Case Reports

Severe Spinal Cord Ischemic Injury

Secondary to Device Embolization after Transcatheter Closure of a Patent Arterial Duct

Liang Tang, MD
Shenghua Zhou, MD, PhD
Xiangqian Shen, MD

Percutaneous closure of patent arterial ducts with the AMPLATZER Ductal Occluder has become an effective and widely accepted alternative to surgical management. Although rarely, the occluder can be dislodged after an initially successful deployment, and with catastrophic consequences. We describe such a case in a 12-month-old girl who underwent transcatheter closure of a patent arterial duct. After device deployment, the occluder embolized in the patient's descending thoracic aorta, and severe spinal cord ischemic injury resulted. To our knowledge, ours is the first report of this complication after the deployment of an AMPLATZER Ductal Occluder. We discuss pathophysiologic mechanisms that could expose patients to the risk of device dislodgment, and we review the relevant medical literature. ***(Tex Heart Inst J 2014;41(1):83-6)***

Transcatheter device closure of patent arterial ducts has become a well-established alternative to surgical management: the procedure is associated with a high success rate and an excellent long-term outcome.[1] Rarely, severe complications can occur, such as device embolization, hemolysis, device-related infective endocarditis, iatrogenic aortic coarctation, and left pulmonary artery stenosis.[1,2] We present the case of an infant in whom an AMPLATZER® Ductal Occluder (ADO) (St. Jude Medical, Inc.; St. Paul, Minn) was used to close a patent arterial duct. The device embolized into the patient's descending thoracic aorta, causing ischemic injury to the spinal cord. We discuss possible reasons and review the relevant medical literature.

Key words: *Aorta, thoracic/surgery; device removal/methods; ductus arteriosus, patent/therapy; embolization, therapeutic/instrumentation; foreign-body migration/surgery; spinal cord injuries/etiology/prevention & control*

From: *Department of Cardiology, the Second Xiangya Hospital of Central South University, Changsha 410011, People's Republic of China*

Address for reprints: *Shenghua Zhou, MD, PhD, Department of Cardiology, the Second Xiangya Hospital of Central South University, No. 139, Middle Ren-min Rd., Changsha 410011, Hunan, PRC*

E-mail: *zhougqin@21cn.com*

Case Report

In July 2011, a 12-month-old girl (weight, 10.2 kg; height, 78 cm) with a patent arterial duct was referred for transcatheter closure of the defect because of recurrent upper respiratory tract infections. The patient's heart rate was 140 beats/min, and her oxygen saturation was 96% on room air with no signs of differential cyanosis. Cardiac examination revealed a palpable thrill, an accentuated pulmonary component of S_2, and a continuous machinery murmur audible at the left upper sternal border. Transthoracic echocardiograms showed a patent arterial duct (tubular and 3 mm in diameter) and left-sided chamber enlargement. After the parents gave informed consent, we placed the patient under general anesthesia and undertook transcatheter closure of the patent arterial duct. Cardiac catheterization data included a pulmonary artery pressure of 54/30 mmHg (median, 37 mmHg), an aortic pressure of 81/42 mmHg (median, 56 mmHg) with a calculated Qp:Qs of 2.5:1, and a pulmonary vascular resistance of 3.0 Wood units. Aortic angiograms showed a short and tubular patent arterial duct (Fig. 1A) with no aortic ampulla, 3.5 mm in diameter at its narrowest, and left-to-right shunting. A 6- to 8-mm ADO was deployed in routine fashion under fluoroscopic guidance. Fifteen minutes after trial occlusion, the patient's pulmonary artery pressure decreased to 30/14 mmHg (median, 19 mmHg), and no decrease in aortic pressure or systemic arterial oxygen saturation was observed. An aortogram confirmed occlusion of the duct and stable positioning of the occluder with slight protrusion toward the aortic arch (Fig. 1B). No pressure gradient was detected upon pullback from the ascending aorta to the descending aorta. The ADO was then completely released (Fig. 1C).

Eight hours after the procedure, the patient began crying irritably. Physical examination revealed a continuous murmur. Emergent fluoroscopic investigation confirmed

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that the ADO had become dislodged and had embolized into the descending thoracic aorta in the transverse plane, perpendicular to the direction of blood flow. Almost the entire descending aorta was obstructed (Fig. 1D). An attempt to retrieve the device percutaneously was unsuccessful, and the patient's lower limb showed signs of hypoperfusion. The ADO was ensnared and moved to the proximal descending aorta (Fig. 1E), and the patient was referred for urgent cardiac surgery. Through a left posterolateral thoracotomy, the device was removed and the duct was directly ligated. The patient was transferred to the cardiac intensive care unit on ventilation and with inotropic support.

