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Scientific Report

Accidental acetamiprid poisoning in a buffalo

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Summary

Acetamiprid is a first generation systemic neonicotinoid insecticide, routinely used for crop protection against sucking type insects. It is likely to be of low toxicity in mammals but severe poisoning may occur if ingested in large amount. A case of buffalo with accidental ingestion of acetamiprid was presented with severe gastrointestinal symptoms and respiratory distress. The patient was managed successfully with symptomatic and supportive treatment. As far as the present report is concerned, it is the first report of acute acetamiprid poisoning in buffalo from India. From this report it is concluded that awareness programs about safe use of pesticides should be implemented.

Key words: Acetamiprid, Buffalo, Neonicotinoid

Introduction

India is an agriculture-based country with a large rural population (60-80%), where pesticides are freely available and is used extensively. Unfortunately, low literacy, poor regulatory frameworks and availability of agrochemicals at very cheap prices have amplified their use. Therefore, agrochemical poisoning is a leading cause of fatalities in India. Among various pesticides, organophosphates (OP) are most commonly used but due to their high toxicity, new compounds with high potency and selective toxicity are being developed continuously (Kumar *et al*., 2013). The neonicotinoids are highly potent insecticides commercially introduced in the 1990s and are presently one of the best-selling insecticides (Chiyozo, 2008).

Neonicotinoids are systemic insecticides used for crop protection and flea control (JMPR, 2001; Tomizawa and Casida, 2005). Because of their worldwide use, cases of human exposures are reported (Mohamed *et al*., 2009; Phua *et al*., 2009; Imamura *et al*., 2010; Iyyadurai et *al*., 2010; Yeh *et al*., 2010; Kumar *et al*., 2013; Lin *et al*., 2013), but to our knowledge, there is no such reported case of acetamiprid poisoning in animals till date. Insecticides within this class include acetamiprid, clothianidin, dinotefuran, imidacloprid, nitenpyram, thiacloprid and thiamethoxam. These are highly selective for nicotinic acetylcholine receptors (nAChRs) in insects as compared to vertebrates (Tomizawa and Casida, 2005). They act as agonists for nAChRs (Tomizawa and Casida, 2003, 2005), and induce neuromuscular paralysis and ultimately death (Nauen and Denholm, 2005; Tomizawa and Casida, 2005). The relatively low risk for vertebrates and the environment; the high specificity for insects as well as versatility in application methods have made neonicotinoids popular insecticides in recent years (Nauen and Denholm, 2005; Tomizawa and Casida, 2005; Elbert *et al*., 2008). With a good safety profile, their use is increasing globally (Tomizawa and Casida, 2005). Acetamiprid along with imidacloprid accounts for 25% of the global insecticide market (Swenson and Casida, 2013). Here, a case report of buffalo with acute acetamiprid poisoning is presented.

Case presentation

The present paper reports the occurrence of acetamiprid poisoning in Udgir (District Latur, Maharashtra), India. A Marathwadi buffalo 2.5 years old, weighing 298 kg was presented to Outpatient Department of Teaching Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Udgir, 2 h after accidental ingestion of approximately 100 g of acetamiprid (Rapid®). On admission, patient had profuse salivation, severe tympany, convulsions, absence of rumination, closed eyes and animal was in lateral recumbency (Fig. 1). The physical examination revealed rectal temperature, 97.4°F; heart rate, 98/min and respiratory rate, 12/min. Cardiac sounds were feeble and reticular sounds were not audible on auscultation. Dyspnoea, stranguria were present, palpebral and corneal reflexes were weak and ocular mucosa was congested. Urine was yellowish and faeces were diarrheic. Complete blood count showed WBC, 16.23 × 10^9/L;

absolute lymphocyte count, 7.74 × 10^9/L; absolute granulocyte count, 8.15 × 10^9/L; Lymphocyte, 47.7%; granulocytes, 50.2%; Hemoglobin, 9.2 gm/dl; RBC, 6.57 × 10^{12}/L; Hematocrit, 25.55%; Platelet count, 202 × 10^9/L; MCV, 39 fl; MCH, 14 pg, and MCHC, 35.9 gm/dl. The pH of urine was 6.5, and urine was positive for glucose and negative for ketone bodies qualitatively. Serum biochemistry revealed total bilirubin, 1.19 mg/dl; direct bilirubin, 0.58 mg/dl; indirect bilirubin, 0.61 mg/dl; creatinine, 2.9 mg/dl; urea nitrogen, 13.92 mg/dl, and glucose, 118 mg/dl.

