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An unusual case of organophosphate poisoning presenting Wellens or Pseudo-Wellens pattern: A diagnostic dilemma

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ABSTRACT

Organophosphate compounds such as Malathion, parathion, dimethoate, chlorpyrifos, tabun etc. Block the acetylcholinesterase enzyme, due to which acetylcholine levels increases. Organophosphate poisoning can occur after dermal, respiratory or oral exposure to organophosphate compounds. Respiratory symptoms are presented frequently in cases of organophosphate poisoning. Salivation, rhinorrhea along with bronchorrhea and bronchospasm are the muscarinic effects contributing to hypoxemia and increase in respiratory rate. Nicotinic effect causes muscular fragility and paralysis which may lead to respiratory failure. In wellens syndrome, the ECG reveals biphasic T waves or deep inverted T waves in leads V2-V3, indicating LAD stenosis, whereas Pseudo-Wellens have a Wellens ECG pattern but normal coronary architecture. In this case a 29-year-old male patient was brought from outside hospital with alleged history binge alcohol consumption and was found in unconscious state in his farm 2 days back.

Keywords: Wellens pattern, pseudo-wellens, LAD stenosis, alcohol, unconscious

1. INTRODUCTION

Poisoning by pesticides continues to be a major global public health issue. More than 250,000 people die each year from pesticide poisoning, according to estimates from the World Health Organization. Due to their high toxicity organophosphate insecticides remains the most common culprit amongst the various pesticides that results in death (Yang and Deng, 2007). Pesticide poisoning has become a major concern in India since 1958, when 100 people died after eating flour laced with the organophosphate insecticide. According to a study, in India, an estimated 230000 people commit themselves each year, from which at least 30% are pesticide suicides (Karunarathne et al., 2021). Most poisoning cases result from intentional consumption, inhalation or

cutaneous absorption. Toxicity can also occur by self-injection, intramuscularly or intravenously (Pandit et al., 2011). All organophosphate insecticides are capable of producing acute toxic symptoms. The muscarinic, nicotinic and central nervous system actions of acetylcholine accumulation precipitate the signs of acute poisoning. Depression of the CNS and fasciculations of skeletal muscles are found in severe cases. Bronchoconstriction, increased bronchial secretions, increased gastrointestinal motility and bradycardia are also observed (Wilson, 1979). Organophosphate stimulates cholinergic and nicotinic receptors by inhibiting the activity of acetylcholinesterase. Atropine is required to reverse cholinergic effects whereas oximes are needed for neurological manifestations, which are mediated through nicotinic receptors (Zafar et al., 2017).

ECG manifestations in organophosphate poisoning are as follows:

- Prolonged Q-Tc interval, Sinus tachycardia, sinus bradycardia, ST elevation, T wave inversion, first degree heart block that is P-R interval > 0.20 sec, Atrial fibrillation, Ventricular tachycardia (Paul and Bhattacharyya, 2012).
- The T-wave changes in the precordial leads along with proximal LAD artery stenosis are common components seen in Wellens syndrome resulting in sudden cardiac death or acute myocardial infarction (Wang et al., 2018).
- Pseudo-wellens syndrome is rather a rare presentation having the same ECG changes as wellens syndrome but without LAD artery stenosis (Yurumez et al., 2007).

2. CASE STUDY

A 29-year-old alcoholic male patient was brought to emergency medicine department by his brother, intubated from outside hospital in ambulance with alleged history of binge alcohol consumption and was found in unconscious state in his farm 2 days back.

As narrated by his brother, patient was seen alright 3 days back when he left his home and had history of binge alcohol consumption and was missing for 1. 5 days, found lying unconscious in his farm. Patient was taken to nearby hospital where he was found to be hypoglycemic and regained consciousness after receiving 100 ml of injection dextrose 25 %. Patient was admitted and monitored. Patient had episode of seizures during night and was intubated. CT brain and CSF analysis were done which were reported to be normal (Figure 1). Patient was referred to tertiary center the next morning.

Figure 1 CT brain plain

On presentation to AVBRH, patient was intubated and on mechanical ventilator with volume controls mode maintaining 100 % O_2 saturation. Bilaterally chest was clear on auscultation. Recorded pulse rate was 112/minute and BP was 130/80mmHg with Glasgow coma score E2V1M5 and random blood Sugar–127mg / dl.

Patient had a history of 1st episode of seizure at the age of 6 years. He also had a history of fecal and urine incontinence. There was no history of trauma, consumption of any substance or ischemic heart disease.

On systemic examination

Patient was unconscious; pupils were constricted, 1mm non-reactive to light with excessive lacrimation. On auscultation bilateral air entry was equal on both sides with S1, S2 normal. Abdomen was soft and non-tender on palpation.