During the next 24 hours, the patient had transient renal failure that rapidly responded to intravenous fluids and low-dose dopamine. On postoperative day 2, the patient was noted to have lower-limb paralysis. A computed tomographic brain scan revealed no signs of cerebral infarction. Magnetic resonance images (MRI) of the spinal cord revealed ischemic damage between the T7 to L2 vertebral levels (Figs. 2A and 2B). Accordingly, the lower-limb neurologic deficits were attributed to spinal cord ischemic injury consequent to device embolization. The patient was given intensive neurologic rehabilitative therapy. Six months later, MRI showed that the ischemic damage had disappeared (Fig. 2C); however, the patient's functional recovery from the systemic neurologic deficits was slow. After rehabilitative therapy for more than 2 years, the muscle force of her lower limbs was beneath expectations. In November 2013, she was taken for stem cell transplantation in an attempt to treat the spinal cord.

Discussion

Embolization of an ADO is rare but potentially life-threatening after the transcatheter closure of a patent arterial duct. A multicenter study of 439 ADO implantations yielded an approximate 0.5% prevalence of device embolization.[2] Embolization typically occurs immediately or within 24 hours after device implantation; however, late embolization has also been reported.[3] Embolization can result from the selection of undersized devices, pulmonary hypertension, unfavorable anatomic features of the patent duct (such as a large, tubular duct without constriction) and inadequate operator experience.[4-6] Reports of ADO embolization usually involve the descending aorta and the right pulmonary artery; the severity of clinical symptoms varies with the degree of blood-flow obstruction.[6-8] When an ADO migrates into the descending aorta and lodges in the transverse plane perpendicular to the direction of blood flow, distal perfusion can be compromised substantially, so the device should be retrieved urgently to prevent catastrophic consequences.

Percutaneous retrieval has proved to be feasible; according to one report, the ADO was retrieved percutaneously in approximately 80% of cases.[9] Regardless, this method is time-consuming and technically challenging, and multiple retrieval attempts might cause aortic injury and further compromise distal systemic perfusion.[4,9] In contrast, surgical device removal is considered to be simple, safe, and efficient, so it is more frequently performed.[6,8] If percutaneous retrieval is unsuccessful or a lower limb shows signs of hypoperfusion, surgical

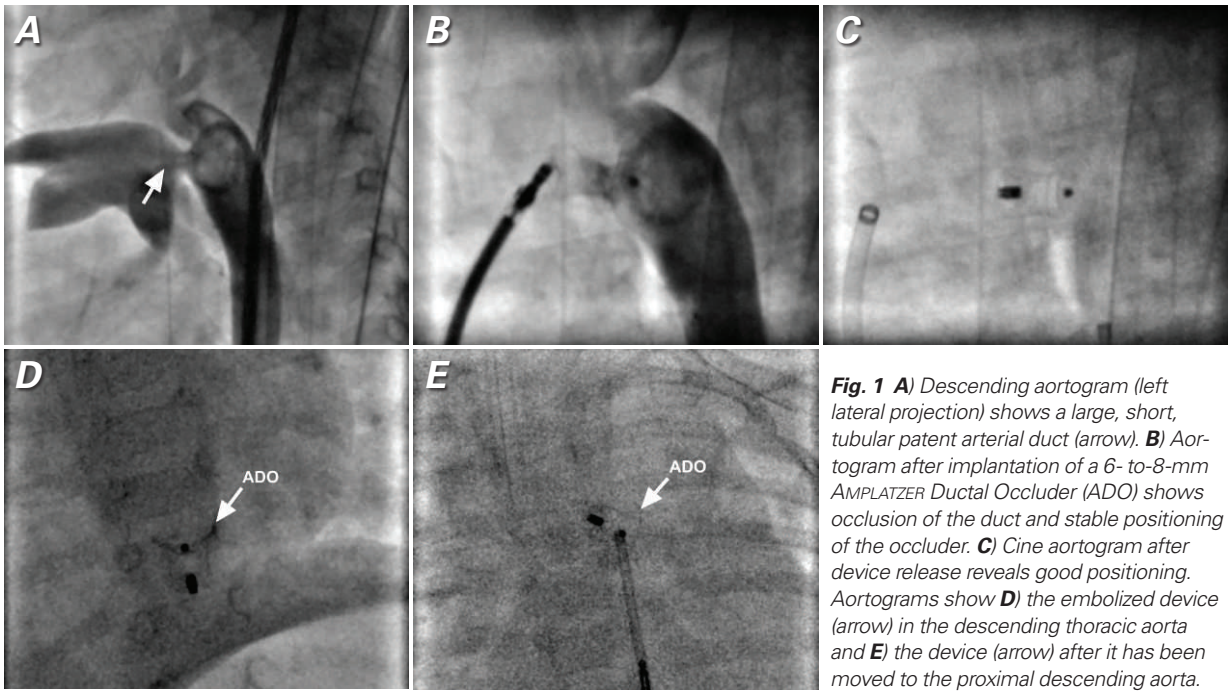

Fig. 1 ***A****) Descending aortogram (left lateral projection) shows a large, short, tubular patent arterial duct (arrow).* ***B****) Aortogram after implantation of a 6- to-8-mm* Amplatzer *Ductal Occluder (ADO) shows occlusion of the duct and stable positioning of the occluder.* ***C****) Cine aortogram after device release reveals good positioning. Aortograms show* ***D****) the embolized device (arrow) in the descending thoracic aorta and* ***E****) the device (arrow) after it has been moved to the proximal descending aorta.*

