke was continued (Figure 2).

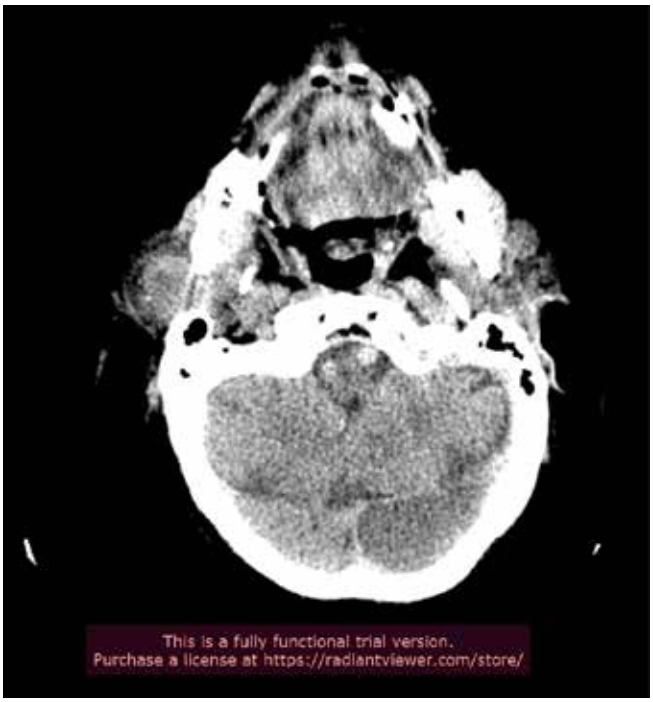

Figure 2. CT scan of the brain: left low temporooccipital zone acute ischemia (ischemic stroke)

The same day ECG (March 11th 2021) showed: sinus rhythm, LBBB, HR 65 bpm, -T to 5mm in D1, aVL, V4-V6 (figure 3).

After clinical stabilization, a decision is made to transfer her to the internal ward for the purpose of performing coronary angiography,for which this time the patient gave the informed consent. This was done in order to later perform surgical treatment of the hip fracture in the orthopedics. Patient is without any complaints, hemodynamically stable, transferred to the internal ward. ECG on admission: sinus rhythm, LBBB, HR 90 bpm, -T of 5mm in D1,aVL.

Coronary angiography was performed on March 18th 2021. There are no angiographically significant stenoses

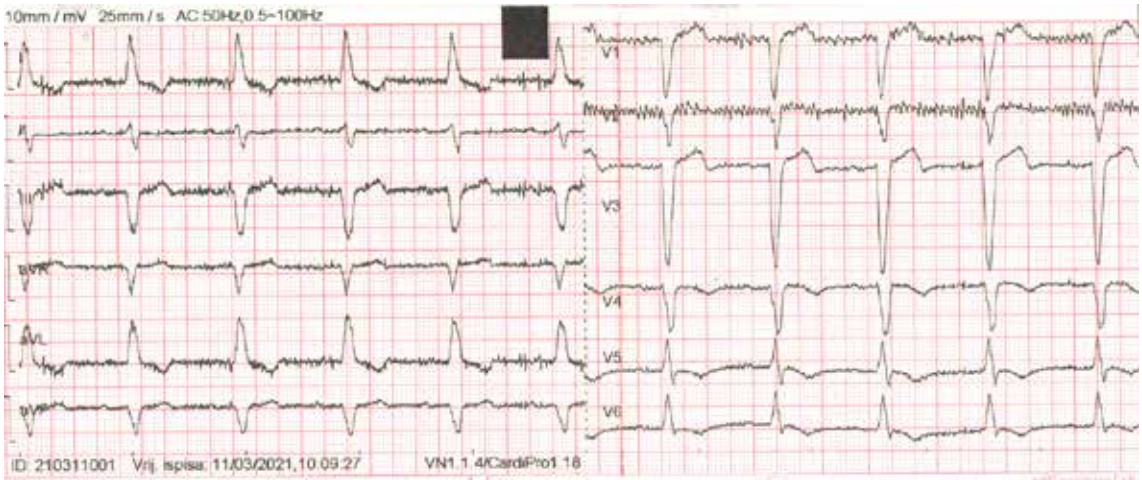

Figure 3. ECG during admission to the neurology department (March 11th 2021)

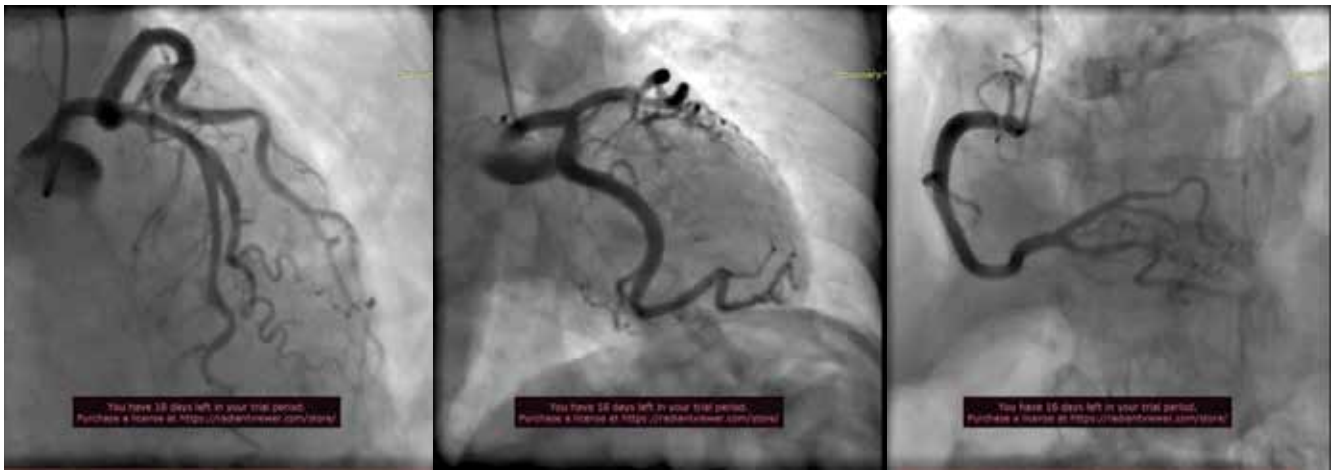

Figure 4. Coronary angiography was performed on March 18th 2021.

