

DEPARTMENT OF CLINICAL SYNTHESIS

**44. PULMONARY EDEMA IN CHRONIC HEMODIALYSIS HYPERTENSIVE PATIENT**

Author: **Iuliana Romaniuc**

Co-authors: Ana Popa, Marina Savca

Scientific adviser: Natalia Caproș, MD, PhD, University professor, Department of Internal Medicine. Clinical Synthesis, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova.

**Background.** Cardiovascular diseases including acute pulmonary edema (EPA) are the most common causes of hospitalization in patients with chronic dialysis. Mortality due to pulmonary edema in hemodialysis patients is 10%. EPA's dramatic presentation calls for emergency care, usually in intensive care units. The purpose of this study is to present a chronic hemodialysis hypertensive patient with pulmonary edema.

**Case report.** The 73-year-old patient presented to the Emergency Medicine department with mixed dyspnea pronounced at the minimal effort, productive cough with poor serous expectations, moderate intensity retrosternal pain, headache, general asthenia, paresthesia in the lower limbs. Known from the records of the family doctor with the diagnosis of renal hypertension for 15 years, type II diabetes mellitus, insulin-independent, terminal hemodialysis-dependent renal failure of 9 months. The patient developed the signs of acute pulmonary edema: severe dyspnea at rest, tachypnea, psychomotor agitation, obnoxious consciousness, peripheral cyanosis. The objective examination revealed evidence of alveolar edema. Respiratory frequency was 22 b/min, data of the blood arterial gases were SpO<sub>2</sub> - 88%, fraction of inspired oxygen (FiO<sub>2</sub>) - 21%, partial pressure of O<sub>2</sub> in the alveolar gas (pO<sub>2</sub>) - 27.3 mmHg, the ratio of partial pressure arterial oxygen and fraction of inspired oxygen (pO<sub>2</sub>/FiO<sub>2</sub>) - 1.30, partial pressure of oxygen in the arterial blood (PaO<sub>2</sub>) <60mmHg. The apexian shock in the V left intercostal space, rhythmic cardiac contractions and accentuated A2. Blood pressure was 160/90 mmHg and heart contractions were 76 b/min. Laboratory analyzes showed: anemic syndrome: hemoglobin - 102 g/dl, erythrocytes - 3.45x10<sup>12</sup>/l, and increased erythrocyte sedimentation rate - 43mm/h. Biochemical examination revealed elevated urea - 30.0 mmol/l and serum creatinine - 1184 mmol/l, hyperkalemia - 6.0mmol/l. The electrocardiogram recorded sinus rhythm, conduction disturbances: I degree of atrio-ventricular block and deflected to the left of the electrical axis. Echocardiographic examination revealed: cardiomegaly, wide aortal stenosis, left and right atrium and ventricle dilation, mild concentric left ventricular hypertrophy and preserved left ventricular ejection fraction - 53%. Severe mitral valve valvulopathy III-IV degree, relaxation of the myocardium VS and signs of moderate pulmonary hypertension, increased systolic pressure in the pulmonary artery - 63 mmHg. The patient immediately was transferred to the intensive care unit. Oxygen therapy through continuous positive pressure with nasal catheter (BiPAP) had also been done. Following the treatment administered diuretics, vasodilators, digitalis, antihypertensives, opioid analgesics, the patient's condition improved.

**Conclusions.** The peculiarities of the case study were: acute pulmonary edema in an chronic hemodialysis hypertensive patient. Early treatment should be instituted because it has a fast evolution and resolves without lasting damages.

**Key words:** Pulmonary edema, Uremic patients, Hemodialysis, Renal failure