

Acute Hemiplegia in Children Complicating Upper Respiratory Infections

Report of Three Cases with Angiographic Findings

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Three children presented with acute onset of hemiplegia following an upper respiratory infection. Angiography revealed irregularities, beading, and slow flow of a peripheral branch of a middle cerebral artery. In addition, one child had narrowing of the cervical segment of the internal carotid artery. An inflammatory arteritis of the cervical internal carotid artery is presumably the site of the formation of cerebral emboli.

ACUTE ONSET OF HEMIPLEGIA in children associated with upper respiratory infection, followed by angiographic demonstration of cerebral artery involvement, seems worthy of being reported. We here present three children, all under (8) years of age, who were hospitalized at the Nassau County Medical Center with acute hemiplegia which began during an upper respiratory infection. Angiographic studies were performed two to 18 days after admission.

Case Reports

Case 1

This seven-year-old boy was well until six days earlier when he developed malaise, cough, and low fever. He was given erythromycin and aspirin. On the next day, he had a generalized tonic-clonic

seizure and was given phenobarbital and dilantin. Over the next two days, he complained of occipital headache and became progressively more lethargic before having a second generalized convulsion. He was taken to the emergency room of a local hospital where a lumbar puncture was performed and the findings interpreted as normal. He remained lethargic and two days later was admitted to the Nassau County Medical Center.

On arrival, he was lethargic but awake and fully oriented. He had left hemiparesis and some decrease of position sensation on the left side of the body. Funduscopic revealed blurring of the temporal margin of the right optic disc. The reflexes were normal. There was no evidence of pharyngitis or otitis.

Blood pressure was 110/70 mm; pulse 110 per minute; respirations 20 per minute; temperature 101 F. His white blood cell count was 12,100 with 74 per cent PMNs. Lumbar puncture after intravenous mannitol revealed an opening pressure of 160 mm of water, protein 14 mg/100 ml, glucose 140 mg/100 ml, 8 red blood cells per cu mm, and numerous lymphocytes. Cultures of cerebrospinal fluid (CSF), throat, nasopharynx, and rectum were negative. An EEG revealed generalized slowing, with high-voltage delta waves predominantly on the right side and posteriorly. Brain scans were normal on admission and on the fifth and 17th

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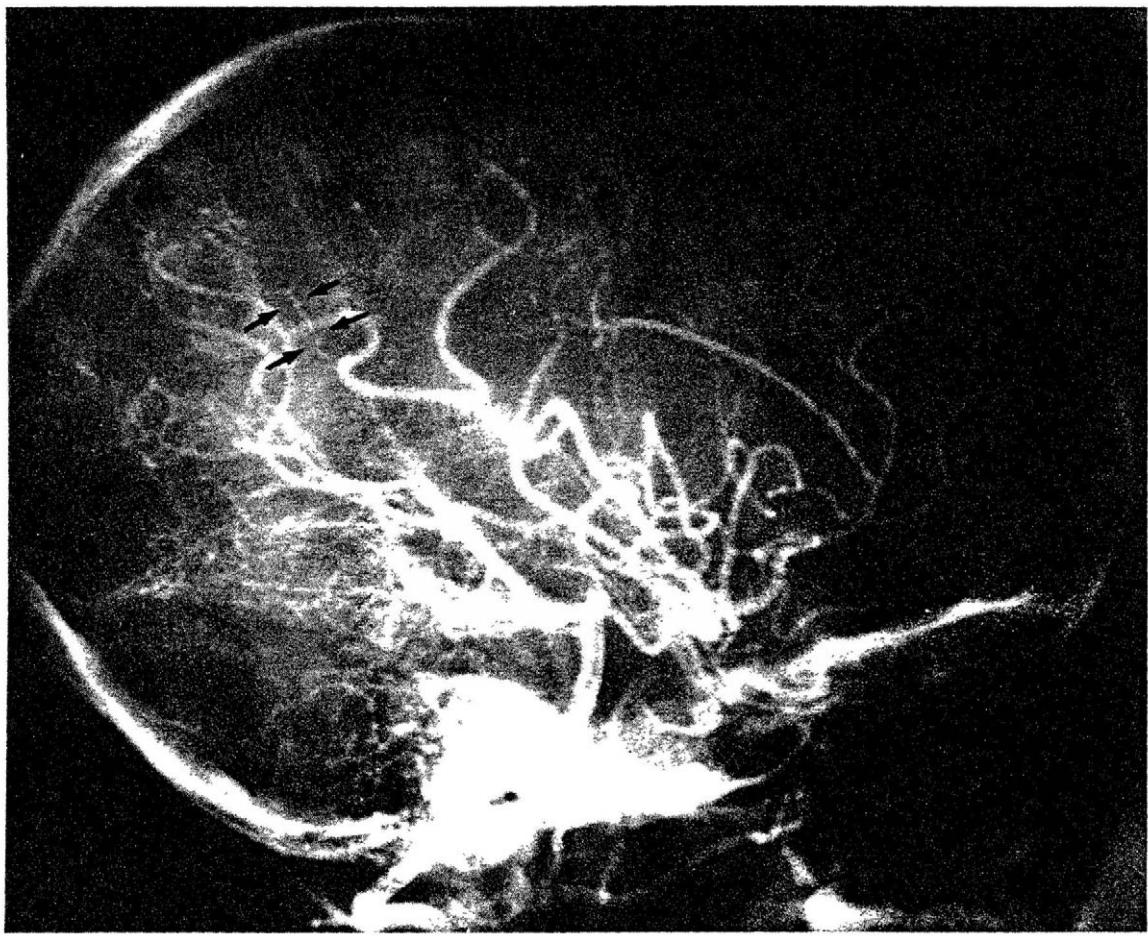


