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Case Report

Acute respiratory failure in pulmonary congestion: Case report and review

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Abstract. Pulmonary Congestion (PC) is one of the most important causes of death in patients with heart failure which can be induced by a functional disorder no macroscopic appreciable in the examination of the heart.

The authors present a case of edema and Respiratory Failure (RF) by PC in absence of clear and significant cardiac pathological elements in autopsy.

Before the edema and then the PC act through an atelectasic mechanism that can be the cause of RF.

The PC is often underestimated in the necropsy findings rather than being considered the main cause of death both in the case of objective cardiac injuries that in functional cardiac deaths.

Key words: Respiratory failure, pulmonary congestion, heart failure

1. Introduction

The literature describes cases of death due to edema and Pulmonary Congestion (PC) both in ventricular fibrillation (1) (the only appreciable signs were cerebral and pulmonary edema and visceral congestion) that in atrial fibrillation (in which a statistically significant association with PC was found) (2,3). But in general there can be many causes of Heart Failure (HF), defined as new-onset or worsening HF signs and symptoms requiring urgent therapy (4), in the absence of macroscopically evident pathological findings at postmortem examination of the heart.

However, in these cases a PC by low cardiac output is appreciable and it is considered the main cause of hospitalization and death among patients with HF (5,6). 25% of patients hospitalized for HF are hospitalized again within 30 days (the highest rate among all causes of medical or surgical hospitalization), and almost half will be readmitted within 6 months (7-10). And the cost of these readmissions in 2010 was quantified in 39.2 billion $ in the United States (10).

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Acutely decompensated heart failure is, therefore, the most frequent cause of hospitalization in adults> 65 years with high rates of morbidity and mortality, and a high economic importance (11-13). The problem is that patients with moderate or severe PC can also be asymptomatic or mildly symptomatic (71%) (14) and the occurrence of symptoms often occur many days after the increase in pulmonary capillary wedge pressure (PCWP) (15,16) even 18 days before of hospitalization (17). In addition, the association between PCWP and severe dyspnea has not been confirmed in the literature because patients with high PCWP may have a minimum dyspnea (6). The recognition, quantification and control of the PC is important for the clinician (18), but it is also important at autopsy to admit a primary role of the PC in the event death. Patients with a PC have a very serious risk of cardiac events (3.2 times higher than those without PC) (14).

The authors present a case of respiratory failure (RF) by edema and PC into a cardiac framework devoid of clear and significant pathological elements at autopsy.

2. Case report

A 55-year-old woman, 170 cm tall and weighing approximately 70 kg has good general health conditions and good normal constitution. She had known hypertension disease and adipose tissue was with a regular distribution and muscle eutropism.

Skull was without appreciable preternatural motility of the bone surface. Patches of ECG recordings on the chest showed no preternatural motility of the bone surface but slightly globular abdomen for fat.

The examination of the thanatological phenomena showed hypostasis underrepresented in dorsal surface of the body and up to the middle axillary line. Stiffness present and winnable with moderate force at all joints. There were no putrefactive phenomena.

After dissecting the soft tissue, there was full integrity of the rib cage, diaphragm and bowel loops with few limpid liquid in the abdominal cavity.

Upon removal of the sternal plastron, the lungs were expanded, coating the heart, with limpid liquid (200cc) in the pleural cables, without adhesions. The pericardium was intact.

Heart was of a globular form (300g weight, 12 cm longitudinal diameter, 11cm transverse diameter, 3.5cm anteroposterior diameter) at its appearance. The lumen of the right and left coronary were pervious.

Apical wall of the left ventricle was approximately 1.2cm thick while the free wall was 1.1cm, and the interventricular septum wall was 1.2cm thick. The thickness of the free wall of the right ventricle was 0.3cm.

In the longitudinal section of the rest of the heart, nothing to report at the level of the myocardium (dissected) chordae and papillary muscles or valvular apparatus.

Histological examination of the left ventricle showed slight level of hypertrophy of the left ventricular with increased cell size and squat appearance (Figure 1).

