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Brain Abscess Following Intracerebral Hemorrhage: A Case Report[1]

Jin Kyung Oh, M.D., Young Joo Kim, M.D., Eun Deok Chang, M.D.[2]

A brain abscess developing at the site of a preceding intracerebral hemorrhage is a rare finding. We report here on a rare case of a brain abscess that developed at the site of an intracerebral hemorrhage after a systemic infection.

Index words : Brain abscess
Cerebral hemorrhage

Brain abscess is an uncommon, serious infection of the brain parenchyma, and this requires prompt administration of high dose antibiotics and surgical drainage. Brain abscesses frequently arise secondary to hematogenous dissemination, by direct inoculation (trauma or surgery), by contiguous dissemination from an extracranial site or as a complication of meningitis. However, the development of a brain abscess at the site of a prior intracerebral hemorrhage is extremely rare, and only several sporadic cases have been reported in the medical literature (1, 2). Differentiation between a resolving intracerebral hematoma and a brain abscess is mandatory for administering the appropriate treatment. We present here a case with a brain abscess at the site of a prior intracerebral hemorrhage and we provide the MR findings that can help differentiate between a brain abscess and a resolving intracerebral hematoma.

Case Report

A 68-year-old man with a medical history of an old infarction at the left basal ganglia developed right hemiplegia and motor aphasia after a fall. There is no external evidence of head trauma, but CT and MRI revealed multifocal acute intracerebral hemorrhages with fluid-blood levels in the left frontal lobe (Fig. 1, 2A, 2B). He was treated conservatively and rehabilitation was then started. However, on the 14th hospital day, the patient developed a high fever with aggravation of his right hemiplegia and aphasia. The patient's chest x-rays were normal. The laboratory findings showed a peripheral blood WBC count of 46700/mm^3, an ESR of 72 mm/hr and a C-reactive protein level of 24.2 mg/dL. A urinary tract infection was present. Three urine cultures yielded

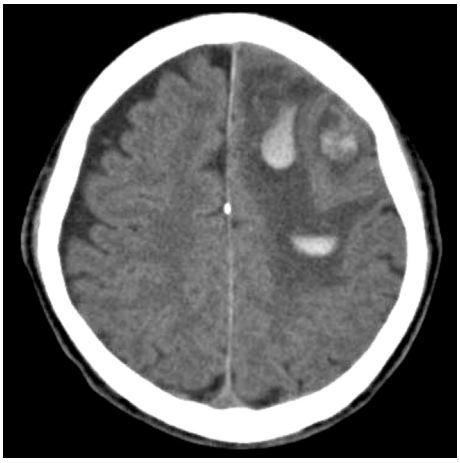

Fig. 1. The initial non-enhanced CT at the time of injury shows multifocal hemorrhages in the left frontal lobe.

[1]Departments of Radiology and [2]Pathology, The Catholic University of Korea
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Address reprint requests to : Young Joo Kim, M.D., Department of Radiology, Uijongbu St. Mary's Hospital, The Catholic University of Korea, 65-1 Kumoh-dong, Uijongbu 480-130, Korea.
Tel. 82-31-820-3599 Fax. 82-31-846-3080 E-mail: violet2@catholic.ac.kr

Pseudomonas aeruginosa. Following administration of antibiotics for two weeks, the fever subsided and the WBC count returned to normal. However, the patient's neurologic symptoms did not improve. The follow up MR scan performed on the 27th hospital day showed cystic masses with marked ring enhancement at the sites of the preceding intracerebral hemorrhages, in addition to the extensive perilesional edema. The center of the lesion had high signal intensity on the T2-weighted images, with a complete dark signal rim, and mixed high signal intensity on the T1-weighted images. Diffusion-weighted imaging (DWI) revealed concentric bands of heterogeneous signal intensity with an inversely heterogeneous afferent diffusion coefficient (ADC) at