FIG. 1. Case 1. Right retrograde brachial angiogram, lateral view. Early arterial phase demonstrating irregularities and beading involving a small parietal branch of the middle cerebral artery (arrows). Earlier views showed a normal internal carotid artery.

days. Ampicillin, mannitol, and dilantin were started. In 24 hours, his consciousness improved as did his hemiparesis. The ampicillin was continued for ten days.

A right brachial angiogram, on the 18th day of hospitalization, revealed irregularities and slow flow in a small parietal branch of the middle cerebral artery (Fig. 1). There was no evidence of space-occupying mass or displacement of the midline structures.

He was discharged three weeks after admission with mild left hemiparesis. Follow-up examination two months later revealed residual motor deficit on the left side. Some change in behavior pattern toward hyperactivity was also noted. Seizures did not recur.

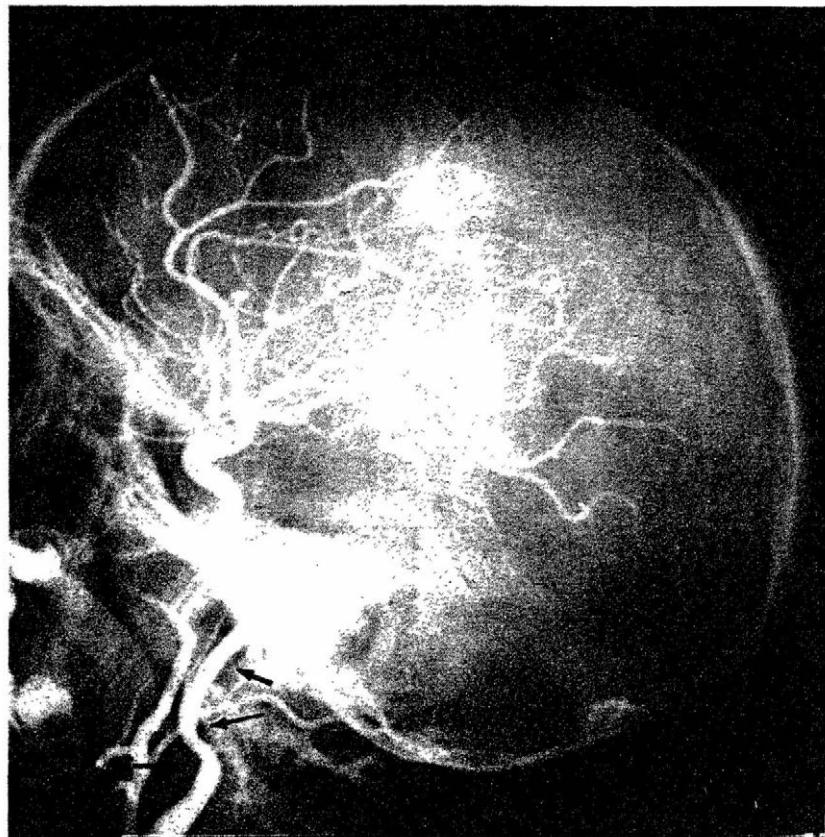
Case 2

This six-year-old girl was hospitalized because of right-sided earache, headache, vomiting for four days, and the onset of diplopia.

