ejpmr, 2024, 11(12), 74-78

SJIF Impact Factor 7.065

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Review Article

ISSN 2394-3211

EJPMR

CLINICAL CASE OF ACUTE CORONARY SYNDROME IN THE PRESENCE OF METABOLIC SYNDROME

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Article Received on 14/10/2024 **Article Revised on 04/11/2024** **Article Accepted on 25/11/2024**

Patient M., 55 years old, employed Medical Record No. 3168/09
Date of Admission: 28.05.2023 Date of Discharge: 09.06.2023

Complaints on Admission: Severe constricting chest pain radiating to the left shoulder blade, palpitations, marked weakness, and sweating.

Medical History: The patient has had arterial hypertension for 7 years, with a maximum blood pressure (BP) reading of 170/100 mmHg, typically controlled at 130/90 mmHg. She has experienced exertional angina (Class II) for approximately 2 years. She denies any history of myocardial infarction (MI) or cerebrovascular accidents (CVA). She has been hospitalized twice for exacerbations of coronary artery disease (CAD), diagnosed as "unstable angina," and has refused the recommended coronary angiography. Her outpatient medications include Arifon Retard 1.5 mg, Thrombo ASS 100 mg, Enalapril 10 mg/day, and sublingual Isoket spray for angina attacks. She denies diabetes mellitus or other glucose metabolism disorders and does not monitor her blood glucose levels.

Current exacerbation began about 24 hours prior, following emotional stress. She noticed an increase in the frequency and intensity of anginal attacks, the onset of rest angina, and reduced exercise tolerance. There has been a significant increase in the need for sublingual nitrates. Emergency Medical Services (EMS) were called, and the patient was admitted to the cardiac intensive care unit with a preliminary diagnosis of CAD: unstable angina. Prehospital treatment included heparin, Isoket, and aspirin. Comorbid conditions: chronic gastritis, spinal osteochondrosis. Habits: Non-smoker, does not abuse alcohol.

Objective Examination: The patient is in moderate condition. Body weight is 96.2 kg, with a Body Mass Index (BMI) of 31.8. Waist-to-Hip Ratio (WHR) is 0.92. Skin is of normal color and moisture. No cyanosis or peripheral edema observed. Lung sounds are vesicular, audible in all areas, with no rales. Respiratory rate is 19 breaths per minute. Heart sounds are muffled, with a regular rhythm. Heart rate is 78 beats per minute, and blood pressure is 160/90 mmHg. The abdomen is soft. The liver is palpable at the edge of the costal margin. The kidney area appears normal upon inspection, with a negative percussion test for kidney tenderness. No focal or meningeal symptoms are present.

ECG Findings: Sinus rhythm, regular, with a heart rate of 76 beats per minute. The electrical axis of the heart is in a horizontal position. Changes in the terminal portion of the ventricular complex are noted in the right chest leads: horizontal ST-segment depression in leads V3-V5 and an inverted T wave in leads V2-V3.

Clinical Blood Analysis: Hemoglobin - 132 g/L, Erythrocytes - 4.0 x 10^12/L, Platelets - 225 x 10^9/L, Leukocytes - 7.3 x 10^9/L, ESR - 12 mm/h.

Urinalysis: Unremarkable.

Biochemical Blood Analysis: Total cholesterol - 5.7 mmol/L, LDL - 4.9 mmol/L, HDL - 0.70 mmol/L, Triglycerides - 1.85 mmol/L, VLDL - 3.76 mmol/L, Uric acid - 440 µmol/L, Max CK - 229 U/L, Max LDH - 401 U/L, Blood glucose on admission - 7.5 mmol/L, Fasting blood glucose (01.06.09, 7:00 am) - 6.1 mmol/L, Postprandial glucose (2 hours after breakfast) - 7.0 mmol/L, Glycated hemoglobin (HbA1c) - 5.8%.

All other parameters are within normal limits.

Admission Diagnosis: CAD: Unstable angina, progressive course. Atherosclerotic cardiosclerosis. CHF I, Functional Class II (NYHA). Hypertension, Grade 2, Risk 4. Obesity, Class I. Dyslipidemia (Type IV by Fredrickson). Impaired glucose tolerance.

Hyperuricemia. Chronic gastritis, remission. Widespread spinal osteochondrosis.

