SINGLE LESION NEUROCYSTICERCOSIS PRESENTING AS STATUS EPILEPTICUS

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ABSTRACT: A 20 year old male who presented in Neuro-Psychiatric clinic in a confusional state, was talking irrelevant, not responding to the surrounding stimulus & did not recognize his family members. There was a history of multiple episodes of seizures (8-10) the previous day. The patient was already under anti-convulsant medication for 4 months and was maintaining full compliance. His routine investigations and electroencephalogram (EEG) study were not contributory. On CT scan of Head, a small ring enhancing focus with central hyperdense dot and perifocal edema was seen in left frontal lobe suggesting possibility of Neurocysticercosis (colloidal vesicular stage).The patient was put on anti-helminthic treatment with Albendazole and steroids for 3 weeks along with the continuation of anti-epileptic treatment. He showed clinical improvement but repeat CT Scan Head done 2 months after that, did not show major changes in the lesion (granulomatous lesion, probably cysticercosis). Neurosurgery and consequent removal & biopsy of the lesion confirmed the diagnosis of Neurocysticercosis (Degenerated cysticercal cyst). This case is reported as an uncommon case of single lesion Neurocysticercosis in frontal lobe presenting as complex partial seizure with secondary generalization and progressing to Status Epilepticus.
KEYWORDS: Neurocysticercosis, Complex partial seizure, Status Epilepticus.

INTRODUCTION: Cysticercosis is a condition that occurs when infested by the larvae of Taenia solium. CNS involvement is seen in approximately 90% of patients with cysticercosis; when cysticercosis involves the CNS, it is called neurocysticercosis (NCC). Neurocysticercosis (NCC) is the main cause of acquired epilepsy in the developing countries (1, 3, 6).

Seizures are the most common clinical manifestation of intraparenchymal NCC at all stages. Neurocysticercosis is typically first seen with seizures (70-90% of acutely symptomatic patients) or headache.

Status Epilepticus refers to the continuous seizures or repetitive, discrete seizures with impaired consciousness in the inter-ictal period. The duration of seizure activity sufficient to meet the definition of status epilepticus has traditionally been specified as 15 to 30 minutes. However a more practical definition is to consider status epilepticus as a situation in which the duration of seizures prompts the acute use of anti-convulsant therapy, typically when the seizures last beyond 5 minutes. (6).

Neurocysticercosis is a common cause of epilepsy syndrome in both rural and urban India**.** V. Rajshekhar and his colleagues conducted a door-to-door survey of 50,617 people between the ages of 2 and 60 years in a rural (38,105 people) and urban setting (12,512 people) in the Vellore district and found that , NCC is the cause of nearly one-third of all cases of AE in both the urban and rural regions (9).

In India, NCC is characterized by small, multiple, diffuse parenchymatous involvement (8). We report an uncommon case of single lesion Neurocysticercosis (NCC) in the left frontal lobe

presenting as complex partial seizure with secondary generalization and progression to Status Epilepticus.

We have found very few reports of Neurocysticercosis presenting as Status Epilepticus in the literature.

CASE: Mr. 'S' a 20 year old, right handed, Hindu male presented in Neuropsychiatric clinic in a confused state with chief complaints of headache, vomiting(4-5 episodes), multiple episodes of seizures (8-10), with loss of consciousness and involuntary passage of urine & stool for 1 day. His past medical history revealed that he had first episode of headache, dizziness and brief unconsciousness of 1-2 minutes about 4 months ago. After that about a month passed and nothing happened but then one day evening, again he was having severe headache followed by 2 bouts of projectile vomiting. Then there was sudden gaze fixation and staring for 1-2 minutes followed by loud cry & use of obscene language (for about 30 seconds), never earlier used in daily life, and then he started having jerky movement of the whole body, rolled on to his right side, had up rolling of eyeballs with frothing from mouth, and incontinence of urine and stool. The whole episode had lasted for about 5 minutes according to the informants. After regaining consciousness the patient remained confused and disoriented for about ½ hour. Later on also he did not remember anything about these events. A similar episode occurred when he was taken to the civil hospital. Then he was referred to the higher centre. He was put on Sodium Valproate 500mg single dose at night and Diazepam 10 mg SOS. Routine investigations and EEG done at that time showed nothing abnormal.

Patient came for follow up 15 days later, and then the dose of Sodium valproate was increased to 750mg at night. He maintained full compliance with the treatment and had regular follow up visit for next 3 months. He remained seizure free during this period.

