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Outcome and Mortality Profile of Patients with Intermediate Syndrome After Acute Organophosphorus Poisoning

Dr. Prodip Kumar Mistry[1]*, Dr. Repon Chandra Saha[2], Dr. Md. Abul Kalam Azad Khan[3], Dr. Prianka Golder[4], Dr. Md. Imtiaz Alam[5]

[1]Assistant Professor, Department of Medicine, Dhaka Community Medical College, Dhaka, Bangladesh
[2]Assistant Professor, Department of Medicine, Brahmanbaria Medical College & Hospital, Brahmanbaria, Bangladesh
[3]Junior Consultant, Department of Gastroenterology, Mugda Medical College Hospital, Dhaka, Bangladesh
[4]Medical Officer, Department of Dermatology, Jahurul Islam Medical College, Dhaka, Bangladesh
[5]Resident, Department of Palliative Medicine, Bangladesh Medical University, Dhaka, Bangladesh

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***Corresponding author:** Dr. Prodip Kumar Mistry
Assistant Professor, Department of Medicine, Dhaka Community Medical College, Dhaka, Bangladesh

Abstract **Original Research Article**

Background: Intermediate syndrome (IMS) is an important neurological complication of acute organophosphorus (OP) poisoning and may cause significant respiratory and neuromuscular morbidity. This study assessed the clinical profile, treatment outcomes, and mortality among patients who developed IMS following acute OP poisoning. ***Objective:*** To assess the clinical profile, treatment outcomes, and mortality among patients with intermediate syndrome following acute organophosphorus poisoning. ***Methods:*** This observational study was conducted in the Department of Medicine, Jahurul Islam Medical College & Hospital, Bajitpur, Bangladesh, over six months from May 2016 to October 2016. Fifty consecutive patients aged >12 years who developed IMS following acute OP poisoning were enrolled. Clinical manifestations, complications, respiratory support, and final outcomes were recorded. Data were analyzed using SPSS version 20.0. Quantitative variables were expressed as mean±SD and qualitative variables as frequency and percentage. A p-value <0.05 was considered statistically significant. ***Results:*** The mean age was 26.33±5.14 years (range 16–69 years), and 38.0% were aged 21–30 years. Females comprised 62.0% of participants. Respiratory muscle weakness was the most common manifestation (44.0%), followed by neck muscle weakness (30.0%) and proximal muscle weakness (28.0%). Cranial nerve palsy occurred in 16.0%. Respiratory failure occurred in 8.0%, multiorgan failure in 22.0%, and 30.0% required mechanical ventilatory support. Overall, 76.0% survived and 24.0% died. Mortality was significantly higher among males than females (52.6% vs. 6.5%; p<0.001). ***Conclusion:*** IMS following acute OP poisoning was associated with substantial respiratory complications and mortality. Early recognition and close respiratory monitoring are essential for timely supportive and ventilatory management.

Keywords: Organophosphorus poisoning; Intermediate syndrome; Respiratory muscle weakness; Mechanical ventilation; Mortality.

INTRODUCTION

Organophosphorus (OP) pesticide poisoning remains an important public health problem, particularly in developing countries where pesticides are widely used in agriculture and are readily accessible to rural populations. Deliberate self-poisoning with pesticides contributes substantially to morbidity and mortality in the developing world, where agricultural chemicals may be easily available and are frequently used for intentional poisoning. [1] Pesticide poisoning has also been recognized as an important cause of poisoning-related mortality in developed countries, indicating that this problem is not restricted to low- and middle-income settings. [2,3] Despite its considerable clinical and public health consequences, pesticide poisoning has historically received inadequate attention as a major health priority in many developing countries. [2]

Organophosphorus compounds produce their principal toxic effects through inhibition of acetylcholinesterase, resulting in excessive accumulation of acetylcholine at muscarinic and nicotinic receptors and within the central nervous system. [4,5] Acute OP poisoning may therefore produce a wide spectrum of clinical manifestations, including salivation, lacrimation,

sweating, miosis, vomiting, diarrhoea, bronchorrhoea, bronchospasm, bradycardia, fasciculations, altered consciousness, seizures and respiratory failure. The severity of poisoning varies according to the particular OP compound, amount and route of exposure, and the time between poisoning and initiation of treatment.

