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Case Report

Hypertensive crisis induced acute pulmonary edema in emergency care unit Sumbawa hospital: a case report

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ABSTRACT

Acute cardiogenic pulmonary edema is a common cause of respiratory distress in emergency department (ED) patients. Pulmonary edema is a problem of major clinical importance resulting from a persistent imbalance between forces that drive water into the air space of the lung and the biological mechanisms for its removal. A patient 66 years old female, with a history hypertency stage II and osteoarthritis admitted to emergency department with acute pulmonary edema. On evaluation, the patient had intense dyspnoe, agitated, diaphoresis without cyanosis, the pulse pressure was normal, rapid and regular. Noninvasive ventilation by noninvasive positive pressure ventilation or continuous positive airway pressure has been studied as a treatment strategy. Noninvasive ventilation and intravenous nitrates are the mainstay of treatment which should be started within minutes of the patient's arrival to the ED. Use of morphine and intravenous loop diuretics, although popular, has poor scientific evidence. We critically evaluate the evidence for the use of noninvasive ventilation on rates of hospital mortality and endotracheal intubation. Although is often results from atrial fibrillation, acute myocardial infarction, hypertension crisis, discontinuation of medication edema. It is important to understand this disease, rapid diagnostic with ultrasound and when treated promptly and effectively, these patients will rapidly recovery. Good oxygenation, intravenous nitrates, intravenous diuretics and low dose sedation which should be started within minutes of the patients arrival to emergency department.

Keywords: Acute lung edema, Diuretic, Emergency department, Hypertensive crisis, Nitroglycerine, Ultrasound, Ventilation

INTRODUCTION

Pulmonary oedema is a consequence of acute heart failure. This type of heart failure results from a sudden decrease in stroke volume, causing an increase in systemic vascular resistance, which in turn further reduces stroke volume, finally leading to pulmonary oedema.[1-3] Pulmonary edema is a problem of major clinical importance resulting from a persistent imbalance between forces that drive water into the airspace of the lung and the biological mechanisms for its removal.[4-6] Under normal conditions, fluid escaping from the capillaries through tiny gaps in the vascularendothelial cell junctions does not enter the alveolar space, because the alveolar epithelial barrier is very tight. Rather, this fluid is removed from the interstitial space and returned to the systemic circulation by the lympathics. Two major mechanisms lead to a disruption of the alveolar epithelial barrier, and in turn, alveolar fluid flooding, namely an exaggerated increase of the hydrostatic pressure in the pulmonary capillaries and an increase in the permeability of the pulmonary blood gas barrier.[4,7,8] Movement of

liquid into the airspace is only one of the factors that determines the amount of fluid in the airspace. Evidence in experimental animal models and humans has accumulated, indicating that in addition to alveolar fluid flooding, fluid removal from the alveolar space also plays an important role in the pathogenesis of pulmonary edema. Fluid removal from the airspace of the lung depends upon active sodium transport across the alveolarepithelium.[4,9,10] The importance of this transport has been demonstrated rather dramatically for the neonatal phase. Mice lacking the alpha subunit of the amiloride-sensitive sodium channel (ENaC) die shortly after birth, because these mice cannot clear the fluid that is normally present at birth from their lungs.[4,11]

Pulmonary oedema is one of the most common emergencies that the clinician faces in the emergency department.[6,12] Although cardiogenic oedema is the most frequent presentation, disorders in which factors other than elevated pulmonary capillary wedge pressure are responsible for fluid accumulation in the alveoliandintersitial must be considered. Spectrum of patient presenting with AHF is wide, ranging from mild to cardiogenic shock. Due to symphatetic activity in the pathophysiology of this patients, is better terminolgy for understanding the syndrome of rapid onset, lifethreatening pulmonary edema. Use of noninvasive positive pressure techniques to treat acute respiratory failure, including continuous positive airway pressure (CPAP) and noninvasive positive pressure ventilation using bilevel devices, has increased dramatically in recent years. Noninvasive positive pressure ventilation reduces the need for endotrachealintubation, shortens the hospital length of stay, and improves survival in patients with acute respiratory failure due to exacerbations of chronic obstructive pulmonary disease (COPD) and immunocom promised states.

