Non-convulsive Status Epilepticus Secondary to Thiamethoxam Poisoning

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ABSTRACT

Introduction: Neonicotinoid compounds are newer group of neuro-active insecticides, widely used for crop protection. They are generally having a favourable toxicity profile in humans because of selective affinity to receptors in insects. However, fatal cases and severe toxicity has been reported in humans.

Case Reoprt: We report a case of 55-year-old male who developed non-convulsive status epilepticus (NCSE) following ingestion of Thiamethoxam (neonicotinoid compound). The patient was consumed 250 mL of Thiamethoxam and brought to emergency department in an unconscious status. Continuous electroencephalography monitoring showed, recurrent non-convulsive generalised electrographic seizures (EGS) occurring at frequency of one EGS per 3-4 minutes with altered sensorium despite not on sedation. He subsequently developed intensive care unit (ICU) acquired weakness and tracheostomised in view of difficult weaning. After 14 day of treatment, the patient was directly discharged from the ICU at stable condition.

Conclusion: This case report emphasises the importance of monitoring NCSE in patients with disorientation. It is desirable to screen for NCSE in patients with neonicotinoid compound poisoning and altered sensorium.

INTRODUCTION

Neonicotinoid compounds are newer group of neuro-active insecticides, widely used for crop protection from insects and pests. It acts on nicotinic acetylcholine receptors, initially it stimulates the agonized receptors and interferes with the transmission of neuronal impulses by fatigue.[1,2] This effect influences the central nervous system and results in dizziness, drowsiness, disorientation and coma. The autonomic nervous system is stimulated through a similar mechanism, and may lead to coronary spasm and cardiac ischaemia, followed by nervous system paralysis. As a result, poisoned patients may present with arrhythmia, hypotension and bradycardia.[3] Mammalian neurotoxicity is less known with neonicotinoids and mostly described with imidacloprid. Here we report a case of non-convulsive status epilepticus (NCSE) secondary to Thiamethoxam ingestion.

CASE REPORT

A 55-year-old male was brought to our emergency department in an unconscious status. According to his

relatives statement, the patient was consumed 250 mL of Thiamethoxam 30% (Neonicotinoid Insecticide; Brand name: Bheema), which consisted of solvents 4.30% w/w Glycerine, Polyethylene Glycol, 1,2 benzisothiazolin 3-one. The container was identified, the time of consumption was not clear. He had few episodes of convulsive seizures, before reaching the hospital. Airway was secured, and invasive mechanical ventilation was initiated. Convulsive seizures were under control after initiation of intravenous Levetiracetam 2g/day. However, on continuous electroencephalography (EEG) monitoring patient was noted to have recurrent non convulsive generalised electrographic seizures (EGS) occurring at frequency of one EGS per 3-4 minutes with altered awareness despite while off sedation. Considering NCSE, patient was initiated on Fosphenytoin. As patient was going on to refractory NCSE, he was tried on propofol infusion up to 4mg/kg/hr with no control of EEG seizures (Figure 1 and 2), later he was switched over to midazolam infusion. With midazolam (2mg/kg/h) the seizure was under control. After 24h of EGS control, midazolam was slowly tapered and switched over to clobazam 20mg/day. Magnetic resonance imaging (MRI) of brain showed features suggestive of post ictal signal changes in the temporal lobe with no other radiological abnormality. Patient sensorium was recovered completely over 48h after stopping midazolam and is being maintained on 3 antiepileptic drugs which includes levetiracetam, phenytoin and clobazam. A repeat EEG showed complete resolution of EGS (Figure 3). In view of poor cough reflex with intensive care unit (ICU) acquired weakness he was electively tracheostomised to facilitate gradual weaning from ventilator. Patient had mild critical illness related myopathy which completely resolved in a week, paralytic ileus resolved after 7 days with prokinetics and electrolyte correction. He developed secondary infection on day10, and blood culture showed an Enterobacter cloaca which was treated with Meropenem. He was discharged at stable condition with tracheostomy tube in situ in view of mucosal edema and dysphagia proven with Functional endoscopy swallow study. Patient was reviewed on day 21 for follow up, after swallow assessment he was decannulated and discharged.

