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Isolated Cerebral Mucormycosis: Case Report with CT and Pathologic Correlation

Ferris Ginsberg,[1] Robert G. Peyster,[2] Eric D. Hoover,[2] and Sydney D. Finkelstein[3]

Intracranial mucormycosis is an uncommon fungal infection showing a predilection for diabetics and immunologically compromised patients [1–3]. In the common rhinocerebral form, cerebral involvement is secondary to direct extension from nasal-sinus disease [2–4]. Isolated cerebral mucormycosis (ICM), occurring by hematogenous seeding, has been reported only rarely [3, 5–10]. We report the second case of ICM with CT depiction of the disease process and the first in which CT is correlated with the gross and histopathologic findings.

Case Report

A 41-year-old man was admitted with a 2-day history of right-sided weakness, headache, and fever. Past medical history included alcohol and IV drug abuse, especially heroin and amphetamines, and was negative for diabetes mellitus.

On admission the patient was slightly lethargic, febrile, and had a flaccid right hemiparesis. The chest X-ray, EKG, complete blood count, and drug screen were negative. Cranial CT was done immediately after admission. The noncontrast CT scan showed a hypodense left basal ganglionic lesion with a very small central area of high density and mild mass effect (Fig. 1A). A contrast-enhanced CT scan showed no abnormal enhancement. A diagnosis of early infarction or cerebritis, possibly secondary to meningitis or vasculitis, was suggested. Lumbar puncture yielded cloudy CSF with WBC count of 3,278 with 70% polymorphonuclear leukocytes, 30% lymphocytes, 100 RBCs, glucose of 59% (serum glucose 173 mg%), and protein of 123 mg%. Gram stain, India ink, and acid fast staining were negative. The patient was started on IV chloramphenicol and penicillin. Left carotid arteriography was normal. CSF cultures for bacteria, viri, fungi, and acid fast bacilli were negative.

On the 5th hospital day the patient was noted to be more lethargic. A repeat contrast-enhanced CT scan showed marked enlargement of the left basal ganglionic lesion with extensive hemorrhage and increased mass effect (Fig. 1B). Again, there was no evidence of abnormal contrast enhancement. Because of the patient's failure to respond to antibiotics and the possibility of cerebral vasculitis he was placed on steroids and fluid restriction.

On the 11th hospital day the patient's condition deteriorated and signs of uncal herniation appeared. A noncontrast CT scan showed further enlargement of the lesion, with extension into the midbrain, and intraventricular hemorrhage. On the 13th hospital day the patient became hypotensive and died in spite of resuscitative efforts.

The brain at autopsy weighed 1400 grams and showed left uncal herniation as well as cerebellar tonsilar herniation. Coronal sections revealed a single large area of necrotic brain tissue, involving the left basal ganglia, thalamus, hypothalamus, and upper midbrain (Figs. 1D and 1E). Microscopic sections showed extensive hemorrhagic infarction due to widespread thrombosis of large and small intracerebral vessels, which contained both intraluminal and intramural fungal hyphae. Breakdown of the vascular walls was also noted. There was no evidence of capsule formation. Nonseptate hyphae consistent with mucormycosis were present throughout the area of necrosis. General autopsy findings consisted of renal mucormycosis and hepatic cirrhosis.

Discussion

Mucormycosis subsumes opportunistic infections caused by the fungi Mucor, Absidia, and Rhizopus, common saprophytes found in soil and decaying vegetable matter [2, 3, 11]. These fungi cause aggressive and fulminant infections in diabetic, immunocompromised, and debilitated patients, and in IV drug abusers [3, 5, 12].

Intracranial mucormycosis is uncommon [13], accounting for only 13% of cerebral fungal infections in a recent postmortem study [14]. There are two forms of intracranial mucormycosis. In the more common rhinocerebral form, seen most often in diabetics, the brain is secondarily involved by direct extension from nasal-sinus disease. In ICM, cerebral involvement, alone or with other organs, occurs by hematogenous seeding. This form is extremely rare and there have been only four previous reports of this disease occurring in IV drug abusers [3, 8–10]. ICM has also been reported in two patients who were treated with long-term steroids for chronic active hepatitis [6, 7], and in one patient after open head trauma [5].

The CT appearance of ICM in our patient reflects the

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[1] Department of Radiology, Monmouth Medical Center, Long Branch, NJ 07740.

