Case Report

Brain abscess complicating a diabetic man with Evans syndrome: a case report

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ABSTRACT

Brain abscess is a relatively rare but potentially life threatening condition. The classic clinical triad of fever, headache and focal neurological sign may not be present in an immuno-compromised patient. The diagnostic delay may result in fatal outcome. A 52-year-old diabetic gentleman with history of chronic immunosuppressive therapy due to Evans syndrome presented with headache, generalized tonic- clonic seizure and right upper limb monoparesis. Clinical ground and neuroimaging was suggestive of pyogenic brain abscess. Patient was treated with antibiotic therapy for prolong period and there was complete clinical and radiological recovery.

Key words: brain abscess, Evans syndrome, monoparesis.

(BIRDEM Med J 2021; 11(2): 137-141)

INTRODUCTION

Brain abscesses is a serious, potentially life-threatening central nervous system infection. Although the overall incidence of brain abscess has declined[1-3], it is increasingly common in specific at-risk populations, including patients who are immunocompromised[4] or those who have undergone neurosurgery or experienced head trauma.[2, 5-7] Bacteria can invade the brain either by direct spread or through hematogenous seeding. There is a classic triad of brain abscess (headache, fever and focal neurologic deficit), but is present only in about 20 percent of patients.[8] Diagnosis is made by neuroimaging studies in appropriate clinical background. Microbiological diagnosis of the etiologic agent is most accurately determined by Gram's stain and culture of abscess material obtained by computed tomography (CT) scan guided stereotactic needle aspiration. Successful management usually requires a combination of antibiotics for prolong period and surgical drainage in some cases.

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Received: December 17, 2020

Revision received: February 25, 2021

Accepted: February 28, 2021

CASE REPORT

A 52-year-old man, diagnosed with diabetes mellitus and Evans syndrome (on mycophenolate mofetil and prednisolone) presented with gradually increasing frontal headache, progressive weakness of the right upper limb and one episode of generalized tonic clonic seizure. On query, the patient gave history of productive cough for one and half months. He gave no history of tobacco use, weakness of any other limbs, fever, vomiting, visual disturbance or altered mental status. There was no history of sinusitis or mastoiditis, previous tuberculosis or contact with tuberculosis patients.

The patient was anxious, ill looking and anemic with normal vitals. On neurological examination, he was conscious and oriented. Cranial nerves were intact and there was no papilledema. His muscle power was 1/5 in right upper limb with brisk reflexes. Planters were bilaterally flexor. There was few coarse crepitation in 4th and 5th intercostal spaces in right mid clavicular line and a just palpable spleen in abdominal examination.

His laboratory investigations including routine urinalysis, renal function tests, serum electrolytes, blood glucose and electrocardiogram were normal. Complete blood count showed anemia (Hb 10.3 mg/dl) with normal

platelet count, glycated haemoglobin (HbA1c) 8.6%. Chest X-ray postero-anterior view showed inhomogeneous opacity in right mid and lower zones with small pleural effusion on left side (Figure 1). On CT scan of head, there was a faint area of higher density with surrounding edematous brain involving the frontal and left parietal lobes (Figure 2). In magnetic resonance imaging (MRI) of brain with contrast, the lesions were hypo intense in T1 weighted image, hyper intense in T2 weighted image, perilesional disproportionate edema in fluid-attenuated inversion recovery (FLAIR) image and restricted diffusion in diffusion-weighted imaging (DWI) (Figure 3) with contrast enhancement in T1 contrast image, distributed in right frontal and both parietal regions suggesting cerebral abscess. Blood culture revealed no growth. Sputum Gram staining showed Gram

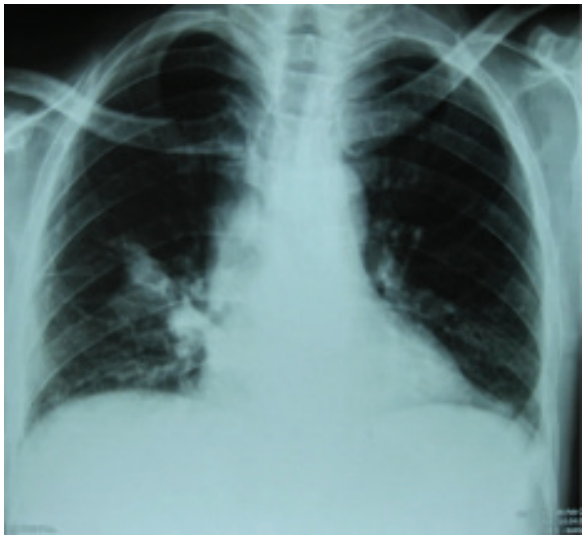

Figure 1 Chest X-ray postero-anterior view showing inhomogeneous opacity in right mid and lower zones and small pleural effusion in left side

