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КЛИНИЧЕСКИЙ СЛУЧАЙ СПОНТАННОЙ ДИССЕКЦИИ ВЕТВЕЙ ЛЕВОЙ КОРОНАРНОЙ АРТЕРИИ У МОЛОДОЙ ЖЕНЩИНЫ

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Clinical Case of Spontaneous Dissection of Branches of The Left Coronary Artery in A Young Woman

Резюме

Спонтанная диссекция коронарных артерий (СДКА) — редкая, но потенциально опасная причина острого коронарного синдрома, особенно у молодых женщин без традиционных факторов риска сердечно-сосудистых заболеваний. Этиологические факторы включают фибромышечную дисплазию, наследственные артериопатии, системные воспалительные заболевания и гормональные изменения. Несмотря на прогресс в диагностике, лечение СДКА остается сложной задачей ввиду высокой вариабельности клинических проявлений и отсутствия единого стандарта терапии. Представлен случай молодой пациентки, госпитализированной с клиникой инфаркта миокарда с подъемом сегмента ST, возникшего на фоне интенсивной физической нагрузки. При проведении коронароангиографии (КАГ) выявлена спонтанная диссекция ветвей левой коронарной артерии. В ходе КАГ отмечено прогрессирование диссекции, потребовавшее повторного стентирования и использования механической поддержки кровообращения (ВА-ЭКМО, ВАБК). Несмотря на проводимое лечение, у пациентки сохранялась нестабильная гемодинамика, прогрессирование полиорганной недостаточности, что привело к летальному исходу.

По данным гистологического исследования не исключена дисплазия соединительной ткани, что могло быть предрасполагающим фактором развития диссекции коронарных артерий. Клинические рекомендации предполагают консервативное ведение стабильных пациентов, однако при наличии осложнений, таких как кардиогенный шок, показано проведение реваскуляризации. В данном случае инвазивная тактика не повлияла на прогноз.

Приведенный случай демонстрирует сложность диагностики и ведения пациентов с СДКА. Необходимы дальнейшие исследования для разработки оптимальных стратегий лечения и выявления генетических маркеров, предрасполагающих к развитию данной патологии.

Ключевые слова: *спонтанная диссекция коронарных артерий, острый коронарный синдром, фибромышечная дисплазия, экстракорпоральная мембранная оксигенация*

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare but potentially dangerous cause of acute coronary syndrome, especially in young women without traditional risk factors for cardiovascular diseases. Etiological factors include fibromuscular dysplasia, hereditary arteriopathies, systemic inflammatory diseases, and hormonal changes. Despite the progress in diagnosis, the treatment of DMCA remains a difficult task due to the high variability of clinical manifestations and the lack of a single standard of therapy. The case of a young patient hospitalized with a ST-segment elevation myocardial infarction clinic, which occurred against the background of intense physical exertion, is presented. Coronary angiography (CAG) revealed spontaneous dissection of the branches of the left coronary artery. The progression of dissection was noted during CAH, which required repeated stenting and the use of mechanical circulatory support (VA-ECMO, IABC). Despite the treatment, the patient maintained unstable hemodynamics and the progression of multiple organ disfunction, which led to death. Histological examination revealed connective tissue dysplasia cannot be excluded, which could be a predisposing factor for the development of coronary artery dissection. Clinical recommendations suggest conservative management of stable patients, however, revascularization is indicated in the presence of complications such as cardiogenic shock. In this case, the invasive tactics did not affect the prognosis.

The above case demonstrates the complexity of diagnosis and management of patients with SDCA. Further research is needed to develop optimal treatment strategies and identify genetic markers predisposing to the development of this pathology.

Key words: *spontaneous coronary artery dissection, acute coronary syndrome, fibromuscular dysplasia, extracorporeal membrane oxygenation*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

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Conformity with the principles of ethics

Informed consent is not required due to the impossibility of identifying the patient

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IgA — immunoglobulin A, IgG — immunoglobulin G, IgM — immunoglobulin M, TAPSE — tricuspid annular plane systolic excursion, TIMI — blood flow scale, VTI LVOT — velocity-time integral of left ventricular outflow tract, BP — blood pressure, ALT — alanine aminotransferase, ANF — antinuclear factor, AST — aspartate aminotransferase, Ab — antibody, VA-ECMO — venoarterial extracorporeal membrane oxygenation, DIC — disseminated intravascular coagulation, DB — diagonal branch, MV — mechanical ventilation, MI — myocardial infarction, STEMI — ST-elevation myocardial infarction, BMI — body mass index, CAG — coronary angiography, CT — computed tomography, LCA — left coronary artery, HDL — high-density lipoproteins, LDL — low-density lipoproteins, CxA — circumflex artery, ACS — acute coronary syndrome, AKI — acute kidney injury, AHF — acute heart failure, RCA — right coronary artery, Ca^{2+} — ionized calcium, SCAD — spontaneous coronary artery dissection, SPAP — systolic pulmonary artery pressure, DES — drug-eluting stent, CRP — C-reactive protein, DUS — Duplex ultrasound, hCG — human chorionic gonadotropin, PCI — percutaneous coronary intervention, RR — respiratory rate, HR — heart rate, ECV — electric cardioversion, ECG — electrocardiogram, EchoCG — echocardiography, PIVB — posterior interventricular branch, RRT — renal replacement therapy

