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Neurobrucellosis

NEUROBRUCELLOSIS: A PANDORA'S BOX

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ABSTRACT Brucella considered a deceptive infectious disease in India. Human Brucellosis is well reported in India; however there are only few cases reports on Neurobrucellosis. Brucella is a Pandora's Box as the clinical picture is variable. There are Diagnostic Criteria, includes clinical picture and/or radio imaging and/or CSF findings. This article enlighten on neurobrucellosis, case report of 17 years with persistent headache, two episode seizures, Photophobia was evaluated and hyper intensity in the frontal lobe with MRI Spectra showing Choline, lactate peak, suggesting a neoplastic lesion or Tuberculoma. Tissue diagnosis was found with inflammatory cells and no neoplastic cells were found, the CSF was sent for IgM and IgG titre which were positive and treated. Patient Improved clinically. Patients with persistent headache or neurological signs should be considered for neurobrucellosis in endemic regions. Brucellosis to be considered in cases where there is no probable cause as the diseases has various pathologies.

KEYWORDS : Brucella; Neurology; Headache; Meningitis

INTRODUCTION

Brucellosis is considered a deceptive infectious disease in India. (Kadri, 2005) Human brucellosis is well reported in India; however there are only few reports on neurobrucellosis. Neurological complications of brucellosis are less frequent however an important clinical entity. The presentation is variable as its clinical pathologies are vast during the course of disease. Meningoencephalitis form is common in Nervous system involvement with basal meningitis that may lead to lymphocytic pleocytosis, cranial nerve involvement, or intracranial hypertension (N. Ceran, 2011) Guven et al (T. Guven, 2013) Observed that headache, blurred vision, loss of vision, hearing loss, and confusion were significantly associated with neurobrucellosis.

Muscular weakness, disorientation, neck rigidity, changes in deep tendon reflexes, and paresthesias were also more common amid the patients. Cranial nerve palsies were common among them the cranial nerves, facial; abducens and vestibulocochlear were affected more in neurobrucellosis. Radiculopathy or polyradiculopathy were observed in Peripheral nerve involvement and Nonspecific symptoms and meningeal signs were seen in neurobrucellosis. The meningeal signs were ought to be seen infrequently present (T. Guven, 2013)

Brucella bacteria may affect the nervous system directly or indirectly, as a result of cytokine or endotoxin on the neural tissue. Cytotoxic T lymphocytes and microglia activation play an immunopathologic role in this disease. Infection triggers the immune mechanism leading to a demyelinating state of cerebral and spinal cord (N. Ceran, 2011)

In neurobrucellosis imaging findings may be found in four types: normal, meningeal contrast enhancement, white matter changes, and vascular changes (N. Ceran, 2011) In addition to non enhancing bilateral white matter lesions deep grey matter involvement has also been documented (R. Rajan, 2013)

Most important differential diagnosis of brucellosis is tuberculosis in our country and endemic region. Both are chronic granulomatous infectious diseases are endemic in our country. (P. Kesav, 2013) There is a clearly overlap between neurobrucellosis and tuberculosis both in terms of clinical presentation, laboratory parameters, and neuroimaging. (R. Rajan, 2013)

The Hearing loss due to vestibulocochlear nerve involvement, deep grey matter involvement, and extensive white matter lesions on neuroimaging mimicking demyelinating disorders seems to be unique for brucellosis (R. Rajan, 2013) (P. Kesav, 2013)

Neurobrucellosis is a diagnostic puzzle as there is a lack of accord in diagnostic criteria. According to Kochlar et al. (D. K. Kochlar, 2000) the criteria necessary for definite diagnosis of neurobrucellosis are (i) neurological dysfunction not explained by other neurologic diseases, this is part of the diagnosis based on exclusion of other diseases (ii) abnormal CSF indicating lymphocytic pleocytosis and increased protein, this was also seen in Guven et al Studies(2013) (iii) and a positive CSF culture for *Brucella* organisms or positive *Brucella* IgG agglutination titer in the blood and CSF.

Recently Guven et al. (T. Guven, 2013) (H. Erdem, 2013) diagnosed neurobrucellosis by the presence of any one of the following criteria:

1. Symptoms and signs suspect of neurobrucellosis,
2. Isolation of *Brucella* species from cerebrospinal fluid (CSF) and/or presence of anti-*Brucella* antibodies in CSF,
3. Decreased glucose, presence of lymphocytosis, increased protein in CSF, or
4. Findings in cranial MRI or computed tomography (CT).

Erdem et al. (H. Erdem, 2013) defined chronic *Brucella* meningitis on the basis of following criteria:

1. The manifestation of clinical neurological symptoms for over 4 weeks
2. The presence of typical CSF evidence with meningitis (protein concentrations >50 mg/dL, pleocytosis over 10/mm^3, and CSF glucose to serum glucose ratios <0.5)
3. Positive bacterial culture or serological test results for brucellosis in CSF (positive Rose Bengal Test or serum tube agglutination) or in blood (positive Rose Bengal Test and serum tube agglutination with a titer ≥1/160) or positive bone marrow culture
4. Nonappearance of any alternative neurological diagnosis.

CASE STUDY

A 17-year-old female was admitted on May 2022, she presented with Headache and seizures since 15 days along with photophobia and one day history of fever. The fever was documented up to 101°F associated with sweating. There was history of insidious onset, with two episodes of generalized tonic clonic seizures associated with history of vomiting, photophobia associated with generalized weakness over the period of 15 days. Patient had headache which had been increasingly and throbbing in nature. She had No history of ear discharge, altered sensorium, or Limb weakness or numbness was reported. Review of other systems was normal. No significant past history was present. The patient is school going belonged to a rural area and was associated with livestock rearing.