on the epicardial coronary arteries that would be a contraindication for the planned orthopedic procedure - left hip arthroplasty (figure 4)

The patient was transferred to the orthopedic department for planned left hip arthroplasty. Therapy on internal ward discharge: bisoprolol 1x5mg, aspirin 1x75mg, pantoprazol 1x20mg, atorvastatin 1x40mg. An anesthesiologist was consulted at the orthopedic department, who concludes that she is currently inoperative. The procedure is contraindicated and the patient is discharged and referred for further treatment to the Internal Department of OB Knjaževac. Therapy on discharge is as follows: bisoprolol 1x5mg, aspirin 1x75mg, pantoprazol 1x20mg, atorvastatin 1x40mg, enoxaparine 40mg o.d. s.c.

Discussion

Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke[1]. The differential diagnosis of a negative T wave on a 12-lead ECG includes: ischemia and myocardial infarction, ventricular hypertrophy, pulmonary embolism, hypertrophic cardiomyopathy, bundle branch block, and increased intracranial pressure.Its frequency seems to be much higher than it is usually thought. And thus is the importance of indicating it and proper diagnosis. According to the early study, in 150 consecutive patients with acute stroke, cerebral T wave occurs in as many as 29%[9].

It is very important to emphasize that ECG changes during acute stroke can mimic those in acute coronary syndrome. This can pose a risk of misdiagnosis of STEMI in a patient with intracranial hemorrhage and to consequently lead to the wrong fibrinolytic therapy. Therefore, it is necessary to differentially think about the possibility of increased intracranial pressure.

Our patient was suspected of having the acute coronary syndrome, based on ECG changes of the newly formed block of the left branch of the His bundle, with deep negative T waves and wall motion abnormalities of the left ventricle on echocardiography with slightly elevated cardiac specific enzymes. It should be noted that there is literature which tested the hypothesis that cerebral T waves are associated with transient cardiac dysfunction. The study included patients diagnosed with hemorrhagic or ischemic brain strokes. The ECG was monitored for cerebral T which was defined as the inversion of a T wave depth ≥5 mm in ≥4 precordial leads. An echocardiographic examination was performed for left ventricular wall motion abnormality. Of all 800 patients, 17 had cerebral T wave (2,1%). Of all patient with cerebral T wave, 14 (82%) had normal wall motion, while only 3 had transient wall motion abnormalities (18%). Of these patients, two had cardiomyopathy similar to

Takotsubo cardiomyopathy, and one globally decreased left ventricular function. Coronary angiography was performed in these patients that showed no significant stenoses to explain left ventricular dysfunction[3].
Some authors believe that the changes on the ECG withdraw very quickly, in proportion to the normalization of intracranial pressure. So most ECG changes will withdraw in 3 days, although they can last up to 8 weeks from the withdrawal of intracranial pressure.
In our case, coronary angography was of great importance for the correct diagnosis and thus therapy, in addition to markers of cardiac necrosis and echocardiography, which is also emphasized in similar works[10].

Conclusion

It is important to emphasize that ECG changes, within a stroke, can mimic changes in acute coronary syndrome. It is important to recognize the cerebral T wave, to differentiate it from other negative T waves etiologies, and thus avoid the pitfall of misdiagnosis of acute coronary syndrome, and thus wrong therapy (ie fibrinolytic, anticoagulant and antithrombotic therapy) which would be dangerous for the patient with cerebral hemorrhage. In this case, the importance of coronary angiography as a diagnostic tool for making a correct diagnosis should be emphasized.

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Sažetak

Cerebralni T talas na EKG-u kod pacijenta sa moždanim udarom

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Cerebralni T talas predstavlja prolazni, duboki, simetrični, negativni T talas na elektrokardiogramu (EKG) kod pacijenata sa moždanim udarom. Važno je prepoznati cerebralni T talas, diferencijalno dijagnostički ga razlikovati od ostalih poremećaja praćenih negativnim T talasom, i na taj način izbeći zamku pogrešne dijagnoze akutnog koronarnog sindroma, a samim tim i primenu pogrešne terapije (npr. fibrinolitičke, antikoagulantne i antitrombocitne), što bi za bolesnika sa moždanom hemoragijom bilo jako opasno. Pacijentkinja starosti 84 godina, hospitalizovana je zbog preloma vrata butne kosti, na ortopedsko odeljenje Zdravstvenog centra u Zaječaru. Od ranije boluje od arteriske hipertenzije i povremenih epizoda atrijalne fibrilacije. Hemodinamski je stabilna, bez subjektivnih tegoba. U okviru preoperativne pripreme, pregledana je od interniste zbog novonastalih EKG promena: sinusni ritam, normogram, sf 65/min, blok leve grane Hissovog snopa (BLG), negativan T talas preko 5mm u prekordijalnim odvodima. Ehokardiografski su viđeni segmentni ispadi u kinetici zidova leve komore.hs troponin je bio 278ng/mL pa je postavljena radna dijagnoza akutnog koronarnog sindroma. Uvedena je terapija ACE inhibitorom, beta blokatorom, statinom i dvojna antitrombocitna terapija. Kod pacijentkinje dolazi do razvoja desnostrane mišićne slabosti. Urađen je CT endokranijuma na kome je opisana zona akutne ishemije nisko temporokcipitalno levo. Nakon kliničke stabilizacije, urađena je koronarografija, sa kojom je ovog puta pacijentkinja saglasna. Koronarni angiogram bio je uredan. Sledstveno, pacijentkinja je, uz optimizaciju medikamentne terapije, vraćena na ortopediju radi daljeg ortopedskog zbrinjavanja.

