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Temporary Right Coronary Artery Flow Disruption during Instrumented Correction of the Spine

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SEVERE hemodynamic deterioration during posterior spine fusion may occur as result of uncontrolled bleeding, venous air embolism, direct compression of the heart and great vessels, or neurologic damage due to compromised spinal cord perfusion.¹⁻³ Patients with Marfan's syndrome present not infrequently for spine surgery; these patients have an increased perioperative morbidity and mortality rate resulting from pulmonary and cardiovascular complications.^{4,5} The anesthetic technique in these patients should aim to decrease myocardial contractility and avoid sudden increases in blood pressure, to minimize the risk for aortic dissection or rupture.⁴ We describe acute myocardial ischemia and subsequently learned results due to right coronary artery obstruction during surgical correction of thoracolumbar junction kyphosis in a patient with Marfan's syndrome and pectus excavatum.

Case Report

A 14-yr-old girl (weight, 65 kg; height, 194 cm) with Marfan's syndrome and pectus excavatum accompanied by progressive thoracolumbar kyphosis (T9-L2, 80°) and thoracic hypokyphosis was admitted for corrective surgery. Due to aortic dilatation, the patient was treated with 50 mg atenolol daily. Anesthesia was induced using 250 mg thiopental, 150 µg fentanyl, and 100 mg succinylcholine, and was maintained with isoflurane, nitrous oxide, and fentanyl. Mon-

itoring consisted of an electrocardiograph (modified lead 2), arterial line, pulse oximetry, capnography, and urinary and pulmonary artery catheters. Deliberate hypotension (mean blood pressure, 50-60 mmHg) was induced using sodium nitroprusside. The first stage of correction consisted of an anterior release and fusion. The second stage consisted of an instrumented posterior spinal fusion from T5 to L3, using the Cotrel-Dubousset system. To correct the thoracolumbar kyphosis, contoured rods were connected to the proximal thoracic hook claws (T5-T8) and then forced manually to the distal lumbar screws (L2-L3). After manual deformity correction by fixing the rods to the screws, S-T segment elevation with negative T waves appeared, followed by multiple premature heart beats, with the sinus rhythm changing to nodal within minutes followed by multiple premature beats and runs of ventricular tachycardia. Although sodium nitroprusside and isoflurane were discontinued, the blood pressure decreased to 50/33 mmHg, and the pulmonary artery pressure was 17/14 mmHg. There was no hemodynamic improvement after administering phenylephrine and lidocaine, followed by volume loading. Only after the rods were completely released from the distal screws did the sinus rhythm return with a blood pressure of 120/60 mmHg. Blood gases, electrolytes, and hemoglobin at this time were within normal limits. A wake-up test showed that the patient could move all four extremities in response to commands. After the patient was given Haemaccel (Behringwerke AG, D-35001 Marburg, Germany) and an autologous unit of blood, a repeated attempt to fix the rods to the distal screws was made. The hemodynamic response was very similar to the previous one. Surgery was completed with a less-than-desirable correction of the thoracolumbar kyphosis. The patient emerged from anesthesia and was extubated immediately after surgery. An electrocardiogram performed in the recovery room did not show any changes and the creatine phosphokinase-MB enzyme was normal.

On the sixth postoperative day, transthoracic echocardiography was performed. The cardiac chambers were normal, as was left ventricular contractility. The aorta was dilated (28 mm diameter) and displaced to the right. The right coronary artery was located between the depressed sternum (pectus excavatum) and the rightward displaced, dilated aorta. Right coronary artery flow was detected by color flow mapping. A flow velocity curve was obtained using the pulsed Doppler technique, with the sample volume placed in the right coronary artery. With the transducer maintained in a stable position and the flow velocity of the right coronary artery recorded, pressure was applied to the sternum. Within seconds rapid decreases in flow velocity amplitude and heart rate were observed (fig. 1). After 4 or 5 s, flow could no longer be detected in the coronary artery. This was immediately reversed by relieving the external thoracic pressure. The maneuver was repeated three times with the same result.

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CASE REPORTS

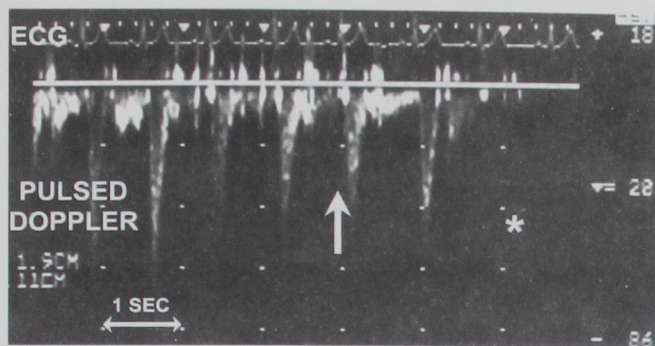


Fig. 1. Doppler velocity signal from the right coronary artery and electrocardiography. The arrow indicates the beginning of pressure on the sternum, and asterisk indicates disappearance of flow signal. Note the bradycardia that accompanies the disappearing flow velocity signal at the seventh cardiac cycle.

Discussion

Unexpected and severe hemodynamic deterioration during instrumental correction of a spinal deformity may result from venous air embolism, compromised venous return, or impaired spinal cord perfusion.¹⁻³ The absence of a sudden decrease in end-expiratory carbon dioxide concentration and the lack of hypoxemia nearly rule out the possibility of venous air embolism. Significant impairment of spinal cord perfusion was excluded by a normal motor response to the wake-

up test. The sequence of hemodynamic changes, beginning with S-T segment changes, arrhythmia, and nodal rhythm, followed only later by hypotension, suggested a primary alteration in coronary artery flow. This hypothesis was further proved by the disappearance of the flow Doppler signal from the right coronary artery during external chest compression. To the best of our knowledge, this is the first description of coronary flow interruption by external chest compression. Even though this life-threatening complication is rare, it should be considered in patients with pressure on the chest during procedures that affect intrathoracic structures.

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