On examination patient was febrile. Rest of the general physical examination was normal. On nervous system examination, our patient was conscious but anxious with the mini-mental state examination score of 30/30. Glasgow Coma Scale E3V5M6. Examination of

cranial nerves revealed Normal, Motor examination revealed normal muscle bulk, no spasticity in upper and lower limbs, grade IV power in proximal muscles of both upper and lower limbs, symmetrically Normal deep tendon reflexes, and bilaterally extensor plantar response. Sensory examination was normal. Meningeal signs were present with Kernig's sign positive. Rest of the neurological examination was normal.

Review of other systemic examinations was normal.

On investigations hemoglobin was 15.9 gm% and total leukocyte count was 8114/cmm. Peripheral smear revealed a microcytic hypochromic picture. Biochemistry showed Elevated liver functions. Widal test and dengue test was Negative

Chest X-ray and ultrasonography of abdomen were normal.

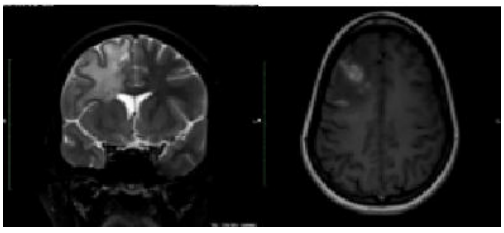

Figure 1 MRI Images Shoring HyperIntense Frontal Lobe Suggestive of Oedematous

The patient was investigated where MRI reported: Multiple small nodular ring enhancing incomplete ring enhancing, gyriform enhancing lesions showing moderate surrounding edema in right frontal lobe causing mild compression of the right lateral ventricle probably represent celebrities.

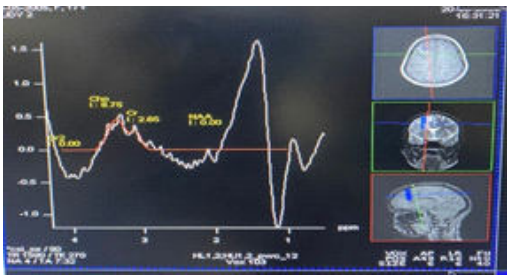

Figure II MRI Spectra Showing Choline Peak and Lipid Lactate Peak

MR Spectroscopy: High Lipid Lactate peak in the enhancing part with a large choline peak & choline creatinine ratio being > 1 suggestive of ?Tuberculoma or Neoplastic Lesion hence Tissue diagnosis for confirmation was advised.

EEG: Abnormal awake record suggestive of generalized epileptiform discharges Cerebrospinal fluid (CSF) analysis was colourless and had proteins: 40.8 mg/dL; glucose: 60.7 mg/dL (concomitant blood glucose: 96 mg/dL); adenosine deaminase: 2.7 U/L. On microscopic examination of CSF total WBCs count was 8/cmm; lymphocytes were 100%; RBCs No organism seen on Gram stain, negative for cryptococcal infection and negative for acid fast bacilli by ZN stain.

Toxoplasmosis IgM was Negative

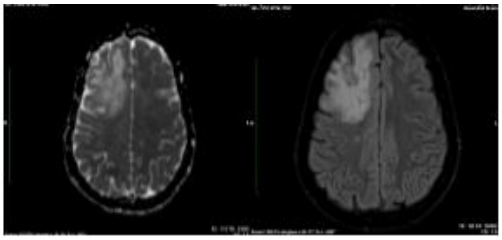

Figure III Shows Hyperintensity in the frontal lobe in MRI

A repeat MRI Showed: Irregular ill- defined area of heterogenous signal intensity and morphological changes in right frontal lobe with enhancement pattern and increased vascularity in right frontal lobe with enlarged right internal cerebral vein. Contrast study shows mild to moderate enhancement in right frontal lobe with few patchy areas in periventricular region in right frontal lobe along with leptomeningeal enhancement. Possibility of infective etiology appears likely. Clinical correlation with CSF and Biopsy was done and IgM and IgG titres Brucellosis were Positive in this patient

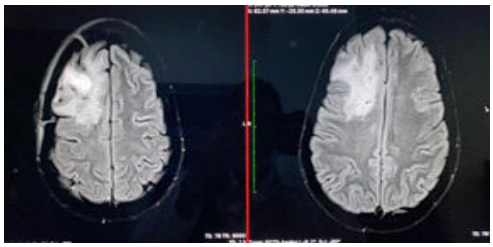

Figure IV MRI FLAIR showing reduction in peri lesional edematous changes

Dual- or triple-combination therapy with doxycycline, rifampicin, trimethoprim-sulfamethoxazole, streptomycin, or ceftriaxone for >2 months (3–6 months) has been recommended.

This patient was treated with Rifampicin, Doxycycline and Ceftrixone in the Hospital and the patient improved markedly on 5th day. The patient was planned for discharge next day.

The patient was discharged on oral Doxycycline, Cefixime and Rifampicin and was followed up as an Outpatient.

CONCLUSION

Patients with severe, persistent headache or neurological signs are possibly screened for neurobrucellosis in endemic regions. Brucellosis could also thought in cases where there is no probable cause found as the clinical picture is vast in the disease course. Accurate diagnosis and proper management of Neurobrucellosis is necessary as the disease mimic various pathologies, hence thorough evaluation is needed.

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