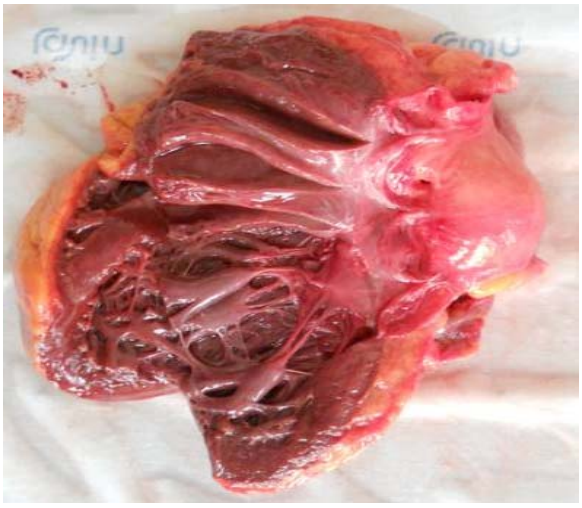

Fig. 1 Internal surface of the left ventricle.

Lungs had increased dimensions and consistency (right lung weight 950 g - left 980 g) and were not crackling.

The large, medium and the small bronchi were with mucosal hyperemia and slightly raised in folds. To the full-thickness section of lungs parenchyma was congested, a finding corroborated by squeezing with frothy bleeding. Histological examination showed abundant interstitial and alveolar infiltration (Figure 2).

Nothing to report from the other organs examined and toxicological screening negative.

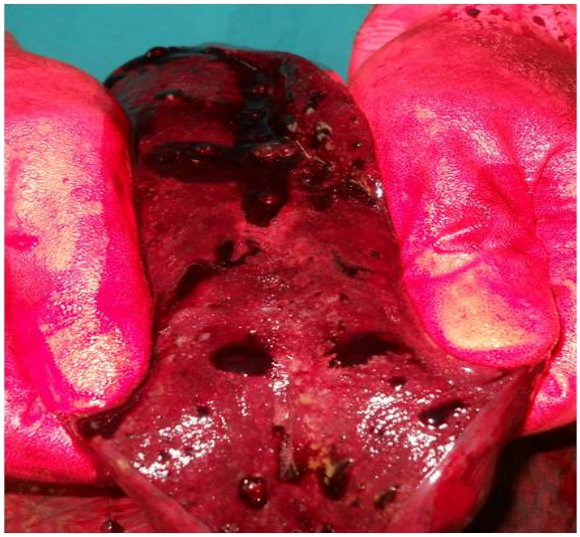

Fig. 2. Pressing of the lung parenchyma.

3. Discussion

The PC is the accumulation of fluid in the lungs, resulting in impaired gas exchange and arterial hypoxemia. It occurs first in the hilar region of the lungs, then in the interstitial space and, finally, in the alveolar side.

PC can also result from non-cardiogenic causes as pulmonary causes (19) or fluid overload by water retention.

In the non-cardiogenic case there is an injury to the alveolar-capillary barrier with leakage of protein-rich fluid in the interstitium (20), while in the cardiogenic form the alveolar-capillary barrier remains intact and the high left ventricular pressure causes pulmonary venous hypertension (increased PCWP) with increased hydrostatic pressure and this is the main mechanism of PC.

The increased PCWP causes the redistribution of excess fluid within the lungs, resulting in interstitial and alveolar edema (4,6,21,22) which is the consequence of an imbalance between the liquid filtration and re-absorption forces (Starling's equation) (23).

Really studies on the relationship of edema / serum protein in patients with cardiogenic and non-cardiogenic PC have shown that there is often an overlap of these two mechanisms with high PCWP and high permeability of the alveolar-capillary barrier (24,25).

The alveolar-capillary barrier, formed by a capillary endothelial layer, an alveolar epithelial layer and from the extracellular matrix must be extremely thin to facilitate the gas exchange by passive diffusion, but must be sufficiently strong, through especially the collagen (26), to resist the stress of hydrostatic pressure. "Stress failure" is called the mechanical injury of alveolar-capillary barrier (detriment of the capillary endothelium and alveolar epithelium) resulting from the rapid increase of PCWP (27). A sustained elevation of PCWP, however, would lead to a thickening of the alveolar-capillary barrier through the deposition of collagen type IV (26), hindering PC in chronic HF (25).

