RE: Pulmonary edema complicating ovarian hyperstimulation syndrome: low-pressure edema, high-pressure edema, or mixed edema?

To the Editor: I read with interest the case report of pulmonary edema complicating ovarian hyperstimulation syndrome (OHSS): low-pressure edema, or mixed edema? by BaHammam[1] and would like to highlight some of my concerns.

First, I agree with the paradigm that OHSS causes a capillary leaky syndrome similar to that seen with major extravascular fluid transudation and third spacing occurs under low-pressure pulmonary edema (if no underlying cardiac disease is present) and elevated right-sided heart pressures. When this pulmonary edema is occurring with high left-side heart filling pressure in the absence of pulmonary veno-occlusive disease or mitral stenosis, we need to think about some sort of either systolic or diastolic myocardial depressant state caused by toxins, sepsis, hyperdynamic, hypermatabolism (high output), arrhythmia, or ischemia, besides the volume overload.

In this report the presence of low systemic vascular resistance, high WBCs, and hypotension can indicate a systemic inflammatory state, infection or sepsis that can cause transient myocardial depression and high filling pressures (CVP and PCWP), a part of the fluid overload state. Despite that the measured cardiac index was 5.6 L/m/m^2, it should have been much higher under such a hypermetabolic condition and hyperdynamic circulation in a young person with normal myocardial function. I think in this situation it would have been very helpful to have information regarding echocardiographic details of cardiac function at the time of ICU admission, a sepsis work up, and repeat follow up echo and full hemodynamic parameters to concur with the final conclusion.

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Reply to RE: Pulmonary edema complicating ovarian hyperstimulation syndrome: low-pressure edema, high-pressure edema, or mixed edema?

To the Editor: I read with interest the valuable comments of Dr. Hassan. Traditionally, diffuse alveolar infiltrate complicating ovarian hyperstimulation syndrome (OHSS) has been attributed to an increased vascular permeability. However, OHSS is consistently associated with a circulatory dysfunction characterized by arterial hypotension, low peripheral vascular resistance, increased activity of the renin-aldosterone system, marked stimulation of the antidiuretic hormone, and sodium retention.[1] Despite different theories, the exact pathogenesis of circulatory dysfunction during OHSS remains unknown. Massive hydration in patients with circulatory dysfunction may lead to further deterioration of pulmonary edema and respiratory symptoms. This theory is supported by the fact that reported cases of alveolar infiltrate complicating severe OHSS demonstrated marked improvement in patients' conditions after fluid restriction and loop diuretics.[3,4] Furthermore, an autopsy case of OHSS reported massive pulmonary edema without diffuse alveolar damage, hyaline membrane formation, or interstitial inflammatory infiltrate supporting the possibility of high pressure edema.[4] The pathogenesis of pulmonary edema complicating severe OHSS appears to be multifactorial. Transient cardiac dysfunction could be one of the pathogenic causes of pulmonary edema. Future research should address different factors that may affect cardiac function in patients with severe OHSS, including the associated physiological and inflammatory changes, with a special focus on the subgroup of patients who develop severe pulmonary edema.

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