Severe Refractory Hypoxemia Following a Gunshot Injury*

Ismail T. Dairywala, MD; Juzar Lokhandwala, MD; Herbert Patrick, MD, FCCP; Raymond Talucci, MD; and Diwakar Jain, MD

We describe the case of a 57-year-old man with severe refractory hypoxemia, despite receiving ventilation therapy with 100% oxygen, following a gunshot wound to his left chest. A limited CT scan of the chest with contrast raised the suspicion of an arteriovenous (AV) fistula. Contrast-enhanced transthoracic echocardiography confirmed the presence of a pulmonary AV fistula. Traumatic pulmonary AV fistula is a rare, but serious and life-threatening condition that should be suspected in patients with thoracic injuries with persistent unexplained hypoxemia. Contrast echocardiography is a relatively simple, inexpensive, and readily available bedside test that can be used to confirm this diagnosis.

(CHEST 2005; 127:398–401)

Key words: contrast echocardiography; penetrating chest trauma; pulmonary arteriovenous fistula

Abbreviations: AV = arteriovenous; LA = left atrium; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography

Pulmonary arteriovenous (AV) fistulas are uncommon. They are usually congenital and are only rarely acquired. Such acquired conditions include cirrhosis, metastatic thyroid carcinoma, schistosomiasis, actinomycosis, Fanconi syndrome, mitral stenosis, and, very rarely, penetrating chest trauma. These pulmonary AV fistulas can be diagnosed with contrast echocardiography. However, we found only one case reporting the use of contrast transesophageal echocardiography (TEE) for the diagnosis of this condition in the setting of trauma. To our knowledge, this is the first reported case of a traumatic pulmonary AV fistula diagnosed in an acute setting utilizing bedside contrast-enhanced transthoracic echocardiography (TTE).

CASE REPORT

A 57-year-old male patient with an unknown medical history was brought into the emergency department after sustaining a gunshot (unknown caliber) wound to his left chest. The wound was allegedly self-inflicted, with the entry point at the left upper sternal border and the exit point near the tip of the left scapula. He was found unresponsive at home and was orally intubated en route to the hospital. On initial examination, the patient had a temperature of 98.3°F, BP of 130/80 mm Hg, heart rate of 110 beats/min, and a ventilation rate of 16 breaths/min. Cardiopulmonary examination revealed the presence of bilateral breath sounds and a regular heart rhythm without any abnormal sounds. The skin was warm, dry, and without cyanosis or telangiectasias. There was no clubbing of the digits. The CBC count was notable for a WBC count of 37,000 cells/μL (neutrophils, 77%; bands, 4%) and a hemoglobin concentration of 10.5 g/dL, with an hematocrit of 31%. The total creatine kinase concentration was 266 U/L (normal range, 0 to 160 U/L), with an MB fraction of 32 U/L (normal range, 0 to 24 U/L) and a creatine kinase index of 12.0 (normal range, 0 to 6). The troponin-I level was 35 ng/mL (normal range, 0.0 to 1.9). Arterial blood gas measurements revealed a pH of 7.32, a Pco2 of 39 mm Hg, a Po2 of 53 mm Hg, and an O2 saturation of 88% on a fraction of inspired oxygen of 100% delivered by mechanical ventilation with no positive end-expiratory pressure. An ECG showed sinus tachycardia, left atrial enlargement, incomplete right bundle-branch block and poor R-wave progression. The results of an emergency department TTE, performed to rule out cardiac trauma, were unremarkable, except for a hyperdynamic left ventricle. No wall motion abnormality or pericardial effusion were noted. A chest radiograph showed a left-sided infiltrate without cardiomegaly (Fig 1, left, A).

A CT scan of the chest and abdomen with IV contrast was obtained on the evening of hospital admission. During the CT scan, the patient’s oxygen saturation level dropped into the 60% range, and the test was aborted halfway through the chest imaging. The CT scan showed a small left-sided pneumothorax, a fracture of the left ninth rib with pulmonary contusion, a left pleural effusion, and a small contrast-filled area in the left lower lobe (Fig 1, right, B). The contrast-filled area in the left lower lobe was initially interpreted as extravasation of contrast into the lung tissues secondary to vascular injury from the penetrating wound. The abdominal CT scan findings were normal. A left-sided thoracostomy was then performed with only minimal drainage (50 mL) of bloody fluid. Hemodynamic data obtained from bedside pulmonary artery catheterization showed a pulmonary artery capillary wedge pressure of 2 mm Hg, a pulmonary artery systolic pressure of 18/4 mm Hg, a central venous pressure of 2 mm Hg, cardiac output of 7.0 L/min, and a cardiac index of 4.0 L/min/m². Despite aggressive cardiopulmonary care, including fluid and blood administra-
tion, the patient’s hypoxemia persisted, with his arterial oxygen saturation level remaining in the 60 to 70% range. A TTE with a “bubble study” using agitated saline solution contrast was performed on the next day in the morning to confirm the presence of a suspected right-to-left shunt as the cause of the refractory hypoxemia. The only available echocardiographic window was at the subcostal location. A four-chamber view of the heart (Fig 2, top right, B) was obtained that showed left ventricular hypertrophy and normal left ventricular function without evidence of any significant pericardial effusion. Saline solution (3 mL) agitated with air (0.25 mL) then was injected via a central venous port. The passage of the contrast material was visualized in the right atrium and the right ventricle (Fig 2, bottom left, C) followed by opacification of the left atrium (LA) after one to two cardiac cycles. The contrast was seen filling the LA from the pulmonary veins on the left side, which is compatible with the presence of a large left pulmonary AV shunt (Fig 2, bottom right, D). The calculated shunt fraction, as determined by the 100% fraction of inspired oxygen method, was 61%. Surgical or interventional radiology repair of the pulmonary AV shunt was sought, but the patient experienced an hypoxic cardiac arrest from which he could not be resuscitated. An autopsy was not performed as per the decision of the medical examiner.