Echocardiography (EchoCG): Aorta thickened, diameter 3.5 cm; Left atrium - 3.5 cm; Interventricular septum (IVS) - 1.2 cm; Posterior wall (PW) - 1.22 cm; End-diastolic volume (EDV) - 144.6 mL; End-systolic volume (ESV) - 89.9 mL; Left ventricular ejection fraction (LVEF) - 52%; Myocardial contractility - no regional wall motion abnormalities; Mitral regurgitation - Grade 1. Doppler Findings: E-wave - 0.48 m/s; A-wave - 0.60 m/s; E/A ratio - 0.80; Diastolic dysfunction - impaired relaxation type.

Holter Monitoring Results: Average HR - 72 bpm; maximum HR - 131 bpm; minimum HR - 66 bpm. A total of 212 ventricular extrasystoles recorded, with 1 episode of ventricular bigeminy. Supraventricular ectopic activity included 15 isolated extrasystoles. Three episodes of ST segment depression were observed (during active periods), with a maximum duration of 8 minutes.

Oxidant and Antioxidant System Indicators: Concentration of AGP - primary products of lipid peroxidation (LPO) in blood serum: 6.28 nmol/mg lipid, indicating increased lipid peroxidation. Activity of erythrocyte antioxidant enzymes: SOD - 21.8 units/mg Hb, GPx - 2.1 units/mg Hb, showing reduced activity of antioxidant defense enzymes. Total antioxidant activity of blood serum (TAA): 54.1 mM ascorbic equivalent, TAA within normal limits.

Indicators of Endothelial Dysfunction and Systemic Inflammation:
Von Willebrand factor: 149% (elevated activity of vWF)
C-reactive protein (CRP) in blood serum: 17 mg/L (elevated)
Fibrinogen: 402 mg/dL (elevated)

The patient received the following therapy Plavix, Heparin, Isoket, Concor, Torvacard, Cardiomagnyl, Noliprel. During hospitalization, the patient experienced recurrent anginal attacks requiring additional sublingual nitrates, with no progression of heart failure symptoms, and BP stabilized at 120-130/80 mmHg. Pre-discharge comprehensive examination results:

ECG: Sinus rhythm, HR 66 bpm. ST segment at baseline, negative T wave in leads AVL, V2-V3.

EchoCG: Aorta thickened, diameter 3.6 cm; Left atrium - 3.8 cm; IVS - 1.2 cm; PW - 1.2 cm; EDV - 150 mL; ESV - 86.3 mL; LVEF - 59%; Mitral regurgitation - Grade 1; E-wave - 0.61 m/s; A-wave - 0.42 m/s; E/A ratio - 1.45; DTe - 150 ms; IVRT - 84 ms; Diastolic dysfunction - pseudonormal type.

Holter ECG Monitoring: Average HR - 68 bpm; maximum HR - 100 bpm; minimum HR - 56 bpm; 73 ventricular and 8 supraventricular extrasystoles recorded; no significant ST segment depression episodes; ventricular arrhythmia classified as Grade 1 by Lown-Wolf.

Biochemical Blood Analysis Total cholesterol - 5.5 mmol/L, LDL - 2.9 mmol/L, HDL - 1.1 mmol/L, Triglycerides - 1.7 mmol/L, ALT - 39 U/L, AST - 56 U/L.

OGTT: Fasting glucose - 6.0 mmol/L, glucose 2 hours after 75g glucose intake - 7.3 mmol/L; fasting insulinemia - 12.3µU/mL, insulinemia 2 hours after 75g glucose intake - 35.8 µU/mL. HOMA-IR - 3.28.

Oxidant and Antioxidant System Indicators: Concentration of AGP - primary products of lipid peroxidation in blood serum: 7.35 nmol/mg lipid, indicating a slight increase in lipid peroxidation.

Activity of erythrocyte antioxidant enzymes: SOD - 28.9 units/mg Hb, GPx - 3.0 units/mg Hb, showing a significant increase in antioxidant enzyme activity. Total antioxidant activity of blood serum (TAA): 58.9 mM ascorbic equivalent, TAA within normal limits.

Total antioxidant activity of blood serum (TAA): 58.9 mM ascorbic equivalent, TAA within normal limits.

Indicators of Endothelial Dysfunction and Systemic Inflammation: Von Willebrand factor: 126% (values decreasing but still above normal).