Ključne reči: cerebralni T talas; ishemijski moždani udar

Srce i krvni sudovi 2022; 41(1): 24-27

Case report

Effects of cardiac rehabilitation, diet and exercise on peak aerobic capacity in obiese patient

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Abstract Obesity is a highly prevalent chronic condition among young population associated with significant morbidity and mortality including increased risk for developing cardiovascular diseases. Cardiac rehabilitation (CR) involving diet and exercise represent basic for improvement of functional capacity and lost of vait in obese patients.

We presented the case of a young obese patient who underwent guided diet and cardiovascular rehabilitation with significant improvement on peak aerobic capacity. At beginning of process our patient was obese with body-mass index of 30.1 kg/m^2, his waste circumference was 110 cm. Laboratory analyses showed elevated total cholesterol 5.8 mmol/L and impaired glicemic control with hemoglobin A1c (HbA1c) 6.0 %. The echocardiography was completely normal. Cardiopulmonary test was performed before guided rehabilitation showing very pour functional abilities with peak oxygen consumption (peak VO2) 32,0 ml/kg/min, at middle of rehabilitation with improvement in peak VO2 42.9 ml/kg/min and at the end of process showing significant improvement of the peak VO2 to 49.1 ml/kg/min, with reduction of BMI to 22.4 kg/m^2 and improvement in laboratory findings.

Guided CR may improve functional capacity. In long-term, regulation of BMI in young patients may be translated into better quality of life and reduction of potential development of obasity-associated morbidities.

Key words cardiovascular rehabilitation, cardiopulmonary test, peak oxygen consumption, obesity

Introduction

Over the past decades, the prevalence of obesity has greatly increased worldwide. Obesity is associated with increased risk for cardiovascular disease, diabetes mellitus, cancer, and osteoarthritis with following increased risk of disability[1]. Lifestyle modification involving guided nutritional education and diet and cardiovascular rehabilitation (CR) through physical activity is considered the standard of care in obesity management of obese patients[2,3]. Other therapeutic interventions including pharmacotherapy and bariatric surgery have been demonstrated to be also beneficial [4]. All these measure can be implemented through programs of personalized or group-based behavioral therapy in specialized medical centers.

We presented a case report of young obese patient who underwent intense inpatient and outpatient rehabilitation program, including behavior intervention, diet, regular exercise and lifestyle counseling.

Presentation

D.N., age 16, is an overweight middle school student, body-mass index (BMI) of 30.1 kg/m^2. His sedentary lifestyle and unhealthy eating habits lead to low functional capacity. Hi struggle with low grades in sport activity school classes and try to lose weight starting different recreative sports disciplines (football, basketball, swimming) unsuccessfully. We started guiding cardiac rehabilitation with diet and cardiac exercise with complete cardiovascular examination and further following his progression through laboratory and cardiopulmonary testing.

In the beginning we performed a physical examination confirmed a normal finding of the heart and lungs. At rest, a 12 – lead channel electrocardiogram (ECG) recorded sinus rhythm, frequency 60 /min, with an image of the right bundle branch block (RBBB) (Picture 1). Echocardiography examination revealed a normal dimension of left ventricular (LV) with completely normal ejection fraction (Table 1). Considering the echocardiography finding without signs of structural heart disease, a cardiopulmonary exercise test (CPET) was performed with Schiller CS200. The test showed very low functional capacity with peak oxygen consumption (peakVO_2) up to 32, 0 mm/kg/min, with early O_2 puls plato fulfillment without signs of myocardial ischemia (Table 2).

The guided program begins with diet leading to weight loss of 8.5 kg trough six months period. After that initial loss of weight, he began with a regular daily walking sessions following guided physical activity in a Special

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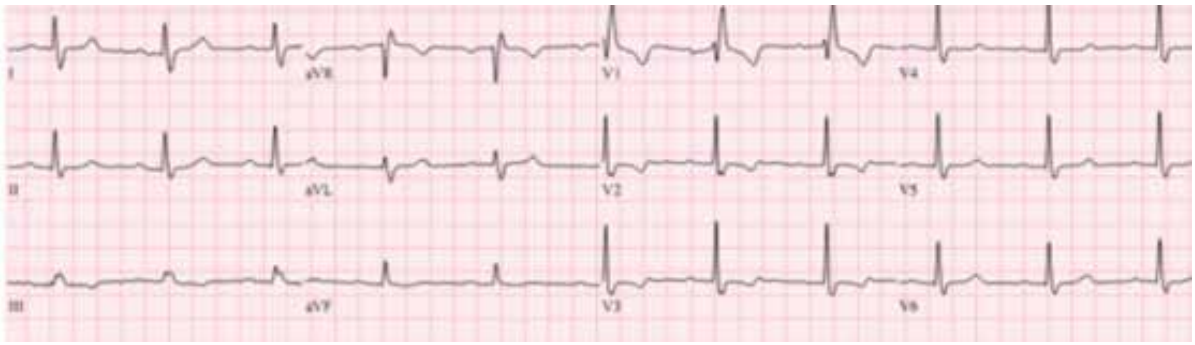

Figure 1. Electrocardiogy of obese young patient with right bundle branch block pattern