On arrival, she was alert and afebrile. Her throat was congested and the tonsils and anterior cervical lymph nodes were enlarged. Both tympanic membranes were dull and the right was bulging and inflamed. Neurologic examination revealed right hemiparesis, right facial weakness, and bilateral sixth nerve palsies with the right more involved than the left. On fundoscopy, she had blurring of both optic discs. Reflexes were normal, and her neck was supple. Her blood pressure and vital signs were normal. Her WBC count was 11,600 with 80 per cent PMNs, 13 per cent lymphocytes, 6 per cent monocytes, and 1 per cent basophiles. A lumbar puncture performed after intravenous mannitol showed an opening pressure of 400 mm of water, protein 14 mg/100 ml, glucose 58 mg/100 ml, 2 red blood cells per cu mm, and no white blood cells. Cultures of CSF fluid, throat, ear swabs, urine, and rectum were negative. An EEG showed a bilateral posterior slow-wave focus, more marked on the right. Brain scans were

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FIG. 2A. Case 2. Left carotid angiogram, lateral view. Early arterial phase showing irregular narrowing of the distal three centimeters of the cervical internal carotid artery (arrows).



normal one week, four weeks, and six weeks after admission.

Cerebral angiography on the second day of hospitalization revealed irregular narrowing of the distal cervical portion of the left internal carotid artery. There were irregularities and loss of the normal undulations of an opercular branch of the middle cerebral artery. No mass or displacement of the midline structures was associated (Figs. 2A and 2B).

The child was given penicillin and chloramphenicol for four weeks. After the WBC and platelet count fell, these antibiotics were replaced by ampicillin for an additional two weeks.

A repeat left carotid angiogram 5½ weeks after admission revealed a normal internal carotid artery, but the changes in the small opercular branch of the middle cerebral artery persisted.

The child slowly improved. When discharged six weeks after admission, only mild right hemiparesis and minimal right sixth nerve palsy remained. Follow-up examinations showed slow resolution of her hemiparesis which had completely cleared 7½ months after discharge.

Case 3

This 14-month-old boy was well until three days prior to admission when he developed a running

nose and a low-grade fever, followed the next day by a nonproductive cough. On the morning of admission, the mother noticed that he was not moving his left arm and was unable to stand on his left leg, or walk. That same day, he had two focal seizures. The first seizure involved the left hand only and lasted five minutes. The second, five minutes later, involved the whole left side of the body and lasted 15 minutes. He was treated with phenobarbital and referred to Nassau County Medical Center.

On admission, the child was alert and responsive. The nasal mucosa was congested. The tonsils were enlarged and hyperemic with specks of white exudate. Anterior cervical lymph nodes were enlarged. The tympanic membranes were red and injected, especially on the right. Examination revealed left hemiparesis, left facial weakness, and left homonymous hemianopsia. The deep tendon reflexes were increased on the left. A Babinski sign was present on the same side. There was no papilledema. The neck was supple.

Blood pressure was 106/50 mm Hg; pulse 145; respirations 38; temperature 104 F. His WBC was 4,700 with 31 per cent PMNs, 64 per cent lymphocytes, and 5 per cent monocytes. His CSF had an opening pressure of 200 mm water, protein 29 mg/100 ml, glucose 100 mg/100 ml, and no

