Intracranial Calcification Caused by a Brain Abscess: A Rare Cause of Intracranial Calcification

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Intracranial calcifications are relatively common computed tomographic findings in the field of neurosurgery, and cysticercosis, tuberculosis, HIV, and cryptococcus are acquired intracranial infections typically associated with calcifications. However, intracranial calcification caused by a bacterial brain abscess is rare. Here, we present a rare case of intracranial calcification caused by a bacterial brain abscess, from which staphylococcus hominis was isolated. To the best of our knowledge, no previous report has been published on intracranial calcification caused by bacterial brain abscess after decompressive craniectomy for traumatic brain injury. In this article, the pathophysiological mechanism of this uncommon entity is discussed and relevant literature reviewed.

Key Words: Brain abscess · Calcification.

INTRODUCTION

Intracranial calcifications observed by computed tomography (CT) are the most common incidental finding in the everyday practice of neurosurgery, because non-contrast enhanced CT of the brain is the preferred imaging modality worldwide for the initial evaluation of patients with head injury. Cysticercosis, tuberculosis, and cryptococcus are acquired intracranial infections typically associated with calcifications. However, intracranial calcification resulting from a brain abscess is extremely rare. We represent a rare case of an unusually calcified brain abscess, from which staphylococcus hominis was isolated, and include a review of the literature.

CASE REPORT

A previously healthy 40-year-old man presented with mental deterioration after a head injury. On neurological examination, he was stuporous with a Glasgow Coma Scale of 8. Routine hematological and serum chemistry testing, including erythrocyte sedimentation rate and C-reactive protein, revealed no abnormality. However, CT scan of the brain revealed severe brain swelling and hemorrhagic contusion in the right frontotempo-parietal (FTP) region. He underwent emergent decompressive craniectomy on the right FTP region (Fig. 1). Postoperatively, he became conscious and was able to obey commands without any neurologic deficit. However, three weeks later, he complained of a febrile sensation and had a temperature of 38.4°C. A CT scan of the brain with intravenous contrast showed a ring-enhanced brain abscess (Fig. 2). Third generation cephalosporin and sufficient hydration were continuously administered. However, despite this management, the fever continued and he complained of mild left hemiparesis (Grade IV+/Grade IV+). A CT scan and magnetic resonance imaging (MRI) with intravenous contrast on hospital day 35 revealed an increase in both the size and extent of the brain abscess (Fig. 3). Navigation-guided culture and biopsy following drainage of the brain abscess using a 5 L catheter were performed. A dark yellowish pus was drained from the abscess. Gram stain and acid-fast bacilli cultures were negative and revealed no organisms. No evidence of granulomas or larval cysts were seen. However, from cultures of the purulent drainage on blood agar, coagulase negative staphylococcus hominis was isolated. His antibiotics were changed to nafcillin and ciprofloxacin based on the results of
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ticercosis, can cause intracranial calcifications1,12. However, bacterial brain abscesses rarely calcify and CT findings of calcified brain abscesses have seldom been reported. The features of cerebral abscesses unveiled by MRI reflect the underlying pathophysiology of abscess formation. During the initial cerebritis stage, an ill-defined subcortical hyperintense zone on T2-weighted images is associated with poorly delineated enhancing areas within an iso-to mildly hypointense edematous region on enhanced T1-weighted images. During the early and late stages, a collagenous abscess capsule is visible on unenhanced scans as a comparatively thin-walled, well-delineated iso- to slightly hypointense ring that becomes hypointense on T2-weighted im-

culture test, and after 6 weeks on antibiotics, he achieved complete clinical recovery except for mild hemiparesis. However, follow-up CT performed 5 months after admission revealed that the brain abscess had developed into a large calcified mass (Fig. 4). Because of its deep-seated location and a lack of clinical symptoms, surgical removal was not performed. Instead, a series of CT scans were taken 3 monthly. No change in either the size or extent of the calcified region was observed over 2 year of follow-up. The patient remains symptom free with mild hemiparesis (Fig. 5).

DISCUSSION

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 capsule5. Brain abscesses develop in response to parenchymal infections by pyogenic bacteria and begin as a localized area of suppuration. Most commonly, infectious agents gain access to the central nervous system from a contiguous site of infection6. It is well known that a number of intracranial infections, such as, tuberculosis and cysticercosis, can cause intracranial calcifications1,12. However, bacterial brain abscesses rarely calcify and CT findings of calcified brain abscesses have seldom been reported. The features of cerebral abscesses unveiled by MRI reflect the underlying pathophysiology of abscess formation. During the initial cerebritis stage, an ill-defined subcortical hyperintense zone on T2-weighted images is associated with poorly delineated enhancing areas within an iso-to mildly hypointense edematous region on enhanced T1-weighted images. During the early and late stages, a collagenous abscess capsule is visible on unenhanced scans as a comparatively thin-walled, well-delineated iso- to slightly hypointense ring that becomes hypointense on T2-weighted im-

Fig. 1. Postoperative computed tomography scan shows successful removal of subdural hematoma after decompressive craniectomy.

Fig. 2. Contrast enhanced CT scan taken 3 weeks later reveals appearance of an intracerebral abscess with a thin walled cavity.

Fig. 3. Gadolinium enhanced magnetic resonance image and contrast enhanced CT taken 35 days later show an increase in both the size and extent of the brain abscess.

Fig. 4. After navigation-guided drainage of the abscess, CT scan performed 5 months after admission show calcified brain abscess.

Fig. 5. Axial and coronal CT scans taken 2 years after admission show remained calcified brain abscess.
CONCLUSION

We report a rare case of intracranial calcification caused by bacterial brain abscess. Although rare, bacterial brain abscess should be borne in mind as a possible cause of intracranial calcification.

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