Anesthesiology 2010; 113:496–8

Traumatic Atrial Septal Defect and Coronary Sinus to Left Atrium Fistula from Coronary Sinus Pacing Lead Removal

Jiapeng Huang, M.D., Ph.D., F.A.S.E.,*
Sebastian Pagni, M.D.,†
Michael J. Bouvette, M.D.,‡
Jing Zhou, M.S.N.A., C.R.N.A.,§
Samuel Morgos, M.D.,* Kishin Dodwani, M.D.*

CARDIAC resynchronization therapy with the placement of a left ventricular pacing electrode in the coronary sinus has been shown to be an effective treatment in selected patients with symptomatic left ventricular dysfunction.[1] Coronary sinus leads can be removed with relative ease because of their small diameter and relatively poor fixation mechanisms.[2,3] However, removal of coronary sinus electrodes could potentially rupture or perforate the coronary sinus, causing tamponade, hemodynamic instability, and death. We report a rare case of atrial septal defect (ASD) and coronary sinus to left atrium fistula without tamponade from coronary sinus lead removal.

CASE REPORTS

An 82-yr-old woman presented for removal of an infected biventricular automatic implantable cardioverter defibrillator/pacemaker generator and leads. Her medical history included coronary artery disease, complete atrioventricular block, hypertension, and dyslipidemia. She had a marked decrease in left ventricular function after right ventricular pacing, possibly because of ventricular dyssychronous contraction, and was treated with biventricular pacing. Her congestive heart failure symptoms significantly improved, and left ventricular ejection fraction increased to 55%; despite that, she needed several lead revision procedures because of persistent diaphragmatic stimulation. A Medtronic model 4195 lead (Medtronic, Minneapolis, MN) had been inserted in her anterolateral cardiac vein 5 months before, and a Guidant model EASYTRAK lead (Boston Scientific, Natick, MA) had been placed in her posterolateral cardiac vein 2 months before. Both leads remained in her coronary sinus.

Anticipating difficulties with lead removal because of previous revisions, we obtained femoral arterial and venous accesses before anesthetic induction with etomidate and rocuronium (Hospira Inc., Lake Forest, IL). General anesthesia was maintained with narcotics and isoflurane (Hospira Inc.). Intraoperative transesophageal echocardiography (TEE) revealed left ventricular ejection fraction of 45% with moderate tricuspid regurgitation and a small pericardial effusion. Slight advancement of the probe from the midesophageal four-chamber view revealed two left ventricular pacing leads in the coronary sinus. The generator, right atrial lead, right ventricular lead, and the Guidant EASYTRAK lead were removed without difficulty. Her blood pressure was 119/89 mmHg with 100% oxygen saturation.

The fixation mechanism of the Medtronic 4195 lead could not be unlocked completely. Traction and countertraction through a Teflon sheath was unsuccessful. During this process, her systolic blood pressure suddenly decreased to 60 mmHg, and the patient desaturated to a low of 90% on pulse oximetry. Suspecting either a hemopericardium or a hemothorax, we administered albumin and 100 μg phenylephrine, which improved her blood pressure and oxygen saturation. However, findings consistent with a clinical diagnosis of cardiac tamponade or pleural effusion were not found after examination of the pericardial and pleural spaces with TEE. Surprisingly, the anteflexed four-chamber view revealed a new large ASD near the fossa ovalis (fig. 1). The pacing wire appeared to be in the left atrium. A bubble study with agitated saline showed passage of bubbles into the left atrium. Color flow Doppler ultrasonography demonstrated a large bidirectional flow jet between the atria (fig. 2; see video, showing a large ASD, Supplemental Digital Content 1, http://links.lww.com/ALN/A596).

