Summary

Pulmonary hypertension (PHT) is a progressive fatal pulmonary circulatory disease that accompanies many conditions (including left to right side shunt) with compensatory elevated cardiac output. PHT also complicates chronic hemodialysis (HD) therapy immediately after the creation of arteriovenous shunt. Affected patients have significantly higher cardiac output.

The arteriovenous shunt formed for hemodialysis therapy is artificial often causing a large left to right shunt whose capacity often increases with time. Currently, there are no standard criteria regarding optimal arterio-venous (AV) shunt size.

In our study we looked at PHT in 80 consecutive patients with ESRD on regular hemodialysis via arterio-venous shunt (Group 1) in Benha university hospital and Kobry El-Kobba military hospital between Feb. 2009 and Feb. 2010 after exclusion of patients with possible secondary pulmonary hypertension. Forty random CKD (predialysis) patients were taken as a control (Group 2).

Patients were subjected to Full history taking, thorough physical examination, chest x-ray, twelve leads ECG, laboratory investigation and transthorcic echocardiography. The Pulmonary artery systolic pressure was obtained through estimation of RVSP though the trans tricuspid systolic

pressure gradient which was estimated according to Bernoulli equation: PAP = 4 x (tricuspid regurge peak velocity m/sec.)² + the right atrial pressure .

In presence of PHT, we reassessed cardiac output and sPAP following temporary compression of the AV shunt.

Comparing group1 and group 2, there were no significant differences between both groups with regard to age, sex, DM, HTN, HR, systolic BP, diastolic BP, Hb%, ECG, Chest X Ray, LVH and diastolic dysfunction ,But group 1 had a significantly higher S.Creatinine, COP, sPAP and EF% than group 2.

In group 1, 16(20 %) patients had PHT and 64 (80%) patients had no PHT with mean sPAP (33.1332±15.57697) mmHg whereas in group 2, 5(12.5 %) patients had PHT and 35 (87.5%) patients had no PHT with mean sPAP (26.0883±8.66754) mmHg.

Of group 1, data of patients with PHT (group A) and without PHT (group B) were compared. There were no significant differences between both groups as regard to age, sex, DM, HTN, shunt site, HR, systolic BP, diastolic BP, S.Creatinine, EF%, LVH, and diastolic dysfunction, but group A had significantly longer duration of dialysis, lower Hb%, and Higher COP than group B.

PAP was estimated before and 1 min after arteriovenous access compression in PHT group. During this maneuver, the mean cardiac output decreased from (9.256±1.7538) L/min to (7.775±1.7842) L/min with highly

significant difference (P-value< 0.001), while the mean systolic PAP decreased from (59.1644±17.28545) mmHg to (49.6300±14.91912) mmHg with highly significant difference (P-value< 0.001).

Our study demonstrated a high prevalence of PHT among patients with ESRD receiving long-term hemodialysis with surgical arteriovenous shunt. Both ESRD and long-term hemodialysis via arteriovenous access may be involved in the pathogenesis of PHT by affecting pulmonary vascular resistance and cardiac output. Pathological elevation of PAP occurs in those patients whose pulmonary circulation can not compensate for arteriovenous access-related high cardiac output. This unrecognized complication of hemodialysis therapy is not uncommon.

Estimation and follow up of PAP using Doppler echocardiography may be indicated in all patients with ESRD undergoing hemodialysis via arteriovenous shunt. Increased unexplained PAP is a call for further investigation, which may include assessment under careful temporary compression of the arteriovenous access. Standardization of arteriovenous access size and a reliable method for shunt estimation will be of great assistance in the future as increased cardiac output and arteriovenous shunt have large impact in development of pulmonary hypertension in those patients.