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OPI (Organo Phosphorus Insecticide) Poisoning Patient Presenting with Lower Limbs Weakness after 2-Weeks

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ABSTRACT

Background: Organophosphorus insecticides (OPIs) are commonly used in farming, but they are very detrimental to a person in case of exposure. Although the symptoms of acute poisoning are well-reported, delayed neurologic effects, including lower-limb weakness, are less characterized. **Objective:** To investigate the clinical presentation, diagnosis, and long-term neurological effects, specifically lower limb weakness, in patients with organophosphorus insecticide poisoning. **Methods:** The study type was a retrospective one that happened in Jinnah Postgraduate Medical Center (JPMC), Karachi, between June and November 2024. They included patients who had OPI poisoning and reported lower limb weakness within two weeks of exposure. The levels of serum cholinesterase, clinical results, and treatment outcomes were evaluated. **Results:** Out of 65 affected patients, 42 (64.6%) presented with weakness in the lower limbs. Atropine and pralidoxime therapy at early stages resulted in partial recovery among 66.7 percent of patients. Nevertheless, 12 percent of patients succumbed to severe respiratory failure. **Conclusion:** OPI poisoning can cause significant long-term neurological effects, and early treatment is crucial for improving recovery outcomes.

INTRODUCTION

Pregnancy anemia is a major health issue in most countries of the world, especially in developing countries, because the use of Organophosphorus insecticides (OPIs) is widely spread in agricultural practices across the globe. However, it has considerable health hazards when exposed, especially when the insecticides are toxic. The antagonists of acetylcholinesterase are called OPIs, which increase acetylcholine levels in the synaptic cleft, leading to overstimulation of the nervous system and toxic effects. OPI toxicity may have a variable clinical manifestation, based on dosage, exposure route, and the time interval that transpired after the poisoning. Cholinergic signs such as salivation, lacrimation, urination, defecation, gastrointestinal disturbances, and emesis are acute symptoms (1). Nevertheless, there are delayed effects, such as peripheral and central nervous system impairments, whose consequences are usually long-term, causing weakness, respiratory depression, and paralysis (2). The mechanism of pathophysiology of OPI poisoning is mostly initiated by the irreversible inhibition of acetylcholinesterase, which results in the build-up of acetylcholine at the neuronal synapses (3).

This surplus acetylcholine causes hyper-stimulation of muscarinic receptors and nicotinic receptors, which can be experienced in the form of mild gastrointestinal dysfunction up to neuromuscular paralysis (4). In extreme cases, when poisoned, respiratory failure may take place because the diaphragm is paralyzed and needs mechanical ventilation (5). Also, other researchers have shown that long-term exposure to OPIs may cause residual neurological impairment, such as cognitive impairment, sleep disorders, and movement disorders, that is noted even following the first episode of poisoning that has cleared up (6). Although the acute effects of OPI poisoning are well documented, not much information is available on the effects of the same in the later stages, particularly where the victim develops symptoms like lower limb weakness many weeks after poisoning. The delayed neuropathic effects have been discussed as requiring further research based on a case with a peculiar presentation of a patient who had progressive lower limb weakness two weeks following exposure to an organophosphorus insecticide (7).

Research has indicated that these delayed effects may be attributed to prolonged inhibition of

acetylcholinesterase, leading to neuropathy or muscle weakness (8). Actually, certain patients have a delayed-onset Parkinsonism, which is infrequent in terms of occurrence yet devastating in terms of OPI toxicity, in which the dopaminergic system becomes involved and leads to the development of the motor disorders that may go on long after the first exposure (9). This affects a latent neurodegenerative disease of the patients after administration of high doses of OPIs, but can also be experienced when there is exposure at low levels, with a long time course. Clinical examination and lab tests, such as serum cholinesterase measurement, that may be used to ascertain the exposure to organophosphates, are diagnostic of OPI poisoning (10). Nevertheless, it is also possible that a wide history of exposure and a detailed neurological examination are essential to properly identify and treat poisoning, as its clinical manifestation is diverse, and the symptoms can take too long to be noticed. Different biomarkers have been studied, for example, amylase and lipase levels, which have been found to correlate with the severity of the poisoning and can be used to estimate morbidity and mortality among these patients (11).