Table 1. Laboratory tests at baseline and after cardiovascular rehabilitation

Parameters	Before rehabilitation	End of rehabilitation	Normal range
WBC, 10*9/L	5.8	6.2	4.0-10.0
RBC, 10*12/L	5.01	5.38	4.0-6.3
Hemoglobine, g/L	156	165	120-180
Hematocrite, L/L	1.44	0.49	0.400-0.520
Trombocite, 10*9/L	191	189	140-440
Total cholesterol, mmol/L	5.8	3.4	<5.2
HDL cholesterol, mmol/L	1.4	1.0	<1.0
LDL cholesterol, mmol/L	4.1	2.5	<4.1
Triglicerides, mmol/L	1.1	0.95	<1.7
Urea, mmol/L	4.8	5.0	3.0-7.5
Creatinin, umol/L	92	98	64-111
eGFR, ml/min/1.73m2	>60	>60	>60
Uric acid, umol/L	326	320	210-460
AST, U/L	19	19	<55
ALT, U/L	15	17	<34
Alkaline Phosphatase, U/L	110	105	40-150
Fasting glucose, mmol/L	6.0	4.2	3.9-5.8
HbA1c, mmol/moL	6.1	4.4	4-5.6
TSH, uIU/ml	3.4	3.7	0.4-4.9
FT4, pmol/L	16	15	9.0-19.1

WBC-white blood cell; RBC-red blood cell; AST- Aspartate amino-transferase; ALT- Alanine amino-transferase.

Table 2. Echocardiographic parameters

Parameters	Value
LA (mm)	35
EDD LV (mm)	52
ESD LV(mm)	30
EFLV (%)	66
Septum (mm)	10
Inferior wall (mm)	10
Mass (mg/m2)	72
E/A	0.63/0.41
E' (m/s)	0.08
E/E'	7.8
RV (mm)	22
TAPSE (mm)	29

LA- left atrium|; EDD LV- end-dijastolic dimension of left ventricule; ESD LV – end-sistolic dimensions of left ventricule EF LV-ejection fraction

hospital for metabolism disease on Zlatibor 'Čigota' with two-stage of one months programs. We performed CPET during CR at one year and at the end after 24 months showed significant improvement in maximal METs with peak VO_2 of 42.3 and 49.1 mm/kg/min respectively. Laboratory analyses showed improvement of lipid profile and regulated level of fasting glucose (Table 3). BMI at the end of program was 22,3 kg/m^2

Discusion

Numerous studies demonstrate the value of peak VO_2 as a prognostic tool ahead of traditional cardiovascular risk factors. Its value has been declared as more important than adiposity level in an obese patient[5]. Still, there are many factors that can affect peak VO_2, such as: body size, weight and body composition[6], physical fitness at the time of test, state of cardiopulmonary function, level of hemoglobin concentration, function of mitochondria and baseline genetic factors[6].

Table 3. Cardiopulmonary test parameters at beginning, during carediac rehabilitation and at the end of rehabilitation

Parameters	Before rehabilitation	After 10 months	End of rehabilitation
BMI (kg/m2)	30.1	24.9	22.3
Duration of test (sec)	415	490	618
SBP baseline (mmHg)	130	120	120
SBP max (mmHg)	170	150	145
DBP baseline (mmHg)	85	80	80
DBP max (mmhg)	90	90	90
HR baseline /min	60	62	61
HR maks /min	165	193	205
VAT VO2 (ml/kg/min)	15.0	18.0	22.0
PeakVO2 (ml/kg/min)	32.0	42.9	49.1
% predicted VO2 max	78	92	98
RER	1.2	1.2	1.2
VE/VCO2 slope	22.78	26.1	26.0

BMI- body mass index; SBP-systolic blood pressure; DBP-diastolic blood pressure; HR-heart rate; VAT- Ventilatory anaerobic threshold; RER-respiratory exchange ratio

In the young population, it is also necessary to favor the influence of growth on both body fat and exercise capacity. In a normal young person, the absolute O_2max increases roughly corresponding to body size[7]. In the beginning of CR, the lower peak VO_2max may be explained by grossly reduced oxygen usage by adipose tissue during exercise[8].

Loss of weight is an important topic in different obese populations, and different intervention treatments (such as diet, guided exercise, bariatric surgery) are constantly being optimized. The improvement in exercise capacity after intervention protocols was associated with improved body architecture[9]. Our patient underwent CR with the regulation of BMI, body composition and regulation of laboratory findings, along with excellent improvement in functional class (A) at the level of a good trained individual with peak VO_2 of 49.1mm/kg/min.

Furthermore, when conducting CR the ratio of peak VO_2 and body composition, especially muscle mass, may provide information useful for further guidance of rehabilitation programs which aims to improve aerobic performance as well as strength determined primarily by muscle mass[10].

Conclusion

Guided CV rehabilitation including diet and exercise lead to significant improvement of functional status and class of fitness of young obese patient with the regulation of all laboratory parameters.

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Sažetak

Efekti rehabilitacije, dijete i fizičke aktivnosti na aerobni kapacitet kod gojaznog pacijenta

Marina Ostojić[1], Jelena Simić[1], Branko Beleslin[1,2], Vojislav Giga[1,2], Ana Đorđević Dikić[1,2], Nikola Bošković[1], Ivana Nedeljković[1,2]

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Gojaznost je hronično stanje sa velikom prevalencom među mlađom populacijom. Povezana je sa značajnim povećanjem morbiditeta i mortaliteta, uključujući povišen rizik za razvoj kardiovaskularnih bolesti. Kardiovaskularna rehabilitacija (KR) podrazumeva promenu načina ishrane uz vođenu fizičku aktivnost i predstavlja osnovu za poboljšanje funkcionalnog kapaciteta i redukciju telesne težine kod gojaznih bolesnika.

