



## An Intracranial Petri Dish? Formation of Abscess in Prior Large Stroke After Decompressive Hemicraniectomy

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### Key words

- Decompressive hemicraniectomy
- Intracranial abscess
- Ischemic stroke
- Secondary infection
- Treatment

### Abbreviations and Acronyms

**CT:** Computed tomography  
**MCA:** Middle cerebral artery  
**MRI:** Magnetic resonance imaging  
**WBC:** White blood cell

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### INTRODUCTION

Stroke is the one of the most common causes of death and major cause of disability worldwide. Because of the aging population, the burden will increase greatly during the next 20 years (1). Patients with a cerebral infarction are vulnerable to severe complications because of the severity of their disease and their comorbidities (13). Secondary complications after stroke commonly include infection, venous thromboembolism, or cardiac disease.

Brain abscess is a life-threatening illness, requiring rapid diagnosis and treatment. The process of development includes seeding of an organism into the brain parenchyma, which often occurs in an area of damaged brain tissue or in a region with poor microcirculation. The lesion evolves from cerebritis stage to capsule formation. Brain abscesses can be caused by contiguous or hematogenous spread of an infection or by head trauma/neurosurgical procedure (4, 12). Areas of ischemia, infarction, and contusions in the brain can provide fertile ground for inoculation or bacterial seeding

■ **OBJECTIVE:** Development of brain abscess after an infarction is a rare clinical condition. There have been 11 cases in the literature. Many patients were treated with potent antibiotics only and did not survive. We present 2 cases in which patients received aggressive surgical resection of brain abscess and survived. The analysis of the literature confirmed our finding that surgical intervention of brain abscess in patients after stroke is advisable.

■ **METHODS:** A 58-year-old man was transferred to our institution with left hemiparesis, hemisensory loss, neglect, and hemianopsia. The computed tomography angiography demonstrated large volume right hemispheric infarct. He underwent decompressive hemicraniectomy but developed fevers and swollen fontanelle 6 weeks later, which did not improve with antibiotics. The magnetic resonance imaging demonstrated progression of ring-enhancement of the old infarct and abscess formation was suspected. In another case, a 42-year-old woman was admitted to our institution with aphasia and weakness on the right side. The computed tomography angiography showed left middle cerebral artery territory infarction and decompressive hemicraniectomy was performed. Patient recovered well but a brain abscess was suspected during a routine preoperative computed tomography before cranioplasty.

■ **RESULTS:** In the first case, patient was then taken to the operating room for resection of the infarcted brain tissue involved. The patient's consciousness improved, and he was discharged with antibiotic treatment. The patient subsequently underwent cranioplasty with synthetic graft with no complications. In the second case, the suspected abscess was resected surgically and culture of abscess grew *Pantoea agglomerans* and *Bacillus macerans*. The patient underwent synthetic cranioplasty 1 month later and remained in good condition.

■ **CONCLUSIONS:** Secondary abscess formation after significant ischemic stroke is a rare condition that carries potential for high morbidity/mortality. The limited body of literature with the addition of our 2 cases supports aggressive management with surgical evacuation of brain abscess to increase survival.

of organisms that result in abscess formation (5).

There is a paucity of literature studying the treatment of a brain abscess in patients with previous ischemic attack of the brain. In completing a literature search, we have identified 13 cases (including our 2 cases) in which patients had developed abscess after an acute ischemic stroke. We report cases in which a brain abscess was formed after an acute cerebral infarction and after life-saving decompressive hemicraniectomy. This literature combined

with our most recent cases, provides some guidance for future management of these complex patients.

### CASE PRESENTATIONS

#### Patient 1

**History and Examination.** A 58-year-old man with past medical history of coronary artery disease (status, after bypass graft), hypertension, and hyperlipidemia was transferred to our institution 9 hours with

