**EMBOLIC PARADOX: WHEN TWO WRONGS MAKE A RIGHT**

**V. Tavakoli**, R. Bindu, C. Philip, R. Grodman, D. Bloomfield

Richmond University Medical Center, Staten Island, NY, USA

We present a rare phenomenon that pulmonary embolus gets trapped in patent foramen ovale (PFO) and then, returns back to the pulmonary circulation. A 74 year old woman presented with worsening dyspnea. Examination revealed increased respiratory and pulse rate. Saturation was 91%. Ventilation-perfusion lung scan showed multiple large segmental perfusion mismatches. Lower extremities duplex scan showed deep venous thrombosis. The patient was started on anticoagulation. An initial Trans-Esophageal Echo (TTE) revealed a serpingenous mass straddling a PFO. A repeat TTE on the third day of admission revealed no evidence of intracardiac masses in either atrias or ventricles. Patient offered no new symptoms between the two echo studies. In light of the vanishing atrial masses, another TEE was performed, confirming the absence of right or left atrial thrombi. A PFO was demonstrated by both color flow Doppler and microbubble contrast echo. Severe RV dilatation and hypokinesis were noted. A massive clot-burden was seen in the proximal pulmonary arteries. A CT angiography, afterward, showed central saddle pulmonary embolus (PE). The patient underwent surgical pulmonary artery thrombectomy that revealed thrombus in pulmonary arteries. The pathophysiology behind this phenomenon is based on the pressure changes in the setting of pulmonary embolism. The initial PE induced acute core pulmonale (as documented by the initial TEE), causing a right to left PFO flow. The direction of the flow caused the emboli coming from the lower extremity to get trapped through the PFO. However, the rapid pressure elevation, caused by the acute pulmonary embolism, was subsequently relieved when PE was lysed over time. Eventually, the right ventricular pressure decreased and the PFO shunt reversed to left-to-right, forcing the embolus to go back toward the pulmonary artery and causing another pulmonary embolism in the pulmonic circulation (as documented by the second TEE).