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General Medicine

RARE PRESENTATION OF EXTRA PULMONARY TB AS MULTIPLE INTRA CRANIAL TUBERCULOMAS- A CASE REPORT

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ABSTRACT TB of CNS is an uncommon yet highly devastating manifestation of TB. It has a hematogenous spread manifesting as meningitis, cerebritis,TB abscess, Tuberculomas and spinal arachnoiditis. We are reporting a case of 26yr old female person presented with short duration of headache, vomiting, neck pain and altered sensorium .Examination revealed left eye Ptosis and right sided classical hemiplegia . Investigations revealed multiple intracranial tuberculomas in MRI brain and feautures suggestive of miliary TB on CT-chest . Multiple tuberculomas are rare presentation of intracranial TB and prognosis is poor in patients with multiple tuberculomas . But in our case clinical improvement is observed with Anti Tubercular Therapy .

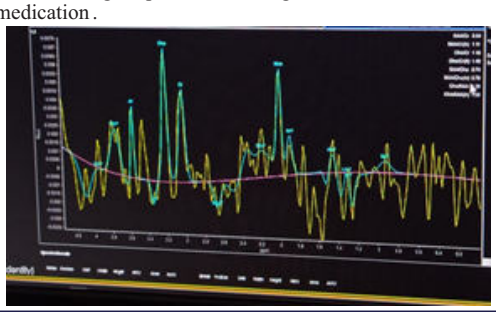
KEYWORDS : TB, CNS, Tuberculoma

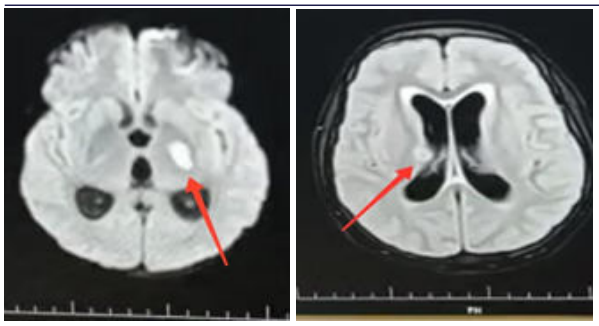
INTRODUCTION :-

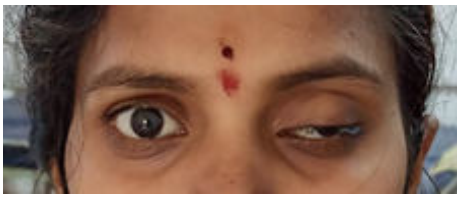
TB of CNS is an uncommon yet highly devastating manifestation of TB which was universally fatal in the era before ATT[1]. CNS TB accounts for approximately 1% of all cases of TB cases[1].It has a hematogenous spread manifesting as meningitis, cerebritis, TB abscess, Tuberculomas and spinal arachnoiditis. Intracranial tuberculomas are least common presentation of CNS TB, found in 1% of these patients. Miliary disease may increase the risk of hematogenous spread to CNS. Mycobacterium tuberculosis may enter the CNS via direct infection of endothelial cells or trafficking through infected phagocytes which is followed by formation of tubercles most commonly in brain cortex or meninges. There is a clear predominance of solitary intracranial tuberculoma with M:F ratio of 1:3.5 , in contrast to almost equal distribution in male and female in multiple intracranial tuberculoma[2].The presence of multiple intracranial lesions in a young patient should warrant consideration of infection over metastatic cancer , in older age group reverse should be considered .While TB remains the most common infectious pathology , Neurocysticercosis and toxoplasmosis should be ruled out . The clinical features of Tuberculomas depend on their anatomical location in the brain related to local mass effect and obstruction of CSF pathway. Neuroimaging is essential for identifying intracranial tuberculous mass lesions with findings determined by composition of the lesion. MRI is preferred modality for the identification of tuberculomas due to superior resolution and better visualization . The radiological appearance of intracranial tuberculomas in MRI is hypointensity with ring shaped enhancement in T1WI. Hyperintensity with central hypointensity on T2WI[3]. Caseating liquid granulomas are hypointense on T1WI and hyperintense on T2WI with ring enhancement which are rare. Isolated bilateral occulomotor nerve palsy in TB meningitis is very rare[3].The mainstay of treatment of intracranial tuberculomas is similar to that of TB meningitis and included TB therapy and and corticosteroids. The WHO Center for Disease Control and prevention of America & British Thoracic Society recommended a 9-18 months course of TB treatment for CNS TB[4]. .Some guidelines suggest adjunctive systemic corticosteroids in all forms of CNS TB , it may be of particular value where there is significant perilesional edema and in cases where there is paradoxical enlargement despite optimal TB therapy[5] .

