Original Article

Management of OPC and Carbamate Poisoning in Intensive Care Unit of Enam Medical College & Hospital, Savar, Dhaka

Easnem Khanum[1], Md Ashraful Islam[2], Mohammad Salim[3], S. M. Rafiqul Islam[4], Md. Rezaul Haque PK[5]

Received: February 3, 2018 Accepted: August 25, 2018
doi: http://dx.doi.org/10.3329/jemc.v8i3.38364

Abstract

***Background**: Bangladesh is an agriculture-dependent country. Organophosphorus insecticides are widely used for the better outcome of cultivation. It is used for suicidal purpose due to its easy availability all over the country. Worldwide, acute pesticide poisoning is causing major health problems with high mortality in developing countries. **Objective**: The objective of our study was to establish that early management of acute poisoning with organophosphorus and carbamate in ICU along with ventilatory support in severe respiratory distress reduces the mortality rate. **Materials and Methods**: This study was conducted in the Department of Anesthesiology & ICU in Enam Medical College & Hospital, Savar, Dhaka from January 2013 to December 2015. Total 84 patients with acute poisoning cases were selected. The diagnosis was confirmed by the history of ingestion of insecticides, observing clinical signs and symptoms and presence of foul smelling of poisonous agent. Management included supportive care, intubation, artificial ventilation in selective cases, administration of antidotes as loading and maintenance dose for atropinization with atropine and pralidoxime, antibiotics, anti-ulcerant, anticonvulsant, inotropic support (in severe hypotension) along with other symptomatic treatment. After stabilization, decontamination was started with removal of contaminated clothes, thorough wash with soap and water, irrigation of eyes with water and normal saline. Gastric lavage was given within 2 to 3 hours of ingestion of poison. **Results**: Acute poisoning was observed more in male (60.71%) than in female (39.29%) and in age group of 21–40 years (60.71%). Suicidal attempt was present in 97.61% cases and causes of suicidal poisoning were familial disharmony (76.19%), financial loss (14.28%) and failure in examination (9.52%). Organophosphate group poison ingestion was in 77.38% cases and carbamate group poison in 21.43% and both agents in 1.19% cases. Ventilatory support was given in 48.80% cases and 78.05% patients were successfully extubated from mechanical ventilator. Mean duration of ventilatory support was 2–14 days. Out of 84 patients, 75 (89.28%) survived and 9 (10.72%) patients expired. Fifty two (61.91%) patients were discharged within 4–6 days, 16 (19.04%) within 2–3 days, and 7 (8.33%) patients were discharged within 7–14 days. **Conclusion**: Early ICU admission and appropriate management of patients after ingestion of poisonous agent results in reduced morbidity and mortality.*

***Keywords**: Organophosphates; Carbamate; Ventilatory support*

J Enam Med Col 2018; 8(3): 144−152

1. *Associate Professor, Department of Anesthesiology & ICU, Enam Medical College & Hospital, Savar, Dhaka*
2. *Associate Professor, Department of Anesthesiology & ICU, Enam Medical College & Hospital, Savar, Dhaka*
3. *Medical Officer, Department of Anesthesiology & ICU, Enam Medical College & Hospital, Savar, Dhaka*
4. *Professor, Department of Anesthesiology & ICU, Enam Medical College & Hospital, Savar, Dhaka*
5. *Professor, Department of Anesthesiology & ICU, Enam Medical College & Hospital, Savar, Dhaka*

***Correspondence** Easnem Khanum, E-mail: easnem@gmail.com*

Introduction

Bangladesh is an agriculture-dependent country. The main food of the country is rice and 75% of the total agricultural land is used only for rice crop cultivation.[1] Pesticides are usually used in agriculture, industries and for domestic purposes[2]; but these are extensively used in the agricultural field for the better outcome of cultivation.[3] An ideal pesticide should have no harmful effect on human and environment, but perfect pesticide has not yet been found.[4] Among the four classes of organic insecticides (organophosphate, organocarbamate, organochlorides and organic pyrathroids), organophosphate and carbamates are the most common poisonous agents used for suicidal purpose due to their easy availability all over the country as over the counter drug (OCT).[3] Humans may be exposed to pesticides in a variety of ways, at different dose levels and for varying periods of time.[5]

