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Bilateral Top of Carotid Occlusion Presenting as Basilar Thrombosis

Bernardo Liberato, Noam Y. Harel, John W. Krakauer

Neurological Institute, Columbia University, New York, N.Y., USA

Introduction

Acute bilateral internal carotid artery (ICA) occlusion is a rare event. It can be seen as a complication of advanced atherosclerotic disease or from distal embolization from a proximal source. The presentation can be dramatic, especially in poorly collateralized patients with embolic occlusion, and can consist of coma and tetraplegia mimicking a basilar artery obstruction. We present a case of a patient who developed acute onset of coma, tetraplegia and loss of brainstem reflexes early in the course of bilateral distal ICA occlusion caused by massive embolization from a cardiogenic source.

Case Report

A right-handed 47-year-old man with AIDS was admitted to the hospital for malfunction of a left ventricular assist device (LVAD). He had been well on antiretroviral therapy, until 6 months prior to presentation when an idiopathic dilated cardiomyopathy was diagnosed. A rapidly progressive deterioration in his cardiac function led to placement of an LVAD (inflow tract in the left ventricle) 4 months later, and the patient was maintained on warfarin, aspirin and clopidogrel on the waiting list for a heart transplantation.

Two months later, the patient was admitted to the hospital for pump failure. An intra-LVAD thrombus was suspected despite his antiplatelet and anticoagulation regimen as described above. Repetitive transthoracic echocardiograms failed to reveal an intracardiac thrombus but because of progressive pump malfunction with hemodynamic compromise, the patient was started on a schedule of intermittent intravenous thrombolysis with alteplase (doses ranging from 50 to 100 mg) as well as intravenous heparin. Two weeks after admission, 1 h after the last scheduled dose of alteplase (50 mg), he became unresponsive and developed clonic movements, without any significant change in his systolic blood pressure or oxygen saturation. This was initially interpreted as generalized tonic-clonic seizures. The patient was loaded with intravenous phosphenytoin (20 mg/kg) and was started on a midazolam drip (after endotracheal intubation) for persistent clonic movements. He was continued on the midazolam drip (titrated to control 'seizure' activity) with little improvement in the clonic movements but with progressive hemodynamic instability requiring pressors. The initial neurological examination within 3 h of onset was remarkable for asynchronous clonic movements of facial muscles and all extremities compatible with multifocal myoclonus in the setting of coma. Small (2 mm) poorly reactive pupils, absent corneal reflexes, absent oculocephalic reflexes and flaccid tetraplegia were also noted. An initial CT scan done within 3 h of onset did not reveal signs of intracranial hemorrhage or brain edema, and the brainstem appeared normal. A presumptive diagnosis of basilar thrombosis was made and the patient was scheduled for an emergent angiogram as soon as his tenuous hemodynamic status improved. After 2 h, when the patient had his hemodynamic status stabilized, a repeat neurological evaluation was done which revealed the same findings as above plus the absence of oculovestibular reflexes on cold water irrigation, but a preserved respiratory drive and gag reflex. An angiogram was done at approximately 7 h from onset which revealed bilateral top of the ICA occlusion with filling of the right and left anterior cerebral arteries through the anterior communicating artery on right ICA injection, no filling of either middle cerebral arteries but a fully patent posterior circulation. Both ophthalmic arteries showed complete anterograde filling from the ipsilateral ICA injection, and there was a fetal origin left posterior cerebral artery which also filled well from a left ICA injection (fig. 1a, b). No endovascular intervention was attempted given the time window and the devastating clinical picture. The patient had persistent brainstem dysfunction on repeat neurological examination and expired 1 day later from progressive hemodynamic failure from a persistently malfunctioning LVAD. An autopsy was performed which revealed a thrombus in the outflow tract of the LVAD, bilateral watershed territory infarcts, bilateral ICA occlusions, diffuse brain edema and uncal herniation. Duret's hemorrhages were identified in the pons and midbrain, compatible with uncal herniation, but no evidence of discrete brainstem infarction was seen. There was no evidence of atherosclerosis in either carotid artery.

Discussion

We describe a case of bilateral top of the ICA occlusion, caused by emboli from a partially lysed intra-LVAD thrombus, with prominent brainstem findings early in the clinical presentation that led to an initial diagnosis of basilar thrombosis. In addition, the bilateral myoclonic movements were also thought to be compatible with a basilar artery occlusion [1].

