SEVERE PULMONARY HYPERTENSION IN A HEMODIALYSIS PATIENT: case report

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INTRODUCTION

Pulmonary arterial hypertension (PAH) is prevalent in hemodialysis patients with many risk factors including fluid overload, cardiac dysfunction, arteriovenous fistula, bone mineral disorders and imbalance between vasodilators and vasoconstrictors.
Here we present a case of severe PAH associated with right sided pleural effusion that partially resolved after drainage

CASE PRESENTATION

This is a 57 years old patient with primary oxalosis that led him to end stage renal disease in 2004. He underwent hemodialysis for one year then received a kidney transplant. Unfortunately his disease recurred and he was back on HD in 2008, with a three times weekly, four hours sessions. The dialysis sessions were initially well tolerated and he had no complaints other than back and joint pain.
Few months ago, he started complaining of anorexia and weight loss and was started on oral supplements.
He then complained of progressive shortness of breath despite lowering his dry weight. CXR showed right sided pleural effusion that revealed to be a transudate upon drainage. Cardiac echocardiography showed preserved systolic and diastolic function with a moderate PAH of 42 mm Hg. Albumin level was low at 20g/l. Dietary counseling was obtained.
During the following HD sessions, he presented several episodes of hypotension, despite stopping his three antihypertensive drugs, precluding efficient dialysis. A new 2D echocardiography showed no cardiac dysfunction but PAH had progressed to 88 mm Hg.
Few days later he was admitted to the ICU with severe systemic hypotension, hypoxia and drowsiness. CXR showed right sided pleural effusion with mediastinal shift, and a chest tube insertion drained 3.4 l of transudate. 2 D echocardiography performed a few hours later revealed partial resolution of the severe PAH, with a PAP of 45 mm Hg. Pulmonary artery catheter found a PAP of 42 mm Hg and a pulmonary capillary wedge pressure of 7 mm Hg.

Baseline	PAP= 42 mm Hg	Normal systolic and diastolic function
Before ICU Admission	PAP= 88 mm Hg	Normal systolic and diastolic function
After pleural effusion drainage	PAP=45 mm Hg	PAP = 42 mm Hg and capillary wedge pressure = 7 mm Hg

DISCUSSION

There are many intricate causes of PAH in our patient. In addition to known pathophysiological mechanisms, low albumin level and high right atrial pressure favored fluid accumulation in the pleural cavity. Our hypothesis is that at some point the pressure in the right pulmonary cavity, increased and transmitted to the major vessels, creating a vicious circle that was broken by the insertion of the permanent chest tube.

CONCLUSION

Massive pleural effusions can have a reversible pathogenic role in pulmonary arterial hypertension in hemodialysis patients.

51ERA-EDTA
468--MP
M) Dialysis. M3) Cardiovascular complications in in CKD 5D.
Hiba AZAR
DOI: 10.3252/pso.eu.51era.2014
Poster presented at: