Atrial septal aneurysm as a cause of abnormal left atrial flow: An echocardiographic study

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Atrial septal aneurysms, although rare, are being found with an ever increasing frequency through the technology of two-dimensional echocardiography. In this article, we report the identification of an atrial septal aneurysm and abnormal systolic flow toward the transducer and mitral valve by use of two-dimensional echocardiography with pulsed-wave Doppler capability.

(Key words: Atrial septal aneurysm, cardiac, defect, malformation, cardiovascular abnormality, echocardiography)

An atrial septal aneurysm is a thin, localized protrusion of the interatrial septum in the region of the fossa ovalis.¹ It occurs at an incidence of 0.2 to 1 per 100 population.^{1,2} The protrusion is often fenestrated^{2,3} and can appear as a primary defect (ie, not associated with congenital heart disease) or a secondary defect (ie, associated with congenital heart disease and the presence of a significant pressure gradient between the two atria). An atrial septal aneurysm is present and defined when there is a bulging of the interatrial septum protruding more than 6 mm into the right or left atrium (or both) during the cardiac cycle.⁴

It has been postulated that an atrial septal aneurysm is caused by a congenital disorder of connective tissue. The aneurysm is absent in childhood, the malformation becoming apparent over time. The fossa ovalis membrane and the mitral, tricuspid, aortic, and pulmonary apparatus are composed of endocardial tis-

sue, and an inherent deficiency that occurs in one might be expected to occur in the others.⁵ An atrial septal aneurysm has been associated with multiple cardiovascular abnormalities (Table).^{1-4,6-12} Most atrial septal aneurysms are thought to be clinically silent, and they are detected only incidentally.¹⁻⁴

Report of case

A 39-year-old woman was evaluated in the echocardiography laboratory because of a complaint of palpitations for the previous 9 months. Her symptoms were exacerbated by caffeine, anxiety, and, occasionally, activity. Her medical and surgical histories were unremarkable. She did not smoke. Her physician had prescribed propranolol hydrochloride on an as-needed basis for palpitations. Results of her physical examination were essentially normal.

The patient's electrocardiogram demonstrated a normal sinus rhythm, incomplete right bundle-branch block, anterior axis rotation, and nonspecific ST-T-wave changes. On the chest roentgenogram, the lung fields and the cardiac silhouette appeared to be within normal limits.

A two-dimensional echocardiogram and Doppler test were performed with a commercially available echocardiograph (Hewlett Packard, Andover, Mass; model 77020AC) and a 2.5-MHz phased-array transducer system with pulsed-wave Doppler capability. This study demonstrated normal chamber size. There was moderate thickening of the anterior mitral leaflet with prolapse and an atrial septal aneurysm involving the entire septum. The aneurysm protruded into the left atrium approximately 1.1 cm during middle-to-late systole and into the right atrium approximately 1.2 cm during early-to-middle diastole (Figs 1 and 2). This motion, in association with a normal right and left atrial pressure gradient, was described by Roberts.⁶ Technical difficulties made it impossible to assess changes in atrial septal motion during inspiration in our patient.

Contrast echocardiography was performed by use of preserved saline solution. The microcavitations

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Table Associated Cardiovascular and Systemic Abnormalities		
Abnormality	Incidence of patients with associated atrial septal defect	Reference(s)
Embolitic phenomena:		
Cerebrovascular accidents	Up to 28%	1, 3, 4
Pulmonary embolism	Unknown	6, 7
Transient ischemic attacks	Up to 25%	8
Mitral valve prolapse	Up to 48%	2, 9, 10
Tricuspid valve prolapse	Unknown	2, 9
Atrial septal defect	Up to 30%	4
Arrhythmias/palpitations	Unknown	11
Mitral, tricuspid, and pulmonary venous obstruction	Unknown	7, 10
Coronary artery disease	Unknown	10
Cardiomyopathies	Unknown	10
Systemic hypertension	Unknown	10
Aortic stenosis	Unknown	7
Congenital heart disease	Unknown	2
Marfan's syndrome	Unknown	12

failed to demonstrate right-to-left or left-to-right shunting by either direct crossover of the microbubbles or negative contrast effect seen with left-to-right shunting. Results of Doppler examination of the atrial septum in the subcostal and apical four-chamber views failed to demonstrate any evidence of right-to-left or left-to-right shunting. This is in contradistinction to the findings of a study by Belkin and associates,³ who observed that all 13 of their patients with an atrial septal aneurysm had evidence of right-to-left shunting at the atrial level.

The pulsed-wave Doppler signal positioned in the middle aspect of the left atrium demonstrated abnormal flow in systole toward the transducer and mitral valve (Fig 3). To our knowledge, this abnormality has not been described before.

Discussion

An atrial septal aneurysm can be classified into any one of three major groups, as described by Hanley and associates,¹⁵ depending on the amount of the atrial septum involved

and on the maximal extent of phasic oscillations of the aneurysmal membrane during the cardiorespiratory cycle. Type 1 atrial septal aneurysm involves the fossa ovalis with maximal oscillations occurring toward the right atrium. Type 2 atrial septal aneurysm involves the fossa ovalis, with maximal oscillations occurring toward the left atrium. Type 3 atrial septal aneurysm, the category to which our patient's aneurysm belongs, involves the entire atrial septum. This defect has been observed previously⁷ in patients with complex congenital malformations of the hypoplastic right ventricular type, all of whom had associated atrial septal defects. Our patient had neither a hypoplastic right ventricle nor an atrial septal defect.

The atrial septal motion, as described in our case presentation, has been described previously and was found to be associated with normal atrial pressure gradients.⁵ The leftward

Figure 1. *Apical four chamber view of the heart in early diastole.*

Figure 2. *Apical four chamber view of the heart in mid-diastole.*

motion of the interatrial septum in systole was postulated to occur because left atrial systole ends before right atrial systole. This pattern is consistent with the normal physiologic variations of blood flow to the atrium.⁸ The findings noted with pulsed-wave Doppler examination can be explained by the fact that the movement of the atrial septum toward the left atrium in systole sets up turbulent flow (eddy currents) toward the transducer and mitral valve.

Conclusion

An atrial septal aneurysm is a rare localized phenomenon of the interatrial septum in the region of the fossa ovalis. It is a disorder of connective tissue⁴ and may be associated with other endocardiac defects. It can be associated with multiple clinical presentations and should always be considered in their differential diagnosis. In the case described, a 39-year-old woman had an atrial septal aneurysm that in-

Figure 3. Pulsed-wave Doppler signal demonstrating abnormal systolic flow.

volved the entire septum without evidence of an atrial septal defect or a hypoplastic right ventricle. At the time this article was written, the abnormal systolic flow demonstrated in the left atrium with pulsed-wave Doppler had not been previously reported.

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see **Warnings**), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol clearance may occur with combined use. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-

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lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°, 2°, 3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, increased urination, spotty menstruation, impotence.

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