Occlusion of the Middle Cerebral Artery Due to Cysticercotic Angiitis

Jesus Rodriguez-Carbajal, MD, Oscar H. Del Brutto, MD, Pedro Penagos, MD, Juan Huebe, MD, and Alfonso Escobar, MD

Three patients with cysticercosis developed a cerebral infarct secondary to the occlusion of the middle cerebral artery or its major branches. Histopathologic examination revealed a large subarachnoid cysticercus surrounding the occluded arteries in two patients and diffuse thickening of the leptomeninges in one. Blood vessels around the parasite showed inflammatory changes that caused either occlusive endarteritis or thrombosis due to disruption of the endothelium. Cysticercosis should be considered as a cause of occlusion of the major intracranial vessels, particularly in young patients living in areas where this disease is endemic. (Stroke 1989;20:1095-1099)

Angiitis of the intracranial vessels is a common complication of several infectious diseases of the central nervous system (CNS) and greatly contributes to the morbidity and mortality of these disorders. Etiologic considerations for the syndrome of infectious angiitis usually include viral, purulent, tuberculous, and syphilitic meningitis. Cysticercosis, the most common parasitic disease of the CNS, has also been associated with intracranial angiitis. In most cases, however, the involved vessels are of small diameter and the neurologic complications that such involvement produces are limited to lacunar syndromes secondary to small cerebral infarcts. This report documents the occurrence of large cerebral infarcts due to occlusion of the middle cerebral artery (MCA) or its major branches in patients with neurocysticercosis.

Case Reports

Case 1

A 23-year-old right-handed woman was evaluated 3 days after the acute onset of headache, vomiting, weakness of her right arm and leg, and the inability to speak. On admission, her blood pressure was 120/70 mm Hg and the results of a general physical examination were normal. Neurologic examination showed an alert patient with motor dysphasia. Cranial nerve function, including pupillary responses and ocular movements, was normal. There was a right hemiparesis and a right Babinski’s sign. The rest of the findings were unremarkable.

Computed tomograms (CT scans) showed a hypodense ring-enhancing lesion in the left sylvian fissure and an area of decreased attenuation in the left basal ganglia (Figure 1). A left carotid angiogram showed occlusion of a major branch of the MCA (Figure 2). A lumbar puncture yielded clear cerebrospinal fluid (CSF) under normal opening pressure; cytochemical analysis revealed 41 mononuclear cells/mm³, 32 mg protein/dl, and 55 mg glucose/dl. Immunologic reactions for the detection of anticysticercus antibodies (enzyme-linked immunosorbent assay and complement fixation test) were negative in the CSF. An open biopsy of the lesion was planned, but the patient experienced sudden deterioration in her neurologic status and died a few days later.

Postmortem examination was confined to the brain and revealed a large encapsulated cysticercus in the left sylvian fissure and an area of infarction in the left putamen and internal capsule (Figure 3). Several branches of the left MCA, including the lenticulostriate, leptomeningeal, and cortical arteries, were occluded by large atheroma-like deposits, and their walls were invaded by an intense inflammatory infiltrate (Figure 4).

Case 2

A 23-year-old right-handed woman was evaluated 1 month after the acute onset of left-sided weakness. She had a history of progressive headache and vomiting for 3 months. On admission, her blood pressure was 110/70 mm Hg and the results of a general physical examination were normal. Neurologic examination showed an alert patient with
bilateral papilledema. Her pupils were 3 mm, round, and reactive; her ocular movements were full. She had a mild left hemiparesis, increased muscle stretch reflexes on the left, and a left extensor plantar response. The rest of the findings were unremarkable.

CT scan showed a hypodense ring-enhancing lesion in the right sylvian fissure and an area of decreased attenuation in the ipsilateral corona radiata and body of the caudate nucleus. A right carotid angiogram showed occlusion of the proximal segment of the MCA. An open biopsy was performed, with removal of a cystic lesion from the right sylvian fissure. Histopathologic examination revealed a large hyalinized cysticercus surrounded by an intense inflammatory infiltrate and occlusion of the leptomeningeal vessels in the vicinity of the cyst, with associated fibrinoid necrosis of the vessel wall.

The patient was treated with prednisone and was discharged with a left hemiparesis that persisted unchanged for 8 months of follow-up.