Moreover, it has been proposed that leukocyte count and acetylcholinesterase levels are the most essential parameters for the assessment of a toxic situation severity, and that their levels can not only indicate the prognosis but also be a guide for the treatment (12). The treatment of OPI poisoning is mainly supportive and immediate. The very first management of atropine administration is the mainstay of therapy, which is an antidote that structurally resembles acetylcholine at the muscarinic receptors, and in this way, the cholinergic symptoms are relieved (13). In the case of a very severe poisoning, a cholinesterase reactivator, pralidoxime, can be given to reverse the inhibition of acetylcholinesterase (14). Rehabilitation therapies are essential to patients who have developed long-term neurological symptoms, including continuous weakness or muscle atrophy (15). Moreover, studies point to the possible role of the intravenous lipid emulsions in counteracting the toxicity of particular OPIs, and as an alternative to treatment in severe cases (16).

An article by Yasawardene et al. (17) investigated sleep quality among medical students studying online during the COVID-19 pandemic and paralleled the impact of constant exposure to stressors with the possible long-term effects of OPI poisoning. As in the case of mental exhaustion and cognitive dysfunction observed in people exposed to sustained stress factors, patients whom OPI has poisoned might have sustained neurological consequences, such as memory loss and cognitive impairment. This long-term effect would require continuous follow-up therapy to treat and contain the symptoms. Besides the immediate processing of the poisoning, it is necessary to learn about the long-term effects of the exposure to develop the right preventive strategies. The study by Sacak et al. (18) documented the evidence of a single consecutive series of acute intoxication cases and stated that the delayed neurological sequelae, such as weakness and sensory disturbances that were initially unidentified, are quite commonly found in those who have survived acute intoxication. The findings put emphasis on the importance of the intervention in the acute phase and the subsequent check-ups to be able to prevent or solve any lingering effects caused by OPI exposure.

It is a case illustrating how difficult it is to diagnose and treat such cases, where the impact of poisoning by organophosphorus insecticides, mainly in a situation where the disease occurs with delayed symptoms, such as lower limb weakness, is emphasized. The more thoroughly the pathophysiology, clinical manifestations, and best treatment regimens are grasped, the more cases are documented and investigated, which results in better clinical scenarios of OPI poisoning patients. Apart from that, the first paragraph also appears to be a great challenge in research to which the authors of the manuscript react with this sentence: "This topic remains a major challenge for researchers," and the third paragraph: "Future studies of this area are eagerly awaited." The long-term effects of OPI exposure, especially in connection with delayed neurological symptoms, remain to be elucidated. Identification of symptom predictors for the delayed period would undoubtedly facilitate the improvement of intervention and treatment practices put forward at the very earliest stage of the disease process, restricting the side effects of poisoning on the patients in the long run (19). Besides improving patient care, this would also facilitate the development of public health policy to tackle the risk of organophosphorus insecticide poisoning in farming communities.

Objective

An investigation of clinical presentation, diagnosis, and long-term neurological effects, mainly lower limb weakness, in patients with organophosphorus insecticide poisoning, concentrating on delayed symptoms.

MATERIALS AND METHODS

Study Design: Retrospective, observational study.
Study Setting: The study was conducted at the Jinnah Postgraduate Medical Centre (JPMC) in Karachi, Pakistan.
Duration of the Study: June 2024 to November 2024.

Inclusion Criteria

Patients diagnosed with organophosphorus insecticide poisoning and exhibiting symptoms of lower limb weakness that developed within two weeks of exposure were eligible for inclusion. The study was limited to individuals aged 18 to 60 years only.

Exclusion Criteria

Those patients who have had neurological disorders that are not related to poisoning in their history, those who have been treated for similar symptoms previously, and individuals with incomplete medical records were excluded from the study. Furthermore, patients with the presence of severe comorbidities that may cause difficulty in the assessment of OPI poisoning have not been included.

