Internal Carotid Artery Thrombus: An Underdiagnosed Source of Brain Emboli in Neonates?

ABSTRACT

We report a full-term neonate with a left middle cerebral artery infarct, narrowing of the internal carotid artery detected by magnetic resonance angiography and B-mode ultrasonography, and a large thrombus at the origin of the internal carotid artery detected by B-mode ultrasonography. Internal carotid arterial thrombus is seldom considered the source of middle cerebral arterial embolus in neonates. We suggest that B-mode ultrasonography of the carotid artery be included in the diagnostic evaluation of middle cerebral artery infarcts in neonates. (*J Child Neurol* 2001;16:446–447).

Most focal arterial ischemic infarcts in full-term neonates involve the middle cerebral artery distribution. The cause of middle cerebral artery infarcts in full-term neonates is often not found despite an exhaustive investigation. This investigation includes coagulation studies, lupus erythematous panel, anticardiolipin antibody level, antiphospholipid antibodies, cardiac ultrasound, placental examination, and brain imaging studies.[1,2] B-mode ultrasonography of the carotid arteries is seldom performed in the evaluation of neonates with middle cerebral artery infarct. We present a neonate with a left middle cerebral artery infarct and internal carotid artery pathology detected by magnetic resonance angiography and B-mode ultrasonography.

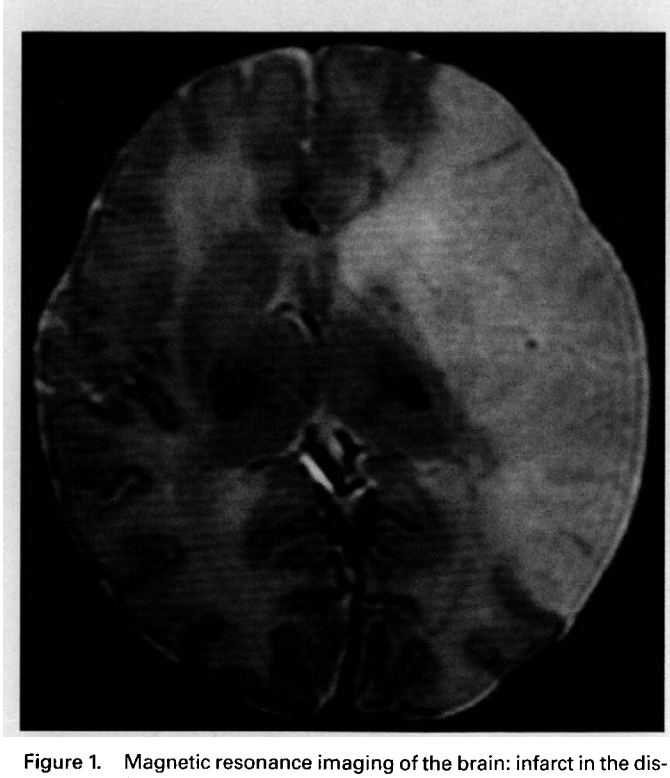

Figure 1. Magnetic resonance imaging of the brain: infarct in the distribution of the left middle cerebral artery.

Case Report

A 3-day-old boy was transferred to Miami Children's Hospital with the diagnosis of seizures and hypotonia. He was the product of an uncomplicated pregnancy and delivery, except for maternal fever during the last week of pregnancy. Apgar scores were 9 and 9 at 1 and 5 minutes. Birthweight was 3.65 kg. Head circumference was 35.5 cm. General examination was normal. Blood cultures were done, and the patient was started on intravenous ampicillin and gentamicin.

At 3 days of age, he had several episodes of arching of the trunk, repetitive limbs movements, and sustained left lateral gaze. Physical examination revealed a hypoactive neonate with a full anterior fontanelle, no rooting reflex, minimal sucking reflex, hypotonia, and poor grasp. The rest of the examination was normal. Laboratory investigation revealed normal serum glucose, calcium, and electrolytes. An unenhanced computed tomography (CT) of the brain revealed a large left-hemisphere infarct. Phenobarbital and fosphenytoin were started because of suspected frequent seizures.

The initial physical examination at Miami Children's Hospital revealed a patient in no acute distress. Head circumference was 35.5 cm. The anterior fontanelle was full. The rest of the general examination was normal except for a grade III/IV systolic ejection murmur. The neurologic examination was remarkable for a depressed level of alertness, poor sucking, and generalized hypotonia. The laboratory investigation revealed a platelet count of 677,000/μL. White and red blood cell counts were normal. A coagulation panel consisting of protein C and S, antithrombin III, plasminogen, and factor V Leiden was normal. A lupus erythematous panel, anticardiolipin antibody levels, antiphospholipid antibodies, cardiac ultrasound, and placental examination did not reveal any abnormality. Phenobarbital and phenytoin levels were in the therapeutic range. On arrival, the patient was placed on continuous-display four-channel electroencephalographic (EEG) monitoring. Continuous video EEG telemetry was started several hours later. Phenobarbital

