British Journal of Medicine & Medical Research
5(2): 286-288, 2015, Article no.BJMMR.2015.030
ISSN: 2231-0614

SCIENCEDOMAIN *international*
www.sciencedomain.org

Monomelic Amyotrophy after Permethrin Poisoning

Dash Sandip Kumar[1*]

[1]*Department of Neurology, Apollo Hospital, Dhaka, Bangladesh.*

Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

Article Information

DOI:10.9734/BJMMR/2015/11574
Editor(s):
(1) Kishore Kumar Hotha, Novel Laboratories, Inc., Somerset, NJ 08873, USA.
Reviewers:
(1) Selma Yazar Urek, Department of Pharmacology and Toxicology, Faculty of Pharmacy, Yeni Yuzyil University, Turkey.
(2) Anonymous, Eötvös Loránd University, Hungary.
(3) Anonymous, University of Pisa, Italy.
(4) Anonymous, Meharry Medical College, USA.
Peer review History: http://www.sciencedomain.org/review-history.php?iid=661&id=12&aid=6016

Case Study

Received 23rd May 2014
Accepted 13th August 2014
Published 9th September 2014

ABSTRACT

Monomelic amyotrophy (MMA) known as Hirayama disease (HD). The first report appeared in 1959 when Hirayama described 12 patients [1] then, a large group of patients was found from Japan [2,3]. MMA from South India was also reported in 1984 [4]. The disease accounted for males over 80% of cases, especially between 15 and 25 years of age. The disorder has been recognized as, unilateral or bilateral asymmetric atrophy of hand and forearm with sparing of brachioradialis giving the characteristic appearance of oblique amyotrophy. Symmetrically bilateral disease has also been recognized. It is believed to be a cervical flexion myelopathy [5].
Pyrethroids are used as insecticides due to their high potency. These are highly toxic to a wide range of insects but have low toxicity to humans. Pyrethroids are known to cause neurotoxicity in humans like seizures, tremors, and dizziness. Motor neuron damage has been reported in acute toxicity due to ingestion of pyrethroids and organochlorines [6] and MND (Motor neuron disease) like features after chronic exposure has been reported [7]. Here a case of monomelic amyotrophy following massive ingestion of permethrin, amytriptyline and benzodiazepine tablets is reported.

Keywords: Monomelic amyotrophy; Hirayama disease; permethrin; Motor neuron disease.

**Corresponding author: Email: drskdash@yahoo.co.in;*

1. INTRODUCTION

A 22 year male ingested 160 gr of Finis insecticidal powder, containing active ingredient Permethrin 0.5%, 20 tablets of 10 mg of amitriptyline and 10 tablets of 5mg of diazepam for committing suicides and was found in a comatose state in his room after 24 hours, before hospitalization. After 5-7 days of symptomatic treatment (the details of treatment not available), he was discharged without any deficit. After 2-3 weeks of discharge from the hospital he noticed gradual weakness of his right upper limb, which slowly progressed to thinning of small muscles of his right hand with weakness .On examination there was moderate weakness of right upper limb with atrophy of first dorsal interosseous, thenar muscles, biceps, triceps and supraspinatus, with areflexia on right upper limb without any sensory loss and plantar reflex of both right and left side were flexor. There was no visible fasciculation, sensory deficit or autonomic involvement. Nerve conduction studies revealed no response to stimulation in right ulnar nerve and F wave was not obtained from right ulnar nerve. Erb"s point stimulation revealed reduced CMAP (Compound Muscle Action Potential) from first dorsal interosseous (FDI) (see Fig. 1), biceps brachii, deltoid, and supraspinatus on right side. Sensory nerve conduction was normal. Electromyography (EMG) revealed fibrillation in abductor digiti minimi, fibrillation and fasciculations in first dorsal interosseous, reduced interference pattern in abductor pollicis brevis, biceps muscle on right side, suggesting monomelic amyotrophy on the right upper limb involving right C5 –TI segments. His brain and cervical spine MRIs were normal. Routine blood tests were normal except for elevation in serum glutamate pyruvate transaminase (SGPT), serum glutamic-oxaloacetic transaminase (SGOT). He was treated with coenzyme Q, vitamins and physiotherapy. After 4 weeks his weakness in the right upper limb improved although the muscle atrophy persists, but there was no further progression of the disease. At 4 months follow up there is no progression of the disease either clinically or EMG wise, and muscle power has improved.

