

Received: 01 February 2025 / Accepted: 25 August 2025 / Published online: 30 October 2025

DOI 10.34689/SH.2025.27.5.032

UDC 616-08-07



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A CASE REPORT OF MYOCARDIAL INFARCT PROCESSED ON POLYMORBID BACKGROUND

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Introduction. Polymorbidity in the form of uncontrolled arterial hypertension, heart failure, severe out-of-hospital polisegmental pneumonia complicated by pleurisy and respiratory failure significantly affects the tactics of myocardial infarction management in polypragmatic conditions and worsen prognosis. Additional difficulties in myocardial infarction management are: Dressler syndrome, recurrent course of infarction and ventricular fibrillation, repeated interventions (coronarography and stenting), contrast induced nephropathy on the background of chronic heart failure with low ejection fraction and pulmonary hypertension. **Material and methods.** Analysis of the medical record data of patient B, 58 years old. **Results and discussion:** This case highlights the challenges faced by physicians in the management of patients with myocardial infarction on polymorbid background, requiring simultaneous intensive treatment of myocardial infarction and combined diseases, which increases the risk of circulatory instability in conditions of forced polypragmasy. **Conclusion:** This case demonstrates the need for active identification of patients with CHD at the outpatient stage and interdisciplinary approach including general practitioners, internists, cardiologists, cardiac surgeons, pulmonologists, intensive care specialists and nephrologists.

Key words: myocardial infarction, polymorbidity, polypragmasy.

For citation:

Pashimov M.O., Zhangelova Sh.B., Kapsultanova D.A., Danyarova L.B., Sakhov O.S., Nurmukhammad F.N., Rakhmetulaeva E.B., Kydykenova A.T. A clinical case of myocardial infarct processed on polymorbid background // *Nauka i Zdravookhranenie* [Science & Healthcare]. 2025. Vol.27 (5), pp. 274-279. doi 10.34689/SH.2025.27.5.032

Резюме

КЛИНИЧЕСКИЙ СЛУЧАЙ ИНФАРКТА МИОКАРДА, ПРОТЕКАЮЩЕГО НА ПОЛИМОРБИДНОМ ФОНЕ

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Введение. Полиморбидность в виде неконтролируемой артериальной гипертензии, сердечной недостаточности, тяжелой внебольничной полисегментарной пневмонией, осложненной плевритом и дыхательной недостаточностью существенно влияет на тактику ведения инфаркта миокарда в условиях полипрагмазии и ухудшают прогноз. Дополнительные сложности в управлении инфарктом миокарда представляют: синдром Дресслера, рецидивирующее течение инфаркта и фибрилляции желудочков, повторные вмешательства (коронарографии и стентирования), контраст индуцированная нефропатия на фоне хронической сердечной недостаточности с низкой фракцией выброса и легочной гипертензии. **Материал и методы.** Анализ данных медицинской карты пациента В, 58 лет. **Результаты и обсуждение:** Данный случай подчеркивает вызовы, с которыми сталкиваются врачи при ведении пациентов с инфарктом миокарда на полиморбидном фоне, требующих одновременного интенсивного лечения инфаркта миокарда и сочетанных заболеваний, что повышает риск нестабильности кровообращения в условиях вынужденной полипрагмазии. **Вывод:** Этот случай демонстрирует необходимость активного выявления пациентов с ИБС на амбулаторном этапе и междисциплинарного подхода, включающего врачей общей практики, терапевтов, кардиологов, кардиохирургов, пульмонологов, реаниматологов и нефрологов.

Ключевые слова: инфаркт миокарда, полиморбидность, полипрагмазия.

Для цитирования:

Пашимов М.О., Жангелова Ш.Б., Капсултанова Д.А., Даньярова Л.Б., Сахов О.С., Нурмухаммад Ф.Н., Рахметулаева Э.Б., Кыдыкенова А.Т. Клинический случай инфаркта миокарда, протекающего на полиморбидном фоне // Наука и Здоровоохранение. 2025. Т.27 (5), С. 274–279. doi: 10.34689/SH.2025.27.5.032

