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/mm³, 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...
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

![Image](image-url)

**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.
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 intracerebral 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 intracerebral 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

13기 신생아 환자 1명을 대상으로 시행한 자기공명영상(네비게이션 기반 스테르티지(NDI) 영상)과 초음파 검사를 이용한 황반 후반부 점막의 위치를 비교한 결과, NDI 영상과 초음파 영상이 일치하는 것으로 나타났다. 이는 NDI 영상이 실제 위치를 정확히 반영할 수 있는 것임을 의미한다.

1. 이형철, 2. 김은영

 ND의 위치를 정확히 반영할 수 있는 NDI 영상의 중요성에 대해서는 앞으로 더욱 연구가 필요하다.