Radiation Tracking in the Cardiac Cath Lab
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Cath Lab Digest

A Product, News and Clinical Update for the Cardiac Catheterization Laboratory Specialist

Cath Lab Spotlight

Saints Medical Center Cardiac Cath Lab is a TEAM is every sense of the word!
Dave Green, RN, BSN, EMT-P; James Waters, MD, Medical Director, Cardiac Cath Lab; Jennifer McCarthy, MSN, RN, Director of Cardiovascular Services; Rich Snyder, RN; Stacey Cayer, RN, ASN; Jeff Peters, RT(R), BA; Carla Hayden-Muse, RT(R); Rosany Rosario, CVT-RCIS; Erin Boutin, RN, ASN, Nurse Manager, Cardiac Cath Lab; Christine Stratton, RN.

Saints Medical Center

Contributions to this article were made by the entire staff in the cardiac cath lab and management team in Lowell, Massachusetts.

What is the size of your cath lab facility and number of staff members?

We currently have one cath lab and have a nurse manager, unit assistant, 5 registered nurses (RNs), 3 cardiovascular technologists (CVTs) and 2 radiologic technologists (RTs). The newest member has been here 3 months and our longest has worked here 4 years. We had a staff turnover when the lab went from diagnostic-only to interventional in 2004. Staff experience ranges from 1.5 years to 24 years.

What type of procedures are performed at your lab?

We perform elective heart caths, angioplasty, stent implants, pacemaker implants, trans-esophageal echos (TEE) and cardioversions.

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Clinical Update

Fibromuscular Dysplasia in Children and Adolescents

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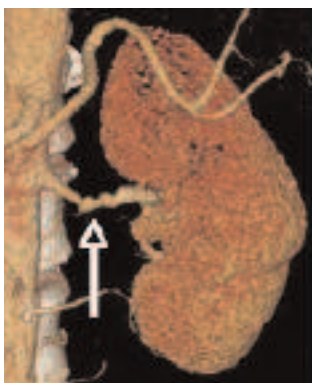
Introduction

FMD is a non-atherosclerotic, non-inflammatory disease that predominately affects the renal and carotid arteries, although it has been described in all vascular beds.[1] Approximately 60–75% of all FMD cases involve the renal rather then the carotid vessels; the renal predilection, however, may be greater in children. FMD more commonly affects women and younger individuals, though the sex distinction has not been proven in children. While its pathogenesis is not completely understood, hormonal, mechanical, and genetic factors, as well as mural ischemia, are thought to play a role. The natural history may be relatively benign, with progression occurring in only a minority of the patients. Depending on the arterial layer that is affected, the disease may be characterized by multifocal, tubular, or focal stenosis, which is a narrowing of the arterial vessel caused by a deposition of collagen that extends into the lumen. In addition to stenosis, vessels with FMD may develop weak points in the vessel wall that then can become aneurysmal. The most prevalent form of FMD identified in children and young adults is intimal fibroplasia, typified by long, irregular or smooth, focal stenosis.[1] Persons with FMD may be asymptomatic and only diagnosed at routine medical visits or even at work up for organ donation.



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Carotid Stenting: An update
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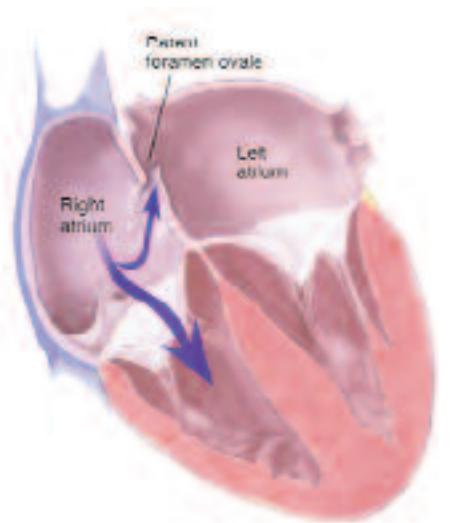
Case Report

Cerebral Vascular Accident Following a Pulmonary Embolism: Search for the Hidden Patent Foramen Ovale

Alex Harrison, MD[1], Ghalib Wahidi, MD[2], Morton Kern, MD[1]

Division of Cardiology, Dept. of Medicine, University of California, Irvine, California[1]; Dept. of Medicine, University of California, Irvine, California[2].



