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CASE REPORT

Acute Pulmonary Embolism, Hemorrhagic Pulmonary Infarction and Ischemic Myocardial Infarction — A Case Report

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Abstract

Deep vein thrombosis (DVT) of the lower extremities often leads to one of the most life-threatening complications such as thromboembolism of the branches of the pulmonary artery. The article describes a clinical case of acute pulmonary embolism with myocardial and lung infarction with a premorbid background after traumatic injury to the deep veins of the left leg. A 57-year-old patient was admitted to our hospital with chest pain that was not relieved by nitrates for 24 hours. The patient was regularly followed by her general practitioner for coronary artery disease and arterial hypertension. The patient had a traumatic injury of the left lower limb about three weeks ago. Later, a trophic ulcer developed at the site of the injury. Death occurred one hour after admission. Dense, easily crumbling blood clots were visualized in the deep veins of the left leg. Numerous thrombi sized from 0.3 to 1.5 cm and up to 2.1 cm long were found in the large, lobar and segmental branches of the pulmonary artery. Microscopic

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signs of acute transmural myocardial infarction of the anterior left ventricular wall were detected with areas of necrotized cardiomyocytes with a demarcation zone of inflammation and undulating deformation of cardiomyocytes. Histological examination revealed necrotic areas of lung tissue impregnated with erythrocytes in the lungs. Such a combined pathology is extremely rare. A sequence of changes is traced not only in the vessels, but also in organs with the development of different types of infarction. This combination of different types of infarction was probably associated with pulmonary hypertension and concomitant diseases such as coronary artery disease, arterial hypertension and atherosclerosis.

Key words: *veins, thrombosytes, thrombotic embolism, pulmonary infarction, myocardial infarction.*

Conflict of interests: none declared.

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Introduction

Lower extremity deep vein thrombosis (DVT) is still one of the major challenges of modern medicine as it is associated with high levels of mortality and disability. According to Russian Phlebology Association and Russian Association of Surgeons DVT cases occur for the first time in 50–70 per 100 000 each year. In 32–45 % of cases, lower extremity DVT is complicated by massive pulmonary embolism (PE) that is fatal in 17 % of patients [1]. Deep vein thrombosis develops when fibrin clots accumulate in the areas where the vessels are damaged and the coagulation cascade activation begins. Activity of antithrombotic proteins such as thrombomodulin and endothelial cell protein C receptor is suppressed and the thrombus is formed [2, 3, 4, 5]. Poor outcomes are associated with co-existent diseases such as coronary artery disease (CAD), arterial hypertension (AH), atherosclerosis as they lead to hypoxic cell injury [6]. Hypoxia can also activate procoagulant factors such as tissue factor (TF) and P-selectin (adhesion molecule) expressed in endothelial cells and lead to leukocyte recruitment. TF initiates coagulation together with P-selectin and are the important components of thrombosis [7–9].

Despite the amount of information we have about thrombosis and its complications, clinical features and pathogenesis, the descriptions of pathomorphological changes are quite limited. Therefore, **the objective** of this paper was to describe the case of DVT with acute pulmonary embolism hemorrhagic pulmonary infarction and ischemic myocardial infarction in a patient with co-existent CAD and after traumatic vessel damage.

Materials and methods

We analyzed the available medical records and clinical and morphological characteristics using the histological techniques (hematoxylin and eosin staining).

Results

A 57-year-old patient was admitted to our hospital with chest pain that was not relieved by nitrates for 24 hours. The patient was regularly followed by her general practitioner for coronary artery disease and arterial hypertension. The patient had a traumatic injury of the left lower limb about three weeks ago. Later, a trophic ulcer developed at the site of the injury. The patients didn't seek medical help and tried to relieve her condition with nonsteroidal anti-inflammatory drugs (NSAIDs).

Objective and instrumental findings

Electrocardiography (ECG) results: sinus rhythm, heart rate — 63 beats per minutes, left axis deviation. ST elevation for 5 mm in leads V3 through V6. Signs of acute transmural anterolateral myocardial infarction. **Troponin test:** positive.

