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An interesting case of Headache

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Abstract :
Brain abscesses are usually seen post surgery and trauma. Brain abscesses are described as a rare complication of bronchieactasis and are usually solitary.We present a case of 42 year old male who had developed bronchiectasis as a sequelae to pulmonary tuberculosis and presented to us with a right facio-brachial monoparesis. Neuro-imaging showed multiple cerebral abscesses which were managed both surgically and with antibiotics.

Keyword :bronchiectasis, brain abscess, tuberculosis Introduction: Brain abscesses are rare in today's world due to availability of higher antibiotics and prompt treatment of underlying condition. [1] However solitary brain abscess are seen as a extension of local pathology or from a hematogenous spread from a septic foci. Multiple brain abscesses are seen commonly in immune compromised individuals[2]. Bronchiectasis is a major cause of brain abscesses.[3] However it is rare to see multiple brain abcesses in a immune-competent person as a result of bronchiectasis.

Case History: A 42 year old male working as a manual laborer came with principal complains of a throbbing type , occipital headache for about 15 days. It aggravated with by bending forward, coughing and he had 6-7 bouts of projectile non-bilious, non-blood stained vomiting over the last 3 days. He took treatment at a local doctor for headache and was prescribed oral analgesics and anti-emetic agents. 6 days before admission, while on the way to work he had a sudden onset giddiness followed by a syncopal attack that lasted for about 2-3 minutes. According to the bystanders there was no seizure like activity. He regained consciousness and there was minimal confusion and carried on his work without any difficulty. One day before admission he had a sudden onset , single episode of Generalised tonic clonic seizure activity that lasted for 5 min involving all the four limbs. However there was no tongue bite , frothing at mouth , bowel and bladder incontinence. On enquiry he denied any aura just before the episode. Following this episode he noticed that he had slurred speech, facial deviation to the left and flaccid weakness in the right upper limb.

He is non diabetic, non hypertensive , occasional alcoholic and smokes 10 beedis per day. He was treated for pulmonary tuberculosis about 15 years back and received treatment for 6 months. He gave history of chronic cough with intermittent increase in the quantity and purulency on the expectoration for which he used to take treatment at local doctor.

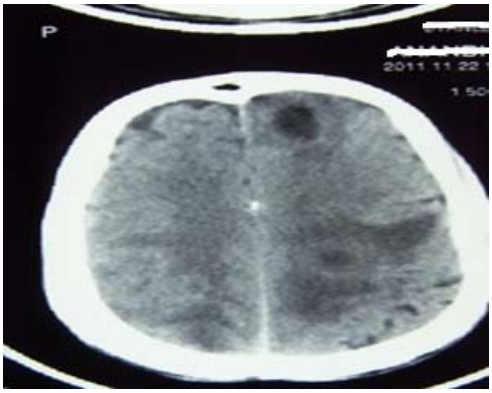

Figure 1(showing hypodense lesion with surrounding edema in the left frntal and parietal regions)

On examination, he was conscious oriented and cooperative. He had grade 3 pandigital clubbing. There was no pallor , icterus , cyanosis , significant lymph node enlargement pedal edema and elevated jugular venous pressure. His blood pressure was 130/90mm Hg in supine position and had a regular pulse of 83 /min . He had a regular rapid and shallow abdomino-thoracic breathing pattern with a rate of 21/min. There was a upper motor neuron type of facial palsy on the right side. The examination of the limbs revealed a right sided predominantly distal flaccid unilateral monoparesis with a power of 4/5 with areflexia. The plantar reflex was bilaterally flexor. There were no sensory disturbances and meningeal irritation signs were absent. His respiratory system examination revealed bilateral (left more than right) biphasic coarse crepitations in both the infrascapular regions. His hemogram , renal function test , serum electrolytes and liver function tests were within normal limits. He was tested negative for HIV ELISA. His chest radiograph showed ectacic changes in the left lower zones. A computed tomography of the brain showed multiple hypodense lesions in the left high parietal ,left frontal and right temporal region with surrounding cerebral edema(figure 1). A computed tomography of the chest showed ectatic changes involving the left lower zones (figure 2). An MRI (magnetic resonance imaging) of the brain was done. It showed T2 hyper intense (figure 3) and T2 FLAIR hypo intense(figure 4) central region which showed diffusion restriction and high lipid- lactate peak on spectroscopic imaging(figure 5) highly suggestive of cerebral

