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RESEARCH ARTICLE OPEN ACCESS

SCRUB TYPHUS INFECTION PRESENTING AS PANCEREBELLAR ATAXIA

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ABSTRACT

Pancerebellar ataxia is a syndrome characterised by bilateral signs of cerebellar dysfunction involving trunk, limb and cranial musculature. We describe an adult female presenting with this syndrome following a febrile illness diagnosed as scrub typhus fever. Since majority of the cases of cerebellitis has been described in children, we highlight this case where the patient presented with an acute onset of tremulousness, imbalance and instability involving all the limbs and trunk, with ocular signs on examination. The parainfectious syndrome was recognised and immediately investigated and started on treatment. Patient made an excellent recovery and became self-ambulatory.

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INTRODUCTION

Acute cerebellitis is an inflammatory syndrome associated with cerebellar swelling and dysfunction, hypothesized to be a result of infection (most commonly viral) and less commonly after vaccination, or secondary to autoimmune diseases like Hashimoto's thyroiditis.[1] It is a rare inflammatory syndrome, and the majority of cases have been described in children and were caused by a primary infection (e.g. West-Nile virus, Mycoplasma pneumoniae) or a postinfectious disorder. [2] In a review of literature on the symptoms at presentation, majority of the children had Ataxia (92%), followed by balance disturbances in 68%. Dysmetria, Dysarthria and Intentional tremors were present in around 25-35% of the cases. Other symptoms like vertigo and nystagmus are less common and seen in nearly 18 & 12% cases respectively.[3] If cerebellar signs are present, the differential diagnosis of cerebellitis needs to be identified for appropriate cause, for which MRI Brain is needed to confirm the diagnosis and rule out other differential disorders. Alternative differential diagnosis needs to be kept in mind, which includes cerebellar stroke, infectious meningoencephalitis, acute disseminated encephalomyelitis, cerebellar tumors, and posterior reversible encephalopathy syndrome. [2] Scrub typhus is a rapidly emerging disease in Southeast Asia and India, predominantly affecting the rural population but increasingly involving the urban areas, and is an important aetiology of acute undifferentiated fever.[4]It is caused by a rickketsial obligate intracellular gram-negative bacterium called Orientia tsutsugamushi.[5]

The clinical presentation of scrub typhus infection ranges from mild febrile illness to severe life-threatening complications, with a wide spectrum of involvement causing liver dysfunction, acute kidney injury, acute respiratory distress syndrome, myocarditis, disseminated intravascular coagulation (DIC) and haemophagocytic syndrome. With the recent increase in the incidence of this disease, there has also been an increase in the reported cases of unusual manifestations of scrub typhus. The range of neurological manifestations seen is also diverse, with case reports of posterior reversible encephalopathy syndrome, Opalski syndrome, parkinsonism, cerebellitis, isolated opsoclonus, acute transverse myelitis, myositis, polyradiculoneuropathy with cranial neuropathy, acute transient behavioral changes. [6]We report aatypical case of pancerebellitis secondary to scrub typhus infections.

CASE HISTORY

A female in her 30s, farmer by occupation, hailing from a village in central India, presented with a history of fever that was high grade not associated with chills and rigors, 12 days prior to admission. Fever lasted for 3 days and after that she developed an acute onset of tremulousness, imbalance and instability that progressed over the next 3 days such that she is unable to make meaningful movements of her limbs & unable to sit/stand without support due to the abnormal jerky movements and tremulousness of her body and limbs as shown in video 1. On examination, blood pressure and pulse rate was within normal limits. Head to toe examination doesn't reveal any tick bite, marks or eschar, rashes, or petechiae. Patient is conscious and

oriented, and higher mental functions are normal with intact comprehension, repetition and naming. Dysarthria is present with broken syllables, varying pitch and loudness suggestive of staccato speech. Cranial nerve examination showed abnormal extraocular movements in the form of impersistent and broken pursuits along with hypometric saccades. Gaze evoked nystagmus was present towards the left. Other cranial nerve examination was within normal limits. Motor system examination was limited due to inability to sustain any movement at all the joints leading to severe imbalance and instability with jerky tremulous movements. Deep Tendon Reflexes were symmetrical with bilateral grading of 2+. Bilateral plantars were also flexor. Sensory examination was within normal limits. Examination showed involvement of cerebellum with positive signs in the form of impaired finger Nose test and finger nose finger test suggestive of severe Dysmetria. Intention tremors and past pointing was also present bilaterally. Heel shin test was also impaired in both the lower limbs. She was not able to perform rapid alternating movements in both upper and lower limbs suggestive of Dysdiadokokinesia. Severe truncal ataxia was also present, with swaying in sitting position. Wide based gait was present with swaying to either sides while walking and also she unable to stand without support of two people. In the background of febrile illness and patient presenting with an acute onset, rapidly progressive truncal and limb Ataxia with staccato type of speech and nystagmus, diagnosis of pancerebellar ataxia secondary to parainfectious process was made. Patient was evaluated for the cause of parainfectious cerebellitis.

