A 53-year-old man presented with a history of depressant syndrome. He had been hospitalized 16 years previously because of penetrating traumatic injury of the left hemithorax by bullet secondary to a suicide attempt. Recently, he came to the emergency department for an illness leading to the discovery of severe hypoxemia refractory to nasal oxygen therapy. The arterial blood gas measurements revealed a pH of 7.41, a PaCO2 of 41 mm Hg, a PaO2 of 45 mm Hg, and O2 saturation of 83% on ambient air; the PaO2 increased to 85 mm Hg on inspired oxygen of 100%, resulting in a shunt of 30%. Multidetector computed tomography–angiography of the chest showed clearly a giant proximal pulmonary arteriovenous fistula (Figure 1A and B and Movies 1 and 2 of the online Data Supplement). Transthoracic contrast echocardiography (TTCE) showed a massive early (in the third cardiac cycle) passage of microbubbles to the left side of the heart (Movie 3 of the online Data Supplement) without evidence of pulmonary artery hypertension or ventricular cavity enlargement. The diagnosis was a laterolateral fistula between the laterobasal segmental pulmonary artery (6 mm in diameter) and an aneurysmal sac draining toward an enlarged pulmonary vein (8 mm in diameter) and the distal pulmonary artery (4 mm in diameter). Clinical examination did not reveal a continuous murmur in the left posterior thorax. The draining artery was occluded first by a plug 10 mm in diameter, and the feeding artery was then occluded by a plug 12 mm in diameter (Figure 2A and B). The PaO2 increased to 75 mm Hg with a residual physiological shunt of 6%. Three-month multidetector computed tomography–angiography follow-up showed a decrease in the aneurysmal sac and the draining vein. One year later, TTCE showed extinction microbubbles (right to left passage) (Movie 4 of the online Data Supplement), and the O2 saturation was 96% on ambient air. The patient recovered uneventfully and at present continues to do well.

Although penetrating chest trauma is a common emergency, the development of traumatic pulmonary arteriovenous fistulas (PAVs) is exceedingly rare.1 Dairywala et
al2 reported 1 case of traumatic PAVF and 4 cases from the literature. The clinical presentation can be acute or can be delayed by many years after the initial injury. Currently, the diagnosis of both congenital and acquired PAVFs is made by combining data from the patient’s history, the physical examination, laboratory test results, chest radiographs, multidetector computed tomography scans, and TTCE.3 The early shunting in this case was related to the proximal location of the fistula and to the massive shunting.4 Pulmonary angiography is currently reserved for therapeutic purposes. Usually, congenital PAVFs, especially in Rendu-Osler-Weber disease, are termino-terminal related to capillary abnormalities. However, traumatic PAVFs, especially proximal, are latero-lateral secondary to injury of the pulmonary artery and vein walls. Treatment of the PAVF consists of occlusion of all feeding arteries to the aneurysmal sac. In our case, occlusion of the feeding pulmonary artery without occlusion of the draining pulmonary artery (the persistent pulmonary artery after the aneurysmal sac) could not be performed efficiently because of reflux of the systemic blood through systemo-pulmonary shunting from the bronchial arteries. Two plugs achieved the occlusion, one within each pulmonary artery. The other therapeutic option was a left lower lobe resection without preservation of lung parenchyma like the endovascular vaso-occlusion.

Disclosures
None.

References
Severe Refractory Hypoxemia 16 Years After a Gunshot Injury: Multidetector CT-Angiography Pattern and Endovascular Treatment
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