Successful innominate thromboembolectomy of a paradoxic embolus

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A 54 year-old man had symptoms of acute right hemispheric cerebral ischemia. He was initially considered for participation in a trial of early thrombolysis in stroke, but an innominate artery embolus was found with no apparent arterial source. The embolus was removed by means of a combined brachial and carotid bifurcation approach to protect the cerebral vasculature from embolic fragmentation during extraction. Further investigation revealed deep venous thrombosis, evidence of pulmonary emboli, and a patent foramen ovale, supporting a diagnosis of paradoxic embolus. Additional treatment included anticoagulation and placement of an inferior vena caval filter. The unusual condition of paradoxic embolus is reviewed, and the management of this patient is discussed. (J Vasc Surg 1998;28:742-5.)

Paradoxic embolus involves passage of a venous embolus into the arterial system. Although the existence of this clinical event has been established for many years, advances in our ability to diagnose intracardiac right-to-left shunts has increased recognition of the potential for paradoxic emboli to occur. Still, management and prevention of this problem are controversial. An unusual case is presented in which the embolus lodged in the innominate artery with a small fragment proceeding further into the cerebral circulation, resulting in stroke. Surgical intervention was necessary to prevent both impending brachial emboli and further cerebral embolism.

CASE REPORT

A 54-year-old man with a 1-hour history of headache and collapse was referred to the Vancouver General Hospital. His history included hypertension, hypercholesterolemia, and myocardial infarction 10 years earlier. He did not have diabetes and had stopped smoking after the myocardial infarction. At examination the Glasgow Coma Score was 15, but he exhibited left hemiparesis and left-sided neglect, and the eyes deviated to the right. Further examination revealed left-sided sensory loss, increased tone, visual field deficit, and hyperreflexia in keeping with a right-hemispheric stroke. The patient also had a cold, pulseless right arm. Cuff blood pressure was obtainable only from the left arm, which was normal to examination. There also was no right carotid impulse.

An electrocardiogram showed normal sinus rhythm with old nonspecific anteroinferior T-wave changes. A computed tomographic scan of the head showed no evidence of intracerebral hemorrhage. Angiography revealed a large embolus in the innominate artery that extended into the proximal right common carotid artery (Fig 1). Right subclavian flow was established in a retrograde manner through the vertebral artery, and the carotid bifurcation could not be clearly visualized. A distal embolus was present in the superior division of the right middle cerebral artery bifurcation, accounting for the neurologic symptoms.

The patient was taken urgently to the operating room, where a standard carotid bifurcation exposure was performed as was dissection of the brachial artery at the antecubital fossa. With electroencephalographic monitoring, the external and internal carotid arteries were clamped, and a Fogarty balloon-tipped catheter was used to remove a portion of the thrombus from the innominate and proximal common carotid arteries. The rest of the thrombus was extracted with a Fogarty catheter passed up from the brachial artery. During this second part of the procedure, the internal carotid artery was kept clamped, and the common carotid artery was flushed out before the arteriotomy was closed. The common and internal carotid arteries were free of atheromatous disease. Normal carotid and brachial circulation was restored. The procedure was completed within 6 hours of the onset of symptoms.

A postoperative transthoracic echocardiogram did not demonstrate an intracardiac source of thrombus, mechanical defect, or right-to-left shunt. A paradoxic embolus was

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suspected, and on further questioning, the patient admitted to increasing right leg pain with mild swelling and progressive dyspnea over the 2-week period before admission. Duplex ultrasonography of the right leg revealed thrombus involving the common femoral vein and extending into the popliteal and calf veins. A ventilation-perfusion scan demonstrated evidence of bilateral pulmonary emboli. A search was made for a venous-arterial communication. Transesophageal echocardiography was performed and demonstrated a patent foramen ovale with marked right-to-left shunting during respiration and coughing.

The patient's neurologic status showed steady improvement, and he quickly regained much of his left-sided motor and sensory function. A computed tomographic scan obtained on the first postoperative day confirmed a 4.5-cm region of effacement in the right temporal region (Fig 2). The dyspnea resolved, and leg swelling responded to rest and elevation. A Greenfield filter was placed in the inferior vena cava on the fifth postoperative day, and the patient was discharged later that day. Three months after the operation he still had a left visual field deficit, a slight left pronator drift, decreased fine sensation in the left hand and arm, slowed fine finger movements, and a decreased left arm swing. The patient continued to be observed for resolution of the cerebral insult and for deep venous thrombosis and pulmonary embolism.

