中文題目：慢性腎臟病患者罹患射出分率正常心衰竭及乳糜胸的個案報告

英文題目：Chylothorax in chronic kidney disease with HFpEF

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Introduction: Chylothorax is the accumulation of lymph in the pleural cavity due to disruption or dysfunction of the flow of chyle through the thoracic duct. The etiologies of chylothorax include malignancy, sarcoidosis, amyloidosis, and traumatic injuries (catheter insertion, surgery etc.). Chylothorax is uncommonly seen in the chronic kidney disease (CKD). Most reported cases were caused by injuries, stenosis or thrombosis of the thoracic duct in central catheters placement. Herein we reported a CKD patient with HFpEF complicated with chylothorax, who recovered after drainage, nutritional support and optimal maintenance hemodialysis.

Case Presentation: An 89-year-old male with type 2 diabetes mellitus in CKD stage 4, was admitted due to dyspea, oliguria and bilateral lower legs edema. Patient was afebrile with blood pressure of 100/62 mmHg. Chest X-ray revealed cardiomegaly and bilateral pleural effusion, which was more prominent over the right side. Despite diuretic uses, patient has exacerbated bilateral pleural effusion and respiratory failure that need endotracheal tube for ventilator support. The NT-pro-BNP was greater than 10000 pg/mL. Albumin level was in normal range 4.0g/dL. Echocardiography demonstrated LVEF 79%, and moderate pulmonary hypertension. Right side thoracentesis revealed milky pleural effusion with lymphocyte predominant (92%). The pleural effusion analysis demonstrated triglyceride 165mg/dL, protein/serum protein=0.5, LDH/serum LDH=0.57. Tuberculosis survey revealed adenosine deaminase 74U/L (normal value <40U/L), while mycobacteria culture and tuberculosis PCR were negative. Serial imaging were arranged to elucidate the etiologies of chylothorax. Chest CT disclosed sub-pleural consolidation patch in LLL, r/o inflammatory process without notable lymphadenopathy or mass lesions. Lymphoscintigraphy neither displayed definite lymphatic leakage. The oxygen support tapered from FiO_2 80% to 30% after pigtail drainage. Maintenance hemodialysis also commenced due to progressive renal function deterioration to the uremic stage. After pigtail drainage, aggressive nutritional support, and optimal hemodialysis, the chylothorax resolved. Patient was discharged after 108 days' hospitalization and continued regular hemodialysis thrice weekly.

Discussion: CKD patients are usually afflicted with cardiorenal syndrome, including HFpEF. Lymphatic system plays the pivotal role in removing interstitial fluid from tissues. In heart failure, the elevated venous pressure renders increased water flux into the interstitial space, and the removal of interstitial fluid back to the venous system is further hampered by elevated central venous pressure. We speculated that the chylothorax of our patient might be caused by increased hydrostatic pressure forcing fluid leaving the vascular space (oliguria-induced volume overload), and decreased osmotic pressure from deranged capillary permeabilities (the uremic toxins) and malnutrition. Moreover, we also wonder whether CKD status might be complicated with disrupted lymphatic contractility and occult lymphatic anatomical abnormalities, which cannot be detected by

conventional lymphoscintigraphy. Advanced imaging modalities as dynamic contrast enhanced magnetic resonance lymphangiography can facilitate anatomical and physiological evaluation of the lymphatic flow, and help elucidate the mechanism underlying chylothorax in heart failure and cardiorenal syndrome

Conclusion: We reported the case of chylothorax in CKD with HFpEF. Pleural effusion is usually attributed to fluid overload or uremic status in CKD. Chylothorax might be overlooked without thoracentesis for diagnosis.