With a slight advancement of the TEE probe, a large fistula between the ostium of coronary sinus and the left atrium was evident. A piece of flail left atrial tissue covered the opening of this fistula. The flail tissue swayed toward the right atrium during systole and opened into the left atrium

* Assistant Clinical Professor, ‡ Associate Clinical Professor, Department of Anesthesia, Jewish Hospital & St. Mary's Healthcare, Louisville, Kentucky, and Department of Anesthesiology and Perioperative Medicine, University of Louisville, Louisville, Kentucky. † Assistant Professor, Division of Thoracic and Cardiovascular Surgery, Department of Surgery, § Staff Nurse Anesthetist, Department of Anesthesiology and Perioperative Medicine, University of Louisville.

Received from the Department of Anesthesia, Jewish Hospital, Louisville, Kentucky. Submitted for publication February 8, 2010. Accepted for publication March 9, 2010. Support was provided solely from institutional and/or departmental sources. Drs. Huang and Pagni contributed equally to this article.

Address correspondence to Dr. Huang: Department of Anesthesiology and Perioperative Medicine, University of Louisville, 201 Abraham Flexner Way, Suite 1200, Louisville, Kentucky 40202. jiapenghuang@yahoo.com.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are available in both the HTML and PDF versions of this article. Links to the digital files are provided in the HTML text of this article on the Journal's Web site (www.anesthesiology.org).

during diastole. The midesophageal two-chamber view further confirmed the new continuity of the coronary sinus and the left atrium. The coronary sinus appeared dilated. A diagnosis of coronary sinus tear with resultant ASD and fistula was made, and emergent repair was planned.

After a standard sternotomy and institution of cardiopulmonary bypass, both atria were explored. A large tear (approximately 3 cm), involving the ostium of the coronary sinus with a linear cut in both the interatrial septum and the posterior left atrial wall with communications between the coronary sinus and the left and right atria, was identified. The coronary sinus was also found to be detached from the mitral valve annulus. The retained lead was transected, and the coronary sinus was reattached to the mitral valve annulus with a running suture. A patch of bovine pericardium was used to close the interatrial septal defect. No residual ASD or fistula was present on postoperative TEE. The tricuspid regurgitation remained unchanged. The patient was weaned from cardiopulmonary bypass easily and extubated on postoperative day 1. She recovered uneventfully and was discharged on postoperative day 12.

Discussion

The coronary sinus is a 3-cm-long thin structure lying within the posterior atrioventricular groove. Functionally, it drains approximately 85% of coronary venous blood and returns it to the heart. It is joined by the great cardiac vein, the lower posterior vein, the middle cardiac vein, and the small cardiac vein. It opens into the posterior lateral wall of the right atrium, near the septal leaflet of the tricuspid valve. It is not only frequently used for the administration of retrograde cardioplegia but also used as an approach for the implantation of biventricular pacing wires and for percutaneous prosthetic mitral valve repair. The coronary sinus is supported by the fibrous trigone between the annulus fibrosus of the tricuspid and mitral valves, and the main coronary sinus vein is often insulated by the adipose tissue. The fatty and fibrous support might allow sequestration of a hematoma in the event of coronary sinus perforation or dissection. A long-axis TEE view of the coronary sinus can be obtained by either

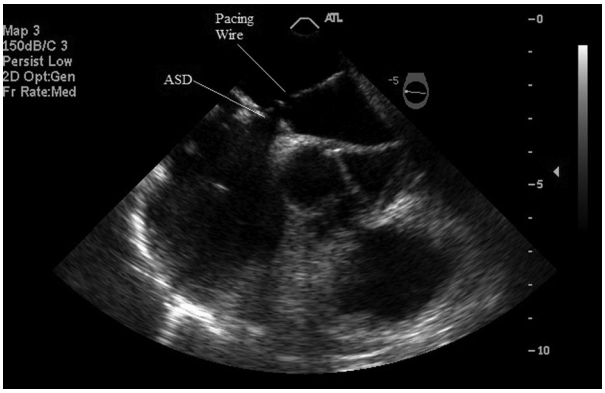

Fig. 1. The anteflexed four-chamber view revealed a new large atrial septal defect (ASD) near the fossa ovalis. A pacing wire was seen in the left atrium.