U ovom prikazu slučaja opisaćemo mladog gojaznog pacijenta koji je prošao vođeni tretman KR sa promenom režima ishrane i postepenim uvođenjem fizičke aktivnosti sa posledičnim značajnim poboljšanjem aerobnog kapaciteta. Na početku procesa naš pacijent je bio gojazan sa indeksim telesne težine (BMI) 30.1 kg/m^2 i obimom struka 110 cm. Laboratorijski je registrovan povišen ukupni holesterol 5.8 mmol/L i poremećena tolerancija glukoze sa vrednostima HbA1c 6.0 %. Ehokardiografski nalaz je bio potpuno normalan. Kardiopulmonalni test je izvršen na početku KR kada je registrovan jako nizak stepen funkcionalnih sposobnosti sa vršnom potrošnjom kiseonika (peak VO_2) 32,0 ml/kg/min. Godinu dana nakon početka rehabilitacije registrovano je poboljšanje peakVO_2 42.9 ml/kg/min, dok je na kraju procesa registrovano značajno unapređenje funkcionalnog kapaciteta sa vršnim VO_2 to 49.1 ml/kg/min, uz redukciju BMI na 22.4 kg/m^2 i poboljšanje laboratorijskih parametara.

Vođena KR može popraviti funkcionalni kapacitet. Dugoročno, uspešna regulacija BMI kod mladog gojaznog pacijenta može voditi boljem kvalitetu života i redukciji potencijalnog razvoja bolesti povezanih sa gojaznošću.

Ključne reči: kardiovaskularna rehabilitacija, kardiopulmonalni test, gojaznost, vršna potrošnja kiseonika

Srce i krvni sudovi 2022; 41(1): 28-32

Case report

Acute coronary syndrome or acute aortic syndrome - correct diagnosis prevents fatal complication

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Abstract A 78-year-old man was admitted to local hospital due to chest pain. Electrocardiogram was performed three times at intervals of one hour, in which the progression of ST elevation on the anterior wall was clearly visible. However, aortic wall dissection was suspected on echocardiography, and CT without contrast also described aortic dissection. Patient was referred to cardiac surgery, but dissection was not confirmed intraoperatively. After two days, the patient developed severe chest pain with severe heart failure and cardiogenic shock. Cardiac echocardiography was performed, and it showed severe ischemic cardiomyopathy with reduced left ventricle ejection fraction (EF 19%). Coronary angiography was performed and it verified three-vessel coronary artery disease but interventional cardiologists estimated that the risk of percutaneous coronary intervention was too high at that time. All recommended therapy was applied and the patient's condition was improved, the symptoms and signs of heart failure were disappeared. The patient was discharged after 10 days in stable condition, compensated with the following therapy: warfarin, clopidogrel 75 mg, bisoprolol 2.5 mg, valsartan/sacubitril 26 mg / 24 mg two times per a day, amiodarone 200 mg, furosemide 2x40 mg, eplerenone 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazole 40 mg. Cardiologists and cardiac surgeons decided to try pecutaneous intervention on two arteries Cx and RCA in the next step.

Key words acute coronary syndrome, aortic dissection

Introduction

Aortic dissection is one of the most urgent and dramatic conditions in clinical medicine with a very high early mortality rate of 1-2% per hour in untreated patients in the first 24 to 48 hours. The most common symptom of aortic dissection, that occurs in over 96% of cases, is chest pain. The chest pain in aortic dissection begins suddenly and the intensity is strongest at the beginning ("decresendo" type of pain), which distinguishes it from pain in acute coronary syndrome, which is usually of a "crescendo" character. About two thirds of patients with ascending aortic dissection have aortic insufficiency, so early diastolic murmur can be heard auscultatory, and also in the physical finding there is a difference in pressure between on the left and right arm, and pulse deficit, ie. weakened or absent arterial pulses and very often neurological manifestations[1,2].

Acute coronary syndrome (ACS) is significantly more common than aortic dissection. It is the most severe form of ischemic heart disease and it is one of the most common causes of urgent hospital admission and sudden cardiac death. In addition to the characteristic chest pain, there are also ischemic changes in the electrocardiography (ECG) and an increase level of cardiac enzymes. Aortic dissection and acute coronary syndrome may have a similar clinical presentation, and careful history, electrocardiogram, and cardiac enzymes are necessary to distinguish these two very serious conditions.

Here we will present the case of a patient in whom the correct diagnosis of acute myocardial infarction was not made, which led to a wrong referral to cardiac surgery and development of severe ischemic cardiomyopathy with severe heart failure and cardiogenic shock.

Case presentation

A 78-year-old man was admitted to local hospital due to chest pain. Electrocardiogram was performed three times at intervals of one hour, in which the progression of ST elevation on the anterior wall was clearly visible. Troponin I (Tn I) was 25.1 pg / mL, CK MB 2.20 ng / mL. Echocardiographic examination showed that the ejection fraction of the left ventricle (EF) was reduced by 44%, the distal two thirds of left ventricle were hypokinetic, as well as the apex of the heart (Figure 1). Also, aortic wall dissection was suspected, and the patient was referred for a CT scan of the chest in a local hospital. CT was performed, but without contrast, and it described that the aortic root was dilated to 48 mm with signs of dissection on a short segment above the aortic valve. Due to this finding, the patient was urgently referred to the Univer-