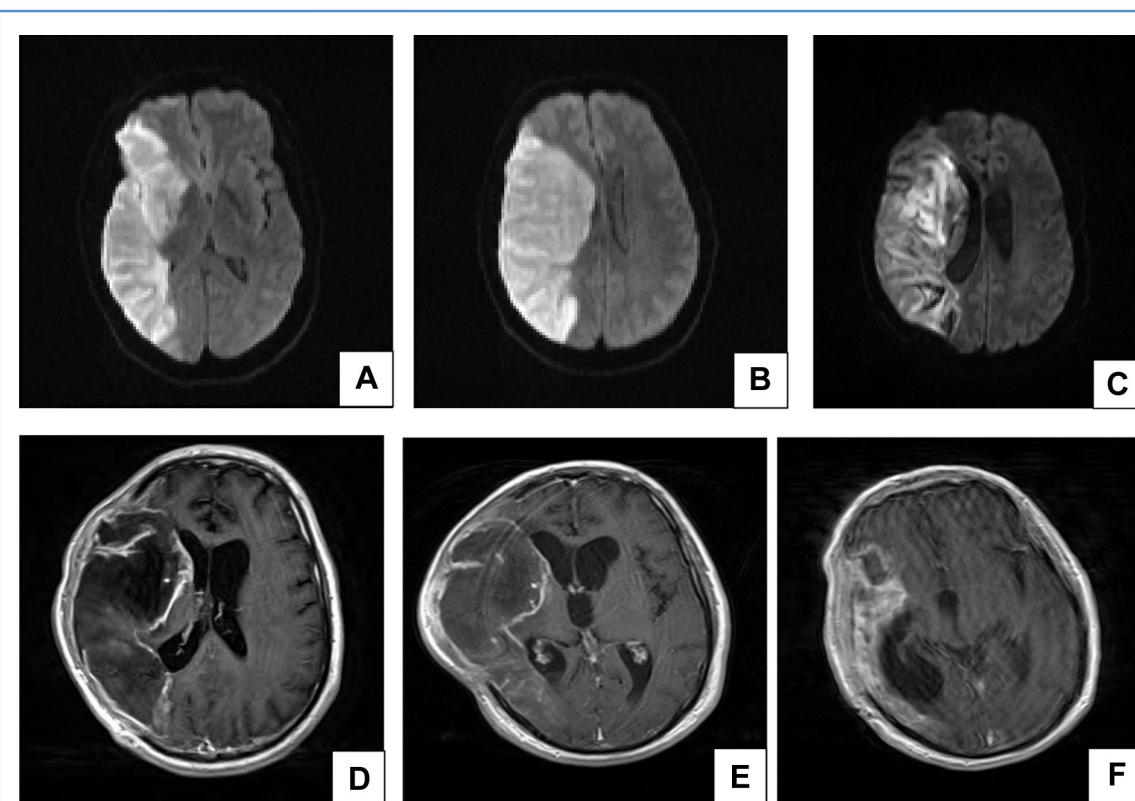
left hemiparesis, hemisensory loss, neglect, and hemianopsia (National Institutes of Health Stroke Scale 16). The computed tomography angiography demonstrated right initial segment of middle cerebral artery occlusion with early findings of edema and large volume right hemispheric infarct. Given his age, presence of early edema, and size of the infarct, he was taken for decompressive hemicraniectomy as a life-saving procedure (Figure 1). As a routine, we do not violate the pia during hemicraniectomy; we did not violate it in this case. Postoperatively, he was awake and alert, following commands, with stable left hemiparesis and hemisensory loss. He was discharged to a rehabilitation hospital in good condition. The patient was readmitted approximately 6 weeks later with fevers and swollen fontanelle. He was started on vancomycin and rocephin at the

rehabilitation hospital before lumbar puncture, and transferred back to our institution for workup and evaluation.

**Investigations.** The patient's laboratory values upon readmission revealed an erythrocyte sedimentation rate of 101 mm/h, C-reactive protein of 6.7 mg/dL and peripheral white blood cell count (WBC) of 9.4. Lumbar puncture showed red blood cell count as 334, WBC as 775 with 77% of neutrophils, glucose as 59, protein as 116. Gram stain was negative and there was no growth on cultures. The magnetic resonance imaging (MRI) demonstrated extensive encephalomalacia throughout the right middle cerebral artery (MCA) territory consistent with evolving infarct (Figure 1). There was some enhancement along the margins of the infarct, possibly indicative of evolving infarct versus infection. Given the laboratory and

radiographic findings, the patient was treated for presumed meningitis/encephalitis with empiric vancomycin, cefepime, and flagyl for 14 days; his cultures did not grow a specific pathogen. One day after stoppage of antibiotics, the patient developed new fever and confusion. A second lumbar puncture showed red blood cell count of 28, WBC count of 921, glucose of 42, protein of 197, and his peripheral WBC was 11.4. Repeat MRI demonstrated progression of ring-enhancement of the old infarct, with increasing restricted diffusion within the old infarct area, along with diffusion positivity in the ventricle, suggestive of pus (Figure 1).

