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Carotid Embolectomy in the Treatment of a Paradoxical Embolus

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Development of a paradoxical embolus to the carotid circulation through a patent foramen ovale is uncommon but well documented. Previous surgical experience with this entity is limited. Treatment for this condition typically involves anticoagulation or the use of an inferior vena cava filter with concurrent closure of the patent foramen itself. We report successful surgical treatment of a woman with a paradoxical embolus lodged in her left carotid artery. In addition, we provide a brief review of this rare topic and treatment rationale.

A paradoxical embolus (PDE) is a rare phenomenon that results most frequently from a patent foramen ovale (PFO).[11] This term refers to the embolic entry of a venous thrombus via a shunt into the arterial circulation. The clinical presentation is acute arterial ischemia with or without clinical evidence of deep venous thrombosis (DVT) or pulmonary embolism (PE). The typical treatment for this PFO is emergent anticoagulation or the use of preventive measures such as an inferior vena caval filter to prevent further progression of end-organ damage, especially cerebral infarction from embolism.[2] Surgical management of PDE in the extracranial cerebrovascular system has been reported previously on two occasions with successful results (Table 1).[3,4] We report an additional case of a PDE that was surgically removed from the left common carotid artery.

CASE REPORT

A 59-year-old female with a past medical history significant for a patent foramen ovale dislocated her patella after a fall. At that time, she consulted an orthopedic surgeon, who recommended conservative management including bedrest and non-weight bearing activities. One month later, she presented to an outside hospital with a right-sided facial droop, right-sided hemiplegia, and aphasia. In addition, the patient was found to be hypoxemic. A CT of the head was performed and was unremarkable. In addition, a spiral CT of the chest was performed to evaluate for pulmonary embolism, given the patient's poor oxygenation. This revealed bilateral pulmonary emboli. The patient was then transferred to our institution with the diagnoses of bilateral pulmonary emboli and embolic stroke.

On admission, the patient was afebrile in no respiratory distress with a regular pulse of 87 bpm; the patient had pulse oximetry of 91% on 4 L oxygen and 100% on 100% face mask. Her physical exam was remarkable for a systolic ejection murmur as well as ecchymosis of the left patella extending down to the shin without erythema or discrete tenderness. The neurological exam was significant for right-sided hemianopsia, a prominent right-sided facial droop, right upper and lower extremity weakness,

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Table I. Carotid embolectomy as treatment of paradoxical embolus

Reference	Age (years)	Sex	Presentation	Location of obstruction	Type of surgery	Outcome	Follow-up treatment
Present case	59	F	Embolic stroke, pulmonary embolism, patent foramen ovale	Left common carotid artery	Carotid embolectomy	Good	Long-term anticoagulation and percutaneous transcatheter closure of PFO
McKinney et al. (2001)[3]	67	M	Embolic stroke, pulmonary hypertension, patent foramen ovale	Right carotid bifurcation	Carotid embolectomy	Good	Long-term anticoagulation
Turnbull et al. (1998)[4]	54	M	Embolic stroke, pulmonary embolism, patent foramen ovale	Innominate artery	Combined carotid bifurcation and brachial embolectomy	Good	Long-term anticoagulation

and aphasia. A bilateral lower extremity duplex scan showed evidence of an acute left lower extremity deep venous thrombosis (DVT) involving the left posterior tibial, peroneal, and anterior tibial veins. Given the history of immobilization and trauma leading to left lower extremity DVT, combined with the patient's patent foramen ovale, it was believed that she likely had a pulmonary embolism as well as an embolus to the left middle cerebral artery through her patent foramen ovale. A Greenfield filter was placed at that time.

The day after admission, a non-contrast head CT showed mass effect consistent with a maturing left cerebral nonhemorrhagic infarct. Three days after admission, the patient complained of increased right-sided weakness and was taken for bilateral carotid duplex imaging to rule out carotid artery disease. The study revealed a heterogeneous embolus in the left common carotid measuring 60-80% diameter stenosis with possible instability; the distal end of the embolus was imaged (Fig. 1A). At this point, the vascular service was called to see the patient.

Thereafter, the patient was taken to the operating room for embolectomy because the embolus appeared to be unstable and had potential to cause further cerebral insult. After heparin was administered, the carotid artery was opened, revealing a large embolic piece of material at the bifurcation. This embolus was removed, but since adequate retrograde flow was not yet established, a #3 Fogarty catheter was carefully passed to extract a tail of thrombus approximately 6 cm in length (Fig. 2). Backbleeding commenced and the Fogarty catheter was passed one additional time to ensure that no additional material was present distally in the internal carotid artery. A #12 shunt was rapidly placed and flow was documented via Doppler flow. The arteriotomy was closed with a Dacron patch. An intraoperative duplex scan revealed excellent flow throughout the carotid and no loose material or debris. After closure, the patient was awakened on the table and taken to the recovery room where she remained stable with an unchanged neurological exam. There was no worsening of neurologic status after the procedure.