Based on these capabilities, noninvasive positive pressure ventilation is now considered the ventilatory mode of first choice for appropriately selected COPD or immunocompromised patients with acute respiratory failure, and it is commonly applied in patients with asthma and pneumonia. In addition, several randomized, controlled trials have demonstrated that noninvasive CPAP alone is highly effective at improving gas exchange and avoiding intubation in patients with acute cardiogenic pulmonary edema, and a recent meta-analysis concluded that itis the preferred noninvasive modality for these patients. Authors here present this case with various underlying pathophysiologic mechanism, with rapid diagnostic and management to patient with acute pulmonary edema.[13-15]

CASE REOPRT

The patient was 66 years old woman, with a history hypertency stage II and osteoarthritis admitted to emergency department with acute pulmonary edema. On evaluation, the patient had intense dyspnoe, agitated, diaphoresis without cyanosis, the pulse pressure was normal, rapid and reguler. Blood pressure 190/120mmhg, HR 130/minutes, RR 32/minutes, Sp.02 88% room air, afebrile. On physical examination extensive bilateral crepititations are present, a later detailed examination after patient improvement.

The ECG evidenced only non specific ST and T abnormalities, tachycardy wave. An urgent bedside ultrasonography was performed in the context of acute pulmonary edema we found 3+ B line bilateral in one viewing field with dilated IVC. Sugar stick 110mg/dl, blood laboratory includes CBC, LFT, RFT, electrolyte, troponin T, and BGA still working.

Acute pulmonary edema is a medical emergency with early diagnostic, early recognition, and prompt initiation of treatment is the key to preventing morbidity and mortality. Immediate ED management of acute pulmonary edema has it impact with on subsequent clinical course, rate of invasive ventilation mechanic and rate in ICU.

Oxygenation skillful use non invasive ventilation (BiPAP/CPAP) or HFNC (high flow nasal canule>15/lpm) can avoid intubation and improved outcome because it decrease preload and after load, thereby decreasing cardiac oxygen demand workload. NIV decrease WOB patient also decrease rate of mechanical invasive ventilation and decrease mortality in patient cardiogenic pulmonary edema.

Nitroglycerine is a vasodilator have benefit effect in acute pulmonary edema with hypertension by causing preload reduction, thereby decreasing cardiac workload. They can be used sublingual until an intravenous access gained. Initial dose nitroglycerine for acute pulmonary edema 20mcg/min infusin gradual up titraton.

Diuretics loop diuretics such as furosemide have been primarily used in the management of acute pulmonary edema. The diuretic effect of furosemide starts in 30min and peaks in 1.5h. However, despite its popularity, there is only poor evidence supporting its current use in acute pulmonary edema. In fact, it may be detrimental to this subset of patients due to its stimulation of RAAS and sympathetic nervous system.

Furthermore, patients with acute pulmonary edema may be systemically hypovolemic or euvolemic due to long-term use of diuretics and high-dose intravenous diuretics may cause further deterioration in clinical condition.

Morphine has long been used as an initial agent for the treatment of cardiogenic pulmonary edema. It causes venodilatation, thereby decreasing preload, it decreases anxiety and pain, thereby decreasing the myocardial oxygen demand and hence has potential benefit during AHFS. However, morphine can cause respiratory

depression, which may lead to worsened hypoxia and respiratory arrest.

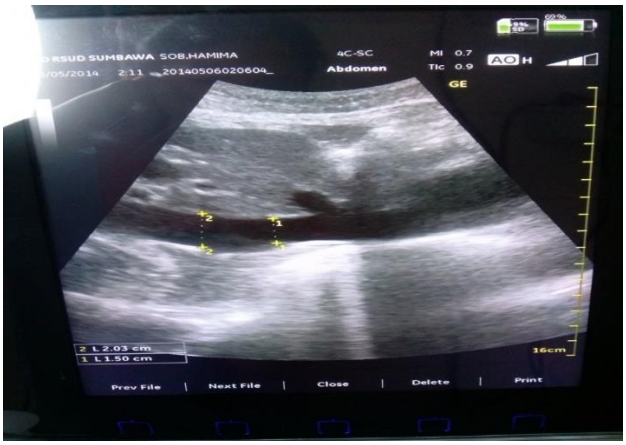

Figure 1: Bedside ultrasound showing the IVC (inferor vena cava) diameter >2.5 with respiratory change <50% suggest high C.