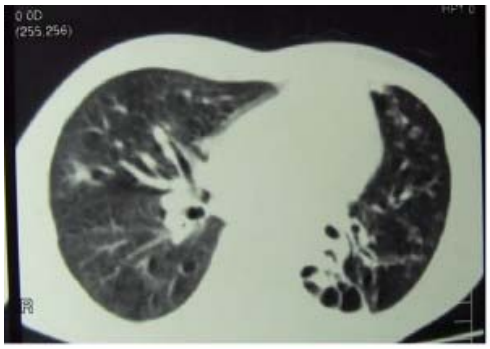

Figure 2

abscesses. These lesions were surrounded by (cerebral oedema. A neurosurgical opinion was taken and they performed an emergency cerebral external drainage. Sputum analysis showed growth of Klebseilla pneumoniae to Ceftazidime and Amikacin. The analysis of the pus from cerebral abscess demonstrated numerous pus cells with Klebseilla pneumonia and patient was treated with antibiotics. Patient's condition improved and was discharged 2 week later.

Discussion:

Cerebral abscess may be caused by direct extension of adjacent infection, blood born or metastatic and locally implanted infected material due to head injury. Local causes of cerebral abscess are either otogenic or rhinogenic in origin[1]. Chronic otitis media is a common cause of otogenic abscesses. Otogenic abscess generally occur in the temporal lobe. Extension occurs through the roof of tympanum or mastoid antrum frequently preceded by extradural abscess. Extension of infection from mastoiditis or labyrinthitis can give rise to cerebellar abscess. Rhinogenic abscess originates

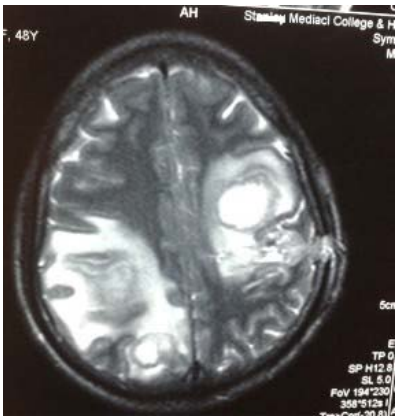

Figure 3 (T2 sequence)

in frontal or ethoidal sinus infection, which spreads to the frontal lobe [3]. Primary foci of infection in metastatic brain abscess lie commonly in the lungs and heart, but may also come from skin, bone or teeth or any other site in the body. It occurs frequently in congenital heart disease with right to left shunts (e.g. tetralogy of Fallot's) and arteriovenous vascular abnormalities of lung as in familial telengiectasia (Osler-Rendu-Weber-syndrome). Infection very often travels from bronchiectasis,empyema thoracis, lung abscess or a broncho-plueral fistula to cause abscess in the brain. Metastastic abscesses are predominantly seen at the junction of gray and white matter as seen in our case. Frontal lobe is the most frequent site of metastatic brain abscess, cerebellum is involved rarely. In our case there were multiple abscess in the parietal , frontal and temporal lobes.[3] Britt and associates were able to define four separate stages in the development of cerebral abscess. Early cerebritis (days 1 to 3) demonstrates a local inflammatory response surrounding the adventitia of blood vessels. The cerebritis is associated with the development of edema and the beginning of a central necrotic region. Late cerebritis stage (days 4 to 9) the most important histological changes take place. Edema reaches its maximum with increase in the size of the necrotic center and the formation of pus. A reticulin network is set down around the periphery of the zone of inflammation by fibroblasts that serves as the precursor to the collagen capsule. Early capsule formation (days 10 to 13) occurs when the collagen network is consolidated and the necrotic center is isolated from the adjacent parenchyma. Obviously, this process is the most crucial one to protect the surrounding tissue from injury. Late capsule formation (day 14 and later) the abscess has five distinct regions: (1) a necrotic center, (2) a peripheral zone of inflammatory cells and fibroblasts, (3) a collagen capsule, (4) an area of neovascularity, and (5) an area of reactive gliosis with edema.The evolution of a well-formed capsule takes about 2 weeks.[5]

