ACUTE CARDIOGENIC PULMONARY EDEMA - ETIOLOGICAL SPECTRUM AND PRECIPITATING FACTORS

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ACUTE CARDIOGENIC PULMONARY EDEMA (APE) - ETIOLOGICAL SPECTRUM AND PRECIPITATING FACTORS (Abstract): **Aim:** the analysis of the predisposing and precipitating factors encountered in the anamnesis of the patients hospitalized with acute pulmonary oedema, in order to establish some correlations with the evolution and prognosis. **Material and methods:** The study included 50 patients, 32 males and 18 females, admitted to the Cardiology I Clinic between 2009 and 2013, diagnosed with acute pulmonary oedema upon admission. The following aspects were investigated: cardiovascular antecedents, prodromal elements of the current episode of acute pulmonary oedema (APE), risk factors and associated comorbidities, previous treatments followed at home as well as the triggering factors of the acute episode. **Results:** The main pathology on which the acute pulmonary oedema episode occurred was represented by: arterial high blood pressure (HBP), dilated cardiomyopathy, ischemic heart disease, valvular heart disease, pre-existing atrial rhythm disorders. The analysis of the factors that precipitated the acute pulmonary oedema episode revealed the following possible triggering conditions: inadequate physical effort on the background of treatment discontinuation, high sodium diet, a prolonged ischemic episode due to inadequate physical effort, rhythm disorder with rapid ventricular response (atrial fibrillation, atrial flutter). In some cases, the risk factors were cumulated. **Conclusions:** Acute pulmonary oedema represents a major emergency that requires immediate admission to hospital and rapid treatment in the emergency department concurrent with the identification of the triggering and precipitating factors. **Keywords:** PULMONARY EDEMA, RISK FACTORS.

Cardiogenic pulmonary oedema represents an important pathology with 10% incidence in patients known with cardiovascular diseases and with a morbidity rate of 50% (1, 2). It is caused by the excessive accumulation of blood plasma at the level of alveoli and pulmonary interstitial space secondary to the increase of the filling pressure in the left heart, which leads to the increase of the hydrostatic pressure in the pulmonary veins and capillaries (1, 2, 3). The main cause is represented by the left heart dysfunction, but there are also some other cardio circulatory conditions that lead to the increase of the pressure in the pulmonary capillaries with the appearance of
the transudate: left heart valvular disorders, cardiac tumours, rhythm disorders with rapid ventricular response (atrial fibrillation, atrial flutter), pulmonary embolism, high blood pressure (2, 3, 4, 5). It represents a major cardiovascular emergency, with life threatening risk, reasons for which the therapeutic intervention has to be fast, prompt, addressing both the involved physiopathological mechanisms and the etiology (5).

Aim of the study: the analysis of the predisposing and precipitating factors encountered in the anamnesis of the patients hospitalized with acute pulmonary oedema, in order to establish some correlations with the evolution and prognosis.

MATERIAL AND METHODS
The study included 50 patients, 32 (64%) males and 18 (36%) females, admitted to the Cardiology I Clinic between 2009 and 2013, diagnosed with acute pulmonary oedema upon admission. All patients were admitted to the emergency unit of the hospital and they were brought by ambulance in most of the cases. Patients were aged between 52 and 85. Being a major emergency, the first therapeutic measures for all patients were initiated in the emergency care unit concomitant with a cardiological examination.

The following steps were undertaken:
- The anamnesis of the patient, with the mention that in the situations in which the condition of the patient did not allow the direct anamnesis, the data was obtained from his relatives or from the previous hospital release documents (patient's previous medical folder respectively).

The following aspects were investigated during the anamnesis:
- Patient's cardiovascular antecedents:
  - high values of blood pressure (HBP), chronic ischemic disease, acute myocardial infarction (IMA), valvular heart diseases, dilated cardiomyopathy (CMD), previously known rhythm and/or conduction disorders, potential surgical interventions at the level of the cardiovascular system;
  - Identifying the number of previous episodes of left ventricular insufficiency (cardiac asthma, acute pulmonary oedema) if these occurred;
  - Identifying some prodromal elements of the current episode of acute pulmonary oedema (paroxysmal nocturnal dyspnoea);
  - The associated risk factors having an impact on the cardiovascular system (smoking, alcohol intake, diabetes);
  - Associated comorbidities (thyroid gland disorders such as hypo or hyperthyroidism, chronic obstructive bronchopneumopathy (BPCO), stroke);
  - Treatments followed at home up to the moment of coming to the emergency care unit;
  - The emphasis was targeted on identifying the precipitating and aggravating factors of the acute pulmonary oedema episode: inadequate physical effort, alcohol intake, paroxysmal rhythm disorder, conduction disorder, prolonged anginous episode, intercurrent respiratory infection, fever, treatment discontinuation, excessive salt intake.

The complete examination of the patients involved: assessing the general state, consciousness, pulmonary auscultation, heart and peripheral arteries (carotid) auscultation, checking of blood pressure (BP).