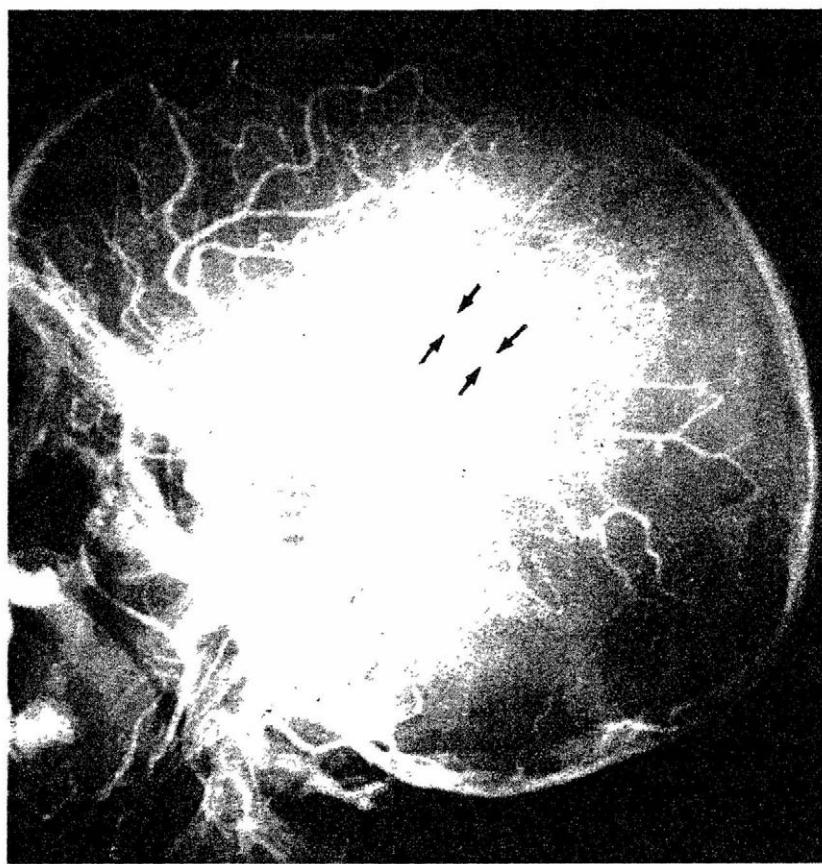


FIG. 2B. Case 2. Mid-arterial phase demonstrating irregularities and straightening involving a small opercular branch of the middle cerebral artery (arrows).

white blood cells. Cultures of CSF, ear canals, urine and rectum were negative. A myxovirus (Influenza A) was grown from the nasopharynx, associated with a significant rise in serum titers. On EEG he showed excess right-sided slowing with phase reversals in the posterior temporal derivations. Brain scans were normal on the second and seventh days of hospitalization.

Right carotid angiogram on the tenth day of hospitalization revealed irregular narrowing with decrease in the caliber of a posterior frontal opercular branch of the middle cerebral artery. There was no associated mass or displacement of the midline structures (Fig. 3).

He was treated initially with penicillin and chloramphenicol. These antibiotics were replaced by ampicillin on the tenth day when the white blood cell count dropped.

The left hemiparesis improved gradually. On discharge four weeks after admission, only mild weakness remained.

Discussion

The neurologic symptoms in these three children began acutely, three to six days after the onset of an upper respiratory infection.

Hemiplegia was present in all three children and seizures were seen in two. The upper respiratory infection was associated with enlarged tonsils in two children, cervical lymphadenopathy in two, and otitis media in two. In none of our cases was any evidence found of bacterial meningitis, brain abscess, intracranial space-occupying lesion, congenital or acquired heart disease, collagen vascular disease or blood dyscrasia. Radioisotope brain scanning was repeatedly negative.

With each child, angiography revealed irregularities, beading, and slow flow in a peripheral branch of a middle cerebral artery. In addition, one child had irregular narrowing of the distal portion of the cervical internal carotid artery, but repeat angiography 5½ weeks later revealed that this arterial segment had returned to normal. From case 3, the influenza A virus was isolated. The etiology in cases 1 and 2 was undetermined. Some degree of encephalitis was present in the three cases, accounting for

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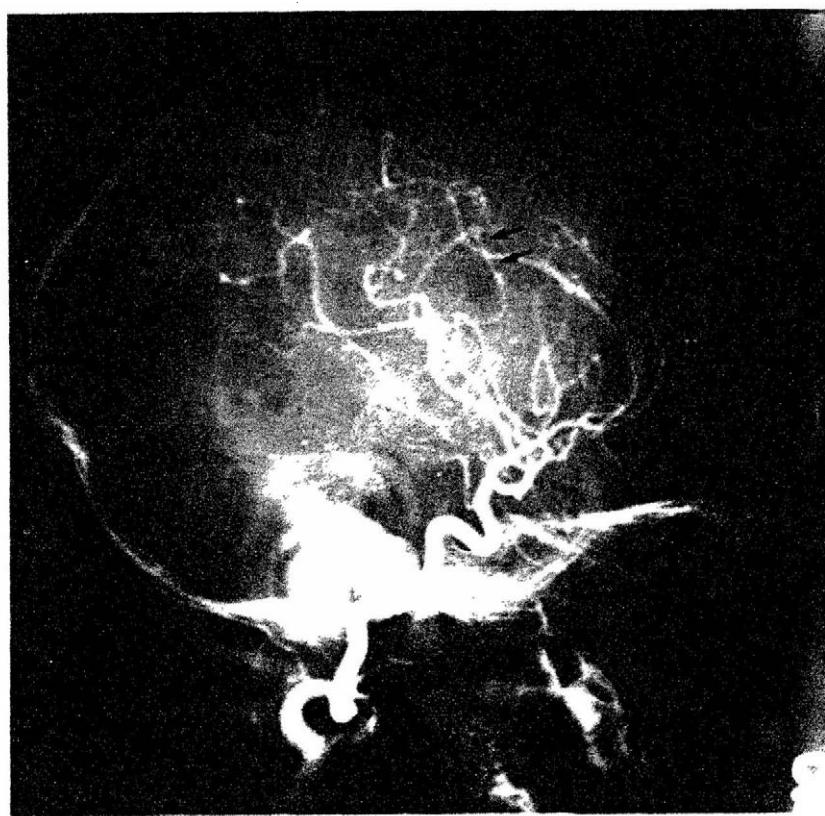