Introduction

Spontaneous coronary artery dissection (SCAD) is an atraumatic, non-iatrogenic, and non-atherosclerotic dissection of the coronary artery wall due to intramural hemorrhage caused by the intimal rupture or spontaneous bleeding from vasa vasorum, which leads to the blood flow obstruction with the dissected endothelium and the emergence of acute coronary syndrome [1].

SCAD is most common among young females with low cardiovascular risks. No significant data about SCAD prevalence have been found due to the absence of a clear diagnostic protocol and variability of clinical signs. It is presumed that SCAD causes 1–4 % of all myocardial infarction cases and 35 % of myocardial infarctions in females under 50 years of age. The female-to-male ratio is approximately 90:10, though in some observational cohorts that can reach 60:40 [2–4].

SCAD is a multifactorial disease. SCAD is most often associated with fibromuscular dysplasia — 25–86 % [5], hereditary arteriopathies and connective tissue diseases (Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome, α1-antitrypsin deficiency, polycystic kidney disease) are observed in 1.2–3 % cases [6], systemic inflammatory diseases — in > 1–8.9 % cases [7]. Meanwhile, hormonal drugs may cause dissection in 10.7–12.6 % cases [8], while pregnancy becomes a provoking factor in 2–8 % cases [9]. Extreme physical exertion or emotional stress, sympathomimetics, delivery, and extensive maneuvers (i.e. Valsalva maneuver) are considered potential triggers in young persons in the setting of altered hormonal background [3].

It is considered that SCAD usually manifests with acute coronary syndrome (ACS). Nevertheless, the latest data demonstrate that SCAD may also manifest with cardiogenic shock, ventricular arrhythmias, and cardiac arrest [10]. ST-elevation myocardial infarction (MI) develops in 26–87 % patients with SCAD, non-ST-elevation — in 13–69 % cases. Cardiogenic shock is reported in 2–5 % patients, ventricular arrhythmias and sudden cardiac death — in 3–11 % cases [10].

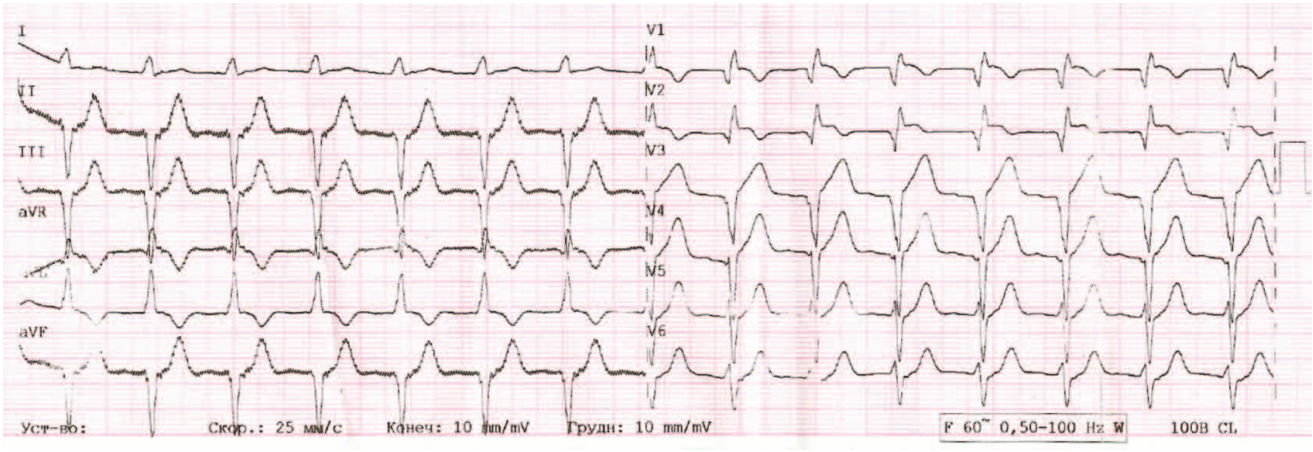

Figure 1. *Electrocardiogram (ECG) of patient K. at the pre-hospital stage*

Below we present a clinical case study of ST-elevation myocardial infarction as a result of spontaneous dissection of a left coronary artery branch in a young patient.

Case Study

The female patient K., 34 years old, was hospitalized into the cardiac intensive care unit of the City Clinical Hospital No. 52, Department of Healthcare of Moscow City (CCH No. 52 DHMC), with the preliminary diagnosis of acute coronary syndrome.

