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# Development of Brain Abscess at the Exact Site of a Previous Intracerebral Hematoma: A Case Report

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Brain abscess is a neurological emergency that requires immediate high-dose antibiotics or surgical intervention. Brain abscess at the site of a previous hemorrhagic stroke that did not receive surgical intervention is rare. Here, we report the case of a 48-year-old man who developed a brain abscess as infective transformation of a preceding right putaminal hemorrhage, 22 days after the stroke. The patient recovered after surgical treatment and systemic antibiotics therapy. An abscess culture test revealed the presence of Group D *Salmonella*, which has never been reported in a case of brain abscess. A comprehensive literature review was performed. We concluded that the possibility of brain abscess formation at the site of intracranial hemorrhage or infarction should be considered when a patient deteriorates clinically after a stroke. ( Tw J Phys Med Rehabil 2011; 39(3): 167 - 173 )

**Key Words:** brain abscess, intracerebral hematoma, putamen, Group D *Salmonella*

## INTRODUCTION

Brain abscess is an uncommon but potentially life-threatening disease. It develops from an infection of local cranial sites in 37% of the cases. In 22% of cases the infection is blood-borne or associated with trauma, meningitis, or postoperative craniotomy. The absence of a clear source of infection is reported in as many as 40% of the cases.<sup>[1,2]</sup> The treatments for brain abscess include antibiotics, aspiration, drainage, and excision. However, irrespective of the treatment performed, the overall mortality rate of patients with brain abscess is as high as 10%.<sup>[1,2]</sup>

Some instances of brain abscess after intracerebral hemorrhage with cranial surgery have been reported.<sup>[1]</sup>

However, brain abscess following ischemic or hemorrhagic stroke without surgical intervention is extremely rare. To the best of our knowledge, only 20 cases have been reported in the medical literature.<sup>[2-6]</sup> Most of the cases reported had an obvious infectious focus. Here, we report another such case and review the literature.

## CASE REPORT

We report the case of a 48-year-old man who experienced left-sided weakness suddenly on July 4, 2009. He went to the emergency room (ER) of a local hospital. A computer tomography (CT) scan showed a right putaminal hemorrhage (Figure 1A, 1B). The patient had clear

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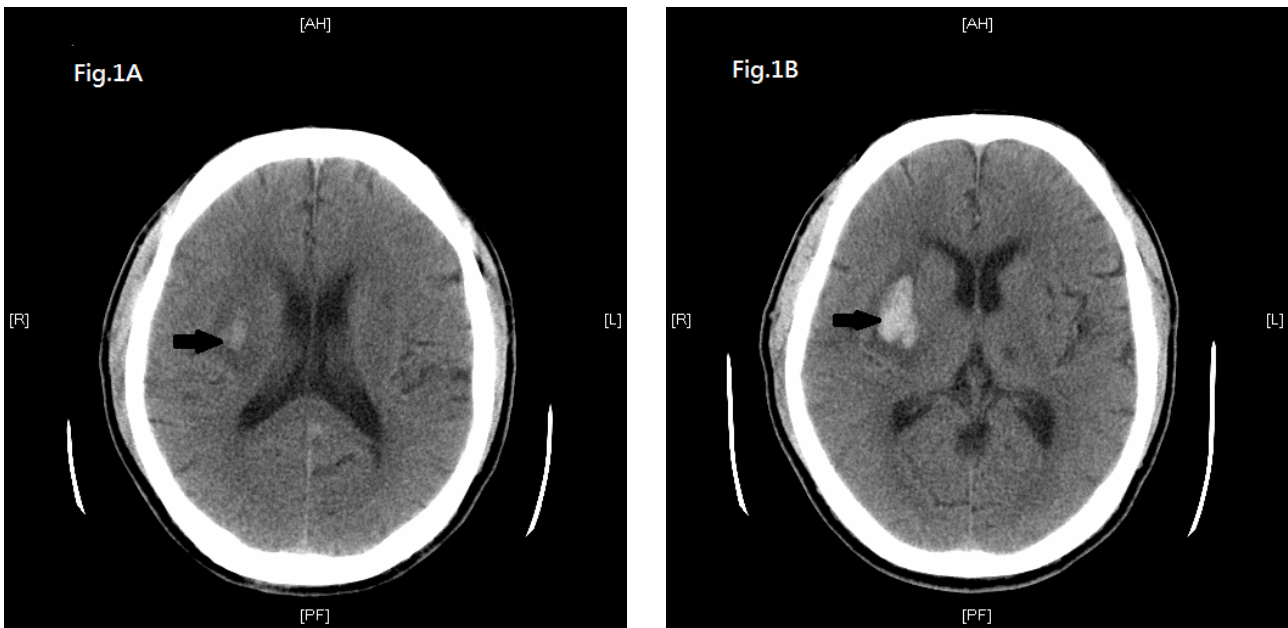