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Cerebral Vascular Accident Following a Pulmonary Embolism: Search for the Hidden Patent Foramen Ovale

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Introduction

We present a case of an 83-year-old man who developed a pulmonary embolism (PE) and then suffered a cerebral vascular accident (CVA) three days later as a result of an unsuspected patent foramen ovale (PFO). This example emphasizes the importance of considering and identifying a PFO as an unsuspected cause of CVA, especially in the setting of proven venous thromboembolic disease.

Case Report

An 83-year-old male with multiple medical problems was admitted to the hospital for intravenous antibiotic treatment of a severe lower extremity cellulitis. He suffered from longstanding diabetes mellitus and resultant chronic kidney disease with nephrotic range proteinuria and hypoalbuminemia. In addition, he was a former smoker and has mild chronic obstructive pulmonary disease (COPD) as well as heart failure with preserved left ventricular ejection fraction. On the third day of his hospitalization, he became progressively short of breath and hypoxic. Physical examination and routine testing did not readily identify a cause for his sudden dyspnea and hypoxia. Empiric treatment with diuretics and nebulized bronchodialators failed to improve his clinical situation. An echocardiogram was obtained and revealed a large right-to-left shunt across a PFO. The shunt was detected by color Doppler as well as agitated saline bubble study where the right-to-left flow was noted at rest without provocation or maneuvers. Figure 1 shows the agitated saline bubble study demonstrating opacification of the entire left atrium and left ventricle by bubbles shunting from the right atrium. His pulmonary arterial systolic pressure (PASP) was estimated at 44 mmHg, elevated from his prior baseline of 28 mmHg a few months prior. Left ventricular function was globally normal with an ejection fraction of 60-65%. His right heart function was also normal and without evidence of right ventricular hypertrophy or dilatation.

Cardiology consultation was requested to assist in the evaluation and management of the right-to-left shunting across the PFO. At this time, physical exam revealed an elderly, ill-appearing male who was afebrile. Blood pressure was 150/80, pulse was 96 and regular, respiratory rate was 22 and his oxygen saturation was 93% on 4 liters of oxygen via nasal cannula. Neck exam revealed mild jugular venous distention estimated at 9 cm of water and no carotid bruits. Cardiac examination demonstrated a regular rate and rhythm with no appreciable murmurs, rubs, or gallops. Pulmonary exam revealed tachypnea, faint scattered wheezing diffusely, but otherwise clear to auscultation and percussion bilaterally. His abdomen was obese, but soft and with normal bowel sounds and no appreciable hepatosplenomegally. He had cellulitis of his lower extremities with bilateral upper and lower extremity 3+ pitting edema without any evidence of clubbing or cyanosis of the nails. His peripheral pulses were intact. Laboratory data revealed a hemoglobin of 9.8 mg/dL, a creatinine of 3 mg/dL, blood urea nitrogen (BUN) of 58 mg/dL, an albumin of 1.7 mg/dL, and a b-type natriuretic peptide (BNP) of 133 pg/mL. His electrocardiogram is shown in Figure 2. The chest x-ray revealed prominent pulmonary vasculature, but was otherwise unremarkable.