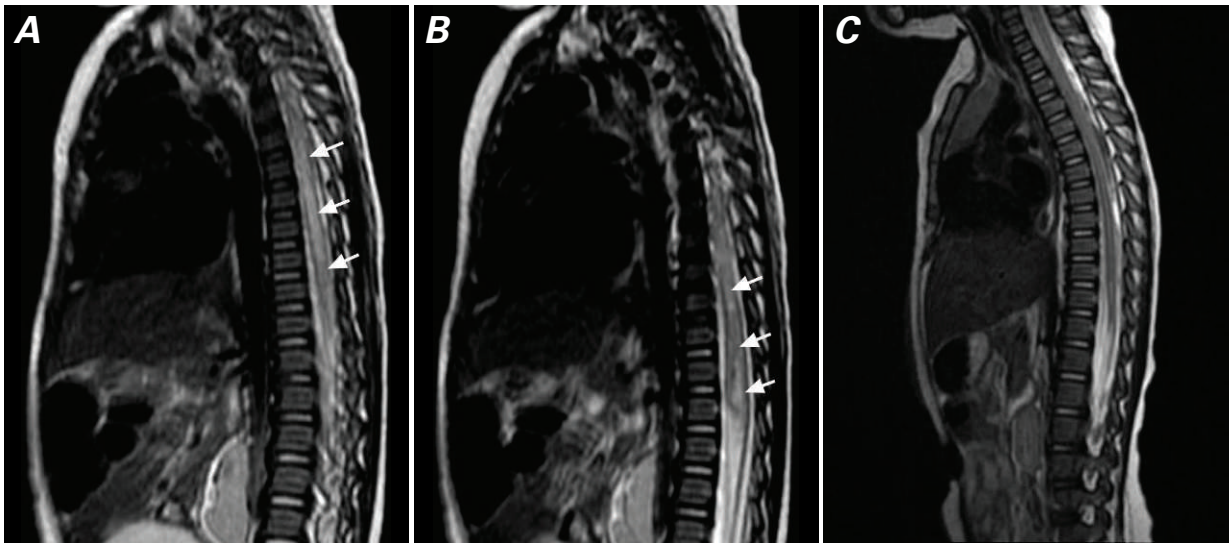

Fig. 2 *Magnetic resonance images of the spinal cord show* ***A****) ischemic damage between the T7 and T12 vertebral levels and* ***B****) ischemic damage extending to the L2 vertebral level.* ***C****) Image from 6 months later shows that the damage has disappeared.*

retrieval should be performed immediately, especially in small children. In such cases, it is important to stabilize the embolized device in a relatively low-risk location until the patient is readied for the operating room. A snare or bioptome can be used to reposition the device in the proximal descending aorta, where harm to the patient is minimized.[9]

In our patient, 3 factors might have contributed to device embolization: the duct was very short and without a constriction within it; the possibly undersized ADO might not have attached firmly to the duct wall; or the patient's pulmonary pressure might have increased acutely during Valsalva maneuvers such as coughing or crying and transiently exceeded her aortic pressure, causing the undersized device to migrate into the descending aorta.

Although our patient promptly underwent surgical device removal, her spinal cord sustained severe ischemic damage. To our knowledge, this is the first report of severe spinal cord ischemic injury to have occurred secondary to device embolization after the transcatheter closure of a patent arterial duct.

Blood to the spinal cord is supplied chiefly by the anterior spinal artery, which in turn is mostly supplied by branches from paired intercostal arteries, branches of the vertebral arteries, and lumbar segmental arterial branches of the descending aorta.[10] The anterior spinal artery is vulnerable to ischemia related to surgery, hypotension, or embolism.[11] Spinal cord ischemia is a severe complication in the surgical repair of aortic coarctation; underlying mechanisms include prolonged distal hypotension, protracted aortic cross-clamping, embolism of the anterior spinal artery, and extensive intraoperative destruction of the intercostal arteries with impaired collateral circulation.[11,12] Our patient's extensive spinal cord ischemia could have developed in similar fashion. After the device embolized, the patient's distal aortic perfusion pressure was acutely and protractedly decreased, which compromised the anterior spinal artery collateral vessels originating from the crucial intercostal arteries at the T6 to T12 vertebral levels and also compromised the downstream lumbar segmental arterial branches.

This case highlights the importance of preventing device embolization and reinforces the necessity of postprocedural monitoring. To minimize the risk of device embolization in similar cases, accurate ductal measurements for occluder selection and careful postimplantation evaluation before device release are crucial. In small children who have a large, tubular duct, the ADO should be at least 4 mm larger than the duct's narrowest portion.[1,6] In addition, in infants with moderate-to-severe pulmonary hypertension, the AMPLATZER Duct Occluder II (St. Jude Medical) might be the device of choice: its 2 symmetric retention discs can secure its position in the pulmonary side of the duct, thus preventing device embolization into the systemic circulation.[13]

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