On admission she complained about prolonged pressure-like retrosternal pain that emerged during the intensive physical exertion (swimming in the pool) and did not disappear at rest. History: similar short-term pain emerged earlier that disappeared spontaneously. Within the previous months the patient had dyspnea on exertion, but she did not undergo any examinations. Life history: no specific cardiovascular diseases were reported in the family history; the patient did not have any comorbidities and bad habits.

Electrocardiogram (ECG): sinus rhythm, heart rate (HR) 100/min, R wave regression in leads II, III, aVF, V1–V4; ST-segment elevation (max. 2 mm) in leads V1–V4, negative T wave in leads aVR, aVL, V1, V2 (Figure 1).

The patient is normosthenic: height 170 cm, body weight 69 kg, body mass index (BMI) 23.8 kg/m2. Physical examination: clear consciousness, clean skin of normal color and moisture level; no edema detected. Respiratory rate (RR): 16 per minute. SpO_2 98 % on room air. Lung auscultation revealed vesicular breathing with no rales. Regular rhythm, physiological accentuation of tones preserved, no murmurs reported; HR 75 bpm, blood pressure (BP) 125/75 mm Hg in the left arm, 120/75 mm Hg in the right arm. No specific features were detected in other organs and systems. Pregnancy was excluded.

Laboratory data are presented in Table 1: increased troponin I levels (twice over the upper limit of normal), thrombocytosis, leukocytosis, electrolyte disorders, hyperglycemia, cytolysis.

Based on clinical signs, ECG, and increased troponin levels, the following diagnosis was established: acute anteroseptal ST-elevation myocardial infarction (STEMI) spreading to the apex and the inferior wall of the left ventricle (LV).

The patient underwent urgent coronary angiography (CAG) (Figure 2 a, b, c, d, e, f, g, h). Left myocardial supply type. No stenotic lesions in the trunk of the left

Table 1. *Laboratory data of patient K.*

Parameters	Results	References
Na+, mmol/l	133 ↓	135-146
K+, mmol/l	2,7 ↓	3,3-5,5
Ca2+, mmol/l	1,03 ↓	1,13-1,23
Glucose, mmol/l	8,5 ↑	3,9-6,4
pH	7,300 ↓	7,320-7,420
Hemoglobin, g/l	127,0	120-140
Platelets, 10^9/l	370,0 ↑	180-320
Leukocytes, 10^9/l	12,8 ↑	4,0-9,0
Troponin I (quantitative), ng/ml	43 ↑	<23
D-dimer	54,00	<230
ALT, U/l	47 ↑	0,0-38,0
AST, U/l	339,9 ↑	0,0-38,0
CRP, mg/l	21,2 ↑	0,00-6,00
Total cholesterol mmol/l	4,1	0-5,3
Triglycerides, mmol/l	0,84	0,68-1,9
LDL cholesterol, mmol/l л	1,94	0-3,38
HDL cholesterol, mmol/l л	1,5	0,78-1,55
hCG, GE/ml	<1,00	0-2,5

Note. Na^+ — sodium, K^+ — potassium, Ca^{2+} — ionized calcium, pH — blood pH, Troponin I (quantitative) — troponin I, D-dimer — fibrin degradation product, ALT — alanine aminotransferase, AST — aspartate aminotransferase, CRP — C-reactive protein, Total cholesterol — total cholesterol, Triglycerides — triglycerides, LDL — low-density lipoprotein, HDL — high-density lipoprotein, hCG — human chorionic gonadotropin