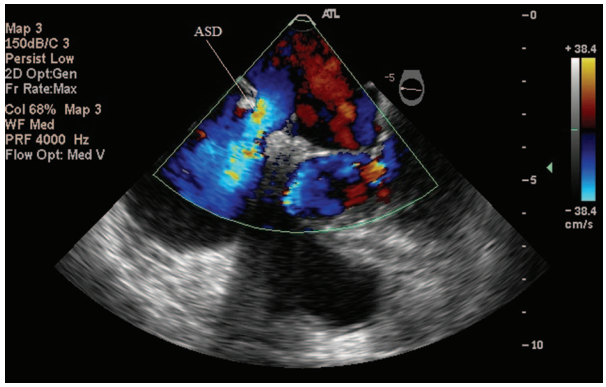

Fig. 2. Color flow Doppler demonstrated flows between the atria. ASD = atrial septal defect.

advancing the probe slightly from the midesophageal four-chamber view coupled with slight retrograde flexion or in a modified midesophageal bicaval view. The short-axis view of the coronary sinus is achieved in the midesophageal position at an array angle of 90°.

Experience with coronary sinus lead removal is limited and warrants surgical backup.[4] The availability of bypass is highly recommended. Femoral arterial and venous accesses ensure an easier transition to bypass if necessary. If the lead does not retract to at least the coronary sinus ostium and out of the branch, a cardiothoracic surgeon should be notified and prepared. The chest preparation should be sufficient to allow for emergent thoracotomy and pericardiocentesis if necessary. Critical to the management of these types of injuries is a rapid and accurate diagnosis. Often these cases are falsely assumed to be relatively simple and low risk. As such, these patients may frequently be inadequately monitored. As illustrated by this case, proper preparation of the patient, including obtaining appropriate access with TEE monitoring, enabled a timely diagnosis and definitive treatment. This approach would be expected to decrease morbidity and mortality resulting from a serious complication, such as the one described here.

In this case, a possible mechanism for injury might have included partial endothelialization and fixation of the pacing lead to the interatrial septum near the coronary sinus ostium. Excessive tension on the lead ruptured the nearby interatrial septum and the posterior left atrial wall, causing ASD and fistula (figs. 1 and 2). The flow from the left atrium to coronary sinus dilated the coronary sinus. Hemodynamic instability during pacemaker lead removal usually triggers a search for cardiac rupture and tamponade. In our case, hypotension and desaturation were induced purely from intracardiac communications, namely the fistula and ASD. If a comprehensive examination had not been performed, this problem could not have been identified. Moderate tricuspid regurgitation might have caused increased right atrial pressure and thus a bidirectional flow through the ASD. After repair of coronary sinus-related injuries, detailed examinations of mitral and tricuspid valves are mandatory because of its close proximity to the annulus of both valves. Also, the repair itself may damage the cardiac con-

duction system, and a temporary pacemaker and defibrillator should be readily available.

We reported a rare complication caused by coronary sinus electrode removal diagnosed and managed with the help of TEE. The coronary sinus and adjacent structures deserve careful examinations after any coronary sinus intervention.

References

1. Abraham WT, Fisher WG, Smith AL, Delurgio DB, Leon AR, Loh E, Kocovic DZ, Packer M, Clavell AL, Hayes DL, Ellestad M, Trupp RJ, Underwood J, Pickering F, Truex C, McAtee P, Messenger J: Cardiac resynchronization in chronic heart failure. N Engl J Med 2002; 346:1845–53
2. De Martino G, Orazi S, Bisignani G, Toscano S, Messano L, Parisi Q, Santamaria M, Pelargonio G, Dello Russo A, Bellocci F, Zecchi P, Crea F: Safety and feasibility of coronary sinus left ventricular leads extraction: A preliminary report. Interv Card Electrophysiol 2005; 13:35–8
3. Kutalek SP: Pacemaker and defibrillator lead extraction. Curr Opin Cardiol 2004; 19:19–22
4. Burke MC: Percutaneous extraction of coronary sinus vein and branch leads. Heart Rhythm 2008; 5:491–5