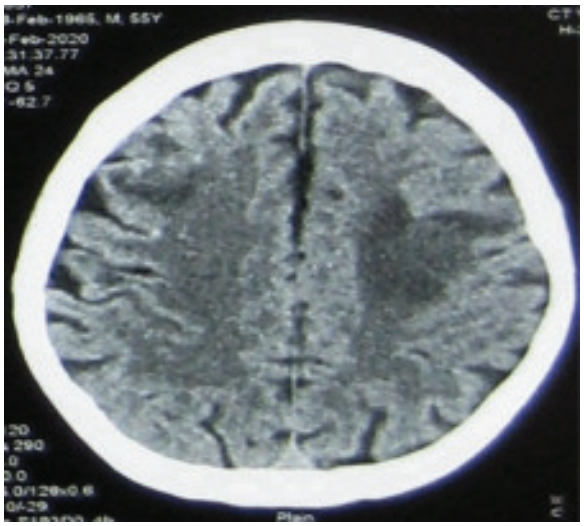

Figure 2 Axial section of computed tomography scan head showing faint area of iso to hypo density with surrounding peri lesional edema in the frontal and left parietal lobe

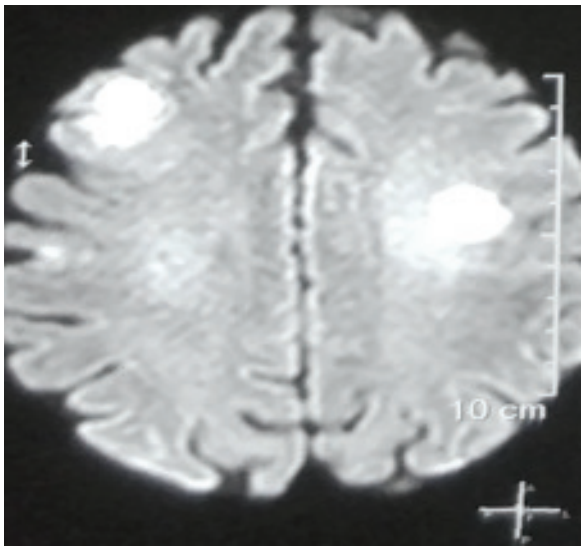

Figure 3 There are hyperintense lesions on diffusion weighted imaging (indicating restricted diffusion, characteristic of viscous materials, such as pus)

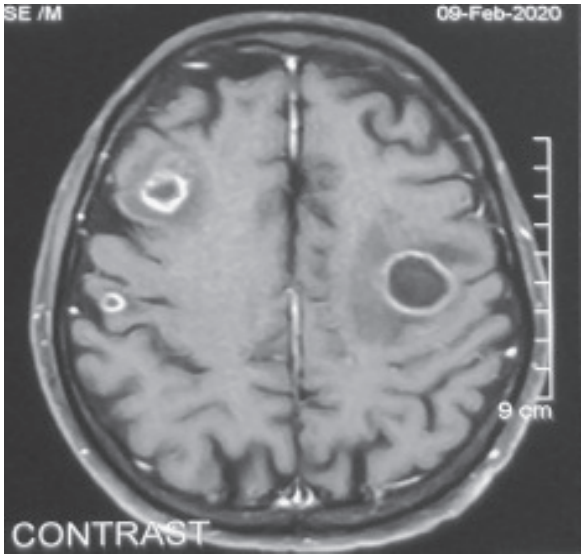

Figure 4 Magnetic resonance imaging with contrast shows peripheral contrast enhancing multiple lesions in both parietal and right frontal region

positive cocci in short and long chains with Gram negative coccobacilli, no acid-fast bacilli (AFB) was found in AFB stain and sputum culture revealed growth of *Klebsiellasp* and Gene X-pert was negative. C-reactive protein (CRP) was raised (164.0 mg/L) with normal procalcitonin level, Monteux test revealed 4 mm induration at 72 hours. Tumor markers (Cancer antigen 19-9 & Carcinoembryonic antigen), CT scan of chest with contrast and CT guided fine needle aspiration cytology (FNAC) of the lung lesion appeared inconclusive. Findings of magnetic resonance (MR) spectroscopy of brain were consistent with pyogenic cerebral abscesses with remote possibility with cerebral tubercular abscess (Figure 6).

