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CASE REPORT

Brain abscess complicating cerebral infarct

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Abstract

Presentation: we report a case of a 68-year-old man who suffered ischemic strokes in the left middle cerebral artery territory and three months later, following urosepsis, developed a cerebral abscess in the infarcted area.
Discussion: a literature search found only eight other cases. We discuss herein the common clinical aspects of brain abscess complicating strokes, the co-existent diseases, and point out the possibility of underreporting this rare but treatable complication.
Conclusion: cerebral abscess should be suspected in patients with a previous brain infarction or haemorrhage, who develop bacteremia and impaired consciousness without a clear explanation to their condition. Advanced age, and medical conditions known adversely to affect immunological competence reinforce the clinical suspicion.

Keywords: *brain abscess, ischemic stroke, complication*

Introduction

Stroke is a leading cause of mortality and morbidity affecting 750,000 people each year in the USA alone [1] with a 30-day mortality rate of up to 14%. Common serious complications after a stroke are pulmonary and urinary infections [2, 3]. Areas of ischemia, infarction and contusions in the brain can provide fertile ground for inoculation or bacteremic seeding of organisms resulting in abscess formation. However, in the absence of cerebral lesions, brain abscesses are extremely rare in septic conditions such as pyelonephritis, pneumonia and vascular catheterization [4].

Abscess formation in patients with endocarditis is usually accompanied by microemboli or cerebrovascular disease resulting in locally decreased oxygenation or perfusion [5]. Recently in a stroke that became infected with abscess formation, 'septic infarction' was reported as a complication of endocarditis [6].

We report a patient whose large cerebral infarct was complicated by a brain abscess in the course of urosepsis.

Case report

A 68-year-old male with a long history of hypertension, non-insulin dependent diabetes mellitus, stable ischemic heart disease, alcohol abuse and an implantation of a penile prosthesis 7 years ago suffered an abrupt onset of right dense hemiparesis with aphasia and impaired consciousness. A left hemispheric middle cerebral artery (MCA) stroke was diagnosed clinically.

A CT scan performed several hours after admission showed mild cerebral atrophy with right basal ganglia lacunar infarcts. The patient was transferred to a rehabilitation hospital where substantial motor improvement was achieved. He remained with a moderate right spastic hemiparesis, yet he was able to stand up and walk with a walker with some assistance.

Two months later, in the rehabilitation hospital, he developed a severe urinary tract infection. Two days later, there was an abrupt deterioration in his neurological status, mainly with respect to his speech and his right-sided weakness. He became aphasic and was unable even to sit in bed due to severe right hemiparesis. A recurrent

left hemispheric stroke was diagnosed, but no imaging was performed at that time. A month later, although some motor improvement had been gradually achieved, he developed a high fever followed by general medical deterioration and was transferred to our department.

On admission, the patient had a temperature of 38.8°C, a blood pressure of 206/94 mmHg and a pulse rate of 100/min. Physical examination revealed a stuporous patient with dense right hemiparesis along with motor and sensory aphasia. Examination of the lungs, heart, abdomen and ocular fundi was unremarkable. An indwelling urinary catheter produced turbid urine.

After blood and urinary cultures were drawn, intravenous ceftriaxone and amikacin were administered. During the next few days, an extensive work up was performed: chest x-ray, urinary and abdominal ultrasound, abdominal and chest CT scans were all normal.

Since his neurological condition did not show any improvement, a brain CT scan without contrast was performed on day 8 of his admission and showed a large hypodense left temporo-parietal area with some oedema (Figure 1). Blood and urine cultures disclosed *Proteus mirabilis*; a lumbar puncture was done, as a diagnosis of complicating meningitis was raised. The cerebrospinal fluid content was consistent with a partially treated bacterial meningitis: 250 cells (52% polymorphonuclear and 48% lympho-monocytic cells), protein 191 mg/dl, glucose 148 mg/dl (serum glucose 282 mg/dl), with a positive culture for *Proteus mirabilis* sp. Consequently, the antibiotics were changed to imipenem and gentamycin.