The paraclinical data were collected concomitantly with the application of the first aid measures in the emergency care unit and therapy:
- Measuring the levels of SaO2, PO2, PCO2, Astrup;
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- 12-lead electrocardiogram;
- Continuous EKG monitoring;
- Continuous monitoring of blood pressure (BP) and SaO2;
- Blood sampling for the absolutely necessary usual blood tests: hemolucogram, renal function, electrolytes, glycaemia, myocardial cytolysis enzymes (aspartate aminotransferase (AST), creatine kinase (CK), creatine kinase MB (CKMB), lactate dehydrogenase (LDH)), coagulation tests;
- Echocardiography in the Emergency Room: 2D, M mode and Doppler in order to assess left ventricular systolic and diastolic function and to confirm the etiology of heart failure.

RESULTS AND DISCUSSION

In our study 50 patients, 32 males and 18 females, aged between 52 and 85, diagnosed with acute pulmonary oedema were admitted to the Cardiology I Clinic, between 2009 and 2013 (fig.1).

*Age distribution of the patients from the group was as follows* (fig.2).

![Fig. 1. Gender distribution of patients](image)

![Fig. 2. Age distribution for the patients with pulmonary oedema](image)

_The anamnesis of the patients in the group revealed the following data:_

- high blood pressure present in 20 patients, 11 males and 9 females;
- dilated cardiomyopathy present in 15 patients, 13 males and 2 females;
- 3 patients presented significant hemodynamic valvular heart disorders: one male presented aortic stenosis and 2 females presented mitral stenosis and aortic stenosis respectively;
- the ischemic cardiac disease was present in 12 patients from the group with high blood pressure, 8 males and 4 females respectively, of which: 2 males had acute myocardial infarction (AMI) before, 6 males presented different forms of angina pectoris; one female had acute myocardial infarction (AMI) before and 3 females, different forms of angina pectori-
is;
- rhythm disorders in 16 patients as follows: 12 with atrial fibrillation (7 males and 5 females), 4 with atrial extrasystolic arrhythmia and ventricular extrasystolic arrhythmia (2 males and 2 females).

Of the total number of patients, 33 (66%) were at their first acute pulmonary oedema episode, and 17 (34%) at their second (fig. 3).

The analysis of the associated risk factors in the patients from the group revealed the following: of the total number of patients, 21 were smokers, 23 reported chronic alcohol consumption, dyslipidemia was present in 18 patients, 6 of them being under treatment with statin. Diabetes was present in 6 males and 3 females, 5 of them being under treatment with insulin; 3 patients had associated thyroid pathology; 4 of them had antecedents of stroke; 5 had chronic obstructive bronchopneumopathy (BPCO) and presented chronic renal disease (fig.4).

**Fig. 3.** Pathological antecedents present in patients in the study

**Fig. 4.** Risk factors present in the patients diagnosed with acute pulmonary oedema.

*Treatments* followed by the patients in the group before the acute pulmonary oedema (APE) episode were different. Patients with high blood pressure were under different therapeutic schemes with calcium blocker, beta blocker, conversion enzyme inhibitor (ACE) and diuretic. The oral anticoagulant was used in 22 patients, 14 of them receiving platelet antiaggregants.

Three patients from the group of those with thyroidian pathology were under treatment with antithyroid drugs or Euthyrox.
Patients known with chronic obstructive bronchopneumopathy had Miofilin in their scheme of treatment (used as a bronchodilator) and sympathomimetic medication in some cases associated with corticotherapy in minimal doses (fig.5).

The analysis of the factors that precipitated the acute pulmonary edema (APE) episode revealed the following possible triggering conditions:

- Inadequate physical effort in the context of treatment discontinuation in 27 patients (20 males and 7 females);
- High sodium diet associated with inadequate physical effort and treatment discontinuation in 12 patients (8 males and 4 females);
- Prolonged ischemic episode in the context of inadequate physical effort in 5 patients (3 males and 2 females). All these patients were known with ischemic cardiac disease;
- Rhythm disorder with rapid ventricular response (atrial fibrillation, atrial flutter) in 15 patients (13 males and 2 females);

Fig. 5. The medication administered before the APE episode.

Fig. 6. Factors that precipitated the onset of acute pulmonary edema.
- Sympathomimetic drugs abuse reported by one patient;
- For 5 patients (all male) the precipitating factors were cumulated: inadequate physical effort associated with high sodium diet, together with alcohol intake, on the background of treatment discontinuation (fig. 6).

**CONCLUSIONS**

The acute pulmonary oedema episode was solved faster in the case of the patients for whom the triggering factor was the high blood pressure (HBP) and of those who were at their first episode.

In patients having dilated cardiomyopathy and mitral or aortic valve disorders as an etiologic substrate, the evolution was more severe, requiring special therapeutic measures. The severity was amplified by the coexistence of the low cardiac output syndrome in the respective cases.

Patients who were at the second episode of acute pulmonary oedema had a more severe evolution.

Treatment discontinuation at home in the case of some patients associated with chronic alcoholism was a clear aggravating factor for the evolution.

**REFERENCES**