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INTERMEDIATE SYNDROME FOLLOWING ORGANOPHOSPHATE INSECTICIDE POISONING IN EMERGENCY DEPARTMENT OF EASTERN NEPAL

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ABSTRACT

Acute organophosphate poisoning is the most common pesticide poisoning which can be manifested in three unique phases of toxic effects e.g. Acute Cholinergic Crisis, Intermediate Syndrome (IMS), Delayed Polyneuropathy. Looking at the previous cases reports the incidence of Intermediate syndrome has been reported to be as high as 80%.

The present study attempts a comparable form of incidence which was encountered in Emergency Department of BP Koirala Institute of Health Sciences (BPKIHS) in Eastern Nepal. The symptoms was analyzed with its manifestation occurred after 24 to 96 hours, affecting conscious patients without cholinergic signs, and involve the muscles of respiration, proximal limb muscles, neck flexors, and muscles innervated by motor cranial nerves. Early recognition and with appropriate supportive therapy that includes artificial respiration complete recovery may occur within or 5–18 days following IMS.

Introduction:

Acute organophosphate poisoning is most common poisoning and accounts nearly 3 million cases every year resulting deaths in more than 250,000. It is a major health problem worldwide and important clinical emergency and contributor to morbidity and mortality.[1]

It can manifest in three different phases of toxic effect e.g. Acute Cholinergic Crisis, Intermediate Syndrome (IMS) and Delayed Polyneuropathy.

Acute cholinergic crisis develops within a few minutes to several hours after exposure, and affects peripheral muscarinic and nicotinic receptors, as well as the central nervous system, through the inhibition of carboxylic esterase enzymes.

Organophosphate- induced delayed neurotoxicity (OPIDN) occurs 2–3 weeks after acute exposure to certain organophosphate insecticides. The clinical features are predominantly motor neuropathy and primarily manifest as numbness and weakness of the lower extremities, followed by progressive ascending weakness of limb muscles.

The disease entity is believed to be due to the inhibition of a poorly characterized esterase called the neuropathy target esterase. 2-5

In between the interval of acute cholinergic crisis and OPIDN, organophosphate compound can cause IMS which was first described by Senanayake et.al.1987.It is mainly characterized by weakness of proximal limb muscles, neck flexors, respiratory muscles, and motor cranial nerves, and was attributed to muscle fiber necrosis following acute cholinergic crisis.

Following previous case studies the incidence of Intermediate syndrome has been reported to be as high as 80 %.6- 12

The present study attempts to report the manifestation of Intermediate Syndrome following Acute Organophosphate Poisoning.

Illustrative case:

Case 1

A 17-year old married female was referred from a local hospital to Emergency Department of BP Koirala Institute of Health Sciences (BPKIHS) with alleged history of ingestion of Cholrpyriphos 40% with suicidal intent week ago with complaints of inability to sit and unable to hold neck since 1 day.

On examination her Glasgow Coma Scale (GCS) was E4 M6 V3, Blood Pressure: 110/60 mm Hg, Heart Rate: 92/min, Respiratory Rate: 24/m, SpO2: 97% at room air. Pupils were less than 2 mm size with Peradeniya Organophosphorus Poisoning (POP) Scaled as moderate poisoning.

Her neck muscles weakness was first manifestation followed by proximal muscles weakness of upper and lower limbs. Her Urea was 17, Creatinine: 03 mg/dl, Total count: 11400 mg/dl, PT/INR: 14/1, SGPT: 14 U/L, SGOT: 15 U/L, GGT: 30 U/L. Her serum acetyl cholinesterase level at index visit was 165 U/L.ABG and Urine routine examination revealed within normal limits.

The patient was transferred to Intensive Care Unit (ICU) and was managed conservatively with intravenous fluids, Pralidoxime and Atropine. The patient condition improved in ICU andwas discharged on 7th day with normal regain of power of neck and limb muscles.

Case 2

A 41-year old lady was brought to Emergency Department by her spouse with alleged history of ingestion of organophosphate compound (Chlorpyriphos 50% and Cypermethrin 5%) ten days prior to admission. She was managed at local hospital and then was referred to emergency ward of BP Koirala Institute of Health Sciences (BPKIHS) for further management. She had history of vomiting multiple episodes, abnormal body movements and dizziness at the index visit in Emergency ward.