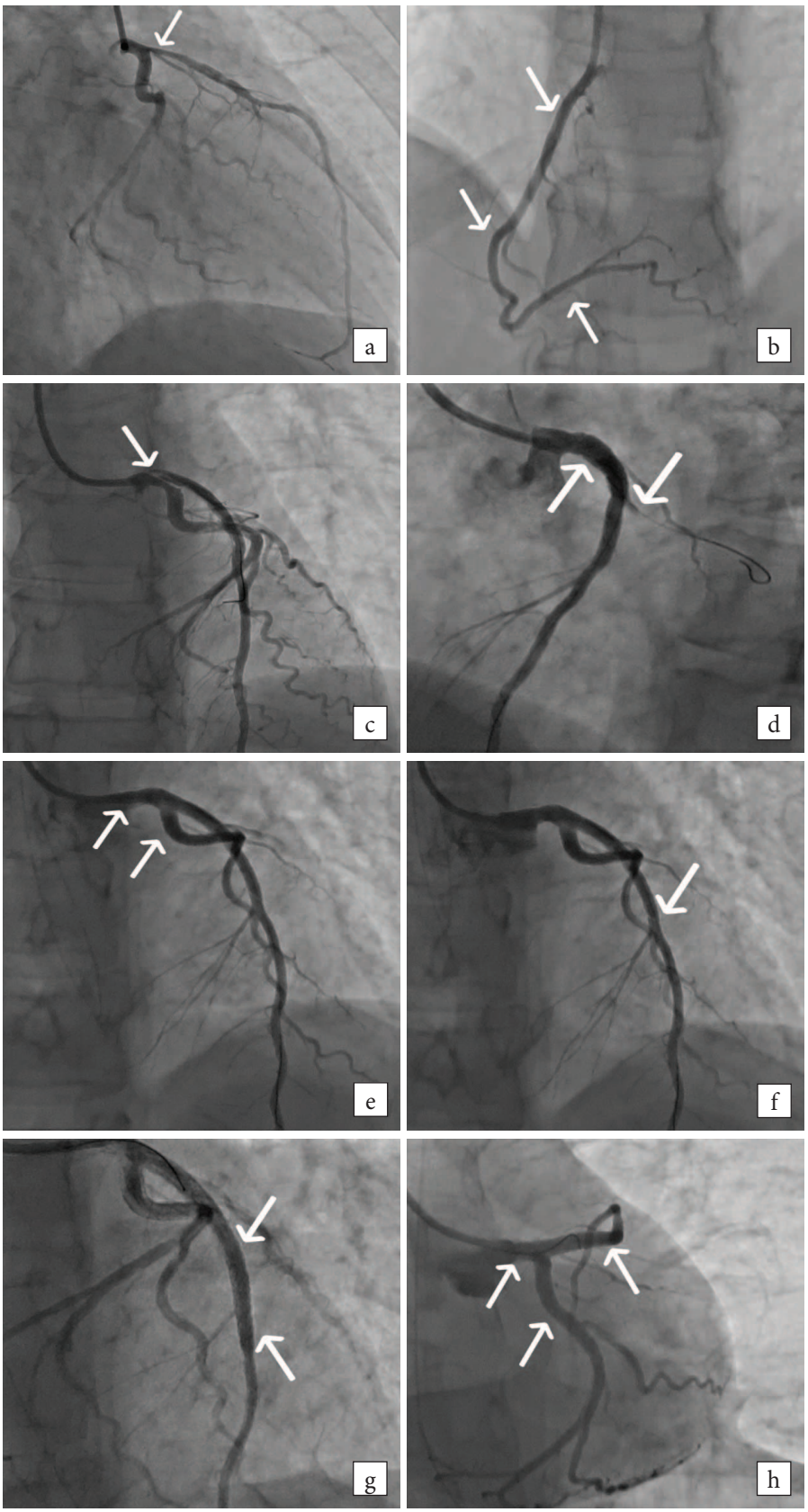

Figure 2 a, b, c, d, e, f, g, h. *Coronary angiography of patient K.*
Note: A step-by-step description of coronary angiography is presented:
a. 80% stenosis of the proximal segment of the LAD,
b. RCA without angiographic abnormalities,
c. Dissection of the left main coronary artery (LMCA) and LAD during guide catheter insertion,
d. Occlusion of the circumflex artery (Cx) from the ostium developed after stenting of the LMCA–LAD,
e. Stenting of the proximal segment of the Cx and the shaft of the LMCA was performed,
f. Dissection is visualized in the mid segment of the LAD and the distal third of the Cx
g. Final result after stenting of the mid segment of the LAD
h. Final result of PCI