Complete blood count: see Table 1 and 2.

C-reactive protein — 100 mg/l (0–5 vg.l). Coagulation studies: fibrinogen 15.6 mmol/l (5.9–11.7 mmol/l), prothrombin index — 186.1 % (80.0–125.0)%, D-dimer — 2297 ng/ml (<250 ng/ml). Alanine aminotransferase — 68 U/L (1.0–45.0) U/L; aspar-

Table 1. **Complete blood count**

Values	Leucocytes	Erythrocytes	Hemoglobin	Thrombocutes	Erythrocyte sedimentation rate
Patient	19×10×9/l	8.64×10×9/l	101 g/l	254×10×9/l	20 mm/h
Reference range	3.80–8.76	4.54–6.00	120–147	173–360	2–8

Table 2. **Leukocyte count and differential**

Values	Segmented neutrophils	Band forms	Neutrophils	Lymphocytes
Patients	77 %	18 %	62.0 %	26.0 %
Reference range	47–72	1–6	40.1–67.0	23.6–48.0

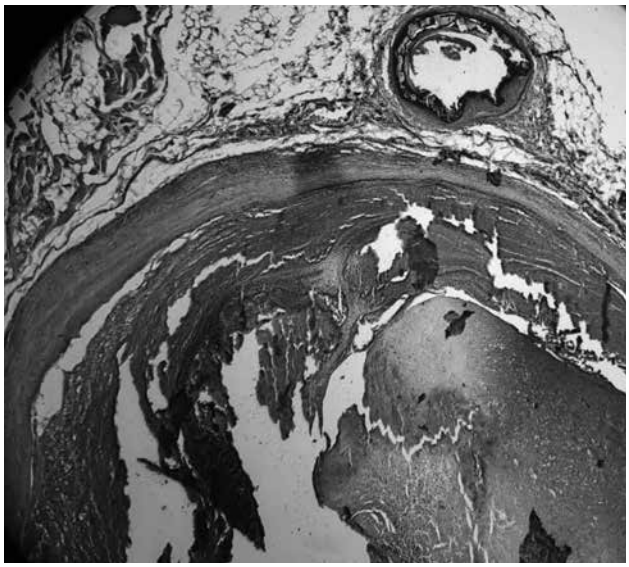
tate aminotransferase — 57 U/L (1.0–35.0) U/L, urea 9.6 (2.8–8.3) mmol/l, creatinine — 94 μmol/l (53–97) μmol /l, glucose 4.86 mmol/l (4.0–5.9 mmol/l), cholesterol — 9 mmol/l (3.1–5 mmol/l).

One hour later the patient acutely deteriorated and died. Post-mortem analysis with pathomorphological evaluation was later performed.

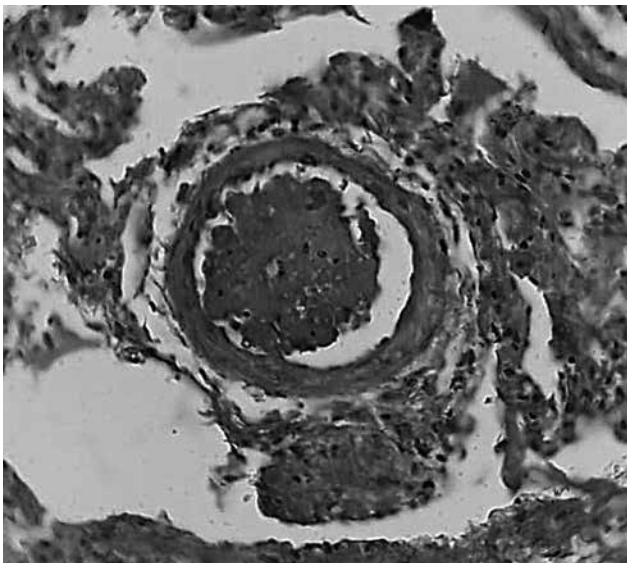
An ulcer 3.2×2.1 cm was found on the anterior surface of the patient's left shin. Atrophic and sclerotic changes were found in the dermal layer.

