Acute Intracerebral Haematoma — An Unusual Presentation of Herpes Simplex Encephalitis

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Abstract
A young man presented with acute headache, behavioral abnormality and CT scan evidence of intracerebral hematoma. Detailed investigation established its infective origin due to herpes simplex virus type-1. The patient made an excellent recovery following a course of acyclovir. Frank intracerebral haematoma in the form of well defined mass on CT scan in patient with herpes simplex encephalitis is extremely rare.

INTRODUCTION
Herpes simplex encephalitis (HSE) is characterized by intense haemorrhagic necrosis of medial part of temporal lobes and orbital parts of frontal lobes. Usually the temporal lobe lesions are bilateral but they are not always symmetrical. The usual radiological features include a low-density lesion involving the medial temporal lobe and/or the insular cortex with or without mass effect in cranial CT. Sometime there may be contrast enhancement of adjoining meninges. CT scan usually detects the hemorrhage necrosis, which is a common neuro-pathological feature of HSE, as ill-defined linear streak of increased intensity. Rarely, however this may produce a well-defined mass. Here we are reporting a case of HSE who presented with acute intracerebral haematoma.

CASE REPORT
A 38 years old young doctor presented with acute severe bursting bifrontal headache, which later on became holocranial, and was disturbing his sleep, without fever, nausea or vomiting. He was initially treated with antibiotic and analgesics, which used to give him short-term relief. His general physical as well as systemic examination were unremarkable, except just palpable liver in per abdominal examination. His investigations at this point of time which included a haemogram, liver function test, urine R/E, skiagram of chest, and X-ray of paranasal sinuses were within normal limit. USG abdomen only confirmed the hepatomegaly. Examination of peripheral smear failed to demonstrate malaria parasite. A cranial CT scan done about a week after the disease onset revealed an acute haematoma in right frontal and temporal regions with perifocal oedema and mass effect (Fig. 1). He was referred to our Department for evaluation. At the time of admission the patient was having holocranial headache with irritability, increased anger, violent behaviour, and disturbed sleep. On questioning it was found that about four weeks before the disease onset patient had a febrile illness associated with follicular tonsillitis which was controlled by oral antibiotic within 10 days. However, he continued to have fatigue, unusual tachycardia, and sweating. There was no history of head injury preceding the onset of this illness. There was no history of bleeding from mucosal surfaces or beneath the skin. His general physical examination
was unremarkable. Neurological examination revealed a normal higher mental function except irritability, restlessness, and increased anger. The optic fundii were healthy with no haemorrhage. There was no meningeal sign. There was no other neurodeficit. Other system examination did not reveal any abnormalities.

His investigation showed a normal haemogram, and coagulation profile. The blood biochemistry including liver function and renal function tests were normal. His skiagram of chest, ECG, echocardiography and Doppler study were within normal limit. The EEG showed a normal awake record. A collagen vascular profile including C-reactive protein, rheumatoid factor, antinuclear antibody, and anti ds DNA were negative. Repeated attempts failed to isolate any pathogenic bacteria by blood cultures. The CSF examination revealed: normal opening pressure, colourless watery fluid with protein - 48 mg/dl, sugar - 80 mg/dl, chloride - 96 mEq/l, with few RBC and no white blood cell. The CSF DNA PCR for HSV-1 was positive. The IgG and IgM antibodies for HSV-1 and HSV-2 were negative. The CSF was also negative for acid fast bacilli and India ink preparation. The Myco-3 DNA PCR and radiometric culture for tubercle bacilli were also negative. A MRI scan of brain revealed (Fig. 2) features of late subacute haematoma in right frontal and temporal region. MR angiogram and venogram did not show any abnormalities.

We managed the patient conservatively with anticonvulsant and cerebral antioedema measures. He was also given a course of acyclovir (30 mg/kg/day for 14 days) through infusion. The patient made a gradual and almost complete recovery. His cranial CT done after six weeks revealed (Fig. 3) near total resolution of haematoma. A repeat MR scan done about eight weeks after the disease onset showed (Fig. 4) almost complete resolution of oedema and regression of haematoma. Whereas his behavioural abnormalities improved significantly, he continued to experience headache at the time of his discharge from hospital. When he was last seen at a follow up after six months he was totally symptom free with no neurodeficit.

**DISCUSSION**

Our patient presented with acute bursting headache with
behavioural abnormalities and CT evidence of intracerebral haematoma (ICH) in right frontal and temporal areas. He had history of febrile illness few weeks before the onset of headache. We considered the following possibilities: a) traumatic ICH, b) herpes simplex encephalitis, c) rupture of mycotic aneurysm following subacute bacterial endocarditis, and d) bleeding due to coagulation disorder. While a positive PCR of HSV-1 in CSF and localized lesion in inferior frontal and medial temporal region in neuroimaging have substantiated the diagnosis of HSE, investigations have excluded the other possibilities. Moreover the response to acyclovir has also favoured the diagnosis.

HSE is a fulminant necrotising meningoencephalitis caused by HSV type 1 and it is distinguished from other encephalitides by its focal and often haemorrhagic nature. Although the infection rapidly disseminates in the brain, the characteristic feature is the early involvement of temporal lobes as demonstrated by CT scan and radionuclide brain scan and EEG. The usual clinical features are fever, headache, seizure, confusion, stupor, and coma. Sometimes symptoms of temporal and inferior frontal lobe lesion e.g. olfactory hallucination, anosmia, temporal lobe seizure, a brief period of personality change, bizarre or psychotic behaviour or a delirium, aphasia, and hemiparesis precede these manifestations.

The three characteristic radiological features are low-density lesion, contrast enhancement and haemorrhage. The haemorrhage usually appears as ill-defined linear streak of increased density. Usually the CT scan findings appear after one week of disease onset. There are however, very few reports of intracerebral haematoma as the only presenting feature of HSE in cranial CT.

In our patient presentation was not fulminating, the usual symptoms like fever, seizure, confusion or altered sensorium were lacking. In the background of such clinical presentation the detection of ICH in cranial CT made this case more challenging. The laboratory evidences supported and therapeutic response had further strengthened the diagnosis of HSE.

**REFERENCES**


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**Announcement**

**CRITICARE 2004 - MUMBAI**

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