Around 21 pesticides were banned by the Pesticide Regulation Committee of Bangladesh Government in between 1996 to 2007, resulting in a decrease in the incidence of suicidal death due to pesticide in the post-ban years (2001–2014) than in pre-ban years (1996–2000).[1] Though more toxic pesticides have been banned, still self-poisoning by pesticides is a serious health problem in our country, which is responsible for about 40% of total poisoning cases admitted to hospital and 8–10% of overall mortality in medical wards.[6] According to government statistics, it is the second most common cause of hospitalization and ninth most common cause of death and about 150,000 deaths occur each year.[1]

Worldwide cases of acute pesticide poisoning account for significant morbidity and mortality, especially in developing countries.[7] Among three million of acute poisoning cases, 220000 deaths occur each year.[8] In China and Southeast Asia about 300000 deaths occur each year.[8] The annual incidence rates among agricultural workers are 18.2 per 100000 full-time workers and 7.4 per million among school children.[9] In Turkey, it also accounts as the most important cause of poisoning like other developing countries.[10]

Some individuals with pesticide poisoning suffer from mental illness. A psychosocial support program may play an important role in preventing suicidal attempt by self-induced poisoning.[11]

Now-a-days the majority of self-induced poisoning is caused by organophosphorus compounds (OPCs) and carbamates which cause a major health problem. In 2007 about 37712 metric ton of pesticides were sold in our country.[12] OPCs and carbamate poisoning are not only a public health problem but also cause a direct threat to our economic growth.

Acute pesticide poisoning usually occurs during agricultural use either by accidental exposure or by suicidal attempt, rarely as homicidal.[2,7,8] Mode of exposure varies including dermal, gastrointestinal, inhalation, and unusually through intravenous and subcutaneous routes.[13] Absorption through stomach depends on several factors, such as fullness of the stomach, the presence of pyloric stenosis, early stomach wash etc. The toxic action depends on the amount of substance taken, distribution to tissues and excretion from the body. After massive ingestion or inhalation symptoms may appear within five minutes or may be delayed up to half an hour to one hour and maximum 2–8 hours. Signs and symptoms appear when serum cholinesterase level falls to 30% of its normal activity. Sometimes the very small amount of these can kill a person.[3]

OPCs inhibit both cholinesterase and pseudocholinesterase activities irreversibly, which leads to accumulation of acetylcholine at synapses, causing overstimulation and disruption of neurotransmission in both central and peripheral nervous systems leading to development of nicotinic and muscarinic features.[14]

Signs and symptoms of organophosphate poisoning can be divided into three broad categories: (i) muscarinic effects, (ii) nicotinic effects, and (iii) central nervous system (CNS) effects.[15,16] Muscarinic features include bronchoconstriction, increased salivation, sweating, lacrimation, constriction of pupil, blurred vision, urinary incontinence, bradycardia, hypotension etc. Nicotinic features include twitching of facial muscles, eyelids, tongue and neuromuscular blockage. Central nervous system effects are restlessness, headache, tremor, drowsiness, ataxia, confusion, convulsion, respiratory depression, coma and death.

OPC poisoning causes an acute cholinergic phase which occasionally may be followed by intermediate syndrome (IM)[17] or organophosphate-induced delayed polyneuropathy (OPIDN)[17]. About 20% of

patients with OPC poisoning develop weakness which spreads rapidly from the muscles of eye to head, neck and proximal limbs. Weakness of respiratory muscles results in respiratory failure. OPIDN is a mixed sensory or motor polyneuropathy which affects long myelinated neurons resulting in muscle cramps followed by numbness and paresthesia to flaccid paralysis of the lower and subsequently the upper limbs. IM usually develops quite rapidly between 1 and 4 days after exposure. Sometimes it occurs even after resolution of acute cholinergic syndrome and may last up to 2–3 weeks. OPIDN usually develop 2–3 weeks after exposure. Recovery is often incomplete and it may persist up to 1–2 years, especially in younger patients.[17]

Death usually occurs as a result of combination of the actions in the central nervous system, increased bronchial secretion, respiratory obstruction by bronchoconstriction and paralysis of respiratory muscles.[18]

The mortality rate of OPC poisoning is high. Fatality usually occurs within 24 hours in untreated cases and within 10 days in unsuccessfully treated cases. Acute effects last for 6 to 30 hours, which disappear in 2 to 3 days but sometimes persist for two weeks.[16] Fatality is often related to delay in diagnosis or an improper management. Early diagnosis, proper management, along with ICU support, especially for respiratory care often reduce mortality.