Түйіндеме

ПОЛИМОРБИДТІ ФОНДА ӨТЕТІН МИОКАРД ИНФАРКТІНІҢ КЛИНИКАЛЫҚ ЖАҒДАЙЫ

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Кіріспе. Бақыланбайтын артериялық гипертензия, жүрек жеткіліксіздігі, плевритпен және тыныс алу жеткіліксіздігімен асқынған ауруханадан тыс полисегментарлық пневмония түріндегі полиморбидтілік полипрагмазия жағдайларында миокард инфарктісін жүргізу тактикасына елеулі әсер етеді және болжамды нашарлатады. Миокард инфарктінің басқарудағы қосымша қиындықтар: Дресслер синдромы, инфарктінің және қарыншалардың фибрилляциясының қайталанатын ағымы, қайталама араласулар (коронарография және стенттеу), төмен шығарынды фракциясы мен өкпе гипертензиясы бар созылмалы жүрек функциясының жеткіліксіздігі фонындағы индукцияланған нефропатияның контрастысы. **Материал және әдістер.** В, 58 жастағы пациенттің медициналық картасының деректерін талдау. **Нәтижелері және талқылауы:** Бұл жағдай мәжбүрлі полипрагмазия жағдайларында қан айналымының тұрақсыздығы қаупін арттыратын миокард инфаркті және аралас ауруларды бір мезгілде қарқынды емдеуді талап ететін полиморбидті фонда миокард инфарктімен ауыратын пациенттерді емдеу кезінде дәрігерлер кездесетін сын-қатерлерді баса көрсетеді. **Қорытынды:** Бұл жағдай жалпы практика дәрігерлерін, терапевтерді, кардиологтарды, кардиохирургтарды, пульмонологтарды, реаниматологтарды және нефрологтарды қамтитын амбулаториялық кезеңде және пәнаралық тәсілмен ЖИА бар пациенттерді белсенді анықтау қажеттілігін көрсетеді.

Түйінді сөздер: миокард инфарктісі, полиморбидтілік, полипрагмазия.

Дәйексөз үшін:

Пашимов М.О., Жангелова Ш.Б., Капсултанова Д.А., Даньярова Л.Б., Сахов О.С., Нурмухаммад Ф.Н., Рахметулаева Э.Б., Кыдыкенова А.Т. Полиморбидті фонда өтетін миокард инфарктінің клиникалық жағдайы // Ғылым және Денсаулық. 2025. Т.27 (5), Б. 274–279. doi: 10.34689/SH.2025.27.5.032

Introduction

In recent decades, the proportion of individuals of working age with cardiovascular pathology has been steadily increasing, presenting a significant socio-economic challenge [1,2,3].

The purpose of this clinical report description is to draw attention to the complexity of managing a multimorbid working-age patient with early-onset cardiovascular disease, which presented with recurrent myocardial infarction and severe community-acquired pneumonia. This case is intended to raise clinicians' awareness of the risks of forced polypharmacy and the importance of timely identification and structured outpatient management.

Materials and Methods:

The material is based on the medical records of patient B., 58 years old, who underwent inpatient treatment at the City Cardiology Center in Almaty. Research method: clinical case description.

Research Results:

The patient presented with complaints of episodes of pressing chest pain, dyspnea at rest, and weakness.

Medical history revealed arterial hypertension for several years, with a maximum recorded blood pressure of 160/90 mmHg and an adapted blood pressure of 120/80 mmHg. The patient was not under regular medical follow-up. Worsening of symptoms was noted over the previous two weeks: recurrent pressing chest pain, fever up to 38°C, and dyspnea on minimal physical exertion. The patient had not received any treatment prior to admission.

On June 18, 2024 (date changed for confidentiality), the patient independently sought medical attention at the emergency department of the cardiology center and was urgently hospitalized in the intensive care unit.

Past medical history: No family history of cardiovascular disease. The patient denies any chronic conditions or unhealthy behaviors (e.g., smoking, alcohol use). No history of allergic reactions.

Physical examination: General condition: severe. Position: orthopnea. BMI: 29.1 kg/m² (overweight). Consciousness clear, patient is adequate. Body temperature: 36.5°C. SpO₂: 96%. Respiratory rate: 18/min. Vesicular breath sounds, weakened in the lower lung fields, with fine moist rales bilaterally. Heart sounds muffled, first

sound diminished, regular rhythm. Blood pressure: 130/80 mmHg. Heart rate: 88 bpm. No peripheral edema.

Based on the complaints, history, and objective, laboratory, and instrumental findings, the following were noted:

