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PNEUMOCOCCAL MENINGITIS ASSOCIATED PYOGENIC VENTRICULITIS: A CASE REPORT.

Medicine

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ABSTRACT

Background: Pyogenic ventriculitis is uncommon yet fatal complication of the inflammation of the ventricular ependymal lining associated with a purulent ventricular system. The commonest organism can be gram negative organism especially if happened after neurological procedure .

Case report: 63 years old male known case of Diabetes mellitus (DM), hypertension (HTN), Parkinson diseases (PD), treated lymphoma on chemotherapy four years ago. Presented with high grade fever, delirium and seizure. Admitted to the Intensive Care Unit (ICU) as a case of septic shock. Patient intubated and started on norepinephrine, intravenous fluids, anti-epileptic (levetiracetam) as well as anti-meningitis antibiotic (ceftriaxone , vancomycin , ampicillin and acyclovir) plus dexamethasone. His Cerebral Spinal Fluid (CSF) analysis showed: glucose 4.6 (high). CSF protein 3.3 (high), CSF WBC 4000. Blood culture showed streptococcus pneumoniae. Brain MRI was done and showed evidence of ventriculitis in form of fluid level at occipital horn of lateral ventricles, no edema no mass effect no hydrocephalus, left hypocampus hyperintensity. Diagnosis of pyogenic ventriculitis was made. Same antibiotics was continued. Repeated brain MRI on day 23 of admission showed some regression of the intraventricular fluid and interval resolution of the left hippocampus signal intensity. Finally, Patient regained full consciousness and improved dramatically and was kept for physical rehabilitation for generalized motor weakness of critical illness polyneuropathy.

Conclusion: Pyogenic ventriculitis is a rare potentially lethal condition. Early diagnosis and intervention is necessary to reduce mortality and morbidity. Imaging plays an important role in establishing diagnosis in adjunct to CSF studies. Once diagnosis is established, appropriate antibiotic treatment must be started based on culture sensitivity.

KEYWORDS

Pyogenic ventriculitis, Pneumococcal Meningitis, co-infection, Non- HIV patient without underlying disease

INTRODUCTION:

Pyogenic ventriculitis (POVE) is defined as exudative inflammation of the ependymal lining of ventricles resulting in accumulation of pus in the ventricular system (1). It is uncommon and lethal condition that happens following meningitis, trauma or iatrogenic CNS procedure (2). It can be triggered by tiny abscess rupture into the ventricles and disruption of the blood brain barrier (3). Causative organism are gram negative organisms, followed by staphylococcus, streptococcus and enterobacter species (1). In immunocompromised individuals Cytomegalovirus, Nocardia and Toxoplasma can cause ventriculitis (1). Clinical manifestations can be high fever, nuchal rigidity, photophobia, delirium, seizures (3). Cerebral spinal fluid analysis and the MRI imaging can be very indicative in reaching the appropriate diagnosis (4).

We report a case of streptococcal pyogenic ventriculitis in a 63years male who developed the complication due to severity of the disease.

Case presentation:

63 years old male known case of Diabetes mellitus (DM), hypertension (HTN), Parkinson diseases (PD), History of treated lymphoma on chemotherapy four years ago. Presented to our emergency department (ED) with high grade fever, along with earache four days prior to presentation. Decreased level of consciousness and on- off confusion one day prior to presentation. The patient had history of head trauma (fall down) one day prior to presentation. No previous history of seizure. No History of previous surgeries nor prior admission. He is on antiparkinson agents (carbidopa/levodopa) and Amlodipine for hypertension. The patient presented to the ED On examination he was found to have high grade fever temperature 38.5. Also, he was found hypotensive and tachycardia (blood pressure 84/43, pulse 130). During physical examination, the patient was confused in general and had witness seizure aborted with diazepam (witnessed by the nurse and the oncall doctor). The examination was limited but in general, both pupils are equal in size and react to light. Initial laboratory findings upon presentation showed leukocytosis (CBC: Hb 13.9, WBC 25.2, PLATLET 335). Renal function test RFT (creatinine 369 µmol/L, BUN 12.9 mmol/L , Na 132 mmol/L , K 5.5 mmol/L , CK > 18141 U/L). Liver function test LFT (total bilirubin 5 umol/L , LDH 2065 U/L , GGT 119 U/L). Arterial blood gases showed high lactate with mixed respiratory and metabolic acidosis (PH 7.010 , HCO3 9.9 , LACTIC ACID 6.2). Brain CT scan was done which revealed normal finding. A provisional diagnosis of meningitis was made. The patient was admitted to the intensive care unit (ICU) as case of septic shock, intubated and started on norepinephrine, Intravenous fluid, anti- epileptic (levetiracetam) as well as anti-meningitis antibiotic (ceftriaxone, vancomycin, ampicillin and acyclovir) plus dexamethasone.

Few hours after admission, patient re-evaluated again, he was found to be anuric since admission, potassium 7.7 (hyperkalemic), Continuous Renal Replacement Therapy (CRRT) was started. Lumbar puncture done by ICU team which revealed turbid whitish cerebral spinal fluid (CSF). Results of the CSF analysis are as shown: CSF glucose 4.6 (reference range 2.22-3.9). CSF protein 3.3 (reference range 0.13 - 0.45), CSF WBC 4000 with polymorphs predominant. Blood culture showed streptococcus pneumoniae, which made the diagnosis of bacterial meningitis with secondary bacteremia more likely. So, patient was continued with antibiotic: namely ceftriaxone for the treatment of bacterial meningitis based on culture and sensitivity with the E-test minimum inhibitory concentration (MIC) of pencillin < 0.06 and for ceftriaxon < 0.12 aim to finish 6 weeks intravenous antibiotic. Continued on the CRRT due to the anuria of acute kidney injury and sever metabolic acidosis, sever hyperkalemia and rhabdomyolysis.

