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The Study of the Clinical Profile and Laboratory Parameters of Acute Neonicotinoid Compound Poisoning at a Rural Tertiary Care Public Hospital in Central India

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Abstract

Context: Pesticide exposures are common health issues in India. Traditionally used pesticides like organophosphates are associated with higher morbidity and mortality. Neonicotinoids are newer class of effective and safer insecticides. However, literature of human exposures is very limited. *Aims:* To study clinical profile, laboratory features and factors associated with mortality after acute human neonicotinoid exposures. *Settings and Design:* This retrospective observational study was performed at department of general medicine in rural tertiary care public hospital. *Methods and Material:* Necessary data of admitted eligible cases of acute neonicotinoid poisoning during five year period of January 2012 to December 2016 were retrieved from medical record section and were analysed. *Statistical Analysis:* Statistical analyses were performed by using Graph pad prism 5. The incidence of Clinical findings, 95% confidence interval, relative risk, and baseline characteristics of patients were calculated by Wilcoxon rank sum test and chi square test. Statistical significance was established at $p < 0.05$ and RR values were considered statistically significant if 95% of CI excluded 1%. *Results:* A total of 141 cases were analyzed. Most exposures involved oral intentional consumptions of Imidacloprid. Clinical manifestations of acute neonicotinoids exposures involved variety of body systems. Severe/fatal cases had higher proportion of respiratory, neurological and cardiovascular manifestations and variety of laboratory and ECG finding. Although most exposures were asymptomatic or non-severe poisoning, 26 cases had severe poisoning with five deaths. *Conclusions:* Even though considered as relatively safer insecticides, large intentional consumption can lead to severe poisoning and even death. Supportive treatment is usually sufficient and severe poisoning needs intensive case.

Keywords: Imidacloprid; Insecticide; Neonicotinoid; Poisoning.

Introduction

Acute pesticide poisonings are among common healthcare issues in India, particularly in settings of low education and poor regulatory frameworks. Among pesticides, highly toxic organophosphates are commonly used and are associated with high morbidity and preventable mortality [1]. Neonicotinoids are newer insecticides that are effective for crop protection, flea control in agricultural and domestic settings [2]. They act on postsynaptic nicotinic acetylcholine receptors (nAChRs) by displacing acetylcholine [2]. Because of relative specificity for target insects, lower risk for non-target organisms, versatility in application and no cross-resistance to other insecticides, they are becoming popular in recent years [2-4]. These are classified as ''moderately hazardous" (Class II WHO; toxicity category II EPA) [5,6]. There are reports which describe

cardiac, neurological, pulmonary, renal, multiorgan failure and death as their exposures [7-11]. Despite increasing use, literature about acute human poisonings is limited to few studies & case reports [12-14]. So, we planned study with objective to study different clinical features, laboratory changes and factors associated with mortality with these neonicotinoids. We hope, this information will help in risk assessment and clinical management of acute neonicotinoids exposures and also help concerned regulatory agencies to decide policies regarding their safe use.

Subjects and Methods

This retrospective observational study was carried out at Rural Tertiary Care Public Hospital in Marathwada region of Maharashtra, India. All patients of neonicotinoid poisonings, who were admitted to our hospital during period of January 2012 to December 2016, were identified from hospital records and were considered for study. Study was approved by institutional ethics committee of our hospital. Patient who had history of exposure to neonicotinoids like Imidacloprid, Acetamiprid, Clothianidin, Thiacloprid, Dinotefuran, Nitenpyram or Thiamethoxam and who was admitted to hospital was defined as neonicotinoid poisoning. Cases that consumed other insecticide, discharged against medical advice, age less than 12 yrs and with incomplete records were excluded from study. The records of all patients of neonicotinoid poisonings admitted during study period were obtained from records section of our hospital. Cases which fulfilled inclusion & exclusion criteria were selected and data regarding demographic profile, clinical features, details of compound exposed, elapsed time, laboratory parameters, complications, treatment received and outcomes were recorded. Clinical features were grouped according to various organ systems. Gastrointestinal effects were defined by symptoms like nausea, vomiting, abdominal pain, gastroesophageal bleeding & odynophagia, central nervous system effects were dizziness, drowsiness, seizures, mydriasis and unconsciousness. Cardiovascular effects included palpitations, chest pain & hypotension. Respiratory effects were sore throat, breathlessness, respiratory failure & aspiration pneumonia. Clinical presentations were classified as "non-severe" and "severe" as per American Association of Poison Control Center data collection system [14]. Patients with manifestations that were potentially life-threatening or caused death (e.g. seizures, respiratory failure, ventricular tachycardia, hypotension, cardiac or respiratory arrest, haematemesis) were categorized as severe. All other presentations were categorized as non-severe. Data collected from medical records were compiled using excel sheet and analysed with Graph pad prism 5. Descriptive statistical method was used to describe frequencies and percentages for categorical data. Statistical analysis was performed to evaluate distribution of baseline characteristics and clinical features between male and female cases. To assess parameters associated with severity, we compared demographic, clinical and laboratory findings between severe/fatal and non-severe cases by Wilcoxon rank sum test for continuous variables and either chi-square test or Fisher's exact test for categorical variables. Death rates for various insecticides were evaluated for statistical significance by calculating ratio of rate for neonicotinoids to rate for other insecticides (rate ratio, RR) and 95% confidence interval (CI) by Newcombe–Wilson method without continuity correction. RRs were considered statistically significant if 95% confidence interval excluded 1.00. Elsewhere, p-value of less than 0.05 was considered statistically significant.