[2] Department of Radiology, Section of Neuroradiology, Hahnemann University Hospital, Mail Stop #206, Broad & Vine Streets, Philadelphia, PA 19102. Address reprint requests to R. G. Peyster.

[3] Department of Pathology, Hahnemann University Hospital, Philadelphia, PA 19102.

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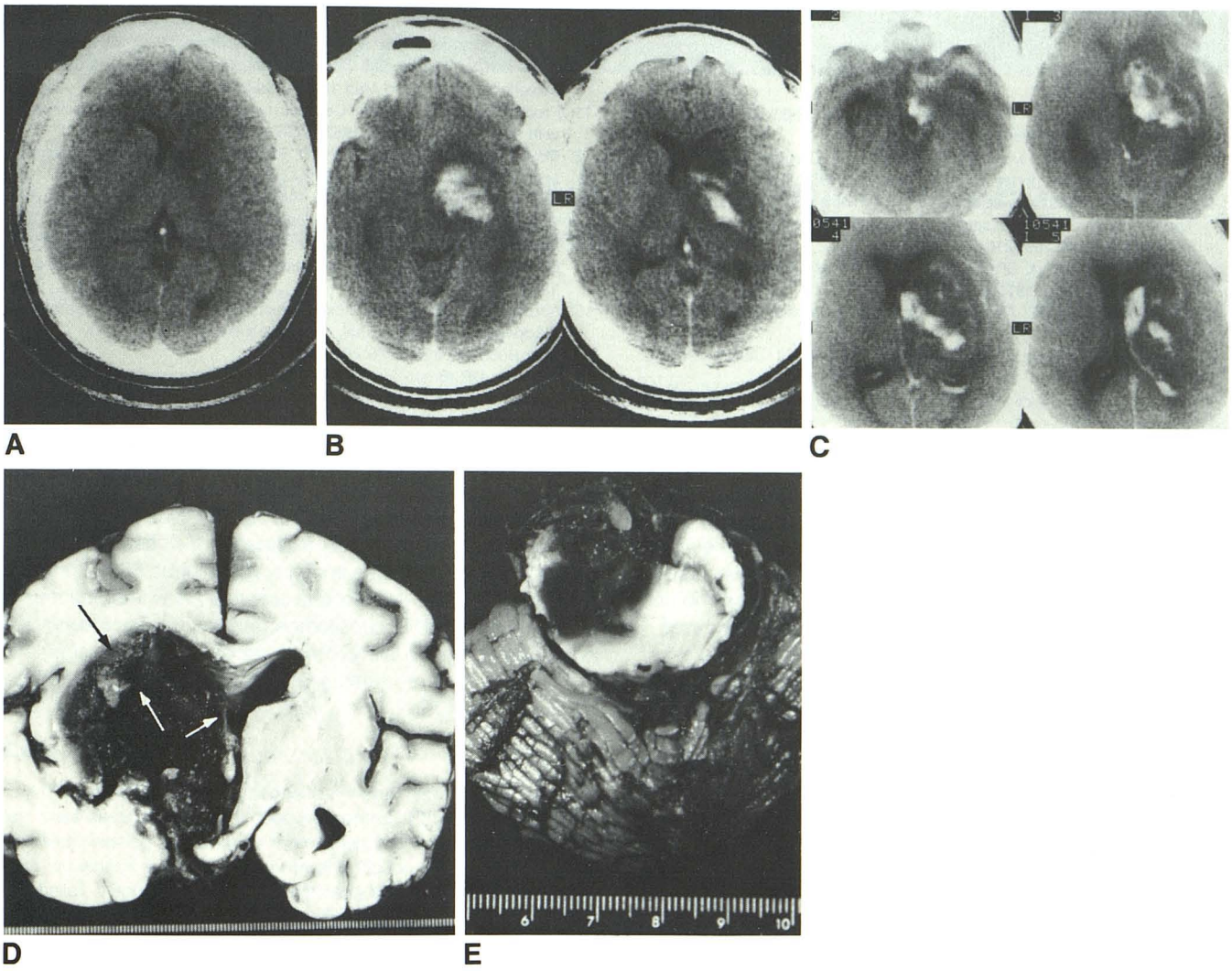

Fig. 1.—*A*, Noncontrast CT scan shows hypodense lesion with small central area of high density representing an infarction with minimal hemorrhage in left basal ganglia. Left frontal horn is compressed.