Fig. 1: Poisoned buffalo admitted to Teaching Veterinary Clinical Complex

Normal saline (4 L), ringer lactate (4 L), Dexamethasone (Dexona®) @ 0.05 mg/kg body weight (BW) were given slowly intravenously to the patient immediately. Along with these, multivitamin (alkoplex®) 10 ml, Flunixin meglumine (Flunimeg®) @ 2.2 mg/kg BW and Pheneramine maleate (Avilin®) @ 0.5 mg/kg BW were given intramuscularly. Convulsions disappear after 18 h of fluid therapy. Dexamethasone was discontinued and rest of the treatment was given for the next 2 days. On the 2nd day animal was anorectic; water intake, rumination and defecation were absent. Rectal temperature was 99.8°F, heart rate and respiratory rate was 68/min and 18/min, respectively. Ocular mucosa was congested, tympany was reduced, but stranguria was still present and animal was not able to walk. On the 3rd day, the patient was showing improvement and rectal temperature was 101.4°F; heart rate, 58/min; respiration rate 22/min. Stranguria reduced, intermittent rumination was observed, animal was able to walk and faeces was normal in consistency. The buffalo was discharged after 3 days of follow up (Fig. 2).

Fig. 2: Improvement in buffalo condition on the third day of treatment

Discussion

Acetamiprid is an agonist of nAChRs, it induces neuromuscular paralysis and ultimately death (Tamura *et al*., 2002; Mohamed *et al*., 2009; Phua *et al*., 2009). It is expected to be safe for mammals, however toxicity can occur through inhalation, ingestion (as in present case) or by dermal contact. Sometimes toxicity may lead to serious conditions that require additional supportive care. There is no report describing acetamiprid toxicity in livestock. This could be the first such report. The symptoms of acute poisoning are similar to those of acute nicotine poisoning except corrosive injuries to gastrointestinal tract (GI) (Chao and Casida, 1997). Respiratory, cardiovascular and certain neurological symptoms *viz* dyspnoea/apnoea, coma, tachycardia, hypotension, mydriasis and bradycardia are warning signs of severe neonicotinoid intoxication (Lin *et al*., 2013).

The symptoms we observed in this study were similar to acute OP and carbamate poisonings *viz*. convulsions, hypotension, diarrhea, hypersalivation and muscle weakness. Therefore, more often atropine and oximes may be administered inadvertently as an antidote but it is still uncertain whether these drugs are effective or may worsen the outcome of neonicotinoid poisoning (Phua *et al*., 2009). However, the main muscarinic signs of acute OP poisoning were not observed. The intoxicated buffalo had tachycardia, hypothermia, stranguria and dyspnoea. The human cases of intoxicated acetamiprid poisoning had nausea, vomiting, symptoms of respiratory failure, tachycardia, hypotension, dry mouth, muscle weakness, hypothermia, and convulsions (Imamura *et al*., 2010). Death after severe intoxication of neonicotinoid has been reported by various authors (Huang *et al*., 2006; Agarwal and Srinivas, 2007; Shadnia and Moghaddam, 2008; Todani *et al*., 2008; Karatas, 2009; Mohamed *et al*., 2009; Panigrahi *et al*., 2009; Phua *et al*., 2009; Imamura *et al*., 2010; Iyyadurai et *al*., 2010; Yeh *et al*., 2010).

Neurological depression decreases airway protection and cardiac depression further aggravates the load of respiration. Besides this, corrosive injury to upper GI tract induces mucosal oedema of the airway, and the resultant inflammatory process precipitates pyrexia and hypotension (Gumaste and Dave, 1992).

The haemato-biochemical parameters revealed leucocytosis, increased level of total bilirubin, direct and indirect bilirubin, creatinine and glucose. The diarrhea and leucocytosis might be due to corrosive injury to GI mucosa. Acetamiprid is extensively absorbed after ingestion reaching the highest concentrations in the adrenal gland, liver and kidney in rats (JMPR, 2011). This might be the reason for increased level of bilirubin and creatinine in the presented case. The high serum

glucose level was probably because of physiological stress. Gastric lavage and activated charcoal should be avoided whenever corrosive injuries to GI mucosa are anticipated. Supportive management is adequate for all neonicotinoid poisoned patients.

Acetamiprid is generally less toxic to mammals causing mild symptoms. However, all the precautions must be taken during its handling. In the case of acute toxicity; respiratory failure and reduced level of consciousness are the most serious but uncommon complications. Care should be taken so that these may not get confused with/mistaken as an OP compound. The clinical consequences of acetamiprid poisoning are not very well described. Therefore, such information is valuable for clinicians, regulatory authorities and the public at large. Furthermore, clinical outcomes depend on early recognition and aggressive management. Awareness programs about safe use of pesticides should be implemented for farmers to minimize the accidental pesticide exposure. All precautions and safety recommendations given on the container should be carefully followed.

Conflict of interest

No potential conflict of interest was reported by the authors.

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