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RESOLUTION OF PERSISTENT NOCTURNAL HYPOXIA IN A PATIENT WITH TREATED OBSTRUCTIVE SLEEP APNEA AFTER CLOSURE OF A PATENT FORAMEN OVALE

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Introduction: A higher prevalence of patent foramen ovale (PFO) is documented in individuals with obstructive sleep apnea (OSA). Concurrent OSA and PFO can lead to more severe nocturnal desaturation although the effect of closure of PFO on nocturnal hypoxia is unclear.

Report of Case: A 38 year old male with hypertension, tobacco use and obesity presented to the WLA-VA sleep center with a prior diagnosis of OSA. A polysomnogram (PSG) performed 7 years prior demonstrated an AHI of 99.0/hr with 4.7% of TST spent at an O2 saturation of <89% and he was successfully titrated to a CPAP of 10 cm H20. The patient was non-compliant with CPAP and was restarted on his prior settings of 10 cm H20 through the VA. Polycythemia (hgb 18) was noted and a workup was initiated. PFTs and CXR were normal and a blood gas showed a daytime room air PaO2 at 67mmHg. A 3-night oximetry showed long periods of time < 88% SaO2 while on CPAP (residual AHI < 5/hr). Supplemental oxygen with CPAP was initiated although nocturnal hypoxia persisted at 3L/min with CPAP. Echocardiogram with bubble study did not demonstrate a shunt but transcranial doppler revealed a high grade right-to-left shunt at rest, increasing with Valsalva maneuver. Cardiac catheterization showed xygenation step up from RA (74%) to RV (82%) indicating significant left to right shunt and TEE showed a PFO. Patient underwent successful percutaneous PFO closure. Repeat PSG on CPAP and room air showed resolution of nocturnal hypoxia and hgb remained normal after cessation of supplemental nocturnal oxygen.

Conclusion: Comorbid OSA and PFO may lead to difficult to treat nocturnal hypoxia. A randomized trial is needed to assess if closure of PFO improves nocturnal hypoxia in these cases.