

Encapsulated Chronic Intracerebral Hematoma Mimicking Neoplasm

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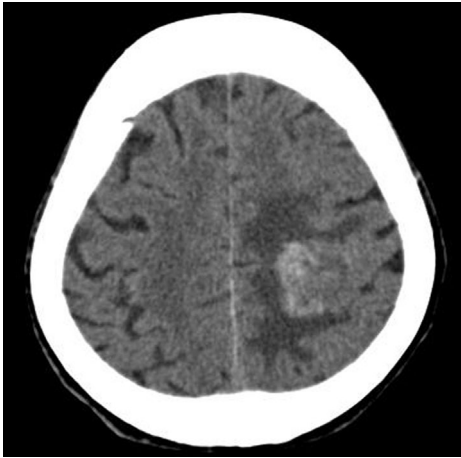


Figure 1. CT scan of brain revealed an irregular, hyperdense mass in the left superior parietal lobe, surrounded by a thin, hyperdense zone.

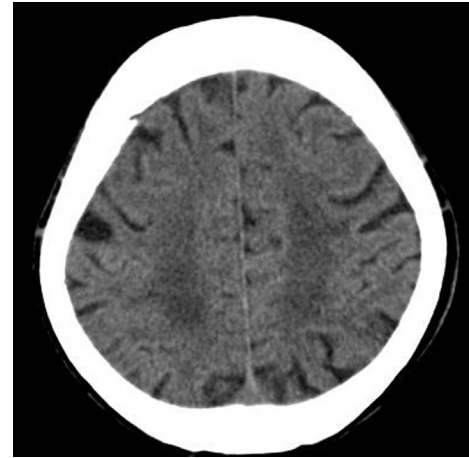


Figure 3. CT scan of brain one month later revealed resolution of hematoma with improvement of perifocal white matter edema.

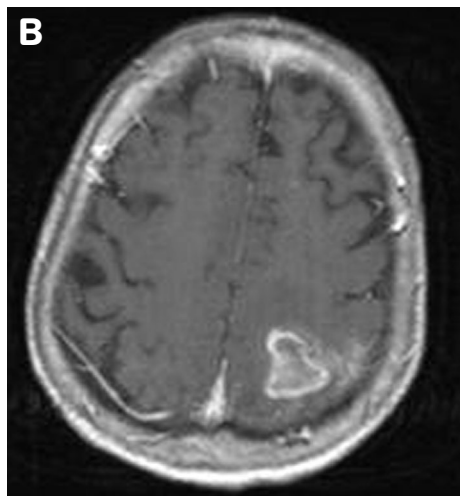
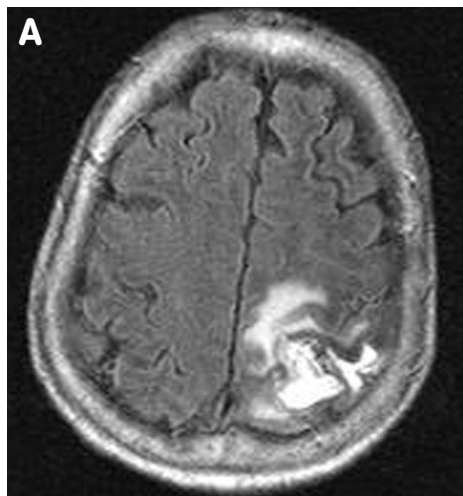


Figure 2. (A) Fluid-attenuated inversion recovery MR image revealed a ring-like and hyperintense lesion with perifocal edema. (B) Post-contrast T1 weighted MR image showed no additional enhancement within or adjacent to the hematoma.

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A 85-year-old woman presented with progressive weakness of right-sided extremities and an unsteady gait, followed by one seizure episode two weeks before admission. Physically, the patient was entirely normal. On neurological examination, she appeared to be drowsy. She had right-sided hemiplegia with hyperreflexia and presence of Babinski's sign. Laboratory examinations revealed no remarkable findings. Computed tomography (CT) scan of brain showed an irregular, hyperdense mass in the left superior parietal lobe, surrounded by a thin, hyperdense zone (Fig. 1). The mass lesion was also associated with perifocal white matter edema, which was not enhanced after contrast injection. Magnetic resonance (MR) imaging revealed a ring-like hyperintense mass lesion with perifocal edema in all sequences (Fig. 2A). The hemosiderin deposition was seen at the periphery of the hematoma with the central being high signal fluid space on gradient echo T2 star image. No additional enhancement within or adjacent to the hematoma was found in the post-contrast T1 weighted images (Fig. 2B). MR angiography revealed unremarkable findings. Although metastatic tumor with bleeding could not be strongly excluded based on the laboratory examination and neuroradiological findings, surgery was recommended for decompression and tissue proof. However, conservative treatment was chosen by the patient and her family. Clinically, the symptoms improved after medications. The follow-up CT scan of brain revealed resolution of hematoma with improvement of perifocal white matter edema (Fig. 3). No recurrence was noted during regular follow-ups.

Encapsulated chronic intracerebral hematoma, first reported by Hirsh et al. in 1981, is a rare disease entity and less than thirty cases have been reported in the literature⁽¹⁾. Commonly, intracerebral hematoma is usually absorbed and disappeared after two or three months, which leaving a residual small scar of cyst⁽²⁾. The forma-

tion of encapsulated intracerebral hematoma has been hypothesized as the exudation or bleeding from capillaries of neovascular channels in the granulated capsule tissue which was originating from fibroblasts⁽³⁾. Unlike the picture of spontaneous intracerebral hematoma, the clinical presentations of encapsulated intracerebral hematoma are characterized as gradual onset, slowly progressive neurological deficits or seizures⁽²⁾. Ring enhancement of intracerebral hematoma, representing both "luxury perfusion" of the surrounding brain and peripheral vascular granulation tissue, may be seen in the CT scan during the absorption of hematoma, but the absorbed hematoma does not commonly result in a mass effect or perifocal white matter edema⁽⁴⁾. Because the ring-like lesion on CT appearance and pseudotumoral clinical course, most patients are considered as harboring a brain tumor. Except for mass effect and deteriorating neurological deficits, encapsulated intracerebral hematoma could be treated by conservative management. Therefore, encapsulated intracerebral hematoma should be considered as one differential diagnosis for a ring-shaped lesion in a patient with progressive neurological deficits.

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