The management of organic insecticide poisoning is a challenging proposition, particularly in the rural area, due to lack of good transport facility and well-equipped emergency medical facilities. The objective of our study was to establish that mortality is reduced by early ICU management along with ventilatory support in case of severe respiratory distress.

Materials and Methods

This observational study was conducted in the Department of Anesthesiology & Intensive Care Unit of Enam Medical College & Hospital, Savar, Dhaka from January 2013 to December 2015. During the study period eighty four patients of acute insecticide poisoning were selected. Among them 65 patients ingested OPCs, 18 carbamates and one patient ingested both OPCs and carbamates.

Diagnosis of acute poisoning cases was confirmed from the history of ingestion of insecticides, observing clinical signs and symptoms, presence of foul smelling of OPC and sometimes from the sample of poisonous substances brought by patients' attendants. In our study plasma cholinesterase levels were not done due to lack of facility in our institution.

All data were recorded in a preformed sheet and analysis was done using Microsoft Word and SPSS version 20.0. p value of <0.01 or less was considered as statistically significant.

For managing a poisoning case in a health care center, skilled and knowledgeable health care providers are necessary for resuscitation and intubation for respiratory care along with appropriate resources[19] which are only possible in an ICU set-up. Management was started from airway, breathing and circulation (ABC) supportive care.[20] For managing airway at first patient was placed in left lateral position with head down to reduce aspiration. High flow oxygen and adequate ventilation were established along with intubation followed by artificial ventilation to prevent respiratory failure along with two secured IV lines or central venous line for starting fluids and other medication. An arterial blood gas (ABG) analysis was done immediately. In patients with convulsion, IV diazepam or IM barbiturate was given for preventing further episodes of convulsion.[20]

For intubation and artificial ventilation[10,21] following guideline was followed in our study:

1. Respiratory rate (R/R) >35 breaths/minute or R/R <8 breaths/minute or apnea
2. GCS <9 out of 15
3. Arterial blood gas analysis

i) Arterial oxygen tension: <50 mm Hg in room air
ii) Arterial CO_2 tension: >50 mm Hg in the absence of metabolic alkalosis.
iii) PaO_2/FiO_2: <250 mm Hg
iv) Pulmonary arterial: alveolar O_2 gradient >350 mm Hg
v) pH: 6.93–7.26
vi) HCO_3: 10–25 mmol/L

After putting on mechanical ventilation controlled ventilation was started along with PEEP (positive end-expiratory pressure) as 4 to 5 cm H_2O and high FiO_2. Both were gradually decreased to keep SaO_2 (arterial

oxygen saturation) above 95% with 40% FiO_2. Weaning was done after stabilization of all vital signs, normal necessary laboratory investigations, correction of desaturation and appropriate atropinization. For weaning, at first ventilator mode was changed from a control mode to SIMV (synchronized intermittent mandatory ventilation), then CPAP (continuous positive airway pressure) and ultimately with a T-tube trial extubation was done very carefully.

After stabilization of the patient, decontamination was started in following ways:

i) Removal of contaminated clothes and thorough wash with soap and water

ii) Irrigation of eyes with water

iii) If patient presented within 2 to 3 hours of ingestion of poisoning, gastric lavage was given using 1:5000 potassium permanganate solution or by 0.9% normal saline.[22,3]

Pharmacological treatment was started by using a loading dose of atropine and pralidoxime. Atropine is a tertiary amine which acts as a competitive antagonist of acetylcholine at the muscarinic postsynaptic membrane and in the CNS.[20] The loading dose of atropine was 1.8 to 3 mg (3–5 vials) through intravenous route, then the dose was doubled every five minutes interval till target endpoint of atropinization (i.e., clear chest on auscultation, pulse >80 beats/minute, pupils no longer pinpoint, dry axilla and systolic BP >80 mm Hg).

After achieving atropinization maintenance dose was started with 10 to 20% of the total dose of atropine needed for the patient to be atropinized but not more than 30 mg approximately at 3–5 mg/hour. Then monitoring was done every 15 minute interval to settle the infusion rate. During the infusion period if toxicity (i.e., confusion or delirium, pyrexia, absent bowel sounds or urinary incontinence) developed, atropine infusion was stopped and symptomatic management was started. Atropine infusion was restarted at 70–80% of the previous infusion rate after disappearance of signs of toxicity. In case of loss of atropinization (e.g., bronchospasm/bradycardia) a bolus dose of atropine was given and infusion rate was increased till the signs disappeared. After stabilization of all parameters, all cases were monitored hourly till required dose of atropine decreased, then cases were monitored 2–3 hourly and continued for 2–5 days. Tapering was done over 3rd to 5th day.

