CASE STUDY

PERIOPERATIVE ANAPHYLAXIS – OUR EXPERIENCE OF TWO CASES

Nikhil Mudgalkar, Nagpadma, Sharley

Department of anaesthesia, Prathima Institute of Medical Sciences, Karimnagar, Andhra Pradesh.

Correspondence:
Dr. Nikhil Mudgalkar
Assistant Professor,
Department of Anaesthesia,
Prathima Institute of Medical Sciences, Karimnagar, Andhra Pradesh.

ABSTRACT

Anaphylactic reactions during intraoperative period are difficult to diagnose because of the anaesthetized state of the patient. During the perioperative period, any event relating to sudden-onset haemodynamic collapse or increased airway pressures during certain surgical procedures should raise suspicion of anaphylaxis. Though cited as rare in the western world, echinococcus infection is common in India, resulting in an increased ratio of cases, as and when compared to the western world. We report of two cases of perioperative anaphylaxis for hydatid disease surgery during 3 years period. Anaphylaxis is a medical emergency requiring prompt intervention. Although protocols vary in some of the details of management, adrenaline is the mainstay of treatment and early administration saves lives. However, even with prompt appropriate treatment, anaphylaxis can still kill, as our first case reports. From our experience, we suggest use of 100 % oxygen during the intraoperative period and maintaining anaesthesia with intravenous agents so that in case of an untoward catastrophic event, cerebral hypoxia may be delayed Increased reports of successfully managed cases will help in future references and in formulating guidelines of management .

Keywords: perioperative anaphylaxis, hydatid cyst, adrenaline

INTRODUCTION

Echinococcus granularis /multilocularis is a common disease found in india. Highest prevalence of hydatid disease is found in Andhra Pradesh and tamil nadu [1]. This explains the fact that we see more number of cases posted for surgery of hydatid disease. Echinococcosis produces cystic lesions primarily found in liver and secondarily in lungs and other tissues. The cyst has got high antigenicity and potential for anaphylaxis with IgE mediated immunity. Following the activation of cellular immunity, mediators released act on different target organs leading to clinical sequlae from urticaria to profound shock. The incidence of intraoperative anaphylaxis is found between 0.2 to 3.3 % [2] .we review in this article our experience of two anaphylaxis cases during intraoperative period and management of such disastrous complication. More reporting of such cases will lead to a better understanding of the associated complications and help in future references. Also successfully managed cases can guide us in treating and formulating protocols for further management.

CASE HISTORY

CASE 1:

A 29 years old female patient from rural background was admitted in the hospital with the diagnosis of hydatid disease of spleen and posted for splenectomy. Patients preoperative history was nil except occasional pain in left flank. Her preoperative investigation revealed haemoglobin of 13.6 gram percent , blood urea 40 mg , serum creatinine of 1.2 mg , serum bilirubin of 0.6mg/dl , bleeding time of 2 minutes and clotting time of 5 minutes , normal chest x ray and normal electrocardiogram . Patient was accepted for anaesthesia as ASA grade 1 case.

On the operative day, she was conscious and coherent, with Nil by mouth for 12 hours, a pulse rate of 90 beats per minute, blood pressure of 124/74 mm Hg with non invasive BP moniter without any additional clinical findings. It was decided to proceed with general anaesthesia with endotracheal intubation. Patient was premedicated with injection midazolam of 1 miligram, injection fentanyl 100 microgram and injection glycopyrolate 0.2miligram. After preoxygenation for 3 minutes, patient was induced with injection thiopentone sodium 250 mg and injection succinylcholine 100 mg. Endotracheal intubation was done with portex cuffed endotracheal tube of 7.5mm internal diameter. After intubation, patients pulse rate was 100 beats per minutes with blood pressure of

140/90 mm Hg with non invasive blood pressure moniter. Injection vecuronium bromide 4 mg was given and patient maintained with oxygen: nitrous ratio of 50:50 with intermittent isoflurane. Surgery started after 10 minutes.