***B*, Contrast-enhanced CT scan shows enlargement of lesion with increased hemorrhage and mass effect.**

***C*, Noncontrast CT scan shows extension of lesion and hemorrhage into left lateral ventricle and midbrain. Loss of white-gray matter distinction indicates cerebral edema.**

***D*, Coronal section of brain showing large area of necrosis involving left basal ganglia, thalamus, and hypothalamus. Infarction of surrounding tissue (*black arrow*) and focal hemorrhage into ventricle (*white arrow*) are seen.**

***E*, Necrosis and cerebritis extend into midbrain.**

underlying pathophysiology of the disease. The hallmark of mucor infections, regardless of anatomic site, is invasion of blood vessel walls. Masses of hyphae may directly occlude the vascular lumen or serve as a nidus for thrombus formation, leading to ischemic infarction [1, 2, 11, 13]. Subsequently, invasion and breakdown of the vessel wall leads to hemorrhage and direct invasion of the brain [15]. These processes result in extensive areas of tissue necrosis.

The absence of a contrast-enhancing rim around the lesion in our case correlates with the lack of capsule formation seen at necropsy. The CT in our case differs from the only other one previously published [7], which showed a low-density lesion with definite rim enhancement and irregular areas of enhancement within the lesion. Enzmann et al. [16] stated that the lack of an enhancing rim around an intracerebral infection in immunocompromised patients indicates the inability of the host to confine the process and implies a poor prognosis.

The possibility of ICM should be considered when cerebral lesions resembling infarcts, especially if atypical or hemorrhagic, are found in a septic, immunocompromised patient with negative blood and CSF cultures.

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REFERENCES

1. Sweeney PJ, Hahn JF, McHenry MC, Mitsumoto H. Mucormycosis presenting as positional nystagmus and hydrocephalus. *J Neurosurg* **1980**;52:270–272
2. Lehrer AI, Howard DH, Sypherd PS, Edwards JE, Segal GP, Winston DJ. Mucormycosis. *Ann Intern Med* **1980**;93:93–108
3. Masucci EF, Fabara JA, Saini N, Kurtzke JF. Cerebral mucormycosis (phycomycosis) in a heroin addict. *Arch Neurol* **1982**;39:304–306
4. Schochett SS. Infectious diseases. In: Rosenberg RN, Schochet SS, eds. *The clinical neurosciences: section III, neuropathology*. New York: Churchill Livingstone, **1984**:209–210
5. Ignelzi RJ, VanderArk GP. Cerebral mucormycosis following open head trauma. *J Neurosurg* **1975**;42:593–596
6. Whalen MJ, Beyt BE. Cryptic cerebral phycomycosis. *Ann Intern Med* **1979**;91:655
7. Mikhael MA. Cerebral Phycomycosis. *J Comput Assist Tomogr* **1979**;3(3):417–420
8. Hameroff SB, Eckholdt JW, Lindenberg R. Cerebral phycomycosis in a heroin addict. *Neurology* **1970**;20:261–265
9. Chmel H, Grieco MH. Cerebral mucormycosis and renal aspirgillosis in heroin addicts without endocarditis. *Am J Med Sci* **1973**;266:225–231
10. Adelman LS, Aronson SM. The neuropathologic complications of narcotic addiction. *Bull NY Acad Med* **1969**;45:225–233
11. Prockop LD, Silva-Hutner M. Cephalic mucormycosis (phycomycosis): case with survival. *Arch Neurol* **1967**;17:379–386
12. Meyer RD, Rosen P, Armstrong D. Phycomycosis complicating leukemia and lymphoma. *Ann Int Med* **1972**;77:871–879
13. Rao VRK, Pillai SM, Mathews G, Radhakrishnan VV. Cerebral mucormycosis—a case report. *Neuroradiology* **1978**;15:291–293
14. Whelan MA, Stern J, deNapoli RA. The computed tomographic spectrum of intracranial mycosis: correlation with histopathology. *Radiology* **1981**;141:703–707
15. Adams JH. Parasitic and fungal infections of the nervous system. In: Blackwood W, Corsellis JAN, eds. *Greenfield's neuropathology*. Chicago: Year Book Medical, **1976**:269–291
16. Enzmann DR, Brant-Zawadzki M, Brit RH. CT of central nervous system in infections in immunocompromised patients. *AJR* **1980**;135:263–267