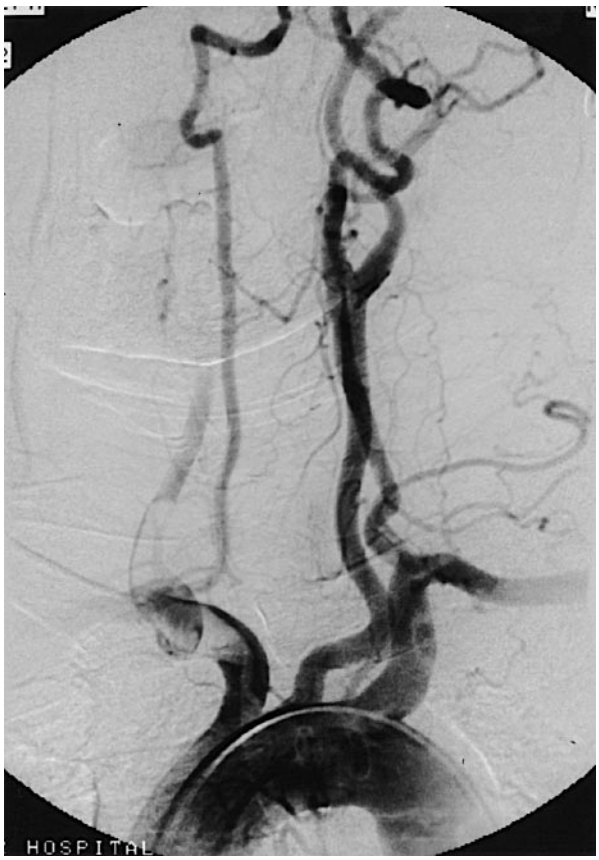

Fig 1. Digital subtraction angiogram demonstrates an embolic filling defect in the innominate artery. Flow into the right subclavian, carotid, and vertebral arteries is markedly impaired. The right carotid bifurcation was not visualized.

DISCUSSION

This case report demonstrates the unusual situation of a massive paradoxic embolism lodged in the innominate artery. A smaller embolic fragment had already made its way into the middle cerebral artery circulation and caused neurologic symptoms. A portion of the innominate embolus would likely have traveled down the brachial circulation.

The patient was treated with emergency surgical removal of the innominate embolus to restore compromised cerebral and brachial circulation and to prevent further embolization of debris. Access was gained through both the right neck and right brachial sites. This allowed effective restoration of both carotid and brachial circulation and protected the cerebral vasculature from embolic debris, which might be dislodged during brachial embolectomy. A more direct approach to the innominate artery through a sternotomy was not favored because of a higher morbidity associated with sternotomy and a need to explore the carotid bifurcation for emboli, because this area was not well visualized on the preoperative angiogram. It was also felt that direct surgical manipulation of the innominate artery might dislodge embolic debris, resulting in further cerebral embolism.

A postoperative search was made for the source of this patient's unusual thrombus. When the echocardiogram was found to be normal, a paradoxic embolus was suspected, and the patient was questioned specifically for signs and symptoms of venous thromboembolism. The shortness of breath and leg swelling were quite mild and had not been brought to light in the preoperative assessment. A transesophageal electrocardiogram was obtained and revealed a patent foramen ovale, supporting the diagnosis. It was chosen not to repair this lesion because of the added morbidity of the procedure. A Greenfield filter was placed to prevent possible further pulmonary and paradoxic emboli from deep venous thrombosis of the right leg. There have been reports of subsequent paradoxic emboli despite insertion of a Greenfield filter,[1,2] and future surgical closure of the patent foramen ovale remains under consideration for this patient. Meanwhile, the patient is receiving anticoagulation therapy to pro-