Third day of hospital course, brain MRI was done and showed evidence of ventriculitis in form of fluid level at occipital horn of lateral ventricles, no edema no mass effect no hydrocephalus, left hypocampus hyperintensity (figure. 1). Neurosurgery were consulted and no need for any surgical intervention.

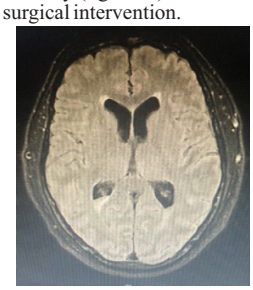

Figure1. Brain MRI done on the third day of admission.

Fourth day of admission, lumbar puncture was repeated and the results were as follows: WBC 230 , CSF protein 1.2 , CSF glucose 6.2

Fifth day of admission, tracheal aspirate culture showed (pseudomonas aeruginosa). so the antibiotic was switched to cefepime 2 g IV Q 8 hour to cover CNS (streptococcus pneumonia) and chest (pseudomonas aeruginosa)

Seventh day, the patient improved dramatically and started opening his eyes and obeying verbal commands.

On day 14 of mechanical ventilation, the decision for tracheotomy was taken and it was successfully done on day 17. Patient required heamodialysis so premicath was done for him without complications. Patient was successfully weaned from mechanical ventilation. After 23 days in intensive care unit, patient improved clinically and biochemically. He became hemodynamically stable and was shifted to medical ward. Brain MRI showed some regression of the interaventricular fluid and interval resolution of the left hippocampus signal intensity (figure. 2).

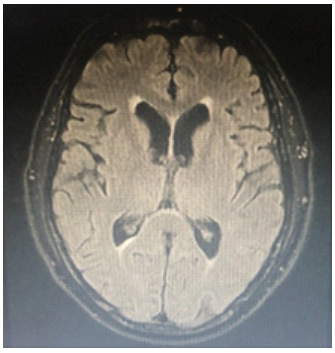

Figure 2. Brain MRI done on day 23 of admission

Patient improved clinically and biochemically, continued the course of antibiotic, regain his consciousness level with glascow coma score 15/15 except for limited motor activity allover that Electromyography (EMG) shows axonal motor and sensory polyneuropathy in correlation with clinical picture going with critical illness polyneuropathy, where full rehabilitation plan and home care for his current condition was arranged but also he had renal failure required renal replacement therapy that improved with time and required one session of dialysis per week and expected to recover over couple of months.

Two weeks after follow up and brain MRI showed further decrease in the amount of bilateral intraventricular fluid. (Figure 3.) shows a comparative between the first brain MRI and the last one and it confirms the further decrease in the amount of fluid.

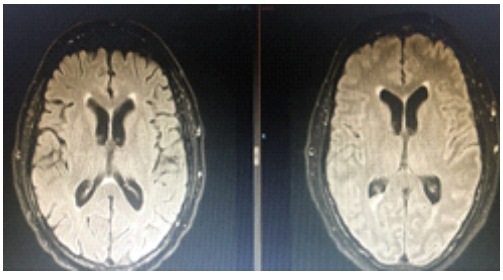

figure 3. shows comparative brain MRI from first one on 12/3/2019, and the last one on 17/4/2019

DISCUSSION:

Neuological complications of pneumococcal meningitis can happen and the most frequent ones are cerebral vasculopathy affect up to 56% , cerebral oedema affect up to 29% and hydrocephalous that affect 16% of the patients (4,5). These complications are associated with poor outcome in general. During the past decade the use of MRI in the patients with pumenococcal meningitis has increased. MRI findings showed parenchyma and sulfa signal changes, pyogenic ventriculitis in some cases (6).

Pyogenic ventriculitis is an uncommon complication of community acquired meningitis. It is frequently fatal infection that results from inflammation of the ventricular ependymal lining associated with a purulent ventricular system (7). This infection can lead to hydrocephalus and death if not promptly recognized and treated. Such infections have increased (infection rate of ventricular-catheter raising up to 20% in some series) and are caused by microorganisms involved in foreign body infections such as staphylococci or antibiotic resistant Gram-negative bacilli (8)

Brain MRI as it shows as periventricular hyperintensity, an ependymal enhancement and irregular intraventricular debris layering in the occipital horns (9). The presence of irregular intraventricular debris is quite specific for pus and helps differentiating from a straight level of acute clotted blood (10). In our case, the patient brain MRI showed inflammatory change encephalitis versus subacute infarction in the left hippocampus region. Also, intraventricular pus ventriculitis versus acute intraventricular haemorrhage.

In a similar case series of 14 patients with pneumococcal meningitis who were treated with intravenous ceftriaxone and ampicillin plus adjuvant dexamethasone initially. Once the pathogen was identified, antibiotic therapy was adjusted to ceftriaxone monotherapy. Brain MRI were done and all 14 patients had hyperintense signals in one or both lateral ventricles. However, the flow up brain MRI showed the intraventricular hyperintensities were resolved and so is the ventriculitis in 7 patients who came for follow up after discharge (11). As far as our patient is concerned, he showed response to the antibiotics intravenously proved by the brain MRI.

As a conclusion, pyogenic ventriculitis is not associated with poor outcome in pneumococcal meningitis. Intravenous antibiotics are sufficient to treat it. However, the timing of the incidence of the ventriculitis cannot be determined by this study nor the previously mentioned case series (11) due to the limitation of the retrospective studies. A more prospective study should be considered in the future.

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