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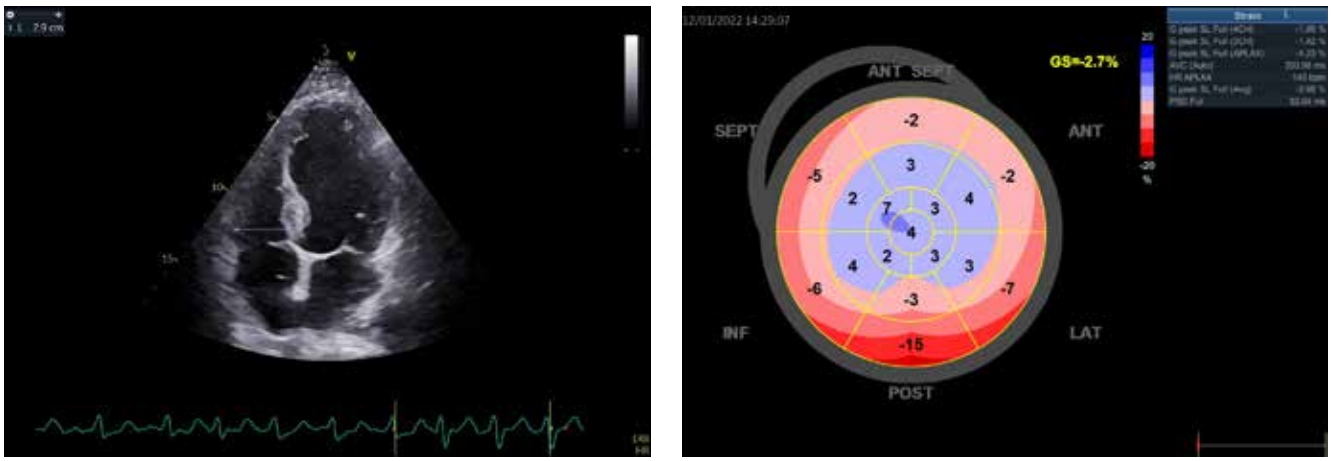

Figure 1a and b. Echocardiography findings with wall motion abnormalities of the anterior, antero-septal wall and the apex

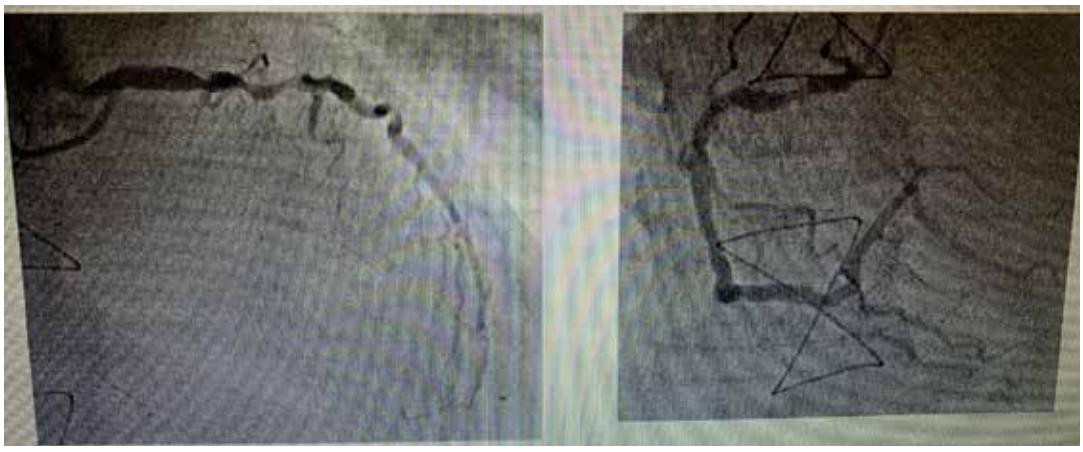

Figure 2. Selective coronarography presenting occluded ostial left main, with significant stenosis in LM, Cx and RCA

sity Clinical Center of Serbia, to the Cardiac Surgery Clinic for urgent surgical treatment. On admission to our clinic, the patient was stabile, with chest pain, blood pressure 135/85 mmHg on both arms, heart rate was 90/min, auscultatory without heart murmurs, all peripheral pulses were palpable. The patient was introduced to the operating room and under general anesthesia, a medial sternotomy was performed. An arterial cannulation of the ascending aorta was performed, cannulation of the right atrium too, and extracorporeal blood flow was started (ECC). The exploration did not show a rupture of the aortal intima, nor a rupture of the aorta. Laboratory analyzes that was taken immediately upon admission to the Clinic of Cardiac Surgery, which arrived subsequently, showed elevated markers of myocardial necrosis (troponin T 3267 ng/L, creatine kinase (CK) 1775 U/L). Intraoperatively, surgeons saw that the coronary arteries were extremely calcified and not suitable for grafting. Next day, selective coronary angiography was performed and it verified three-vessel coronary artery disease (Figure 2) with stenosis in the distal part of the left main stenosis, ostial left anterior descending (LAD) occlusion, 90% proximal circumflex artery (Cx) stenosis, and 80% stenosis in the right coronary artery (RCA). In the first postoperative day, there was a further increase in troponin T (Tn T 6959 ng/L), and the next two days there was a gradual decrease (3595 ng/L, then 2391 ng/L). After two days, the patient developed severe chest pain, ECG showed atrial fibrillation, heart rate 150/min, ST elevation in V1-V3 leads, Q wave in V2-V3, ST depression in D2, D3, aVf leads (Figure 3). Clinically, severe heart failure and cardiac shock was developed. Intravenous antiarrhythmic therapy (amiodarone) was prescribed, and electroconversion was attempted but without success. Inotropic support was prescribed. Due to the cessation of diuresis, continuous intravenous diuretic therapy was prescribed, too. Laboratory analyzes showed a new elevation in markers of myocardial necrosis (troponin T 4094 ng/L), an elevation in transaminases (AST 190 U/L, ALT 134 U/L). Cardiac echocardiography was performed, and it showed severe ischemic cardiomyopathy with a large part of akinesia that involved the entire septum, medio-apical segment of the anterior, posterior and lateral wall, better contractility was only in basal segments of the postero-lateral wall (Figure 4). Ejection fraction was severe reduced (EF 19%). Longitudinal LV function was also severe decreased (AFI: EDV 106 ml, ESV 85 ml, EF 19%, GLS -2.7%, PSD -52), signs of diastolic dysfunction was presented, too and also significant increased in filling pressure of LV (DCT -92, E'lat- 7.0, E's-5.0, E/e'avg-18.0), moderate mitral regurgitation, right ventricle was with normal size, decreased systolic function (TAPSE 1.1 cm), severe tricuspid regurgitation 2-3 +, pressure in the right heart was 50 mmHg. In the further