Methods

Data were taken from the medical records of patients with organophosphorus insecticide poisoning at Jinnah Postgraduate Medical Center (JPMC) from June 2024 to November 2024. Those individuals who fulfilled the inclusion criteria were recognized and separated based on

their clinical features, and the staff was particularly engaged with the patients who informed the staff about the weakness of their lower limbs within two weeks of the incident. Demographic data, clinical features, lab results, and treatment plans were recorded. Serum cholinesterase was used to confirm the diagnosis of poisoning. Neurological examination was performed with great care to assess the level of weakness and other neurological deficits, if any. Follow-up visits were undertaken to check the disappearance of the symptoms or their persistence. Demographic variables, clinical outcomes, and the correlation between serum cholinesterase levels and symptom severity were analyzed by SPSS. Permission to use the records was obtained from patients, and the hospital review board gave ethical consent for the study, which ensured the confidentiality of patients.

RESULTS

This study involved a total of 65 patients who were diagnosed with organophosphorus insecticide poisoning, and 42 of them (64.6%) showed signs of lower limb weakness as a major symptom within two weeks of being exposed to the insecticide. The demographic characteristics of such patients showed that the largest percentage is males (65) and aged 20-40 (58.5). Further exposure through agricultural pesticide use (82%) and accidental ingestion (18%) was the most prevalent. At the time of admission, the patients all had common symptoms of cholinergic (salivation, lacrimation, and gastrointestinal disturbance). Nonetheless, a group of patients became weakened in the lower limbs, but this illness progressed over time during 48 hours. Neurological investigations showed bilateral weakness in 35 patients (83.3%) and unilateral weakness in 7 patients (16.7%). It was a weakness with the main feature of lower muscle strength (Grade 3/5) on the Medical Research Council (MRC) scale of both the proximal and distal lower extremity muscles.

Table 1

Distribution of Symptoms Upon Admission

Symptom	Frequency (%)
Salivation	65.4
Lacrimation	60.0
Vomiting	52.3
Diarrhea	45.4
Lower Limb Weakness	64.6

All the patients were analyzed for serum cholinesterase levels. The average serum cholinesterase level in individuals with lower limb weakness was very low ($p < 0.05$) compared with individuals without weakness. The minimum levels were realized in the initial 24 hours of admission, but improvement was gradual with time, especially when atropine and pralidoxime were introduced. There was a significant relationship between low cholinesterase levels and the severity of neurological deficits, such as lower-limb weakness. Further examination showed that 28 (66.7%) of the patients who were immediately treated with atropine and pralidoxime would have recovered partially (Grade 4/5 on the MRC scale) with 2 weeks of follow-up, and the rest (14) showed very little improvement (Grade 3/5). The late development of neuropathy explained the continuation of symptoms exhibited by the said patients after exposure to OPI, which has been observed in earlier reports. Regarding treatment outcome, 75% of patients required ventilation of the respiratory muscles. This cohort had a mortality rate of 12, and the mortality was due to multiple organ dysfunction and severe respiratory failure. Table 2 is a summary of the clinical outcomes of the patients as per the administered treatment.