After that, one evening patient again started having headache, dizziness and blurring of vision in the evening. Suddenly he developed gaze fixation / staring in one direction for 1-2 minutes, gave a sudden loud cry and started using abusive language with slangs. There were no specific content or repetition of sentences. His uncle and grandmother who tried to catch hold of him were violently pushed away. Then he developed jerky movement of the whole body in somewhat flexed position, frothing from the mouth, facial deviation with facial muscle twitching, up rolling of eyeballs and then loss of consciousness. This lasted for about 4-5 minutes but no incontinence of urine or stool was reported. When he opened his eyes after that he remained confused for about ½ hour, could not recognize his family members and could not answer any question. Despite this he was given the scheduled dose of Sodium valproate 750mg at night.

But after about 2 hours he developed a second similar episode. This started with little vomiting, followed by gaze fixation, sudden loud cry, use of abusive language, jerky body movement, frothing from the mouth and involuntary passage of urine and stool. The patient remained unconscious for about 15-20 minutes and after regaining senses also, was confused and could not recognize people or answer their question. Then after about 45min-1 hour, 3rd episode started in the similar fashion and almost similar events were repeated. Thereafter the events kept repeating themselves at an interval of every 30-45 minutes and the patient never gained full consciousness in between. He was described by attendants as restless, anxious, confused and disoriented during the intervals of seizures. In total about 8-10 episodes of seizures occurred in the time span of 6-8 hours.

No medical facilities were available in their locality so finally in the morning he was brought to our centre.

At the time of presentation the patient was confused, looked in motionless stare, had poor muscle tone, did not respond to verbal command and was disoriented to time, place and person. Afterwards also he had no recollection of the events of the previous night. The investigations done were – Blood & Urine routine examination, LFT, KFT, VDRL, RBS & Serum Electrolytes and they were found to be within normal limits. But the CT scan of Head showed "small ring enhancing focus of size 13mm, with central hyperdense dot and perifocal edema in left frontal lobe – suggesting possibility of Neurocysticercosis (colloidal vesicular stage)." He was put on Injection Diazepam 10mg i.v slowly stat & SOS, Sodium Valproate 500 mg twice daily and Clobazam 10 mg at night. Steroids (Prednisolone), PPI, were added to the treatment regimen along with Albendazole 400 mg twice daily and the supportive treatment continued as well. Later on the other investigations like stool routine examination showed normal results & absence of egg/larva/cysts, and ELISA for anti Taenia Solium antibody (IgG) came out to be Non Reactive. Fundoscopic examination of eye revealed no abnormality.

There was no history of Head injury/DM/TB/HTN/Metabolic Disease/Congenital anomalies/febrile seizures in childhood or family history of seizures. The patient was a teetotaler but relished non-vegetarian food and had once taken pork also. Being from a rural background, he also enjoyed taking fresh vegetables (cooked, uncooked) and fruits. His General and Systemic examination did not reveal any abnormality except Plantar indeterminate on right side. His higher cognitive functions tested later on were found to be intact.

The patient was kept in the hospital for a week and was then discharged with Sodium Valproate 500 mg twice daily, Clobazam 10 mg at night, Albendazole 400 mg twice daily, Pantoprazole 40 mg in the morning empty stomach and multivitamins along with Prednisolone in titrated doses. His first follow-up was planned after two weeks, which he complied to. He was then put on Sodium Valproate 500 mg twice daily, Clobazam 10 mg at night. He was seizure free till his 2nd follow-up after 2 months. But repeat CT Scan of Head done at this time, reported "well defined, rounded, ring enhancing hyperdense lesion measuring (1.1x1.0cm), with perilesional white matter edema in the left frontal lobe - Granulomatous lesion. Possibly Neurocysticercosis".

Though the patient was seizure free with the above medication but headache persisted, so, considering the non resolution of intracranial lesion and fearing poorer prognostic outcome in the future the patient and his attendants opted to go for the surgical treatment and removal of the lesion. Neurosurgery was performed consisting of left frontal craniotomy and excision of SOL on 07.10.09 under ETGA. Follow-up CT Scan Head done on 09.10.09 showed post-operative changes and residual white matter edema in the left frontal lobe. No abnormal enhancing lesion was noted - suggestive of evacuation of the granuloma. Rest of the Brain parenchyma appeared normal. Histopathology of the single nodular lesion done at NIMHANS, Bangalore on 14.10.09 confirmed the diagnosis of NCC stating in the final impression that it was degenerated cysticercal cyst: left frontal. Patient was post-operatively treated with antibiotics, analgesics and AED'S and other supportive measures. His general condition improved and at the time of discharge he was conscious and oriented, obeyed verbal command, moved all 4 limbs, was able to take food orally and his wound was healthy.

He was discharged on 15.10.09 with Sodium Valproate 500 mg thrice daily, Clobazam 10 mg at night. He has also been advised follow-up in the Neurosurgery OPD after 3 months. Till latest report he is said to be seizure free and maintaining follow up regularly.

