Anaphylactic Shock Following Thiopentone: A Case Report

L Mathews

Citation

L Mathews. *Anaphylactic Shock Following Thiopentone: A Case Report*. The Internet Journal of Anesthesiology. 2004 Volume 10 Number 1.

Abstract

A case of carcinoma rectum with right pulmonary nodule was posted for right upper lobectomy. The patient sustained anaphylactic shock following induction with thiopentone. He was successfully resuscitated and surgery was postponed. He was posted for surgery a week later. Thiopentone was avoided and he was induced with ketamine. The intra-operative and postoperative periods were uneventful and he was discharged a week later. Anaphylactic shock following thiopentone, the timely management instituted and precautions taken when he was posted the next time are discussed.

CASE REPORT

A 41 year old male with a diagnosed case of carcinoma rectum (underwent abdominoperineal resection 8 years ago under general anesthesia) with right pulmonary nodule was electively posted for right upper lobectomy. Pre-operative investigations were within normal limits. Lignocaine sensitivity test was negative. He was adequately premedicated with i/m diazepam 7.5 mg and i/v Tidigesic 0.2 mg. On reaching the operating room, under all aseptic precautions, an epidural catheter was inserted at T12 – L1 space. The position of the epidural cannula was confirmed with 3 ml of 2% lignocaine following which 15 ml of 2% lignocaine with adnernalin was administered. Heart rate, blood pressure, respiratory rate and SaO_2 were maintained. 15 minutes later, he was induced with i/v thiopentone 250 mg + O_2 + i/v scoline 100 mg and intubated with a cuffed portex ETT no 8.5. Following induction, there was a fall in blood pressure and rashes appeared over the upper limbs and chest. The pulse was not palpable and blood pressure not recordable. He developed erythema and rashes all over the body and edema of face and tongue. On auscultation, there were rhonchi all over the chest.

He was ventilated with 100% oxygen, Inj. Atropine 0.6 mg + 0.6 mg, Inj. Adrenaline 1 mg i/v was administered immediately and external cardiac massage instituted. Dopamine infusion was started, and Inj. Aminophylline 250 mg i/v given. Following the above measures, the pulse was palpable and the blood pressure showed 80 – 90 / 70 mm Hg. The ECG displayed sinus trachycardia. I/v fluids were administered to maintain a CVP of 10 cm of water. IPPV was continued with 100% oxygen. Surgery was postponed and he was shifted to the recovery room.

After 2 hours, he had spontaneous respiratory effort and heart rate, blood pressure and SaO_2 were maintained. 4 hours later, he was disconnected from ventilator and was on spontaneous respiration via ETT. Oxygen was continued via T piece. He was extubated the next morning when he was fully awake and obeying commands and pupils were equal and reacting to light. Arterial blood gases were within normal limits. Edema had subsided and chest was clear. Dopamine was tapered off.

The patient was posted for surgery a week later. He was premedicated with i/m diazepam 7.5 mg, i/m hydrocortisone 100 mg and i/v tidigesic 0.2 mg. Before induction i/v hydrocortisone 100 mg and i/v aminophylline 250 mg were given. Induction was commenced with i/v ketamine 125 mg + oxygen + i/v pavulon 5 mg and intubated with cuffed portex ETT no 8.0. Anesthesia was maintained using oxygen nitrous oxide, isoflurane and pavulon. Ketamine infusion 500 mg / 500 ml of normal saline @ 60 mg per hour was given intra operatively. The intra operative and postoperative periods were uneventful. He made a complete recovery and was discharged on the 12^{th} postoperative day.

DISCUSSION

The term anaphylaxis means against protection. It is a life threatening antigen-antibody reaction. Prior exposure of an antigen (drug, food, and venom) leads to production of antigen specific IgE antibodies thus sensitizing the host. Subsequent exposure causes antibody-antigen interaction which initiated degrannulation of mast cells and basophils

leading to release of vasoactive mediators like histamine leucotrienes, prostaglandins and platelet activating factor. Their physiological effects are increased capillary permeability, peripheral vasodilatation, broncho-constriction, negative inotropy, coronary artery constriction, platelet aggregation and release of vasoactive amines[1,2].

Although only a small proportion of the over all number of adverse reaction to anesthetic agents are due to hypersensitivity, this number is apparently increasing. This is largely the result of an increase in the number and magnitude of surgical operation in recent years which has led inevitably to a considerable increase in the frequency of repeat anaesthesia.

Although hypersensitivity to barbiturates, in particular thiopentone, are relatively rare, they are often severe. Their incidence is significantly reduced by the avoidance of the drug in atopic patients with chronically elevated concentration of IgE[3].

After injection of barbiturates, there may be an urticarial rash of the upper chest, neck and face that fades after a few minutes. Anaphylactoid reaction such as hives, facial edema, bronchospasm and shock occasionally occur after thiobarbiturate induction. The absence of reaction to oral barbiturates does not ensure a lack of sensitivity to intravenous barbiturates[4].

Causes of anaphylaxis during the perioperative period are muscle relaxants, barbiturates, propofol. local anesthetics, opioids, volatile anesthetics, protamine, antibiotics, blood and plasma volume expanders and intravascular contrast media.

Clinical manifestations include:

Figure 1

Skin -	urticaria, flushing, pruritis, angioedima
Upper airway -	sneezing, rhinorrhoea, nasal obstruction, edema of the tongue, soft palate, epiglottis, larynx, trachea - stridor, cyanosis, respiratory arrest
Lower airway -	tightness in the chest, cough, shortness of breath, wheezing -pulmonary edema
Eyes -	epiphora, conjunctivitis
Cardio vascular system -	hypotension, hypovolemic shock, coronary artery spasm, heart rate and rhythm disturbances

Investigations are plasma IgE concentration, plasma histamine concentration, plasma tryptase concentration and ELISA test[5].

Management of anaphylactic shock aim at 1) reversal of arterial hypoxaemia 2) replacement of intravascular fluid 3) stabilize the circulation 4) inhibition of further cellular degrannulation with release of vasoactive mediators[6].

References

1. Sprung J, Schoenwald P. K and Shwartz L.B " Cardiovascular collapse resulting from thiopentanal induced histamine release" Anaesthesiology, 86 (1997) 1006
2. Elter M.S., Helrich M and Mackenzie C. F "Immunoglobulin L fluctuation in thiopentanal anaphylaxis" Anaesthesiology, 52 (1980) 181-83
3. Fox G.S., Wilkinson R.D and Rabow R.I "Thiopentanal anaphylaxis a case and method of diagnosis". Anaesthesiology, 35 (1971) 655-57.
4. Thomson D.S, Lason C.N, Hacke J. W "Thiamyral anaphylaxis" Anaesthesiology, 39 (1973) 556-560
5. Lilly J.K and Hoy R.H "Thiopentanal anaphylaxis and reagin involment" Anaesthesiology, 53 (1980) 335-337.
6. Fisher M, "Treatment of acute anaphylaxis" BMJ 311 (1995) 731-33

Author Information

Lailu Mathews, M.D.
Associate Professor, Department Of Anaesthesiology And Critical Care, Pondicherry Institute Of Medical Sciences