Modern Management of Cardiogenic Pulmonary Edema

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Pathophysiology

CPE results from leakage of fluid from the pulmonary capillaries and venules into the alveolar space as a result of increased hydrostatic pressure. When the pulmonary capillary hydrostatic pressure exceeds pulmonary interstitial pressure, fluid transudates into the pulmonary alveoli and interstitium [2]. Hydrostatic pressure rises when the left ventricle (LV) is unable to e

myocardial performance pulmonary edema infarction occurs
least 25% of LV myocardial mass. Acute severe hypertension can also

Several other conditions can also result in CPE by exacerbating LV function, including sepsis, sgoxicosiumdisis,

impairs image quality or may mimic vascular redistribution. Image quality is also often impaired in obese patients and patients who develop COPD [10].

The clinical diagnosis of acute heart failure is estimated to be incorrect in more than 50% of cases, with frequent overdiagnosis and underdiagnosis [11–13]. Because of the limitations of clinical assessment and chest

Most patients who experience CPE, however, do not have ECG evidence of an acute dysrhythmia or AMI. Treatment should therefore be aimed at redistributing the excessive pulmonary interstitial fluid into the systemic circulation, which improves alveolar oxygen-carbon dioxide exchange and hypoxia; therefore, pharmacologic agents that provide preload reduction and afterload reduction should be administered. In some cases, inotropic support is required also.

Pharmacologic therapy for CPE includes pharmacologic agents that should be admac

when patients require anxiolysis. Low-dose benzodiazepines have a more preferable side-e

(captopril, enalapril) forms of ACEIs to patients who develop CPE is associated with reductions in systemic vascular resistance (afterload) and

The catecholamine inotropes include dobutamine, dopamine, and norepinephrine. No studies to date have demonstrated a mortality benefit of these over the others in the ED setting. Dobutamine provides the advantage of inducing mild reductions in preload and afterload in addition

cardiac output and PCWP. Patients who received levosimendan also had a lower 180-day mortality rate (26% versus 38%) compared with patients who received dobutamine. Although the study demonstrated in-hospital and

minutes, and they have been demonstrated to reduce ICU use, intubation rates, and hospital costs.

Inotropes should not be used routinely in patients who present with CPE; however, in patients who develop CS or in patients who cannot tolerate preload- and afterload-reducing8((wh446)-7.1(bo)-6ca.8cing)-446ofcingita8-376.1cingus

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