Table 1 Representing reflexes

Reflexes	Right	Left
Plantar	Decreased	Decreased
Ankle	Hyporeflexia	Hyporeflexia
Knee	Hyporeflexia	Hyporeflexia
Biceps	Hyporeflexia	Hyporeflexia
Triceps	Absent	Absent

Investigations

Blood investigations were indicative of the following: Urea-19mg/dl, creatinine-0.6mg/dl, sodium-146mmol/L, Potassium-3.4mmol/L. ALT-52U/L, AST-99U/L, Alkaline phosphatase-71U/L, Total protein count-5.2 g/dl, Albumin-2.8g/dl Globulin-2.4g/dl, Total Bilirubin-0.8mg/dl. CKMB-43U/L. hsTroponin-l-571.2 pg/ml,

Serum cholinesterase levels were found to be 0.3U/ml.

Complete blood count with peripheral smear and coagulation profile was found to be within normal limits.

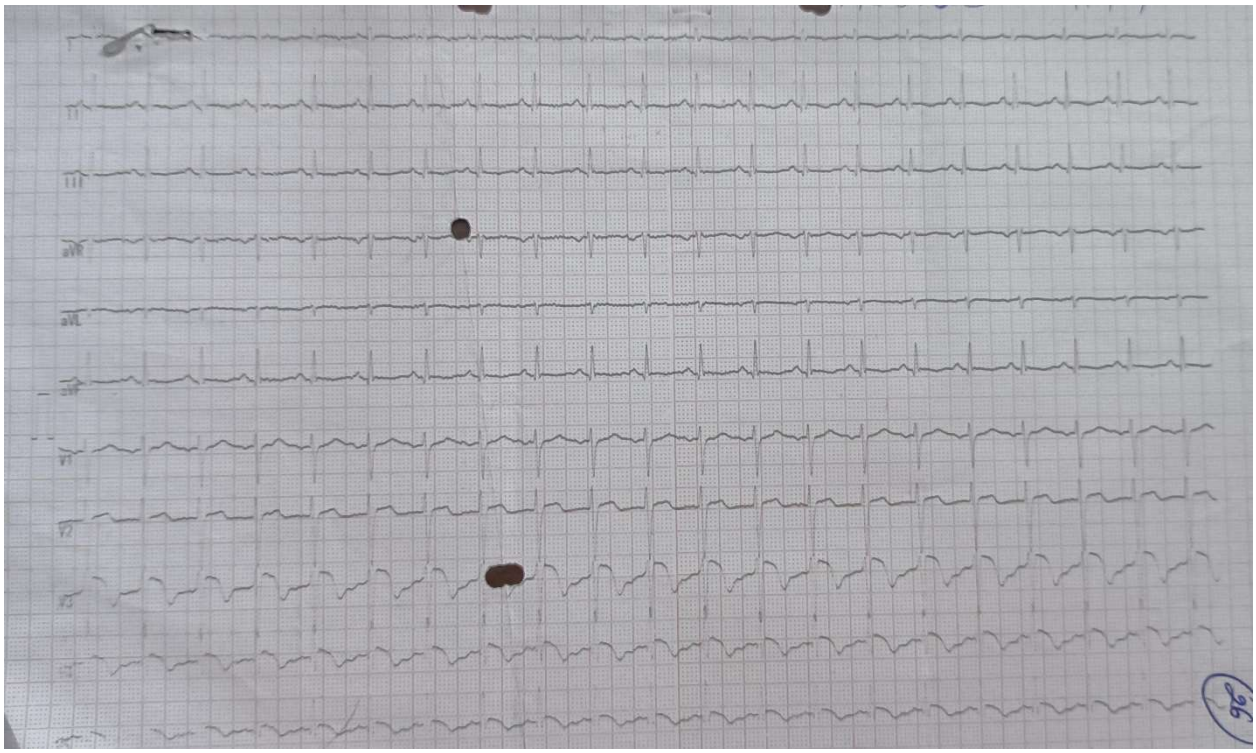

Figure 2 ECG changes

According to the cardiologist, an ECG was advised and revealed a biphasic T wave in leads V2, V3, V4 and V5, indicating a left anterior descending artery lesion (Figure 2). 2D echo was done which was not significant.

Treatment

Treatment given in EMD: Patient was started on Inj. Thiamine 500 mg IV, Inj. Levetiracetam 1gm IV, Inj. Ondansetron 4mg IV, Inj. Tazobactum 4.5gm IV, Inj. B-compplex 1 ampule IV BD, patient was given Tab. Aspirin 300mg, Tab. Clopidogrel 300mg, Tab.

Atorvastatin 80mg, Inj. Unfractional Heparin 2000IU bolus slowly every 6hourly. Ryle's tube insertion was done. In view of serum cholinesterase levels, Patient was started on Inj. Atropine in divided doses, followed by Inj. Atropine infusion at the rate of 2ml/hour and Inj. Pralidoxime 2gm IV stat, followed by Inj. Pralidoxime infusion at the rate of 8mg/kg/hour. Patient vitals improved and thus was shifted to medicine ICU.