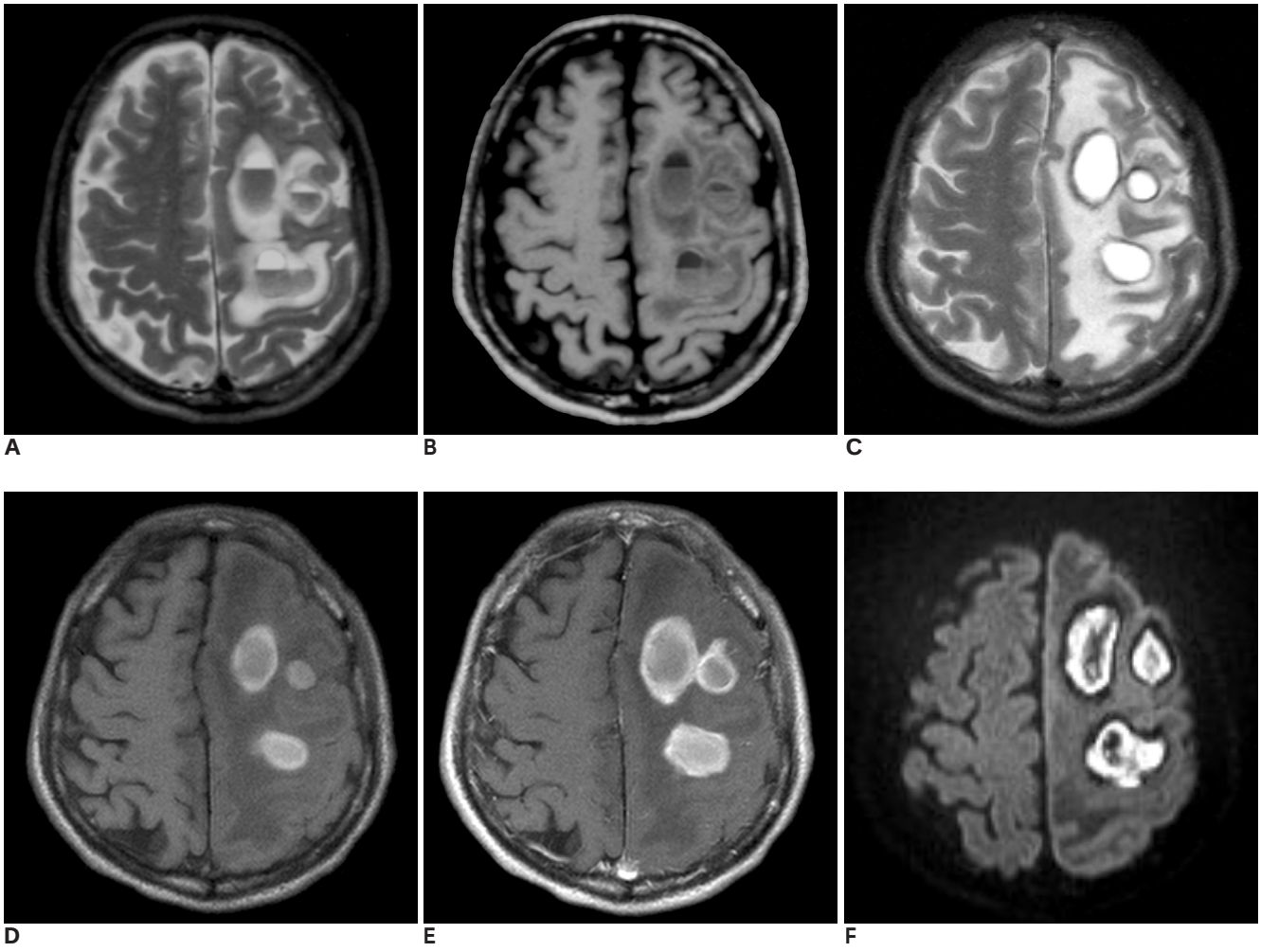

Fig. 2. A, B. The initial MR images at the time of injury. The axial T2-weighted (**A**) and T1-weighted images (**B**) show the layered, acute stage hematomas with minimal perilesional edema. The lesions are not enhanced (not shown).
C-G. The MR images taken 3 weeks after the hemorrhage. The axial T2-weighted image (**C**) shows multiple well-defined hyperintensity lesions with hypointense walls, and these lesions appear hyperintense on the axial T1-weighted image (**D**) with isointense walls. Note the prominent perilesional edema. The postcontrast T1-weighted image (**E**) demonstrates uniform peripheral wall enhancements, which accurately corresponded to the low signal rim on the T2-weighted image. The diffusion-weighted image (**F**) reveals layered heterogeneous signal intensities with inversely heterogeneous afferent diffusion coefficient values (**G**) at the center of the lesions and a marked hypointense rim at the periphery. Ring enhancement with a complete hypointense rim on both the T2-weighted images and the DWI, and the extensive edema indicate that brain abscess developed at the site of the preceding hemorrhage.

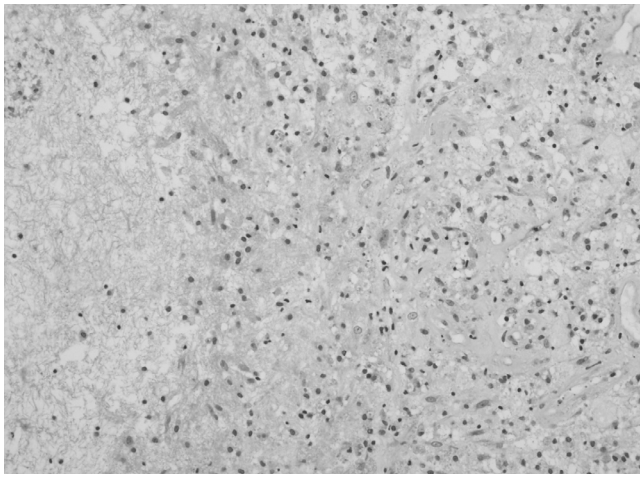

Fig. 3. Photomicrograph of the histologic specimen reveals fibroblasts, mononuclear infiltrates and macrophages associated with hemosiderin pigments in the abscess capsule, and amorphous proteinaceous materials with degenerating erythrocytes (upper left) within the abscess cavity (Hematoxylin-eosin stain, ×200).

the center of the lesions and a marked hypointense rim at the periphery of the lesions. The low signal rim at the periphery on the DWI and T2-weighted images accurately corresponded to the enhanced rim on the contrast enhanced images (Fig. 2C - G). Given the possibility of brain abscesses, the patient underwent surgical drainage via open craniotomy. A yellowish, cheese like purulent material was aspirated and three well capsulated abscesses were removed. The histology revealed macrophages, a mononuclear infiltrate, revascularization and gliosis in the wall, and all this was suggestive of a brain abscess (Fig. 3). The cultures of the surgical specimen and the necrotic fluid were negative. The patient was treated with broad-spectrum antimicrobial coverage for 6 weeks. Follow up CT was performed 2 weeks after completion of the antibiotic course and it revealed near complete resolution of the ring enhancing lesions and the brain edema. The patient has fared well and he has been followed for 12 months.