DISCUSSION: Neurocysticercosis can affect people of any age group. There are reports of NCC in a girl of 2.5 years age presenting with fever, vomiting, neck rigidity, headache, and seizures (11). Samir Vora and his colleagues studied 450 persons in a village from Goa with Elisa test to diagnose Cysticercosis. The sero-prevalence rate was 22.4%, one of the highest in the world. The sero-prevalence increased with age and peaked at around 65 years. There were no statistical association between seropositivity and religion or pork consumption (12). This indicates the generalized exposure to T. Solium eggs through fruits and vegetables.

In a group of 203 patients with parenchymal NCC studied by Del Brutto et.al in 1996, 86 single lesion NCC were found to result in GTCS(52,60%) and simple partial seizures [34,40%] , the remaining cases(117) had multiple lesions which could result in GTCS [69, 59%] simple partial seizures [44,38%] or complex partial seizures [4, 3%] (1).

Complex partial seizure is a rare presentation of solitary neurocysticercosis as is in our case. Complex Partial Seizures can arise from the medial temporal lobe or inferior frontal lobe (6). Those arising from the temporal lobe often begin with a motionless stare followed by simple oral or motor automatism. In contrast frontal lobe seizures often begin with vigorous motor automatism or stereotyped clonic or tonic activity (7). In our case we had mixed presentation. It also shows that NCC is a pleomorphic disease. This may be due to variations in the locations of lesion, the number of parasites and the host's immune response (4).

Status Epilepticus is a life threatening condition and needs emergency medical care. There are very few reports of Neurocysticercosis presenting as status epilepticus. G E Peachell reported a case of Status Epilepticus and death due to cerebral cysticercosis (10).

The other varied presentations may be headache, intracranial hypertension, strokes, neuropsychiatric disturbances, diplopia, hydrocephalus etc. (1, 4, 6). But vomiting is an unusual feature of NCC presenting with Epilepsy.

The diagnosis and treatment of NCC have been considerably improved in recent years. Del Brutto and colleagues (13) provide diagnostic criteria for neurocysticercosis based on objective clinical, imaging, immunologic, and epidemiologic data. These include four categories of criteria stratified on the basis of their diagnostic strength, including the following: 1) absolute, 2) major, 3) minor and 4) epidemiologic. Interpretation of these criteria permits two degrees of diagnostic certainty: 1) definitive diagnosis, in patients who have one absolute criterion or in those who have two major plus one minor and one epidemiologic criterion; and 2) probable diagnosis, in patients who have one major plus two minor criteria, in those who have one major plus one minor and one epidemiologic criterion, and in those who have three minor plus one epidemiologic criterion. Our case fulfilled one absolute (histopathological), one major (highly suggestive cystic lesion in CT scan) and one epidemiologic criteria (coming from an endemic area).So we had a definite diagnosis of Neurocysticercosis.

Albendazole has been found to be more effective than Praziquantel in reducing the total number of cysts in the CT scan Head (5). Albendazole has better penetration into CSF, is cheaper and its concentrations are not affected when given with steroids (2). To avoid exacerbation of

neurological symptoms attributed to local inflammation due to death of the larvae both Albendazole and Praziquantel are generally given simultaneously with steroids, in order to control the edema and intracranial hypertension. In a meta-analysis of randomized trials assessing the effect of cysticidal drugs on neuroimaging and clinical outcomes of patients with neurocysticercosis, Del Brutto and colleagues found that Cysticidal drug therapy results in better resolution of colloidal and vesicular cysticerci, lower risk for recurrence of seizures in patients with colloidal cysticerci, and a reduction in the rate of generalized seizures in patients with vesicular cysticerci (14). However our patient continued to have headache and non resolution of the cyst in neuroimaging even after 3 weeks of albendazole therapy.

The patient's treatment from the drug therapy to neurosurgery was according to the Current Consensus Guidelines for the treatment of Neurocysticercosis (2).

CONCLUSION:-

1. Neurocysticercosis is one of the serious public health problems in India. Need of Community Mental Health, Public Health care awareness regarding this condition is urgently felt.
2. Aggressive multidimensional approach for early identification of N.C.C with M.R.I., C.T. Scan & Serological test should be considered before invasive neurosurgical approach & antituberculous therapy.
3. N.C.C. should be considered in all cases of Epilepsy, Headache, and mental deterioration in the patients from endemic area.
4. N.C.C. may be treated with ABZ, which is safer, effective & least toxic.
5. Oral steroid is to be started prior to starting ABZ & tapered off after conclusion of treatment. Anti-convulsant & analgesic medication needs to be given as per requirement.

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