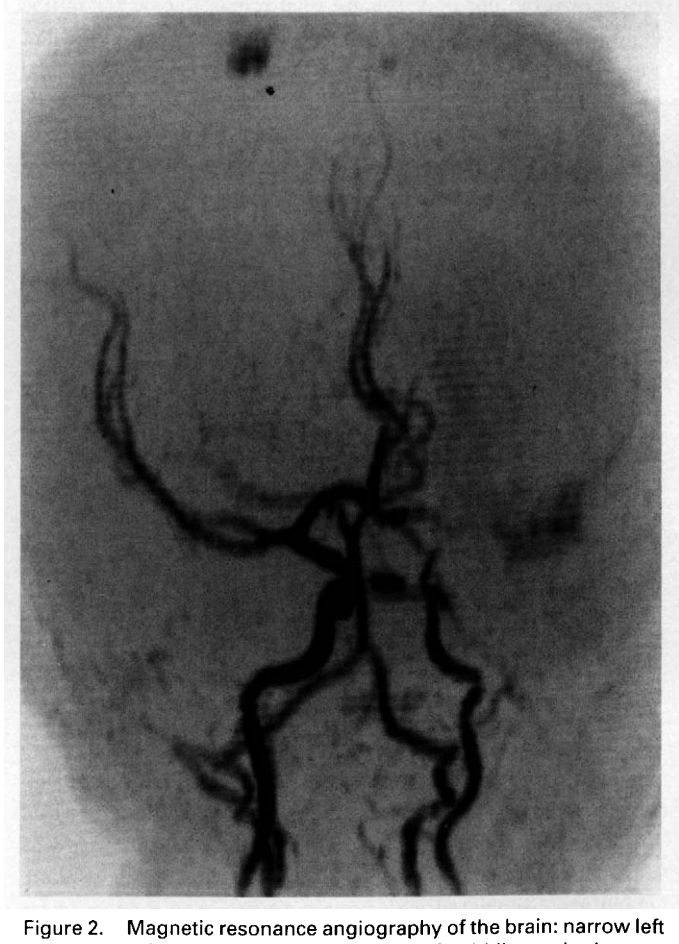

Figure 2. Magnetic resonance angiography of the brain: narrow left internal carotid artery and absence of the left middle cerebral artery.

Figure 3. B-mode ultrasonography: partially calcified thrombus at the proximal segment of the internal carotid artery and narrowing of the internal carotid artery lumen.

and fosphenytoin were stopped after no EEG seizures had occurred in 3 days. Antibiotics were stopped once blood cultures were negative.

A magnetic resonance imaging (MRI) of the brain confirmed the presence of a left middle cerebral artery infarct (Figure 1). Magnetic resonance angiography of the brain revealed narrowing of the left internal carotid artery and absence of the middle cerebral artery (Figure 2). A B-mode ultrasound of the left carotid artery revealed an echogenic thrombus and narrowing of the proximal portion of the internal carotid artery (Figure 3).

He began to improve after 5 days of age and was discharged at 18 days of age. At the time, his clinical examination was normal. The patient was lost to follow-up. Imaging studies were not repeated.

Discussion

Magnetic resonance imaging of the brain in this patient revealed an infarct in the distribution of the middle cerebral artery (see Figure 1). Middle cerebral artery infarcts may be due to generalized cerebral hypoperfusion[3–5] or middle cerebral artery pathology.[5,6] Middle cerebral artery pathology has been associated with spasm, dissecting aneurysm, in situ thrombus formation, or embolus from sources other than the internal carotid artery.[5,6]

In this patient, the previously mentioned conditions were excluded based on clinical, laboratory, and radiologic findings. Generalized cerebral hypoperfusion occurs during perinatal asphyxia.[3–5] Middle cerebral artery spasm occurs in neonates exposed to cocaine or prolonged systemic hypertension during pregnancy.[7,8] Dissecting aneurysm of the middle cerebral artery is related to trauma and has typical radiographic findings. In situ middle cerebral artery thrombosis occurs with birth trauma, polycythemia, meningitis, and coagulopathies.[3,9] Middle cerebral artery embolism occurs with placental infarcts, twin-to-twin transfusion syndrome, patent ductus arteriosus, congenital heart disease, sepsis, temporal artery catheterization, and mural or atrial myxoma.[3,8,10]

Three radiographic findings suggest that the cause of the infarct was the migration of material originating at the internal carotid artery. These radiologic findings are the narrowing of the internal carotid artery (see Figures 2 and 3), the absence of the middle cerebral artery (see Figure 2), and the echogenic thrombus at the proximal segment of the internal carotid artery (see Figure 3). B-mode ultrasonography demonstrated the thrombus at the proximal segment of the internal carotid artery and the narrowing of the internal carotid artery.

The lack of reports of internal carotid artery thrombus in neonates with middle cerebral artery infarct may reflect that such a lesion is rare or underdiagnosed. The cause for the thrombus in the internal carotid artery in this patient was not found.

We suggest that B-mode ultrasonography of the carotid arteries be included in the evaluation of middle cerebral artery infarcts in neonates.

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