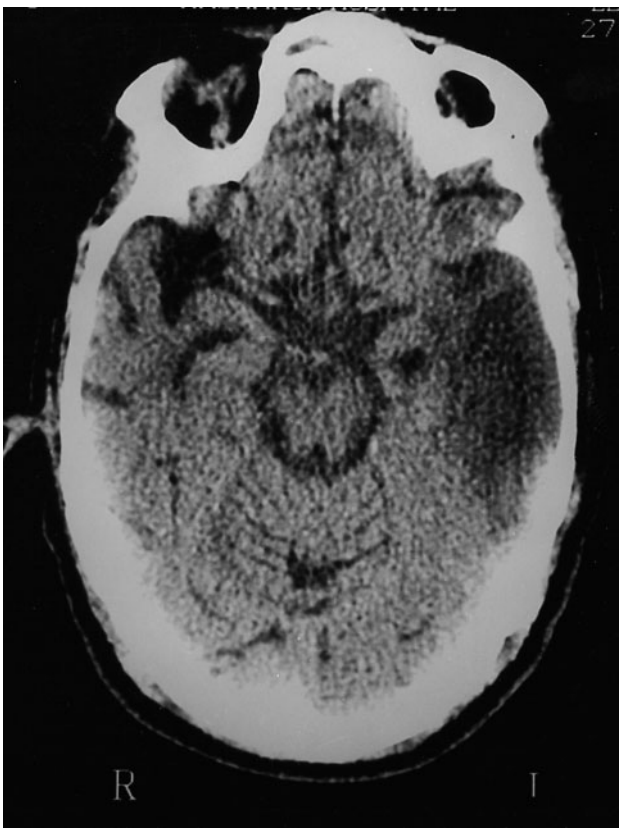

Figure 1. An unenhanced brain CT scan cut, showing a large hypodense lesion with some surrounding oedema compatible with old and recent temporal infarcts.

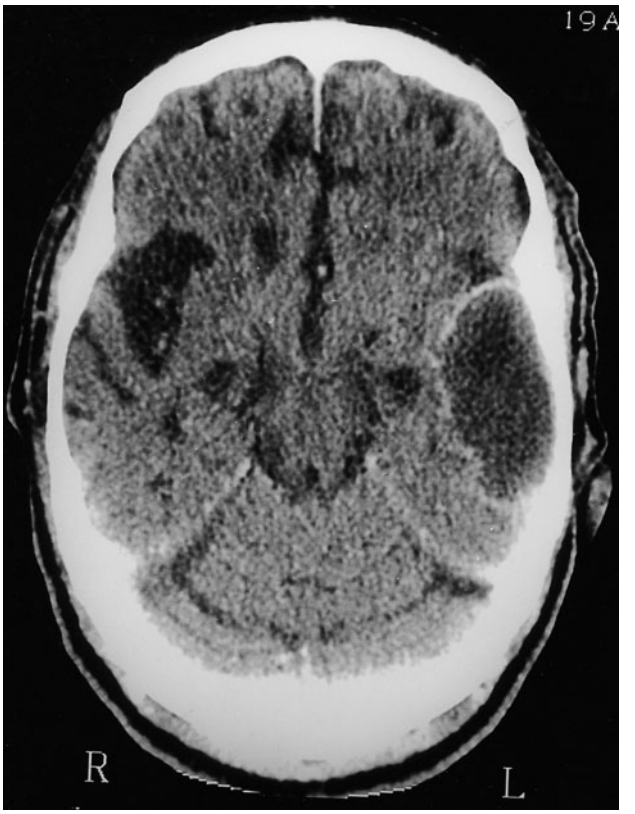

Figure 2. A similar CT scan cut done (with contrast) two weeks later, showing large ring enhancement in the previously hypodense area compatible with a brain abscess.

Two weeks later, despite appropriate treatment, the patient's condition did not improve. A new brain CT scan with contrast demonstrated ring enhancement surrounding the large hypodense area seen in the previous brain CT scan (Figure 2).

The patient was transferred to the neurosurgical department with the diagnosis of a brain abscess. A left temporal craniotomy was done exposing a partially vascular, moderately thick capsular abscess. The capsule was separated from the surrounding oedematous brain tissue, and purulent material was drained, from which *Proteus mirabilis* was subsequently isolated. During the next few days, a net amelioration was noted but unfortunately the patient died a week later from cardio-respiratory arrest.

Discussion

The combination of a sudden onset of a right dense hemiparesis with aphasia along with a mild impairment

of consciousness indicated that the patient initially had had a fairly large infarct in the left MCA territory. The first CT scan, performed early in order to exclude haemorrhage, did not show the infarct. After 2 months of rehabilitation, while suffering from a urinary infection, the patient had a second stroke. The increased risk of stroke in the course of bacterial infections or septicemia is well known [7]. The mechanisms that could cause thrombo-embolic complications in septic patients are numerous but activation of the coagulation system is probably the most important [8].

