Visualized Embolization of Inferior Vena Cava Thrombus During the Abdominal Compression Maneuver to Evaluate for a Patent Foramen Ovale

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INTRODUCTION

Transthoracic echocardiography (TTE) remains the initial imaging modality of choice in diagnosing a patent foramen ovale (PFO) due to its availability, cost-effectiveness, and ease of use.[1,2] A transesophageal echocardiogram (TEE) is the reference standard imaging modality for confirming the presence or absence of a PFO.[3] As the Valsalva maneuver is difficult or impossible to perform during TEE due to sedation, inferior vena cava (IVC) compression may be used to increase the probability of shunt detection.[4]

The IVC or abdominal compression maneuver was first described in the year 2017 as a novel method to detect PFO during TEE. A limitation of this method is moderate IVC compression in obese patients and its relative contraindication in patients with low cardiac output states or active gastroduodenal or hepatobiliary-pancreatic diseases.[4] The probable transit of an IVC thrombus immediately upon performing the compression maneuver during a TEE to diagnose a PFO has yet to be reported. We report a 57-year-old man who had a TEE following an ischemic stroke during which time there was visualized embolization of an IVC thrombus into the right heart chambers within minutes after performing the IVC compression maneuver.

HISTORY OF PRESENTATION

A 57-year-old man presented to the emergency department with new-onset right-sided weakness, right facial droop, and inability to speak for 3 hours. On examination, the patient was aphasic, had a right-sided facial droop, and power in the right upper and lower extremities was 1/5 and 2/5, respectively. A stroke code was called, and a stat computed tomography (CT) head scan was done that showed no intracranial abnormality. A CT angiogram of the head and neck showed an occlusion of the left M2 branches of the middle cerebral artery and a filling defect measuring 13 mm in diameter suggesting occlusion of the left internal carotid artery immediately above the bifurcation. The patient's prothrombin time was 14.2 seconds (12.0-14.8 seconds), international normalized ratio was 1.15 (0.9-1.12) and activated partial thromboplastin time was 27.8 seconds (22.8-36.5 seconds). The patient received tissue plasminogen activator and underwent a thrombectomy of the occluded M2 vessel, which was unsuccessful. Subsequent placement of 2 carotid stents did not recanalize the internal carotid artery occlusion. The patient was intubated for airway protection due to sedation from general anesthesia for the procedure. A repeat CT head scan was done on day 2 of hospital admission, which showed an evolving left middle cerebral artery infarct and a new attenuation in the right central sulcus representing a subarachnoid hemorrhage (SAH). Subsequent CTs showed that the SAH was stable and required no further intervention.

A TTE was done on day 2, which did not show any PFO using either color-flow Doppler or agitated saline bubble study. The left ventricular (LV) size and visually estimated LV ejection fraction were reported to be normal (ejection fraction ~ 60%-65%), and the LV diastolic function was indeterminate. The left atrial volume index could not be measured, but the left atrium (LA) was visually estimated to be normal, the right ventricle (RV) was poorly seen with mild tricuspid regurgitation, and a normal IVC dimension with failure to collapse by 50% consistent with a right atrial (RA) pressure of ~8 mm Hg was observed. There was no apical wall motion abnormality or an apical clot.

The patient was extubated on hospital admission day 3. Additional laboratory results showed that D-dimer was elevated at 3.14 μg/mL (0-0.52 μg/mL); lupus anticoagulant, protein S, antithrombin III, mixing studies, and factor V Leiden R506Q variant were all negative. Beta 2 glycoprotein (IgG, IgA, IgM), phosphatidylserine (IgG, IgA, IgM), and anti-cardiolipin (IgG, IgA, IgM) were also within normal limits. Prothrombin gene mutation G20210 A was not detected.

The patient was alert and responded to commands, although aphasic, and was discharged to an acute rehabilitation facility for continued care with a high dose of atorvastatin (80 mg orally). No antiplatelet medication was given due to SAH. At the rehabilitation facility, due to swelling of the patient's right lower extremity, an ultrasound was performed that showed acute clots in the superficial femoral vein, popliteal vein, posterior tibial vein, and peroneal vein. The patient was transferred back to this hospital due to acute onset of a right lower extremity deep venous thrombosis (DVT). They were started on a heparin drip while awaiting IVC filter placement and monitored closely for clinical signs and symptoms of intracranial hemorrhage due to the recent history of SAH.

An IVC filter was placed in the infrarenal portion of the IVC (Figure 1). The filter placement was confirmed by fluoroscopy without any evidence of IVC or pelvic vein thrombus.

