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Research Article

Organophosphorus Poisoning In Pregnancy. A Case Report and Review of Literature

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ABSTRACT
Organophosphorus poisoning is a significant public problem specially in developing countries where domestic abuse, poverty and gender inequity is a matter of great concern. Organophosphorus which is present in pesticides is easily accessible in developing countries for homicidal as well as suicidal cases. A case of 26-year-old intubated primi (G1POLO) at 34 weeks gestation presented in emergency with history of domestic fight followed by some pesticide intake 24 hours back.
Patient was intubated at some local hospital due to respiratory depression. There was history of multiple episodes of vomiting, excessive salivation, sweating followed by unconsciousness.
Patient was given Intravenous atropine to reverse the cholinergic side effects. Her plasma pseudocholinesterase levels were also very low (150 lU) which further confirmed the diagnosis. Intravenous infusion atropine dose continued for a duration of 5 days till full recovery was achieved. Patient was discharged in stable condition and delivered vaginally at 37 weeks of gestation. There were no residual neurological defects in fetus as well as mother.

Keywords: Organophosphorus Poisoning, Pregnancy.
Abbreviations- OP- organophosphorus poisoning,

INTRODUCTION

Pesticide misuse in homicide and suicidal cases is common in developing countries due to easy accessibility. Pesticides cause 2 million suicidal cases worldwide annually. Use of organophosphorus in pregnancy as suicidal or homicidal agent is scarcely reported in literature hence the importance of this case report. The diagnosis is made after history taking, signs and symptoms and ultimate confirmation is made by low. plasma pseudocholinesterase levels. Most common organophosphorus group implicated is Diazinon 60 EC 2. [1]
Organophosphorus inhibits the cholinesterase enzyme which results in excessive cholinergic activity and respiratory depression being the catastrophic event.3 The antidote for cholinergic symptoms is atropine and is also considered the treatment of choice in pregnant women.?. The use of Pralidoxime in cases of pregnant women lacks standardization and very few case reports have mentioned their safety in pregnancy. [2]

CASE REPORT

A 26-year-old G1P0L0 at 34 weeks came to emergency department with history of marital discord and fight followed by self-consumption of kalla nag (chlorpyriphos and pyremethrin) which was taken 10 hours back. After the intake there were multiple episodes of vomiting, excessive salivation, sweating followed by sudden unconsciousness. Patient was rushed to nearby hospital where she was intubated in view of severe respiratory depression and was referred to the tertiary hospital for further follow up. On arrival, patient pupils were meiotic and was on ventilatory support at (FI02 60%,PEEP-6) maintaining saturation of 98%, respiratory rate 26 / minute, pulse 150 /minute, blood pressure was 110 / 70mm HG, temperature 101 degree Farenheit. Basal crepitus was

present on auscultation. On abdominal examination uterus was 34 weeks, relaxed, cephalic, FHS 140/minute and the os was closed.

Catheter bag showed urine output as 700 ml. USG showed single live foetus of 34 weeks, placenta anterior, liquor adequate with normal colour doppler. Her symptoms were highly suggestive of organophosphorus poisoning and hence atropinization was started at a dose 1.2 mg followed by infusion at 0.6 mg/hr which continued for 5 days until complete reversal of atropinization was attained. Her plasma pseudocholinesterase levels at admission was 200 IU which increased to 500 on day 5. Other supportive measures like betnasol coverage for lung maturity in anticipation of threat of preterm delivery and intravenous Lasix was also given to clear lung congestion. Tapering of ventilatory support started after 12 hrs and patient was on room air on day 5 of admission with improving lung function. Both mother with baby were discharged in stable condition on 10 days. On follow up the patient went into labour at 39 weeks and delivered healthy baby of 3 kg vaginally with good APGAR score. There were no neurological deficits observed in the neonate.

DISCUSSION

Organophosphorus (OP) poisoning causes inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at nerve synapses and resultant overstimulation of both muscarinic and nicotinic receptors [4]. According to the World Health Organization (WHO) classification of pesticides, chlorpyrifos and pyrimethamine are categorized under slightly hazardous Class III compounds [5]. Pregnant women are particularly vulnerable due to physiological changes such as increased cardiac output, altered respiratory dynamics, and enhanced renal clearance, all of which may influence the absorption and metabolism of OP compounds. Additionally, reduced plasma cholinesterase levels during pregnancy may exacerbate toxicity, thereby complicating management [6]. Psychological stressors, including family pressure and peripartum depression, contribute significantly to suicidal tendencies, with OP compounds being among the most readily accessible agents, accounting for more than 200,000 deaths annually worldwide [7].

Clinical manifestations include nausea, vomiting, excessive secretions (bronchorrhea, urination, diarrhea), headache, confusion, miosis, bradycardia, respiratory abnormalities (tachypnea or bradypnea), blood pressure fluctuations, and in severe cases, convulsions, coma, and death [3]. OP poisoning typically progresses through three phases: acute cholinergic crisis, intermediate syndrome, and delayed neuropathy [8]. These compounds can be absorbed via inhalation, dermal exposure, and gastrointestinal routes. Since the clinical presentation is predominantly cholinergic, atropinization remains the cornerstone of treatment, with atropine administered until signs of adequate atropinization are achieved, as in the present case [9].

Pralidoxime, an acetylcholinesterase reactivator, acts at the nicotinic receptor site and alleviates symptoms such as muscle weakness and paralysis if administered within 48 hours; however, it was not used in this case. To date, no significant adverse effects of pralidoxime use during pregnancy have been consistently reported [10]. Exposure to OP compounds in the third trimester has been associated with adverse fetal outcomes including preterm labor, miscarriage, fetal growth restriction (FGR), attention deficit hyperactivity disorder (ADHD), and cognitive impairment.

CONCLUSION

Organophosphorus poisoning in pregnancy has a favourable outcome in most cases if timely diagnosis and management is done. Atropine is the cornerstone for reversal of cholinergic features. Low Plasma pseudocholinestrase levels are the hall mark of OP poisoning. Use of Pralidoxime in pregnant women with OP poisoning needs more exploration and validation. Long term effect of OP poisoning on neonates needs to be explored and studied.

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