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Tamponade without the Tamponade: A Case of Severe Decompensated Pulmonary Hypertension

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Background:

Pericardial effusion is a known complication of pulmonary artery hypertension (PAH), and while its incidence is as high as 44% (1), only approximately 0.02% of patients with PAH will develop an effusion large enough to cause cardiac tamponade. Pericardial effusion is a strong indicator of mortality in PAH and even more so with acute cardiac tamponade (2,7). The PAH patient's ability to compensate for the presence of pericardial effusion is very limited. Our case presentation serves to illustrate the complex hemodynamics of the PAH patient with tamponade and reviews its management.

Methods: A 45-year-old Hispanic woman with morbid obesity presented with significant worsening of her progressive, chronic dyspnea of two years duration. On presentation, she was tachypneic and hypoxemic but without hypotension. She had no prior history of pulmonary or cardiac disease, and rheumatologic work had been negative. She had evidence of jugular venous distension, a loud S2, and pulsus paradoxus. Transthoracic echocardiogram revealed a severely dilated, hypokinetic right ventricle, moderate tricuspid regurgitation, paradoxical septal motion and a large, concentric pericardial effusion, with a bigger acoustic window posterior to the left chambers while the patient laid supine. Echocardiographic criteria for tamponade were not met. Right heart catheterization was promptly performed.

Results: Inhaled nitrous oxide (iNO) at 20 ppm was promptly initiated. iNO was eventually replaced for epoprostenol infusion with the goal of reducing mean pulmonary artery pressure (PAPm) below 60 mmHg in preparation for pericardiocentesis. Percutaneous pericardiocentesis was never attempted due to the absence of a safe anterior acoustic window, as most of the pericardial fluid collected in dependent areas of the pericardial sac, with the largest diameter measuring 3 cm. The cardiothoracic surgery consultant felt a surgically performed pericardial window was too risky, with low likelihood of survivability. The patient's condition continued to deteriorate, requiring initiation of norepinephrine infusion on fifth day of hospitalization. Ultimately, as the patient's clinical status continued to decline, her family decided to concentrate on comfort interventions only. The patient was liberated from mechanical ventilation and expired shortly after.

Conclusions: The clinical presentation, sonographic findings, and hemodynamic information of the PAH-tamponade patient is different from the traditional pericardial tamponade without pulmonary hypertension patient. Any large pericardial effusion in a subject with PAH, even if only located posteriorly in the pericardial sac, must raise suspicion for tamponade physiology. The diagnosis of tamponade is complex and all information must be carefully integrated. Traditional therapeutic interventions, particularly abrupt drainage, may be disastrous. We highly recommend a multidisciplinary approach, including input from cardiology, cardiothoracic surgery and the pulmonary hypertension specialist. Despite a multidisciplinary approach, mortality remains very high with any intervention.

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