**Case 3**

A 38-year-old right-handed man complained of progressive headache for 1 year. One month before admission, he experienced several episodes of generalized tonic-clonic seizures that were controlled with phenytoin therapy. After this bout of seizures, he noticed severe weakness of the left side of his body that persisted until admission to the hospital. His blood pressure was 120/90 mm Hg, and the results of a general physical examination were normal. Neurologic examination showed bilateral papilledema, a left spastic hemiparesis with brachial dominance, and decreased pin and temperature sensation on the left.

A right carotid angiogram showed forward and upward displacement of the anterior cerebral artery and occlusion of the proximal segment of the MCA. A ventricular shunt was placed for relief of the hydrocephalus. Ventricular CSF examination revealed 29 mononuclear cells/mm³, 42 mg protein/dl, and 49 mg glucose/dl; Nieto’s complement fixation test for the detection of anticysticercus antibodies was strongly positive. The patient presented systemic complications after surgery and died a few days later.

Postmortem examination was confined to the brain and revealed diffuse thickening of the leptomeninges at the base of the skull, with entrapment of blood vessels arising from the circle of Willis; some parasitic membranes were identified within this leptomeningeal thickening. The right MCA was occluded, with thickening of the adventitia and severe endothelial hyperplasia. There was also an...
Discussion

Cysticercosis occurs when humans become the intermediate host in the life cycle of the tapeworm *Taenia solium* by ingesting its eggs from contaminated food. Once in the human stomach, eggs hatch into oncospheres by action of the gastric juices. Oncospheres cross the intestinal wall, enter the bloodstream, and are carried into the tissues of the host where the larvae (cysticerci) develop. One of the main target organs of cysticerci is the CNS, where parasites may lodge in the brain parenchyma,

old infarct in the right basal ganglia and subcortical white matter.

FIGURE 2. Case 1. Left carotid angiogram showing avascular area in territory of middle cerebral artery.

FIGURE 3. Case 1. Macroscopic brain slices showing (left) large cysticercus in left sylvian fissure (solid arrow) and (right) infarct in left basal ganglia (open arrow).
Subarachnoid cysticerci may be scattered over the convexity of the cerebral hemispheres or may form large clumps of cysts within the cisterns of the CSF. Frequently, abnormal thickening of the leptomeninges occurs at the base of the skull, secondary to an inflammatory exudate composed of collagen fibers, hyalinized parasitic membranes, lymphocytes, plasma cells, eosinophils, and multinucleated giant cells. Blood vessels arising from the circle of Willis are usually entrapped within this dense exudate, with subsequent invasion of the vessel wall by inflammatory cells, leading to endarteritis with thickening of the adventitia, fibrosis of the media, and endothelial hyperplasia. A cerebral infarct secondary to the occlusion of a blood vessel may be the final result of this vascular involvement.

Occlusion of the major intracranial vessels has been reported as an unusual complication of subarachnoid cysticercosis. In one of these cases, a clump of cysticerci was found surrounding a thrombosed internal carotid artery. Similar findings were noticed in two of our patients in whom a large cysticercus in the sylvian fissure induced segmental arteritis in the proximal segment of the ipsilateral MCA and its major branches (Figures 3 and 4). In the third patient, no macroscopic cyst was identified and the occlusion of the MCA was explained on
FIGURE 5. Case 3. Right carotid angiogram showing upward and forward displacement of anterior cerebral artery and occlusion of proximal segment of middle cerebral artery.

the basis of a dense inflammatory exudate of cysticercotic origin within the cisterns of the CSF. Our cases provide further evidence that cysticercosis is a possible cause of ischemic cerebrovascular disease and that it should be considered in the differential diagnosis of patients with angiographic evidence of occlusion of the major intracranial vessels, particularly when the patients come from areas of the world where this disease is endemic.

References

KEY WORDS • cerebral infarction • cysticercosis • vasculitis
Occlusion of the middle cerebral artery due to cysticercotic angiitis.
J Rodriguez-Carbajal, O H Del Brutto, P Penagos, J Huebe and A Escobar

Stroke. 1989;20:1095-1099
doi: 10.1161/01.STR.20.8.1095

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/20/8/1095

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/