We are quite certain that most of the large non-congruent hypodense temporo-parietal area demonstrated in the second brain CT scan was the result of the two previous infarcts in the MCA territory (Figure 1). The absence of marked brain oedema and mass effect in the follow up CT scan, in spite of the abscess formation, is explained by the large previously infarcted area enabling the development of inflammatory and oedematous process, without marked pressure on adjacent cerebral tissue. Although such an abscess can develop in a deep lacune and expand, or arise de novo as a septicemic abscess, it is most unlikely in our patient who had two sudden, dense hemiparetic and aphasic strokes.

Since the second CT (Figure 1) scan was performed without contrast medium, we cannot firmly determine when exactly the infarcted area became infected and the abscess formation began. Nevertheless, there are no significant volume or configuration differences between the second and the third CT scans (Figure 2). We therefore conclude that the abscess formation began during the first week while the patient suffered severe sepsis. An enhanced CT might have achieved earlier diagnosis of abscess formation. However, it is well known that in the subacute stage of a stroke, contrast medium can appear as vascular malformation due to luxury perfusion or as an enhancing 'ring lesion' suggesting a brain tumour. For technical and logistic reasons brain MRI was not done. Undoubtedly, an early MRI is more sensitive than a CT in detecting a brain abscess in the cerebritis phases of its development, and in detecting associated cerebral oedema [4]. Cerebritis appears on MRI as an area of low signal intensity on T1-weighted images with irregular post-gadolinium enhancement and as an area of increased signal intensity on T2-weighted images. Yet on top of an infarct this may not show up so well. MRI spectroscopy may permit differentiation of brain abscesses from necrotic or cystic tumours, subacute infarcts, demyelinating diseases and other non-neoplastic processes [9]. The antibiotic therapy stabilized and limited the bacteremia but not the abscess process, once the infarcted area was affected.

Brain abscess formation in previously haemorrhagic or infarcted areas is very rare. A literature search found only five cases of abscesses in infarcted tissues [10–14] and three in haemorrhages [12, 15, 16].

Several authors suggested that the disruption of the blood-brain barrier, followed by bacteremia may predispose the affected brain tissue to infection and thus to abscess formation [12, 14]. Others proposed that in endocarditis emboli, once the infarction had occurred, luxury perfusion-reperfusion or infection of collateral vasculature might allow secondary infection of the penumbral zone [6].

Although the information on these eight cases is limited, it is worthwhile to note that some of these cases had immunological defects known to facilitate infections: transient pancytopenia [14], renal failure [11], diabetes [10] and in our case the association of diabetes and alcoholism. Clinical evidence of bacterial meningitis preceded the definite diagnosis of brain abscesses in two cases [12, 14] as in ours. Indeed, brain abscess can complicate bacterial meningitis. Although this is rare in adults, it is common in neonates particularly in meningitis due to *Proteus* sp. or *Citrobacter* [17, 18]. The rarity of abscess development in an infarcted area renders this diagnosis difficult and necessitates a high index of suspicion. Retrospectively, we should have repeated the contrast CT scan earlier or demanded an MRI in order to diagnose and treat this uncommon complication sooner. Lumbar puncture was done before the contrast CT scan, thus prior to diagnosing the brain abscess; spinal tap could be hazardous in the setting of a ring-enhancing lesion producing mass effect and a midline shift but not when minimal oedema is present as in this case.

In the majority of the cases described [10, 12–14, 16] including our own, several months elapsed between the initial brain damage and the abscess formation, with the focal damage remaining a vulnerable zone for further infection.

Previous authors were intrigued by the rarity of this complication of stroke, given the frequency of potential sources of septicemia after an acute stroke [14]. Others estimated that there might be more underreported cases of this complication [19]. One can argue that the rarity of this complication on one hand and the common features shared by both sepsis and cerebral abscesses on the other hand (in stroke patients), might be the cause of some other undiagnosed cases.

We suggest that cerebral abscess should be suspected in patients with previous brain infarction or haemorrhage, suffering from bacteremia, progressive focal signs and deteriorating level of consciousness. Advanced age, meningitis and medical comorbidities known to adversely affect the immunological potency reinforce the clinical suspicion.

Key points

- Bacterial infections and septicemia increase the risk of stroke.
- Cerebral abscess should be suspected in aged patients with previous brain infarction or haemorrhage, suffering from bacteremia, progressive focal signs and deteriorating level of consciousness without other clear explanation.

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