Acute bilateral ICA distribution strokes are uncommon, and in a series of first ischemic strokes in the anterior circulation distribution, Bogousslavsky et al. [2] reported that approximately 1.3% had acute bilateral hemispheric infarcts, with a significant number of these having a cardioembolic etiology. More recently, Kwon et al. [3] reported a series of 6 patients with acute bilateral ICA occlusion who presented with tetraplegia and coma suggesting basilar artery thrombosis. In their series, the patients had bilateral motor involvement with coma, but all had preserved brainstem reflexes within 3 h of presentation. All of the patients lost brainstem reflexes within 1 day (but after 3 h) and died within 3 days of onset. This observation is in accordance with Fisher's description of bilateral ICA

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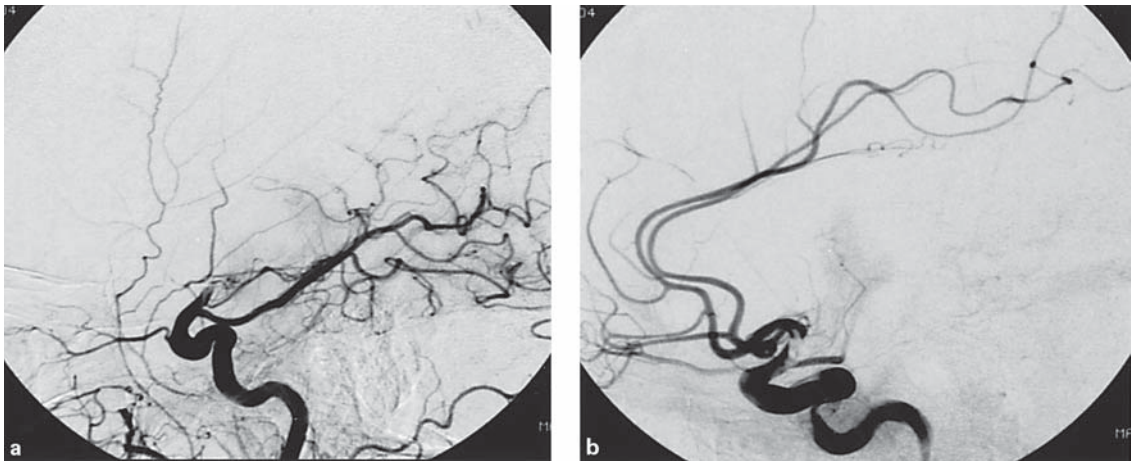

Fig. 1. Angiographic lateral views of both ICAs. **a** Left ICA injection showing embolus to distal ICA with complete occlusion of the middle cerebral artery and anterior cerebral artery origins. **b** Right ICA injection revealing occlusion at the right middle cerebral artery origin and decreased flow in both anterior cerebral arteries.

occlusion mimicking acute basilar artery thrombosis [4]. Our patient differs from the previous descriptions given the early (less than 3 h) loss of most brainstem reflexes. This finding is not explained by brainstem distortion (given the lack of brainstem involvement by early imaging) or by brainstem infarction (given the autopsy results).

Absent oculocephalic and oculovestibular reflexes can be observed in cases of toxic-metabolic coma, such as drug overdose (barbiturates, tricyclics) or hypothermia. The combination of midazolam and phenytoin may, at least in part, have contributed to the complete ophthalmoplegia observed in this patient. Spector at al. [5] have reported a series of patients who developed complete external ophthalmoplegia after excessive doses of phenytoin had been administered. No level of anticonvulsant was available for our patient so no correlation could be made regarding toxic serum level and degree of ophthalmoplegia. However, the dosing was strictly weight based, and with his normal liver and renal functions there was no reason to suspect toxic levels were achieved. The absent pupillary and corneal reflexes could also have been a consequence of CNS-depressant agents. Midazolam when used for anesthesia causes dose-dependent depressive effects on the corneal reflex [6], an effect also seen with excessive doses of phenytoin [5]. Although a drug effect on the brainstem in combination with bilateral cortical infarction may explain our findings, this case does raise the issue of whether massive cortical infarction can lead to acute deafferentation of the brainstem. Transient loss of all brainstem reflexes is described in postanoxia and can reverse [7], suggesting that physiological brainstem dysfunction can occur in the absence of tissue damage.

Conclusion

This case illustrates the catastrophic presentation of acute bilateral ICA occlusion and a clinical scenario that led to the pre-angiogram diagnosis of basilar thrombosis. A number of important issues are raised by this case. First, although evidence supports the use of intermittent intravenous thrombolysis for prosthetic valvular thrombosis [8], there is increased risk of distal embolization. Second, medications plus a structural lesion may mimic another structural lesion and lead to erroneous localization. Third, a constant awareness of the possibility of simultaneous bilateral ICA occlusion is important considering the therapeutic possibilities of modern interventional neuroradiological techniques and the possible differences regarding timing of anterior and posterior circulation angiographic intervention.

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John W. Krakauer, MD
Department of Neurology – Columbia University
710 West 168th Street, Room 640, New York, NY 10032 (USA)
Tel. +1 212 305 1710, Fax +1 212 305 1658
E-Mail jwk18@columbia.edu