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TEE, upper transgastric horizontal (0°) view before (*left*) and 7 minutes after (*right*) IVC compression maneuver demonstrates the absence of (*arrow*) and then the newly identified, large serpiginous, highly mobile FFRHT (*arrow*) in the RA and RV. *TV*, Tricuspid valve.

Video 2: Two-dimensional TEE, midesophageal bicaval (120°) view during agitated saline bubble study obtained 1 minute after IVC compression, demonstrates the PFO with small right-to-left shunt and bubbles (*arrow*) within the LA.

Video 3: Two-dimensional TTE, subcostal view obtained 2 days post–IVC compression, demonstrates a highly mobile serpiginous thrombus emanating from the IVC into the RA.

Video 4: Two-dimensional TTE, subcostal view obtained 6 days post–IVC compression, demonstrates partial resolution of the thrombus (*arrow*).

View the video content online at www.cvcasejournal.com.

A TEE was done on hospital day 2 due to the possibility of a PFO having caused the stroke. Soon (i.e., approximately 7 minutes) after performing the IVC compression maneuver to diagnose a PFO, a new serpiginous echo density from the IVC-right atrium (RA) junction extending into the RV was seen in transit, which was absent during the beginning of the study (Table 1, Video 1, Figure 2). This was consistent with a thrombus that may have embolized during the IVC compression maneuver. A PFO was diagnosed with bubble passage from the RA to the LA after release of the compression maneuver (Video 2, Figure 3). No left atrial or left atrial appendage thrombi were detected.

A repeat TTE was done 2 days later and showed that the thrombus was still present in the distal IVC, RA, and RV (Video 3, Figure 4). The patient did not have any sequelae from thrombus embolization. A repeat TTE done after another 4 days showed partial resolution of the thrombus (Video 4, Figure 5). On discharge, the patient was instructed to start Eliquis 5 mg every 12 hours. Additionally, planned placement of a PFO closure device was arranged.

Our findings suggest the following diagnoses: acute ischemic stroke with subsequent SAH, acute DVT with placement of an IVC filter due to relative anticoagulation contraindication, probable embolization of an IVC thrombus during IVC abdominal compression maneuver, and a PFO.

DISCUSSION

The foramen ovale is a defect in the interatrial septum during the embryonic stage that spontaneously closes in 75% of the patients following increase in blood flow to the lungs and a drop in pulmonary vascular resistance. Failure of this defect to close is termed a PFO.[5] It is seen in about 15% to 35% of the adult population.[6] PFO is of potential clinical significance as it serves as a pathway between the venous and systemic circulation and may increase the chance of thrombi migrating between these 2 circulatory systems.[7] The presence of a PFO, an atrial septal aneurysm, or both increases the odds of development of an ischemic stroke by 3-fold in patients less than 55 years old.[8] The diagnosis of a PFO becomes important in patients presenting with a stroke as surgical closure is at times superior to medical therapy in secondary prevention and risk reduction of recurrent cryptogenic stroke.[9]

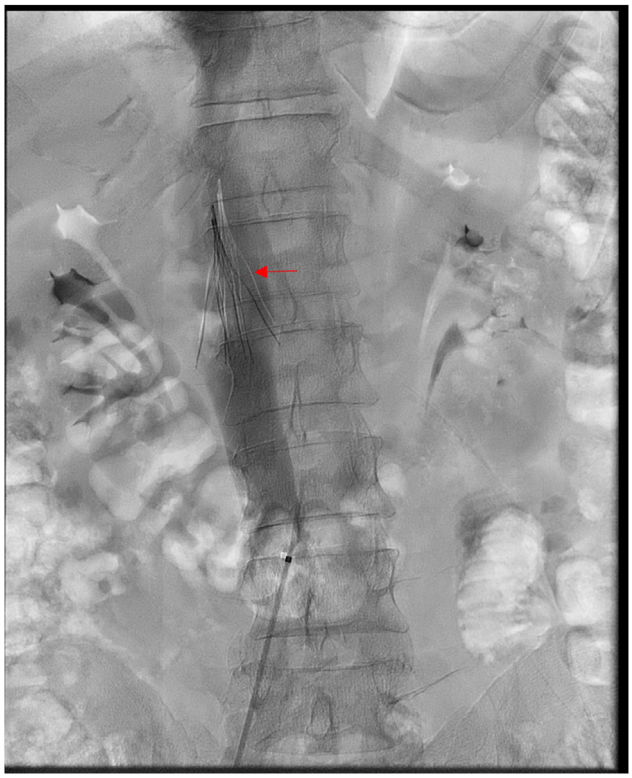

Figure 1 Venogram showing an IVC filter (*red arrow*), which was placed in the infrarenal portion of the IVC.