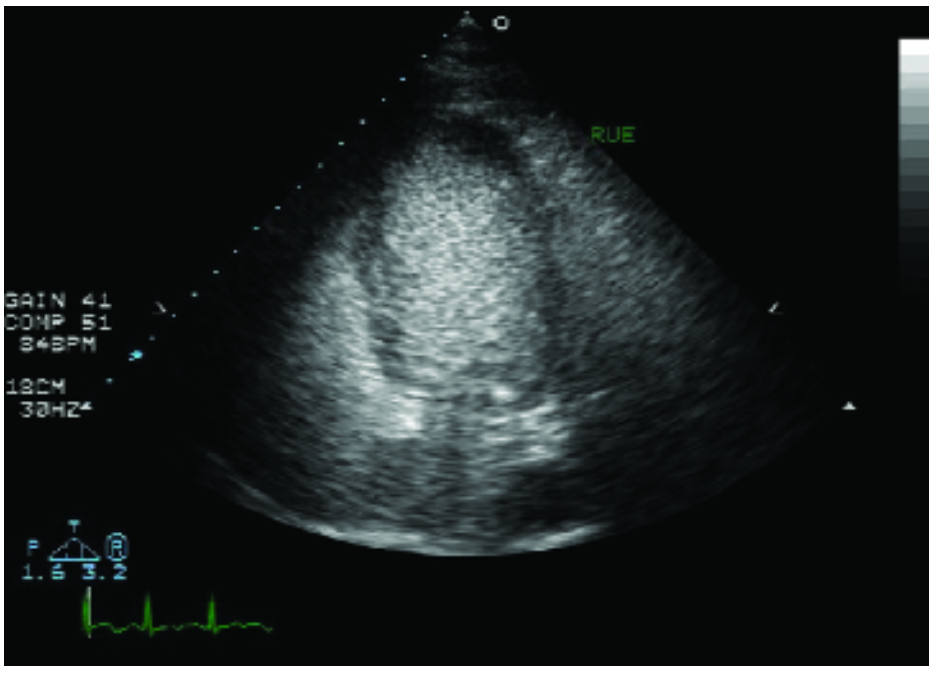

Figure 1. Apical four-chamber echocardiographic view demonstrating agitated saline bubble study where all four chambers are opacified, suggesting a right-to-left intracardiac shunt.

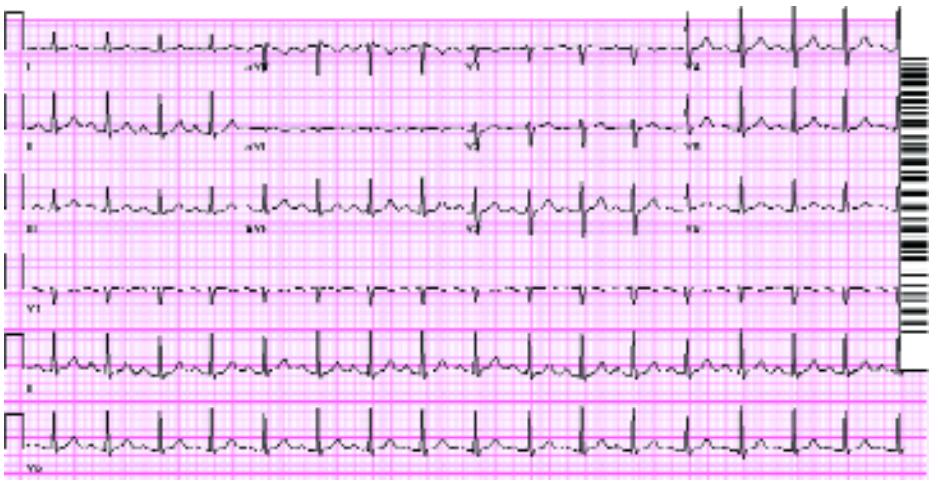

Figure 2. ECG showing sinus tachycardia but otherwise normal.

An acute pulmonary embolism was highest on the differential, given the clinical picture of acute onset of dyspnea and hypoxia in a patient who has been bedridden in the hospital. In addition, despite being on prophylactic subcutaneous heparin against deep venous thrombosis (DVT), his nephrotic syndrome puts him at even higher risk because of his renal losses of protein C and S. A lower-extremity duplex ultrasound to evaluate for lower extremity DVT did not reveal any identifiable venous thrombus but was a difficult exam due to significant edema. A contrast computed tomography (CT) pulmonary angiogram was deferred given the high risk for developing contrast-induced nephropathy. A ventilation perfusion (V/Q) scan demonstrated a large right lower lobe perfusion defect suggestive of a PE. Anticoagulation treatment with intravenous heparin was started.

The intra-atrial communication was determined to be a PFO. Prior echocardiograms were reviewed and did not have any findings of an intra-atrial communication or shunting by color Doppler when the patient had normal PASP. The PE was thought to increase pulmonary vascular resistance and thereby increase right atrial pressures, which opened a previously closed PFO and allowed for right-to-left shunting.