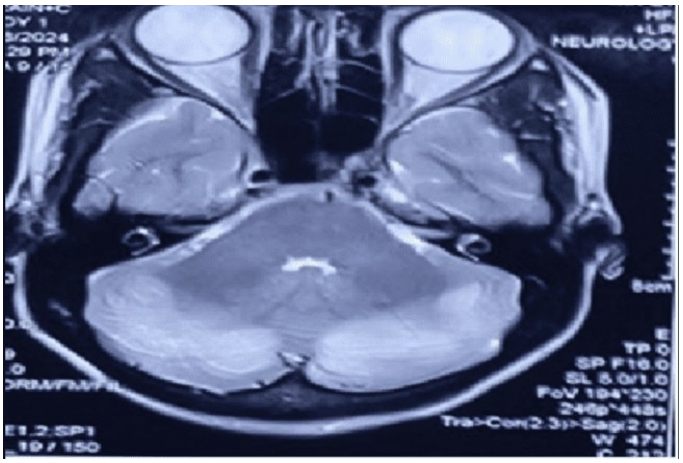

Figure 1. Bilateral symmetrical T2 hyperintensity involving the cerebellar hemispheres and cerebellar tonsils with effacement of adjacent folia in MRI Brain

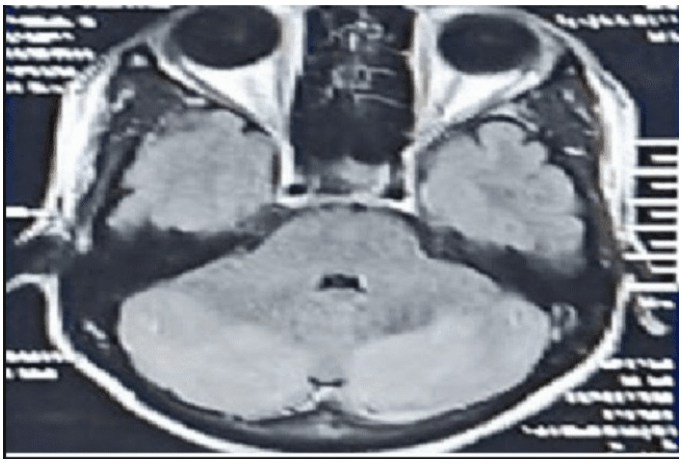

Figure 2. Bilateral symmetrical FLAIR hyperintensity involving the cerebellar hemispheres and cerebellar tonsils with effacement of adjacent folia in MRI Brain

Blood investigations including complete blood counts, and comprehensive metabolic panel was sent including liver function tests, renal function tests, thyroid profile and vitamin B12 levels and was found to be within normal limits, except for a minor elevation of SGOT and SGPT with levels of 47.3 IU/L & 36.7 IU/L respectively (Normal <35 IU/L). NCCT Head was done, which was showing bilateral cerebellar hemisphere hypodensity, without any mass effect, midline shift or hydrocephalus. Fundus examination was not showing papilledema. Lumbar puncture and Cerebrospinal Fluid (CSF) analysis was done on 12th day of illness which was normal- with 5 cells (all lymphocyte), protein of 33.25 mg/dl and glucose of 46.1 mg/dl (corresponding Blood sugar of 78 mg/dl). Gram stain & culture of CSF was negative, Staining for Acid Fast Bacilli & Cartridge Based Nucleic Acid Amplification Test (CBNAAT) for Tuberculosis was negative. CSF molecular diagnostic test for common pathogens causing meningitis/encephalitis (CSF BioFire) was negative.