In addition to the acute cholinergic manifestations, a proportion of patients develop a characteristic neurological complication known as intermediate syndrome (IMS). The syndrome was first described by Senanayake and Karalliedde in 1987 and was termed "intermediate" because it occurs after the acute cholinergic phase but before the development of organophosphate-induced delayed neuropathy. [4] IMS is characterized predominantly by weakness of the neck flexors, proximal limb muscles, respiratory muscles and motor cranial nerves. Distal muscle groups are relatively spared. The syndrome generally develops within approximately 24–96 hours following acute OP poisoning, although the timing and severity may vary between patients and different OP compounds. [5,6]

The exact pathophysiological mechanism of IMS remains incompletely understood. Persistent cholinesterase inhibition and dysfunction of the neuromuscular junction have been implicated, while abnormalities of nicotinic acetylcholine receptor function and associated myopathic changes have also been proposed.[5] The clinical presentation may range from mild proximal muscle weakness to severe generalized weakness with respiratory muscle paralysis. Because IMS occurs after the initial cholinergic crisis has apparently improved, its development may be overlooked unless patients remain under close clinical observation. [6]

The most serious manifestation of IMS is respiratory muscle weakness leading to respiratory insufficiency or respiratory failure. Patients may initially appear clinically improved but subsequently develop progressive weakness of the respiratory and bulbar muscles. Respiratory failure may require endotracheal intubation and prolonged mechanical ventilation and is an important determinant of morbidity and mortality in affected patients. [6,7] Consequently, continuous assessment of respiratory function and early recognition of neuromuscular deterioration are essential components of the management of patients at risk of IMS. Timely ventilatory support may allow patients with severe respiratory muscle involvement to recover completely, although the duration of respiratory support and clinical course can vary considerably. [7]

IMS should be distinguished from organophosphate-induced delayed neuropathy (OPIDN), another neurological complication of OP exposure. OPIDN generally occurs after a longer latent period and is characterized predominantly by a symmetrical sensorimotor axonopathy, particularly affecting long axons. [8] In contrast, IMS develops during the early recovery period following acute poisoning and is primarily characterized by neuromuscular weakness involving proximal, neck, cranial and respiratory muscles. [4,6] Recognition of this distinction is clinically important because IMS may require urgent respiratory support.

The outcome of acute OP poisoning can vary from complete recovery to severe complications and death. Clinical severity at presentation has been investigated as a predictor of outcome, and measures such as the Poison Severity Score and Glasgow Coma Scale have been evaluated for identifying patients at increased risk of adverse outcomes. [9] However, patients who develop IMS may deteriorate after the initial cholinergic manifestations have subsided, making continued monitoring particularly important. The development of respiratory failure, requirement for mechanical ventilation, duration of ventilatory support and associated complications may substantially influence the final outcome. [6,7]

Although recovery from IMS is possible with appropriate supportive management, severe cases may experience prolonged respiratory support, complications and mortality. Therefore, assessment of the clinical characteristics and outcomes of patients who develop IMS is important for understanding the clinical burden of this complication and for improving recognition and management in hospital practice. The present study was therefore undertaken to assess the outcome and mortality profile of patients with intermediate syndrome following acute organophosphorus poisoning, with particular emphasis on their clinical manifestations, respiratory complications, requirement for ventilatory support, duration of treatment, recovery and mortality.

Objectives

The main objective was to assess the clinical profile, treatment outcomes, and mortality among patients who develop intermediate syndrome following acute organophosphorus poisoning.

METHODOLOGY & MATERIALS

The observational study was conducted in the Department of Medicine, Jahurul Islam Medical College & Hospital (JIMCH), Bajitpur, Bangladesh. The study was carried out over six months from May 2016 to October 2016. The study population consisted of patients with acute organophosphorus compound poisoning admitted to the Department of Medicine who subsequently developed intermediate syndrome.