Patient maintained haemodynamic stability for next 2 hours. During this period patient received 2 litres of lactated ringer solution, and her output was 150 ml clear. After ligation of all ligaments, , patient developed sudden bradycardia and cardiac arrest without any prior changes in rate or rhythm either by moniter or auscultation. Immediately nitrous oxide was put off, patient ventilated with only 100 % oxygen and cardiopulmonary resuscitation was started with external cardiac massage .injection atropine 0.6 mg Intravenous and injection adrenaline 1 mg (1:1000) was given. After 3 cycles of Cardiopulmonary resuscitation with continuous external cardiac massage and 3 doses of injection adrenaline patient regained spontaneous rhythm around 6 minutes after the initial event. Though peripheral pulses were palpable they were weak, heart rate on moniter showed 150 beats with sinus rhythm, blood pressure recorded was 80 mm Hg systolic on non invasive blood pressure moniter.Noradrenaline infusion was started with rate of 10 microgram /kg /min. Fluid boluses with lactated ringer were given to augment the preload. Surgery was started again and completed within next 15 minutes. The diagnosis of anaphylactic shock to spillage of contents of hydatid cyst was made. Patient was given injection hydrocortisone 200 mg, injection diphenhydramine 50 mg and injection ranitidine 50 mg. Throughout the period patient heart rate remained between 140-150 beats per minute and blood pressure of around 80 mmhg systolic on vasopressor (noradrenaline) support.

Patient was shifted to high dependency unit and put on ventilatory support with controlled ventilation; another vasopressor dopamine was added at the rate of 10 microgram/kg /minute along with noradrenalie infusion. Central venous line was put (internal jugular) and her central venous pressure was found to be 8cm of water. Patient's investigation immediate post surgery showed eosinophilia and severe metabolic acidosis. Injection soda bicarbonate was given in standard doses to tackle acidosis. After 2 hour of surgery, patient showed spontaneous respiration but efforts were very poor, though patient did not regain consciousness.

6 hours post surgery, patient developed pulseless electric activity and even after 1 hour of cardiopulmonary resuscitation, patient could not be survived. Patients relative did not consent for autopsy.

CASE 2:

30 years old female patient from rural background admitted in hospital with a diagnosis of hydatid cyst of right lobe of liver on ultrasonography. Her previous history was uneventful except mild pain in right side of abdomen. She was posted for laprascopic aspiration of hydatid cyst. On preoperative examination, she did not give any history of comorbid conditions. Her haemogblobin was 11 grams% , platelet count 1.5 lakhs , normal kidney function tests , liver function tests , electrocardiogram and chest x ray.she was accepted for anaesthesia as ASA grade 1 case.

On the day of surgery, she was conscious and coherent; her pulse rate was 86 beats per minute, blood pressure of 124/80 mm Hg with non invasive blood pressure monitor, and without any additional clinical findings. It was decided to proceed with general anaesthesia with endotracheal intubation .In preoperative room, patient received premedication in the form of injection glycopyrolate 0.2 mg, injection ranitidine 50 mg, injection ondensetron 4 mg, injection midazolam 1.5 mg and injection fentanyl 80 mcg. She was preoxygenated with 100 % oxygen for 3 minutes. Induction of anaesthesia was done with injection propofol 100 mg .patient was intubated with portex cuffed endotracheal tube of 7.5 mm internal diameter under effect of injection succinylcholine 100 mg .Anaesthesia was maintained with oxygen : Nitrous oxide : isoflurane and injection vecuronium bromide. Intraoperative course was uneventful except one episode of hypertension (180/110 mm Hg) which was managed with nitroglycerine drip.Intraoperative fluids were 2 litres ringer lactate and urine output was 300 ml in 2 hours period. Procedure was uneventful. After surgical closure and deflation of pneumoperitonium , nitrous oxide was cut off and patient ventilated with 100 % oxygen while anaesthesia team was waiting for recovery of the patient. Patient did not regain spontaneous activity even after 20 minutes of controlled ventilation, so reversal was not attempted. After 20 minutes of surgical closure patient developed sudden bradycardia and cardiac arrest within a few seconds without any pre-existing changes in rate or rhythm on monitor. Immediately cardiopulmonary resuscitation was started with effective external cardiac compression and injection adrenaline 1:1000 1 mg intravenous given. After 3 cycles of cardiopulmonary resuscitation, patient developed ventricular fibrillation which was treated with biphasic DC shock of 150 joules. Rhythm reverted to sinus rhythm immediately post DC shock and peripherals were palpable though blood pressure recorded was low (80 /50 mm Hg). Nor adrenaline infusion was started and patient shifted to high dependency unit on ventilator.Injection ranitidine 50 mg , injection diphenhydramine 25 mg also given. Central venous catheter was put and CVP showed 6 cm water. Additional fluid boluses were infused to achieve target CVP of 8 cm of water, when blood pressure recorded was 120 /70 mm Hg, and nor adrenaline infusion was stopped after 4 hours. In view of delayed occurrence of the reaction, other pathologies like severe hypovolemia, tension pnuemothorax and myocardial infarction were ruled out suitably and diagnosis of anaphylactic shock was made. Postoperative blood examination revealed eosinophilia of 10, though patient did not afford serum tryptase. Patient regained consciousness after 6 hours, but

sedated with midazolam and kept on ventilator till next day morning and extubated on second day uneventfully without any neurologic deficits. Patient was dischared home on 5th postoperative day.