C-reactive protein (CRP) in blood serum: 11 mg/L (elevated). Fibrinogen: 400 mg/dL.

Recommended Treatment: Cardiomagnyl 75 mg/day, Concor 5 mg/day, Noliprel, Torvacard 20 mg/day, Cardiket 40 mg twice daily.

During the year-long outpatient follow-up period, the patient felt generally satisfactory. She occasionally experienced angina attacks during moderate physical exertion, which were alleviated by taking nitroglycerin.

A repeat comprehensive examination was conducted after one year:

ECG: Sinus rhythm, HR 68 bpm, horizontal QRS axis. ST segment at baseline, negative T wave in leads aVL, V2, V3.

EchoCG: Follow-up showed no significant changes in echocardiographic parameters, with persistent pseudonormal type diastolic dysfunction.

Holter ECG Monitoring: Three orthogonal leads (X, Y, Z) were used, and ECG data (excluding artifacts) was analyzed over 19 hours. Average HR - 70 bpm; maximum HR - 110 bpm; minimum HR - 48 bpm.

Recorded 68 ventricular and 5 supraventricular extrasystoles, with no significant ST segment depression episodes. Ventricular arrhythmia classified as Grade 1 by Lown. A reduction in heart rate and a decrease in ventricular and supraventricular ectopic activity were observed on therapy.

Biochemical Blood Analysis: Total cholesterol - 4.3 mmol/L, LDL - 2.7 mmol/L, HDL - 1.3 mmol/L, Triglycerides - 1.6 mmol/L, ALT - 38 U/L, AST - 25 U/L.

OGTT: Fasting glucose - 6.0 mmol/L, glucose 2 hours after 75g glucose - 7.3 mmol/L; fasting insulin - 12.3 µU/mL, insulin 2 hours after 75g glucose - 35.8 µU/mL. HOMA-IR - 3.28.

Oxidant and Antioxidant System Indicators

•AGP concentration (primary lipid peroxidation products): 7.6 nmol/mg lipid, indicating a decrease though still above "normal" levels.
•Erythrocyte antioxidant enzyme activity: SOD - 27.5 units/mg Hb, GPx - 2.8 units/mg Hb, showing decreased antioxidant enzyme activity.
•Total antioxidant activity of blood serum (TAA): 56.0 mM ascorbic equivalent, within normal range.

There is a trend toward balance restoration between oxidant and antioxidant systems, although lipid peroxidation remains dominant.

Endothelial Dysfunction and Systemic Inflammation Indicators

•Von Willebrand factor: 122.8%
•C-reactive protein (CRP): 6 mg/L
•Fibrinogen: 390 mg/dL

Significant reductions in Von Willebrand factor activity and CRP levels were observed, although values remain above normal.

This clinical case demonstrates that the combined pathology of Acute Coronary Syndrome (ACS) and Metabolic Syndrome (MS) adversely affects microcirculation, oxidant and antioxidant systems, and endothelial function in patients with acute coronary heart disease. There is an increase in spasmodic events and the appearance of congestive components in microcirculation, increased lipid peroxidation, reduced antioxidant enzyme activity, sustained hyperactivation of neurohumoral systems, pro-inflammatory state, and endothelial dysfunction—all contributing to impaired microhemodynamics and predicting a poor prognosis.

The observed correlations in this study between insulin resistance, Von Willebrand factor activity, microcirculation indicators, lipid peroxidation, and CRP levels are confirmed by this case. In the outcome of ACS, the patient exhibited high lipid peroxidation activity, low antioxidant defense, elevated Von Willebrand factor activity, and increased CRP concentration.

Clinical Case #2

Patient C., 65 years old, employed Medical Record No. 4120/09

Complaints on Admission: Severe, pressing chest pain radiating to the left arm, "cold sweat," and weakness.

Medical History: The patient has a 15-year history of arterial hypertension with peak BP reaching 180/100 mmHg, typically controlled at 130/70 mmHg. For the past 10 years, he has experienced occasional pressing chest pain during moderate physical exertion, relieved by rest. No history of myocardial infarction (MI) or stroke. Previous outpatient medications included Cardiket, Enalapril, Thrombo ASS, Vazocardin, and Amlotop. The current deterioration began at 4:00 am on 15.03.2023, with intense, pressing chest pain and "cold sweat." Emergency Medical Services (EMS) were called, and fentanyl and intravenous nitrate infusion were administered. The patient was admitted to the cardiac intensive care unit.