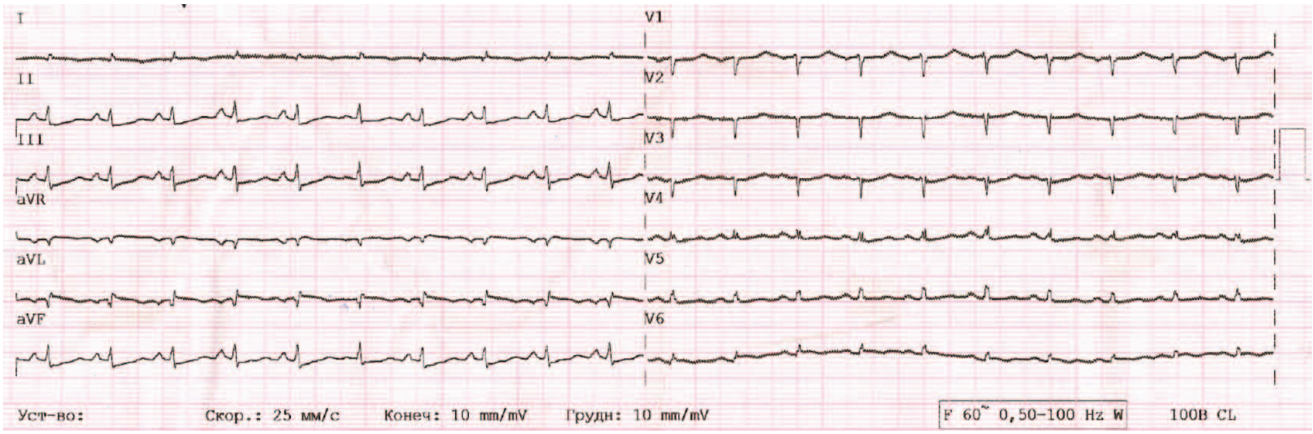

Figure 3. *Electrocardiogram (ECG) of patient K. after surgery*

coronary artery (LCA). Anterior interventricular artery (AIVA): 80–85 % stenosis in the proximal segment (Figure 2a) with smooth contours over 20 mm long. The 1st diagonal branch (DB-1): acute prolonged sub-occlusion from the ostium to the proximal-middle DB segment, TIMI blood flow 0-1. Spontaneous DB dissection was diagnosed, with the intravascular hematoma spreading from the DB to the proximal AIVA segment. The circumflex artery (CxA) and the right coronary artery (RCA) (Figure 2b) did not have stenotic lesions. During the percutaneous coronary intervention (PCI), intimal dissection of the proximal AIVA segment spreading to the LCA trunk was observed in the infarction-associated AIVA-DB territory (Figure 2c). The drug-eluting stent (DES) was implanted in the dissection area. Occlusion of the CxA ostium was observed in the follow-up CAG (Figure 2d). DES was implanted into the recanalized and dilated segment from the ostium to the middle CxA segment (Figure 2e). Due to the no-reflow phenomenon, integrilin (2b/3a receptor blocker) infusion was arranged. After the infusion, the blood flow in the CxA restored. The follow-up CAG detected intimal dissection in the LCA trunk along the proximal edge of the stent implanted earlier, which required DES implantation from the ostium, overlying the proximal edge of the stent implanted earlier. During the next follow-up, LCA lumen was restored, though intimal dissection was diagnosed in the middle AIVA segment and in the ostium of CxA PIVB, leading to its occlusion (Figure 2f). DES was implanted into the middle third of AIVA (Figure 2g). During the follow-up, intimal dissection was reported along the proximal stent edge, due to which DES was implanted, overlapping stents implanted earlier. Attempts of DB recanalization were unsuccessful. CxA PIVB was recanalized wth subsequent angioplasty, but due to metallization of the LCA trunk and CxA, as well an acute angle of CxA branching, it was not possible to implant a stent. The follow-up examination did not determine intimal dissection. TIMI 3 blood flow was reported in the LCA trunk, AIVA, CxA, and their branches (Figure 2h).

The patient intraoperatively developed clinical signs of cardiogenic shock, requiring drug-induced and mechanical circulatory support. The patient was transferred to mechanical ventilation (MV), venoarterial extracorporeal membrane oxygenation (VA-ECMO) was initiated, and the intraaortic balloon pump (IABP) was installed.

ECG after CAG with stenting (Figure 3): sinus rhythm, HR 125/min, R wave regression in leads I, aVL, V1–V3; ST-segment depression (max. 2 mm) in leads II, III, aVF.

Echocardiography (EchoCG) after CAG with stenting: significantly decreased general systolic LV function (ejection fraction (EF) 12–13 %) along with diffuse hypokinesis; circular akinesis of the LV apex spreading to the middle segments of the posterior, inferior, lateral, and anterior walls; VTI in the left ventricular outflow tract (VTI LVOT) 3.3 cm; decreased right ventricular (RV) contractility (tricuspid annular plane systolic excursion (TAPSE) 1.4–1.5 cm). No aortic pathology was detected. Cardiac chambers were not dilated, no valvular regurgitations were detected; systolic pulmonary artery pressure (SPAP) 35 mm Hg.

Accounting for the young age of the patient, absence of risk factors and atherosclerotic lesions of coronary arteries, the following diagnosis was established based on CAG data: myocardial infarction caused by the spontaneous coronary artery dissection.

Systemic immune inflammatory diseases (systemic vasculitides, antiphospholipid syndrome, systemic lupus erythematosus, etc.) were considered in the differential diagnosis. However, no clinical signs, negative results of immunology tests (titers of antinuclear antibodies (Abs), anti-myeloperoxidase, protease, cardiolipin Abs, cryoglobulin levels) helped to exclude systemic connective tissue disorders. Decreased C3 complement levels and IgA levels were detected (Table 2).

Table 2. Immunological studies of patient K.

