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ST-segment Elevation during Cardiac Electrophysiologic Surgery

CHARLES W. WHITTEN, M.D.,* TERRY W. LATSON, M.D.,† RICHARD P. COCHRAN, M.D.,‡
JEFFREY C. ELMORE, M.D.,* JAMES D. GRIFFIN, M.D.§

Coronary artery spasm leading to ST-segment elevation has not been reported as a complication of cardiac electrophysiologic surgery. We report such an instance that occurred during epicardial mapping and atrioventricular (AV) nodal cryomodification in the course of normothermic cardiopulmonary bypass (CPB) in an otherwise healthy 14-yr-old girl.

CASE REPORT

A 14-yr-old, morbidly obese girl was referred to our institution for evaluation of possible Wolff-Parkinson-White syndrome.

She was in a normal state of health until 1 yr prior to admission, when she experienced the sudden onset of palpitations and light-headedness while walking. She had no associated syncopal, seizure, or focal neurologic symptoms with this episode, although she did have "burning" anterior chest pain without radiation. The episode lasted 1.5 h and resolved spontaneously. She experienced a similar episode, which lasted 2 h, 6 months prior to admission. Three weeks prior to her admission she experienced a third episode, this time associated with shortness of breath and nausea. This episode lasted until verapamil was administered intravenously (iv) in her local emergency room to resolve a supraventricular tachycardia. Verapamil 80 mg orally three times per day was prescribed, and she had no recurrences of supraventricular tachycardia prior to her admission.

Upon admission, vital signs and physical examination were remarkable only for a 1/6 systolic ejection murmur at the left lower sternal border. Her admission ECG showed a normal sinus rhythm at 75 beats per min, an axis of 20°, a PR interval of 0.12 s, a QRS interval of 0.08 s, and a QT interval of 0.37 s, with inverted P waves in lead III. Admission chest x-ray and laboratory were unremarkable.

Electrophysiology studies revealed inducible atrial fibrillation, dual AV nodal pathways, and inducible preexcitation with reciprocating tachycardia *via* a Mahaim fiber pathway.

The patient was scheduled for epicardial mapping and cryomodification of her AV node. After arrival in the operating room, a five-lead ECG was placed and a radial arterial catheter inserted. Anesthesia was induced with thiopental, succinylcholine, midazolam, and sufentanil, and the trachea was intubated without complications. After induction, two catheters were inserted into the right internal jugular vein. Anesthesia was maintained with vecuronium, sufentanil, and midazolam in an air/oxygen mixture.

The patient's prebypass course was uneventful, and warm CPB was instituted 35 min after the beginning of surgery. No aortic cross-clamp was required during the procedure. One hour and 42 min after the institution of CPB, and as open endocardial cryomodification *via* the right atrium was nearing completion, an elevation of the ST segment of greater than 5 mm was noted in lead II. Smaller elevations also were noted in leads III and aVF, with reciprocal anterior changes. The patient was still on total bypass at this point, with a mean perfusion pressure of 50–60 mmHg and a heart rate of 50 beats per min. After careful search for visual evidence of air along the entire course of the right coronary artery, no gross evidence of air, kinking, or obstruction was discovered by the surgeon. The ST-segment changes persisted even when the perfusion pressure was increased to 95 mmHg with phenylephrine.

An iv infusion of nitroglycerin was begun, and the rate of infusion gradually increased over several minutes to 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, with persistence of the ST-segment elevation. Nifedipine 10 mg was administered intranasally, followed by another administration of the same dose 10 min later. Due to concern about unresponsive coronary artery spasm, the surgeon then placed papaverine 20 mg topically along the course of the right coronary artery. Within 2 min after placement of papaverine, the ST-segment elevation began to resolve, and complete normalization took place over the next 5 min.

