

Ischemic Stroke Complicated by a Brain Abscess: A Case Report and Review of the Literature

ABSTRACT

Ischemic stroke and brain abscess are life threatening, severe medical conditions requiring urgent and comprehensive evaluation. Brain abscess is a very rare and dangerous complication of ischemic stroke that can be treated successfully when diagnosed. Association of these two life threatening condition may cause catastrophic results.

In this report, we present our experience with an ischemic stroke case complicated by a brain abscess under the light of previous literature. A 64-year-old patient with a cerebral infarction complicated by a cerebral abscess is reported with its clinical and radiological features.

Because of their insidious and opportunistic nature, brain abscess complicating cerebral infarcts are pathologies requiring a high index of suspicion for proper treatment and favorable results.

KEY WORDS: Brain abscess, Cerebral infarction, Ischemic stroke, Cerebrovascular accident.

INTRODUCTION

Ischemic stroke remains the third leading cause of death after heart disease and cancer in the United States (10). Patients with a cerebral infarction are vulnerable to severe complications because of their morbidities. Although it is rare, abscess formation over the ischemic brain tissue can be one of these complications leading to further challenges in medical management. We describe a case with a cerebral infarction complicated by a brain abscess and try to highlight the important issues about this complication and difficulties in reaching the exact diagnosis with the help of previous reports.

CASE REPORT

A 64-year-old man suffering from rapid onset aphasia and progressive weakness in his right leg was evaluated on July 13, 2003. His neurological examination revealed moderate deterioration in his speech and a mild monoparesis (4/5) in his right leg. His routine laboratory investigation was normal. His computerized tomography (CT) was compatible with the changes attributable to his age, without any signs of an acute process. His medical history was unremarkable except for hypertension and a surgery for knee prosthesis. The diagnosis was transient ischemic attack after rapid improvement in his neurological signs. Further investigation revealed a small plaque in his left internal carotid artery with no hemodynamic alterations and an aneurismal dilatation at the cardiac apex with a suspicious thrombus within. The decision was to follow him up with antiplatelet therapy. Unfortunately

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he presented with alteration in consciousness and right hemiparesis (3/5) on September 23. His radiological and laboratory investigation on admission were again normal. However, his control CT revealed a left occipito-parietal hypodense lesion compatible with left posterior cerebral artery infarction (Figure 1). The patient was hospitalized and given proper treatment. The diagnosis was ischemic stroke.

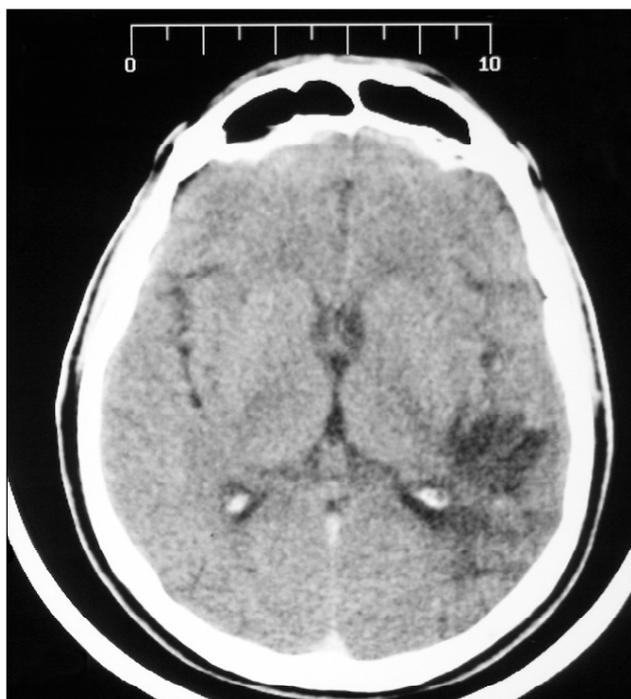


Figure 1: CT image demonstrating left occipito-parietal hypodense lesion compatible with left posterior cerebral artery infarction.