Parameters	Results	References
Serum complement component C4 (C4, C4f), mg/dL	12	10,0-40,0
Serum complement component C3 (C3, C3NCF, C3a), mg/dL	62 ↓	90,0-170,0
Antinuclear antibodies (ANA), optical density units	0,12	0,00-1,0
Anti-β2-glycoprotein antibodies, GE/mL	1,6	0,0-10,0
Anti-myeloperoxidase (anti-MPO) antibodies — IgG, IU/L	0,1	0,0-20,0
Anti-proteinase 3 (PR3) antibodies — IgG, IU/L	2,5	0,0-20,0
Cryoglobulin	отр	
Total anticardiolipin antibodies, GE/mL	3,4	0,0-10,0
Immunoglobulin G (IgG), mg/dL	545 ↓	1000-1400
Immunoglobulin M (IgM), mg/dL	52 ↓	130-170
Immunoglobulin E (IgE), IU/mL	24,9	0,0-130,0
Immunoglobulin A (IgA), mg/dL	162 ↓	210-290

Note: C4 — Serum complement component C4 (C4, C4f), C3 — Serum complement component C3 (C3, C3NCF, C3a), ANA — Antinuclear antibodies, optical density units, Anti-β2GPI antibodies — Antibodies to β2-glycoprotein I, Anti-MPO antibodies — Antibodies to myeloperoxidase (IgG), Anti-PR3 antibodies — Antibodies to proteinase 3 (IgG), Cryoglobulin — Immunoglobulins that precipitate at low temperatures and dissolve on warming, ACA (Total) — Total anticardiolipin antibodies, IgA — immunoglobulin A, IgG — immunoglobulin G, IgM — immunoglobulin M, IgE — immunoglobulin E

Duplex ultrasound (DUS) of vessels of upper and lower extremities, including brachiocephalic arteries, demonstrated their complete patency; the intima-media complex was not thickened.

Computed tomography (CT)-angiography of the aorta and its branches, arteries of lower extremities did not reveal any vascular pathology (stenosis, aneurysms, contrast defects, pathological tortuosity). Chest CT was normal. Abdominal CT demonstrated dolichosigmoid.

The treatment was arranged in accordance with clinical guidelines of the Ministry of Health of Russia concerning the diagnosis and treatment of acute ST-elevation myocardial infarction (2020) [11]. Despite the intensive multicomponent therapy, hemodynamics remained unstable, paroxysmal ventricular tachycardia relapsed, and multiorgan failure progressed in the setting of systemic hypoperfusion. Death occurred 27 days after the hospital admission.

The following main diagnosis was formulated before autopsy: "Spontaneous DB dissection with the emergence of AIVA hematoma. Acute anterior (spreading) ST-segment elevation myocardial infarction. CAG: intimal dissection in the proximal AIVA segment spreading to the LCA trunk. LCA trunk dissection. LCA trunk stenting spreading to AIVA, intraluminal recanalization of CxA, balloon CxA angioplasty, CxA stenting, kissing dilation of the LCA trunk bifurcation. No-reflow phenomenon. Balloon CxA angioplasty. Intimal dissection in the ostium of CxA PIVB. AIVA stenting.

Complications: Acute heart failure (AHF) Killip IV. MV, VA-ECMO, IABP. Paroxysmal ventricular tachycardia, electrical cardioversion (ECV). Multiorgan (respiratory, cardiovascular, hepatic, cerebral, renal) failure syndrome. Coagulopathy. Acute kidney injury (AKI). DIC. Polyneuromyopathy of critical conditions. Renal replacement therapy (RRT) sessions. Pulmonary edema. Cerebral edema."

Autopsy data: all coronary arteries had similar alterations — wall dissection at the medial level (~3/4 along the circumference) (Figure 4). The contracted intima and partially media (arrow) lie in the arterial lumen with the blood spilled in the dissection area. Destructive foci in the media without rupture are observed in several large arteries (Figure 5).

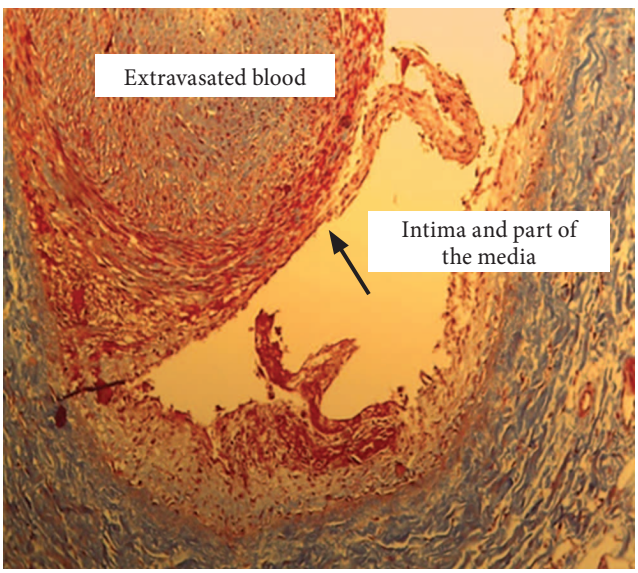

Figure 4. *Coronary artery with medial layer rupture and false lumen formation. Masson's trichrome stain.*