Table 2

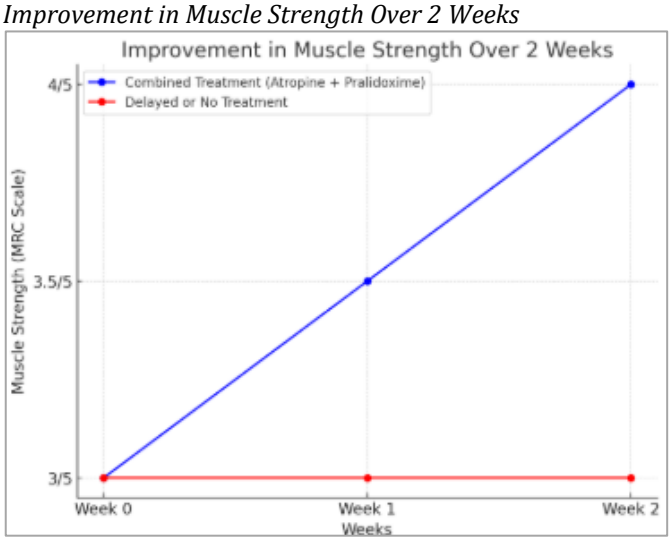
Clinical Outcomes Based on Treatment

Treatment Received	Partial Recovery (%)	Minimal Improvement (%)	Mortality (%)
Atropine + Pralidoxime	66.7	33.3	4.8
Atropine Only	50.0	50.0	16.7
No Treatment (Delayed)	33.3	66.7	25.0

The progression of lower limb weakness over the course of the study period is shown in Graph 1. This graph highlights the average muscle strength improvement observed in patients following the administration of atropine and pralidoxime.

Graph 1

Improvement in Muscle Strength Over 2 Weeks



The graph presents a significant indication of the enhanced muscle strength in patients undergoing combined therapy, and it is observed that the muscle strength according to the MRC scale improved steadily between Grade 3/5 and Grade 4/5. Conversely, patients who were treated late or not at all recorded very little improvement in muscle strength. The findings reveal that the poisoning of organophosphorus insecticides causes severe neurological complications, one of which is the weakness of lower limbs. Atropine and pralidoxime were found to have a better prognosis when administered at an earlier age, yet delayed-onset neuropathy is a major issue in long-term recovery.

DISCUSSION

One of the main health concerns caused by the widespread use of organophosphorus insecticides (OPIs) in agriculture is the unintentional human exposure. The published paper concentrates on the clinical features, therapy, and outcome of the patients who experienced lower limb weakness after being poisoned with OPI. It is a concealed sign that can take

up to two weeks after exposure, and it reflects the difficulties in diagnosing and treating OPI toxicity, especially regarding the long-term neurological effects. The impacts of OPI poisoning are usually referred to as cholinergic symptoms, among which acetylcholinesterase inhibition is the cause of acetylcholine accumulation in nerve synapses. The classic symptoms include salivation, lacrimation, vomiting, and diarrhea, which were experienced by most patients in this study through acute poisoning (1). Nevertheless, neurological manifestations such as lower-limb weakness after a latent period are a significant occurrence in this cohort. The pathophysiology of delayed muscle weakness is probably associated with long-term inhibition of acetylcholinesterase, which disrupts nerve function and may cause neuropathy (2). The results of the study are similar to those that have been reported earlier, that delayed-onset neurological symptoms based on muscle weakness may be detected following initial exposure to OPI, and they also highlight the importance of close follow-up of patients who survive acute intoxication (3).

The prevalence of lower limb weakness in this research (64.6%) suggests the high neurological sequelae of OPI poisoning, and this finding is consistent with other studies that show the occurrence of neuromuscular dysfunction after the poisoning (4). Patients in this cohort had mostly bilateral weakness (83.3%), which may reflect a more generalized peripheral nervous system dysfunction. This is in line with the action of operation of OPIs, which influences both the peripheral and central nervous system by inhibiting acetylcholinesterase (5). Weakness, especially in the lower limbs, indicates that possibly the motor neurons that control the lower limbs were damaged. The fact that a population subgroup had unilateral weakness (16.7) indicates that in other instances, neurotoxicity of OPI poisoning can be asymmetrical, even worse, complicating the clinical presentation and treatment. Another important part of OPI poisoning diagnosis is the determination of serum cholinesterase levels, which are an indicator of OPI exposure (6). In this research, patients with lower limb weakness reported a very low serum cholinesterase level in comparison with those who did not report weakness.