A sustained elevation of PCWP, however, would result in a thickening of the alveolar-capillary barrier through the deposition of collagen type IV (26), making it difficult PC chronic HF (25).

So the presence of edema and PC in chronic heart disease acquires much more significance.

Also inflammation, however, plays an important pathogenic role (28,29) as demonstrated by the high concentrations of neutrophils, pro-inflammatory cytokines (30,31) and biomarkers of oxidative stress in HF. The hypothesis is that the pulmonary endothelium responds to mechanical stress signals activating cascade involving the release of inflammatory cytokines, the activation of macrophages (with the consequent release of TNF-alpha) (32,33) and, therefore, the damage the alveolar-capillary barrier (34).

RF is established by reduction of the V / Q ratio and pulmonary shunt caused by airway resistance distal for constrictive atelectasis and for hypoxic pulmonary vasodilation (35,36).

4. Conclusion

According to autopsy findings, death may be related to a hypertensive crisis with PC and HF. PC by low cardiac output is considered the main cause of hospitalization and death in patients with HF (5,6), to consider, therefore, not only as an effect, but also cause of a left ventricular dysfunction leading to cardiac events.

References

1. Kamm RC. Fatal arrhythmia following deodorant inhalation: case report. Forensic Sci 1975; 5: 91-93.
2. Madias JE, Patel DC, Singh D. Atrial fibrillation in acute myocardial infarction: a prospective study based on data from a consecutive series of patients admitted to the coronary care unit. Clin Cardiol 1996; 19: 180-186.
3. Silva RG, Lima GG, Laranjeira A, et al. Risk factors, morbidity, and mortality associated with atrial fibrillation in the postoperative period of cardiac surgery. Arq Bras Cardiol 2004; 83: 105-110.
4. Gheorghiade M, Zannad F, Sopko G, et al; International Working Group on Acute Heart Failure Syndromes. Acute heart failure syndromes: current state and framework for future research. Circulation 2005; 112: 3958-3968.
5. Komajda M, Follath F, Swedberg K, et al; Study Group on Diagnosis of the Working Group on Heart Failure of the European Society of Cardiology. The EuroHeart Failure Survey programme--a survey on the quality of care among patients with heart failure in Europe. Part 2: treatment. Eur Heart J 2003; 24: 464-474.
6. Gheorghiade M, Filippatos G, De Luca L, Burnett J. Congestion in acute heart failure syndromes: an essential target of evaluation and treatment. Am J Med 2006; 119: 3-10.
7. Ross JS, Chen J, Lin Z, et al. Recent national trends in readmission rates after heart failure hospitalization. Circ Heart Fail 2010; 3: 97-103.
8. Jencks SF, Williams MV, Coleman EA. Rehospitalizations among patients in the Medicare fee-for-service program. N Engl J Med 2009; 360: 1418-1428.
9. Keenan PS, Normand SL, Lin Z, et al. An administrative claims measure suitable for profiling hospital performance on the basis of 30-day all-cause readmission rates among patients with heart failure. Circ Cardiovasc Qual Outcomes 2008: 29-37.
10. Lloyd-Jones D, Adams RJ, Brown TM, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics--2010 update: a report from the American Heart Association. Circulation 2010; 121: 46-215.
11. Russo MJ, Gelijns AC, Stevenson LW, et al. The cost of medical management in advanced heart failure during the final two years of life. J Card Fail 2008; 14: 651-658.
12. Giamouzis G, Kalogeropoulos A, Georgiopoulou V, et al. Hospitalization epidemic in patients with heart failure: risk factors, risk prediction, knowledge gaps, and future directions. J Card Fail 2011; 17: 54-75.
13. Butler J, Kalogeropoulos A. Worsening heart failure hospitalization epidemic we do not know how to prevent and we do not know how to treat! J Am Coll Cardiol 2008; 52: 435-437.
14. Zoccali C, Torino C, Tripepi R, et al. Pulmonary congestion predicts cardiac events and mortality in ESRD. J Am Soc Nephrol 2013; 24: 639-646.
15. Adamson PB, Magalski A, Braunschweig F, et al. Ongoing right ventricular hemodynamics in heart failure: clinical value of measurements derived from an implantable monitoring system. J Am Coll Cardiol 2003; 41: 565-571.