Results

Total 141cases of acute neonicotinoid exposures, which qualified inclusion and exclusion criteria, were studied. Among the cases, males were 105 (74.46%) and females were 36 (25.54%) (Table 1). During year 2012-2013, there were ten and 18 cases respectively. Number of cases increased after 2014 and there were 31, 34 and 48 cases for these respective years. Median age of cases was 41 years for males (13–64 year) and 29 years for females (12-77 year). Exposure involved oral ingestion in 89 (63.12%) cases, 14 (9.93%) had inhalational contact, nine (6.38%) had dermal and 29 (20.57%) had mixed exposures (Table 1). Reason for exposure was intentional consumption in 83 (58.87%) cases while remaining 58 (41.13%) had accidental exposures and 46 (43.81%) had alcohol co-ingestion, all males. Out of 141 cases, 53 (37.59%) were asymptomatic, 62 (43.97%) had symptomatic & non-severe poisoning while 26 (18.44%) had severe/fatal poisoning with five (3.55%) deaths. There was no significant difference in male and females for year of exposure, route of exposure, reason for exposure and severity of poisoning. However, males had significantly higher age and number of alcohol co-ingestion than females. Most commonly observed neonicotinoid compound was Imidacloprid, reported

Table 1: Distribution of baseline characteristics of the cases with acute neonicotinoid exposures in the study

Characteristics	Males N (%) **105** (74.46)	Females N (%) **36 (25.54)**	P-value
Calendar year of poisoning			
2012	7 (6.66)	3 (8.33)	
2013	15 (14.28)	3 (8.33)	
2014	24 (22.85)	7 (19.44)	0.854
2015	25 (23.81)	9 (25.00)	
2016	34 (32.38)	14 (38.89)	
Age (median and range, years)	41 (13–64)	29 (12-77)	0.013
Reason of exposure			
Intentional	62 (59.04)	21 (58.33)	0.940
Accidental	43 (40.95)	15 (41.67)	
Route of exposure			
Oral	67 (63.81)	22 (61.11)	
Inhalational	10 (9.52)	4 (11.11)	
Dermal	7 (6.67)	2 (5.55)	0.975
Non-oral Mixed	21 (20.00)	8 (22.22)	
Simultaneous alcohol intake.	46 (43.81)	0 (0)	0.0001
Severity			
Asymptomatic	34 (32.38)	19 (52.78)	
Symptomatic & non-severe	49 (46.67)	13 (36.11)	
Symptomatic & severe	18 (17.14)	3 (8.33)	
Death	4 (3.81)	1 (2.78)	0.189

Table 2: Distribution of the individual compounds and various clinical features among all acute neonicotinoid exposures in the study