Unfortunately, 3 days after the initiation of anticoagulation, the patient was observed to have right upper extremity weakness. A magnetic resonance imaging (MRI) of the brain showed an acute right parietal lobe infarct with surrounding edema measuring approximately 3.3 x 3.8 cm. After several days, the patient's right upper extremity strength and respiratory status returned to his baseline. A repeat echocardiogram with agitated saline bubble study was obtained 10 days after the initiation of treatment with anticoagulation to reassess the degree of right-to-left

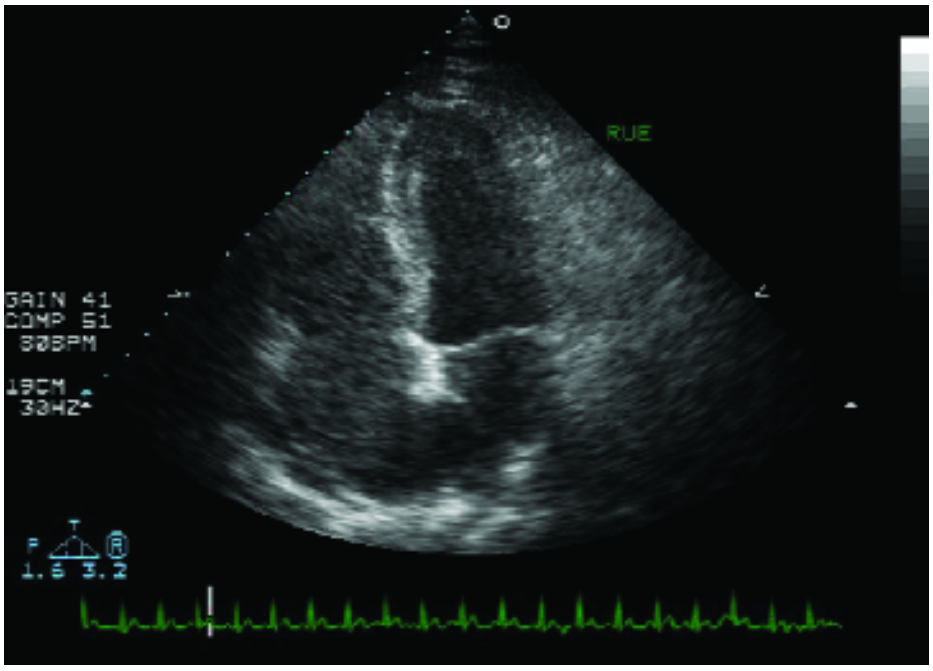

Figure 3. Apical four-chamber echocardiographic view demonstrating agitated saline bubble study where only the right-sided cardiac chambers are opacified, suggesting that the right-to-left intracardiac shunt is no longer present.

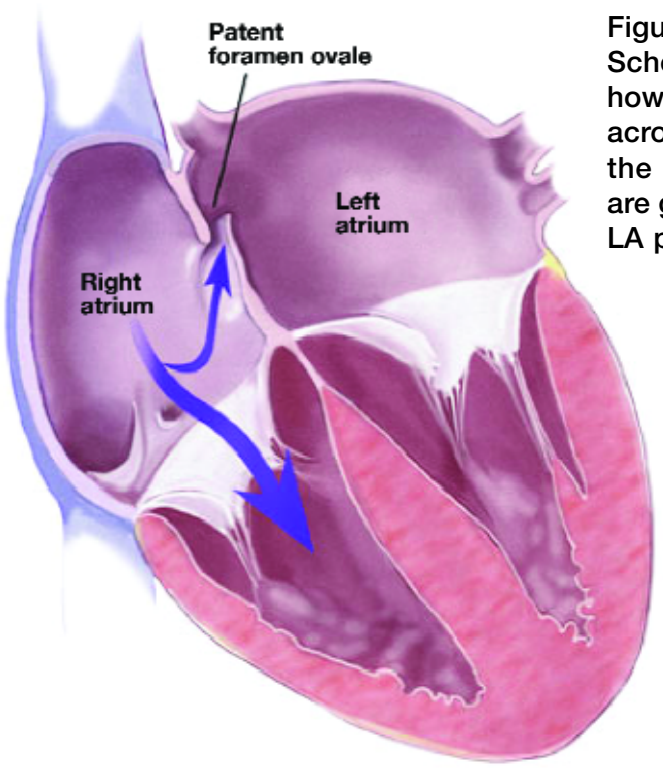

Figure 4. Schematic showing how blood can flow across a PFO when the RA pressures are greater than the LA pressures.

shunting and demonstrated no shunting at rest (Figure 3). Only a few bubbles could be visualized crossing into the left atrium with Valsalva maneuver. The patient is doing well six months later.

Discussion

This case demonstrates a hidden cause of CVA and how a PFO allows right-to-left shunting with elevated right-sided cardiac pressures, posing a significant risk for paradoxical embolism.

