Sudden Impolite Behavior in a Polite Girl: a Case Report

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Abstract- In this case we present a previously healthy child with sudden behavioral change and acute confusional state (ACS). ACS is a neurologic emergency. The clinical manifestations of ACS are; confused affect, limited verbal response, disturbance in performing orders, some automatism, stable vital signs and absence of tonic-clonic movements and it has a wide range of differential diagnosis which should be assessed by detailed medical history, medical examination and laboratory tests. The only abnormal test found in this patient, was cerebrospinal fluid abnormality, consistent with viral encephalitis.

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Case Report

A 9 year-old girl presented to the emergency department with sudden onset of aggressive behavior. According to her mother's statement, she was absolutely well until the previous day. She was a polite child at home and a well organized student in school.

The previous day, her mother noticed absent-mindedness behavior and no directional gaze of the patient, and the child started asking irrelevant questions like asking her mother "who are you?" ‘‘Who are these people in our house?" " Are you my mother?".

Her mother ignored this behavior and asked her to wear her scarf and getting ready for going to a travel, the girl didn't pay attention and by insisting of her mother, she burst to cry and got restless and shouted that she doesn't know how to wear her scarf and asked her mother to teach her how to wear the scarf. She repeated the same questions several times.

From the next day she became very aggressive, and when taken to hospital, she attacked and insulted the physicians who were trying to examine her. Since being admitted in hospital she was restless, aggressive but allowed a doctor to examine her just once with full cooperation and appeared normal in physical and mental state exam but just after the exam finished, she started being aggressive again.

She didn't have any drug consumption, vomiting, fever and head trauma recently, but she sustained an intermittent headache since 10 days ago.

Her past medical history was not notable.

In family history we notified that her father has a history of severe headaches for many years and her mother has had the history of hypothyroid disease.

The personality history of the child revealed that she has always had a strong guilt feeling and by beginning the routine religious assignments since 1 year ago, she started having severe obsessive behavior. About 8 months ago she was punished severely by her mother following being curious about sexual issues in school and became very isolated for one month afterward and started to talk to herself.

The psychiatric consultation reported; short term memory defect and disorientation and suggested encephalopathy.

In the previous month her mother increased the amount of meat in her daily meals.

The general physical and neurological exam and vital signs were normal and stable.

2 days after of being admitted she became more agitated and walked fast aimlessly in the ward. Risperidone 1 mg daily administered to control the agitation.

The next day she refused any eye contact, any verbal response and eating food. She lost urine and stool control and didn't come down of the bed for the whole day.

The lab work results showed:

Complete Blood Count: white blood cell=9770/µl

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(reference range: 4000-10000/µl), neutrophil=73%, lymphocyte=20%, hemoglobin=13.9, platelet=260,000/µl.

The urine screening test for toxins and medications were negative.

Blood lab work of electrolytes; sodium, potassium, calcium, magnesium, blood urea nitrogen, creatinine, ammonia, lactate, arterial blood gas, thyroid function tests, hepatic function tests, were within normal range.

Wilson disease tests performed and were negative.

Brain CT scan was suspicious of mild brain edema, and brains MRI were normal. Because of the possibility of brain edema, dexamethasone and mannitol prescribed for 2 days.

A lumbar puncture performed with following results: white blood cell=33/µl, lymphocyte=90%, red blood cell=4/µl, glucose=62 mg/dl, protein=16 mg/dl. Because of the possibility of viral encephalitis, acyclovir prescribed.

Discussion

Recurrent the child with acute confusional state (ACS) is a diagnostic challenge because it has several differential diagnoses. ACS is a neurologic emergency (4).

The clinical presentations of ACS are; dazed affect, limited verbal reactions, disturbance in performing orders, automatism, normal vital signs and absence of tonic-clonic movements (4).

Some of the differential diagnoses are; acute confusional migraine (ACM), non-convulsive status epilepticus (NCSE), metabolic disorders (eg. Hypoglycemia, diabetic ketoacidosis, Wilson disease), fluid and electrolyte disturbances (eg. hyponatremia), thyroid storm (rarely can be associated with levothyroxin use), uremic encephalopathy, central nervous system(CNS) diseases, head injuries, encephalitis, toxic substances digestion, non-convulsive status epilepticus, subarachnoid hemorrhages (SAH), rheumatologic problems and autoimmune diseases (systemic lupus erythematosus, antiphospholipid syndrome), central nervous system vascular diseases, stroke, cerebral hypoxia and disturbed brain perfusion (choking, drowning, heart arrhythmia, hypotension, carbon monoxide toxicity), demyelinating diseases (acute disseminated encephalomyelitis), epileptic disorders, postictal situations and psychiatric disorders (1,4,5,8-10,12,14,17,29,20,22,23,25,27-30,31).