Inclusion Criteria:

- Patients of either sex with a history of ingestion and/or exposure to organophosphorus compounds.
- Patients aged >12 years.

- Patients who developed intermediate syndrome following acute organophosphorus poisoning.
- Patients who could be clinically evaluated and followed during their hospital stay.
- Patients or their legal guardians who provided informed written consent.

Exclusion Criteria:

- Patients with poisoning due to compounds other than organophosphorus compounds.
- Patients with co-existing serious cardiac or other major medical conditions that could independently affect the outcome.
- Pregnant women.
- Patients aged ≤12 years.
- Patients with hypokalaemia due to diarrhoea, vomiting, or diuretic use.
- Patients in whom the clinical features were insufficient to establish intermediate syndrome.

A total of 50 patients with intermediate syndrome following acute organophosphorus poisoning who fulfilled the inclusion criteria were enrolled consecutively during the study period. Intermediate syndrome was identified clinically by the development of neuromuscular weakness occurring approximately 24–96 hours after organophosphorus exposure, usually following improvement of the acute cholinergic manifestations. Particular attention was given to weakness involving the ocular muscles, muscles of the head and neck, proximal limb muscles, and respiratory muscles, with or without progression to ventilatory failure. Data were collected using a pretested structured questionnaire. Baseline demographic and clinical information, including age, sex, history and route of poisoning, time from poisoning to development of intermediate syndrome, clinical manifestations, neurological findings, and respiratory status, was recorded. During hospital admission, patients were regularly monitored for vital signs, respiratory function, oxygenation, electrocardiographic changes, progression of muscle weakness, and development of respiratory failure. Treatment details, including supportive management, pralidoxime therapy where indicated, antibiotic therapy, endotracheal intubation, and mechanical ventilation, were documented. The duration of ventilatory support, ICU stay, complications, clinical recovery, and final outcome were also recorded. The principal outcome measures were recovery and mortality, while respiratory failure, requirement for mechanical ventilation, duration of ventilation, complications, and length of hospital stay were assessed as important secondary outcomes.

Ethical principles were maintained throughout the study. Permission was obtained from the appropriate hospital authority before commencement of data collection. Written informed consent was obtained from each patient, or from a legal guardian when the patient was seriously ill or unable to provide consent. Participation was voluntary, and participants were free to withdraw at any stage without affecting their treatment. Confidentiality and anonymity of all participants were strictly maintained, and the collected information was used solely for research purposes.

Statistical Analysis:

All data were recorded systematically in a preformed data collection sheet and entered into Statistical Package for the Social Sciences (SPSS), Version 20.0 for analysis. Quantitative variables were expressed as mean ± standard deviation, while qualitative variables were presented as frequency and percentage. Appropriate statistical tests were applied where applicable to compare clinical characteristics and outcomes. A p-value <0.05 was considered statistically significant. Confidentiality of all patient information was strictly maintained throughout the study.

RESULT

Table 1: Baseline demographic characteristics of patients with intermediate syndrome following acute organophosphorus poisoning (n=50)

Characteristic	Frequency (n)	Percentage (%)
Age group, years		
12–20	7	14
21–30	19	38
31–40	15	30
41–50	4	8
51–60	3	6
61–70	2	4
Age (Mean ± SD)	26.33 ± 5.14	
Range*	16–69	
Sex		
Male	19	38
Female	31	62
Male: Female ratio	01:01.6	

Table 1 shows that the largest proportion of patients were aged 21–30 years (38.0%), followed by those aged 31–40 years (30.0%). Females constituted 62.0% of the study population, while males constituted 38.0%, giving a male-to-female ratio of 1:1.63.

Table 2: Clinical manifestations of intermediate syndrome among the study participants (n=50)

Clinical manifestation	Frequency (n)	Percentage (%)
Respiratory muscle weakness	22	44
Neck muscle weakness	15	30
Proximal muscle weakness	14	28
Cranial nerve palsy	8	16

Table 2 shows that respiratory muscle weakness was the most frequently reported manifestation (44.0%), followed by neck muscle weakness (30.0%) and proximal muscle weakness (28.0%). Cranial nerve palsy was reported in 16.0% of patients.