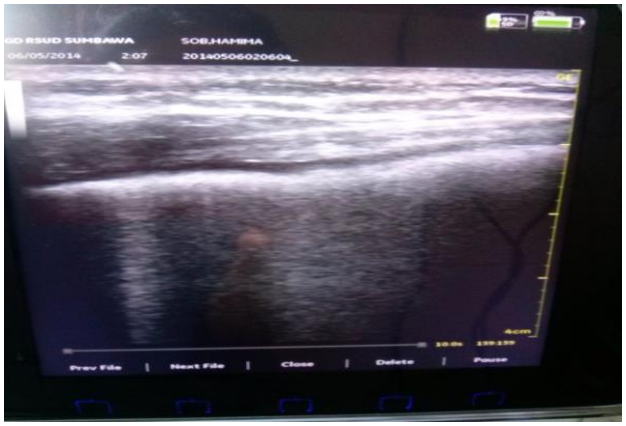

Figure 2: Bedside ultrasound showing B line bilateral with + lung sliding.

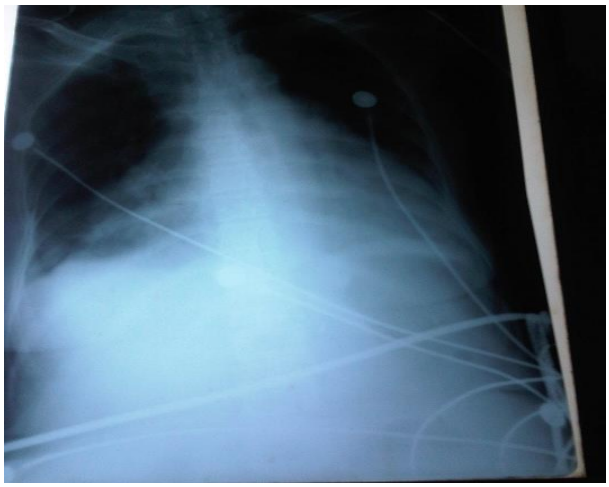

Figure 3: CXR performed show cardiomegaly with pulmonary congestion.

DISCUSSION

Authors undertook this study to test the hypothesis that acute pulmonary edema in association with hypertension is frequently due to transient systolic dysfunction. Contrary to our supposition, authors found that simple diagnostic by ultrasound during the acute episode of hypertensive pulmonary edema were similar to those measured after the resolution of the congestion, when the blood pressure was controlled.[16-18]

On admission, this patients had clinical and radiographic evidence of pulmonary edema that subsequently resolved with diuresis and control of hypertension; these observations ruled out unrecognized pulmonary disease as the cause of the acute problem. Pulmonary fluid homeostasis is equilibrium between forces that drive fluid into the alveolar spaces and the mechanisms responsible for its clearance.

Acute pulmonary edema is associated with increased cardiac filling pressures, which are transmitted to pulmonary capillaries and eventually drive intravascular fluid into pulmonary interstitium and alveoli. There are three stages of progression of acute pulmonary edema:

- Stage 1: Distension of small pulmonary capillaries due to increased left atrial pressure,
- Stage 2: Interstitial edema,
- Stage 3: Flooding of alveolar space causing hypoxia.

Underlying pathophysiologic mechanisms are similar for AHF and acute pulmonary edema. However, there are notable differences between the two. Acute pulmonary edema develops over minutes-to-hours into a life-threatening condition. Important distinguishing features are the pathophysiologic changes resulting in stress failure and increased permeability of the pulmonary capillaries causing abrupt onset and rapid progression.[19-21]

Sympathetic system is an important factor in cardiovascular physiology. Left ventricular dysfunction predisposes to abrupt increase in sympathetic tone and release of catecholamines that can precipitate flash pulmonary edema. Increased catecholamines cause increased heart rate and decreased diastolic time. It causes activation of renin-angiotensin-aldosterone system (RAAS), which further worsens diastolic stiffening and increased diastolic pressures causing pulmonary fluid overload. Increased sympathetic tone adversely affects the pulmonary circulation by increasing permeability and/or provoking stress failure of the pulmonary capillaries. Other factors contributing to flash pulmonary edema include decreased nitric oxide and increased endothelin activity.[22-26]