After his discharge with moderate deficits, he was readmitted because of high fever and decreased level of consciousness on December 19, 2003. His body temperature was 39.3 C. His laboratory findings supported an acute infectious process: erythrocyte sedimentation rate 90 mm/h, C-reactive protein 58 mg/dl, hemoglobin 15.2 g/dl, white blood cell count 18700/mm³ and slight hepatic dysfunction with relevant values. His neurological examination revealed a right hemiparesis that was interpreted as a sequel of his previous stroke, and diminished consciousness. The chest x-ray and urinalysis results were normal. His cranial tomography with contrast medium showed the previous infarct site at the left occipito-parietal region. Additionally, there was another lesion closer

to the lateral margin of the infarct site that was shaped like a ring, showed hyperdensity and was nearly 1 cm in diameter (Figure 2). The lesion was not demonstrating any mass effect and there was no obvious edema around it. The radiological interpretation was not clear because of the doubt whether the lesion was a hematoma in the absorption phase or an abscess. Magnetic resonance imaging (MRI) could not be performed to rule out



Figure 2: Patient's enhanced CT revealing a ring-like shaped lesion over the previous infarct

any of these diagnoses because of his incompatible knee prosthesis. Given the possibility of a brain abscess, a cerebrospinal fluid (CSF) examination was performed. The opening pressure was 160 mmH₂O. The laboratory results were as follows: leukocyte count 2700/mm³ with 96% neutrophils, glucose level 0.3 mmol/L and protein level 5.8 g/L. The gram stain and the culture of the CSF were negative, just like the other samples (urine, sputum and blood). Since the CSF findings were compatible with a central nervous system infection, he was administered high dose, empirical antibiotic therapy (imipenem 6 gr/day). We did not perform a surgery not only because of the patient's unstable cardiac condition but also because the lesion's small size allowed us to follow the patient up. The patient improved gradually with antibiotics

and supportive management. The control CT on the 2nd week of therapy showed a decrease in the size and enhancement of the lesion. Following intravenous antibiotic therapy for 6 weeks, He was discharged with mild deficits. His post-discharge control tomography was free of the described lesion (Figure 3).

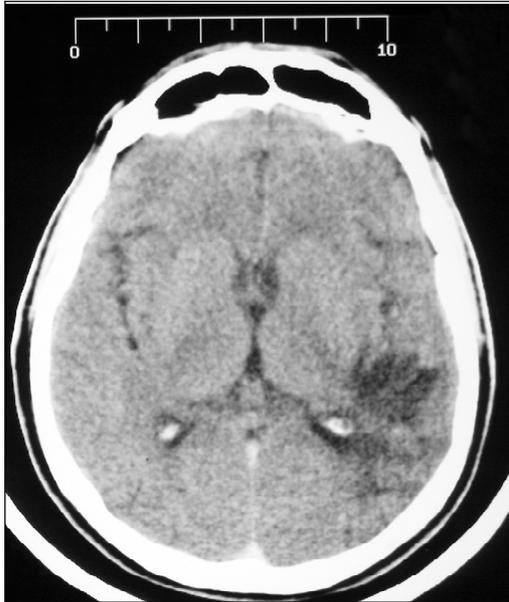


Figure 3: Patient’s post discharge control tomography was free of the described lesion.



Figure 4: The CT image of Borm et al’s patient that was misinterpreted as an infarct. Notice the finger-like edema through the cerebral cortex (image reprinted with permission).

DISCUSSION

Stroke has been defined as "rapidly developing clinical signs of focal or global disturbance of cerebral function with symptoms lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin". Ischemic stroke is the most common type and is the result of decreased blood flow to a portion of the brain; hemorrhagic stroke is the result of bleeding into brain.

Ischemic stroke remains the third leading cause of death after heart disease and cancer in the United States (10). Rates for males range from 340.3 cases per 100,000 population in the Russian Federation to 58.7 cases per 100,000 in the United States (16). An estimated 700,000 strokes occurred in the U.S. in 2002, with nearly 500,000 cases being first events with mortality rates ranging from 39 cases per 100,000 in New York State to 80.8 per 100,000 in South Carolina. The global burden of mortality was estimated at 4.7 million in 1995 (16).