**Management.** Because of the radiographic progression and clinical confusion, the patient was taken for exploration and resection of the infarcted brain tissue



**Figure 1.** **A, B:** DWI of patient in first case upon initial admission demonstrating a large right MCA territory stroke with early edema. **(C)** and post contrast T1 **(D)** 6 weeks after initial stroke of patient in first case demonstrated of extensive encephalomalacia throughout the right MCA territory, but notable contrast enhancement along the edges and some persistent diffusion weighted positivity. No definitive abscess or empyema was seen. **E:** Post contrast T1 antibiotics treatment of patient in first case demonstrated persistent ring-enhancement of the prior infarct site with increased edema and diffusion restriction. Overall changes were suspicious for early organized abscess formation. **F:** Post contrast T1 following surgery of patient in first case demonstrated significant improvement on restricted diffusion within the resected region with reduction of mass effect.

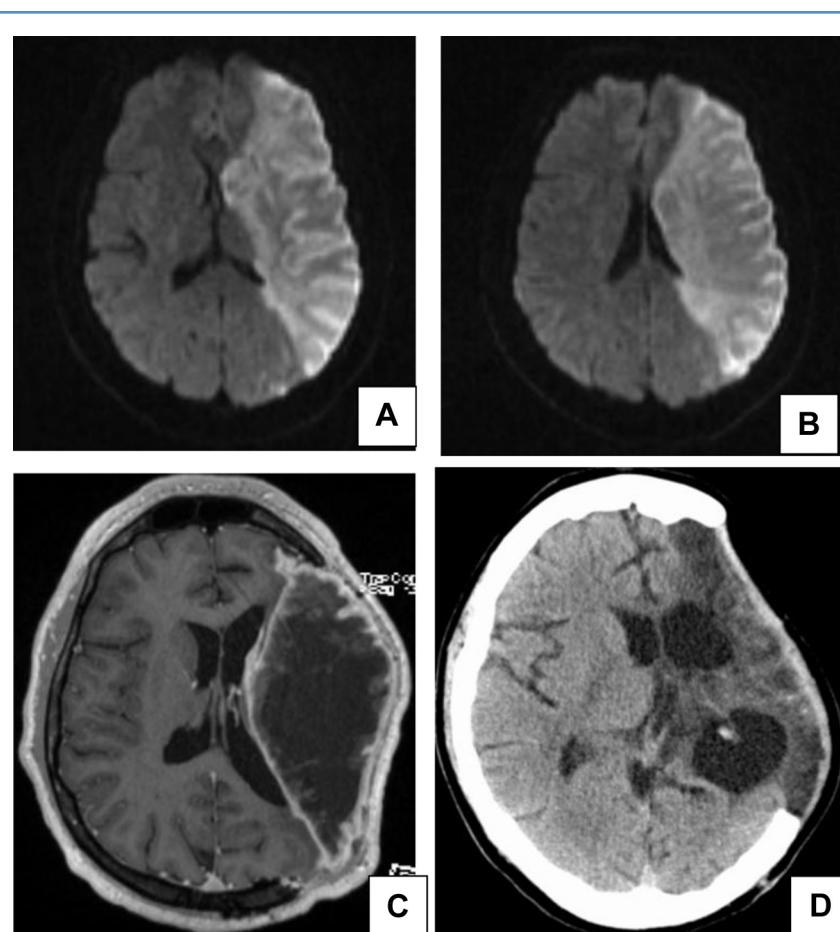
involved. Grossly, the patient's prior craniectomy wound was clean with no extradural evidence of infection. The infarcted brain tissue was entered and resected, with notable pus mixed with necrotic tissue. Pathology demonstrated extensive tissue necrosis with bacterial colonization and WBC reaction, although no specific organisms grew on culture. Furthermore, although bacteria were seen by the pathologists, Gram stain and culture were negative in the microbiology laboratory.

**Postoperative Course.** The postoperative MRI showed significantly improved restricted diffusion in the resected area (Figure 1). The patient's consciousness was also improved and he was discharged to a rehabilitation hospital for recovery with 6 weeks of treatment of combined vancomycin (1000 mg every 12 hours)/cefepime (2 g every 8 hours)/flagyl (500 mg every 8 hours). The patient subsequently underwent cranioplasty after completion of antibiotics with synthetic bone graft (Synthes PEEK Patient Specific Implant), and recovery was uneventful. At 3 months after cranioplasty, he is doing well with no further signs/symptoms of infection, and is within his baseline hemiparesis. He was planned for follow-up imaging, but died from myocardial infarction 6 months after his cranioplasty.