Comorbid Conditions: Peptic ulcer disease of the duodenum (2006), in remission.

Lifestyle: Never smoked, does not abuse alcohol.

Objective Examination on Admission: Moderate condition, BMI = 23.6. Pale skin, cyanosis of lips and acrocyanosis. No peripheral edema. Vesicular breath sounds, respiratory rate 19/min, regular heart rhythm with muffled tones, HR 64 bpm, BP 110/70 mmHg. Soft, non-tender abdomen, liver at costal margin. No focal or meningeal symptoms.

ECG: Sinus rhythm, HR 52 bpm. QS complex with ST-segment elevation in leads V1-V5, ST elevation in I, aVL, V6, flattened T-wave in III, aVF. Anterior fascicular block of the left bundle branch.

Clinical Blood Analysis: Hemoglobin - 138 g/L, Erythrocytes - 4.2 x 10^12/L, Platelets - 310 x 10^9/L, Leukocytes - 12 x 10^9/L, ESR - 14 mm/h.

Urinalysis: Unremarkable.

Biochemical Blood Analysis: Total cholesterol - 5.5 mmol/L, LDL - 3.7 mmol/L, HDL - 0.5 mmol/L, Triglycerides - 2.8 mmol/L, VLDL - 3.7 mmol/L, Uric acid - 398 µmol/L, Max CK - 1635 U/L, Max LDH - 1021 U/L, Blood glucose on admission - 7.1 mmol/L, Fasting blood glucose (18.03.09, 7:00 am) - 5.8 mmol/L, Glycated hemoglobin - 4.1%. Other parameters within normal limits.

Admission Diagnosis: CAD: Acute anterior Q-wave myocardial infarction from 15.03.2023. Post-infarction cardiosclerosis. Anterior fascicular block of the left

bundle branch. CHF IIA, NYHA Functional Class III. Hypertension, Grade 3, Risk 4. Peptic ulcer disease in remission. Dyslipidemia Type II B (Fredrickson).

Comprehensive Examination Results

EchoCG: Aorta - thickened, 3.4 cm in diameter; Left atrium - 3.5 cm; IVS - 1.1 cm; PW - 1.2 cm; EDV - 146.8 mL; ESV - 73.2 mL; LVEF - 49%. Hypokinesis of mid (lateral, anterior, anterior-septal) segments. Akinesis of apical (lateral, anterior, septal) segments. Wall motion score index - 1.56. Sphericity index - 0.48. E - 0.49 m/s, A - 0.62 m/s, E/A - 0.79, DTe - 230 ms, IVRT - 108 ms. Diastolic dysfunction with impaired relaxation.

Holter ECG Monitoring: Average HR - 56 bpm, maximum HR - 120 bpm, minimum HR - 51 bpm. Recorded 40 ventricular and 73 supraventricular extrasystoles. One run of supraventricular tachycardia with a maximum HR of 120 bpm for 5 complexes. No significant ST-segment changes.

Microcirculation (LDF): PM - 4.7 perfusion units; ASp/PM - 5.9 perfusion units; ABp/TSCO - 57.2%; ANp/TSCO - 26.3%; ASp/TSCO - 13.4%; IEM - 1.28 perfusion units. Predominance of spastic component in microcirculation.

Oxidant and Antioxidant System Indicators: AGP concentration - 6.9 nmol/mg lipid, indicating increased lipid peroxidation. Erythrocyte antioxidant enzyme activity: SOD - 30.1 units/mg Hb, GPx - 3.4 units/mg Hb, showing slight reduction in antioxidant defense. TAA - 104.2 mM ascorbic equivalent, indicating high total antioxidant activity.

Endothelial Dysfunction and Systemic Inflammation Indicators: Von Willebrand factor - 167.8% (elevated), CRP - 27 mg/L (elevated), Fibrinogen - 420 mg/dL (elevated).

Therapy: Heparin, Isoket, Pentoxifylline, Egilok, Simvastol. Thrombolytic therapy was not administered due to peptic ulcer history. Hospital stay was uneventful.