These huge numbers underlines the importance of the event itself.

On the other hand, stroke is one of the most complicated pathologies. Patients with stroke are at risk of developing a wide range of complications secondary to their stroke that increase the mortality and morbidity rates. Although studies prove that the leading cause of death within a few days of stroke are usually the result of the brain damage itself, it is clear that deaths occurring after a few weeks are mainly because of potentially preventable problems such as infection, venous thromboembolism or cardiac disease (7) (Figure 4). The proper management of these complications such as infections, deep venous thrombosis or cardiac problems is therefore as vital as management of stroke. There are many reports in the literature about this issue (10).

Intracranial abscesses are also life-threatening medical conditions with a mortality rate of 30 – 70% (14), and a yearly incidence of 1.3 cases per 100,000 population. There is a 3:1 male preponderance of brain abscess, the reason for which is unclear. As expected, the incidence among children and patients older than 60 years is much higher (15).

Brain abscess is a focal intracerebral infection that begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well-vascularized capsule. There are four major routes for an infection to reach the brain: (a) spread from a contiguous focus (i.e. most often from middle ear,

mastoid cells and paranasal sinuses), (b) hematogenous spread to the brain from a distant focus, (c) trauma, (d) through the peripheral nerves (i.e. rabies or herpes). One other group can be constituted by immune compromised patients regarding their special condition. Among these routes, brain abscesses due to hematogenous spread have the highest mortality rates (12, 18).

Brain abscesses can be bacterial, fungal, or parasitic. Certain bacteria, fungi, and protozoa have been observed with increasing frequency as etiologic agents in immune compromised patients.

Diagnosis is made in the light of clinical findings such as fever, headache, focal neurological deficit, or seizures. Although it could be easy to interpret a lesion with a surrounding ring-like enhancement as an abscess in enhanced CT, one should always keep in mind that there are various lesions that can mimic the classic ring-like appearance of an abscess, most notably a necrotic tumor. On the other hand, it is well known that the contrast medium itself can cause a misinterpretation. Because of the increased vascularity of the penumbra after the stroke, contrast enhancement, which is known as luxury perfusion, can be observed and this may lead to a diagnosis of a brain tumor or an abscess. When possible, further investigations such as MRI, lumbar puncture or even biopsy should be utilized rapidly to clarify the exact nature of the lesion. The reverse correlation between a favorable outcome and the time spent until the initiation of the therapy should be kept in mind.

The major therapeutic choices are antibiotic therapy and surgical treatment. In fact, these two are complementary approaches rather than being individual treatments. Surgery is preferred for decompression and obtaining samples. Mostly, a combined regime is the treatment of choice with wide spectrum antibiotics. A 6-week course of intravenous antibiotics is commonly prescribed. More recently, a short intravenous therapy in the hospital followed prolonged oral treatment on an outpatient basis has been proposed (15).

Brain abscess following ischemic stroke is a rare situation as a complication. Although infectious challenges such as sepsis, and urinary or pulmonary infections are always at the scene for an infarcted patient, brain abscess formation is not so common. Several authors suggest that the disruption of the blood brain barrier, followed by bacteremia may

lead to abscess formation (3, 4, 5). Since stroke patients already harbor lots of medical pathologies that are also predisposing factors for stroke, such as diabetes, another point of view is that the sum of all of these disorders after stroke is a facilitating factor for infections and brain abscess.

Although there is always the possibility of a septic embolism from an infectious focus to the brain, brain abscess formation over a cerebral infarct is a rare entity. Since our patient is one of these rare cases, we found it worthy of note in the light of previous reports on this subject.

A comprehensive search of the English literature using Pub-Med revealed only seven reports of ischemic stroke cases complicated by a cerebral abscess. There are also two other case reports, one published in German in 1984 (1) and another in Japanese in 1989 (9) on this subject but we were not able to review and include them in our paper because of difficulties in reaching these papers, mainly due to their old publishing dates. We also added the data of our case to the review.