The mechanism of stroke in this patient is postulated as follows: the acutely elevated right-sided cardiac pressures caused by the PE resulted in the patient's PFO to open and allow for right-to-left shunting of blood. This shunting created a situation where a paradoxical embolus could occur where a venous thromboembolism could cross the intra-atrial communication and cause an arterial embolic phenomenon, *i.e.*, an ischemic stroke.

Acute PE causes increased pulmonary vascular resistance (PVR) that impedes right ventricular outflow. PVR is increased by the physical obstruction of the vascular bed with thrombus as well as vasoconstriction caused by inflammatory mediators and hypoxia. The correlation of pulmonary artery pressure (PAP) to thrombus size and/or burden is limited by the variable contribution of vasoconstriction among subjects. However, when obstruction of the vascular bed approaches 75%, the right ventricle must generate a systolic pressure in excess of 50 mmHg and a mean PAP approximating 40 mmHg to preserve pulmonary perfusion.[1]

A PFO is a congenital cardiac lesion that can persist into adulthood and is extremely common, with a prevalence estimated at 25–30% of the general adult population.[2,3] The septum primum and septum secundum fuse by age two in about 70 to 75 percent of children, with the remaining 25 to 30 percent having a PFO. This anatomy can lead to right-to-left shunting when the right atrial (RA) pressure is greater than the left atrial (LA) pressure (Figure 4). This can be demonstrated with maneuvers that increase RA pressures, such as the Valsalva maneuver, or in pathologic conditions that increase right-sided cardiac pressures. An important distinction is Eisenmenger's syndrome.

Table I.
Prevalence of a PFO in patients with cryptogenic stroke

Author	Year	Study Type	Number of Subjects (N)	PFO Prevalence	Comparison Group
Lechat[4]	1988	Case-control	60	40% vs. 10%	100 "normals"
Webster[5]	1988	Case-control	40	50% vs. 15% ($p < 0.001$)	40 age/sex matched
Di Tullio[6]	1992	Cross-sectional with nested case-control	146	48% vs. 4% < 55 yrs old ($p < 0.001$) 38% vs. 8% > 55 yrs old ($p < 0.001$)	Cryptogenic stroke vs. Identifiable origin of stroke
Overell[7]	2000	Meta-analysis of case-control studies	892	40% vs. 18% < 55 yrs old ($p < 0.001$)	721 controls
Mas[8]	2001	Prospective observational	581	46%	None
Homma[9]	2002	Prospective multicenter randomized controlled treatment trial	250	39%	None

Table II.
Prevalence of DVT in patients with a PFO and cryptogenic stroke

Author	Year	Study Type	Number of Subjects (N)	DVT Prevalence	Comparison Group
Stollberger[10]	1993	Cross-sectional observational	42	57%	Venography
Lethen[11]	1997	Cross-sectional observational	53	10%	Venography
Cramer[12]	2004	Cross-sectional observational	46	20%	MRI Venogram (MRV)

Individuals with Eisenmenger's syndrome initially have substantial left-to-right shunting — generally through an ASD or ventricular septal defect (VSD) — and, as a result, morphologic alterations occur in the small pulmonary arteries and arterioles leading to pulmonary hypertension and the resultant reversal of the intracardiac shunt, *i.e.*, right-to-left. These individuals demonstrate central cyanosis and digital clubbing on exam, ECG shows right atrial and ventricular enlargement with right axis deviation, and echocardiography shows signs of chronic right ventricular volume and pressure overload with ventricular hypertrophy and enlargement. Our patient had none of these findings.

Literature Review of PFO and DVT in Cryptogenic Stroke

On review of the literature, there are six large studies that report a prevalence of PFO in cryptogenic stroke ranging from 38 to 50% as compared to controls (4 to 18%) (Table I).[4-9] Furthermore, a few studies have assessed the prevalence of DVT in patients with a PFO and cryptogenic stroke and found 10 to 57% of patients to have demonstrable DVT (Table II).[10-12]

In the setting of a PFO, an increase in right-sided cardiac pressures — like that which occurs with an acute PE — can result in right-to-left shunting and pose a risk for a paradoxical embolism.

Summary

In summary, the evaluation of patients with cryptogenic stroke should include a search for the hidden PFO. And in the setting of a PFO, an increase in right-sided cardiac pressures — like that which occurs with an acute PE — can result in right-to-left shunting and pose a risk for a paradoxical embolism. ■

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Note: This article underwent double-blind peer review by members of the **Cath Lab Digest** *Editorial Board.*

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