Discussion

An intact blood brain barrier in the normal brain provides resistance to infection. Disruption of the blood brain barrier by hemorrhage may make the affected brain tissue susceptible to infection by blood-borne bacteria with subsequent abscess formation (1). In the previously reported cases, the first episodes of high fever, which indicated systemic infection and bacteremia, occurred 0-90 days after the onset of the intracereberal hemorrhages. Our patient had an episode of high fever 2 weeks after the hemorrhage. A urinary tract infection was considered to be the source of hematogenous seeding of the infection that spread to the brain. Although uniform ring enhancement is an important radiologic finding for the diagnosis of a brain abscess, it is not a specific finding for a brain abscess and it must be distinguished from a necrotic neoplasm and other cystic lesions. Intracerebral hematomas usually resolve spontaneously or they form a cavity over several months. As the hemorrhage evolves, different characteristic appearances can be identified on CT & MRI, depending on the age of the bleed. From 1 - 6 weeks, peripheral enhancement can be seen because there is a breakdown of the blood-brain barrier in the vascularized capsule that surrounds the hematoma (3) and this mimics the appearance of an abscess. Because a brain abscess is an emergency condition that requires prompt administration of high dose antibiotics and surgical drainage, it is mandatory to differentiate a brain abscess from a resolving hematoma. However, this differentiation can be difficult due to the overlapping radiological features.

More recently, DWI has demonstrated significant potential to further delineate and diagnose ring-enhancing mass lesions (4, 5). Many studies have confirmed the presence of restricted diffusion in those abscesses with high signal intensity in the central cavity and a correspondingly low ADC value. The probable factors for the restricted diffusion in brain abscesses are the microscopic organization of the tissues, the high viscosity of the pus that's caused by a high protein level and the different types of viable or dead cells along with the necrotic tissue, bacteria and exuded plasma. Additionally, water molecules are bound to amino acid groups on the surface of macromolecules, which further restrict their translational motion (5). However, these findings are not confined to an abscess and they might present in various other brain diseases like hemorrhagic primary or secondary tumors and resolving hematomas (4). It is known that one DWI finding of hyperacute and late subacute hematomas is hyperintensity on DWI with a low ADC value (6). The precise biophysical explanation for the decreased ADC in hyperacute and late subacute hematomas is uncertain. We found layered low and high signal intensities on the DWI with an inversely heterogeneous ADC at the center of the lesions in our case. Correlation of our surgical and pathologic specimens demonstrated that the central heterogeneous signal intensities on the DWI reflected the proteinaceous and

necrotic debris with a bloody background in the abscess tissues.

The mature abscess often has a rim that gives a signal that's similar to or slightly higher than the white matter on the T1-weighted images and a lower signal for the rim is seen on the T2-weighted images. These signal properties have been ascribed to collagen and the paramagnetic free radicals that are released from the phagocytosing macrophages (7). A low signal rim on T2-weighted images may also be seen in other lesions such as subacute and chronic hematomas, metastases, granulomatous lesions and, on rare occasions, gliomas (8). In our case, a complete low signal rim was seen on the T2-weighted image and the DWI. Although paramagnetic hemosiderin-laden macrophages begin to take up residence at the periphery of the hemorrhage at the late subacute stage of an intracerebral hematoma, the presence of scant amounts of hemosiderin is unlikely to have been the primary cause of a complete T2 hypointense rim on the subacute hematoma in our case. According to Kang et al, a hypointense rim on both the DWI and the T2-weighted images showed up only at the chronic stage of an intracerebral hematoma (6). Furthermore, a hypointense rim at the periphery on the DWI and T2-weighted images accurately corresponded to the enhanced rim observed on the contrast enhanced images. In a study on 221 patients reported by Schwartz et al, an abscess was the most common pathology that manifested as a ring-enhancing lesion with complete hypointense rims on the T2-weighted images (8). Schwartz et al also reported that an intracerebral hemorrhage was a rare condition with ring enhancement and a T2 hypointense border (8). The histology for our case confirmed the presence of macrophages, a mononuclear infiltrate, revascularization and gliosis, and hemosiderin in the wall, which all contributed to the complete low signal rim seen on T2WI.