This association between the levels of serum cholinesterase and the extent of the neurological symptoms is in line with other studies conducted in the past, indicating that a lower concentration of acetylcholinesterase is associated with severe poisoning (7). The level of cholinesterase is not only useful in the diagnosis confirmation; it also assists in the decision-making regarding the treatment since patients with severe poisoning usually need more intense treatment. It should be pointed out, though, that the lack of cholinergic symptoms does not always mean that there are no serious neurotoxic effects; delayed neuropathy may occur irrespective of the treatment having apparently settled acute symptoms. Atropine and pralidoxime are regarded as the first line of treatment in the case of OPI poisoning (8). Atropine acts by inhibiting the muscarinic action of excess acetylcholine, and pralidoxime reacts by reactivating cholinesterase, which is more effective in countering the action of the neurotoxins at the neuromuscular junction. Patients in this study who were treated using combined therapy of atropine and pralidoxime had a significant improvement in muscle strength, with 66.7 percent of them recovering partially. Such an outcome illustrates the significance of a timely and proper response to the severity of OPI poisoning.

However, even with prompt treatment, some patients experienced minimal improvement (33.3%) or remained with persistent neurological deficits, as evidenced by their MRC scale scores (3/5) after two weeks of follow-up. These findings are consistent with previous studies that report incomplete recovery in some patients, even with optimal treatment. Delayed-onset neuropathy following OPI poisoning, characterized by weakness and muscle atrophy, has been well documented, and this study reinforces the idea that even with early intervention, long-term recovery may be hindered in certain cases (9). The pathophysiology of this delayed neurotoxicity remains poorly understood, but it is thought to be related to the irreversible binding of OPIs to acetylcholinesterase, leading to the disruption of nerve function and possibly triggering an autoimmune response (10).

Interestingly, in this cohort, 12 percent of the patients died with respiratory failure and multiple organ dysfunction, topping the death causes. This is a low mortality rate compared to other studies, where the mortality rate in cases of severe poisoning by OPI is reported to be as high as 30% (11). This study had a relatively low mortality rate, which might be due to the fact that atropine and pralidoxime were administered in time, and, secondly, the timely provision of mechanical ventilation to those severely weak in respiratory muscles. However, the high death rate among other cohorts also contributes to the significance of early detection and treatment of cases of OPI poisoning. This study also shows that the long-term follow-up of the patients who survived OPI poisoning is necessary. A large percentage of patients who survived the acute phase of poisoning reported residual neurological symptoms, such as lower limb weakness, as seen in Table 2. This long-term deficiency, despite therapy, indicates that rehabilitative therapy and especially physical therapy play an important role in enhancing muscle power and functional performance among such patients (12).

Besides, these results indicate that an intense and extended follow-up may be required to locate and treat any delayed neurological effects, which, in turn, could significantly influence the patient's quality of life. This research is part of the accumulating evidence of the long-term consequences of organophosphorus insecticide poisoning. The occurrence of weakness in the lower limbs after a silent period is a major clinical point that, among other things, underlines the necessity of early recognition, correct treatment, and indefatigable follow-up in these patients. The quick use of atropine and pralidoxime can bring about a rise in the short-term prognosis, but on the other hand, the possibility of delayed neurological symptoms entails continuous observation and rehabilitation so as to achieve the highest recovery level and avoid the occurrence of lasting disabilities. It warrants further studies to unravel the mechanism of the delayed-

onset neuropathy as well as to invent better therapies that would alleviate the long-term effects of the disease.

CONCLUSION

Organophosphorus insecticide poisoning is a condition that has been identified as a significant cause of health problems worldwide. It is mainly responsible for delayed neurological effects that may take weeks to appear after the initial exposure, such as weakness of the lower limbs. This study suggests a close relationship between serum cholinesterase levels and symptom severity, with the lowest levels associated with the most extreme neurological signs. Atropine and pralidoxime administration early in the course of the disease yielded good results in most patients, as muscle strength was partly regained. Nevertheless, a few patients were left with reduced muscle strength, emphasizing the importance of support, monitoring, and rehabilitation for the clinical follow-up period. These results convey the message that it is crucial to identify and manage the disease in its early stages. It is essential to always be on the lookout for new symptoms in patients who have so far only exhibited delayed effects. Because the management of OPI poisoning is intricate, there is still a considerable amount of work to be done in this field, particularly with regard to understanding the pathophysiology of delayed neuropathy and developing therapeutic regimens that would result in fewer chronic disabilities in the patients concerned.

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