

Cranial Arteritis Associated with Herpes Zoster

Case Report with Angiographic Findings¹

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ABSTRACT—Angiographic findings of unilateral angiitis in a case of herpes zoster ophthalmicus with contralateral hemiplegia are described. Herpes zoster is often associated with major CNS involvement and a vascular etiology was previously postulated. Recent pathological reports suggest that cerebral angiitis secondary to herpes virus infection may be more common than heretofore realized. Our findings suggest a means by which suspected cases can be more fully evaluated.

INDEX TERMS: Arteries, inflammation • Cerebral Angiography, indications • Cerebral Blood Vessels, diseases • Hemiplegia • Herpes Zoster • Meningoencephalitis

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HERPES ZOSTER produces localized lesions of the skin and the peripheral nervous system. There have also been reports in which central nervous system dysfunction has followed attacks by the varicella virus, and some pathological evidence for selective involvement of blood vessels supplying the central nervous system (9). The purpose of this paper is to describe the cerebral angiographic picture seen in a patient presenting with hemiplegia following contralateral ophthalmic herpes zoster. We believe the hemiplegia resulted from cerebral angiitis.

CASE REPORT

A 7-year old Caucasian boy presented at Eugene Talmadge Memorial Hospital with progressive onset of right hemiplegia which had begun during the previous week. Six months prior to admission, a rash had developed over his face in the distribution of the ophthalmic division of the left trigeminal nerve. A diagnosis of Herpes zoster ophthalmicus was made. The lesions had slowly regressed until admission to this hospital, when only stellate cutaneous scars and mild corneal opacities remained. One week prior to admission, the boy noticed progressive onset of weakness in his right arm and leg over a period of 12 hours. During the next six days, the weakness progressed to complete right hemiplegia. There was also increasing dysphasia.

On physical examination, the child was alert with expressive dysphasia. Areas of hyperpigmentation and scarring were noted in the distribution of the ophthalmic division of the left trigeminal nerve. There was also mild scarring of the left cornea and a spastic right hemiplegia. The pupils were asymmetrical, larger on the left, and there was mild right central facial weakness.

Initial blood studies revealed hemoglobin 12.5 g/100 ml, hematocrit 38.7 ml/100 ml, white blood count 16,800/mm³ with 51% polymorphonuclear leukocytes, 24% eosinophils, 17% lymphocytes, and 8% monocytes. The eosinophilia was explained by a *Necator americanus* infestation, subsequently demonstrated in stool examination. Lumbar puncture revealed an opening pressure of 90 mm H₂O of crystal clear fluid with a cell count of 1 lymphocyte and 13 red blood

cells/ml. Cerebrospinal cultures for pathogens and acid fast organisms were negative. Immunoglobulin electrophoresis revealed IgG 1050, IgA 231, IgM 97, all within the normal range. Antinuclear factor, anti-streptolysin-O (ASO) titers, and lupus erythematosus smears were normal.

An electroencephalogram showed high voltage delta activity over the left cerebral hemisphere, more marked posteriorly. A ^{99m}Tc pertechnetate scintigram demonstrated a markedly abnormal flow pattern. There was initially more activity on the right with a delay of maximum visualization of the radionuclide on the left. Static views (Fig. 1) demonstrated a triangular-shaped area of increased activity in the left frontoparietal area.

A left percutaneous carotid arteriogram demonstrated normal ventricular size and no midline shift. The left middle cerebral branches had numerous areas of segmental constriction. There was also involvement of the proximal left anterior cerebral artery with a localized area of narrowing distally below the corpus callosum (Figs. 2 and 3, A). In subsequent films, stasis of contrast media in the left middle cerebral artery branches was noted (Fig. 3, B). Stasis and segmental narrowing, localized to the left, primarily affecting the middle and anterior cerebral arteries, were felt to be consistent with arteritis. A right carotid arteriogram showed normal findings.

During his hospitalization of two weeks, a gradual lessening of the patient's right hemiplegia was noted.

DISCUSSION

Central nervous system (CNS) involvement with herpes zoster occurs in the spinal cord, producing myelitis throughout the CNS with diffuse meningoencephalitis, and, following herpes zoster, ophthalmicus in which a contralateral hemiplegia may result. The various modes of involvement of the CNS have been reviewed by Acers (1).

In 1964, Rose *et al.* (8) reviewed 40 cases of herpes zoster meningoencephalitis. Autopsied cases often had lesions of the blood vessels supplying the central nervous system. The lesions included infiltration of the intima and adventitia with giant and mononuclear cells. In addition, infarction was encountered in the brain and spinal cord. Kolodny *et al.* (6) provide the only previous angiographic description of a patient with CNS involvement associated with herpes zoster in the case of a 21-year-old man who had slowly progressive

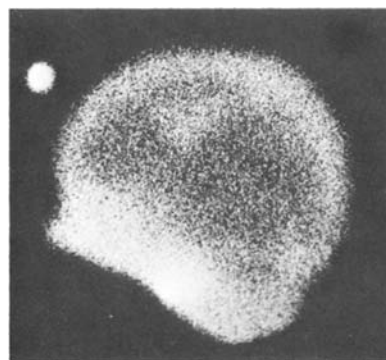


Fig. 1. Left lateral static brain scan showing a triangular area of increased uptake high in the posterior frontal region.