Figure 1. 1A, B Serial computed tomography without contrast enhancement showing an intracerebral hemorrhage in the right putamen (black arrow) of our patient.

consciousness and could still walk independently. He refused hospitalization, even been advised. However, he developed progressive weakness of his left limbs and experienced frequent falls. As a result, the patient was admitted to the rehabilitation ward of our hospital for further evaluation and management 12 days after his stroke (July 16, 2009). The medical history of the patient included hypertension but not diabetes mellitus, congenital heart disease, or immunocompromised disease. The patient had alcoholism, with a sorghum liquor intake of 600-1200 cc per day for more than 30 years. Assessment with the Glasgow Coma Scale (E4V5M6) indicated clear consciousness upon admission. He was impaired in judgment, orientation, memory, abstract thinking, and calculation. The patient had muscle power grade 2 in his left arm and grade 3 in his left leg. Fever up to 38.5°C without chills was noted on the day of admission. Laboratory testing revealed a leukocyte count of 11,400/mm<sup>3</sup> with 83.8% neutrophils. The patient's fever subsided after intravenous fluid 1000mL was given. Both blood and urine cultures showed negative findings. The patient was started on rehabilitation programs but experienced a sudden loss of consciousness on July 26, 2009 (22 days after his stroke). The left pupil was 3.0 mm in diameter

without light reflex. In contrast, the right pupil was 2.0 mm in diameter with light reflex. The patient also experienced a partial seizure of his right arm. Body temperature remained normal. Emergent non-contrast enhanced brain CT was performed and showed a cystic mass at the site of the preceding hemorrhage and a shift of the midline to the left. The patient's laboratory test results revealed a leukocyte count of 16,240/mm<sup>3</sup> and a C reactive protein concentration of 0.77 mg/dl. All the GOT, GPT and ammonia level were normal. The patient was transferred to the neurological intensive care unit. Endotracheal tube was inserted. Hyperventilation was performed first. Glycerin injection was given. For better image quality, brain CT with contrast was performed, which showed a brain abscess with marked ring-like enhancement (Figure 2). The neurological status of the patient deteriorated gradually over 13 hours, with dilation (6.5 mm) of the right pupil without light reflex and normal size (2.0 mm) of the left pupil. Craniectomy with drainage of the ring-like mass was performed on the same day. Viscous purulent material was aspirated and a pus culture showed Salmonella Group D1. However, there was no microorganism growth with either blood or urine culture tests. A later stool culture test was also negative.

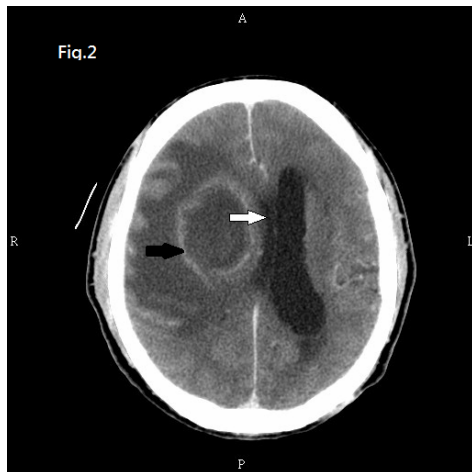


Figure 2. Computed tomography with contrast agent in our patient 3 weeks after stroke showing a well-defined, thin-walled ring-enhancing mass that was 4.8 cm × 3.3 cm × 4 cm in size (black arrow). There was a marked shift of the midline (white arrow) and extensive mass effect. The lesion was located in the area affected by the preceding hemorrhagic stroke.