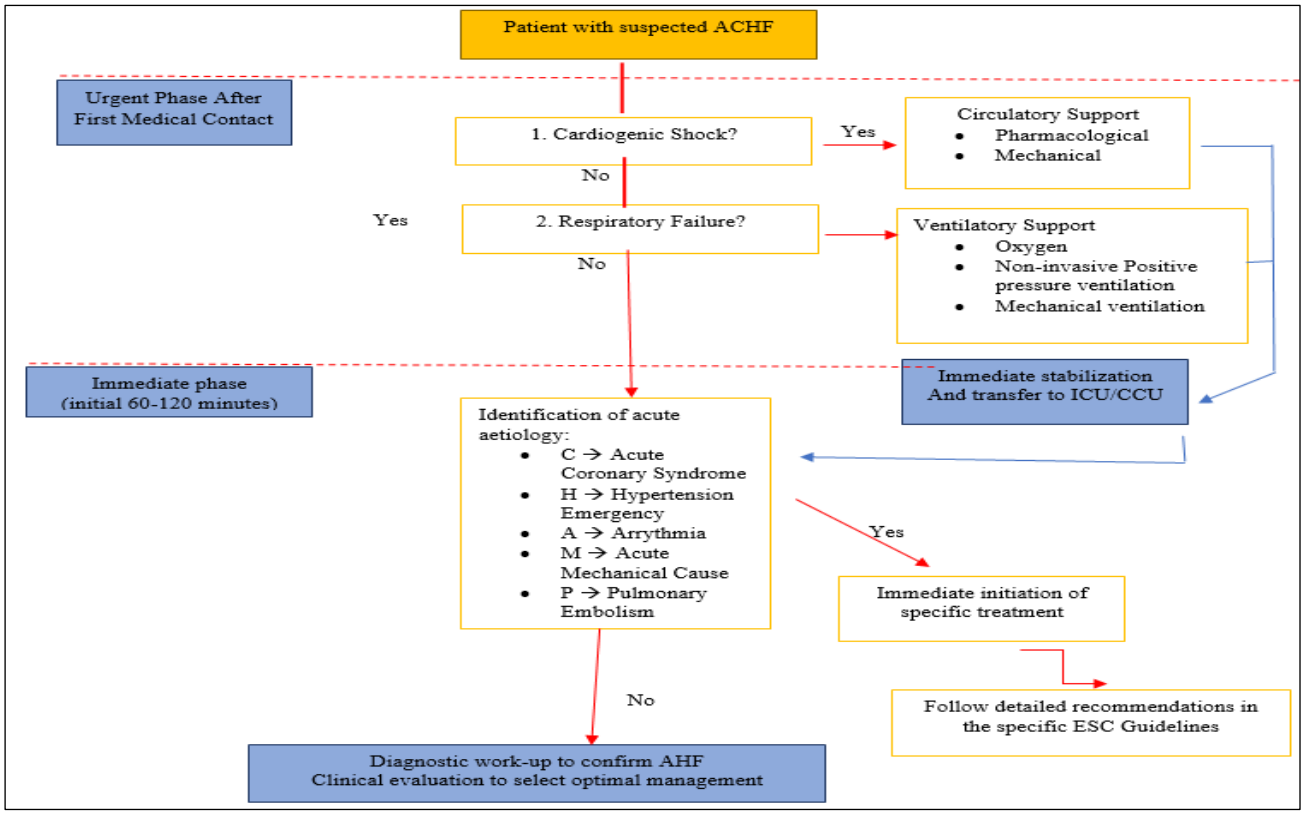

Figure 4: Algorithm management for patient with acute heart failure ESC 2016[25].

