Case Report

Unexplained Hypotension Under General Anaesthesia

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Case Report

A 39 years aged young male with hypertension and ischemic heart disease of 4 years duration on medical management, presented with lumbar canal stenosis involving L3-L4 and L4-L5 levels for surgical decompression. Routine preoperative assessment including cardiac evaluation revealed regional wall motion abnormality in the echocardiogram with LVEF of 56%. He had moderate effort tolerance and was planned for surgery under ASA grade II. The patient was adequately fasted and pre-medicated with a benzodiazapine and H2-antagonist. He was advised to skip ACE inhibitors on the morning of surgery and to continue with antiplatelet agents. The patient was monitored as per ASA standard including invasive BP (right radial artery) monitoring. He had a stable induction with IV Fentanyl 100 mcg and IV Propofol 180 mg and was intubated after muscle relaxation with IV Vecuronium 8 mg. In addition the patient was also given 10mg of IV Esmolol to attenuate the stress response due to larngoscopy and intubation.

The surgery was done in the prone position and anaesthesia was maintained with Isoflurane, air and oxygen. Since the patient's intraoperative BP was gradually dropping and the urine output was falling, he was managed initially with 2 litres of intravenous crystalloids and then 500ml of colloid(Gelofusine) to maintain the MAP above 70-80 mmHg. However, following administration of the colloid, there was sudden drop in BP to 60/40 mmHg. He was given additional intravenous boluses of crystalloids. IV Ephedrine 6 mg boluses (to a total of 36 mg) and IV Phenylephrine 100 mcg boluses (to a total of 300 mcg)were administered and then a dopamine infusion was started at 10 mcg $kg^{-1}min^{-1}$ with no improvement in the BP. Given his past medical history, a cardiac etiology was suspected for the hypotension.

At the end of surgery (in about 10 minutes time), when the drapes were removed and the skin was exposed, extensive erythrodermic rashes were noticed over the thorax, abdomen, arms and legs raising the doubt of a possible hypersensitivity reaction to one of the anaesthetic agents. We suspected anaphylaxis and the patient was immediately given IV Adrenaline (1 cc of 1:10000 solution), IV Chloramphenicramine 25 mg and IV Hydrocortisone 100 mg. The BP rapidly improved to 100/72 mmHg with a stable heart rate. Other signs of anaphylaxis were ruled out. The patient was extubated after adequate reversal and shifted to the ICU in a stable condition for observation.

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Discussion

Anaphylactic and anaphylactoid reactions are sudden and dose independent. They occur mostly in individuals who are already sensitized but may occur even on first exposure to any of the anaesthetic drugs or colloids used during the surgery. The manifestation of anaphylactic and anaphylactoid reactions are clinically indistinguishable. Anaphylactic reactions are IgE mediated and are detected by the presence of positive in-vitro and in-vivo tests and the release of tryptase, a mast cell protease enzyme during the reactions. Anaphylactoid reactions occur through a direct nonimmune mediated release of mediators or complement activation. Though they are IgE independent anaphylactoid reactions can be associated with activation of mast cells and/or basophils and raised tryptase levels[1]. Though anaphylactic reactions are rare in occurrence, they may occur intra-operatively in patients under anaesthesia which often go unnoticed.

Since the main features of anaphylaxis such as hypotension and bronchospasm have many other common causes, the recognition of such hypersensitivity reactions during anaesthesia is usually delayed. Anaphylactic reactions in operation rooms can occur due to various allergens, particularly drugs like muscle relaxants, NSAIDS, antibiotics, hypnotics and certain intravenous colloid solutions. Natural rubber latex materials like gloves and the Foley catheter are also common trigger factors for hypersensitivity reactions. In this case we had not given any antibiotics or NSAIDS intra-operatively, so it was thought that either the muscle relaxant or the colloid solution could have been the trigger for anaphylactic reaction. Muscle relaxant was given at induction and was associated with no haemodynamic changes. But the sudden hypotension following the administration of intravenous gelofusine lead us to conclude that the colloid solution was the cause of anaphylaxis. There are very few reports of colloids causing anaphylaxis. Polyzoiset al[2] has reported a similar kind of anaphylactic reaction due to gelofusine during an orthopedic surgical procedure.

Plasma expanders such as colloids are commonly used during major surgeries with excessive blood loss. They play a major role in resuscitation of the severely hypovolaemic patients. The incidence rate of anaphylactoid reaction to gelofusine that contains

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succinylated gelatin and other plasma expanders varies between 0.07–0.15%[3,4]. However with increasing use of such plasma expanders, the reports of such adverse reactions are increasing in the literature[5-8].

