Conclusions: PFO closure may resolve symptomatic postural dyspnea and hypoxemia and is an effective method for treating OPs.

TCT-160
The Genetic Basis Of Patent Foramen Ovale
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Background: Patent foramen ovale (PFO) is a remnant of normal fetal development that persists in >25% of the population. It is a canal connecting the atria that allows the transit of blood from venous to arterial circulation, bypassing the pulmonary circuit. PFO is associated with cryptogenic stroke, migraine, visual auras, and other medical conditions. Based on the analysis of family pedigrees, previous studies suggest that PFO is an inherited condition. The aim of this novel study was to elucidate the specific genes involved in maintaining patency of the foramen ovale after birth.

Methods: Medical records of patients with identified PFO were reviewed for family history of PFO prevalence. Of 750 patients with PFO, 26 families were identified having multiple members diagnosed with PFO. Five families (16 individuals) were recruited for genetic testing. PFO was diagnosed using transcranial Doppler imaging with agitated saline. Serum DNA was collected from the 16 subjects, and was exome sequencing revealed 25 mutations on 13 unique genes involved in maintaining patency of the foramen ovale after birth.

Results: Patients with IVC rim deficiency had significantly larger defect size and increased Qp/Qs ratio compared to patients without IVC rim deficiency. Although patients with IVC rim deficiency had significantly lower procedure success rate, more than 90% patients with IVC deficiency could be treated with low complication rate such as device migration or cardiac erosion. (Table)

Conclusions: Under the appropriate device selection and device deployment technique, majority of patients with IVC rim deficiency can be treated by transcatheter closure with low complication rate.

TCT-162
Paradoxical embolic myocardial infarction
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Background: Despite several negative prospective randomized trials on the efficacy of patent foramen ovale (PFO) occlusions the discussion on the validity of these studies and on new indications is ongoing. We investigated the risk of paradoxical embolic myocardial infarction over a time period of more than 13 years.

Methods: Our effort to describe the epidemiology of paradoxical embolic myocardial infarction included a retrospective and a prospective approach: We analyzed retrospectively the incidence of paradoxical coronary embolism over 10 years and then prospectively undertook the effort to prove this using the same methodology over 39 months in another center. All patients with acute myocardial infarction (AMI) according to ECG criteria and elevation of cardiac troponin or creatin kinase and normal coronary arteries were screened for PFO and if no other reasons for the AMI could be excluded PFO was considered as presumed paradoxical embolism.

Results: In a retrospective analysis over 10 years amongst 4848 AMIs 22 patients (0.45 %) were identified to have had presumed paradoxical embolic pathogenesis based on strict criteria of absence of atherosclerosis and virtual absence of atherosclerosis in any vascular territory. To further elucidate the epidemiologic size of this disease entity we prospectively studied 1654 AMIs in another tertiary referral center over a time period of 39 months and found an incidence of 13 presumed paradoxical embolic AMIs (0.79 %) based on application of the same criteria. The patients in the paradoxical embolic cohorts were younger (45.7 ± 11.5 years) as compared to the overall AMI population (65.0 ± 13.2 years), most likely based on the criteria applied (exclusion of detectable atherosclerosis).

Conclusions: Paradoxical embolism through a PFO is the most likely cause of AMI in roughly 5/1000 patients. While this is a rather low rate, the incidence is still high enough to warrant clinical attention.