Central pontine myelinolysis presenting with depressive features
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Abstract: INTRODUCTION Central pontine myelinolysis is defined as a symmetric area of myelin disruption associated with rapid correction of hyponatremia. Clinical presentation is with flaccid paralysis, altered consciousness and often facial or bulbar weakness. Here we report this case of central pontine myelinolysis, who initially presented with symptoms of depression. CASE REPORT 45 year old female was referred for complaints of decreased appetite, decreased communication, sleep disturbance, crying spells for past 10 days from medicine ward where she was admitted for the same. She is a known case of Autosomal dominant polycystic kidney disease, treated for urosepsis in the nephrology ward, 20 days back. There was a past history of suicide attempt five years back following interpersonal conflicts. Physical examination revealed stable vital parameters, brisk deep tendon reflexes in the neurological examination. She was found to be restless, irritable and answering in monosyllables for most of the questions. Her attention was arousable, but ill sustained. All the features mentioned above, were not consistent with a functional aetiology and neurologist opinion was sought. Neuroimaging revealed hyper intense lesions in the central pons, caudate nucleus and Putamen, suggestive of central pontine myelinolysis. Conclusion The series of events presented here highlight the need, to consider psycho pathological behaviors as sequel, of electrolyte abnormalities. It is imperative to rule out neurological and metabolic abnormalities before entertaining a psychiatric diagnosis in any patient.