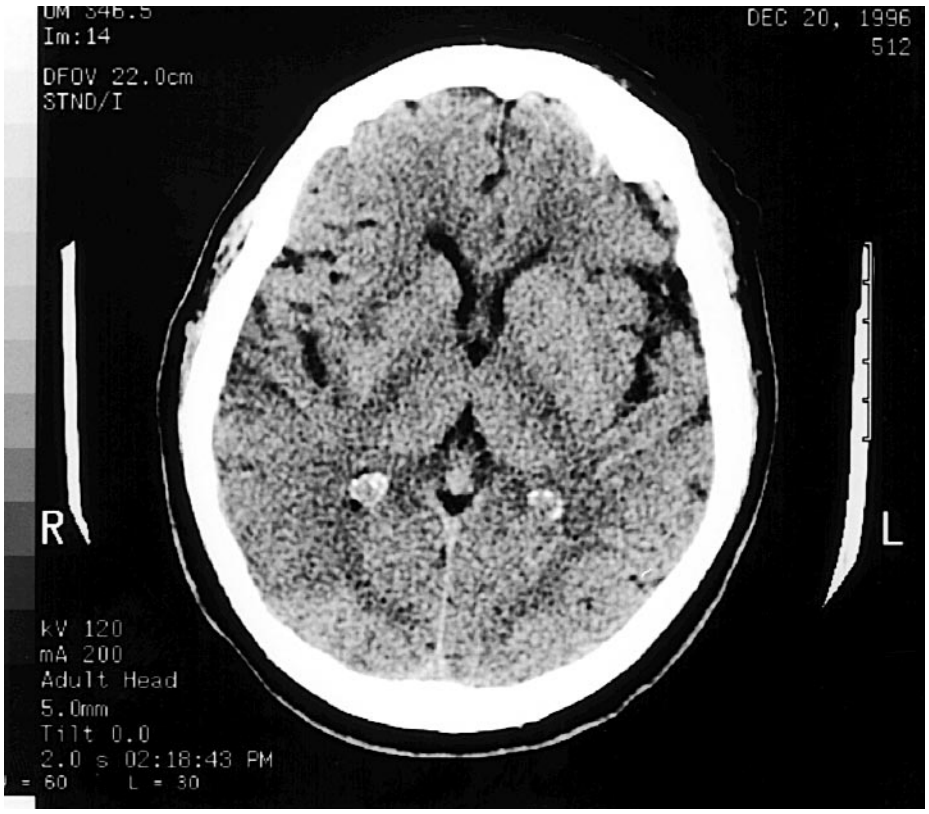

Fig 2. Computed tomographic scan obtained 24 hours after the onset of symptom shows early evidence of ischemic brain injury in the distribution of the left middle cerebral artery. There is cortical effacement of the right temporal region.

tect him from further thromboembolism. An underlying cause of the venous thrombosis could not be elucidated.

Paradoxic embolism was first described by Cohnheim in 1877[3] and is an infrequent event, accounting for fewer than 2% of all arterial emboli.[4] It may be defined as passage of a venous thrombus through a right-to-left shunt resulting in systemic arterial embolization. Diagnosis is typically based on the presence of an arterial embolism without a corresponding source in the left heart or the proximal arterial tree, an embolic source within the venous system with or without pulmonary embolism, an abnormal intracardiac or intrapulmonary communication between right and left circulations, and a pressure gradient that promotes right-to-left shunting at some point in the cardiac cycle.[5] Although most of these emboli are venous thrombus from branches of the inferior vena cava,[6,7] reports exist of paradoxic embolization of fat,[8] tumor,[9] amniotic fluid,[5] and air.[10,11]

Sites of paradoxic arterial embolization usually involve the cerebral vasculature or the extremities,[1,12] although coronary, renal, and splenic arterial emboli have been described.[13] The site for right-to-left shunting among such patients usually is a patent foramen ovale; however, other atrial septal defects, ventricular septal defects, and aortocaval and pulmonary arteriovenous fistulas have been implicated.[13-15] Patent foramen ovale has been found to be present in 20% to 30% of the healthy population.[16,17] The actual overall risk for paradoxic embolus in the presence of a patent foramen ovale is unknown[12] but likely correlates with the size and hemodynamic features of the defect.