Pralidoxime therapy

Current WHO guideline for pralidoxime therapy is as follows: 30 mg/kg over 10–20 minutes as loading dose followed by a continuous infusion of 8–10 mg/kg/hour until clinical recovery (after 12–24 hours atropine is no longer required or patient is extubated) or 7 days.[20] In our study, it was not possible to continue the continuous infusion as above in all cases due to high cost; rather 1 gm two times daily was used after loading dose till atropine was no longer required or up to 24 hours of extubation.

Pralidoxime is a quaternary ammonium compound with poor CNS penetration which reactivates acetylcholine by removing the phosphoryl group from OP compound and causes recovery of neuromuscular transmission.[23] Pralidoxime is not helpful in carbamate poisoning, but it does no harm if used. In our study, it was also avoided in carbamate poisoning. Besides atropine and pralidoxime, an antiulcerant and an antibiotic (3rd generation cephalosporin) were routinely used in every case along with other symptomatic treatment.

Routine investigations were done as part of management, e.g., ABG, ECG, chest radiography, complete blood count, serum electrolytes, serum creatinine, SGPT and urine R/M/E. Close monitoring of all vital parameters was done as per ICU rule. Regular consultation was done with specialists of Internal Medicine and Respiratory Medicine.

Results

In this study, among 84 patients, 51 (60.71%) were male and 33 (39.29%) were female. Acute poisoning was observed more in married subjects (55, 65.47%; male 28 and female 27) than in unmarried (29, 34.52%; male 23 and female 6). p value was found 0.000 which was significant.

There were four age groups. The common age group was found 21–40 years (51, 60.71%), the less common group is above 60 years. The age group of the patients is summarized in Table I.

Table I: Distribution of subjects according to age (N=84)

Age group in years	Number	Percentage
8–20	21	25
21–40	51	60.71
41–60	10	11.90
61–80	02	2.39

Regarding occupation, 33 (39.28%) cases were housewives followed by students 18 (21.43%), service holders 09 (10.71%), businessmen 10 (11.90%), farmers 12 (14.29%) and retired persons 2 (2.38%) (Table II).

Table II: Distribution of subjects according to occupation (N=84)

Occupation	Number	Percentage
Housewives	33	39.29
Students	18	21.43
Farmers	12	14.29
Businessmen	10	11.90
Service holder	09	10.71
Retired person	02	02.38

Table III shows that educated persons (84.43%) ingested poison more than the illiterate persons (15.47%). A significant p value (0.000) is found.

Table III: Distribution of patients according to educational qualifications (N=84)

Education level	Number	Percentage
Illiterate	13	15.48
Primary (I-V)	27	32.14
Secondary (VI-X)	35	41.66
Higher secondary (XI-XII)	07	8.33
Graduate	02	2.38

The motive of poisoning was suicidal and accidental. Most of the cases were suicidal 82 (97.62%) and only two (2.38%) cases were accidental. Causes of suicidal poisoning were familial disharmony (64, 76.19%) followed by financial loss (12, 14.29%) and failure in examination (8, 9.52%).

Out of 84 patients, 77.38% ingested OPCs, 21.24% cases ingested carbamates and 1.19% cases ingested both carbamate and organophosphates. Most of the patients (45, 53.57%) presented within 2–4 hours of ingestion of poison, 13 (15.47%) within 5–8 hours, 8 (9.52%) within 10–24 hours and 18 (21.42%) after 24 hours of ingestion (Table IV).

Table IV: Admission time after ingestion of poison

Admission time in hours	Frequency	Percentage
2–4	45	53.57
5–8	13	15.47
10–24	08	9.52
>24	18	21.42
Total	84	100

The incidence of toxicity may be reduced by early gastric lavage. In our study gastric lavage was commonly given in patients who were admitted within 2–3 hours of ingestion of poison. Among 84 cases, gastric lavage was given in 42 (50%) cases in EMCH and in 22 (26.19%) at Upazilla Health Complex/local community clinic where they visited first. Gastric lavage was not given in 20 (23.80%) cases. p value was found significant (0.000).

After confirming as acute poisoning cases loading dose of atropine was started. Most of the patients needed 38.4 mg to 153.6 mg of atropine. Maximum doses needed in different cases are summarized in Table V. Atropine toxicity was observed in 15.47% cases and was managed effectively.

