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Published In/Presented At

Saif, H., Ahmed, S., Ayub, B., & Kluck, B. (2013, April 10-12). *Intracardiac echocardiogram in patient with patent foramen ovale and obstructive sleep apnea*. Poster presented at: The American College of Physicians Internal Medicine 2014, Orlando, FL.

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Intracardiac Echocardiogram in Patient with Patent Foramen Ovale and Obstructive Sleep Apnea

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Background and Purpose:

Summary of case:

We report intracardiographic evidence of right to left flow and transient atrial septal aneurysm during sonorous breathing. The purpose of this case report is to provide echocardiographic evidence of the mechanism of patent foramen ovale (PFO) in stroke patients. This case report emphasizes the significance of sonorous respiration in patients with obstructive sleep apnea and PFO.

Case:

62 year old male presented to ED with L sided weakness, persistent dizziness and nausea.

- He has a past medical history of hypertension, hyperlipidemia, gout, obstructive sleep apnea, and over past few months had early morning dizziness, nausea, and vomiting.
- Except for mild bilateral ataxia his physical examination was unremarkable.
- Brain MRI revealed large area of acute ischemia in left posterior inferior cerebellar area.
- Head and Neck MRA showed occluded left posterior inferior cerebellar artery (PICA), 2.5 mm right pericallosal artery aneurysm and TcD demonstrated high intensity transient signals, also known as cerebral microembolic signals, with and without Valsalva.
- Transthoracic echocardiogram (TTE) verified ejection fraction of 55 %. It also demonstrated PFO with mild left to right color flow without right to left flow.

Treatment:

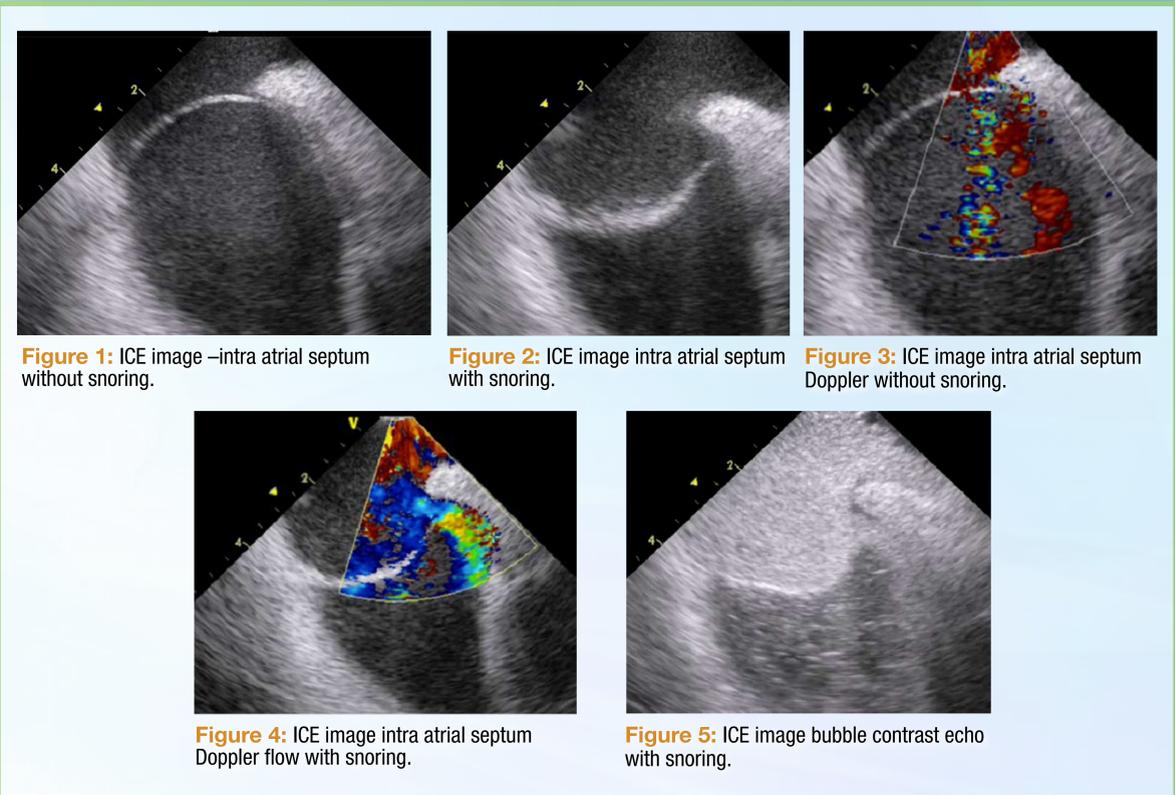
- After discussing options of treatments, percutaneous PFO closure was attempted.
- Image was performed using an 8 French AcuNav Intracardiac echo probe under sedation, which quickly produced sonorous respirations.
- Without snoring - virtually no right to left flow or atrial septal aneurysm (ASA).
- With snoring - a significant mobility of the septum primum leftward consistent with a large atrial septal aneurysm (ASA).
- In addition, there was a large right to left flow seen on color imaging and a significant flow of agitated saline contrast from right to left with sonorous breathing.
- Successful closure of PFO with 30 mm Gore Helix Device eliminated shunt flow

Conclusion:

- We are unaware of any publications demonstrating Intracardiac echocardiographic evidence of transient ASA and significant right to left flow across PFO only with sonorous breathing.
- What is the mechanism of PFO in stroke patients?
 - If, as in this patient, the mobility of the septum and opening of the foramen occurs only with snoring, the traditional screening tools to identify risk for subsequent stroke might be inadequate.
 - The patient is awake during TcD and TEE probe placement may disrupt intracardiac pressure changes associated with snoring.
 - This mechanism identified raises the possibility of an additional mechanism to explain wake-up stroke.

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Discussion:

For a stroke to occur in this fashion, four pre-requisites have to be met as described by Ryan et al.

- 1 Arterial embolus leading to cerebrovascular ischemia
- 2 Venous thromboembolism
- 3 Intra-arterial communication such as PFO
- 4 A gradient favoring right to left shunting.

All these four conditions are rarely demonstrated at the same time in a patient with cryptogenic stroke and PFO.

It has also been suggested that the PFO tunnel itself may be a potential source of thrombus.¹ Beelke et al demonstrated a high prevalence of PFO in patient with OSA with trans cranial doppler (TcD) with intravenous application of agitated physiological saline solution.²

Sleep apnea itself on the other hand leads to changes in intracardiac pressures leading to pulmonary hypertension which facilitates flow from right to left through PFO.^{3,4} Guchlerner et al demonstrated in their 100 patient study that the prevalence of right to left shunt is in fact high in Obstructive sleep apnea (OSA) population.⁵ Beelke et al verified through their study that even a single obstructive event is enough to cause right to left shunt with concomitant PFO.⁶

Snoring has been studied well in sleep apnea as a very useful indicator of apnea or hypopnea in patients with OSA.^{7,8} It has been speculated that there may be a functional hypercoagulable state in patients with OSA and that this may be contributory to increased rate of stroke in patients with OSA^{9,10} although sleep apnea has not been demonstrated as a single etiological factor for hypercoagulable state.

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