The sensitivity of diagnosing a PFO on TTE has improved over the years from 50% to 91% and continues to improve with the advent of three-dimensional TTE.[1,2] To confirm a diagnosis, contrast material crossing the RA into the LA needs to be demonstrated, which is usually done by injecting agitated saline into a peripheral vein and looking for entry of more than 1 microbubble in the LA within 3 cardiac cycles.[10] Maneuvers such as coughing or Valsalva can be used at the time of agitated saline injection to increase RA pressure, facilitating bubble entry into the LA. Demonstrating color-flow Doppler or contrast across the interatrial septum on TEE has a sensitivity of 100% and 89%, respectively.[3]

The study involving the utility of IVC compression maneuver in diagnosing PFO was conducted in 293 patients and compared with the current standard of practice of performing the Valsalva maneuver. The maneuver is described as compression of the right epigastric region for a period of up to 30 seconds with immediate release following RA opacification with microbubbles from peripherally injected bolus of agitated saline. It significantly improved detection of PFO compared with the Valsalva maneuver. IVC compression not only increased the RA pressure but also showed a significant reduction in LA pressures due to a decrease in preload during compression, thereby increasing RA-to-LA shunting.[4] In addition, it may also potentiate flap separation uncovering a PFO.[11] The advantage of this method is that it can be performed in individuals that have poor cognition, under deep sedation, and can be used as an adjunct to the Valsalva maneuver to diagnose PFOs. Our patient was not obese

Table 1 Timeline of TTE and TTE procedures during second hospital admission

Day/time	Modality	Findings
Hospital admission 1		
Day 2	TTE	No PFO, no clot
Hospital admission 2		
Day 2/time 0 minutes	TEE, precompression maneuver	No thrombus
Day 2/time 0 + 1 minute	TEE, during compression maneuver	PFO with small right-to-left shunt
Day 2/time 0 + 7 minutes	TEE, postcompression maneuver	Thrombus present
Day 4/time 0 + 2 days	TTE	Thrombus still present
Day 8/time 0 + 6 days	TTE	Partial resolution of thrombus

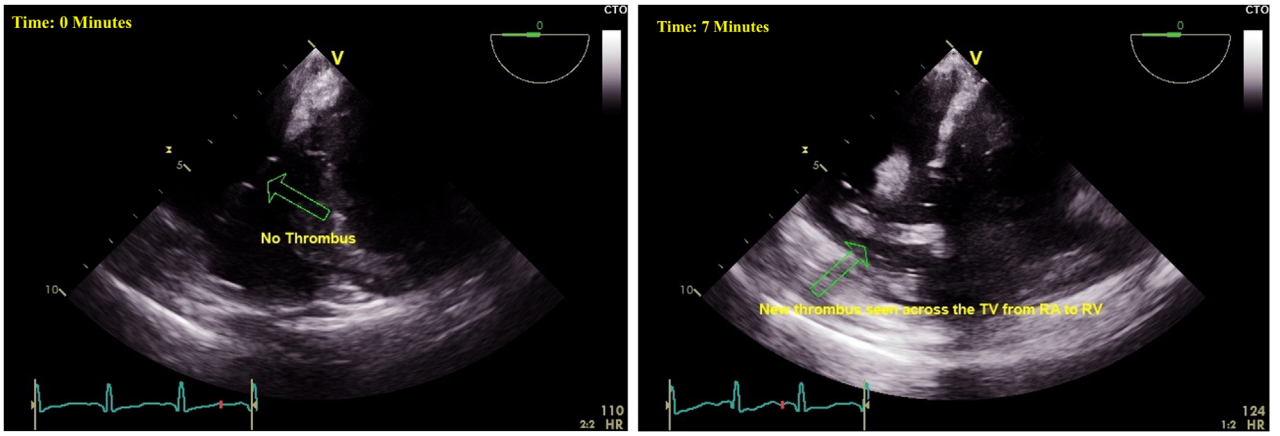

Figure 2 Two-dimensional TEE, upper transgastric horizontal (0°) view during diastole before (*left*) and 7 minutes after (*right*) IVC compression maneuver, demonstrates the absence of and then the newly identified, large serpiginous, highly mobile FFRHT (*arrows*) in the RA and RV.