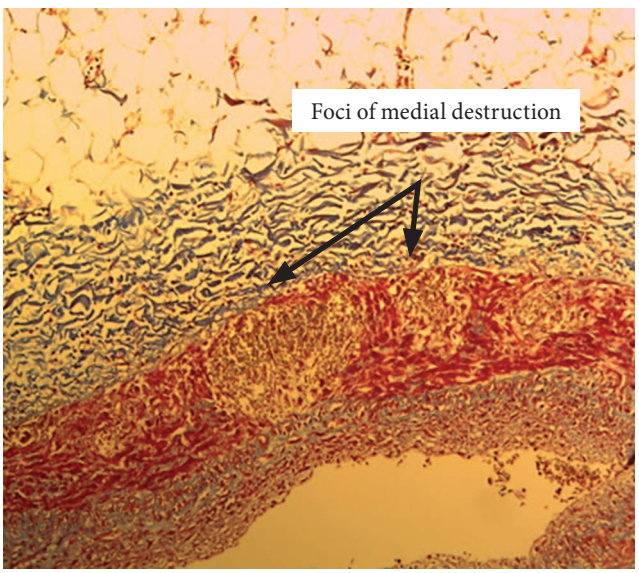

Figure 5. *Coronary artery with medial layer destruction without rupture. Masson's trichrome stain.*

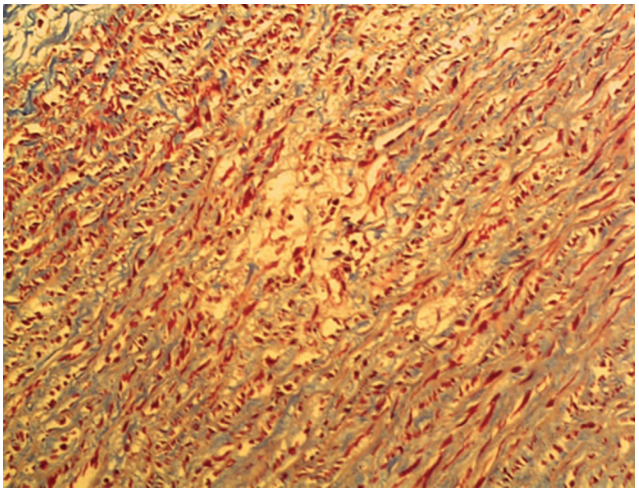

Figure 6. *Aorta. Marked myocyte edema. Masson's trichrome stain*

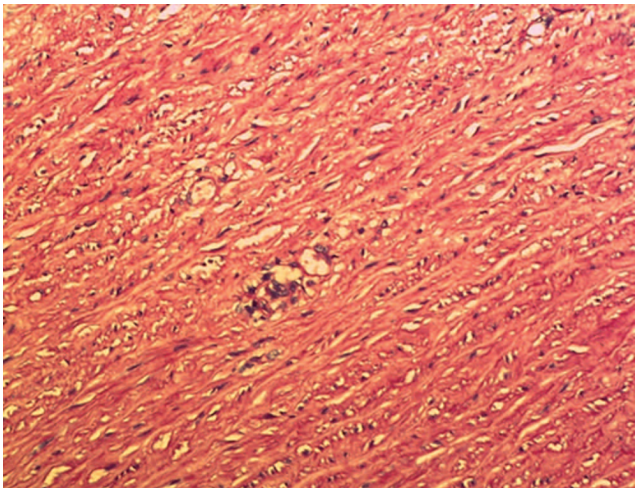

Figure 7. *Aorta. Myocyte vacuolization with nuclear dystrophy presenting as hyperchromatosis*

Along the whole length the aortic intima is ivory-colored and smooth. Histology revealed signs of destruction in the form of myocyte vacuolization (Figure 6) and nuclear dystrophy in the form of hyperchromatosis (Figure 7).

Connective tissue dysplasia could not be excluded based on histology findings.

Thus, a young female patient developed acute transmural LV MI caused by the spontaneous dissection of the left coronary artery branches complicated with resistant cardiogenic shock. Interpretation of clinical signs, ECG patterns helped to select invasive tactics — PCI with stenting and subsequent mechanical circulatory support (IABP, VA-ECMO), which did not affect the outcome.

Discussion

Three SCAD types are defined by J. Saw (2014). In that case angiography signs corresponded to the most common type II [12]. This type features lesions of the middle and distal segments of coronary arteries. Significant (often mild) quick alterations of the arterial caliper are observed, from their normal diameters to diffuse narrowing. Diffuse (usually > 20 mm) and commonly smooth narrowing may vary in severity from a slight, mild stenosis to complete occlusion [13]. Accounting for histology results, inside-out mechanism was observed, with sudden intimal rupture and blood entering the medium layer, forming a false lumen that increased due to intramural pressure — as a result, intramural hematoma was formed [10].