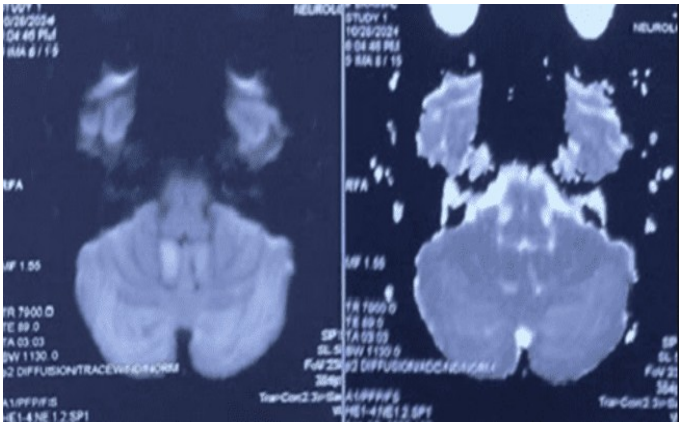

Figure 3. MRI Brain DWI sequence showing bright signal and isointense signal on ADC

As part of tropical diseases workup, test for Scrub typhus IgM ELISA was positive and other tropical fever workup including Malaria parasite, Dengue and Leptospira was negative. Inflammatory markers were normal with ESR of 17, and CRP of 3.66 mg/L (Normal <5 mg/L). Thyroid profile was within normal limits with T3, T4 and TSH values of 0.49 ng/ml, 7.57 µg/dl, and 0.46 µIU/mL respectively. Electroencephalography (EEG) was obtained which had normal background activity, without any epileptiform discharges. MRI Brain with contrast showed bilateral symmetrical T2/FLAIR hyperintensities involving the cerebellar hemispheres and cerebellar tonsils with effacement of adjacent folia (Figure 1 and 2). These areas show bright signal on DWI and appear isointense on ADC (Figure 3) suggestive of cerebellitis. Patient was treated with intravenous Doxycycline, with IV steroids Dexamethasone 8mg TDS for 14 days. Patient's ataxia improved significantly during the course of hospital stay such that she was able to walk without any support and was able to perform all her activities after the course of antibiotics and steroids. Cerebellar signs had significantly reduced as compared to admission (video 2).

DISCUSSION

Scrub typhus is a vector-borne zoonotic disease which presents as an acute undifferentiated febrile illness 7–10 days after the bite of an infected larval trombiculid mite (chigger) [7] Eschar may not be detected in up to 40%–50% of the cases [8,9] even though its considered one of the hallmarks of this disease, and it was absent in our patient also. The neurological manifestations in scrub typhus is diverse, involving both central and peripheral nervous system, important being meningoencephalitis, [10] acute disseminated meningoencephalitis, isolated sixth palsy, seventh nerve palsy, polyneuropathy, cerebral infarction, transient Parkinsonism, opsoclonus–myoclonus [11] and Guillain-Barre syndrome.[12] The neurological injury occurs due to focal vasculitis and lymphocytic infiltration of blood vessels and perivascular spaces, secondary to invasion of vascular endothelial cells by the organism as revealed by autopsy studies of these patients.[13] Scrub typhus causing Acute

Encephalitis and meningoencephalitis is known in the Indian subcontinent, especially among the paediatric population [14] Acute cerebellitis is a relatively rare finding in scrub typhus cases, and has been found to be re-emerging disease in past few years, especially in Indian population [13] A number of tests are available for diagnosis of scrub typhus, but ELISA is one of the most commonly used and widely available and has high sensitivity & specificity of 92% & 94% respectively. [15] According to J. Granerod [16] et al definition used for defining causality of acute encephalitis, a single high titre amounts to Probable causative role in the disease. Extending the same in our case, serum ELISA was positive for Scrub typhus and patient was labelled as probable scrub typhus infection related acute cerebellitis. Imaging plays a major role in determining the aetiology as well as ruling out other causes like Metabolic diseases, neoplasms, meningitis, encephalitis, abscess, acute disseminated encephalomyelitis, or hereditary degenerative disorders. The most common finding is that of bilateral diffuse hemispheric abnormalities in upto 73% cases of Acute Cerebellitis, [17] which was seen in our patient also. Other findings that may be seen include involvement of one hemisphere, isolated vermis and superior cerebellar peduncle involvement.[18]There is no guidelines for treatment currently, and case reports describe improvement with steroids,[19] and in the absence of response to steroids IVIG has also been used with good outcomes.[20] Our patient was administered dexamethasone along with antibiotic Doxycycline for 14 days, during which the patients symptoms improved significantly. Patient was then placed on oral tapering dose of Prednisolone for next 14 days. She began performing ADLs independently and ataxia resolved tremendously with the above treatment.

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