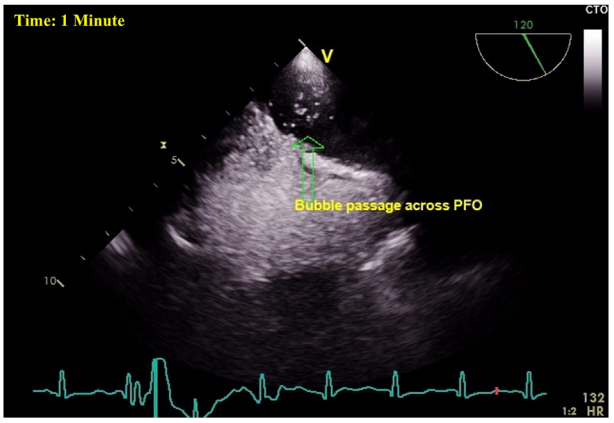

Figure 3 Two-dimensional TEE, midesophageal bicaval (120°) view during agitated saline bubble study, demonstrates the PFO with small right-to-left shunt and bubbles (*arrow*) within the LA.

and did not have any history of low cardiac output state or gastroduodenal or hepatobiliary-pancreatic diseases. On readmission with lower-extremity DVT and given the recent diagnosis of SAH, placement of an IVC filter in the infrarenal region was warranted. We believe that the IVC compression maneuver performed during TEE evaluation for this procedure contributed to the probable dislodgement of a thrombus from the IVC into the RA.

Free-floating right heart thrombi (FFRHTs) are a relatively rare phenomenon that can be detected by TTE, with CT being an alternative imaging modality.[12,13] The exact prevalence of FFRHTs is unknown due to underutilization of TTE, but it is estimated to be in the range of 4% to 18% among patients with pulmonary embolism (PE).[14] The thrombi can originate from peripheral veins in the leg and travel to the lungs via the right heart chambers; these are classified as type A thrombi. In other reported patients, the thrombi can originate from the right heart chambers, due to dilatation and stasis of blood within, and these are classified as type B thrombi. Type A thrombi are mobile, worm-shaped, unstable structures that are not attached to the walls of the right heart chambers, making them susceptible to cause PE, right heart failure, left heart failure, and subsequent cardiogenic shock. However, type B thrombi are attached to the walls of the right heart chambers and rarely embolize.[12] Clinicians must be extremely vigilante of patients confirmed to have FFRHTs on TTE as the in-hospital mortality in the context of PE could be as high as 45%, with 20% of the patients dying on day 1 of admission.[14] Commonly reported conditions that predispose to the development of FFRHTs include coronary artery disease, atrial fibrillation, valvular heart disease, COVID-19, and PFO.[12,15] PFO is considered a significant risk factor due to its anatomy that leads to stasis of blood and thrombus formation.[5]

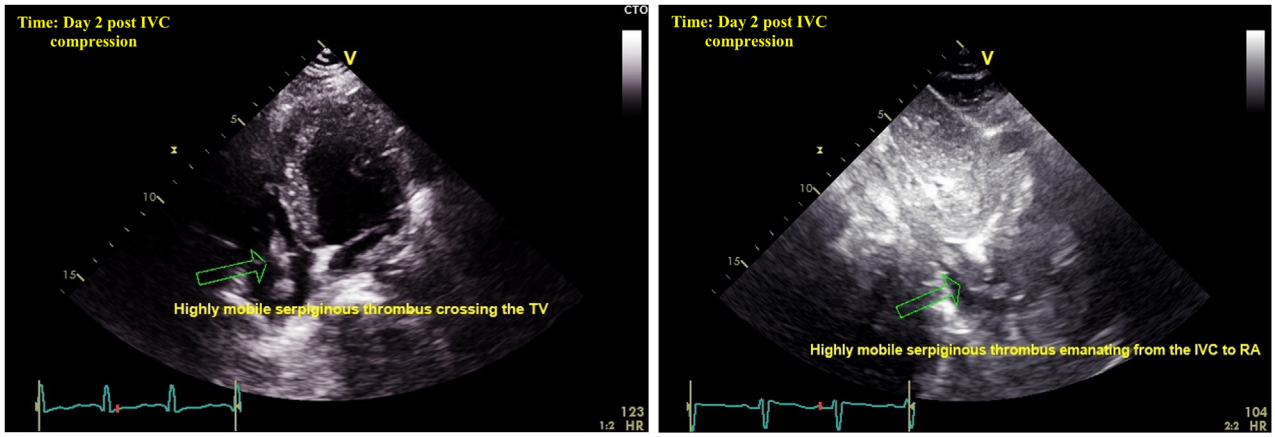

Figure 4 Two-dimensional TTE, apical 4-chamber view (*left*) and subcostal view (*right*) obtained 2 days later, demonstrates the thrombus (*arrow*) in the IVC, RA, and RV.