Neonicotinoid compound		Number (n)	Percentage (%)
	Imidacloprid	108	76.60
	Acetamiprid	9	6.38
	Thiamethoxam	9	6.38
	Clothianidin	6	4.26
	Dinotefuran	4	2.83
	Nitenpyram	3	2.12
	Thiacloprid	2	1.42
	total	141	100
Clinical features		**Number (n)**	**Percentage (%)**
	Nausea	77	54.61
	Vomiting	59	41.84
	Sore throat	42	29.79
	Abdominal pain	42	29.79
	Chest pain	24	17.02
	Dizziness	23	16.31
	Odynophagia	22	15.60
	Dermal irritation	19	13.48
	Ocular irritation	17	12.06
	Breathlessness	17	12.06
	Drowsiness	15	10.64
	Respiratory failure	15	10.64
	Palpitations	13	9.22
	Unconsciousness	9	6.38
	Gastroesophageal bleeding	9	6.38
	Hypotension	8	5.67
	Aspiration pneumonia	6	4.25
	Seizures	3	2.13
	Mydriasis	2	1.42
	Rhabdomyolysis	1	0.71

in 108 (76.60%) cases, followed by Acetamiprid & Thiamethoxam, each nine cases (6.38%) and exposures with Clothianidin (4.26%), Dinotefuran (2.83%), Nitenpyram (2.12%) & Thiacloprid (1.42%) were less commonly reported (Table 2). Among cases, variety of clinical features involving gastrointestinal, cardiovascular, respiratory, nervous, renal system and other local effects involving eyes and skin were observed (Table 2). To find out various factors associated with development of severe/fatal poisoning, we classified 141 cases into severe/fatal poisoning and non-severe poisoning (Table 3). All of 26 cases with severe/fatal poisoning had exposure to imidacloprid. Out of these 26 cases, 25 had oral ingestion (96.15%) while one had mixed inhalational & dermal exposure (55.65%) and this difference was significant. Other parameters like older age (39 vs 32), delay for treatment (2.5 vs 1.5), systemic manifestations of Gastrointestinal system (88.46% vs 35.06%), Cardiovascular system (76.92% vs 7.83%), nervous system (73.08 % vs 15.65%), Respiratory system (69.23% vs 28.70%), abnormal laboratory findings (50.00% vs 0%) and alcohol use (76.92% vs 22.61%) were significantly more common in severe/fatal poisoning (Table 3). Varieties of ECG findings were noted in study cases. (Table 4). The ECG was abnormal in 21 (80.77%) cases of severe/fatal poisoning while three (2.60%) cases of non-severe exposures had abnormal ECG and this difference was significant. Table 5 compares fatal outcome rate of Neonicotinoids to other insecticides poisonings reported to our hospital. Majority of patients (96.45%) recovered and were discharged; however, five cases died. The proportion of deaths for neonicotinoids was 3.55%, which is significantly lower than Organophosphates (15.56%), Organochlorides/Carbamates (17.18%) and Herbicides (10.16%), which are commonly used

Table 3: Comparison of demographic and clinical characteristics between patients with severe/fatal and non-severe neonicotinoid insecticide exposures in the study

Characteristics	Severe/fatal poisoning n=26 (%)	Non-severe poisoning n=115 (%)	p-value
Mean Age (years)	39 (19-54)	32 (12-77)	0.049
Delay for medical treatment (h)	2.5 (0.5-9)	1.5 (0.25-9.5)	0.034
Gastrointestinal effects	23 (88.46)	65 (35.06)	0.0002
Nausea	21 (80.77)	56 (48.70)	0.0002
Vomiting	20 (76.92)	39 (33.91)	0.0001
Abdominal pain	10 (38.46)	32 (27.82)	0.284
Gastroesophageal bleeding	9 (34.61)	0 (0)	0.0001
Odynophagia	5 (19.23)	17 (14.78)	0.575
Cardiovascular effects	20 (76.92)	9 (7.83)	0.0001
Palpitations	9 (34.61)	4 (3.48)	0.0001
Chest pain	17 (65.38)	7 (6.09)	0.0001
hypotension	8 (30.77)	0 (0)	0.0001
Respiratory tract effects	18 (69.23)	33 (28.70)	0.0001
Sore throat	9 (34.62)	33 (28.70)	0.548
Breathlessness	16 (61.54)	1 (0.87)	0.0001
Respiratory Failure (type 1 & 2)	15 (57.69)	0 (0)	0.0001
Aspiration/ ventilator associated pneumonia	6 (23.07)	0 (0)	0.0001
Central nervous system effects	19 (73.08)	18 (15.65)	0.0001
Dizziness	5 (19.23)	18 (15.65)	0.652
Drowsiness	15 (57.69)	0 (0)	0.0001
Seizures	3 (11.54)	0 (0)	0.0002
Unconsciousness	9 (34.61)	0 (0)	0.0001
Mydriasis	2 (7.69)	0 (0)	0.0027
Other effects	6 (23.07)	23 (20.00)	0.726
Ocular irritation	3 (11.54)	14 (12.17)	0.928
Dermal irritation	4 (15.38)	15 (13.04)	0.748
Abnormal ECG findings	21 (80.77)	3 (2.60)	0.0001
Laboratory findings	13 (50.00)	0 (0)	
Hypokalemia	8 (30.77)	0 (0)	0.0001
Renal failure	2 (7.69)	0 (0)	0.0027
Abnormal liver enzymes	5 (19.23)	0 (0)	0.0001
Metabolic acidosis	3 (11.54)	0 (0)	0.0002
Alcohol intake	20 (76.92)	26 (22.61)	0.0001
Route of exposure			
oral	25 (96.15)	64 (55.65)	0.0001
Non-oral	1 (3.85)	51 (44.35)	0.0001

