Spontaneous Echocardiographic Contrast in a Normal Heart

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Citation

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Abstract

Spontaneous echocardiographic contrast (SEC), is a swirling pattern of increased blood flow echogenecity, distinct from white noise artifacts in the left atrium or left atrial appendage (LAA). It is seen in conditions of blood stasis or low-velocity blood flow. It is associated with the formation of left atrial thrombus and increased thromboembolic complications, mostly seen in patients with manifest cardiac disease, during atrial fibrillation.
We present a case of a 65 year old female who was admitted for a transient ischemic attack. Post resolution she remained completely asymptomatic and had a negative diagnostic inpatient workup. Holter monitoring performed during both inpatient and outpatient management failed to demonstrate any arrhythmia. Transesophageal echocardiogram showed SEC in the LAA and a structurally normal heart. We believe this to be an unusual occurrence as the patient had no underlying structural heart disease and remained in sinus rhythm during extended inpatient and outpatient monitoring.

CASE REPORT

A 65 year old female presented to our hospital with an acute onset of headache, difficulty swallowing and expressing herself, right sided facial droop and drooling. These symptoms began approximately an hour earlier at home. She had no other complaints and denied similar complaints in the past.

The patient's past history was significant for breast cancer status post lumpectomy, melanoma removal, partial nephrectomy secondary to pyelonephritis and hypertension. Her medications included anastrozole, atenolol, megestrol, clorazepate and temazepam. She was allergic to compazine. She drank one glass of wine per day, and denied tobacco or illicit drug use.

Initial neurological examination showed mild right sided facial droop, and slurred speech. Her symptoms gradually began to improve, to resolution within two hours from time of presentation. Repeat neurological examination was completely normal. Patient's temperature was 98.4 degrees Fahrenheit, pulse of 76 beats/min; regular, respiration 16 /min and a blood pressure of 162/96 mmHg. Cardiac examination showed a regular rhythm, and otherwise unremarkable.

Serum electrolytes were within normal limits, with no cardiac enzyme leaks; electrocardiogram showed sinus rhythm with nonspecific ST and T wave changes. A computerized tomography (CT) scan of her brain showed no acute intracranial process. Patient was admitted for observation, placed on twenty four hour Holter monitoring, and started on a heparin drip. The following day an ultrasound of carotid arteries showed no evidence of hemodynamically significant stenosis. A transthoracic echocardiogram showed mild concentric left ventricular hypertrophy and an ejection fraction of 60 percent, with mild tricuspid regurgitation and no evidence of pulmonary hypertension, patent foramen ovale (PFO) or thrombi. She remained in sinus rhythm throughout her hospital stay ; her blood pressure was consistently below 150/90 mmHg.

Based on these findings and complete resolution of patients symptoms, the patient was discharged home with a diagnoses of transient ischemia attack (TIA), started on clopidogrel, and was to follow up for a transesophageal echocardiogram (TEE) in two weeks. The TEE demonstrated spontaneous echocardiographic contrast (SEC) in the left atrial appendage (LAA) (Fig.1A&B), which was poorly contractile with a left atrial appendage emptying flow velocity (LAAEV) of 25-30 cm/sec. The left atrium was normal in size and no thrombus was identified within the chamber. The left ventricle showed concentric hypertrophy; with normal systolic function. Also noted were mild mitral

valve prolapse with mild regurgitation, mild pulmonary hypertension with a pulmonary artery systolic pressure of 35-40 mmHg and a small PFO with a left-to-right shunt. Based on these findings she was started on warfarin for six months. Repeat TEE at that time showed resolution of SEC and improved LAA contractility with LAAEV of 60 cm/sec and bidirectional flow through the PFO.

Video: Transesophageal echocardiogram (TEE) demonstrating spontaneous echo contrast in the left atrial appendage.