Deep veins of the left shin were full of blood, with multiple fibrotic changes and round cell infiltration with surface injury and fibrin deposition in the intima. Dense, easily crumbling dark-red and mixed blood clots with uneven surface were visualized in the deep veins of the left shin (Picture 1).

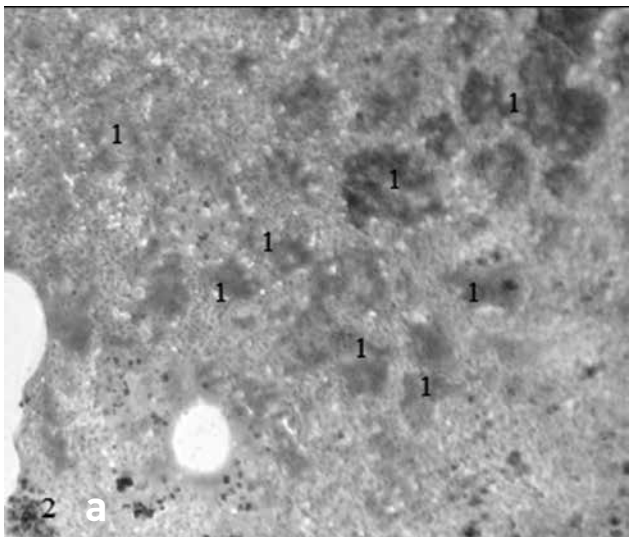
Numerous thrombi sized from 0.3 to 1.5 cm and up to 2.1 cm long that could be easily squeezed out from blood vesseld were found in the large, lobar and segmental branches of the pulmonary artery (Picture 2). Histological examination of the lungs revealed necrotic areas of lung tissue impregnated with erythrocytes (Picture 3). Erythrocytes, macrophages and fluid was found in the alveoli. The capillaries in the

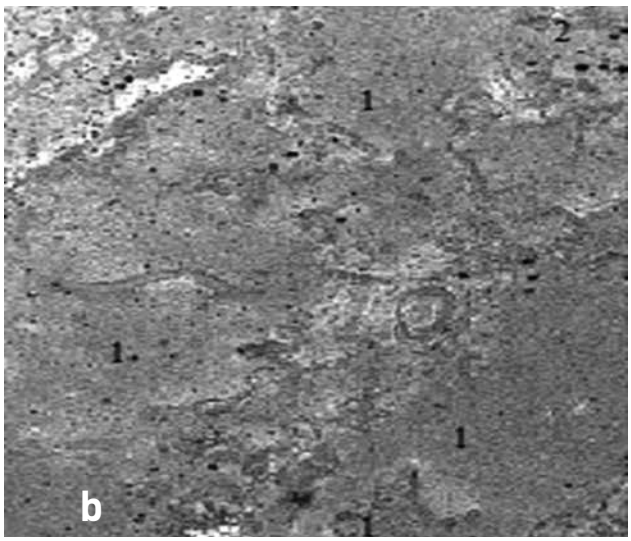


Picture 1. Thrombus in the deep vein of the left sheen. Hematoxylin & Eosin, × 400



Picture 2. Thrombus in the pulmonary artery. Hematoxylin & Eosin × 400





Picture 3. a-b. Microscopy of hemorrhagic infarction of the lung: 1 - necrotic areas of lung tissue impregnated with erythrocytes. Alveoli filled with erythrocytes. 2 - Areas of hemosiderin. Hematoxylin & Eosin × 400