In our case of brain abscess, the patient's brain edema was aggravated on the follow up imaging study. After intracereberal hemorrhage, penetration of the serum protein from the clot into the surrounding white matter, followed by breakdown of the blood-brain barrier due to inflammation, have been proposed as mechanisms leading to edema formation in the extracellular component (9). This edema usually subsides and the mass effect gradually diminishes in cases with a resolving hemorrhage. It is known that pronounced or persistent edema is one of the signs of hemorrhagic intracranial neoplasm rather than a benign hemorrhage. In the case of a brain abscess, exuberant neovascularization around the margin of the necrotic brain is responsible for the marked vasogenic edema. The brain edema may be greater in volume than the abscess itself, and this causes much of the associated mass effect. In spite of encapsulation, a circumscribed disturbance of the blood-brain barrier persisted, and this was responsible for the belated resolution of the patient's edema and a slow decrease of the intracranial pressure (10).

This case illustrates that a cerebral hematoma can be transformed into an abscess when systemic infection complicates a hematoma. Abscess formation in a hematoma cavity should be considered in the differential diagnosis of patients who deteriorate after a febrile episode and who also have a history of an intracerebral hemorrhage.

In this case with a brain abscess, the presence of ring enhancement with a complete hypointense rim both on the T2-weighted images and DWI and the unusual extensive edema along with the clinical findings made us diagnose a brain abscess rather than a resolving subacute hematoma.

References

1. Chen ST, Tang LM, Ro LS. Brain abscess as a complication of stroke. *Stroke* 1995;26:696-698
2. Siatouni A, Mpouras T, Boviatsis EJ, Gatzonis S, Stefanatou M, Sakas D. Brain abscess following intracerebral haemorrhage. *J Clin Neurosci* 2007;14:986-989
3. Zimmerman RD, Leeds NE, Naidich TP. Ring blush associated with intracerebral hematoma. *Radiology* 1977;122:707-711
4. Hartmann M, Jansen O, Heiland S, Sommer C, Munkel K, Sartor K. Restricted diffusion within ring enhancement is not pathognomonic for brain abscess. *AJNR Am J Neuroradiol* 2001; 22:1738-1742
5. Mishra AM, Gupta RK, Jaggi RS, Reddy JS, Jha DK, Husain N, et al. Role of diffusion-weighted imaging and in vivo proton magnetic resonance spectroscopy in the differential diagnosis of ring-enhancing intracranial cystic mass lesions. *J Comput Assist Tomogr* 2004;28:540-547
6. Kang BK, Na DG, Ryoo JW, Byun HS, Roh HG, Pyeun YS. Diffusion-weighted MR imaging of intracerebral hemorrhage. *Korean J Radiol* 2001;2:183-191
7. Haimes AB, Zimmerman RD, Morgello S, Weingarten K, Becker RD, Jennis R, et al. MR imaging of brain abscesses. *AJR Am J Roentgenol* 1989;152:1073-1085
8. Schwartz KM, Erickson BJ, Lucchinetti C. Pattern of T2 hypointensity associated with ring-enhancing brain lesions can help to differentiate pathology. *Neuroradiology* 2006;48:143-149
9. Wagner KR, Xi G, Hua Y, Kleinholz M, de Courten-Myers GM, Myers RE, et al. Lobar intracerebral hemorrhage model in pigs: rapid edema development in perihematomal white matter. *Stroke* 1996; 27:490-497
10. Wallenfang T, Bohl J, Kretzschmar K. Evolution of brain abscess in cats formation of capsule and resolution of brain edema. *Neurosurg Rev* 1980; 3:101-111

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뇌 실질 내 혈종과 동반된 뇌 농양: 증례 보고[1]

[1]가톨릭대학교 영상의학과, [2]병리과

오진경 · 김영주 · 장은덕[2]

뇌 실질 내 혈종이 있었던 위치에 뇌 농양이 병발되는 경우는 현재까지 13예가 보고된 매우 드문 증례이다. 저자들은 뇌 실질 내 혈종이 있었던 위치에 전신 패혈증으로 인한 뇌 농양이 병발되었던 환자 1예를 경험하였기에 영상소견과 함께 보고하는 바이다.