The diagnostic search was limited due to the severe patient's condition. Arteriopathies were considered an etiological factor of dissection, in particular the most common cause of SCAD, i.e. fibromuscular dysplasia (FMD). The diagnosis of coronary artery FMD is difficult, as no pathognomonic disease symptoms are present, while diagnostic criteria are still not available. Based on investigators, renal, carotid, and vertebral arteries are most commonly affected [14]. FMD of coronary arteries is mainly characterized by lesions of middle and distal segments, with smooth lumen narrowing. FMD is often detected when other arterial territories are affected. In this case, proximal AIVA segment was the one affected. CT-angiography and DUS did not detect stenosis, aneurysms, tortuosity in other vascular territories.

Based on literature, multi-vessel dissection with the LCA trunk involvement and higher percent of complications were reported when SCAD was detected in pregnancy [15]; however, pregnancy was excluded in our patient.

No specific data were reported for non-specific aortoarteriitis (NAA, Takayasu disease), as no significant difference in blood pressure values and pulse features in both upper and lower extremities was observed. No angiographic signs of aortic pathology were detected. Laboratory NAA markers are not developed; however, according to clinical guidelines, increased immunoglobulin, C3-complement, anti-cardiolipin and b2-glycoprotein Ab levels are possible [16], which were not confirmed in our patient. Titers of antinuclear antibodies (Abs), anti-myeloperoxidase, protease, cardiolipin Abs in our patient did not exceed acceptable values, i.e. no systemic vasculitis was detected.

Several genetic connective tissue diseases are associated with SCAD (Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome (type 4), Alport syndrome, polycystic kidney disease, osteogenesis imperfecta), but they form a small proportion among all SCAD cases [17], and these were not diagnosed in our case. Vascular dissection or rupture may manifest as a vascular syndrome in non-differentiated connective tissue dysplasia (NCTD), which is most commonly detected among females of reproductive age [18].

Based on the histology results in the clinical case discussed, the destructive process was detected in the media of coronary arteries and aorta together with vacuolization. This description confirms altered architectonics of the elastic carcass, defect of fibrous structures and proper substance of the connective tissue that form the basis for dysplasia pathogenesis.

28 syndromes have been currently described in NCTD [19]. A history of cardialgia (one of the leading symptoms in NCTD), dolichosigmoid (syndrome of digestive system pathology), decreased C3-complement and immunoglobulin (including IgA) levels (immunological disorder syndrome) may indirectly define this disease in our patient. Smetanin M. Yu. et al. (2018) demonstrated that patients of reproductive age with connective tissue dysplasia had bone metabolism disorders with deficiency of vitamins, macro- and microelements participating in bone mineralization, collagen synthesis and maturation [20]. Decreased plasma Ca2+ levels were diagnosed in a patient. Detected alterations can allow to suspect NCTD syndrome in the patient.

Genetic screening was not arranged due to the current absence of clear data on those hereditary diseases that may lead to SCAD and insufficiently analyzed genetic basis of SCAD. An association has been established for adverse NCTD cardiovascular manifestations with the homozygous T80807T phenotype of the SP4 polymorphic gene, homozygous AA phenotype of the β-1 adrenoceptor polymorphic gene, G allele of the MMP9 polymorphic gene (-8202 A/G), heterozygous 5A/6A phenotype of the MMP3 polymorphic gene [21]. Data demonstrating the association of SCAD with the PHACTR1/EDN1 locus have also been discovered. Antonutti M. et al. (2021) described gene mutations in COL3A1, COL5A2, FBN2, LTBP2, NOTCH1, ELN genes, as well as their associations with the major adverse cardiovascular events, including SCAD relapse, cardiogenic shock, and heart failure [22].

According to Kotecha D. et al. (2021), wide dissection area and location in proximal segments are associated with a higher risk of complications, while the simultaneous dissection of more than one artery may be associated with worse prognosis compared to patients with single-vessel lesion [23]. According to an expert opinion, SCAD treatment in stable patients without relapsing chest pain should be conservative. Revascularization is considered in high-risk patients who correspond to at least one of the following criteria: unstable hemodynamics, cardiogenic shock, ventricular arrhythmias, ventricular fibrillation, persisting and recurrent SCAD, intramural hematoma > 10 mm long or its continued prolongation, LCA trunk dissection, prolonged proximal dissection of AIVA, circumflex artery (CxA) or RCA, ostium dissection of AIVA, multi-vessel dissection [24]. The patient discussed in the clinical was in the high-risk group of poor outcomes due to proximal AIVA dissection, unstable hemodynamics, and cardiogenic shock. Successful revascularization was arranged, ~~but~~ it did not affect the prognosis.

Conclusion

Spontaneous coronary artery dissection (SCAD) remains a rare, but potentially life-threatening condition, which diagnosis and management are rather difficult, especially in young females without traditional risk factors. With the limited evidence and absence of general treatment standards, the choice between the conservative and invasive tactics requires individual approach based on clinical signs, hemodynamic patient stability, angiography data, and the hospital capabilities. The presented clinical case underlines the importance of multidisciplinary interactions and required further studies aimed at compiling clear recommendations on the management of this patient category.

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