Table 4: Various ECG findings noted and Treatment modality used among the patients with acute neonicotinoid exposures in the study

ECG finding		Number (n)	Percentage (%)
	Normal ECG	86	60.99
	Sinus tachycardia	31	21.99
	ST-T Changes	9	6.38
	Prolonged QTc interval	5	3.55
	Atrial fibrillation	4	2.84
	Sinus Bradycardia	3	2.13
	Ventricular tachycardia	1	0.71
	Ventricular ectopic	2	1.42
Treatment modality used		**Number (n)**	**Percentage (%)**
	Decontamination	129	91.49
	H2 Antihistamines Or Proton Pump Inhibitors Or Antiemetics	65	46.10
	IV Fluids	49	34.75
	Bronchodilators	21	14.89
	Oxygen	19	13.48
	Antibiotics	14	9.93
	Ventilatory Support	14	9.93
	Anti-Convulsant/ Sedative Drugs	10	7.09
	Potassium Chloride	8	5.67
	Inotropes	6	4.26
	Atropine & Pralidoxime	5	3.55
	Anti-Arrhythmic Drugs	4	2.84
	Blood Transfusion	3	2.13
	DC Shock	3	2.13
	No treatment	12	8.51

Table 5: Death rates for neonicotinoid and other insecticides exposures during the study period

Insecticide	Total	Death (N)	Death rate (%)	RR	95% CI
Neonicotinoids	141	5	3.55	-	-
Organophosphates	842	131	15.56	0.25	0.10-0.61
organochlorines/ carbamates	390	67	17.18	0.26	0.11-0.61
Pyrethroids	423	6	1.42	1.8	0.93-3.52
Herbicides	305	31	10.16	0.43	0.19-0.98

insecticides in our region. Treatments modalities used were recorded and treatment received was all symptomatic and supportive (Table 5).

Discussion

In this study, we studied cases of acute neonicotinoid poisoning for period of January 2012 to December 2016. There was gradual increase in number of cases from 2012 to 2016 with most cases occurring in year 2016. This observation suggests that these compounds are becoming popular and are being used increasingly in recent years and number of acute human exposures might increase in future [2,4]. This observation is similar to earlier studies by Phua et al and Forrester who also described increasing trend number of cases [5,12]. Imidacloprid (76.60%) was most commonly reported, followed by Acetamiprid & Thiamethoxam (each 6.38%) while Thiacloprid (1.42%) was least common. Retrospective analyses of poison control center data by Forrester (76.5%) & Phua et al (90%) and prospective observational cohort study by Mohamed et al, reported similar observation regarding Imidacloprid to be the most commonly exposed neonicotinoid [5,6,12]. Lin et al, in review concluded that Imidacloprid was major poison among Neonicotinoids, which constituted 94% of intoxication events [15]. We conclude that widespread use and easy availability were reasons that most patients were exposed to Imidacloprid [16]. We noted more number of intentional oral consumptions than accidental inhalational and/or dermal exposures. This is in contrast to study by Forrester where majority of exposures were unintentional and below 2% were