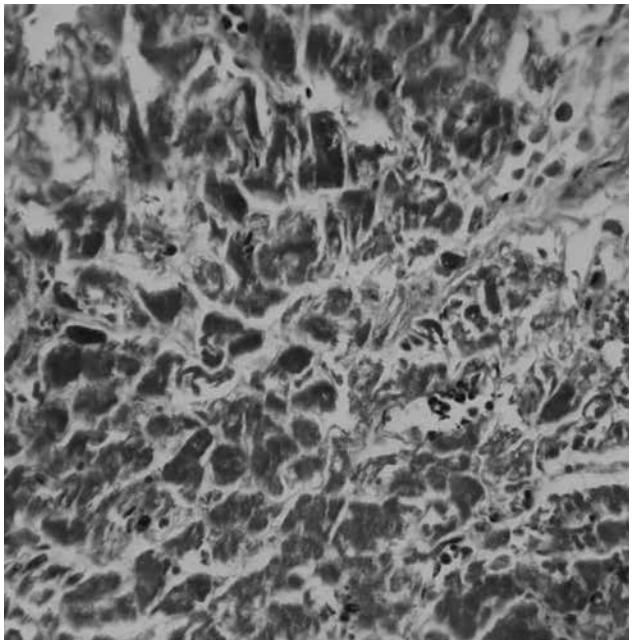
lung tissue in the area of the vessel occlusion are full of blood and there are intraalveolar hemorrhages.

Microscopic signs of acute transmural myocardial infarction of the anterior left ventricular wall were detected with areas of necrotized cardiomyocytes with a demarcation zone of inflammation and undulating deformation of cardiomyocytes (Picture 4). The nuclei became homogenous, partly fragmentated. Stromal tissue became metachromatic. Fibrotic changes were noted in the blood vessel walls and the lumen was filled with a mass of disintegrated erythrocytes. Necrotic areas were demarcated by leucocytes. Apart from them, hypertrophied LV cardiomyocytes sized up to 1.9 cm were noted, heart mass — 350 grams. Histologic evaluation showed areas of fragmentation, hypertrophied cardiomyocytes and perivascular connective tissue (Picture 5). Coronary arteries with atherosclerotic changes, fibrotic plaques and calcinosis (9/4 mm). Left coronary artery is filled with dark-red microthrombi that are tightly attached to vessel walls.

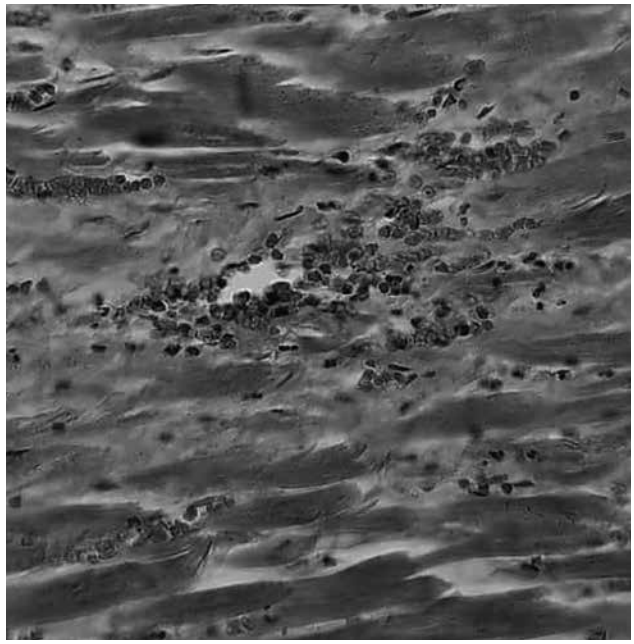
A pathology report contained the following: Deep vein thrombosis of the left shin. Acute pulmonary embolism. Hemorrhagic pulmonary infarction. Acute transmural myocardial infarction. Pulmonary edema. Acute respiratory and cardiac failure. Left knee joint posttraumatic arthritis. Atherosclerosis. Arterial hypertension, left ventricular hypertrophy (1.9 cm), diffuse perivascular cardiac sclerosis.