- History of uncontrolled arterial hypertension;
- Delayed presentation: for two weeks the patient had experienced recurrent chest pain and progressive weakness;
- Fever up to 38°C;
- Clinical signs of myocardial infarction lasting more than two weeks, with elevated troponin I at admission: 2.51 ng/ml and 2.66 ng/ml after 3 hours. The positive result without dynamic increase/decrease supports a diagnosis of myocardial infarction that occurred at least a week prior;
- ECG on 18.06.2024: sinus rhythm, HR 80 bpm. ST-segment depression in leads I, II, III, aVL, aVF, V3–V6. No R wave progression in V1–V4;
- Echocardiography: conducted during arrhythmia. Aortic wall and valve leaflets thickened. Dilation of the left heart chambers. Left ventricular (LV) myocardial hypertrophy. Hypo- and akinesis of the LV apex. Diffuse hypokinesis of other LV walls. LV systolic function reduced (Simpson's EF 32%), right ventricular function preserved. Pericardial effusion: ~100 ml. Mild pulmonary hypertension: systolic pulmonary artery pressure 26 mmHg. Pleural effusion: right side 460 ml, left side 660 ml;
- Dyslipidemia on admission: Total cholesterol – 5.12 mmol/L; HDL – 0.91 mmol/L; LDL – 3.92 mmol/L; Triglycerides – 1.46 mmol/L;
- Pneumonia symptoms: febrile syndrome, progressive inflammatory markers (Table 1), elevated procalcitonin – 2.43 ng/ml; D-dimer – 1600 ng/ml; fibrinogen – 7.44 g/L. Chest X-ray: multisegmental pneumonia (50% lung involvement). Positive dynamics with combined parenteral antibiotic therapy (ceftriaxone 2 g/day IV and levofloxacin 500 mg/day IV), as shown by decreased CRP and a slight reduction in band neutrophils (Table 1);
- Pleural effusion findings: moderate bilateral effusion on ultrasound (19.06.2024, 12:22) – ~400 ml on the right, ~380 ml on the left;
- Elevated creatinine and reduced GFR – indicating contrast-induced nephropathy following coronary angiography.

Table 1.

Dynamics of Laboratory Parameters Over Time.

Parameter	18.06.2024	19.06.2024	28.06.2024	30.06.2024	02.07.2024
Leukocytes	11,19 x10 ⁹ /л	16,70 x10 ⁹ /л	13,35 x10 ⁹ /л	12,04 x10 ⁹ /л	13,68 x10 ⁹ /л
Band neutrophils (%)	10	23	14	14	12
Eosinophils (%)	8	6	6	5	4
ESR (mm/hour)	36,06	31,65	57,79	71,46	53,50
Creatinine (μmol/L)	84	84	260	107	84
GFR(CKD-EPI) (mL/min/1.73 m ²)	88	88	22	66	88
Potassium (mmol/L)	2,8	4,2	3,2	3,6	3,6
hs-CRP (mg/L)	70,70	80,86	87,39	114,05	70,70

- **Persistent hypokalemia** (Table 1);

- **Ultrasound Doppler (03.07.2024):** Echo signs of atherosclerotic lesions in the brachiocephalic arteries and

lower extremity arteries, with atherosclerotic plaques causing up to 37% luminal stenosis.

Final Diagnosis:

Primary diagnosis: Ischemic heart disease. Primary anterior extensive non-ST elevation myocardial infarction (NSTEMI) dated 12.06.2024, recurrent course (date changed for confidentiality, likely occurred one week prior to admission). Type 1.

Procedure: Myocardial revascularization via stenting of the left anterior descending (LAD) and circumflex artery (LCx) on 18.06.2024 (day of admission).

Concurrent disease: Community-acquired bilateral multisegmental pneumonia of mixed etiology (hemodynamic and inflammatory), severe course.

Complications: Acute heart failure, type 1 (ESC);
Moderate pulmonary hypertension;
Dressler's syndrome;
Pericarditis with mild effusion;
HFrEF (LVEF by Simpson's method 32–39%), NYHA functional class III, ACC/AHA Stage C;
Recurrent myocardial infarction as of 24.06.2024;
Hypokalemia;
Recurrent ventricular fibrillation;
Bilateral exudative pleuritis;
Respiratory failure grade II–III;
Contrast-induced nephropathy.

Procedure on 24.06.2024: Coronary angiography and stenting of the right coronary artery (RCA). Temporary pacemaker implantation.

Procedure on 03.07.2024: Implantation of automatic implantable cardioverter-defibrillator (AICD) Biotronik Rivacor 5 DR-T.

Comorbidity:
Grade I arterial hypertension, very high cardiovascular risk;
Secondary dyslipidemia;
Metabolic syndrome;
Ischemic heart disease: multivessel obstructive coronary artery disease;
Multifocal atherosclerosis.

On this multimorbid background, despite potassium supplementation, persistent hypokalemia led to multiple (8 total) episodes of ventricular fibrillation. Rapid normalization of glomerular filtration rate and creatinine levels during therapy confirmed the diagnosis of contrast-induced nephropathy. Sepsis and pulmonary embolism were excluded based on the diagnostic data and clinical dynamics.

Coronary angiography revealed chronic occlusions in four vessels; infarct-related arteries—the LAD and LCx—were stented on 18.06.2024.