Table 3: Complications and respiratory support among patients with intermediate syndrome (n=50)

Clinical outcome/intervention	Frequency (n)	Percentage (%)
Respiratory failure	4	8
Multiorgan failure	11	22
Mechanical ventilatory support required	15	30

Table 3 shows that respiratory failure occurred in 8.0% of patients, while multiorgan failure occurred in 22.0%. Mechanical ventilatory support was required in 30.0% of patients.

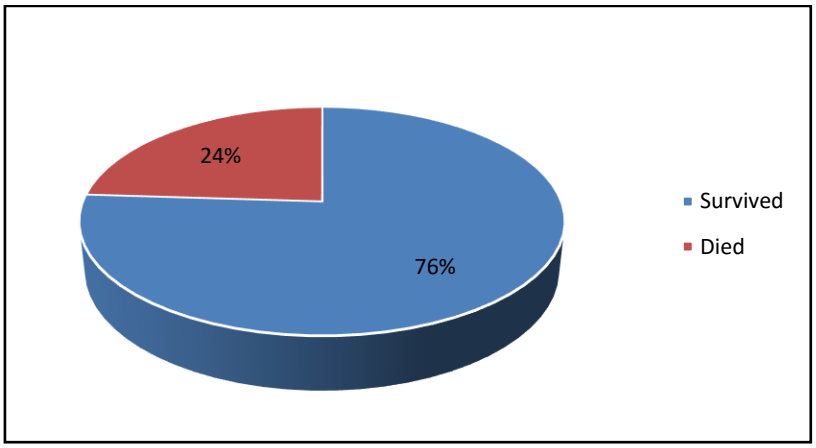

Figure 1: Overall clinical outcome and mortality among patients with intermediate syndrome (n=50)

Figure 1 shows that 38 patients (76.0%) survived, whereas 12 patients (24.0%) died, giving an overall mortality rate of 24.0%.

Table 4: Association between sex and final outcome among patients with intermediate syndrome (n=50)

Sex	Survived, n (%)	Died, n (%)	Total, n (%)	p-value
Male	9 (47.4)	10 (52.6)	19 (100.0)	<0.001
Female	29 (93.5)	2 (6.5)	31 (100.0)	
Total	38 (76.0)	12 (24.0)	50 (100.0)	

Fisher's exact test

Table 6 shows a significant association between sex and final outcome among patients with intermediate syndrome. Among the 19 male patients, 9 (47.4%) survived and 10 (52.6%) died. In contrast, among the 31 female patients, 29 (93.5%) survived and 2 (6.5%) died. Overall, 38 (76.0%) patients survived, while 12 (24.0%) died. The difference in outcome between male and female patients was statistically significant ($p < 0.001$).

DISCUSSION

Acute organophosphorus (OP) poisoning remains an important clinical problem, particularly in developing countries, where pesticide exposure contributes substantially to poisoning-related morbidity and mortality. [10] Intermediate syndrome (IMS) is a

recognized neurological complication of acute OP poisoning that develops after the acute cholinergic phase and is characterized predominantly by weakness of the neck, proximal limb, cranial, and respiratory muscles. [11,12] The present study assessed the clinical manifestations, complications, respiratory support requirements, and final outcomes among 50 patients who developed IMS following acute OP poisoning.

In the present study, the patients had a mean age of 26.33±5.14 years, with an age range of 16–69 years. The largest proportion belonged to the 21–30-year age group (38.0%), followed by the 31–40-year group (30.0%). Overall, 82.0% of the patients were between 16 and 40 years of age. This predominance of young adults is consistent with previous observations of OP poisoning and IMS. In one previous study, patients had an age range of 13–50 years with a mean age of 31.5 years. [13] Hayden *et al.,* reported an age range of 13–47 years with a mean age of 23 years, which is close to the mean age observed in the present study. [12] Khan MN *et al.,* also reported that the majority of affected patients were between 15 and 35 years of age. [14] Similarly, other investigators reported that the 21–30-year age group was the most commonly affected. [15] The predominance of younger adults in the present study may therefore reflect the demographic pattern commonly observed among patients presenting with acute pesticide poisoning and its neurological complications.