All patients got adequate respiratory care. Ventilatory support was given in 41 (48.80%) cases and 43 (51.2%) cases were managed with oxygen support. Thirty two (78.05%) patients were successfully extubated from mechanical ventilator and weaning from ventilation was not successful in 9 (21.95%) patients. Mean duration of ventilatory support was 2–14 days. About 14 (34.14%) patients got ventilatory support for 2–4 days, 15 (36.58%) patients for 5–7 days and 12 (29.26%) for >7 days. p values for ventilatory support (0.182) and duration of total ventilator time (0.250) were found non-significant.

GCS level was <9 in 50% cases, 9–11 in 32.14% and 12–15 in 17.85% cases. After getting treatment, 75 (89.28%) patients survived and 9 (10.72%) patients expired. Mortality was found more in case of patients who were admitted after 24 hours of ingestion of poisoning and were with severe respiratory distress

Table V: Loading doses of atropine in acute poisoning cases (N=84)

Dose in mg	Number of ampoules	Number of patients	Percentage
19.2	32	06	7.1
38.4	64	22	26.19
76.8	128	20	23.80
153.6	256	21	25
307.2	512	13	15.47
614.4	1024	02	2.38

along with GCS <9. At the end of successful management 52 (61.91%) patients were discharged within 4–6 days, 16 (19.04%) within 2–3 days and 7 (8.33%) were discharged within 7–14 days. During the study period several complications and abnormal laboratory findings were observed (Table VI).

Table VI: Complications and abnormal laboratory findings

Complications and abnormal laboratory findings	Frequency	Percentage
Aspiration pneumonia	29	34.52
ARDS	11	13.90
Pleural effusion	17	20.23
Septicemia	07	8.33
Psychosis	14	16.66
Convulsion	13	15.47
Intermediate syndrome	12	14.28
Renal impairment	07	8.33
Altered liver function	08	9.52
Leukocytosis	52	61.90
Hypokalemia	37	44.04
Hyperglycemia	19	22.61
Ischemic changes in ECG	09	10.71

Discussion

Insecticide poisoning as suicidal attempt is a common problem in the rural and semi-urban areas of developing countries. In our study suicidal cases were observed in 97.61% and accidental in 2.38% cases which are almost similar to a study done in Rangpur Medical College Hospital (RMCH) by Sarker et al[8] where suicidal cases were in 92.2% cases and accidental in 7.8% cases. Most common cause of suicidal attempt was familial disharmony (76.19%) in our study which was also the top most cause in RMCH study[8].

Several studies have shown that incidence of poisoning in males are marginally higher than females.[12,24,25] In this study 60.7% subjects were males and 39.29% were females which is almost similar to a study[3] conducted in Dhamrai Upazilla Health Complex where males (61.30%) were predominant than females (38.70%).

In our study 65.47% subjects were married and 34.52% were unmarried, which is consistent with the study[3] done in Dhamrai Upazilla Health Complex where 68.64% were married and 31.36% were unmarried. In RMCH study[8] 71.45% subjects were married and 28.55% were unmarried. The most common age group in our study was found 21–40 years, which is almost similar to a study done by Godhwani et al[26] in India.

In this study poisoning was observed more in housewives (39.29%) followed by students (21.43%), farmers (14.29%), businessmen (11.90%) and service holders (10.71%). These results are almost similar to the study done in Rangpur Medical College Hospital[8] where 25.8% were housewives, 14% farmers and 16.1% were students.

In our study, illiterate persons (15.48%) were fewer than educated persons (84.52%) (primary 32.14%, secondary 41.66%, higher secondary 8.33% and graduate 2.3%). All these values are almost similar to RMCH study[8] (primary 35.7%, secondary 47.8%, higher secondary 10.8% and graduate 2.3%).

In this study, 18 (21.24%) patients consumed carbamates, 65 (77.38%) organophosphate and only one (1.19%) patient ingested mixed (both carbamates and organophosphate group) group of insecticides.

This is consistent with a study done by Godhwani et al[26] where 29.2% patients ingested carbamates and 60.68% organophosphates.

The time interval between ingestion and ICU admission plays an important role because early management reduces morbidity and mortality. In our study, it was observed that patients who were admitted early (53.57% within 2 to 4 hours) needed less amount of bolus dose of atropine and mechanical ventilation along with early recovery than the patients (21.42%) who were admitted more than 24 hours after exposure. Stomach wash also plays the same role because early wash reduces the toxicity.