16. Chaudhry SI, Wang Y, Concato J, Gill TM, Krumholz HM. Patterns of weight change preceding hospitalization for heart failure. Circulation 2007; 116: 1549-1554.
17. Yu CM, Wang L, Chau E, et al. Intrathoracic impedance monitoring in patients with heart failure: correlation with fluid status and feasibility of early warning preceding hospitalization. Circulation 2005; 112: 841-848.
18. Picano E, Gargani L, Gheorghiade M. Why, when, and how to assess pulmonary congestion in heart failure: pathophysiological, clinical, and methodological implications. Heart Fail Rev 2010; 15: 63-72.
19. Ware LB, Matthay MA. Clinical practice. Acute pulmonary edema. N Engl J Med 2005; 353: 2788-2796.
20. Bernard GR, Artigas A, Brigham KL, et al. The American-European Consensus Conference on ARDS. Definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med 1994; 149: 818-824.
21. Gheorghiade M, Shin DD, Thomas TO, et al. Congestion is an important diagnostic and therapeutic target in heart failure. Rev Cardiovasc Med 2006; 7: 12-24.
22. Fonarow GC; ADHERE Scientific Advisory Committee. The Acute Decompensated Heart Failure National Registry (ADHERE): opportunities to improve care of patients hospitalized with acute decompensated heart failure. Rev Cardiovasc Med 2003; 4: 21-30.
23. Starling EH. On the Absorption of Fluids from the Connective Tissue Spaces. J Physiol 1896; 19: 312-326.
24. Fein A, Grossman RF, Jones JG, et al. The value of edema fluid protein measurement in patients with pulmonary edema. Am J Med 1979; 67: 32-38.
25. Sprung CL, Rackow EC, Fein IA, Jacob AI, Isikoff SK. The spectrum of pulmonary edema: differentiation of cardiogenic, intermediate,and noncardiogenic forms of pulmonary edema. Am Rev Respir Dis 1981; 124: 718-722.
26. West JB, Mathieu-Costello O. Strength of the pulmonary blood-gas barrier. Respir Physiol 1992; 88: 141-148.
27. West JB, Tsukimoto K, Mathieu-Costello O, Prediletto R. Stress failure in pulmonary capillaries. J Appl Physiol (1985) 1991; 70: 1731-1742.
28. Haupt MT. Cardiogenic pulmonary edema: an inflammatory disorder? Crit Care Med 2003; 31: 1282-1283.
29. De Pasquale CG, Arnolda LF, Doyle IR, et al. Prolonged alveolocapillary barrier damage after acute cardiogenic pulmonary edema. Crit Care Med 2003; 31: 1060-1067.
30. Cohen AB, Stevens MD, Miller EJ, et al. Neutrophil-activating peptide-2 in patients with pulmonary edema from congestive heart failure or ARDS. Am J Physiol 1993; 264: 490-495.
31. Nakos G, Pneumatikos J, Tsangaris I, Tellis C, Lekka M. Proteins and phospholipids in BAL from patients with hydrostatic pulmonary edema. Am J Respir Crit Care Med 1997; 155: 945-951.
32. Filippatos G, Leche C, Sunga R, et al. Expression of FAS adjacent to fibrotic foci in the failing human heart is not associated with increased apoptosis. Am J Physiol 1999; 277: 445-451.
33. Parissis JT, Venetsanou KF, Mentzikof DG, et al. Tumor necrosis factor-alpha serum activity during treatment of acute decompensation of cachectic and non-cachectic patients with advanced congestive heart failure. Scand Cardiovasc J 1999; 33: 344-350.
34. Birukov KG. Cyclic stretch, reactive oxygen species, and vascular remodeling. Antioxid Redox Signal 2009; 11: 1651-1667.
35. Benumof JL, Wahrenbrock EA. Blunted hypoxic pulmonary vasoconstriction by increased lung vascular pressures. J Appl Physiol 1975; 38: 846-850.
36. Gehlbach BK, Geppert E. The pulmonary manifestations of left heart failure. Chest 2004; 125: 669-682.