Keywords: pontine myelinolysis, hyponatremia, depression

Introduction: Central pontine myelinolysis (CPM) is a clinically heterogeneous, demyelinating condition originally thought to occur only in the central pons. When demyelination is found in areas outside of the Pons, the disorder is referred to as either extrapontine myelinolysis (EPM) or central and extrapontine Myelinolysis (CPEPM). Common presentation of CPM are a combination of neuropsychiatric (e.g., emotional lability, disinhibition, and other bizarre behaviors) and neurologic (e.g., confusion, impaired cognition, dysarthria, dysphasia, gait instability weakness or paralysis, generalized seizures) symptoms. Catatonic symptoms, auditory hallucinations & movement disorders associated with central pontine myelinolysis have been reported, but CPM presenting with features of depressive stupor, has been rare.
neuropsychiatric medications have hyponatremia as a potential complaint, neurological examination and mental state findings, considered as a case of depression. With the above said attempt in the past and histrionic personality traits, the patient was stressors, suicide is secondary to impairment in potassium siphoning, as has been mentioned to reestablish normal cell volume. An alternative possibility, causing brain cells (primarily astrocytes) to swell. Brain cells quickly on the onset of hyponatremia, water moves from the blood into the brain, hyponatremia can portend a higher risk for CPM. During acute a more critical patient presentation, but more chronically developing (48 hour development), as compared with slowly progressive (SIADH), renal disease, cancer, pregnancy, and in high-endurance abnormalities, the syndrome of inappropriate secretion of ADH is better explained on the basis of an organic pathology, rather than increased psychomotor activity, irritable mood, ill sustained attention & brisk deep tendon reflexes in both the lower limbs which is better explained on the basis of an organic etiology. Because there have been many atypical features for depression such as poorly established rapport, facial grimaces, irritable mood and brisk deep tendon reflexes in the examination, which prompted us to think in terms of an organic etiological basis. In the back ground of autosomal polysonic dominant kidney disease, central pontine myelinolysis seems plausible on the basis of hyponatremia induced demyelinating changes. Central pontine myelinolysis (CPM) is a rare neurologic disorder of demyelination that was first reported by Adams et al over 50 years ago in a malnourished 38-year-old man with alcohol dependence. In addition to malnourishment and alcohol dependence, the Adams et al case-report patient was also described as having hyponatremia. Since that time, several studies have shown that rapid overcorrection of hyponatremia exacerbates CPM as well. Common symptoms of CPM include weakness, quadriplegia, pseudobulbar palsy, behavioral changes, psychosis, and cognitive disturbances. By definition, it is a non-inflammatory, frequently symmetrical central pontine demyelination.(Adams et al). Osmotic demyelination syndromes are rapidly progressive and usually fatal. The most common cause of CPM is an overly-rapid correction of hyponatremia in patients with conditions leading to nutritional or electrolyte stress, such as alcoholism, liver disease, immune suppression after transplantation, malnourishment with underlying medical disease, gastrointestinal disease with acute electrolyte abnormalities, the syndrome of inappropriate secretion of ADH (SIADH), renal disease, cancer, pregnancy, and in high-endurance exercise (in triathletes, marathon runners, etc.). Acute hyponatremia (48 hour development), as compared with slowly progressive chronic hyponatremia (48 hours in development), generally causes a more critical patient presentation, but more chronically developing hyponatremia can portend a higher risk for CPM. During acute onset of hyponatremia, water moves from the blood into the brain, causing brain cells (primarily astrocytes) to swell. Brain cells quickly adapt to hyponatremia by losing inorganic osmolytes (electrolytes) in order to reestablish normal cell volume. An alternative possibility, suggested by the presence of structures similar to Rosenthal fibers within perivascular astrocytes (see below), is that myelin destruction issecondary to impairment in potassium siphoning, as has been recently proposed for several other demyelinating brain diseases. Psychiatric illnesses (in addition to alcoholism) and many neuropsychiatric medications have hyponatremia as a potential complication. Myelinolysis may occur as a severe complication of eating disorders, especially anorexia nervosa (AN). One of the most important reasons can be a rapid correction of hyponatremia caused by tubulopathy, water intoxication (WI), or abuse of diuretics in individuals with AN. Among patients with chronic mental illness, 10%–25% may have primary polydipsia, increasing the risk of hyponatremia and premature death. Primary medications or classes of medications used daily in neuropsychiatry in which hyponatremia is a side effect or reported to occur after use include carbamazepine, oxcarbazepine, the serotonin reuptake inhibitors (SSRIs), lithium, tricyclic antidepressants, opioids, and poly pharmacy with multiple antipsychotic drugs. Other classes of commonly used medications that increase risk of hyponatremia include salt-losing diuretics, nicotine, non steroidal anti-inflammatory drugs, and acetaminophen. Central pontine myelinolysis is frequently associated with loss of consciousness, progressive lethargy and coma, ataxia, and changes in reflexes which develop over two to seven days after the onset of the treatment of the underlying disease or correction of hydroelectrolytic imbalance. As a consequence, a typical pseudo coma, also known as the locked-in syndrome (quadriplegia, anarthria, a capacity to follow the examiner with the eyes but not to follow command, bilateral Babinski sign) develops. Clinical features are tetra paresis; usually spas tic although flaccidity has been described especially soon after onset, pseudo bulbar palsy: dysarthria, dysphagia , weakness of tongue and palatal movement, exaggerated jaw jerk and emotional lability early depression of conscious level progression to ‘locked-in’ state , other brainstem features according to extent of myelinolysis; failure of ocular movement, decreased or absent pupil reactions , cerebellar ataxia if cerebellar peduncles or cerebellar hemispheres are involved ,seizures may occur if associated with hyponatraemic encephalopathy. Autosomal dominant polycystic kidney disease is an inherited systemic disorder that predominantly affects the kidneys, but may affect other organs including the liver, pancreas, brain, and arterial blood vessels. Approximately 50% of people with this disease will develop end-stage kidney disease and require dialysis or kidney transplantation. In 85% of patients, ADPKD is caused by mutations in the gene PKD1 on chromosome 16 (TRPP1); in 15% of patients mutations in PKD2 (TRPP2) are causative. Autosomal polycystic kidney disease results in failure of the concentrating ability of the renal tubules, thus resulting in compensatory hyponatremia. The rapid therapeutic intervention leads to demyelination of the neurons in the pontine & extrapontine areas & thus explains the current picture. There have been studies on hyponatremia induced psychosis, hyponatremia being a predisposing factor for a catatonic symptoms, factor in the causation of alcoholic hallucinosis and a frequent association with the alcohol withdrawal state. In term of assessment, the possibility of an organic cause should always be kept in mind while assessing patients with psychiatric symptoms. There may be clues that patient has an organic disorder, some factors which suggest an organic cause of psychiatric symptoms are: atypical psychiatric presentations, failure to respond to treatment, different to previous psychiatric presentations, abnormal physical examination, no clear aetiology. Conclusion: The series of events presented here highlight the need, to consider psychopathological behaviors as sequelae of acute urosepsis and electrolyte abnormalities. It is imperative to rule out metabolic and neurological abnormalities prior to diagnosing a patient with a psychiatric diagnosis. It is deduced to think about functional etiology whenever there is an atypical presentation but it should be borne in mind that...
atypical initial presentation of psychological symptoms do occur in diseases with organic basis. It is mandatory to rule out organic causes considering the impending Consequences

References:
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