## Patient 2

**History and Examination.** A 42-year-old woman with aphasia, right hemiparesis (National Institutes of Health Stroke Scale 16) was admitted to our institution's stroke service 9 hours after she was last known as normal. The computed tomography angiography and MRI demonstrated a large acute left MCA stroke and she underwent decompressive hemisplenectomy (Figure 2). As noted in the prior case, we do not routinely violate the pia, and we did not do so in this case. She was discharged within 2 weeks in stable condition. The patient was readmitted 15 weeks after surgery due to finding of enlarged pseudofontanelle and possible abscess formation during preoperative computed tomography (CT) scan before cranioplasty.

**Investigations.** The patient's laboratory values upon readmission revealed peripheral WBC of 11.8, erythrocyte sedimentation rate of 40 mm/h, and C-reactive



**Figure 2.** **A, B:** DWI of patient in second case upon initial admission showing a large left MCA territory infarct. Post contrast T1 (**C**) on overall asymptomatic patient in second case revealed rim-enhancing parenchyma lesion suggesting abscess formation. **D:** CT of the second case 5 weeks after surgical drainage showed resolution of abscess.

protein of 1.8 mg/dL. MRI demonstrated a rim-enhancing collection in the previous infarcted area of left MCA territory, which was highly suspicious for tissue necrosis and superinfection forming an abscess (Figure 2).

**Management.** Because of the radiographic evidence, the patient was taken to the operating room for exploration and resection of the infarcted brain tissue involved. The previous craniectomy wound was well-healed with no evidence of extradural infection or inflammation. The infarct was entered, yielding pus combined with necrotic brain tissue. Culture of abscess grew *Pantoea agglomerans* and *Bacillus macerans*.

**Postoperative Course.** The patient was started on a course of 6 weeks of treatment of

combined vancomycin (1250 mg every 12 hours)/cefepime (2 g every 8 hours)/flagyl (500 mg every 8 hours). The patient was discharged and underwent cranioplasty with synthetic bone graft approximately 6 weeks later after completion of antibiotics. The patient remained in good condition, and is 16 months from her initial stroke with no clinical or radiographic evidence of recurrence of infection.

## DISCUSSION

The reported incidence of brain abscess formation in a prior infarct is rare. In the literature, we identified 11 prior cases in addition to our own (Table 1). In evaluating these cases, some patterns emerge. The duration between stroke and diagnosis of

**Table 1.** Analyses of Previously Reported Cases and Our Current Cases

Study ID	Age	Gender	Occluded Vessel	Thrombolytic?	Decompressive Surgery?	Concomitant Infection	Interval from Stroke (weeks)				Infection Treatment	Species	Outcome
							Stroke	Peak	Onset	Resolution			
1	Beloosesky et al., 2002 (3)	68	M	Left MCA	No	No	UTI, Fever, <i>Proteus mirabilis</i> in blood and urine, partially infected CSF	12 weeks			Antibiotics and surgery	<i>Proteus mirabilis</i>	Death
2	Amonn and Müller, 1984 (1)	68	M	Right MCA	No	No	Fever	46 weeks			Died before treatment	<i>Staphylococcus aureus</i>	Death
3	Xue and Wang, 2005 (13)	56	F	Right temporal/occipital lobe ischemia	Yes	No	Fever	9 weeks			Antibiotics and surgery	<i>Staphylococcus aureus</i>	Death
4	Chen et al., 1995 (4)	70	M	Right MCA	No	No	Aspiration pneumonia followed by fever	5 weeks			Antibiotics and surgery	Not found	Death
5	Shintani et al., 1996 (11)	40	M	Right PCA	No	No	Fever	2 weeks			Antibiotics and surgery	<i>Streptococcus</i> spp.	Recovered with homonymous hemianopsia
6	Miyazaki et al., 2004 (10)	77	M	Left temporal lobe ischemia	No	No	Pneumonia with a sputum culture of <i>Staphylococcus aureus</i> followed by fever	58 weeks			Antibiotics and surgery	<i>Staphylococcus aureus</i>	Recovered
7	Kraemer et al., 2008 (9)	33	F	Left MCA and ACA	Yes	Yes	Fever and detection of <i>Pseudomonas aeruginosa</i> and <i>Staphylococcus aureus</i> in tracheal aspirate	7 weeks			Antibiotics and surgery	Group C <i>Streptococcus</i> spp.	Recovered with hemiparesis
8	Yamanaka et al., 2011 (14)	75	M	Left MCA	Yes	No	None	13 weeks			Antibiotics and surgery	<i>Staphylococcus epidermidis</i>	Recovered
9	Arentoft et al., 1993 (2)	59	F	Right MCA	No	No	Fever with lung infiltration with blood and feces revealed <i>Salmonella</i>	12 days			Antibiotics	<i>Salmonella typhimurium</i>	Death
10	Emmez et al., 2007 (7)	64	M	Left PCA	No	No	Fever	13 weeks			Antibiotics	Unknown	Recovered with mild deficits
11	Ichimi et al., 1989 (8)	73	M	Left MCA	No	No	Fever	11 weeks			Antibiotics and surgery	<i>Proteus vulgaris</i>	Alive (no follow-up reported)
12	Present case (patient 1)	58	M	Right MCA	No	Yes	Fever	6 weeks			Antibiotics and surgery	Unknown	Recovered (died 6 months after operation due to unrelated cause)
13	Present case (patient 2)	42	F	Left MCA	No	Yes	None	15 weeks			Surgery	<i>Pantoea agglomerans</i> and <i>Bacillus macerans</i>	Recovered

