Middle Cerebral Artery Occlusion Due to Hydatid Cysts of Myocardial and Intraventricular Cavity Cardiac Origin

Two Cases

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Background

Hemispheric strokes of hydatid origin are very rare. We describe two cases of middle cerebral artery occlusion by a cyst of cardiac origin.

Case Descriptions

Cerebral angiography demonstrated occlusion of the initial segment of the middle cerebral artery. Myocardial and pericardial cysts were diagnosed by echography and pathological examination. Subsequent development of hydatid cysts within the necrotic area of the infarcted hemisphere suggests cerebral hydatid embolism of cardiac origin.

Conclusions

In endemic areas, embolism of hydatid cysts should be considered in the diagnosis of stroke in young patients. (Stroke. 1994;25:886-888.)

Key Words

• echinococcosis • embolism • young adults

Although hydatid cysts are common, particularly in Morocco, where hydatidosis is endemic, only three cases involving large blood vessels have been reported. We describe two cases in which occlusion of the middle cerebral artery (MCA) was secondary to embolism of myocardial cysts, with subsequent development of cerebral cysts in the region of the infarct.

Case Reports

Case 1

A 21-year-old woman, without significant medical history, acutely developed a left hemiparesis with left hypoesthesia. Three days earlier she had complained of itching with nettle rash, headache, and fever (38°C). A noncontrast computed tomographic (CT) scan (Fig 1A) revealed a right MCA infarct. Cerebral angiography showed an abrupt cutoff of the right proximal M1 segment, which was compatible with embolism (Fig 1B). There was no evidence of other occlusions or vasculitis. Cerebrospinal fluid and blood tests (including white blood cell count) were normal. Hemiparesis improved progressively over 3 months. Nine months later intracranial hypertension developed, and hemiparesis worsened. CT scans showed a cyst in the territory of the right MCA infarct (Fig 1C). Hydatid cysts were also found in the pericardium (Fig 2A) and kidneys, liver, and spleen (Fig 2B). Subsequently, cysts also developed in the brain stem. Cysts in the heart (left atrial cavity, ventricular myocardium, and pericardium) and the brain (right temporal lobe) were surgically removed. Scolecis and hooks of Echinococcus were found in the cyst fluid. Two years later a new cyst in the pons was removed. The patient died shortly thereafter; autopsy was not performed.

Case 2

A 40-year-old woman, without significant medical history, acutely developed a right hemiparesis with aphasia. A noncontrast CT scan revealed a left MCA infarct (Fig 3A). Cerebral angiography showed an abrupt cutoff of the left proximal M1 segment (Fig 3B). Cerebrospinal fluid and extensive blood tests were normal, showing no eosinophilia. A fluorescent antibody test for hydatidosis was positive in serum (1/80). A two-dimensional transthoracic echocardiogram showed two liquid-containing circular masses in the myocardium protruding in the ventricular cavity (posterolateral part of the left ventricle). Hydatid cysts were also found in kidneys, liver, and spleen. The patient was treated with mebendazole 2500 mg/d for 3 months. After 1 year no clinical change was observed, but a CT scan showed two cystic lesions, one of which was in the previously infarcted zone (Fig 3C).

Discussion

Estimates of the incidence of hydatid cysts in the myocardium or pericardium range from 0.3% to 3.3%. Cysts in the central nervous system are found in approximately 2% of infected patients, frequently in association with systemic dissemination, particularly to the liver (65%) and lungs (25%); however, isolated involvement of the brain or spinal cord has also been observed, especially in children. The usual manifestation of an intracranial hydatid cyst is intracranial hypertension, which may be associated with hemiparesis, seizures, visual disturbances, or altered levels of consciousness.
Only three cases of large-vessel occlusion in hydatidosis have been reported, in one case after cardiac hydatid surgery.

The neurological symptoms of our patients suggested acute occlusion of the M1 segment of the MCA. The abrupt cutoff of the M1 segment and the absence of vasculitis suggested vascular occlusion secondary to embolism. Laboratory studies and angiography disclosed no risk factors for vascular occlusion other than hydatidosis. The fact that hydatid cysts subsequently developed within the MCA cerebral infarct suggests that rupture of the cardiac cysts into the ventricular cavity may directly result in embolism of the cerebral arteries. Cardiac echinococcosis is rare (less than 2%). Cysts can lodge on tricuspid valves or in the left ventricular myocardium, septum, or atrium. The clinical picture is dependent on the location of the cyst, but the cyst can be discovered incidentally. In every case with documented hydatidosis, even in the absence of cardiac complaints, two-dimensional echocardiography is a reliable method to detect those cysts. Because of the unpredictability of the cyst rupture and the severity of the embolisms, early diagnosis and surgical extirpation of the lesions should be recommended. In our observation, the presence of a cyst in the MCA territory infarct and multiple cysts in the kidney and liver supports the hypothesis of rupture (in case 2, with presence of the cysts in the myocardium close to the ventricular cavity) and embolization (in case 1, with a left atrial cyst, and in case 2) of the cardiac cysts. However, the multiple localizations could also be related to systemic dissemination of the parasite, which in our observations is less probable.

In conclusion, hydatidosis is the most common parasitic disease of the central nervous system. Stroke is an infrequent complication, but hydatid cyst embolism should be considered in the diagnosis of stroke in young patients, particularly in geographic locations where the disease is endemic.

Acknowledgments

The authors are grateful to Pr Najat Boukhrissi and Drs Jidanne Mohamed, Ohayon Victor, and Hda Hamid for per-
forming the radiological investigations; Drs Ait M'Barek Abdellah, Hanae Benomar Benabdallah, and Hamid Ouhabi for providing valuable information; and Dr Merle Ruberg for critical reading of the manuscript.

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Stroke. 1994;25:886-888
doi: 10.1161/01.STR.25.4.886

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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