DISCUSSION

Anaphylactic shock, an extreme manifestation is a systemic presentation of type-I (IgE) mediated immediate hypersensitivity reaction. Hydatid cyst may trigger off this reaction because of its high antigenicity.

In case 1, the anaphylaxis occurred within minutes after puncture of the hydatid cyst of spleen. Patient went immediately into cardiac arrest and was successfully revived after 5 minutes, though patient remained hypotensive throughout the procedure and into High dependency unit, which did not respond to any vasopressor agents and patient expired after episode of pulseless electric activity 6 hours of surgery .patient was young and without any co morbidities.

In case 2, anaphylaxis occurred almost after completion of surgery, when anaesthesia team was waiting for recovery of the patient and patient was being ventilated with 100 % oxygen for 15 minutes. Patient developed sudden cardiac arrest and was revived after 7 minutes of Cardiopulmonary resuscitation. Patients showed improvement in blood pressure in contrast to the first patient, shifted to high dependency unit and uneventfully extubated after 28 hours of surgery.

Though anaphylaxis is cited as rare, [2] we report of 2 cases within a span of a 3 year period from august 2008 to august 2011, where we did 18 cases of hydatid cyst of various organs.

Both cases were females, which is in line with the findings by other researchers [3,] who demonstrated 3 times more incidence of anaphylaxis in females.

Both cases were managed with the standard line of management of anaphylactic shock on table , including epinephrine , volume loading , hydrocortisone , diphenhydramine and ranitidine.[4] ,we were able to revive the second case , probably because patient was being ventilated with 100% oxygen 20 minutes prior to the anaphylactic reaction occurrence , providing greater time for cerebral protection.

The timing for this reaction is also interesting in second case. As previous experiences indicates , IgE mediated immune reactions occur within 30 minutes of exposure to the allergen [5] , When we reviewed other reported cases , we found that in most of the cases anaphylaxis occurred within minutes of spillage of the contents into body.[6] our second case did not show the corresponding findings. For this reason, other causes mimicking anaphylaxis (myocardial infarction, tension pneumothorax, sudden hypovolemia) were ruled out and diagnosis of anaphylaxis was made. The patient responded to the treatment of adrenaline which also supports the diagnosis of anaphylaxis.

The evidence base for the management of acute anaphylaxis is limited, given the ethical and practical difficulties inherent in performing randomized clinical trials in medical emergencies. So the guidelines for management of acute episodes also vary. However, all protocols agree that adrenaline is mainstay of treatment of anaphylaxis and should be given early in the course of the process. Moreover, identification of anaphylaxis on table is difficult, but, often the first sign is pulselessness and or severe bronchospasm [7], in both our cases , pulselessness was the first sign , while neither of the cases showed bronchospasm .

From our experience of these two cases, we can have some conclusion.

1)Perioperative anaphylaxis in hydatid disease is not uncommon in endemic areas.

2)Avoiding the use of inhalational agents to increase the oxygen concentration at near 100 % ,while maintaining anaesthesia with intravenous agents like propofol infusion and opioids like fentanyl can give us precious time to revive the patient ,in case of any eventuality in hydatid .

REFERENCES

1. Reddy CRRM. Epidemiology of hydatid disease in Kurnool.Ind J Med Res 1968; 56:1205-20.
2. Jakubowski MS, Barnard DE. Anaphylactic shock during operation for hydatid disease. Anesthesiology 1971; 34: 197-99.
3. Laxenaire MC, Mertes PM, Benabes B, et al. Anaphylaxis during anaesthesia: results of a two-year survey in France. Br J Anaesth 2001; 87:549 –58.
4. David L. Hepner, MD, and Mariana C. Castells. Anaphylaxis during the Perioperative Period. Anesth Analg 2003; 97:1381–95.
5. Pumphrey R. Lessons for the management of anaphylaxis from a study of fatal reactions. Clin Exp Allergy. 2000; 30:1144–50.
6. Khanna P, Garg R, Pawar D. Intraoperative anaphylaxis caused by a hepatic hydatid cyst. Singapore Med J 2011; 52(2): e18.
7. The Association of Anaesthetists of Great Britain and Ireland. Suspected anaphylactic reactions associated with anaesthesia. 2003. Available at: http://www.aagbi.org/publications/guidelines/docs/anaphylaxis03.pdf (accessed 6 January 2008).