On the morning of 24.06.2024, the patient experienced cardiac arrest due to ventricular fibrillation in the cardiology department. After successful resuscitation, he was transferred to the ICU, where a second episode of ventricular fibrillation occurred. Emergency coronary angiography was repeated for life-saving indications. During the procedure, another episode of VF occurred, requiring defibrillation by the resuscitation team. Sinus rhythm was restored and the patient regained consciousness. Given repeated VF episodes and new ST elevation in leads II, III, and aVF, stenting of the chronically occluded RCA was performed. Control angiography showed good angiographic results with full restoration of the vessel lumen and satisfactory TIMI III blood flow.

Due to recurrent episodes of VF, temporary pacemaker implantation was performed on 24.06.2024. A multidisciplinary team decided to implant an AICD (Biotronik Rivacor 5 DR-T) on 03.07.2024 due to:

- Persistent hypokalemia;
- Prolonged QT interval;
- Eight episodes of ventricular fibrillation during hospitalization;
- Reduced left ventricular ejection fraction.

Polypharmacy:

According to the medication chart, the 58-year-old patient received 13–15 drugs per day, including:

- Four medications for heart failure;
- Diuretic;
- Two antibiotics;
- Ibuprofen (later switched to colchicine);
- Intravenous fluid therapy with potassium and magnesium supplementation;
- Proton pump inhibitors;
- Two antiplatelet agents;
- Anticoagulant;
- Statin;
- Nitrates.

Discussion:

This clinical report highlights the complexity of managing a patient with uncontrolled arterial hypertension and a very high cardiovascular risk. Notably, the presence of prolonged chest pain and worsening symptoms over two weeks points to the possibility of a “silent” myocardial infarction, which is commonly seen in patients with hypertension. Upon admission, Dressler's syndrome was diagnosed.

The patient's extremely serious condition was exacerbated by pneumonia and persistent hypokalemia, which led to eight episodes of ventricular fibrillation, despite stenting of three coronary arteries. Coronary angiography confirmed multivessel coronary disease with chronic occlusions, indicating long-standing ischemic heart disease.

Doppler ultrasound of the neck and lower extremity vessels also revealed atherosclerotic plaques. Decreased ejection fraction and left ventricular hypertrophy may have resulted from both ischemic heart disease and long-standing hypertension.

Pneumonia and pleuritis: Laboratory findings (elevated CRP and procalcitonin) and imaging confirmed bilateral multisegmental pneumonia. The etiology is likely mixed—infection and hemodynamic changes—supporting the hypothesis of a combined origin. Pleural effusion could be caused by inflammation, Dressler's syndrome, or heart failure, requiring a comprehensive treatment strategy.

Contrast-induced nephropathy: A sharp rise in creatinine and decline in GFR are consistent with contrast-induced nephropathy, which may also be linked to polypharmacy [4].

In summary, this 58-year-old patient residing in a metropolitan area with access to routine screening for coronary artery disease and hypertension experienced delayed diagnosis of multifocal atherosclerosis during inpatient care.

A key concern often raised by clinical pharmacologists and medical chart reviewers is the presence of

polypharmacy, where patients receive five or more drugs per day. Future research should focus on improving diagnostic and treatment protocols to enhance care for multimorbid patients and to explore new definitions and evaluation standards for justifiable polypharmacy in modern clinical medicine [5,6].

Conclusion:

This clinical report describes a 58-year-old working-age patient with early-onset cardiovascular and multimorbid pathology who required extensive pharmacotherapy. The case emphasizes the importance of interdisciplinary collaboration in managing multimorbid patients, necessitating individualized treatment strategies and continuous monitoring. This approach optimizes therapy and improves prognosis and quality of life in the context of inevitable polypharmacy. It also highlights the need to redefine polypharmacy in the context of contemporary clinical practice.

Prognosis: Extremely serious; scheduled cardiac rehabilitation by a multidisciplinary team is necessary. Further evaluation is required to identify the cause of treatment-resistant hypokalemia.

Informed Consent: Informed consent was obtained for the publication of this clinical case and use of medical data for scientific purposes.

Acknowledgment: We express our gratitude to the staff of the City Cardiology Center of Almaty, under the leadership of Director Roza Tokhtanalievna Kuanishbekova, where the inpatient care was provided.

Funding: This clinical case was prepared as part of the research project "Improving outcomes of surgical myocardial revascularization through the development of an integrated innovative patient management strategy after intervention," funded by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan, Grant No. IRN AP19680319.

Conflict of Interest: The authors declare no potential or actual conflicts of interest that could influence the results or conclusions presented in this manuscript.

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