Treatment given in medicine ICU: Patient was started on IV antibiotics, Inj. Tazobactum 4.5 gm IV tds, Inj. Levofloxacin 500mg IV OD, Inj. Pantaprazole 40 mg OD, Inj. Ondensetron 4mg IV tds, Inj. Thiamine 100 mg IV tds, Inj. D25 IV, Inj. levetiracetam 500 mg IV BD and other supportive measures. Patient was kept on mechanical ventilator. During the course of hospital stay, patient's condition was not stable. He went into sudden cardiorespiratory arrest and cardiopulmonary resuscitation was done according to ACLS protocol. Inspite of all resuscitative measures patient could not be revivied and was declared dead.

3. DISCUSSION

This patient was found lying unconscious in his farm, with inadequate history narrated by the brother. His CT brain, CSF examination and complete blood profile were found to be normal but pupils were contstricted and lacrimation was present, so serum cholinesterase was sent which was decreased suggesting OP poisoning. Diagnosis of acute OP poisoning is based on the history of exposure as well as red cell and plasma cholinesterase levels. RBC cholinesterase is a true cholinesterase but cannot be measured quickly, although serum cholinesterase is a pseudocholinesterase, it can be measured quickly in emergencies. More than 50% of serum cholinesterase must be suppressed before clinical symptoms start to appear. Current therapies for organophosphate poisoning include decontamination, atropine reversal of muscarinic symptoms, pralidoxime regeneration of acetylcholinesterases, and supportive pulmonary care. Inadequate atropinization is the most prevalent reason of therapy failure (Yurumez et al., 2007). When ECG of the patient was done in leads V2, V3, V4 and V5, a biphasic T wave was observed, which was suggestive of left anterior descending artery stenosis. As our patient was young and there was no history of any ischemic heart disease or other comorbidities, there may be chances of ECG showing pseudowellens pattern but this cannot be justified without coronary angiography. However, patient died within 12 hours so coronary angiography could not be done. When clinical signs and ECG changes are distinctly suggestive of myocardial injury, immediate non-invasive coronary angiography may be the best approach to make early diagnosis. In 1979, Gerson and associates obsereved inverted terminal T-waves in the precordial leads in individuals with proximal LAD stenosis induced by exercise and were the first to describe ECG abnormalities associated with Wellens syndrome (Ju et al., 2021). Various factors can cause wellens syndrome, which may include hypoxia, coronary artery disease, increased myocardial demand and coronary vasospasm. Wellens pattern can be classified as type A that is characterized by biphasic T wave and type B which shows deep T wave inversion in leads V2 and V3 (Milne et al., 2022). A clinical entity with wellens ECG pattern but with normal coronary anatomy is said to be Pseudo-wellens syndrome, seen in patients with myocardial bridge due to external coronary artery compression or coronary spasm from coccaine (Ola and Tak, 2019). The higher incidence of ECG changes in patients with moderate or severe poisoning may be considered as evidence that some ECG changes could be directly related to cholinesterase inhibition (Dalvi et al., 1986). In fact, there can be a wide range of anomalies on the ECG in cases of acute organophosphate poisoning. These so-called "toxic repolarization abnormalities," which include pathological repolarizations such as diffuse ST and T-inversion abnormalities and QTc prolongations, were seen in a large series. These ECG abnormalities are frequently present and eventually result in ventricular tachycardia know as torsade de pointes. Furthermore, depending upon the amount of the intoxication, these spectacular ECG changes were recorded from first to twentieth day which mimics that of severe ischemia. A theory has been suggested that there are distinct phases; including a brief period of intensely elevated sympathetic tone that causes severe tachycardia, which is succeeded by a protracted interval of increased parasympathetic activity, which results in sinus bradycardia, atrioventricular block, ST segment and T wave abnormalities and QTc interval broadening. As we know, QTc prolongation results from strong and uneven sympathetic stimulation of myocardial fibres making it the main reason that we observe these ECG changes in acute cases of OP poisoning with autonomic discharge or altered consciousness (Jorens et al., 2008).

4. CONCLUSION

Appropriate use of organophosphate compounds, public awareness in reference to their dreadful effects and restrictions of their unauthorised sales can help to minimise the prevalance of OP poisoning. Furthermore, being aware of the consequences in these cases, early investigations should be encouraged because an early diagnosis and course of treatment may enhance the patient's prognosis.

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Author Contributions

Jeenat Khan, Gajanan Umalkar and Akhilesh Singh contributed in selection of case and drafting the manuscript, Charuta Gadkari guided us in perfecting the manuscript.

Informed consent

Written and oral informed consent was obtained from the patient included in the study.

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data collected during this study are available upon reasonable request from the corresponding author.

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