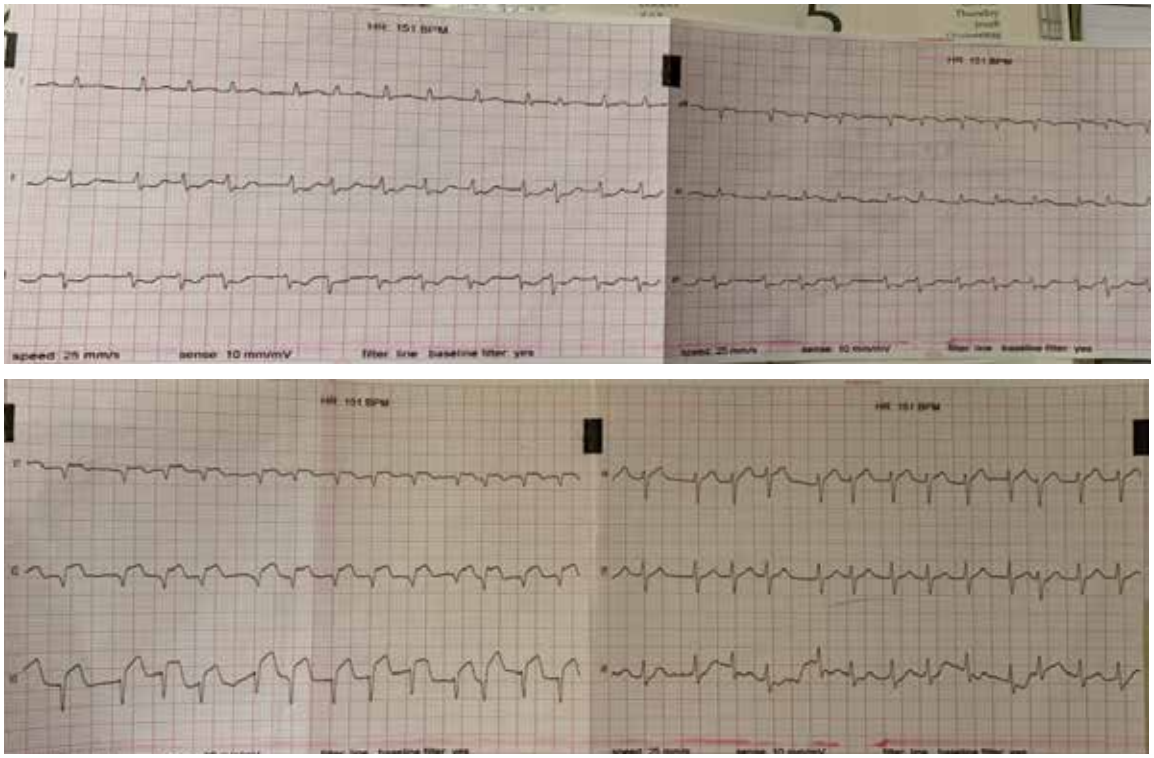

Figure 3. Electrocardiogram-atrial fibrillation, Q wave in V2-V3 leads with ST elevation, ST depression in D2, D3, aVf leads

course, the patient's condition was improved, the symptoms and signs of heart failure were disappeared. The sinus rhythm was established. ECG showed a scar in V2-V4 leads with persistent ST elevation. The patient was discharged after 10 days in stable condition, compensated with the following therapy: warfarin, clopidogrel 75 mg, bisoprolol 2.5 mg, valsartan/ sacubitril 26 mg / 24 mg two times per a day, amiodarone 200 mg, furosemide 2x40 mg, eplerenone 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazole 40 mg. Cardiologists and cardiac surgeons decided to try pecutaneous intervention on two arteries Cx and RCA in the next step.

Discussion

Here we present a typical patient that is seeing every day in cardiac ambulances. The patient came with chest pain, first ECG showed unconvincing elevation of the ST segment, troponin was taken but troponin I, which was not elevated, then an echocardiography of the heart where performed ant it showed disorder in segmental kinetics but aortic wall stratification was suspected. However, the next two ECGs, showed a clear progression of elevation, but due to the suspicion of dissection, a chest CT scan was performed and it showed misdiagnosed of ascending aortic dissection and the patient was referred to wrong direction.

In all patients with chest pain electrocardiogram (ECG) should be performed first. If acute coronary syndrome is ruled out, aortic dissection should be considered. In the IRAD study, ECG was normal in 31% of patients, nonspecific ST and T changes were found in 42% of patients, ischemic changes in 15% of patients, and signs of acute myocardial infarction in 5% of patients with ascending

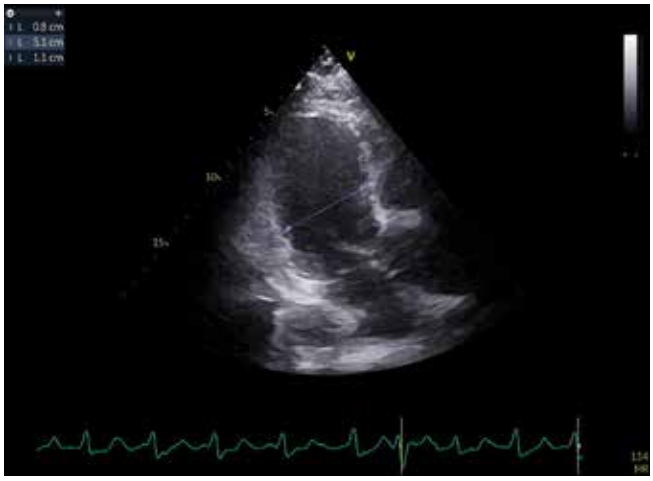

Figure 4. Echocardiography findings showing deterioration of LV function and ejection fraction

aortic dissection[3]. Aortic dissection can be extended to the branches of the aorta, as well as to the coronary arteries, mainly the right coronary artery, so it can give a picture of inferior myocardial infarction with ST-segment elevation in the inferior leads in the ECG.