DISCUSSION

Spontaneous echocardiographic contrast (SEC), also known as “smoke”, is a swirling pattern of increased blood flow echogenecity, distinct from white noise artifacts in the left atrium or LAA. It is caused by ultrasonic backscatter from red blood cell aggregates, seen in conditions of blood stasis or low-velocity of blood flow. The clinical importance of SEC is its association with left atrial thrombus and increased thromboembolic complications and is a frequent finding in patients with atrial fibrillation (AF), mitral stenosis and in patients with prior thromboembolism, occurring in 30%-60% of such patients[1]. Thrombus and/or SEC develop in LAA in patients with LAA dysfunction[2], mostly seen in patients with manifest cardiac disease[1].

Evaluation of our patients past medical history revealed no predisposing factors to the development of SEC in the LAA. All diagnostic studies performed both during her hospital stay and as an outpatient were normal. No structural or functional cardiac abnormalities were found that could predispose the formation of SEC in the LAA.

It has been demonstrated that >15% of strokes originate from the heart and from the LAA in particular[3]. The LAA is a blind-ended, complex structure embryologically distinct from the body of the left atrium which is routinely analyzed by TEE looking for thrombi, SEC[4], and abnormalities in emptying flow velocities, especially in patients with CVA. LAA function is determined with the help of TEE evaluation of LAAEV. The LAAEV is measured by positioning the Doppler sample volume in the proximal third of the appendage during end-diastole. It has been shown that LAAEV < 55 cm/s is coupled with elevated risk for the formation of SEC, while patients with AF an LAAEV of < 35 cm/s is associated with elevated embolic risk[5].

In AF, SEC occurs in 45% to 60% of patients and is associated with large LA dimension and decreased atrial mechanical function[1]. Sadanandan et al.[1] in a study of 1,288 patients demonstrated SEC in the LA in 24 patients in sinus rhythm, 2% of the overall TEE population, 50% of which had been referred to detect a source of CVA. They demonstrated that prior CVA, large LA size and decreased mean LAAEV were the only independent factors predisposing to SEC in patients in SR[1]. Studies have shown various factors affect the LAA and the formation of SEC. Elevation of left ventricular and/or left atrial pressures can independently or together result in reduction of LAA function[6], while grade 2 or grade 3 untreated hypertension can lead to increased left atrial afterload and impairment of LAA function, especially in patients with left ventricular systolic dysfuntion[7]. Reduced left ventricular function predisposes to the formation of SEC/thrombi, a study by Handke et al.[5] showed that LAAEV < 55cm/s, left ventricular ejection fraction <35 % and left atrial diameter >45 mm were all independent predictors of SEC in patients with stroke and reduced left ventricular function in sinus rhythm. Kaneko et al[8] compared LAA function in patients with and without cardiogenic brain embolization (CBE) with paroxysmal AF (PAF) in sinus rhythm and found that the group with CBE had significantly larger LAA area, smaller LAA-fractional area change and a higher incidence of LAA-SEC. Reexamination of LAA function done two weeks post initial evaluation showed significant improvement of all abnormalities observed initially, demonstrating LA and LAA stunning secondary to PAF. Enlarged LA size/diameter is a consistent finding in all studies evaluating factors affecting LAA dysfunction and/or SEC and thrombi formation[1,2,3,4,5,6,7,8].

The presence of SEC and LAA dysfunction on TEE is not an unusual finding. Our patient is unique as no structural or functional abnormality could be found to explain SEC and LAA dysfunction found on TEE. Both her left atrial size and function were normal as were her left ventricular size and function, with no valvular or structural abnormalities predisposing to SEC or LAA dysfunction. Based on the Kaneko study, paroxysmal atrial fibrillation could be a possible cause, but with no past history of symptoms, a structurally normal heart, and consistently negative cardiac monitoring (in hospital as well as on outpatient basis) the probability decreases. Infarction of the arteries to the LAA can result in a decrease of LAAEV, but subsequent resolution would not be seen. We did not perform any coagulation studies on our patient during her hospital stay or at follow up, though this could possibly explain the SEC, it would not explain the LAA dysfunction.

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