One hour after resolution of the initial ST-segment elevation, a greater than 5-mm ST-segment elevation was again noted in leads II, III, and aVF, with reciprocal anterior changes. The perfusion pressure was again increased with phenylephrine. The infusion of iv nitroglycerin was continued at 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Twenty milligrams of nifedipine was administered intranasally, and again, papaverine 20 mg was placed topically along the course of the right coronary artery. An additional 20 mg nifedipine was administered intranasally 10 min later. With continuing elevation of the ST segments, papaverine 20 mg was introduced into the coronary circulation *via* a retrograde cardioplegia cannula (15-Fr Gundry retrograde coronary sinus perfusion (RCSP) cannula with cuff, DLP Inc., Grand Rapids, MI) placed in the coronary sinus. Again, within 2 min after papaverine administration, the ST segments began to resolve. The total duration of ST segment elevation during this episode, from onset until complete resolution, was 24 min.

After 3 h and 58 min of warm CPB, the patient was separated from CPB while receiving dobutamine 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, nitroglycerin 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and lidocaine 2 mg/min. She tolerated the remainder of the surgical procedure without incident and was transferred to the intensive care unit while continuing to receive dobutamine 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$, nitroglycerin 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and lidocaine 2 $mg \cdot min^{-1}$. After arrival in the intensive care unit, the patient was given nifedipine 10 mg sublingually in an effort to prevent additional coronary spasm. Her immediate postoperative ECG showed no evidence of acute myocardial injury, and serial ECGs over the next 3 days showed no evidence of ischemic myocardial injury.

* Assistant Professor, Department of Anesthesiology.
† Associate Professor, Department of Anesthesiology.
‡ Assistant Professor, Department of Surgery.
§ Resident, Department of Anesthesiology.

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Address reprint requests to Dr. Whitten: Department of Anesthesiology, The University of Texas Southwestern Medical Center–Dallas, 5323 Harry Hines Boulevard, Dallas, Texas 75235-8894.

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Discussion

With the increasing interest in the diagnosis and surgical management of cardiac arrhythmias, a new group of patients are undergoing CPB. These patients generally are young, otherwise healthy, and have no other underlying medical problems except for conduction abnormalities; generally, they are unlikely to have significant coronary atherosclerosis.

Acute ST-segment elevation during normothermic CPB with a spontaneously beating heart can occur as a result of: 1) air and/or particulate emboli in the coronary circulation; 2) coronary artery thrombosis; 3) coronary artery spasm; 4) obstruction of a coronary artery (*e.g.*, due to a poorly positioned aortic cannula or aortic dissection). Several observations support our belief that the episodes described in this case report were caused probably by spasm of an epicardial coronary artery: 1) ECG evidence of acute transmural ischemia developed recurrently in a myocardial zone presumably perfused by a coronary artery without atherosclerotic lesions; 2) the ST segment elevation was severe, acute in onset, transient, and recurrent; 3) the pattern of the ST-segment elevation was essentially identical in both situations; and 4) spasm of the right coronary artery occurs more frequently than does spasm of the vessels comprising the left system.[1]

The definitive diagnosis of coronary artery spasm requires direct evidence from coronary arteriography of spasm of the artery supplying blood to the area of myocardium corresponding to the area indicated to be ischemic by the ST-segment elevation.

It is possible that recurrent air embolization is responsible for the sequence of ST-segment events we observed. Air embolization also most frequently occurs to the right coronary artery. However, we believe that air embolization was not the most likely diagnosis, for the following reasons: 1) air was not visualized by the surgeons; 2) no left-sided chambers were opened, and no septal defects were present to serve as possible conduits for air embolization; 3) the episodes did not respond to increases in perfusion pressure (the primary therapy for air or particulate embolization of the coronary circulation); 4) the episodes recurred with identical manifestations; and 5) the episodes responded in a timely fashion to topical and coronary sinus papaverine administration.

There are four major types of coronary arterial spasm: 1) iatrogenically induced spasm; 2) coronary spasm superimposed on well-defined coronary obstruction; 3) spasm secondary to nitrate withdrawal; and 4) spontaneously occurring spasm. Causes underlying the pathophysiology of coronary artery spasm include abnormalities in autonomic control of vascular smooth muscle and excessive responsiveness of smooth muscle to local metabolites or circulating vasoactive substances. Maseri *et al.*[2] recently argued that the development of segmental occlusive spasm is caused by a local hyperreactivity of the arterial vessel wall in response to a variety of stimuli acting on different receptors rather than by a dysfunction of the autonomic nervous system. Not all coronary artery spasm leads to transmural myocardial ischemia with subsequent ST segment elevation. If coronary spasm leads to incomplete vessel occlusion, or if collateral flow is sufficient, ischemia may be limited to the more vulnerable subendocardium, and ST-segment depression may result.