There was no evidence of hepatic infection found with abdominal sonography. An antibiotics susceptibility test led to intravenous (IV) antibiotics (ampicillin 2.0 g q12h) being prescribed for 8 days. We subsequently consulted an infectious disease physician, who suggested the administration of IV ceftriaxone (2.0 g q12h). However, the patient developed ceftriaxone-related fever and the drug was discontinued after 9 days. We replaced this antibiotic with levofloxacin, which we administered as 750 mg IV qd for 26 days before changing to the oral form for 2 weeks. The patient regained consciousness after the operation and was transferred to our rehabilitation ward for continuing rehabilitation. Follow-up contrast-enhanced brain CT scans performed 3 weeks, 6 weeks and 7 months separately after the operation showed a marked reduction in the brain abscess cavity (Figure 3A, B, C). Though experiencing left hemiplegia, the patient was doing well 3 months after the operation and could walk using a quadricane with moderate assistance.

## DISCUSSION

Brain abscess can develop either by the direct spread of an infection, which accounts for 20-60% of cases, or

through hematogenous seeding. No primary site or underlying condition can be identified in 20-40% of patients with brain abscess. The mean duration between the onset of the symptoms and diagnosis of brain abscess is 13-14 days.<sup>[1,2,7]</sup> Increased intracranial pressure is evident in two-thirds of cases and, as a focal sign, hemiparesis is present in one-third. Fever is present in 62% of brain abscess cases.<sup>[1]</sup>

Brain abscess after stroke is uncommon but when it does occur the stroke location is usually deep in the brain. In such cases, brain abscess formation occurs mainly via hematogenous seeding, because most indirect infection of the brain involves deep in the brain. To the best of our knowledge, only 20 cases of brain abscess after hemorrhagic stroke<sup>[2-6]</sup> and 9 cases after ischemic stroke<sup>[6,8-15]</sup> have been reported in the medical literature: These cases are summarized in Table 1.

The blood brain barrier (BBB) becomes fragile after intracerebral hemorrhage and perihematoma edema may damage the vascular endothelium. Disruption of the BBB by hemorrhage may make localized brain tissue susceptible to infection by blood-borne bacteria and subsequent abscess formation.<sup>[2]</sup> In addition, a hematoma may serve as a medium in which bacteria can grow. This would explain why there is a higher incidence of brain abscess after hemorrhagic stroke than after ischemic stroke.

Among all patients with brain abscess, the most common bacteria cultured are *Staphylococcus aureus* (14%) and *Proteus* species (14%).<sup>[1]</sup> In only those patients with brain abscess after stroke, *S. aureus* is the most common pathogen.<sup>[4]</sup> *Proteus* species are the only pathogen reportedly found in patients with brain abscess after infarction.<sup>[12]</sup> The present case study is only the second report of a brain abscess involving *Salmonella* species<sup>[5]</sup> and the first involving Group D *Salmonella*. Our patient developed a fever on the 12th day after a stroke but this ceased after 12 hours. The patient was retrospectively suspected as having transient non-typhoid *Salmonella* bacteremia that his immune system may have temporarily overcome. However, some bacteria might pass through BBB to brain tissues and a brain abscess developed. A CT scan of the patient's brain abscess 10 days after the fever showed a thin-walled ring enhancement, which is consistent with previous reports of brain abscess images taken within 10-14 days of fever.<sup>[17]</sup>