Abscesses, being acute inflammatory processes, usually present with a short clinical course. The short history is the main clinical difference with other intracranial mass lesions Symptoms frequently are present for less than 1 week and in 75 per cent of cases for less than 2 weeks. The predominant symptom reported in most patients (70 to 95 per

cent) is headache.Focal neurological deficits are noted in 50 to 80 per cent of patients and relate to location and include hemiparesis, aphasia, visual field defects, and with cerebellar lesions, nystagmus and ataxia. Seizures present in 30 to 50 per cent of patients preoperatively. About half of patients develop low-grade fevers, with temperatures seldom above 39 degrees. Meningismus is present in about 20 per cent of patients and suggests meningitis. Papilledema ---23 to 50 per cent of patients. Some patients have sudden deterioration. In such cases uncal or tonsillar herniation or subarachnoid or intraventricular rupture must be suspected.[3]

Treatment:

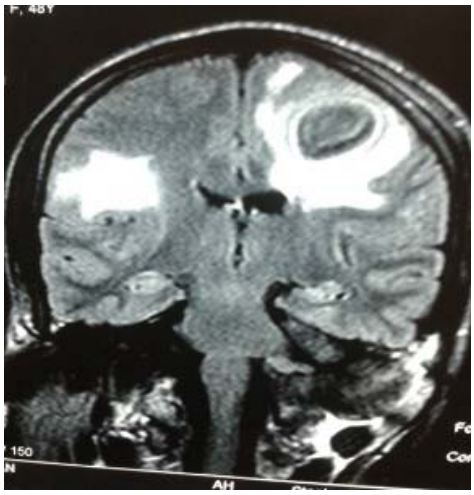

Figure 4 (T2 FLAIR sequence)

Optimal therapy of brain involves a combination of both dose parenteral antibiotics and neurosurgical drainage. Empirical therapy of community acquired brain abscess in immune competent individual involves a third generation cephalosporin [cefotaxime or ceftiaxone] and metronidazole. Aspiration and drainage of abscess under stereotactic guidance are beneficial for both diagnosis and therapy. Empirical antibiotic therapy can be modified based on the results of gram's stain and pus culture and sensitivity. Complete excision of an abscess via a craniotomy or craniectomy is generally reserved for multiloculated abscess. Medical therapy alone is not optimal for adequate treatment of brain abscess and should be reserved for patients whose abscesses are neurosurgically inaccessible, for patients with small [2-3 cm] or non encapsulated abscesses and patients whose condition is too tenuos to allow performance of a neurosurgical procedure.[5] All patients should receive a minimum of 6-8 weeks of parenteral antibiotic therapy. Prophylactic anticonvulsant therapy should be given because of high risk of focal or generalized seizures and it is continued for at least 3 months after resolution of abscesses, and decision regarding withdrawal is based on electroencephalograpgy (EEG). If EEG is abnormal treatment should be continued. If EEG is normal therapy can be slowly withdrawn, with close follow up and repeat EEG after the medication has been discontinued. Glucocorticoids should not be given routinely to patients with brain abscesses. Intravenous dexamethasone therapy is reserved for patients with substantial peri abscess edema and associated mass effect and raised intracranial pressure. Dexamethasone should be tapered as rapidly as possible to avoid delaying the natural process of encapsulation of the abscess. The treatment for bronchiectasis includes appropriate chest physiotherapy and teaching the patient appropriate maneuvers to help postural drainage and clearing of secretions.[5] During acute exacerbations of increased purulent

expectoration , appropriate antibiotic therapy should be initiated. Serial contrast enhanced computed tomography or MRI(magnetic resonance imaging) scans should be obtained on monthly or twice monthly basis to document resolution of the abscess.

Figure 5 (spectroscopic image at the centre of abscess showing a increased high positive lipid and negetive high lactate peak)

References:

1. Levy RM. Brain abscess and subdural empyema. *Curr Opin Neurol. Jun 1994;7 (3):223-8*

2. Tseng JH, Tseng MY. Brain abscess in 142 patients: factors influencing outcome and mortality. *Surg Neurol. Jun*2006;65 (6):557-62

3. Le Moal G, Landron C, Grollier G, et al. Characteristics of brain abscess with isolation of anaerobic bacteria. *Scand J Infect Dis. 2003;35(5):318-21*

4. Bernardini GL. Diagnosis and management of brain abscess and subdural empyema. *Curr Neurol Neurosci Rep. Nov 2004;4(6):448-56*

5. Muzumdar D, Jhawar S, Goel A. Brain abscess: an overview. *Int J Surg. 2011;9(2):136-44.*