Regarding sex distribution, females constituted 62.0% of the study population, whereas males constituted 38.0%, resulting in a male-to-female ratio of 1:1.63. This finding differs from some previous reports in which males predominated. Ather reported a female-to-male ratio of approximately 1:1, whereas Tall *et al.,* reported a female-to-male ratio of 1:1.8. The difference in sex distribution among studies may be related to differences in study setting, population characteristics, type and circumstances of poisoning, and inclusion criteria. In the present study, all enrolled patients had developed IMS, and therefore the observed sex distribution represents the selected population with this complication rather than the sex distribution of all patients with OP poisoning.

The clinical manifestations observed in the present study were predominantly related to respiratory and neuromuscular weakness. Respiratory muscle weakness was the most frequent manifestation, occurring in 22 patients (44.0%), followed by neck muscle weakness in 15 patients (30.0%) and proximal muscle weakness in 14 patients (28.0%). Cranial nerve palsy was observed in 8 patients (16.0%). These findings are consistent with the recognized clinical spectrum of IMS, which includes weakness of the respiratory, neck, proximal limb, and cranial muscles. [11,12] Senanayake and Karalliedde originally described patients developing paralysis of the proximal limb muscles, neck flexors, motor cranial nerves, and respiratory muscles approximately 24–96 hours after acute OP poisoning.[12] The predominance of respiratory muscle weakness in the present study is also consistent with previous observations in which respiratory muscle involvement was reported as an important manifestation of IMS. [16]

The relative frequency of individual muscle-group involvement has varied among previous studies. Cranial nerve palsy was reported more prominently in the Sri Lankan study by Senanayake, whereas proximal muscle involvement was emphasized in earlier observations by Wadia. Such variation may be related to differences in the severity and timing of clinical assessment, characteristics of the poisoning agent, and criteria used for identifying IMS. [11,17] Nevertheless, the pattern observed in the present study supports the characteristic neuromuscular presentation of IMS.

Several mechanisms have been proposed to explain the development of IMS. Wadia [21] suggested that persistence of nicotinic effects, potentially related to inadequate or delayed oxime therapy, may contribute to the development of paralysis. [11] Gadoth and Fischer proposed that delayed release of organophosphorus compounds from adipose tissue may result in continued effects on nicotinic receptors. [18] In contrast, Senanayake emphasized neuromuscular-junction dysfunction as an important mechanism underlying the syndrome. [17] Electrophysiological observations in subsequent studies have also supported neuromuscular-junction dysfunction as an important component of IMS. [12,23] The different proposed mechanisms indicate that the pathophysiology of IMS is complex and may vary according to the particular OP compound and severity of poisoning.

Respiratory involvement was an important finding in the present study. Respiratory failure occurred in 4 patients (8.0%), while 15 patients (30.0%) required mechanical ventilatory support. The relatively frequent requirement for ventilatory support is clinically important because respiratory muscle weakness is a characteristic component of IMS and may progress to respiratory failure. Previous studies have similarly demonstrated that delayed respiratory failure is an important manifestation of OP poisoning and may occur after apparent improvement from the initial cholinergic phase.[17] [40] In a large prospective study, delayed respiratory failure requiring intubation was observed after the initial acute phase in a proportion of patients, emphasizing the need for continued respiratory monitoring. [19]

Multiorgan failure was documented in 11 patients (22.0%) in the present study. The occurrence of multiorgan failure indicates that severe OP poisoning complicated by IMS may be associated with systemic complications in addition to neuromuscular manifestations. In the original thesis observations, the organs involved in multiorgan failure included the lungs,

cardiovascular system, and brain. However, because the present results provide only the overall frequency of multiorgan failure and do not provide organ-specific frequencies, no further quantitative comparison can be made from the current dataset.