Finally, he was diagnosed as a case of brain abscess, pneumonia (right middle lobe), Evans syndrome and diabetes mellitus. His treatment included ceftriaxone, vancomycin, metronidazole along with short course intravenous steroid to reduce cerebral oedema and levetiracetam. Neurosurgeons opined for conservative management. After 7 weeks of antibiotic treatment, his muscle power was became normal with no residual disability or seizure. During this period his steroid was tapered to the required dose for Evans syndrome. Bisphosphonate was also advised. His CT scan of head,

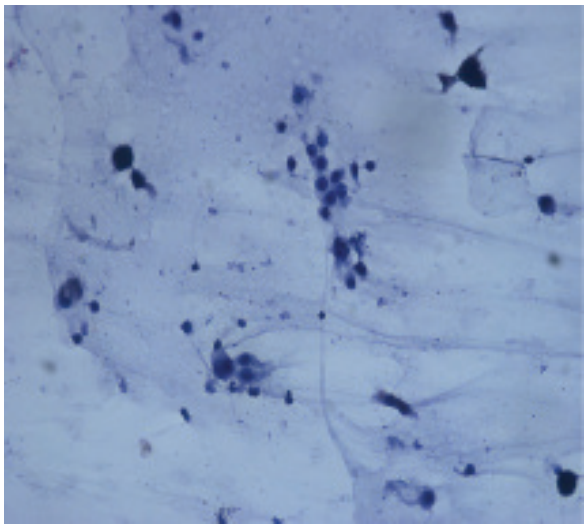

Figure 5 Smear from the lung lesion shows moderate cellular material containing plenty of pulmonary macrophages, moderate number of polymorphs, lymphocytes and reactive bronchial cells in the background of blood mixed with debris. No malignant cell or granuloma is seen.

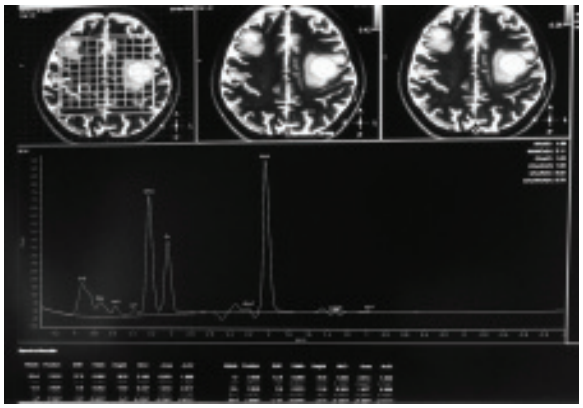

Figure 6 Magnetic resonance spectroscopy shows increased N-acetyl aspartate peak

done in follow up after 7 weeks was normal with no area of altered attenuation in the brain parenchyma (Figure 7), a chest X-ray was unremarkable and in bone mineral density (BMD): T score was 2.7 and Z score was 2.4.

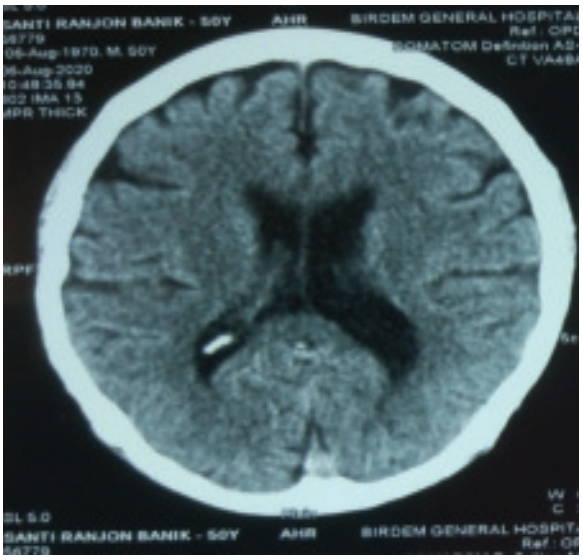

Figure 7 On axial section of computed tomography scan of head, there is no focal or diffuse area of altered attenuation seen in the brain parenchyma

