Case Report

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Anaesthetic Management of a Patient with Parotid Abscess with Concomitant Organophosphorous Compound Poisoning

Preethi Rathnasabapathy[1], Sujatha MP[2]

Author's Affiliation:

[1]Post Graduate, [2]Associate Professor, Sri Devaraj Urs Medical College, Sri Devaraj Urs Academy of Higher Education & Research R L Jalappa Hospital, Kolar, Karnataka 563101, India.

Abstract

Introduction: Organophosphorous compounds are chemical agents in wide spread use throughout the world in agricultural industries. This poses a major challenge to an anesthesiologist; there is accumulation of acetylcholine causing overstimulation of muscarinic and nicotinic receptors disruption of transmission of nerve impulses in both peripheral and central nervous system.

Case Report: A 47 year old male presented with alleged history of OP compound consumption with complaints of pain and swelling on the left cheek. On examination Diffuse swelling with warmth and tenderness was present. A diagnosis of parotid abscess was made. Systemic examination - CNS - GCS-E4M5V4 with bilateral pupil -2mm reacting to light. Pseudo cholinesterase was 380.

Anaesthetic Management: Preloaded with 500ml RL. Monitors connected. Patient was on continuous Atropine infusion, Inj Midazolam and Inj fentanyl. Preoxygenated with 100% oxygen. Induced with Inj Propofol. Laryngeal mask airway(LMA) number 5- Proseal was inserted. Anaesthesia maintained with 50% nitrous oxide in oxygen, IV propofol with intermittent positive pressure ventilation. Inj Atropine infusion was continued at 3ml/hr and titrated according to heart rate. The haemodynamic parameters remained stable throughout the procedure. The patient was extubated, Postoperative recovery was uneventful.

Conclusion: The use of muscle relaxants can produce bradycardia and prolong the motor recovery.Volatile anesthetics can also cause bradyarrythmias. In this case report we found that patient with OP compound consumption can safely be managed under general anaesthesia with IV propofol, without muscle relaxant and inhalational volatile anaesthetics.

Keywords: Organophosphorous; Parotid abscess.

Key Messages: Organophosphorous compounds are commonly used in agricultural industries. Patients who consume them pose a major challenge to an anaesthesiologist.They have effect on both muscarinic and nicotinic receptors.In such cases with other concomitant conditions,patients can be managed successfully without usage of inhalational agents and muscle relaxants.

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Corresponding Author: Sujatha MP, Associate Professor, Sri Devaraj Urs Academy of Higher Education & Research, R L Jalappa Hospital, Kolar, Karnataka 563101, India.
Email: drsujathamp@yahoo.co.in

Introduction

Organophosphorous compounds are chemical agents in wide spread use throughout out the world in agricultural industries. In patients who have consumed OP compound, they pose a major challenge to an anaesthesiologist as there is accumulation of acetylcholine causing overstimulation of both muscarinic and nicotinic receptors causing disruption of transmission of nerve impulses in both peripheral and central nervous system.Hence leading to airway compromises by excessive secretions and neuromuscular weakness and cardiac arrhythmias.

Case Report

A 47 year old male presented with alleged history of OP compound consumption. He also complained of pain and swelling on the left cheek. On examination- Diffuse swelling with warmth and tenderness was present.A diagnosis of parotid abscess was made. Systemic examination - CNS - GCS-E4M5V4 with B/l pupil -2mm,RTL.Other systems were normal. Routine investigations were done and was within normal limits. Pseudocholinesterase was 380.

Anaesthetic Management

General anaesthesia was planned. 18G IVC secured in right forearm, preloaded with 500ml RL. Monitoring includes pulse oximetry, ECG, non-invasive blood pressure, end-tidal carbondioxide. Patient was on continuous atropine infusion. Pre medicated with Inj Midazolam and Inj. Fentanyl. Preoxygenation done with 100% oxygen. Induced with Inj Propofol. After elimination of reflexes and reaching enough depth of anaesthesia laryngeal mask airway(LMA) number 5 Proseal was inserted and was confirmed by chest rise and $ETCO_2$, and LMA fixed in place. Anaesthesia was maintained with 50% nitrous oxide in oxygen, IVpropofol with intermittent positive pressure ventilation. Inj.Atropine infusion was continued at 3ml/ hr and titrated according to heart rate. Further analgesia was supplemented by inj Paracetamol. The haemodynamic parameters remained stable throughout the procedure. The procedure lasted for 45 min during which 2 crystalloids was given. The patient was extubated when he was awake and obeyed to open eye for command and shifted to recovery room. Postoperative recovery was uneventful.

Discussion

Organophosphorus compounds phosphorylate cholinesterase, an enzyme which hydrolyzes acetylcholine and leads to excessive parasympathomimetic activity. After examining the patient, if the patient is not adequately treated then atropine and pralidoxime should be repeated. Patient can be pre-medicated with midazolam/ diazepam if he is very restless.

Before induction Ryles tube aspiration and thorough oral suction must be done. As succinylcholine is contraindicated, non-depolarising muscle relaxant can be used for intubation. Sellick's manoeuvre is advised for prevention of aspiration. The concentration of acetylcholine is high in these patients. The relaxant of choice is pancuronium. Vecuronium may produce severe bradycardia. Inhalational agent like halothane should be used extremely carefully as chances of bradyarrhythmias are very high. Asystole and life-threatening bradycardia can occur without warning even if the patient has tachycardia. Recurrent bradyarrhythmias may be managed more easily by inserting transvenous pacing electrode.[1]

Sympathomimetics should be avoided as there is an increased susceptibility to ventricular fibrillation. Continuous cardioscope monitoring should be done. Drugs like ketamine hydrochloride and enflurane are contraindicated for fear of convulsions.

Reversal of muscle relaxant is not required as the level of acetylcholine is already very high and they may accelerate the toxicity of organophosphorus compound. Patient should be mechanically ventilated if breathing is inadequate. Postoperatively, atropinisation should be continued for seven days. Phenobarbitone or diazepam should be administered for 48 hours and pralidoxime 1 gm should be given 6 hrly for the first 24 hours.[2]

Skeletal muscle weakness appears within first 4 days. The peripheral neuropathy after poisoning may become evident within two or five weeks. The low pseudo-cholinesterase levels may persist for 15 days to one month.[3]

Conclusion

Organophosphorous compounds phosphorylate cholinesterase, an enzyme which hydrolyze Acetylcholine and leads to excessive parasympathomimetic activity. The use of muscle relaxants can produce bradycardia and prolong the motor recovery. Volatile anasthetics can also cause bradyarrythmias. In this case report we found that patient with OP compound consumption can safely be managed under general anaesthesia with IV

propofol, without muscle relaxant and inhalational volatile anaesthetics.

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Conflict of Interest: NIL

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