Discharge Examination

ECG: Sinus rhythm, HR 64 bpm, anterior fascicular block of the left bundle branch. Pathological Q wave in leads V1-V5, persistent ST elevation in V2-V4 with negative T wave in I, aVL, V2-V3.

Repeat EchoCG: Aorta - thickened, 3.5 cm; Left atrium - 3.5 cm; IVS - 1.1 cm; PW - 1.2 cm; EDV - 154.6 mL; ESV - 79.3 mL; LVEF - 53%. Hypokinesis of mid (lateral, anterior, anterior-septal) segments, aneurysm at the left ventricular apex. Wall motion score index - 1.5. Sphericity index - 0.53. E - 0.53 m/s, A - 0.61 m/s, E/A - 0.86, DTe - 220 ms, IVRT - 90 ms. Diastolic dysfunction with impaired relaxation.

Holter Monitoring: Average HR - 57 bpm, maximum HR - 95 bpm, minimum HR - 50 bpm. Recorded 28 isolated supraventricular extrasystoles. No significant ST-segment changes or ventricular ectopic activity.

Biochemical Blood Analysis: Total cholesterol - 4.6 mmol/L, LDL - 2.9 mmol/L, HDL - 1.0 mmol/L, Triglycerides - 2.2 mmol/L, ALT - 45 U/L, AST - 41 U/L. Blood glucose (several measurements): 6.2, 5.4, and 6.6 mmol/L.

Microcirculation: Improved blood flow with reduced spastic phenomena.

Oxidant and Antioxidant System: AGP - 7.4 nmol/mg lipid, increased lipid peroxidation continues. High antioxidant enzyme activity: SOD - 40.6 units/mg Hb, GPx - 4.8 units/mg Hb, with increased TAA (112.3 mM ascorbic equivalent).

Endothelial Dysfunction and Systemic Inflammation: Von Willebrand factor - 134.8%, CRP - 9 mg/L, Fibrinogen - 360 mg/dL.

Annual Follow-Up

Final Comprehensive Examination (one year): Mild dyspnea with exertion; no chest pain, no use of nitroglycerin. Overall satisfactory condition. No peripheral edema.

ECG: Sinus rhythm, HR 65 bpm, anterior fascicular block of the left bundle branch, pathological Q wave in V1-V3, persistent ST elevation in V2-V4, negative T wave in V2-V3.

EchoCG: EDV - 151.3 mL; ESV - 75.4 mL; LVEF - 54%. Hypokinesis of mid segments, apical aneurysm of LV. Sphericity index - 0.51. E/A - 1.1, DTe - 210 ms, IVRT - 90 ms. Slight reduction in heart size with improved LV contractility and diastolic function.

Holter Monitoring: Average HR - 58 bpm, maximum HR - 100 bpm, minimum HR - 49 bpm. No ventricular ectopic activity, 10 isolated supraventricular extrasystoles. No pauses >2.5 sec or arrhythmia detected.

Blood Biochemistry: Total cholesterol - 4.5 mmol/L, LDL - 2.2 mmol/L, HDL - 1.0 mmol/L, Triglycerides - 1.8 mmol/L, ALT - 20 U/L, AST - 21 U/L, fasting glucose - 5.5 mmol/L.

Microcirculation: No significant microcirculation abnormalities.

Oxidant and Antioxidant System: AGP - 5.6 nmol/mg lipid, reduced lipid peroxidation, though not normalized. Antioxidant enzyme activity: SOD - 28.9 units/mg Hb, GPx - 3.6 units/mg Hb. TAA - 99.6 mM ascorbic equivalent. Balance trend between oxidant and antioxidant systems observed.

Endothelial Dysfunction and Systemic Inflammation: Von Willebrand factor - 87%, CRP - 5 mg/L, Fibrinogen - 380 mg/dL, showing significant improvements.

CONCLUSION

Over 12 months, positive changes in central hemodynamics, normalization of LV diastolic function, improved microcirculation, reduced oxidative stress, and endothelial dysfunction were observed. The absence of metabolic syndrome contributed to a more favorable disease course, more rapid stabilization of macro- and microhemodynamic parameters, balanced oxidant and antioxidant systems, reduced endothelial dysfunction, and slower LV post-infarction remodeling. This case demonstrates that the lack of metabolic syndrome positively influences disease outcomes and decreases the risk of adverse cardiovascular events.