In a large multi-centred prospective trial involving 2,00,906 infusions of colloid substitutes conducted by Ring and Messner, 69 cases of anaphylactoid reactions were observed. Specifically, the incidence of severe reactions such as shock, cardiac and/or respiratory arrest was found to be 0.003% for plasma protein solutions, 0.006% for hydroxyethylstarch, 0.008% for dextran and 0.038% for gelatin solutions[4]. Vervloet et al, reported 3 cases of anaphylaxis due to Plasmagel, a modified fluid gelatin. One of these occurred on first exposure during the surgery and in another patient a repeated infusion of plasmagel caused anaphylactic shock[6].

Diagnosis

The sudden increase in serum tryptase levels are indicative of mast cell degranulation and are the only indicative test to narrow down the diagnosis to anaphylaxis. Concentrations peak after an hour of the hypersensitivity reaction and usually last for several hours thereafter. Serial measurements are more specific and sensitive than a single measurement in the confirmation of anaphylaxis. The collected blood samples are needed to be refrigerated before sending to the laboratory. Comparison with the baseline values, taken during convalescence may confirm or exclude the diagnosis. In this case we did not check the serum tryptase level since it was not available in the institute. The activated basophils express lysosomal membrane glycoprotein CD63 on their surface which can be detected in a method called basophil activation test(BAT). Apostolou et al[9] have reported the use of basophil activation test as a reliable assay to detect gelofusine sensitivity. In addition anaesthesiologists should also recognize other possible non-allergic causes for the reaction.

Management

Initial management of anaphylaxis should follow the ABC approach, limb elevation and discontinuation of the suspected drug or intravenous colloid solution. Adrenaline (epinephrine) is the most effective drug in anaphylaxis and should be given as early as possible. It effectively reverses peripheral vasodilation and reduces oedema. Its beta-receptor stimulation helps in dilatation of the bronchial airways, potentiates myocardial contraction, and suppresses the release of histamine and leukotriene. Early administration of adrenaline attenuates the severity of IgE-mediated allergic reactions. There is greater margin of safety for the intramuscular route though experienced specialists give adrenaline 1mcg Kg-1 intravenously for anaphylaxis. Subcutaneous or inhaled route for adrenaline may not be effective and is not recommended.

Intravenous fluid challenge with crystalloids 20ml Kg-1 and oxygen supplementation should be administered as soon as possible. Antihistamines (H1-antihistamine) as a second line drug in the management of anaphylaxis may help counter histamine-mediated vasodilation and bronchoconstriction. Corticosteroids may help prevent or control the reactions. The dose of IV hydrocortisone varies from 100-200mg depending on the age and weight of the patient. In case of severe bradycardia which develops in some patients after an anaphylactic reaction IV atropine may be considered. Glucagon may be considered to treat an anaphylactic reaction in patients on beta-blockers.

Conclusion

Anaesthesiologists and surgeons should always be suspicious and vigilant to diagnose rare causes of uncontrolled hypotension which may be hidden under the surgical drapes like in this case. A portion of the skin has to be made visible to be observed by the anaesthesiologist through the duration of surgery irrespective of the nature of the surgery and position of the patient. Though anaphylactoid reactions occur commonly with drugs used during anaesthesia, the colloid plasma expanders also carry the risk of hypersensitivity reactions. Plasma expanders provide intravascular volume expansion and help reduce transfusion requirements. However, these agents should be handled with caution. A high index of suspicion and a prompt diagnosis should ensure successful resuscitation in the event of anaphylaxis.

References

1) Hepner DL, Castells MC. Anaphylaxis during the perioperative period. Anesth Analg 2003; 7:1381-95.

2) Polyzois I, Lampard A, Mohanlal P, Tsiridis E, Manidakis N, Tsiridis E. Intraoperative anaphylaxis due to gelofusine in a patient undergoing intramedullary nailing of the femur: a case report. Cases Journal 2009; 2:12.

3) Mishler JMt. Synthetic plasma volume expanders – their pharmacology,safety and clinical efficacy. Clin aematol 1984; 13:75-92.

4) Ring J, Messmer K. Incidence and severity of anaphylactoid reactions to colloid volume substitutes. Lancet 1977; 1:466-9.

5) Jenkins SC, Clifton MA: Gelofusine allergy – the need for identification jewellery. Ann R Coll Surg Engl 2002; 84:206-7.

6) Vervloet D, Senft M, Dugue P, Arnaud A, Charpin J. Anaphylactic reactions to modified fluid gelatins. J Allergy Clin Immunol 1983; 71:535-40.

7) Freeman MK. Fatal reaction to haemaccel. Anaesthesia 1979; 34:341-3.

8) Prevedoros HP, Bradburn NT, Harrison GA. Three cases of anaphylactoid reaction to Haemaccel. Anaesth Intensive Care 1990; 18:409-12.

9) Apostolou E, Deckert K, Puy R, Sandrini A, de Leon MP, Douglass JA, et al. Anaphylaxis to Gelofusine confirmed by in vitro basophil activation test: a case series. Anaesthesia 2006;61:264-8.