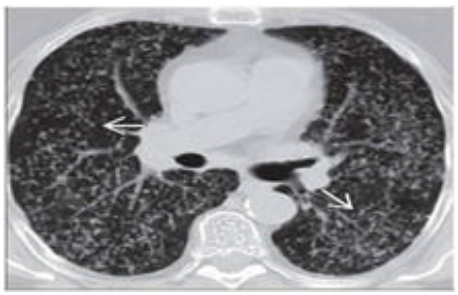
CASE REPORT :

A 26 year old female individual presented to the OPD with 15 days duration of Headache associated with neck pain and non-projectile vomitings with altered sensorium and weakness of right upper limb and lower limb with loss of speech and drooping of left eye from 1 week. History of weight loss was present since 1 month. Patient had no history of fever, cough, shortness of breath and seizures. She had no history of contact with tuberculosis patient or ear discharge or earache. On examination– her GCS score is 9/15 and pallor is present without lymphadenopathy. Her vitals are stable. Respiratory system revealed decreased breath sounds on both sides of chest with crepitations in the basal areas. CNS examination revealed partial ptosis of left eye(MRD)Marginal reflex distance :OD+4,OS 0, & left eye is deviated downwards & laterally with normal pupillary reactions suggestive of oculomotor nerve palsy. There is slight deviation of angle of mouth to left side. Tone is decreased in right upper limb & lower limb with hypoactive reflexes on right side. Power is 0/5 on right side with extensor plantar response on right side. Terminal neck stiffness is present but Kernig's and Brudzinski's signs are negative. Examination of other systems are Normal. Our provisional diagnosis is TB meningitis with Vasculitis. Laboratory investigations revealed low haemoglobin levels , normal total leukocyte count with elevated lymphocytes . ESR is 80mmHg. Random Blood Sugars, Liver Function Tests, Renal Function Tests, Serum electrolytes are within normal range. Patient is seronegative for HIV, HbsAg, HCV. Mycobacterium tuberculosis is not detected in sputum by CBNAAT.CT chest revealed multiple variable sized air spaced nodules with adjacent reticular septal thickening & ground glass attenuation in bilateral lung fields suggestive of military tuberculosis.CT Brain revealed cortical based hyperdense lesion in grey matter of left temporo -parietal lobe which is further evaluated with MRI Brain which showed Multiple small ring enhancing lesions scattered diffusely in bilateral frontal lobes ,left high parietal lobe, Right capsuloganglionic region , cerebellar hemispheres with mild perilesional edema and mild hydrocephalus, there is abnormal enhancement at basal cistern. All these findings are suggestive of multiple intracranial infective granulomas probably tuberculomas or Neurocysticercosis. Further evaluation done with MR Spectroscopy which showed multiple ring enchancing lesions with largest lesion measuring 12 to 13mm.These lesions showed mild lipid lactate elevation with elevated choline to creatinine ratio & relatively maintained NAA peak. FLAIR hyperintensity is noted along ventricular walls & basal cisterns with abnormal meningeal enhancement. There is mild dilatation of supratentorial ventricular system. All these findings are highly suggestive of multiple intracranial tuberculomas. Lumbar puncture is not done. Our final diagnosis is Disseminated TB with multiple tuberculomas. Anti tubercular therapy ,Dexamethasone with tapering doses are advised to the patients . During the course of hospital stay patient has improved sensorium and regain power on the right side. Patient is discharged with medication .