intentional [12]. However, study by Phua et al reported 69% cases of suicides and study by Mohamed et al reported 89.71% cases of intentional poisoning [5,6]. Nearly all reported case studies with both fatal and favourable outcomes, reported intentional consumptions as cause of exposure [15]. These differences might be due to different study criteria, poison center operations, or types of reported exposures. In our study, majority of cases were males (74.46%) while rest were females. These observations were similar to Taiwan study where males were majority while UK study had evenly distributed male and females [5,17]. However, in study by Forrester, female were more than males [12]. These differences can be due to differences in cases reported to poison centres and various socio-demographic background. Majority of our cases (62.41%) were symptomatic while 37.59% cases were asymptomatic. Neonicotinoids have agonistic action at nAChRs; their toxic effects, therefore, may be similar to nicotine. Activation of nicotinic receptors by nicotine classically shows a biphasic clinical pattern with initial stimulation followed by inhibition [2,5,18]. The most commonly reported clinical features in our study were gastrointestinal with variable degree of respiratory, neurological, cardiovascular, ocular, dermal & other symptoms and were consistent with available literature of acute exposures [5-12,15,19-21]. In our study, severe/fatal poisoning were observed in 26 cases (18.44%) with five (3.55%) deaths. In study by Forrester, having 1,142 exposures with more than 98% unintentional exposures, only 32 (2.9%) resulted in serious outcomes with no deaths [12]. Another study by Phua et al with total 46 exposures, reported ten cases (21.74%) of severe poisoning with two (4.35%) death [5]. Mohammad et al studied 68 cases of intentional Imidacloprid poisoning and reported severe features requiring intensive care in two cases (2.94%) and no deaths [6]. Double-blind crossover study of 19 planters by Elfman reported no adverse effects with Imidacloprid [22]. Lin et al observed severe manifestations in 22 cases and six deaths from total 66 cases having detailed clinical records [15]. The differences in proportion of cases having severe/fatal outcome in these studies could be due to differences in study design, types of exposures reported, differences in exposure assessment methods and different definitions used to define severe poisoning. The definition used in our study and by Phua et al was similar and proportion of severe cases (18.44% vs 21.74%) and fatal outcome (3.55% vs 4.35%) were almost similar [5]. Moreover, fatal outcome rate for neonicotinoids (3.55%) was significantly lower than carbamate/organochlorines (17.18%), organophosphates (15.56%) and herbicides (10.16%), however, it was marginally but not significantly more than pyrethroids (1.42%). This is consistent with other three poison center investigations. Study by Adams et al observed that Neonicotinoids have less serious medical outcomes than pyrethroids and carbamates [17]. In study by Phua et al, mortality for Neonicotinoids was lower than organophosphates and carbamates but was similar to Pyrethroids [5]. In study by Forrester, serious outcome rate for neonicotinoid insecticides was substantially lower than carbamates/chlorinated hydrocarbon/ organophosphates and pyrethroids [12]. Therefore, it can be proposed that acute exposures of neonicotinoids are relatively safer than other insecticides. This finding can be explained by their selective action at insect nAChRs and high water solubility reducing ability to penetrate mammalian blood–brain barrier rendering them less toxic to CNS [2,23,24]. However, it must be remembered that severe toxic effects and even death have occurred following acute neonicotinoid exposures, especially following large ingestions [5,7-8,10,11,13,15,21,25]. All cases with major severity or death in our study were exposed to Imidacloprid alone. This may be related to fact that Imidacloprid was most frequently encountered neonicotinoid in study and it is expected to be more toxic than other Neonicotinoids because of higher selectivity of other neonicotinoids [2]. However, it is worth to note that Neonicotinoids like Acetamiprid and Thiacloprid can cause severe poisoning and even death [9,13,15,21,26]. Average age of severe/fatal poisoning group was significantly higher than that of non-severe group. Phua et al and Lin et al also noted similar observations with older patients having more severe poisoning [5,14,15]. Inhalational and dermal exposures were significantly associated with non-severe poisonings and oral exposures with severe/ fatal poisonings, a finding consistent with studies from Sri Lanka, Taiwan and review by Lin et al [5,6,15]. We noted higher proportion of severe/fatal outcomes in males than females but this difference was not significant. This finding might be due to fact that study had higher proportion of males having oral ingestions. Patients with co-ingestion of alcohol had significantly higher proportion of severe poisoning and four of five cases with fatal outcome had consumed alcohol. In study by Mohamed et al, prolonged sedation and respiratory depression was noted in two patients who had co-ingestion of ethanol [6]. There are case reports of severe/fatal poisoning with co-ingestion of alcohol and Neonicotinoids. Yeh et al reported case of ingestion of alcohol with Imidacloprid and manifestations included disorientation, bradycardia,