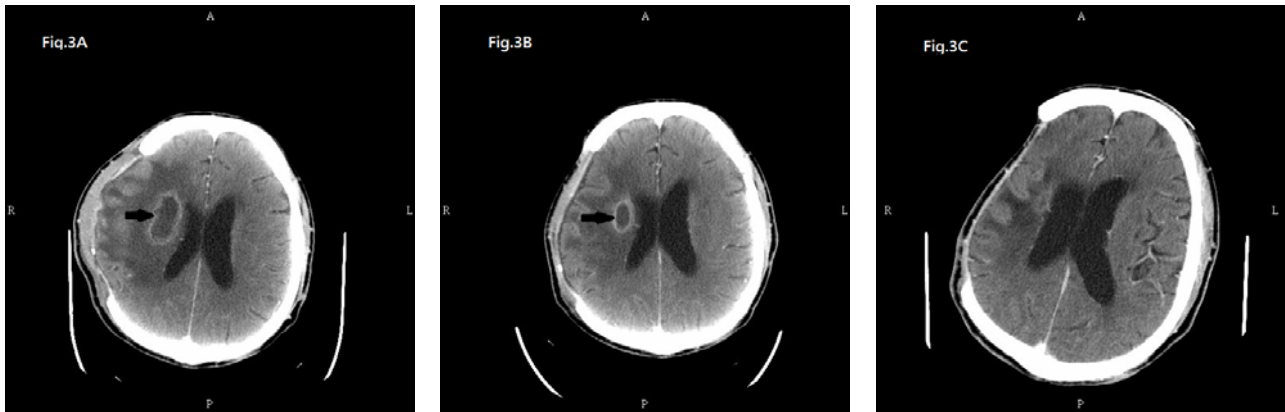


Figure 3. Computed tomography with contrast agent in our patient 3 weeks after operation (black arrow) (A), 6 weeks after operation (black arrow) (B), and 7 months after operation (C).

Table 1. Summary of reported cases of cerebral abscess after intracerebral hemorrhage<sup>[3,4]</sup>

No.	Authors and year of publication	Age/Sex	Previous disease	Site of hemorrhage	Route of infection	Pathogen	Time to abscess formation (weeks)*	Country
1	Israel et al <sup>[18]</sup>	27 F	Eclampsia	L basal ganglia	Unknown	<i>Pneumococcus</i>	n.r.	n.r.
2	Busse et al <sup>[19]</sup>	45 M	n.r.	R putamen	Sepsis	Sterile	8	Germany
3	Biller et al <sup>[20]</sup>	62 M	Hypertension	R frontal lobe	Infected wound of a carotid endarterectomy <sup>(a)</sup>	<i>Staphylococcus aureus</i>	> 2	USA
4	Biller et al <sup>[21]</sup>	34 F	Preeclampsia	R basal ganglia	Infected episiotomy <sup>(a)</sup>	<i>Staphylococcus epidermidis</i> and alpha-haemolytic <i>Streptococcus</i>	4	USA
5	Kurihara et al <sup>[22]</sup>	53 M	n.r.	R putamen	Phelebitis <sup>(b)</sup>	<i>Staphylococcus</i>	20	Japan
6	Mashimoto et al <sup>[23]</sup>	74 M	n.r.	R basal ganglia	Endocarditis <sup>(a)</sup>	<i>Enterococcus faecalis</i>	n.r.	Japan
7	Arentoft et al <sup>[5]</sup>	59 F	Chronic renal failure	R frontal lobe	Gastroenteritis <sup>(a)</sup> Bacteremia	<i>Salmonella typhimurium</i>	15	Denmark
8	Lee et al <sup>[24]</sup>	64 M	n.r.	R putamen	Sepsis of unknown origin	Sterile	4	Korea
9	Iida et al <sup>[25]</sup>	71 M	n.r.	Cerebellum	Pneumonia <sup>(b)</sup>	<i>Staphylococcus aureus</i>	3	Japan
10	Chen et al <sup>[6]</sup>	56 F	Hypertension	L putamen	Sepsis of unknown origin	<i>Klebsiella pneumoniae</i>	5	Taiwan
11	Bert et al <sup>[26]</sup>	42 F	n.r.	L temporoparietal	Phelebitis <sup>(a)</sup>	<i>Staphylococcus aureus</i>	8	France
12	Sumioka et al <sup>[27]</sup>	55 M	n.r.	R putamen	Unknown	<i>Morganella morgani</i>	8	Japan
13	Okami et al <sup>[28]</sup>	51 M	n.r.	L thalamus	Bacteremia <sup>(a)</sup>	<i>Staphylococcus aureus</i>	12	Japan
14	Amayo et al <sup>[29]</sup>	66 M	n.r.	L basal ganglia	Pneumonia <sup>(a)</sup>	Sterile	4	Kenya
15	Inamasu et al <sup>[30]</sup>	48 M	Diabetes mellitus, Hypertension	R putamen	Pneumonia <sup>(a)</sup>	<i>Enterococcus faecalis</i>	4	n.r.
16	Nowak et al <sup>[31]</sup>	58 M	Hypertension	L thalamus	Catheter-induced sepsis <sup>(a)</sup>	<i>Staphylococcus aureus</i>	9	Germany
17	Nakai et al <sup>[4]</sup>	75 M	n.r.	R temporoparietal	Unknown	Gram-positive coccus	4	Japan
18	Nakai et al <sup>[4]</sup>	32 M	n.r.	R occipital	Bacteremia <sup>(b)</sup>	Sterile	3	Japan
19	Siatouni et al <sup>[3]</sup>	79 M	Idiopathic leukemia, psoriasis (on immunosuppressive medication)	L parietal lobe	UTI <sup>(a)</sup>	<i>Enterococcus faecalis</i>	4	Greece
20	Dashti et al <sup>[2]</sup>	30 F	Alcoholism	L basal ganglia	Unknown	<i>Citrobacter koseri</i>	4	USA
21	Present case	48 M	Hypertension, Alcoholism	R putamen	Bacteremia <sup>(b)</sup>	<i>Salmonella Group D1</i>	3	Taiwan

