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# Saccadic Intrusions: Don't Miss Toxins & Drugs

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Saccadic intrusions are involuntary conjugate saccades (rapid eye movements) that interrupt fixation. Although, some of these may be seen in normal individuals, most of the time these are pathologic. Generally, they are almost always pathologic if they are symptomatic. They often reflect dysfunction of the brainstem, cerebellum, superior colliculus, basal ganglia, and/or cerebral hemispheres1,2.

First, we have to differentiate nystagmus from saccadic intrusions. Nystagmus is classically described as rapid jerky movements followed by slow corrective saccades whereas in intrusions, the movements are purely saccadic.

The next step would be to distinguish two groups of saccadic intrusions by the presence or absence of an intersaccadic interval.

Saccadic intrusions with intersaccadic intervals such as square wave jerks, macro saccadic oscillations and saccadic pulses may be seen in neurodegenerative diseases and demyelinating diseases1.

Saccadic intrusions without intersaccadic intervals such as ocular flutter and opsoclonus can be seen in various conditions5. This includes parainfectious brainstem encephalitis, metabolic toxic states, demyelinating diseases, inherited disorders, and paraneoplastic conditions (primarily neuroblastoma in children, and small cell lung carcinoma, breast carcinoma or ovarian carcinoma in adults), although in many cases, the cause remains unknown1,2,4.

Ocular flutter and opsoclonus are rarely caused by drugs and toxins. This association has been reported in drugs/toxins such as cocaine, phenytoin, lithium, amitriptyline, phencyclidine and more recently venlafaxine.

We reported a case of a 21-year-old male presented acutely with deterioration of his GCS and seizures. Further he was complaining of a non-specific general ill health for 2-3 days. He started to rapidly deteriorate after admission. His GCS dropped to 10/15. He developed a tachycardia (140–160 beats/min), increased blood pressure (BP 170/100 mmHg), and an elevated body temperature of 40°C.

There was profuse sweating and hyper salivation but no evidence of increased muscle tone or rigidity. More specifically we were at times able to note ocular flutter and at times opsoclonus.

The patient was not on prior medications including neuroleptics. Even on direct questioning on admission he denied taking any toxins or recreational drugs.

His liver enzymes were elevated. AST>ALT. The creatine kinase (CK) was 40,000 µg/l suggestive of rhabdomyolysis.

Electroencephalogram demonstrated generalized delta activity, suggestive ofencephalopathy/generalized cerebral dysfunction. Non-contrast computed tomography brain (CT brain), septic screen and cerebrospinal fluid analysis all were negative or within normal limits.

Urine for toxicological screening was positive for PCP.

Despite the initial care, our patient rapidly deteriorated. His CK levels increased to 152,000 µg/l within 24 h and his renal functions declined rapidly. Unfortunately, the patient succumbed to acute renal failure secondary to rhabdomyolysis.

The fact we are trying to emphasize is that, if we didn't suspect and do the screening, we would have missed the diagnosis. The patient's history is not always reliable. It's true that we have lost the patient, this case is clearly a lesson for us.

Acute encephalopathy/encephalitis is one of the common scenarios we encounter in acute medical wards. The differential diagnoses for this kind of presentations are huge. Further they can deteriorate very rapidly as in this case. Therefore, high index of suspicion is the vital initial step for the accurate diagnosis.

A toxicology screen is a test that determines the approximate amount and type of legal or illegal drugs that you've taken. It may be used to screen for drug abuse, to monitor a substance abuse, or to evaluate drug intoxication.

Toxicology screening is a simple test which can be done quickly. It is most often done using a urine or serum sample. Occasionally hair follicles or saliva is also used. Initial test would be a qualitative assessment and if we need quantification, we have to test it separately. Sometimes the recreational drugs are taken in combinations in which case the test can reveal the presence of multiple drugs/toxins.

Depending on the drug, it may show up in the blood or urine within a few hours or weeks after being ingested. Certain substances, such as alcohol, are eliminated from the body quickly whereas some other drugs can be detected for several weeks after being used.

Many factors affect the length of time that a test can detect a certain drug in the body. These factors include body mass, hydration levels, the pH level of the urine and how long ago the person took the drug.

Obviously in case of repetitive use and heavy use the duration in which it can be tested will be longer.

Some examples are as follows4:

Alcohol 7-12 h

Amhetamines 48h

Heroin 48h

Short- acting benzodiazepines 3 days

Phencyclidine 8 days

Delaying in getting a relevant sample will increase the chances to get a negative result4.

Diagnosis of drug or toxin induced encephalopathy will be based on history of taking the drug, Clinical picture and confirming the presence of such drug or its metabolite in either serum or urine.

The problem arises when the patient denies taking such drug which happened in our case or not in a clinical state to mention it. Therefore, suspecting it early and getting the appropriate sample to test is vital. If not,we will end up in investigating extensively without fruitful outcome. Further, losing the tract may lead to increased morbidity and mortality.

There were no studies in literature which shows the percentage of saccadic intrusions in drug induced cases compared with other etiologies. Further, the options for symptomatic management of saccadic intrusions were also limited to hand full of case reports.

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Although rare, possibilities of drugs and toxins as the causative agents have to be considered even if it is a doubtful circumstance. On the other hand, we have to actively look for this clinical sign in confirmed patient with minor symptoms.

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