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A CASE STUDY ON EXPLORING TRICYCLIC ANTIDEPRESSANT OVERDOSE-INFUSED WITH HYPOTHYROIDISM

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ABSTRACT:

Tricyclic Antidepressants (TCAs) have a narrow therapeutic range and the patient became strong poisons of the central nervous system and cardiovascular system, they are the primary cause of harmful antidepressant overdoses. In this case report we have analysed a patient with Tricyclic Antidepressant overdose and the treatment was provided to the patient.

PRESENTATION OF CASE:

A 55 years old female patient was confirmed with tricyclic antidepressant poisoning. The patient took 12 tablets of Amitriptyline at her residence and she became drowsy. The patient had diagnosed with Hypothyroidism five years back with depression disorder for past two years on regular treatment. The Patient was treated with Anti-Emetics, Benzodiazepines, Calcium salts, Proton Pump Inhibitors, Hydrophobic amino acids.

KEY WORDS:

Anti Tricyclic Depressant overdose, Hypothyroidism, Amitriptyline.

INTRODUCTION:

Tricyclic antidepressants, or TCAs for short, are a class of medications that share a common pharmacological structure and mode of action. They could be applied to depression treatment.[2]

These drugs work by preventing the reuptake of neurotransmitters that influence mood, attention, and pain perception in people, such as norepinephrine and serotonin. Several TCAs have been approved by the U.S. Food and Drug Administration (FDA) for particular applications based on clinical trials and solid proof of their safety and efficacy for particular medical problems. TCAs are advised by evidence-based guidelines as a second-line treatment for major depressive disorder (MDD), after selective serotonin reuptake inhibitors (SSRIs). TCAs show mixed results when used with SSRIs to treat MDD; however, because of their

anticholinergic action and lower threshold for overdose. TCAs have been shown to be quite successful in treating severe or therapy-refractory depression; nonetheless, because of these features, they are generally not regarded as the first-line treatment for MDD. In order to improve the competency of interprofessional healthcare team members when providing care for patients with MDD and related conditions, this activity highlights the mechanism of action, indications, contraindications, adverse event profile, monitoring protocols, pertinent interactions, off-label uses, and other crucial aspects of TCA therapy on a clinical level.[1]

Since their introduction in the late 1950s, TCAs have been shown to be beneficial in the treatment of depression. They are not frequently employed as a first line of treatment, though.[2]

Certain TCAs have a greater effect on these neurotransmitters than others, which helps to explain why some TCAs are more effective in treating diseases other than depression, including pain, or why they are more prone to have negative side effects, such drowsiness, dry mouth, and constipation.[1]

EPIDEMIOLOGY:

The American Association of Poison Control Centres' 2021 National Poison Data System Annual report connected TCAs to 4705 single exposures and 15 fatalities. The most often used CA, amitriptyline, was responsible for 2106 exposures and five fatalities. Nortriptyline had 372 exposures and one death, while doxepin had 556 exposures and one fatality. Moreover, among the tetracyclic antidepressants, mirtazapine was responsible for 1613 single exposures and no fatalities. [3]

According to the database, TCA overdose was linked to 1.12 exposures per 10,000 individuals in 1992. Lately, victims of antidepressant overdose have been more likely to have taken SSRI overdoses. However, hospitalization rates for TCA overdose cases are higher due to their narrower therapeutic index compared to SSRIs. [4]

GENERAL MECHANISM:

This medication's exact mode of action is unknown. Amitriptyline may increase the concentration of transmitter amines at the brain's synaptic clefts by blocking the membrane pump mechanism that allows chemicals like serotonin and norepinephrine to be reabsorbed. These amines play a significant role in mood regulation. One of the first theories on depression is the monoamine hypothesis, which suggests that depression results from deficits in serotonin (5-HT) and/or norepinephrine (NE) neurotransmission in the brain. Amitriptyline may work by counteracting these systems, which is how it helps to alleviate depression symptoms.

Whether its analgesic effects are related to its mood-altering activities or attributable to a different, less obvious pharmacological action (or a combination of both) is unknown. [5]

MECHANISM FOR POISONING:[6]

TCAs are quickly absorbed in the digestive system. As TCAs have natural anticholinergic properties

an overdose may result in decreased gastrointestinal motility, delayed absorption, and toxicity.

More irregular absorption may result with co-administration of various anticholinergic drugs.

TCAs are mostly metabolized by CYP2D6, and this pathway's enzyme inducers and inhibitors may change how these compounds are metabolized.

Either the original chemical or one of its metabolites may be toxic.

The detrimental effects of TCA may be amplified and the unbound percentage may rise in respiratory or metabolic acidosis.