Pyrethrins are obtained by extracting dried pyrethrum flowers. Pyrethroids are classified into two classes based on their manifestations produced by toxic doses on animals. Type I poisoning syndrome is called tremor or T syndrome [8]. Type II poisoning syndrome is known as choreoathetosis/salivation or CS syndrome [8]. Pyrethroids disturb ion channels like voltage gated sodium channels and voltage sensitive chloride channels and cause neuronal excitation and impaired axonal transport [9]. Due to their action on sodium channels, there is increased Ca^{++} influx causing synaptic excitotoxicity due to excessive glutaminergic neurotransmission [10]. Pyrethroids are metabolized via Cytochrome P 450 enzyme system and also by esterase cleavage, following which two products are formed, which are further metabolized then eliminated [11]. Some humans are known to be poor metabolizers, and are prone to suffer motor neuron death. In our case, excessive ingestion of diazepam might have ameliorated type II syndrome possibly by facilitating inhibitory pathways through its action at the GABA receptor. Some people are thought to be poor metabolizers of pyrethroids and carboxyl esterase inhibitors can enhance pyrethroids toxicity. Amitriptyline is also metabolized by cytochrome P450 system. Cytochrome P450 activity depends on various factors including genetic constitution. Polymorphism in enzymes is an important factor in the individual variability in metabolic capacity [12]. It is speculated that as both permethrin and amitriptyline are metabolized in the same pathway, there may be drug interaction causing slowing degradation of permethrin, thereby causing death of motor neurons, leading to monomelic amyotrophy in this patient.

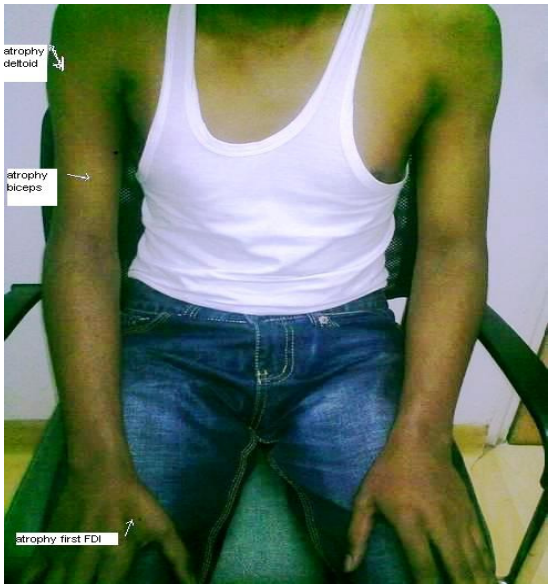

Fig. 1. Showing atrophy of first dorsal interosseous (FDI), deltoid and biceps on right upper limb

2. CONCLUSION

Here a case of monomelic amyotrophy of right upper limb following massive ingestion of permethrin, amytriptyline and benzodiazepine tablets is reported. The amyotrophy developed 2-3 weeks following the ingestion of the offending agent and the probable cause of motor neuron damage is discussed.

CONSENT

The patient has given informed and written consent for the case report to be published.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. Hirayama K, Toyokura Y, Tsubaki T. Juvenile muscular atrophy of unilateral upper extremity – a new clinical entity. Psychiatr. Neurol. Jpn. 1959;61:2190–7.
2. Hirayama K, Tsubaki T, Toyokura Y, Okinaka S. Juvenile muscular atrophy of unilateral upper extremity. Neurology. 1959;13:373–80.
3. Sobue I, Saito N, Iida M, Ando K. Juvenile type of distal and segmental muscular atrophy of upper extremities. Annals of Neurology. 1978;3:429–32.
4. Gourie-Devi M, Suresh TG, Shankar SK. Monomelic amyotrophy. Archives of Neurology. 1984;41(4):388–394.
5. Chen C, Chen C, Wu C, Ro L, Chen S, Lee T. Hirayama disease: MR diagnosis. American Journal of Neuroradiology. 1998;19(2):365–368.
6. Doi H, Kikuchi H, Murai H, Kawano Y, ShigetomH, Ohyagi Y, Kira J. Motor neuron disorder simulating ALS induced by chronic inhalation of pyrethroid insecticides. Neurology. 2006;67:1894-1895.
7. Pall SH, WilliamsAC, Waring R, Elias E. Motorneuron disease as manifestation of pesticide toxicity. Lancet. 1987;19:685.
8. Verschoyle RD, Aldridge WN. Structure activity relationship of some pyrethroids in rats. Arch Toxicol. 1980;45:325-329.
9. Rey DE, Fry JR. Reassessment of the neurotoxicity of pyrethroid insecticides. Pharmcol Ther. 2006;11:174-193.
10. Sen I, Joshi DC, Joschi PG, Joschi NB. NMDA receptor mediated differential Ca^{++} load and greater vulnerability of motor neuronsin spinal cord cultures. Neurochem Int. 2008;52:247-255.
11. Steventon GB, Waring RH, William AC. Pesticide toxicity and motor neuron disease. J Neurol Neurosurg Psychiatry. 1990;53:621-622.
12. Weide J vander, Hinrichs John WJ. The influence of cytochrome P450 pharmacogenetics on disposition of common anti depressant and anti psychotic medication. Clin Biochem Rev. 2006;27:17-25.

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sciencedomain.org/review-history.php?iid=661&id=12&aid=6016