Aortic dissection can mimic many diseases, so early recognition of this entity is crucial in order to reduce early mortality. Analyzing 26 clinical variables at initial presentation of aortic dissection, Von Kodolish singled out the following variables as independent predictors for the diagnosis of acute aortic dissection: (1) pain (sudden onset, tearing, tearing), (2) dilated aorta or mediastinum at radiography (3) difference in pulse or arterial pressure. If all three predictors are present, the probability of aortic dissection is 96%. The probability is lower if there are one or two predictors and is 35%, and only 7% if none of these predictors exist[4].

The choice of diagnostic methods depend on the patient's condition, the availability of diagnostic techniques and experience in interpreting the results. Diagnostic techniques should primarily confirm the existence of aortic dissection and the type of dissection. Aortography has long been the gold standard for the diagnosis of dissection. Its sensitivity is 86-88% and specificity 75-94%. However, with the development of non-invasive methods, primarily contrast computed tomography, aortography as an invasive method has been suppressed[5]. Transthoracic echocardiographic examination is very useful, with sensitivity for proximal aortic dissection of 78-100%, but for distal is lower 31-55%[6,7,8]). The most significant sign is intimate flap (floating intimate membrane) that separates the true from the false lumen. Using the color Doppler technique, the flow through the false lumen can be seen. However, today, computed tomography (CT) with the use of intravenous contrast is used for the diagnosis and confirmation of dissection, which is a very sensitive and specific method for the diagnosis of aortic dissection. Multidetector CT (MDCT) with the possibility of three-dimensional image reconstruction further improves the diagnostic accuracy of this method. The sensitivity and specificity of CT for the diagnosis of aortic dissection is 96-100%[9,10]. What is important to point out is that CT must be done with contrast in order to avoid situations like the one that happened to our patient.

If an acute myocardial infarction is not diagnosed on time a number of complications resulting from myocardial necrosis can be developed. The most common complication of infarction, especially anterior wall infarction, is heart failure. Our patient developed severe ischemic cardiomyopathy with a significantly reduced left ventricular ejection fraction, which was resulted in the development of acute heart failure. Cardiac shock is one of the manifestations of acute heart failure[11]. Treatment of cardiac shock should be started as early as possible. Early identification and treatment of the underlying causes, along with hemodynamic stabilization and treatment of organ dysfunction, are essential components of treatment[11]. In the case of our patient, hemodynamic stabilization was performed, therapy was prescribed according to recommendations, but treatment of the underlying cause, ie. treatment of coronary heart disease was not possible, because interventional cardiologists estimated that the risk of percutaneous coronary intervention was too high at that time. Therefore, it was decided to stabilize the patient with medication. However, severe ischemic cardiomyopathy and the potential new decompensation and the development of malignant heart rhythm disorders, remain a problem. Therefore, in accordance with the latest recommendations, the patient received therapy (valsartan/sacubitril, empagliflozin, eplerenone and bisoprolol), which has been shown to reduce mortality and prolong survival in heart failure. Of course, loop diuretics, which prevent congestion, are important parts of therapy. It remains to be seen how the situation with our patient will continue.

Conclusion

Acute coronary syndrome is much more common than aortic dissection, so in patients with chest pain, ACS should be confirmed or ruled out first. Although CT is a more sophisticated method than electrocardiograms, if it is not done adequately, or like in this case without contrast, or if it is read by someone with insufficient experience in recognizing of aortic dissection, misdiagnosis and misdirection and treatment of patients can occur. A new approach to the treatment of patients with heart failure, provides us new opportunities in the treatment of these patients, with prolonging their lives, reducing mortality and improving quality of life.

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Sažetak

Akutni koronarni sindrom ili akutni aortni sindrom – pravilna dijagnoza sprečava fatalne komplikacije

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Muškarac star 78 godina primljen je u regionalnu bolnicu zbog bolova u grudima. Urađeni su elektrokardiogrami u tri navrata u razmaku od po sat vremena, u kojima se jasno vidi progresija ST elevacije na prednjem zidu, ali je ehokardiografski (EHO) postavljena sumnja na raslojavanje zida aorte, te je urađena CT grudnog koša, ali bez kontrasta, gde je opisana disekcija ushodne aorte. Pacijent je htno upućen na kardiohirurgiju, ali disekcija nije potvrđena intraoperativno. Posle dva dana, pacijent je dobio jak bol u grudima uz razvoj teške srčane insuficijencije i kardiogenog šoka. Urađen je EHO srca koji je pokazao tešku ishemijsku kardiomiopatiju sa smanjenom ejekcionom frakcijom leve komore (EF 19%). Urađena je koronarna angiografija i verifikovana trosudovna koronarna bolest, ali su interventni kardiolozi procenili da je rizik od perkutane koronarne intervencije tada bio previsok. Primenjena je sva terapija prema preporukama i stanje pacijenta se poboljšano, simptomi i znaci srčane insuficijencije su nestali. Pacijent je otpušten posle 10 dana u stabilnom stanju, sa sledećom terapijom: varfarin, klopidogrel 75 mg, bisoprolol 2,5 mg, valsartan/akubitril 26 mg/24 mg dva puta dnevno, amiodaron 200 mg, furosemid 2k40 mg, 25 mg, empagliflozin 10 mg, rosuvastatin 20 mg, pantoprazol 40 mg. Kardiohirurški konzilijum je odlučio da se naknadno pokuša PCI preostale dve arterije C xi RCA.

Ključne reči: akutni koronarni sindrom, disekcija aorte

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