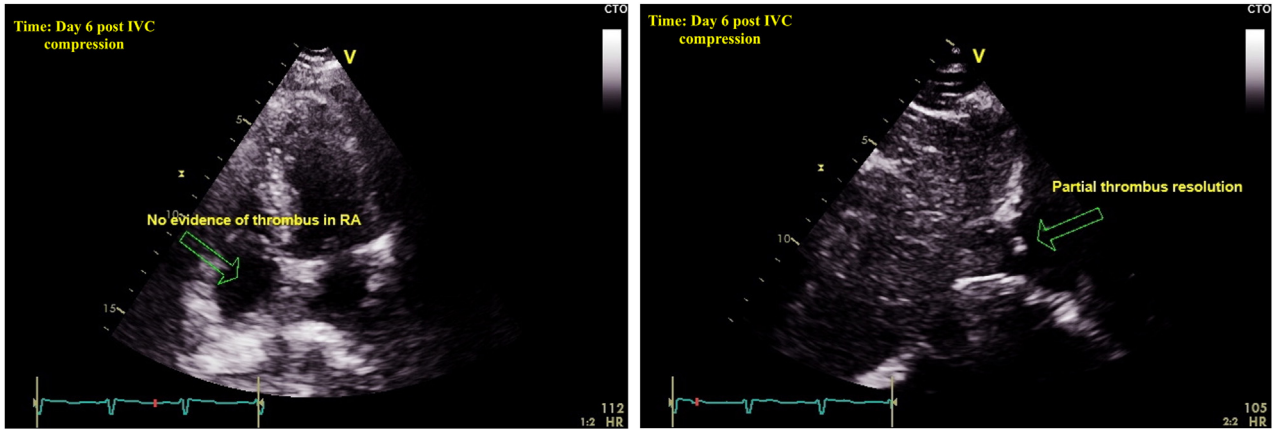

Figure 5 Two-dimensional TTE, apical 4-chamber view (*left*) and subcostal view (*right*) obtained 6 days later, demonstrates partial resolution of thrombus (*arrow*).

Some reports suggest that FFRHTs can lead to the development of a massive PE in almost 90% of the reported patients. However, that development was absent in our patient. Concomitant DVT is found in 28.8% of cases and is one of the important risk factors for FFRHTs, which was confirmed in our patient. In addition, PFO has also been shown to be more common in 35% of patients with an acute PE. The mortality rate in this subset of patients is also higher when compared to a subset of patients with an absent PFO. Our patient was also shown to have a PFO on TEE. Furthermore, patients with FFRHT often develop massive PEs associated with cardiogenic or obstructive shock, which was absent in our patient. Treatment options usually include thrombolysis, interventional catheter aspiration, or surgical thrombectomy. Heparin has also been shown to have favorable outcomes.[12] Our patient was treated with heparin with close monitoring of the mental status due to a recent history of SAH. The patient surprisingly did not have any complications, and subsequent TTEs showed eventual near resolution of the thrombus. Hence visualization of the IVC to rule out a thrombus must be performed in high-risk patients to prevent embolization into the right heart chambers. In such patients, performing the IVC compression maneuver may be associated with an increased risk and considered a relative contraindication based upon this report.

The IVC was not completely visualized, but this is a learning point moving forward and we will now take careful effort to visualize the IVC in all patients with proven DVT or undergoing a compression maneuver. Had a thrombus been seen, we would now consider aborting this maneuver to prevent the small potential risk of dislodgement and embolization. A risk versus benefit comparison should be weighed and discussed as a part of shared decision-making with the patient prior to performing this maneuver and procedure.

CONCLUSION

This case report highlights the importance of visualizing the IVC prior to performing the IVC compression maneuver during TEE for the diagnosis of a PFO in patients with extensive DVT and cardiovascular comorbidities. A risk versus benefit comparison of the procedure must be weighed during shared decision-making with patients to prevent possible formation and embolization of an FFRHT leading to associated potentially life-threatening complications.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical

Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

FUNDING STATEMENT

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DISCLOSURE STATEMENT

The authors report no conflict of interest.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2023.05.008.

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