Diphenhydramin overdose

The woman had a brief episode of ventricular, tachycardia (ventricular tachycardia can be a fatal cardiac rhythm if not treated immediately), that spontaneously converted to a sinus tachycardia (a fast normal cardiac rhythm). She was given 2mg of intravenous naloxone (Narcan a narcotic antagonist), as well as one ampule (50g) of 50% dextrose for a blood glucose of 42 mg/dL. Neither drug improved her level of consciousness or respiratory status. The patient was subsequently endotracheally intubated prior to transport to the Emergency Department.

Upon arrival at the emergency department, the patient’s vital signs were as follows: temperature 38. 2 C, (100. 7F); heart rate, 126 beats per minute; respirations, 26 breaths per minute (ventilator rate of 12); blood pressure, 116/63mm/Hg; and oxygen saturation, 96%. The patient’s boyfriend reported that the woman had a history of depression and multiple suicide attempts. Physical assessment revealed erythematous (red), dry, warm skin and mucous membranes were pink and dry. Pupils were dilated and did not

After stabilization in the emergency department, the patient was transferred to the intensive care unit. Several days later, she was discharged from the hospital to a mental health facility. “ Discussion: Although the body contains receptor sites for histamine 1, 2, 3, and 4 substances that contain histamine 1 receptor antagonists are responsible for the majority of antihistamines poisonings. In 2003, Poison Control Centers in the United States reported more than 28, 000 exposures to diphenhydramine. Approximately 11, 000 exposed patients presented to an emergency department.

From 1985 through 2002, Poison Control Centers attributed 48 fatalities to diphenhydramine ingestion. Histamine receptor sites play a wide variety of roles in the body including, but not limited to bronchial constriction, increased vascular permeability, peripheral vasodilatation, gastric acid secretion, arousal, pituitary, hormone secretion, and thermoregulation. First-generation H1 antagonists (eg. diphenhydramine, promethazine, and hydroxyzine) also block muscarinic receptor sites, disrupting cortical neuro-transmission, which leads to sedation and seizure activity.

Second-generation H1 antagonist (eg. loratadine, cetirizine, and fexofenadine), also known as non-sedating antihistamines , have far fewer effects on the central nervous system (CNS) and produce fewer anticholinergic symptoms. Antihistamines also block fast sodium channels, which can result in cardiac conduction problems including sinus tachycardia, ventricular tachycardia, and torsades de pointes. When taken orally, diphenhydramine’s onset of action begins in 15-45 minutes. Peak effect occurs in 1 to 4 hours, and diphenhydramine’s half life is 2 to 7 hours.

When ingested in larger doses, such as in the case study patient’s case, anticholinergic syndrome can develop. The classic signs and symptoms of anticholinergic syndrome can be recalled by the mneumonic “ red as a beet, dry as a bone, blind as a bat, mad as a hatter, and hotter than Hades. ” This paints a picture of a patient of a patient with dry mucous membranes and hot, dry, flushed skin caused by inhibition of salivary and sweat glands. Redness, especially of the face, is produced by peripheral vasodilation. Increases in body temperature result from an inability to sweat combined with impaired CNS thermoregulation.

Agitation is associated with altered CNS regulation, and visual disturbances occur when the pupils are significantly dilated and lose their ability to accommodate to light. Other common signs of anticholinergic syndrome include paralytic ileus, sinus tachycardia, and urinary retention. Such as the patient in this case. ” (Alvin D. Jeffery, 2008). Activated charcoal administration is the initial treatment for many oral drug overdoses, including diphenhydramine. However, in this case, the patient already exhibited signs of paralytic ileus, which is a contraindication to activated charcoal therapy.

Treatment for antihistamine overdose and anticholinergic syndrome is largely aimed at correcting the anticholinergic response associated with the medication. According to (Alvin D. Jeffery, 2008), to increase acetylcholine availability and prolong acetylconline effects at the receptor sites, physostigmine (an acetylcholine inhibitor) is administered. The usual dose is 0. 5 to 2mg given intravenously over 1 to 3 minutes. The onset of action is within 5 minutes with effects lasting approximately 1 hour.

As more acetylcholine becomes available to the body, an increase in parasympathetic acivity is seen. Therefore, the emergency nurse must be aware of hypotension, bradycardia, and excessive salivation. These adverse effects can be treated easily with intravenous atrophine. ” Because of the ptentially fatal cardiac adverse effects of physostigimine, its use should be limited to situation in which supportive therapy is not enough such as in the with increasing agitation, altered mental status, refractory seizures, or narrow complex supraventricular dysrhythmias.

Gasstric emptying, although not beneficial if initiated more than 1 hour after ingestion, was performed in this patient’s case because she presented within the appropriate time frame. The intravenous sodium bicarbonate was administered not only to reverse the patent’s metabolic acidosis but also because if effectively treats QRS complex widening and quinidine –like cardiac conduction delays associated with antihistamine toxicity. The isotonic intravenous fluid solution was administered to compensate for the increased vascular permeability associated with the cholinergic properties of physostigimine.