After admission loading dose of atropine was started in all patients as the first line of treatment according to WHO guideline. Most of the patients in our study needed 38.4 mg to 153.6 mg of atropine, only two patients needed 614.4 mg of loading dose of atropine. Of these two patients, one patient survived and one expired. Though pralidoxime is a specific antidote, it was not possible to follow the current WHO guideline after loading dose due to its high cost. Godhwani et al[26] also did not follow current guideline, but their overall mortality rate was only 12% which is similar to our study (10.72%). Another study done by Sungur and Guven[10] showed that they also could not give pralidoxime in 14 patients due to lack of government supply and two patients were not given due to delayed admission (48 and 96 hours). But they were treated successfully with atropine alone; this may be due to rapid reactivation of dimethyl phosphorylated acetylcholinesterase although reactivation of diethyl phosphorrylated acetylcholinesterase needed a significant amount of oxime therapy. Another study done by De Silva et al[27] showed that pralidoxime plus atropine therapy do not have any benefit over atropine alone because the need for mechanical ventilation, ventilation duration, median days of intensive care unit stay, the frequency of the intermediate syndrome and the mortality rate were similar in each group.[27] In the present study, we also observed that mortality rate is not significantly different whether or not treated with pralidoxime.

Immediate management with ventilation may reduce mortality rate in patients with acute pesticide poisoning when present with severe respiratory distress. In our study 41 (48.80%) patients were given mechanical ventilation and 32 (78.05%) patients were successfully weaned from ventilatory support with an average duration of 5 to 7 days (36.58%). Eddleston et al[28] showed that out of 376 patients with acute pesticide poisoning 90 (24%) required ventilatory support and their ventilatory success rate was 49%, which is less than in our study (78.05%) and 51% intubated patients died.

In the study of Godhwani et al[26], out of 82 patients, 53 (64.63%) needed mechanical ventilation, success rate was 79.29% which is higher than the findings in our study with an average duration of 5.97 days. The higher rate of their incidence of ventilatory support was due to already developed aspiration pneumonitis, long time lag between ingestion of poison and admission to ICU. The overall mortality of their study was 12% which is higher than findings in our study (10.72%).

The intermediate syndrome is a state of muscle paralysis due to postsynaptic neuromuscular junction dysfunction. It usually occurs after recovery from acute cholinergic crisis period and before the onset of delayed polyneuropathy.[29]

Eddleston et al[30] showed that organophosphate (OP) pesticides were responsible for the major cause of death in most series of self-poisoning cases, especially from rural areas in contrast to carbamates which are less toxic. In our study we found almost similar result — out of nine patients three died due to ingestion of carbamate (2 by rodenticide and 1 by carbofuran). Ventilatory support was required less in carbamate group (12, 29.26%) and high in OPC group (29, 70.73%).

Sungur and Guven[10] observed several abnormal laboratory findings such as hyperglycemia in 15 (31.91%) patients, which may be due to secondary release of catecholamine from adrenal medulla, elevated liver enzymes in 11 (23.40%) and leukocytosis in 34 (72.34%) patients. In our study hyperglycemia was observed in 19 (10.71%) patients, leukocytosis in 52 (61.90%), and elevated liver enzyme (high SGPT) in 8 (9.52%), impaired renal function in 7 (8.33%) patients, ischemic changes in ECG in 9 (10.71%) patients and hypokalemia in 37 (44.04%) patients, which may be due to excessive vomiting.

OPC poisoning is a serious health problem in our

country. Early diagnosis and management is essential. As respiratory failure is the main cause of preventable death, early endotracheal intubation and mechanical ventilation for appropriate duration along with other pharmacological treatment, close monitoring and early recognition will reduce the morbidity and mortality. This can be possible by increasing the government ICU facilities with skilled manpower and logistics.

A national network on poison information service and management should be established and more studies should be done to share the experiences, problems and complications during the entire period of management for better outcome along with banning of more toxic poisonous agents to reduce the respiratory hazards. Strong psychiatric counseling and increased social awareness should be established to prevent the suicidal attempt.

Acknowledgement

We are grateful to Dr. Md. Enamur Rahman, Chairman, Enam Medical College & Hospital for his support to complete the treatment of the poor patients. We are thankful to Dr. Mohiuddin Ahmed, Associate Professor of Medicine for helping us to prepare the preformed data sheet for record keeping and guidance during the study period. We are also thankful to all our junior doctors and nursing staff for their enormous support.

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