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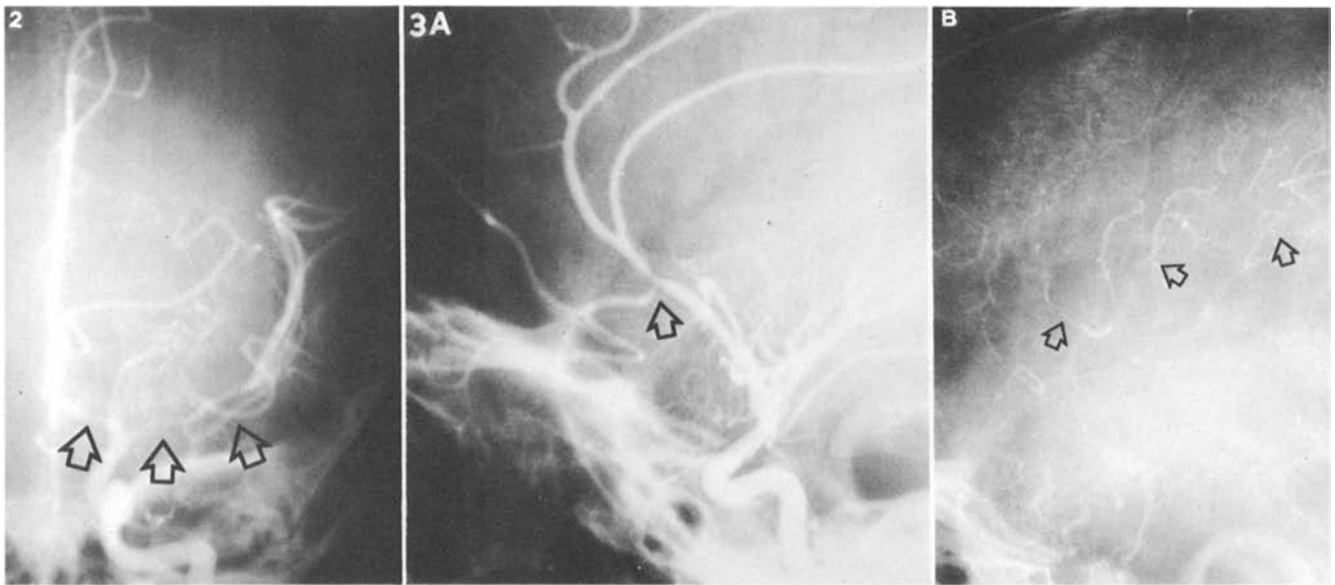


Fig. 2. Anteroposterior view of the left internal carotid arteriogram showed multiple areas of narrowing in the proximal middle and anterior cerebral arteries (arrows).

Fig. 3. A. The lateral left internal carotid arteriogram shows a localized area of constriction of the anterior cerebral artery involving the origin of the frontopolar artery (arrow). Note slow circulation in the middle cerebral artery branches.

B. Late arterial phase showing stasis of contrast material in branches of the middle cerebral arteries in the posterior frontal region.

CNS involvement leading to death two and one half years after his initial complaint.

In our case, the onset of contralateral hemiplegia followed ophthalmic herpes by six months. In most of the previously reported cases, evidence of CNS involvement occurred two to fourteen weeks after the onset of ophthalmic herpes zoster (1). In one case, reported by Norris *et al.* (7), the hemiplegia occurred six months after ophthalmic herpes zoster. Mention has already been made of a patient with diffuse CNS angiitis associated with herpes zoster which ran a course of two and one half years. Thus, the temporal span of our case is not unusual.

Angiographic findings in our patient included segmental narrowing of the middle cerebral artery in the sylvian fissure and the anterior cerebral artery below the genu of the corpus callosum. The abnormal isotopic flow studies and the triangular area of increased uptake seen on the static brain scan were highly suggestive of cerebral infarction. Since our patient recovered, we are unable to make a clinico-pathological correlation of the demonstrated lesions.

Possible explanations for contralateral hemiplegia associated with herpes zoster ophthalmicus have been reviewed by Acers. Cope and Jones (3) suggested spread through the corticospinal tracts. Schiff and Brain (10), and Gordon and Tucker (4) have suggested a diffuse invasion of the tissue adjacent to the gasserian ganglion by the inflammatory process. The latter theorized that the neurologic changes were due to vascular thrombosis secondary to this. Biggart (2) suggested spread of the inflammatory process through the perivascular spaces.

We would postulate two different mechanisms of involvement which could explain the angiographic picture. A localized attack on the internal carotid artery adjacent to the gasserian ganglion could occur with the development of a thrombus and subsequent distal embolization. However, the angiogram fails to demonstrate pathologic lesions of the carotid siphon, and the lesions distal to this were not typical

of emboli. The angiographic findings reported here simulate the angiographic pictures of tuberculous meningitis reported by Greitz (5). This might suggest another explanation whereby the inflammatory process spreads from the gasserian ganglion through the ipsilateral subarachnoid spaces.

Rosenblum and Hadfield (9) have recently offered pathological evidence for cerebral angiitis associated with herpes zoster and suggested that this may be more common than previously realized. We feel the angiographic findings in our case offer a mechanism by which the CNS symptomatology may be evaluated.

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