FIG. 3. Case 3. Right carotid angiogram. Lateral view. Arterial phase. There is an abrupt change in the caliber, and irregular narrowing involving a posterior frontal opercular branch of the middle cerebral artery (arrows).

the lymphocytes in the CSF of the first patient, and intracranial pressure rises in the other two patients.

It is highly unusual to find the cerebral arteries, whether extracranial or intracranial, injured during an upper respiratory infection. Major cerebral arteries are affected more often than smaller ones. The internal carotid artery may be involved in its extracranial or intracranial segment, often with extension to its terminal branches.²⁻⁸ The proximal middle or anterior cerebral arteries may be occluded separately, without involvement of the internal carotid artery.^{1,4,8} The smaller caliber cerebral arteries are rarely involved in the absence of changes in major cerebral arteries.^{1,4} The arterial changes consist of irregularities, narrowing or occlusion of vessels in the neck or skull.

The prior upper respiratory infection in these cases may be mild, or more often is associated with prominent cervical lymph nodes, pharyngitis, tonsillitis, or retropharyngeal abscess. Otitis media or sinusitis may also be

present. The neurologic symptoms usually appear a few days or weeks after the onset of the upper respiratory infection.^{2,3,6,7} Hemiplegia is the main neurologic symptom. Convulsions may also be seen. These may be present without hemiplegia, as we noted in another patient with upper respiratory infection in whom angiography revealed irregularities and beading of a small branch of a middle cerebral artery.

The pathogenesis of the cerebral artery involvement in these cases is not fully understood. The internal carotid arteries lie close to the tonsils and some cervical lymph nodes. The inflammatory process may extend directly from an infected tonsil or lymph node to the contiguous adventitia of the internal carotid artery, producing a localized arteritis with intimal damage, thrombus formation, and peripheral embolization later.² The intracerebral vascular changes result from these emboli. More than one intracranial artery may be thus affected. This hypothesis may explain the findings in our second patient who

demonstrated irregularities of the internal carotid artery and of a small distal branch of the middle cerebral artery.

The other two patients, however, had an angiographically normal internal carotid artery. In these instances, there may have been an underlying arteritis of the internal carotid artery, too mild to be detected angiographically, yet capable of producing distal emboli. In case 2, if the initial angiogram had not been performed early, the irregularities of the internal carotid artery would not have been demonstrated.

On the other hand, when changes in the internal carotid artery only become apparent in later angiograms, these may be expressions of the formation of scar tissue. As in the child reported by Harwood-Nash,⁴ in some symptomatic children with an apparently normal cerebral angiogram, the emboli may be too peripheral to be detectable by angiography.²

A history of upper respiratory infection may not be clinically evident,⁷ or only a history of a febrile episode may be elicited in some of the children who present with involvement of the cervical internal carotid artery.⁵ In all such cases the vascular involvement leads to ischemia and hemiplegia. Structural damage to the brain results in various degrees of residual neurologic deficit.

Another alternative explanation for the peripheral vascular changes with angiographically normal carotid arteries is focal arteritis caused by an inflammation or allergic

reaction, localized to that segment of the artery.⁹ There is, however, no convincing evidence to substantiate this hypothesis.

With the increasing use of cerebral angiography in the investigation of children with acute hemiplegia, and a more careful evaluation and awareness of the angiographic findings, positive results may prove more prevalent than has been previously suspected.

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