**DISCUSSION**

Although penetrating chest trauma is a common emergency, the development of traumatic pulmonary AV fistulas is exceedingly rare. In three case series from the Mayo Clinic involving 204 patients with pulmonary AV fistulas, there were only two trauma-associated cases over a period of almost 45 years. We found seven other individual cases reported in the English-language literature. Four of these reports were the result of gunshot wounds. Interestingly, the presentation was delayed by many years after the initial injury in four of the seven cases.

Generally, the diagnosis of both congenital and acquired pulmonary AV fistulas has been made using a combination of data from the patient’s history, the physical examination, laboratory testing, chest radiographs, CT scans, contrast echocardiography, and pulmonary angiography. Although contrast echocardiography can be helpful in the diagnosis of this condition, we found only one report documenting the use of this modality in the setting of acute penetrating chest trauma. In that particular case, TEE was used. To our knowledge, this is the first case report in which the diagnosis of a traumatic pulmonary AV fistula secondary to a gunshot wound was confirmed using bedside contrast-enhanced TTE.

Our case also demonstrates an interesting feature with regard to the timing of the appearance of the contrast in the LA. The timing of the appearance of the contrast agent in the LA assists in differentiating an intrapulmonary shunt from an intracardiac shunt. In an intrapulmonary shunt, it takes three to five cardiac cycles after right atrial opacification before the contrast appears in the LA. In our case, the contrast was visualized in the LA within one to two cardiac cycles, which is more suggestive of an intracardiac shunt, such as an atrial septal defect. However, the contrast was clearly seen filling the LA from the pulmonary veins on the left side, which is compatible with the presence of an intrapulmonary shunt (Fig 2, bottom right, D). We think that the early appearance of the contrast in the LA was the result of an extraordinarily large AV fistula. This
is evidenced by the calculated shunt fraction of 61%, with the suggestion of a possible direct artery-to-vein connection. We found no other evidence for a right-to-left intracardiac shunt on both two-dimensional imaging of the cardiac chambers and color Doppler interrogation of the interatrial and interventricular septa. As the patient died prior to further diagnostic and therapeutic interventions, and due to the fact that an autopsy was not performed, we were not able to determine the exact location and extent of the fistula. It seems likely that the contrast-filled area seen on the chest CT scan was the site of the AV fistula. The fact that the contrast-filled area was sharply demarcated raises the possibility of an aneurysm with or without an associated AV fistula. Also, an aneurysm is more ovoid rather than rounded, which makes an AV fistula more likely. Because of the limited nature of the chest CT scan, the entire extent of the fistulous communication could not be defined.

It can be suggested that the patient may have had a pulmonary AV fistula prior to his injury, but this seems unlikely in the absence of other clinical findings such as telangiectasias and clubbing. We also found no evidence of any nontraumatic conditions that have been associated with pulmonary AV fistulas. Another unusual finding in this case was the lack of significant left pleural hemorrhage despite evidence of an obvious high-velocity perforating vascular chest injury, perhaps because of the tracking of the hematoma into a pulmonary vein.

The role of echocardiography in the evaluation of acute chest trauma using both TTE and TEE has been well documented. This patient underwent an emergency department TTE for the evaluation of potential cardiac injury as per the trauma evaluation protocol, the findings of which were entirely normal. With earlier suspicion, a contrast “bubble study” may have been combined with this study, resulting in an earlier diagnosis of pulmonary AV fistula. Our patient had progressively worsening hypoxemia despite maximal oxygen therapy. Because of its rarity, the suspicion on a traumatic pulmonary AV fistula was not raised until several hours after hospital admission, after a thorough search for all other possible causes failed. The chest CT scan was limited but nevertheless may have raised the
Identification and Treatment of Bronchoconstriction Induced by a Vagus Nerve Stimulator Employed for Management of Seizure Disorder*

Jagdeep S. Bijwadia, MD, FCCP; Robert C. Hoch, MD, FCCP; and Donn D. Dexter, MD

We evaluated a 63-year-old woman who developed dyspnea with a sensation of chest tightness that was temporally associated with discharges from a vagus nerve stimulator that had been implanted for the control of intractable seizures. Spirometry demonstrated the development of significant airflow obstruction associated with the firing of the stimulator. Adjustment of the stimulator settings resolved the discharge-associated bronchoconstrictive phenomenon. These findings highlight an important association between vagus nerve stimulators and dyspnea that should be considered in the differential diagnosis of patients with these devices who present with dyspnea and/or chest tightness. The relative importance of vagal stimulation to bronchoconstriction is suggested by the findings.

Key words: bronchoconstriction; dyspnea; seizure; vagus nerve stimulator

Vagus nerve stimulators are relatively new devices that are finding increased use in the nonpharmacologic management of seizure disorders. They are implanted in the subcutaneous tissues of the chest, with a wire lead inserted into the vagus nerve to deliver regularly timed cycles of electrical pulses that suppress epileptogenic foci.1 We report a case in which a vagus nerve stimulator had the unintended effect of inducing dyspnea that was associated with the activation of the device, which was found to temporally correlate with bronchoconstriction as demonstrated by serial pulmonary function assessments. Nerve stimulator setting adjustments were found to alleviate the bronchoconstriction and associated dyspnea.

CASE PRESENTATION

A 63-year-old, right-handed white woman with a 20-year history of psychomotor seizures with intractable features, despite management with levetiracetam and carbamazepine, underwent implantation of a vagus nerve stimulator (NeuroCybernetic Pros...