Reports of perioperative coronary artery spasm are common.[3-9] Several factors in the perioperative period may interact to provoke spasm in a susceptible patient (*e.g.*, acid–base abnormalities, imbalance of the adrenergic nervous system, acute electrolyte imbalances, and imbalance of vasoactive substances not under neural control). In the case presented here, additional factors may have included mechanical trauma secondary to the procedure and/or the direct application of a cryoablation probe at −60° C along the tendon of Todaro and in the area of the tricuspid valve. Such trauma and/or hypothermia of tissues in proximity to the right coronary artery may have caused neural and/or metabolic abnormalities instrumental in eliciting the episodes of spasm.

Cohen and colleagues[10] have provided a scheme for the management of coronary artery spasm in patients undergoing CPB. They suggested: 1) direct injection of 1 mg of nitroglycerin into each coronary artery graft or into the affected coronary artery; 2) administration of nifedipine 30 mg through a nasogastric tube immediately and then every 6 h; 3) a peripheral intravenous infusion of nitroglycerin 60–100 $\mu g \cdot min^{-1}$; 4) substitution of isosorbide dinitrate (20–40 mg sublingually every 4 h) for iv nitroglycerin 12–24 h after postoperative stability has been obtained; and 5) maintenance on long-term dosages of isosorbide dinitrate 10–20 mg every 4 h or nitropaste, plus nifedipine 30 mg every 6 h.[10]

Both nitroglycerin and calcium channel blockers, given either iv or orally, may reverse coronary artery spasm. However, they are not always effective by either of these routes. Both drugs also have been administered by intracoronary injection to treat more resistant cases of coronary artery spasm. Intracoronary papaverine has also been shown to reverse coronary artery spasm. Papaverine, an opiate derivative, has potent direct arterial and smooth muscle relaxing properties.[11] In the absence of epicardial coronary obstruction, it has been shown to cause maximal increases in coronary blood flow at intracoronary doses of 8–12 mg[12] and to increase flow to all layers of the myocardium.[13] Intracoronary papaverine generally causes very little change in systemic arterial pressure, heart rate, or left atrial pressure.[13] Although it is not certain that

resolution of the ST-segment changes in this patient was due to papaverine (*versus* a delayed response to nifedipine), in both instances the ST-segment resolution began within 2 min after papaverine administration.

Our review of the literature suggests that this is the first reported case of the use of papaverine administered *via* a retrograde cardioplegia cannula for the treatment of coronary artery spasm in a patient undergoing CPB. We believe that this mode of administration has potential advantages. These include the ability to perfuse the coronary circulation without the application of an aortic cross clamp and the absence of direct damage to the coronary circulation, which might occur with direct injection into a coronary artery. In theory, there should be minimal arterial inflow into the affected arterial system distal to the area of spasm. In the absence of arterial inflow to compete with retrograde distribution of medication, the papaverine administered *via* the coronary sinus should be able to reach the affected arterial bed (similar to when the aorta is cross-clamped with administration of retrograde cardioplegia).[14] Although the efficacy of this approach cannot be proven on the basis of this single case, we believe this technique merits consideration.

In summary, we report a case of recurrent ST-segment elevation occurring during electrophysiologic surgery in a patient without known coronary artery disease. We believe that coronary artery spasm was the most likely etiology for these ST-segment changes. Local hypothermia or mechanical trauma in the area of the right coronary artery may have been instrumental in provoking these episodes of spasm. Management included administration of calcium channel blockers, iv nitroglycerin, the direct administration of papaverine upon the affected coronary artery, and retrograde perfusion of the coronary circulation with papaverine *via* a retrograde cardioplegia cannula.

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