The cases were published between 1995 and 2005. Five of the reports were prepared as case reports (2, 3, 6, 13, 17); two as a case report and review of the literature (11 and this report) and one was described in a letter to the editor (8) which was a response to the previously published report of Chen et al (6) on the same subject. In two of the reports, the one from Chen et al (6) and the one from Kaplan et al (11), patients with hemorrhagic strokes complicated by brain abscess were also presented. Since we aimed to interpret complicated ischemic strokes, the most common type of stroke, these data were excluded for consistency concerns.

Data obtained from the reports and features of the cases are presented in (Table I).

The mean age of the cases reviewed was 55.7 (ranging between 16 and 77). There were 3 females and 5 males. A medical history was mentioned in most of the cases. The medical histories included the most common clinical entities that, in particular, are usually described as predisposing factors for stroke, such as, diabetes mellitus, hypertension, ischemic heart disease and atrial fibrillation. Besides these findings, one of the cases was healthy previous to the stroke (17) and one had a diagnosis of intermittent porphyria (10), which can also be a rare cause of ischemic stroke via cerebral arterial narrowing (4).

Table I: Analyses of Previously Reported Cases and Our Case

Author / Publication Date	Age / Sex	Medical History	Neurological findings after stroke	Infarct	Systemic findings prior to abscess detection	Neurological alteration leading to abscess detection
Arentoft et al, 1993 Autopsy	59 / F	Chronic renal failure	Coma and clonic seizures	Right MCA	Patient died before abscess diagnosis	Patient died before abscess diagnosis
Chen et al, 1995	70 / M	NA	Left hemiparesis	Right MCA	Vomiting	Sudden onset anisocoria
Davenport et al, 1995	16 / F	Intermittent Porphyria	Aphasia, right hemiparesis	Left MCA	Worsening headache, vomiting, drowsiness	Deterioration in language functions
Shintani et al, 1996	40 / M	Healthy subject	Sudden visual decrease in visual acuity of the left eye	Right PCA	Worsening headache	Stable
Beloosesky et al, 2002	68 / M	HT, NIDDM, IHD, alcohol	Aphasia, right hemiparesis	Left MCA	Fever, general medical deterioration	Deterioration in language functions and progress in right sided weakness
Miyazaki et al, 2004	77 / M	AF, HT, TIA	Diminished conscious, aphasia, right hemiparesis	Left MCA	Fever	Deterioration in conscious
Kaplan et al, 2005	52 / F	NA	Left facial paralysis and left hemiparesis	Right MCA	Worsening headache	Stable
Present case	64 / M	HT, Prosthesis surgery	Diminished conscious, aphasia, right hemiparesis	Left PCA	Fever	Deterioration in conscious

NA: Not available; HT: Hypertension; NIDDM: Non-insulin dependent diabetes mellitus; IHD: Ischemic heart disease; AF: Atrial fibrillation; TIA: Transient ischemic attack; MCA: Middle cerebral artery; PCA: Posterior cerebral artery.

Table I: Continued

Author / Publication Date	Interval till abscess detection	Intervention	Source of infection / origin	Pathogen	Result
Arentoft et al, 1993	Detected on autopsy	NA	Unknown Origin	Salmonella typhimurium	Exitus
Chen et al, 1995	5 weeks	Stx. Asp and antibiotics	Aspiration pneumonia	Culture negative	Exitus
Davenport et al, 1995 Letter to the editor	4 weeks	Stx. Asp and antibiotics	Septicemia	Escherichia coli Enterococcus faecalis	Recovery
Shintani et al, 1996	2 weeks	Surgery and antibiotics	Unknown Origin	Streptococcus	Recovery
Beloosesky et al, 2002	16 weeks	Surgery and antibiotics	Urinary infection	Proteus mirabilis	Exitus
Miyazaki et al, 2004	55 weeks	Stx. Asp and antibiotics	Pneumonia	Methicillin resistance staphylococcus aureus	Recovery
Kaplan et al, 2005	8 weeks	Aspiration and antibiotics	Pneumonia	Staphylococcus aureus	Recovery
Present case	6 weeks	Empirical antibiotic treatment	Not known	Not known	Recovery

All the infarcts determined (6 cases) were in the middle cerebral artery (MCA) territory except two cases which had an infarction in posterior cerebral artery (PCA) territory.

