Short Communication

Sudden Hemiparesis as the Presenting Sign in Cryptococcal Meningoencephalitis

ROBERT F. SAUL, M.D.,* JAMES G. GALLAGHER, M.D., PH.D.,† AND JOHN E. MATEER, M.D.*

SUMMARY A previously healthy young man presented with an acute stroke syndrome and was found to have cryptococcal organisms in the CSF. Though an initial CSF examination for an infectious etiology was negative, a second lumbar puncture was performed because of hypoglycorrhachia, which established the diagnosis. An uneventful recovery followed the administration of Amphotericin B and 5-Flucytosine.

A literature search revealed only one previously reported case of cryptococcal meningoencephalitis presenting as a stroke. The need for performing a CSF examination on young patients presenting with a cerebrovascular event, and the aggressive investigation of unexplained hypoglycorrhachia are emphasized.

Stroke Vol 17, No 4, 1986

CRYPTOCOCCUS NEOFORMANS invading the central nervous system most commonly presents with symptoms and signs characteristic of meningitis, meningoencephalitis, or a mass lesion.1,2 Headache, with concomitant nuchal rigidity, decline in mental function, papilledema, and later progressive focal signs, are the most commonly encountered features of this illness. A sudden hemiparesis as the presenting sign of cryptococcal meningoencephalitis is rare, having been reported only once in the literature.3 We report a second case which we believe represents a sudden occlusion of a small hemispheric branch of the right middle cerebral artery secondary to a focal area of vasculitis, or a septic embolism to the right internal capsular branches.

Case Report

A 36-year-old male university meteorologist was in good health until nine months prior to admission, when he developed the insidious onset of bioccipital and retro-orbital headaches that required regular analgesia for relief. He was able to continue his occupation, but was plagued by the nagging headache. A diagnosis of sinusitis had been made by one physician, and he was treated with a course of an antibiotic without relief. Two weeks prior to his current admission his headaches worsened, and one day prior to admission, he noted the sudden inability to place his left hand on a computer keyboard. There was some associated left leg and thigh numbness, and his wife noted that his speech was slurred. His colleagues noted a facial weakness. He was taken to a local hospital where his examination showed a normal mental status, an easily recognizable left facial weakness, a mild left hemiparesis, left hyperreflexia, a left Babinski sign, and no sensory loss. A CT scan of the head without contrast was normal. A lumbar puncture showed clear fluid, two undifferentiated white blood cells, and 24 red blood cells per high powered field. The CSF glucose was 31 mg/dl, with a concomitant serum glucose of 108 mg/dl. The protein was 69 mg/dl. India Ink and gram stain examinations revealed no organisms.

He was transferred to our institution the following day. Further history revealed hands-on exposure to some cockatoos at an exotic bird farm ten months earlier. He was afebrile and his general physical examination was normal. The neurological examination was unchanged. A repeat CT scan with contrast was normal. The peripheral white blood cell count was 5,500/mm³, with 68% polymorphonuclear leukocytes, 1% bands, 21% lymphocytes, 5% mononuclear cells, 4% eosinophils, and 1% basophils. The hematocrit was 39%, Westergren sedimentation rate was 5 mm/hr, BUN was 11 mg/dl, and creatinine was 1.1 mg/dl. A chest X ray was normal. An EKG showed left axis deviation. A repeat lumbar puncture revealed an opening pressure of 55 mm H₂O, 74 white blood cells per high powered field, of which 98% were lymphocytes, and 2% monocytes. The glucose was 33 mg/dl with a concomitant serum glucose of 112 mg/dl. An India Ink preparation revealed rare budding yeast consistent with Cryptococcus neoformans. A CSF cryptococcal antigen latex agglutination titer was markedly elevated at 1:32. Subsequent cultures of the spinal fluid grew Cryptococcus neoformans. A CSF cryptococcal antigen latex agglutination titer was markedly elevated at 1:32. Subsequent cultures of the spinal fluid grew Cryptococcus neoformans. The patient was started on Amphotericin B and 5-Flucytosine.4 Within 48 hours his neurological deficit was rapidly improving and cerebral arteriography was therefore not performed. By ten days, his entire examination was normal. An uneventful recovery followed.

Discussion

Acute cerebrovascular syndromes are uncommonly reported in cryptococcal meningoencephalitis. Aber-
fled and Gladstone reported a 28-year-old man with no antecedent symptoms or signs, who awakened with a left cerebral infarction. This was attributed to a small vessel occlusion in the distribution of the left middle cerebral artery. After numerous CSF examinations *Cryptococcus neoformans* was identified, and it was postulated that a septic embolus caused the infarction.

In a 1983 clinicopathologic conference, Little et al., discussed a 70-year-old man presenting the fever, skin rash, and mental confusion, who one month later developed a mild left hemiparesis, and was found to have a right basal ganglia infarct due to a proliferative endarteritis secondary to *Cryptococcus neoformans*. This patient had been evaluated extensively for one month for the presenting symptoms listed above, and in the course of his illness developed the hemiparesis.

Hemiparesis and focal cerebral signs occur in cryptococcal central nervous system infections, which are more commonly seen in patients with granuloma, or what are known as torulomas.6,7 These patients generally have a slowly progressive deficit, unlike patients with acute vascular insults.

Sebetta and Andriole reviewed three major series of cases, and found 10% of the patients had an aphasia. There was no mention of hemiparesis or long tract signs.8 Edwards et al., in a series from Australia, noted long tract signs in 15 to 29 patients late in the course of their disease, but none of these patients had the signs early in their illness.9

Though our patient was plagued by mild headaches for nine months, there were no signs on physical examination of meningeal inflammation. Neurological consultation was requested for the acute hemiparesis. There was no evidence of a mass lesion on two CT scans, and though no cerebral arteriogram was performed, the patient presented with a clear cerebrovascular syndrome that resolved rapidly and completely.

We feel our patient probably occluded a small superficial branch of the middle cerebral artery, or a right internal capsular branch, either from a vasculitis, or an embolus, respectively.

Pathologically, the inflammatory meningeal infiltrate in Cryptococcal infections, may spread along the perivascular Virchow-Robin space into the cortex, and sometimes deeper into the cerebral tissue. An unreactive granulomatous meningoencephalitis may occur, with the development of cystic cavities teaming with yeast organisms. A mesenchymal cellular reaction, resembling yeasts with morphology compatible with *Cryptoccus neoformans* only after prolonged examination. The gram stains revealed even fewer recognizable organisms. The assay of cryptococcal antigen was not performed on the initial specimen, but would, most likely, have suggested the diagnosis even in the absence of cultural or light microscopic evidence of the yeast. In central nervous system cryptococcosis, serologic analysis will reveal approximately 92% demonstrable antigen in the CSF, and/or antibodies in the serum.3

The prompt response to antifungal therapy in our case was gratifying. However, prolonged administration of antifungals is required to sterilize the CSF. Some patients will have persistent low antigen titers in the CSF and positive smears, even though cultures are negative and the symptoms have resolved. Relapses are common.

Acknowledgment

The authors would like to thank Dr. J. Alfred Jones of State College Pennsylvania for referring the patient.

References

Sudden hemiparesis as the presenting sign in cryptococcal meningoencephalitis.
R F Saul, J G Gallagher and J E Mateer

*Stroke*. 1986;17:753-754
doi: 10.1161/01.STR.17.4.753

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

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/17/4/753

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/