DISCUSSION

There are currently seven members in the class of neonicotinoid insecticides, which include imidacloprid, acetamiprid, clothianidin, thiacloprid, dinotefuran, nitenpyram and thiamethoxam. Thiamethoxam is a systemic insecticide in the class of neonicotinoids, it has a broad spectrum of activity against many types of insects. Generally, mammalian neurotoxicity is less known with neonicotinoids and mostly described with imidacloprid. Thiamethoxam is relatively a better choice of insecticide compared to organophosphorus in terms of prevention of acute poisoning. Few case reports of attempted suicides have described signs of possible neurotoxicity such as drowsiness, dizziness, disorientation, impaired papillary function, hypothermia, tremors.[4,5] PEI-CHEN LIN et al, in their review, reported 152 cases of neonicotinoid poisoning through different routes. Out of whom 21 (13.8%) cases were severe and 110 (72.4%) were non-severe cases due to oral ingestion of neonicotinoid.[6] The most common route of intoxication was oral route and the amount of ingestion correlated with severity. Imidacloprid was the most common neonicotinoid compound consumption. NCSE is a clinico-electrophysiological diagnosis, Salzburg criteria being the most accepted one for diagnosis of NCSE.[7] In the setting of a non-primary neurological patient, it may be easily overlooked as it requires high index of suspicion. The etiological spectrum of NCSE is as wide as that of convulsive status epilepticus. The etiology may be broadly classified into NCSE in patients with or without established epilepsy. The latter setting may especially be more challenging especially since there is another underlying pathology and seizure is a symptomatic phenomenon rather than the primary issue in these cases especially wherein it may be difficult to say whether the altered sensorium is due to primary pathology or due to seizure. If remained unrecognised, NCSE can potentially cause significant neurological significant sequelae.[4] In our present case, on day 2 patient remained unresponsive, hence EEG monitoring was done which showed NCSE. After instituting appropriate anti-epileptics, patient sensorium was improved.

CONCLUSION

NCSE is a rare phenomenon with neonicotinoid poisoning. This case report emphasises the importance of monitoring for NCSE in patient with no improvement in sensorium after initial treatment. It also highlights the role of high clinical suspicion in cases of even the so called 'safe' neonicotinoids insecticide poisoning, especially to monitor for NCSE.

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Fig 1: Right hemispheric onset of epileptiform discharges noted on an electroencephalogram.

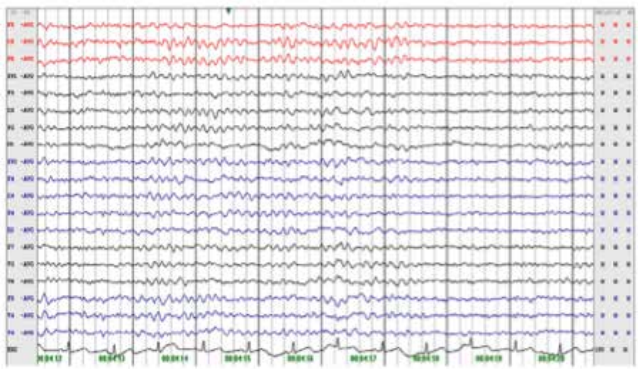


Fig 2: Right hemispheric onset of epileptiform discharges evolving into electrographic seizures with bihemispheric epileptiform discharges (more prominently right hemispheric) noted as above on an electroencephalogram.

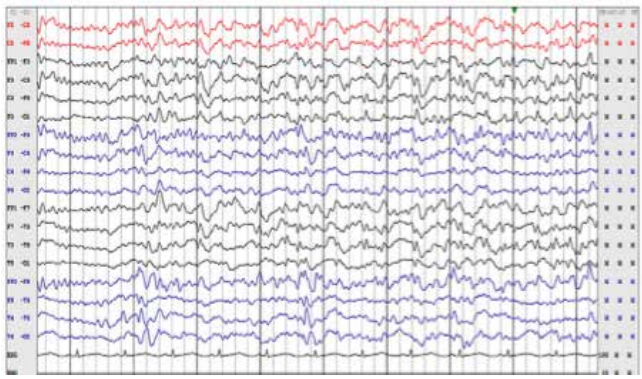
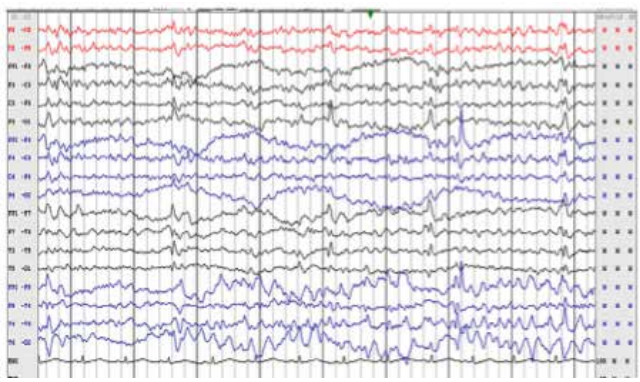


Fig 1: An electroencephalogram after treatment of NCSE showed, resolution of the electrographic seizures and epileptiform discharges and showed a Delta 1 background activity.



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