MCA, middle cerebral artery; UTI, urinary tract infection; CSF, cerebrospinal fluid; PCA, posterior cerebral artery; ACA, anterior cerebral artery.

brain abscess varies significantly in the published cases; it ranged from 12 days to 58 weeks. Although the pathogens varied, the most common was *Staphylococcus* species. Most of the patients who had developed brain abscess presented with fever but often had negative systemic cultures. Therefore, providers who treat patients with severe stroke must be mindful of the possibility of an intracranial abscess in the febrile patient without positive systemic cultures, regardless of time interval from the stroke.

The pathogenesis of brain abscess within a previous stroke is understandable in the context of the tissue changes that occur in ischemia. The vascular event produces local blood-brain barrier disruption, which makes this region vulnerable to microbial seeding in the event of bacteremia secondary to systemic infection, leading to the formation of cerebral abscess (6). Furthermore, in the case of ischemia, lack of normal blood flow limits the ability of the immune system to combat invading pathogens. Of the 13 cases, only 3 patients had decompressive surgeries whereas the 10 other patients had no surgery. This suggests that the decompressive surgery for stroke is not the offending risk factor. In our patient, the abscess formation occurred within the infarcted cortex with no other tissues affected (skin, soft tissue, dura) at the time of wound exploration. In neither case was infection found grossly in the wound or in the extradural space. Furthermore, the pia was not violated in either case. As such, although possible, it is unlikely that the infection was introduced through the craniectomy. Both cases presented after craniectomy with convex and enlarged pseudofontanelle. In cases of large stroke, we typically expect a sunken or concave fontanelle. Therefore, failure of the fontanelle to sink after craniectomy for large stroke should prompt radiographic examination before cranioplasty to delineate potential abscess from other causes such as hydrocephalus or contralateral subdural hematoma development.

Treatment of these reported abscesses varied among reports. In 1 case out of the 13

reported, the patient recovered with medical therapy only (antibiotics). The overall mortality was 5 of 13 patients (38.5%). All but 1 reported that survivors had undergone surgery to evacuate the abscess. Our own experience mimics that, Although our first patient did not worsen on antibiotics clinically, the abscess progressed, and he required evacuation of the affected area. One limitation is the likelihood of publication bias, given the small number of cases reported and the nature of retrospective case reports. Despite this, the dismal outcomes reported in the literature and our experience support extensive surgical removal of abscess located in a previous stroke. With regard to timing of our cranioplasty, there is no specific literature discussing timing after intracranial abscess drainage. We performed each cranioplasty after the completion of the antibiotic course, with imaging (CT) showing no recurrence of abscess, and with a sunken pseudofontanelle.

## CONCLUSION

Intracranial abscess is a rare but reported complication of stroke. However, if not treated properly and timely, the abscess could lead to fatal outcome. It is imperative for clinicians to be aware of the possibility of abscess formation after an infarction to make the correct diagnosis in a timely fashion. Radiographs, particularly a ring-enhancing hyperdense lesion on diffusion weighted MRI or a ring-enhancing hypodense lesion on CT, reflects the presence of an abscess. The literature review, as well as our own case presentations, suggests that treating the abscess with conservative antibiotics was usually not enough. Extensive surgical resection of the abscess with antibiotics treatment after surgery offers the best outcome for patients.

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