Discussion. Despite the fact that this patient had poor prognosis due to the signs and symptoms of cardiovascular decompensation associated with various CAD forms, understanding the pathomorphological features of thrombosis development associated with progressive thrombophlebitis and deep vein thrombosis is important and extremely relevant [1]. The presented clinical case shows the effects of traumatic vessel injury, as well as the existing coronary atherosclerosis that causes subendothelial thrombi formation in the area of the damaged atherosclerotic plaque and leads to the development of myocardial ischemia, PE with complications and hemorrhagic lung infarction. PE is associated with two pathological processes: mechanical vessel obstruction and humoral changes. Major thromboembolic lung vessel obstruction (reduction of the total arterial lumen by 40–50 % that corresponds to the obstruction of 2–3 branches of pulmonary artery) increases total pulmonary vessel resistance. This, in turn, obstructs blood flow from the right ventricle, decreases the filling of left ventricle and, eventually, leads to the reduction of cardiac output and arterial pressure [4, 6, 7, 8].

Such a combined pathology is extremely rare. Therefore, the described case is unique and important. A sequence of changes is traced not only in the vessels, but also in organs with the development of different types of infarction. Obviously, ischemic myocardial infarction caused by thrombosis developed first, and led to the reduction of functional myocardium mass, ventricular dilation, changes in the neurohumoral regulation of heart function and vessel tone, changes in the intracardiac and central haemodynamics and PE and hemorrhagic pulmonary infarc-



Picture 4. Microscopy of fragmented hypertrophied cardiomyocytes, cardiac sclerosis. Hematoxylin & Eosin, × 400



Picture 5. Microscopy of acute transmural infarction: areas of necrotic cardiomyocytes with cardiolysis. Hematoxylin & Eosin, × 900

tion due to the obstruction of major lobar branches of the pulmonary artery [2, 3, 5, 7, 8]. This combination of different types of infarction was probably associated with pulmonary hypertension and concomitant diseases such as coronary artery disease, arterial hypertension and atherosclerosis. The clinical case described in this report shows that PE can develop without the classic mechanism — pulmonocoronary reflex.

Conclusion. The described clinical case proves the key role of CAD complicated by chronic cardiac failure, traumatic limb injury in the development and progression of DVT. Pre-existing heart disease reduces compensatory functions of cardio-vascular system and worsen prognosis.

Conflict of interest: none declared.

References

1. Burleva E.P., Brazhnikov A.Yu., Belova A.D. Experience of outpatient multidisciplinary management of patients with venous thrombosis. Hospital-replacing technologies: Ambulatory surgery. 2017;(3–4): 105–111. Russian.
2. Vorobieva O. V., Gimaldinova N. E., Lastochkin A. V. Atherosclerotic gangrene of the intestine: a case report. Cardiovascular therapy and prevention. 2020; 19(6): 92–93. Russian.
3. Vorobyeva O.V., Lastochkin A.V. Clinical and morphological case of diabetes mellitus complicated by myocardial infarction. Preventive medicine. 2020; 23(3): 104–107. Russian.
4. Abdulrahman A.O., Weina J., Dahui S., Baochang Q. Review Article Deep venous thrombosis: a literature review. Int J Clin Exp Med. 2018; 11(3): 1551–1561.
5. Dennis J, Johnson CY, Adediran AS. The endothelial protein C receptor (PROCR) Ser219Gly variant and risk of common thrombotic disorders: a HuGE review and meta-analysis of evidence from observational studies. Blood. 2012; 119(10): 2392–400.
6. Jonathan S., Patrick H., Hassan A., Alex W. Deep vein thrombosis: pathogenesis, diagnosis, and medical management. Cardiovasc Diagn Ther. 2017 Dec; 7(Suppl 3): 276–284.
7. Khoury H, Lyons R, Sanaiha Y. Deep Venous Thrombosis and Pulmonary Embolism in Cardiac Surgical Patients. Thorac Surg. 2020; 109(6): 1804–1810.
8. Paul C K., John W E., James D Douketis and Graeme J. Hankey. Deep vein thrombosis: update on diagnosis and management. Med J Aust 2019; 210 (11).