Excluding the case whose brain abscess was diagnosed by an autopsy (2), the mean time interval between the diagnosis of a cerebral infarction and the detection of a brain abscess was 13 weeks (ranging from 2 weeks to 55 weeks). The mean time interval is almost 7 weeks after excluding the great influence of the case described by Miyazeki et al (13), whose abscess was diagnosed 55 weeks after stroke.

The clinical progress and the final outcomes of the cases, after appropriate management, are relatively acceptable. There were 3 (37.5%) deaths and 5 (62.5%) recoveries. For cases with previously injured brain tissues due to an ischemic stroke, it seems that an abscess forming in the damaged area is not as fulminant as a brain abscess developing in the normal brain.

One may claim that interpreting the time interval between the first stroke and the abscess detection is not an appropriate method since the patients certainly did not harbor a brain abscess when they had their first stroke. However, in our opinion this time interval is important for several reasons. First of all, as described previously, most of the cases are insidious and opportunistic without any remarkable signs of a developing brain abscess. Even healthy and stable patients are vulnerable to this kind of complication from the very first day of their stroke. Second, the time of this complication cannot be anticipated. The diagnosis is nearly always challenging and the most important factor seems to be the physician's index of suspicion. These patients should be followed meticulously and a comprehensive evaluation is required when there are any signs of any medical alteration which could be as simple as persistent fever. Although most of the authors find their management inadequate, attributing this to "malpractice" would be unfair since the complexity of these cases is obvious. In the majority of the cases described, including our own, the time interval between the initial damage and the abscess formation is too long. The most logical explanation is that disruption of the blood-brain barrier facilitates abscess formation and the damaged brain, which is a preferred target for infectious agents, remains a vulnerable zone for a long time.

In this era of CT and MRI, it is nearly a must to perform a CT when the patient demonstrates new neurological deficits. However, performing a CT can sometimes provide misleading results. We therefore recommend performing at least an enhanced CT or even MRI when possible for evaluating stroke patients demonstrating new focal deficits or alterations in their neurological condition. The case described by Borm et al (5) supports our recommendation. A 56-year-old non-immunocompromised man who experienced acute right-sided hemiparesis and aphasia without any signs of infection was presented. The patient's initial enhanced CT revealed a hypodense area with slight contrast enhancement that was attributed to post-infarction luxury perfusion by a radiologist. Two months after the initial event, the patient deteriorated again and this time the MRI scan revealed a polycystic, enhancing mass. The patient again did not have any infectious symptoms. He was operated on and a *Nocardia asteroides* brain abscess was confirmed. Borm et al. link their case's initial symptoms to the brain abscess that was already present in his initial CT rather than cerebral ischemia. They think that their patient's initial CT was misinterpreted by an inexperienced radiologist who falsely diagnosed a developing brain abscess in its cerebritis phase as an infarction (Figure 4). They conclude that "an early infection of the brain can occur with symptoms of stroke and a developing brain abscess may present without laboratory findings." This case represents a good example of the importance of radiological and laboratory findings in patients demonstrating acute focal neurological deficits and how insidious and silent brain pathologies can be sometimes.

Although there are limited numbers of cases complicated by a cerebral abscess, the true incidence of this complication is not known. There are various reports in the literature considering infections in stroke patients as general complications. Taking into account the similar findings of sepsis and brain abscess in stroke patients, it is hard to distinguish these cases and the true incidence remains unclear and may be higher than assumed since we do not know whether reported cases include a brain abscess or not.

CONCLUSION

In this era of evidence-based medicine with various technical improvements, amazing radiological devices and effective therapeutic measures, it is surprising to see that diagnosis of a cerebral abscess complicating a cerebral infarct is still depended on general medical practice principles and the physician's suspicion.

Cerebral abscess should be suspected in patients with previous cerebral infarcts suffering from bacteremia and progressive focal signs. The fact that these patients may not demonstrate any clinical and neurological alterations necessitates comprehensive evaluation. Since these patients are usually old and harbor various medical problems besides their stroke, every effort should be made to prevent such insidious complications.

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