Poisoning symptoms often surface two hours after intake.

The patient has most likely not taken a large overdose if, six hours after ingestion, there are no clinically apparent signs of toxicity or abnormal ECG results, and the patient is able to be medically cleared for further testing, if necessary.

Significant toxicity typically lasts for 24 to 48 hours after it starts.

TREATMENT:

In situations of TCA poisoning, proper ventilation, circulation, and airway care are essential. Activated charcoal intestinal cleansing should only be carried out under the right circumstances, with the airway safeguarded. Up to two hours after ingestion, charcoal cleansing may be beneficial, particularly if the bowel noises are reduced. All possible measures should be taken to prevent acidosis from developing since it can worsen cardiac and neurological damage. Benzodiazepines are typically effective in treating seizures; but, in instances where seizures are unresponsive, anticonvulsants such propanol or phenobarbital should be promptly administered. In extreme cases, total anaesthesia may even be necessary. Patients who are hemodynamically unstable, have seizures, or have a QRS prolongation of more than 100 msec should be administered sodium bicarbonate. One meq/kg of sodium bicarbonate is administered as a bolus, and then the substance is infused intravenously. Reducing the QRS and maintaining a serum pH in the range of 7.5 to 7.55 are the objectives of this therapy. Patients with hypotension should get sodium bicarbonate and IV fluids.[7]

Activated charcoal lessens the amount of CAs absorbed. When more than one item is used, it could also be advantageous. For tricyclic antidepressant (TCA) poisoning, the American Association of Poison Control Centres (AAPCC) offers evidence-based management guidelines. This protocol is designed to help poison control centre staff treat patients who may have overdosed or ingested TCAs, as well as to aid in prehospital triage.[8]

TREAMENT PROCEDURE FOR INTAKING ACTIVATED CHARCOAL:

60g of activate charcoal was taken

It was mixed with water to make a slurry

It was administered to the patient through Nasogastric or Orogastric Tubes

It absorbs poison in the stomach and prevent bloodstream absorption

Activated charcoal bind with toxins and act as antidote

ALGORITHM:[9]

Managing tricyclic antidepressants (TCA) Poisoning involves several steps involves:

Access the patients Airway, Breathing and Circulation (ABC)

Consider Gastric lavage if ingesion occurred within one hour

Did the Patient was Stable?

Yes

No

Administer Activated Charcoal

Immediate Life-Saving Interventions

Monitor Vital Signs, ECG and Electrolytes

Did the Patient Experienced Seizures?

Yes No

Administer Benzodiazepines QRS Widening (or) Arrythmiasis ?

Yes No

Administer

Sodium Bicarbonate through IV

Intralipid Emulsion Therapy for Refractory Cardiac Arrest or Instability

Consider Hemolysis for Severe Toxicity or Overdose

Monitoring and follow-up

Continuously Monitor vital signs, ECG, Electrolytes and Mental status

Reassess patient Regularly.

CASE DESCRIPTION:

Mrs. X a female patient of 55 years presented to the emergency department with the complaints of intake of Amitriptyline 12 tables at her residence and she became drowsy and unconscious. She had a medical history of known case of hypothyroidism for five years and depression disorder for two years on regular treatment She had a past medication history of Tablet. Thyroxin 25 mg OD, Tablet. Amitriptyline 10 mg TID. On physical examination the patient was unconscious, disoriented and afebrile. On admission the patient has elevated blood pressure (140/90mmHg). The other vitals like pulse rate (87 beats/min) and Temperature (97.6°F) are normal. The Patient was treated with the following drugs for four days: Injection Emeset 4mg TID through IV (Anti Emetic), Injection Cysteine 1g BD through IV (Hydrophobic Amino Acid), Injection Calcium Gluconate 1g BD through IV (Calcium Salts), Injection Pantoprazole 40mg BD through IV (Proton Pump Inhibitors) Tablet Clonazepam 0.5mg HS through PO (Benzodiazepines) Active Charcoal 60g for every 6 hours through Nasogastric or Orogastric Tubes(Antidote), Injection Sodium Bicarbonate 60ml OD through IV (Alkalinizing Agents),Injection Ceftriaxone 1g BD through IV (Cephalosporin Antibiotics). The

Patient Condition was improved on the course of treatment and the patient was discharged in stable condition.

CONCLUSION:

We have presented a 55 years female patient diagnosed with Tricyclic Antidepressants Overdose. The patient was treated with Anti- Emetic, Hydrophobic Amino Acid, Calcium Salts, Benzodiazepines, Proton Pump Inhibitors, Antidote, Alkalinizing Agents and Cephalosporin Antibiotics. The patient condition improved on subsequent days and was discharged.

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