\*Duration from intracerebral hemorrhage to abscess formation; (a)proven; (b)presumed; n.r.: not reported; M: male; F: female; UTI: urinary tract infection; R: right; L: left.

The existing literature suggests that over half of all patients with brain abscess after hemorrhagic stroke had diabetes<sup>[3]</sup> or a number of immunosuppressive factors, such as taking immunosuppressive drugs or chronic nephropathy.<sup>[5]</sup> Our patient did not have diabetes mellitus, rheumatoid disease, renal disease, liver disease, or AIDS. However, he had long-term alcohol abuse. Non-typhoid *Salmonella* infection is not uncommon in immune competent patients in Taiwan.<sup>[16]</sup> Bacterial factors seem to play an important role in the pathogenesis of such infections. In a previous study, a tendency was found for Group D1 *Salmonella* to cause bacteremia with a higher frequency than other serotypes.<sup>[16]</sup> Since the patient was immunocompromised, he was more easily infected by *Salmonella*.

On average, one case of brain abscess after stroke has been reported for each year since 2000 (Table 1). Half of these cases involved Asians (Japanese, Taiwanese, and South Koreans), which may indicate a higher incidence of brain abscess after stroke in Asians than in Westerners.

In conclusion, we reported the case of brain abscess after hemorrhagic stroke that was caused by *Salmonella* D1 infection. With quick diagnosis and treatment, the patient experienced good functional recovery.

## ACKNOWLEDGMENT

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## 腦膿瘍發生於原出血性腦中風的位置：病例報告

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腦膿瘍是神經科上的急症，需要立即性的給予抗生素或是開刀處理。在先前沒接受過手術處理的出血性中風的病人身上，腦膿瘍出現在先前出血的位置是很罕見的。本案例是一位 48 歲男性，因殼核出血性腦中風之後，步態不穩而入院接受復健治療，入院時有認知功能受損，復健表現被動的現象，在殼核出血性腦中風之後 22 天，這位病人在出血的位置出現了腦膿瘍，經過手術及抗生素治療之後，這位病人的腦膿瘍痊癒，培養出的細菌是沙門氏菌 D1 群。本報告針對腦中風之後併發腦膿瘍做一系列的文獻回顧，提供給臨床醫師參考。（台灣復健醫誌 2011；39(3)：167 - 173）

**關鍵詞：**腦膿瘍(brain abscess)，腦內出血(intracerebral hematoma)，殼核(putamen)，沙門氏菌 D 群(Group D Salmonella)