On examination her Glasgow Coma Scale (GCS) was E2 M3 V3, Blood Pressure: 150/80 mm Hg, Heart Rate: 105/min, Respiratory Rate: 28/m, SpO2: 89% at room air. Pupils were less than 2 mm size with Peradeniya Organophosphorus Poisoning (POP) Scaled as moderate poisoning. Weakness was noticed in proximal muscles of upper and lower limbs. Her Urea was 10, Creatinine: 1.2 mg/dl, Total count: 24200 mg/dl, PT/INR: 14/1, SGPT: 39 U/L, SGOT: 29 U/L, GGT: 12 U/L, Lipase 149U/L and Amylase was 232U/L. Her serum acetyl cholinesterase level was 139 U/L. On ABG (Arterial Blood Gas) her PH was 7.44, PCO2 32.1, PO2 80.5, Lactate 1.0 and HCO3 was 21.8.Urine routine examination and ECG revealed within normal limits. The patient was conservatively with intravenous fluids, atropine and pralidoxime and Benzodiazepines. Airway was managed by endotracheal intubation. On the 3rd day patient was expired.

Case 3

An 18-year old married female was brought to our Emergency Department by her father with alleged history of ingestion of

unknown compound 5days ago. She was admitted and managed at local private hospital for last three days with intravenous fluids and atropine1.8mg bolus. When the symptoms did not improve she was then referred here for further management. She had history of vomiting and unable to hold her neck. On examination her Glasgow Coma Scale (GCS) was E4 M4 V4, Blood Pressure: 110/80 mm Hg, Heart Rate: 68/min, Respiratory Rate: 28/m, SpO2: 91% at room air. Pupils were less than 2 mm size with Peradeniya Organophosphorus Poisoning (POP) Scaled as mild poisoning. Neck holding weakness was noticed and also in proximal muscles of upper limbs.

Her Urea was 19, Creatinine: 0.6 mg/dl, Total count: 15000 mg/dl, PT/INR: 14/1, SGPT: 39 U/L, SGOT: 29 U/L, GGT: 12 U/L, Lipase 149U/L and Amylase was 232U/L. Her serum acetyl cholinesterase level was 1320 U/L (Reference ranged 3167- 6333U/L). On ABG (Arterial Blood Gas) her PH was 7.39, PCO2 35.2, PO2 100, Lactate 0.8 and HCO3 was 21.3.Urine routine examination revealed Albumin ++; Sugar : negative; Pus cells: 0-1/hpf; RBC: plenty. ECG revealed normal sinus rhythm.

She was been managed in emergency ward with intravenous fluids, Atropinized with 25 ampoules and Pralidoxime 2 grams stat. The patient was followed up in medical ward and was managed conservatively with same medication. The patient condition improved and was discharged on th day with normal regain of power of neck and limb muscles.

Discussions:

Intermediate syndrome is a major cause of morbidity and mortality in patients with acute organophosphate insecticide poisoning. It is well recognized as a disorder of neuromuscular junctions but its exact etiology, incidence, and risk factors are not clearly defined because a previous study are largely small-scale case series and was irrelevant for a consistent and rigorous definition of IMS.[13]

IMS has been commonly associated with organophosphate compounds like diazinon, dimethoate, methylparathion, methamidaphos, monocrotophos, fenthion and ethylparathion which are quite different in our study that is chlopyriphos.[14, 19]

In one study done by Aygun D et. al. 2002 found that the level of plasma acetylcholinesterase level poorly correlated in IMS. 15.There may be a considerable variations between the methods used for estimation of IMS and plasma acetylcholinesterase level to detect its toxicity. 16 Therefore, serial estimations of plasma acetylcholinesterase may be more useful in diagnosis of Intermediate Syndrome.

Involvement of Cranial nerve palsy as reported from Srilanka and the involvement of proximal muscles in other studies were quite similar to our study. In most of the study found that mortality varied form 10.5% to 41.6%. [2, 17- 18]

From the study done by Vikramet. al, 2005 the time taken for recovery from the manifestations varied from 3-12 days which was quite consistent with our case report who completely recovered and discharged on 7th day. [19]

Conclusion:

In a view of high mortality early recognition and supportive therapy remains the cornerstone of the patient of Intermediate Syndrome. It should always be borne in mind in treating physician during treating organophosphate poisoning patient. The appropriate treatment protocol must be established in every Emergency Ward of district and private hospitals in Nepal.

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