Six days postembolectomy, the patient underwent PFO closure in the cardiac catheterization laboratory for her patent foramen ovale via a transhepatic route. The presence of the Greenfield filter made placement through her inferior vene cava hazardous. The patent foramen ovale was sized with an Amplatzer sizing balloon and the Amplatzer atrial septal occluder device was delivered by fluoroscopic guidance.

After the operation, hemodynamic data indicated no significant shunt by oximetry. Two days later, the patient's care was transferred to the acute rehabilitation service where she spent 4 weeks and was eventually placed on long-term anticoagulation with warfarin for her lower extremity DVT. At 6 week follow-up, a duplex scan of the left carotid showed no residual or recurrent embolic material (Fig. 1B). The patient was well at discharge from rehabilitation with a stable neurologic examination.

DISCUSSION

Paradoxical embolus (PDE) refers to the embolic entry of a venous thrombus via a shunt into the arterial circulation. Patent foramen ovale is the major predisposing factor of PDE[5] and has been found to be present in 9 to 35% of the populations.[6,7] PDE has been reported to be quite infrequent, causing 2% of arterial emboli.[1] Some evidence, however, suggests that this figure may underestimate the true incidence of this phenomenon. The National Institute of Neurological Disorders and Stroke Data Bank has estimated that approximately 40% of strokes are without identifiable anatomical cause.[8] Approximately one-quarter of these cryptogenic strokes occur in

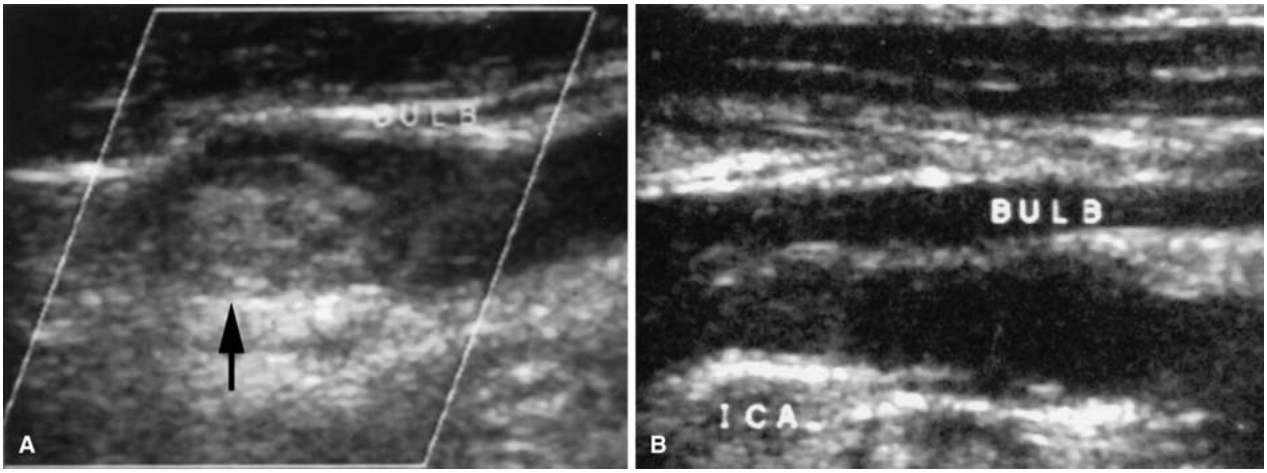

Fig. 1. Left carotid duplex imaging scan preoperatively **A** and at 6-week follow-up **B**. *Arrow* delineates echogenic embolus sitting past left carotid bulb. Note that area of occlusion is clear at 6-week follow-up.

patients with a PFO,[7] which suggests that a PDE may be involved in the pathophysiology of many unexplained cerebrovascular accidents. Clinically, the diagnosis of PDE requires the following criteria: (1) presence of DVT or PE; (2) abnormal communication between the venous and systemic circulations; (3) clinical, angiographic, or pathologic evidence for arterial embolism; and (4) presence of a gradient favoring right-to-left shunting.[9] A PDE should be suspected when large amounts of thrombus or embolus are removed during embolectomy or the embolus is well formed and rubbery in texture. On occasion, even the indentations caused by venous valves may be noted on the surface of the embolus.

The treatment of PDE typically involves immediate anticoagulation once an arterial embolism has been diagnosed.[2] This strategy is complicated in patients who suffer acute stroke with increased risk of intracranial hemorrhage. For these patients, other strategies such as the use of a Greenfield filter for prevention of further emboli should be considered. It should be noted, however, that the efficacy of such filters in the prevention of small embolic events affecting the intracranial circulation is controversial.[10] Since most patients presenting with PDE have a PFO, definitive treatment in these patients also involves closure of this defect either by percutaneous transcatheter or surgical closure. Martin et al. have reported procedural success with percutaneous transcatheter closure of a PFO as an alternative to surgical closure or extended anticoagulation as treatment of PDE.[11] Although controversial, many today believe that PFO should be initially approached via an endovascular route with surgical closure reserved for patients with large atrial septal defects or unusual atrial septal anatomy, which might cause a device to impinge on additional structures of the heart.