Treatment of patients with paradoxic emboli is controversial and must be individualized. The acute ischemic event must be addressed, and further embolism must be prevented. In general anticoagulation is indicated[1] and is continued for 3 to 6 months or indefinitely if the patient has a patent foramen ovale or chronic obstructive pulmonary disease–induced pulmonary hypertension, which would enhance right-to-left flow.[5,13] If deep venous throm-

bosis is present, a vena caval filter should be considered, especially if pulmonary embolism is present or if anticoagulation is contraindicated. Peripheral emboli should be extracted when possible or managed with thrombolysis. Surgical closure of a patent foramen ovale, transcatheter closure, fibrinolysis, and anticoagulation and antiplatelet therapy all have been described[5,12,13,18] with no prominent advantage evident from a particular management strategy. Hanna et al.[19] observed no recurrences during a mean follow-up period of 28 months among 15 patients with paradoxic cerebral embolus who underwent treatment with aspirin, warfarin, open heart closure, or a septal occlusion device.

Paradoxic embolism is a rare diagnosis that should be entertained when a patient has arterial embolism with no clear embolic source. A search should be made for deep venous thrombosis and a patent foramen ovale, both of which may be subclinical. This case study illustrates a novel approach to a large paradoxic embolus in an unusual location, which if left untreated may have fragmented with devastating consequences.

REFERENCES

1. Katz S, Andros G, Kohl R, Harris R, Dulawa L, Oblath R. Arterial emboli of venous origin. Surg Gynecol Obstet 1992;174:17-21.
2. Dalman R, Kohler TR. Cerebrovascular accident after Greenfield filter placement for paradoxical embolism. J Vasc Surg 1989;9:452-4.
3. Cohnheim J. Thrombose und embolie. In: Vorlesungen uber allgemeine pathologie. Vol 1. Berlin: Hirschwald, 1877:134.
4. Biller J, Johnson MR, Adams HP Jr, Kerber RE, Corbett JJ, Bruno A, et al. Further observations on cerebral or retinal ischemia in patients with right-left intracardiac shunts. Arch Neurol 1987;44:740-3.
5. Ward R, Jones D, Haponik EF. Paradoxical embolism: an underrecognized problem. Chest 1995;108:549-58.
6. Stollberger C, Slany J, Schuster I, Leitner H, Winkler W, Karnik R. The prevalence of deep venous thrombosis in patients with suspected paradoxical embolism. Ann Intern Med 1993;119:461-5.
7. Jungbluth A, Erbel R, Darius H, Rumpelt H, Meyer J. Paradoxical coronary embolism: case report and review of the literature. Am Heart J 1988;116:879-85.
8. Pell ACH, Hughes D, Keating J, Christie J, Busuttil A, Sutherland GR. Brief report: fulminating fat embolism syndrome caused by paradoxical embolism through a patent foramen ovale. N Engl J Med 1993;329:926-9.
9. Dahl-Iverson E. Embolie paradoxale de l'artere iliaque externe gauche-embolectomie. Lyon Cir 1930;1:39-42.
10. Marquez J, Sladen A, Gendaell H, Boehnke M, Mendelow H. Paradoxical cerebral air embolism without an intracardiac defect. J Neurosurg 1981;55:997-1000.
11. Gronert GA, Messick JM, Cucchiara RF, Michenfelder JD. Paradoxical air embolism from a patent foramen ovale. Anesthesiology 1979;50:548-9.
12. Chaikof EL, Campbell BE, Smith RB. Paradoxical embolism and acute arterial occlusion: rare or unsuspected? J Vasc Surg 1994;20:377-84.
13. Loscalzo J. Paradoxical embolism: clinical presentation, diagnostic strategies, and therapeutic options. Am Heart J 1986;112:141-5.
14. Hunter DD. Pulmonary arteriovenous malformation: an unusual case of cerebral embolism. Can Med Assoc J 1965;93:662-5.
15. Bridger JE. Aortocaval fistula: a rare cause of paradoxical pulmonary embolism. Postgrad Med J 1994;70:122-3.
16. Wilmshurst PT, deBelder MA. Patent foramen ovale in adult life. Br Heart J 1994;71:209-12.
17. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc 1984;59:17-20.
18. Zeiro C, Canterin FA, Pavan D, Nicolosi GL. Spontaneous closure of a patent foramen ovale and disappearance of impending paradoxical embolism after fibrinolytic therapy in the course of massive pulmonary embolism. Am J Cardiol 1995;76:422-4.
19. Hanna JP, Sun JP, Furlan AJ, Stewart WJ, Sila CA, Tan M. Patent foramen ovale and brain infarct: echocardiographic predictors, recurrence and prevention. Stroke 1994;25:782-6.

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