Transudative Chylothorax in the Setting of Acute Right Heart Failure in a Patient with Pulmonary Arterial Hypertension (PAH): A Case Report.

Reid J¹, Garcia K², Brumley K², Fried J², Taylor T², Sager J.S².
¹Keck School of Medicine of USC, ²Santa Barbara Cottage Health System; United States

Background: Transudative chylothorax is a rare finding, with only 15 reported cases in the medical literature. Suggested etiologies include translocation of chylous ascites across the diaphragm, described in patients with nephrotic syndrome or cirrhosis; or by increased lymph production secondary to high pulmonary pressures, decreased thoracic duct inflow, or formation of lymphatic venous collaterals, described in patients with heart failure. We report a case of transudative chylothorax in the setting of acute right heart failure from pulmonary arterial hypertension (PAH).

Case Report: Patient is a 62 year-old man with a past medical history significant for idiopathic PAH transferred to our medical center after developing acute hypoxemic respiratory failure and right heart failure secondary to fluid overload after an elective left hip arthroplasty three days prior. We stabilized his acute right failure with continuous diuresis, inhaled epoprostenol, and ventilator and pressor support. Echocardiogram on admission demonstrated severe right atrial and ventricular enlargement, severe pulmonary hypertension with estimated RVSP of 60-70mmHg, a severely dilated pulmonary artery at 3.2cm, and a dilated non-compressible IVC. Right atrial pressure measured on admission was 20 mmHg. Initial imaging showed a right-sided pleural effusion as well as moderate ascites. Additionally, the patient presented with acute renal failure that corrected over the ensuing week with aggressive diuresis. His serum creatinine dropped from 2.43 to 0.92 with a negative fluid balance of 20L at the end of his hospital stay. Despite diuresis, follow-up imaging demonstrated persistence of the right pleural effusion. Thoracentesis revealed milky pleural fluid with triglyceride level 207, pH of 6.98, amylase of 17, glucose of 119, LDH of 99, and total protein of 3.3. Microbiology cultures were negative and white cell count was 1421, monocyte/lymphocyte predominant. The protein pleural effusion to serum ratio was calculated as 0.452. The LDH effusion to serum ratio was calculated as 0.478. This confirms an acidic transudative chylothorax. Paracentesis revealed serum ascites albumin gradient (SAAG) of 1.0. Ascitic fluid triglycerides were 131, ascites fluid total protein was 4.0. Of note, the patient’s serum triglyceride level was 77. Both thoracentesis and paracentesis were negative for infection or malignancy. He had normal synthetic and structural liver function and imaging revealed an enlarged liver consistent with “nutmeg” congestive hepatopathy. Dietary recommendations were made to reduce long chain fatty acid intake and to supplement with medium chain fatty acids in his diet. Patient did very well with resolution of the pleural effusion.

Conclusions: Transudative chylothorax is a rare type of pleural effusion; most often reported secondary to chyle leak associated with liver cirrhosis or heart failure. This report describes concurrent transudative chylothorax with borderline chylous ascites in a patient with acute decompensated right heart failure associated with PAH. The etiology of concurrent chylothorax with chylous ascites remains poorly understood. We postulate that acute increased right-sided heart pressures led to elevated pressure in the superior vena cava and back pressure into the thoracic duct. Reducing the pressure in the system resolved the chylothorax. We believe this to be the first reported case of transudative chylothorax in the setting of acute decompensated right heart failure secondary to PAH, which resolved with reduction of right-sided pressures.