There have seldom been references to the use of thrombolysis in patients with PDE. The therapy is largely undefined, but has been shown to have utility in treating ischemic infarcts, the etiology of which have been linked to a paradoxical embolus. One well-documented case reported successful treatment via intravenous recombinant tissue plasminogen activator of a paradoxical embolus associated with pelvic vein thrombosis in a 16-year-old female with acute onset of left hemiplegia, left hemisensory deficit, and dysarthria.[12] While such results are encouraging, further effort is needed to define indications for thrombolysis in patients with PDE.

Surgical treatment of carotid embolism caused by PDE is rare. Previous to this case, only two other reported cases of surgical management have been described. McKinney et al.[3] reported the case of a 67-year-old man with a saddle embolus to the carotid bifurcation and a demonstrable large patent foramen ovale, as well as DVT and multiple pulmonary emboli treated successfully with embolectomy. The authors chose not to repair the patient's foramen ovale via surgical closure because of what they termed the uncertain risks and benefits of PFO closure versus long-term anticoagulation. Turnbull et al.[4] described the surgical removal of a paradoxical saddle embolus to the innominate artery, which caused cerebral ischemia in a 54-year-old patient who also had a DVT, multiple PE, and a patent foramen ovale. The authors of this case decided to place the patient on long-term anticoagulation as definitive treatment in lieu of PFO closure

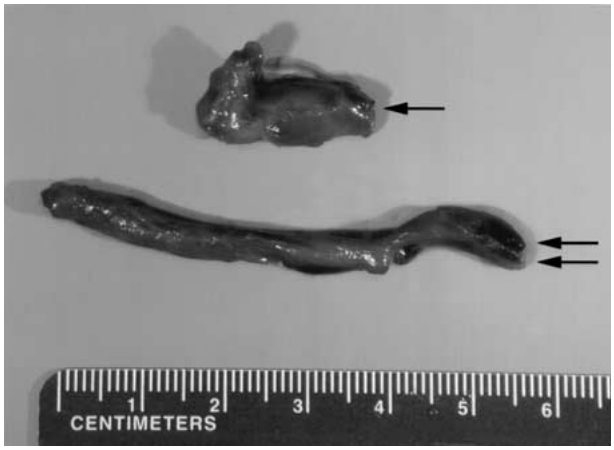

Fig. 2. Operative specimen revealing thrombus. *Arrow* corresponds to echogenic embolus shown on preoperative duplex imaging scan (Fig. 1A). *Double arrow* denotes end of thrombus tail.

because they felt there was no prominent advantage in performing the procedure.[4]

The patient presented in this report had the important features facilitating a work-up of PDE to the carotid circulation. She had an embolizing lower extremity DVT causing bilateral pulmonary emboli, the etiology of which likely extended back to her patellar injury and bedrest a month before the ischemic events. The patient also had a PFO documented by transesophageal echocardiography (TEE), the gold standard for detection of PFO.[13] Such echocardiography should be routinely performed when patients demonstrate large amounts of well-formed chronic thrombi during embolectomy. The right-to-left flow through the PFO may have been facilitated by pulmonary hypertension secondary to pulmonary emboli. This progression likely led to the thrombus in her left common carotid artery and the ensuing cranial ischemic events.

The guidelines for when to proceed with TEE in patients with arterial embolism have not yet been strictly defined.[14] When an obvious impending source of embolism has been elucidated by transthoracic echocardiography, that technique is deemed to be adequate in most cases. However, TEE is performed when possible in patients found to have both peripheral arterial embolism and negative transthoracic echocardiograms. Studies comparing transthoracic echocardiography with TEE for the detection of potential sources of arterial embolism have all found higher rates of detection with TEE than with transthoracic echocardiography. Given our emergent situation and the patient's ability to tolerate TEE long enough to complete the examination, TEE was our preferred imaging modality.

We chose to perform a carotid embolectomy to prevent further cerebrovascular insult. The major risk of carotid embolectomy is that of further ischemia caused by either embolic events or ischemic territory reperfusion. We chose to proceed because we felt that the potential benefits of performing the operation outweighed the risks of intraoperative and postoperative stroke extension. The patient still had movement in her right lower extremity, as well as improving aphasia at the time of embolectomy. If she had had evidence of a massive stroke or had been unconscious, embolectomy would not have been indicated. However, in this case, not removing the embolic material would have left her susceptible to further cerebral infarction and risked further decrease in her functional status. Also, the carotid duplex scan performed preoperatively showed flow around the thrombus. If the nidus present were to cause further thrombosis, complete occlusion of the carotid might have occurred and ultimately have led to further ischemic insult to the brain. As the duplex scan revealed an unstable situation, we did not feel additional diagnostic studies such as magnetic resonance angiography were indicated, as the situation in the internal carotid artery needed to be immediately addressed.

In conclusion, we present a case of PDE causing a cerebrovascular event, successfully treated with carotid embolectomy after placement of a Greenfield filter.

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