

Case Report

Acute Circulatory Collapse Followed by Cardiac Arrest during Thiopentone Induction

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Abstract

Acute circulatory collapse followed by cardiac arrest is a rare but serious complication of thiopentone anaesthesia. A 5 years old boy admitted in North Bengal Medical College Hospital with history of 'Recurrent Tonsillitis'. He was prepared for tonsillectomy operation. The reports of his clinical investigations were almost normal except E.S.R which was a bit elevated-09%. X-Ray chest was normal, other investigations did not show any abnormalities.

During induction with Thiopentone Sodium the patient had sudden cardiac arrest. In spite of all life support given to him the patient did not recover.

Key words: Sudden severe hypotension, Anaphylactic shock, Cardiac arrest.

Introduction

Thiopentone sodium was introduced in anaesthesia by Silus Landy in 1934. This is sodium ethyl thiobarbiturate^{1,2}. It is the sulphur analogue of pentobarbitone. Introduced commercially as pentothal sodium. In modern anaesthesia it is used only for induction in other word to produce sleep^{3,4}. It has got many side effects and complications, such as respiratory depression, bronchospasm, laryngospasm; myocardial depression, hypotension, anaphylactic shock etc. Acute circulatory collapse & cardiac arrest is also a rare but a serious complication of it which may be a life threatening^{1,5,6}.

Case Report

A 5 years old boy named-Walid, S/O of Mohammad Alamgir of Kazipur, Sirajgonj was admitted in E.N.T ward of North Bengal Medical College Hospital with history of recurrent tonsillitis. He was prepared for tonsillectomy operation. He was given anaesthetic fitness as his clinical examination & investigations

revealed nothing abnormalities except E.S.R reading which was 09%. He was in empty stomach for about 6 hrs. In the operation theater he was quite. Pre-oxygenation was given for 3-5 minutes. Then induction with thiopentone sodium was given at a dose of 05 mg. per/kg. 5% solution was used.

On completion of injection the patient suddenly became pale. O.T sister reported that no pulse was felt. I immediately ausculted heart but no sounds were found, which was completely stopped. I gave a smart blow over the sternum but found no response. Then I rapidly intubated the patient & started C.P.R (vigorous Ext.Cardiac massage) with 100% oxygen but failed to achieved any response. In the mean time oxygen saturation started to fall which came to 75%. Then I administered 1 ampoule of Inj. Adrenaline (1:10000) intracardiacly within a few seconds the circulation returned & heart sound heard clearly & loudly. The operation was postponed, the patient was being resuscitated accordingly. Within 10 minutes the patient had second arrest.

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I immediately sought help from our cardiologist who rushed in to the O.T with all monitors. Asystole was diagnosed & advanced cardiac life support had been started. A massive dose of hydrocortisone, Inj. Dopamine infusion, Inj 7.5% Sodi-bicarb, all were tried.

I left no stone unturned to get back the life of the patient, but all in vein. The ill-fated boy never returned alived. We resuscitated the patient for about 45 minutes, then the cardiologist gave-up all hopes& declared the patients death.

Discussion

Thiopentone sodium, commercially used as pentothal sodium is a very potent anaesthetic agent, which was used in anaesthesia by Silus Landy in 1934. Then it was used as sole anaesthetic agent. But now in modern anaesthesia it is used as induction agent at sleeping dose^{3,5}. This is sodium ethyl thiobarbiturate. It is the sulphur analogue of pentobarbitone. It is soluble in water and alcohol and forms a 2.5 or 5% solution which is highly alkaline^{4,7}.

It causes sedation, hypnosis, anaesthesia and is anticonvulsant. Average anaesthetic dose is 4-7 mg/kg I/V. After injection of an anaesthetic dose of thiopentone, the blood level rises within a minute, then falls rapidly and the patient regains consciousness due to the rapid distribution of the drug to viscera, lean body mass, etc. It rapidly crosses the blood-brain barrier due to its high lipid solubility. Elimination half-life is 9 hrs. Eliminated more rapidly in the young than in the old^{8,9}. Thiopentone depresses myocardium directly as well as respiratory centre. Total peripheral resistance also reduced. Induction dose of intravenous administered cause a fall of blood pressure markedly & elevation of heart rate. Depression of the medullary centre vasodilates peripheral capacitance vessels which increases peripheral pooling of blood & decreases venous return to the right atrium. The tachycardia is probably due to a central vagolytic effect^{8,11}. Cardiac output & arterial blood pressure may fall dramatically owing to uncompensated peripheral pooling and unmasked direct myocardial depression^{5,6}.

Patients with poorly controlled hypertension are particularly prone to wild swings in B.P during induction. The cardiovascular effects of barbiturates therefore vary markedly. A slow rate of injection and adequate pre-operative hydration attenuated these changes in most patients⁶. It constrict the cerebral

vasculature causing a decrease in cerebral blood flow & intracranial pressure. The drop in ICP exceeds the decline in arterial blood pressure so that cerebral perfusion pressure is increased. It may cause severe bronchospasm following induction with thiopental due to histamine release causing apnoea & cyanosis^{10,11}. Tidal volume and respiratory rate both are decreased. Hypotension caused by peripheral vasodilation will not necessarily cause a fall in tissue blood perfusion providing that cardiac output is maintained¹⁰. Once tissue oxygenation becomes inadequate cell function becomes impaired. In the brain this is associated with impaired as is sometimes seen during spinal/epidural anaesthesia & is a vulnerable warning sign that the blood pressure is too low. In the heart this is associated with ECG changes, ischaemic changes affecting the ST segments and dysrhythmias followed by circulatory collapse^{5,6,11}.

Conclusion

As thiopentone is a potent intravenous anaesthetic, the drug should be administered very slowly & with extreme caution. 2.5% solution should always be used but never 5% solution. Prior to induction 2 ml of 2.5% should be administered & reaction should be observed carefully. In susceptible cases all the investigations should be done, especially allergy test. (IgE level)^{9,11}. Then alternative induction agents (ketamine, propofol) or inhalational induction agents (halothane, ether, sevflurane) should be tried¹¹. All the resuscitatory equipments should be ready at hand. Above all it is the anaesthetist whose alertness, carefulness, observation & aquired knowledge can overcome this complication & can save a patients life¹⁰.

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