Knowing the wide range of differential diagnosis of ACS, it has also been reported in a case of polyarteritis nodosa which caused inappropriate antidiuretic hormone secretion and hyponatremia (8).

In this case, we faced a previously healthy child without head trauma and previous medical conditions. They denied any drug digestion or history of epilepsy and normal physical and neurological exam.

The electrolyte panel, thyroid, renal and hepatic functions were normal and cannot be considered as the cause of the problem. Imaging studies ruled out the possibility of demyelinating diseases.

Wilson disease ruled out. Hypoglycemia which is an important cause of psychosis and hallucination in children (9) was not found and no hypoglycemic medication (*e.g.* ethanol, sulfonylureas or beta blocker) was ingested.

Many medications can be the cause of ACS, such as drugs with anticholinergic effects (*e.g*. atropine, diphenhydramine, hyoscyamine, chlorpheniramine, typical and atypical antipsychotics), sympathomimetics like amphetamine (10-12). Dextromethorphan and ketamine have hallucinogens effects too (13). Withdrawal syndromes like benzodiazepine withdrawal are also of the causes of psychosis in children (14). Steroid-induced psychosis and antibiotic-induced mania even by commonly used antibiotics like amoxicillin, clarithromycin and erythromycin are known entities (15,16).

CNS abnormalities (17), brain infections by agents like Chlamydia, mycoplasma, Epstein-Barr virus and cerebral malaria have been associated with psychosis (18).

A history of headache in the previous days could lead the doctors to consider CNS problems like ACM and encephalitis.

ACM is a rare form of migraine and electroencephalogram changes are nonspecific (7) and often occurs in late childhood and early adolescence and is a diagnosis of exclusion. Attacks terminate with sleeping and are usually self limited and don't need any specific treatment. 50% of the patients of ACM have a history of migraine disorder. Head injuries could be a trigger of ACM (1).

Non-convulsive status epilepticus (NCSE) should be considered in all children who admitted with encephalopathy and performing Electroencephalogram (EEG) must be a part of evaluation. NCSE should be considered in a child with cognitive and behavioral changes which last at least for 30 minutes and with evidences of EEG changes of epilepsy (6). The diagnosis of NCSE is given when a child is nonresponsive, rigid or in a child with previous known epileptic disorder. EEG in Absence Status Epilepticus

shows bilaterally synchronous and rhythmic 1.5 to 4 Hertz slow spike wave discharge (4).

Recently has been shown that some of the cases of encephalitis in children and adults has been caused by autoimmune conditions and production of antibodies against surface proteins of CNS synapses such as "excitatory glutamine N-methyl-d-aspartate receptor" (NMDAR), 'alfa-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor' (AMPAR), the "inhibitory gamma-aminobutyric acid-B" ($GABA_B$) receptor and the" leucine-rich, glioma-inactivated 1" (LGI1) protein. Patients with each of these auto antibodies can present typical limbic encephalitis syndrome with short term memory defects, behavioral disorders, seizure and confusion. In the case of NMDAR auto antibodies the patient would have extensive encephalitis with psychosis, catatonia, dyskinesia, autonomic disturbances and central hypoventilation. These diseases can be idiopathic or as a part of paraneoplastic syndrome (31).

The most relevant abnormal test found in our patient, was the lumbar puncture and cerebrospinal changes which was in favor of viral infection. So however the PCR exam of herpes virus of cerebrospinal fluid was negative, we deducted that our patient was afflicted by viral encephalitis.

At the sixth day of her admission she had secondary generalized partial seizure and phenytoin administered. The seizure started as twitches of left side of her face which last for less than one minute and repeated 3 times till phenytoin administered.

Her general condition got better and improved gradually and discharged after 3 weeks. 3 days afer discharge she had an episode of seizure again and carbamazepine was added to her treatment.

She was scheduled for the child psychiatric follow-up, her general condition improved but became reluctant to her religious assignments which she was committed to previously and showed more obsessive behavior.

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