DISCUSSION :-

TB remains to continue as the major health problem until today. A total of 1.4 million people died from TB in 2019 , an estimated 10 million people fell ill with TB worldwide. The WHO has recently launched a new global TB strategy for the "post 2015 era " aimed at" ending the global TB epidemic by 2035". Extrapulmonary TB defined as TB at sites other than lungs, such as lymph nodes genitourinary tract, pleura, bones, joints, meninges, CNS, peritoneum and other abdominal organs[6]. CNS TB is one of the most devastating type of TB because it is associated with high mortality and high rate of neurological sequelae[2]. Therefore it is essential to have an approach towards its diagnosis and treatment. The clinical features of tuberculoma spans from subtle to severe illness[7]. It depends on time of presentation, location and size of lesion. In the initial stage , there may be no clinical features or asymptomatic, later it emerges as features like headache and epilepsy. Tuberculomas may present with altered mental status, focal neurological signs or increased intracranial pressure.If the patient presents in delayed condition, there will be increased intracranial pressure and weakness of extremities[7] .In our case patient presents with headache, vomitings, altered sensorium and 3rd and 7 th cranial nerves palsy with right sided hemiplegia. Gaur et al have reported bilateral occulomotor nerve palsy with pupil sparing in a 19 yr old male due to tuberculoma in midbrain[8]. Similar studies were done in 11 patients involving cranial nerves due to brain stem tuberculoma by Talamas et al[9]. Kumudini Sharma et al presented a case of brain stem tuberculoma with isolated oculomotor nerve palsy[10] . Cranial nerve involvement may occur due to mass effect from tuberculoma / abscess , due to brainstem infarction or a false localizing sign due to raised intracranial tension. . The important causes of III nerve palsy are posterior communicating artery aneurysm, Stroke, diabetes ,vasculitis , tumour , meningitis, trauma .The motor nucleus of III cranial nerve is situated in midbrain , at the level of superior colliculus[11].It emerges from ventral surface of brainstem and enters the orbit through superior orbital fissure .Its motor fibres innervate levator palpebrae superioris except superior oblique and lateral rectus. The parasympathetic fibres originate for EdingerWestphal Nucleus and they innervate the ciliary ganglion from where they supply sphincter pupillae via short ciliary nerves[11].Occulomotor nerve palsy may be complete or incomplete depending on extent of damage and number of fibres involved. In our case, multiple intracranial tuberculomas are seen in bilateral frontal lobes, left high parietal lobe, right capsuloganglionic region & bilateral cerebellar hemispheres. Turgut et al reported distribution of brain tuberculomas was as follows cerebral hemispheres 41%, cerebellum 35%, brainstem 6%, intraspinal 6%, multiple 12%[12]. Multiple intracranial lesions could be suggestive of neurocysticercosis, glioma, metastatic tumours, toxoplasmosis. CT is reported to have sensitivity of 100% and specificity of 85.7%[7] . MRI is reported superior to CT in diagnosis of tuberculoma but its availability should be a concern. Its superiority is in visualising the morphological detail of tuberculoma, especially in the tiny lesion in the brain stem[7]. In our case the supporting diagnostic modality is MRI and MR Spectroscopy. MRI shows multiple intracranial tuberculomas in bilateral cerebral , cerebellar hemispheres with abnormal meningeal enhancement at basal cistern and mild hydrocephalus. MR Spectroscopy reveals lipid lactate elevation at lesion with elevated choline to creatinine ratio and relatively maintained NAA peak, thus going in favour of multiple tuberculomas. Tuberculoma in MRI will show conglomerated ring enhanced mass on gadolinium enhanced T1 weighted imaging. MRI will also distinguish the lesion of tuberculoma with others with T1 and T2 based on its nature, either caseating or noncaseating[7]. Tuberculoma is distinguished with other in its lower T2 weighted as it contains more lipid .MR Spectroscopy may increase specificity of diagnosis by identifying lipids with in the lesion that are considered characteristic for TB[7]. Tuberculomas are characterised by prominent decrease NAA/cr and slight decrease in NAA/Cho-lipid lactate peaks are usually elevated[13] . Venkatatram Krishnan et al presented cases highlighting the importance of imaging in diagnosing CNS TB[14]. The recommended use of the regimen and duration of therapy are extrapolated from the standard regimen for pulmonary TB, since no randomised control trail has been established an optimal treatment course for CNS TB[5]. Recommended dose of ATT Is at least 9 to 18 months, depending on patients clinical and radiological response, but, may have to be continued for longer or changed to second line medication[5]. The use of steroids in cerebral TB is controversial issue. It reduces the inflammation with in the sub-arachnoid space[5]. Initial literature is filled with the reports of successful treatments with surgical excision of tuberculomas, but with introduction of better medications and with reports of equal or even better results with ATT alone, so the paradigm shifted toward conservative management[5].

CONCLUSION:

Since clinical manifestations of disseminated TB are non-specific, timely recognition of signs & symptoms are essential to establish the diagnosis. Delayed diagnosis or misdiagnosis of disseminated TB can delay the initiation of treatment, leading to various complications and increasing the risk of mortality. b High clinical suspicion and investigations like MRI will lead to early diagnosis and treatment, so that we can prevent neurological sequelae.

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