The overall outcome of the present study showed that 38 patients (76.0%) survived, whereas 12 patients (24.0%) died, giving an overall mortality rate of 24.0%. This mortality is within the broad range reported in previous studies of IMS.[11] The mortality observed in the present study was comparable to the findings reported from Vellore. [20] Other studies have reported mortality rates ranging from approximately 10.5% to 41.6%. [21] Yamashita reported considerable variation in mortality associated with OP poisoning, ranging from 4% to 30%, whereas Malik reported a mortality rate of 5.5%. [21,22] Differences in mortality among studies may reflect variation in poisoning severity, time to hospital presentation, availability of intensive care facilities, respiratory support, and other aspects of supportive treatment.

The mortality observed in the present study was lower than that reported by Numidasa UA *et al.,* and Pandyal BP *et al.,*[47], as noted in the original thesis.[23,24] Such differences may be related to variations in patient characteristics, severity of poisoning, treatment facilities, and clinical management. Suliman MI *et al.,*[19] also reported an association between delay in treatment and adverse outcome. However, although treatment delay was discussed in the original thesis, the present results provided here do not include a quantitative analysis of treatment delay in relation to mortality. Therefore, treatment delay cannot be identified as an independent determinant of mortality from the current results.

An important finding of the present study was the significant association between sex and final outcome. Among the 19 male patients, 9 (47.4%) survived and 10 (52.6%) died. In contrast, among the 31 female patients, 29 (93.5%) survived and only 2 (6.5%) died. Thus, mortality was proportionally higher among male patients than female patients, and the difference was statistically significant ($p<0.001$). This finding indicates an association between sex and final outcome in this study population. However, because this was an observational study with a relatively small sample size, the observed association should not be interpreted as evidence that sex itself caused the difference in mortality. Other clinical factors, such as severity of poisoning, respiratory involvement, multiorgan failure, treatment delay, and requirement for mechanical ventilation, may potentially contribute to differences in outcome. These factors were not simultaneously adjusted for in the present analysis.

The present findings also emphasize the importance of early recognition and close monitoring of patients who develop IMS after acute OP poisoning. The characteristic involvement of respiratory, neck, proximal, and cranial muscles requires repeated clinical assessment, particularly because respiratory weakness may progress to respiratory failure. Previous literature has emphasized the importance of recognizing delayed respiratory deterioration and providing appropriate ventilatory support when required. [12, 19] Adequate respiratory care is considered a crucial component of management in patients with clinically significant IMS. [17]

Overall, the present study demonstrates that IMS following acute OP poisoning predominantly affects young adults and is characterized by important respiratory and neuromuscular manifestations. Respiratory muscle weakness was the most common clinical manifestation, while 30.0% of patients required mechanical ventilatory support. Despite supportive management, the overall mortality was 24.0%. A statistically significant association was also observed between sex and final outcome, with a higher proportion of deaths among male patients. These findings are broadly consistent with the clinical characteristics and outcome patterns described in previous studies and highlight the importance of early identification of IMS, continuous respiratory monitoring, and timely supportive management.

Limitations of the study

This study was limited by its single-center design and relatively small sample size of 50 patients, which may restrict the generalizability of the findings. The observational design also limits causal inference, and the absence of a comparison group prevented assessment of factors associated with the development of intermediate syndrome. The small number of deaths further limited detailed analysis of independent predictors of mortality.

CONCLUSION

Intermediate syndrome following acute organophosphorus poisoning was associated with substantial respiratory and neuromuscular morbidity and considerable mortality. Young adults constituted the majority of affected patients, with respiratory muscle weakness being the most common clinical manifestation. Mechanical ventilatory support was required in 30.0% of patients, while the overall mortality was 24.0%. A significant association was observed between sex and final outcome, with a higher proportion of mortality among male patients in this study. These findings emphasize the importance of early recognition of intermediate syndrome, close monitoring for respiratory deterioration, and timely provision of appropriate supportive and ventilatory care. Larger, multicenter prospective studies are warranted to better identify independent predictors of mortality and optimize

management strategies for patients with intermediate syndrome.

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