DISCUSSION

Brain abscess is collection of pus in the brain parenchyma. In most patients, it results from predisposing factors, such as underlying disease (e.g., infection with the human immunodeficiency virus [HIV]), a history of treatment with immunosuppressive drugs,

disruption of the natural protective barriers surrounding the brain (e.g., due to an operative procedure, trauma, mastoiditis, sinusitis, or dental infection) or a systemic source of infection.[9] Brain abscess from direct spread usually cause a single abscess whereas hematogenous seeding usually results in multiple abscesses. In around 27 percent cases source of the infection or exact organism can not be identified.[1] Headache is the most common symptom of brain abscess which is usually severe and does not respond to analgesics.[2] Fever is not a reliable indicator of brain abscess since only 45 to 53 percent of patients have this sign.[9,10] Incidence of seizures is 25 percent of cases and can be the first manifestation.[10] Focal neurological deficit usually observed days to weeks after the onset of headache.

The symptoms of our case correspond with the typical presentations of brain abscess. Though he did not have fever but the background of immunocompromised status with the radiological findings of hypo-intense lesion in T1 and hyper-intense in T2 weighted image perilesional disproportionate edema in FLAIR image and restricted diffusion in DWI with contrast enhancement in T1 contrast image in MRI of brain with contrast and MR spectroscopy findings sustained the diagnosis. Negative tumor marker with inconclusive CT guided FNAC report of the lung lesion excluded the possibility of malignancy. The complete clinical and radiological recovery of the patient following prolong antibiotic therapy further support the diagnosis.

Regarding the choice of neuro-imaging technique, MRI of brain with contrast is preferable than CT scan because of its high sensitivity for detecting early cerebritis and satellite lesions, better visualization of brain stem, the extent of ring enhancement and accurate estimation of central necrosis. CT scan can be done in emergency basis but must be done with contrast. DWI is capable of differentiating ring-enhancing lesions due to brain abscess from neoplastic lesions.[11] One prospective study involving 115 patients with 147 cystic brain lesions, which included 97 patients with brain abscess, showed that diffusion-weighted imaging had a sensitivity and specificity for the differentiation of brain abscesses from primary or metastatic cancers of 96% (positive predictive value, 98%; negative predictive value, 92%).[12] It may be possible to differentiate between pyogenic, tubercular and fungal abscess with the help of combining the information of conventional MRI, DWI and proton MR spectroscopy.[4] The MR spectroscopy shows cytosolic amino acids, acetate and succinate in the pyogenic abscesses, whereas in the tubercular abscesses lipid/lactate was seen. The fungal abscesses showed lipid, lactate and amino acids.[4] The presence of amino acids on MR spectroscopy is a sensitive marker of pyogenic abscess. Lumber puncture is contraindicated in presence of focal symptoms and signs or papilledema. The definitive diagnosis is made from pathologic examination of brain tissue obtained by open or stereotactic brain biopsy.

The management of brain abscess usually requires antibiotic therapy and surgical drainage. The antibiotics are usually chosen according to the probable site and likely pathogens involved. In case of unknown source empirical therapy include vancomycin plus metronidazole plus ceftriaxone. Cefepime should be used if pseudomonas infection is suspected. When substantial mass effect is present glucocorticoids are used usually in the form of dexamethasone. Needle aspiration is preferred to surgical excision but drainage is not required in case of early cerebritis without evidence of cerebral necrosis or abscesses located in vital regions of the brain or those inaccessible to aspiration. The treatment duration is guided by both clinical course and imaging study. Usually the treatment duration is between four to six weeks when abscess has been drained or excised and six to eight weeks when no intervention has been done.[6] We have treated our patient with empirical antibiotic therapy and continued for seven weeks. CT scan was done to monitor the response because in MRI the abnormality might persist for months and should not be a guide for prolonging antibiotic treatment.[7]

The mortality rate of brain abscess is 8% to 25% which was about 60% before 1970. This has been possible with the help of new anti-bacterial approaches and new imaging techniques.[8] Meticulous history taking and clinical examination may point to earlier diagnosis of any case. Since brain abscess presents with headache, focal neurological sign which are the common presenting symptoms in our daily practice, this report aims at taking into account of a strong clinical suspicion for brain abscess with choice of appropriate investigation, which might enable earlier diagnosis, prompt management and lead to better outcome and reduced mortality.

Authors' contribution: DA managed the case, did literature review and drafted the manuscript. MM did literature search and helped in drafting the manuscript. RH diagnosed the case and was in overall supervision. MRH helped in literature search and managing the case. All authors read and approved the final manuscript

Conflict of interest: Nothing to declare.

Consent: Informed written consent was obtained from the patient for the publication of this case report and any accompanying images

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