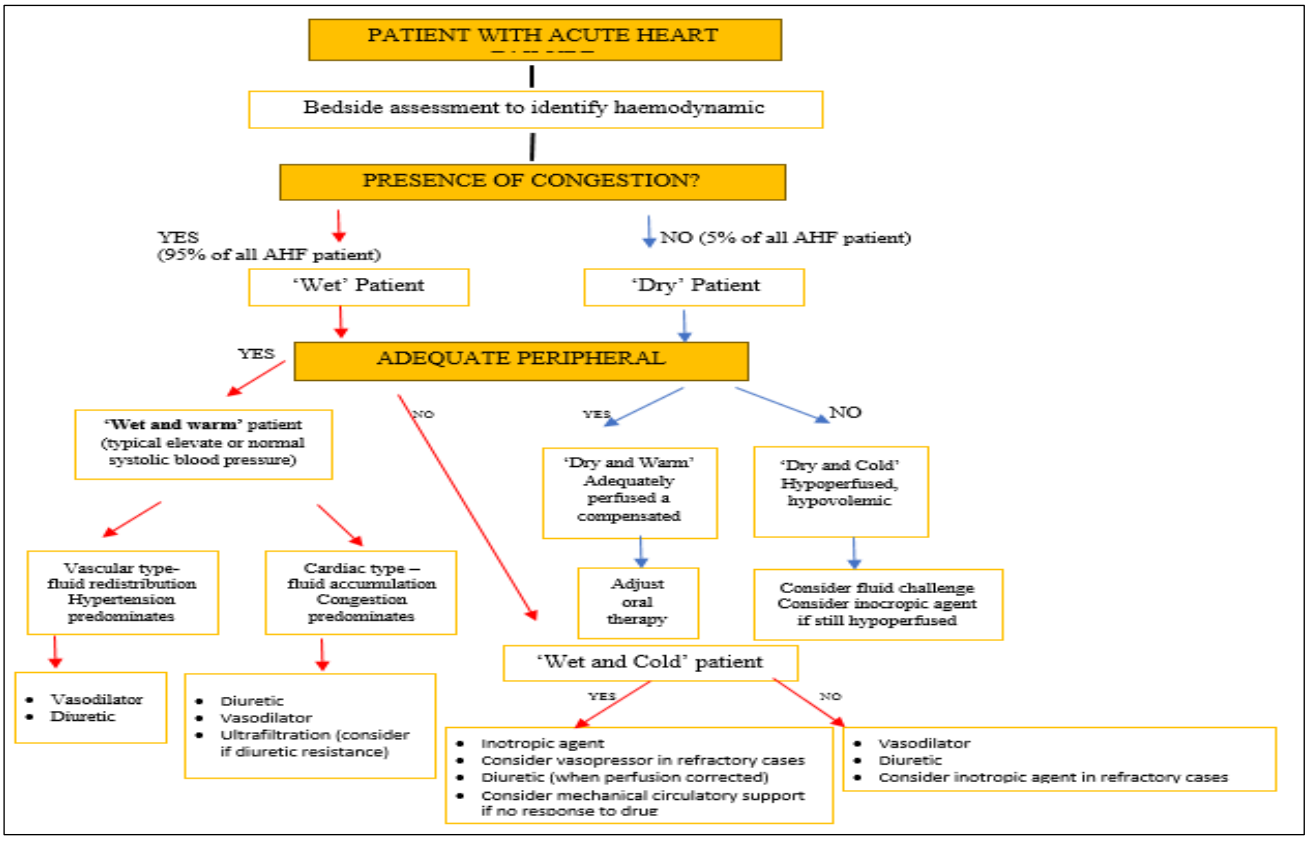

Figure 5: Algorithm management for patient with acute heart failure ESC 2016.[25,26]

CONCLUSION

We need to understand this pathophysiology and its very prompt management in the ED cannot be emphasized more. Hypertensve crisis is the extreme end of the spectrum of acute pulmonary edema. It is one of those high-yield emergencies presenting to the ED which when treated quickly and effectively will not only save a life but also obviate the need for intubation and ICU care. In this subset of patients, there is redistribution of fluid in the body mainly in the lung while patient may still be hypovolemic or euvolemic. The patients present to the emergency with extreme respiratory distress associated with restlessness, diaphoresis, and high blood pressure (usually systolic above 180mmHg). The mainstay of management is NIV/HFNC and high-dose nitrate infusions within minutes of the patient's arrival to the ED. Use of intravenous diuretics and morphine, although popular, is not supported by good scientific evidence.

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