ventricular arrhythmia, and cardiopulmonary arrest [7]. However, we could not find any study assessing role of alcohol co-ingestion in severity of neonicotinoid poisoning and thus, warrants further research. We found that different symptoms like abdominal pain, odynophagia, sore throat, dizziness, eye and skin irritation occurred equally in both severe/fatal and non-severe groups and there was no significant difference. Conversely, majority of respiratory, cardiovascular and neurological symptoms occurred more commonly in severe/fatal group and these differences were significant. Study by Phua et al & Mohamed et al noted similar observations and proposed that coma, respiratory depressions, respiratory muscle weakness, cardiac arrhythmia and aspiration pneumonia are associated with severe/fatal cases [5,6]. Lin et al in a review noted that respiratory, cardiovascular and some neurological symptoms occurred more commonly in severely intoxicated patients and meticulous observation is indicated in neonicotinoid-poisoned patients presenting with these warning signs [15]. We could study different ECG findings in cases of acute neonicotinoid exposures and noted that ECG was either normal or had sinus tachycardia in majority of cases. We observed abnormal ECG findings like ST-T changes, prolonged QTc, atrial fibrillation, sinus bradycardia, ventricular ectopic, and ventricular tachycardia in order of frequency of occurrence. Except for fatal ventricular tachycardia in one patient and atrial fibrillation in other, most of ECG changes were reversible. We could not find any literature which studied different ECG findings in acute neonicotinoid poisoning. Few case reports have noted abnormal ECG findings. Huang et al reported case of fatal ventricular fibrillation following ingestion of Imidacloprid compound which was refractory to DC shock and IV anti-arrhythmics [8]. Yeh et al reported case of fatal ventricular tachycardia following ingestion of imidacloprid and alcohol [7]. Case report by Todani et al reported atrial fibrillation lasting for 11 hours with Acetamiprid poisoning [26]. Here, we can conclude that life threatening arrhythmias do occur with neonicotinoid poisoning and can be fatal. The cause of arrhythmias can be multifactorial including activation of autonomic system with resultant coronary spasm & cardiac ischemia, hypoxia, electrolyte imbalance, direct toxic effects on myocardium and alcohol co-ingestion. There is no specific antidote for neonicotinoid poisoning in humans [2]. Treatment given to cases in our study was mainly supportive, that involved decontamination, administration of H2 antihistamines/proton pump inhibitors/antiemetic drugs, fluids, antibiotics, oxygen, bronchodilators, DC shock, anti-arrhythmics, potassium chloride, ventilatory support, blood transfusion, atropine & pralidoxime, anti-convulsants/sedatives and inotropic agents. Review of available literature demonstrated similar findings and treatment given was mainly supportive [5,6,12,13,15].

We noted use of atropine and pralidoxime in few cases where clinical features were similar to organophosphate poisoning and were misdiagnosed initially in unavailability of compound details on presentation, which later turned out to be Imidacloprid poisoning. Similarly, there are descriptions of Imidacloprid poisoning getting misdiagnosed as organophosphate poisoning due to similar manifestations and were given treatment with atropine and pralidoxime [5,9,27]. Oximes in absence of organophosphate poisoning have inhibitory effect on acetylcholinesterase activity and therefore, might increase nicotinic effects [6]. Thus, treatment with oxime in neonicotinoid poisoning might be ineffective and may be contraindicated. Mohamed et al noticed that two most seriously poisoned cases received treatment with pralidoxime [6]. Therefore, it can be said that poisoning with Neonicotinoids should be considered in differential diagnosis of patients having features suggestive of organophosphate poisoning and use of pralidoxime should be avoided in these cases.

Limitations

Being a hospital based retrospective study of admitted cases of only neonicotinoids, out of hospital deaths, combinations with other insecticides and cases not admitted, were likely to be missed. Although, we accessed key data of most patients, accurate information on exact timing, elapsed time before treatment and minor clinical information may be incomplete. We could not measure exposed quantity, solvent present in preparations and blood levels of insecticides. In our study, majority of exposures were due to imidacloprid, so evaluation may miss differences in clinical presentations for other neonicotinoids due to their limited number.

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Key Messages

Neonicotinoids, being used increasingly, their human exposures tend to increase in future. Though, they have specific mode of action on insects and considered less toxic to humans, can cause death, especially after intentional Imidacloprid consumptions. Treatment is Supportive and severe poisoning with respiratory, nervous & cardiovascular manifestations needs intensive case.

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