

CASE REPORT

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CARDIAC ARREST;

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ABSTRACT... We present hereby a case report of 65 years old female scheduled for laparotomy for excision of hydatid cyst liver diagnosed on ultrasonography and CT scan, but on laparotomy unexpectedly it turned out to be pheochromocytoma right adrenal and was further complicated by rupture of tumour, avulsion of inferior vena cava, massive haemorrhage, leading to cardiac arrest. Detailed in this report is the management of unexpected pheochromocytoma, treatment of acute severe haemorrhage with massive transfusion and resuscitation and revival of patient from cardiac arrest with CPR.

Key words: Pheochromocytoma, acute massive blood loss, cardiac arrest, CPR, massive transfusion, postoperative bleeding, thoracolaparotomy.

On Apr 09, 2003, a 65 years old lady weighing 50 Kg, was anaesthetized for excision of hydatid cyst right liver diagnosed by ultrasound and computerised scan. During manipulation of tumour by surgeon the patient suddenly developed severe sympathetic response with pulse rate 160/min and blood pressure 230/140 mm hg. Pheochromocytoma was suspected and surgeon also confirmed adrenal location of the tumour extending and attached to under surface of liver. Surgeon was asked to stop manipulation of the tumour and patient was stabilized with injection loproressor 5 mg and injection largectil 25 mg (as no injectable alpha blocker was available).

Blood pressure, pulse oximetry, electrocardiography, temperature, urine output was monitored. Facility of invasive blood pressure monitoring was not available.

Central venous line was also passed in right internal jugular vein to monitor fluid balance.

During dissection of tumour inferior vena cava got avulsed as it was entangled in the tumour resulting in acute massive blood loss leading to cardiac arrest (EMD type). All anaesthetics were stopped. Cardiopulmonary resuscitation was done with 100% O₂ through endotracheal tube, rapid intravenous fluid through already established two peripheral and one central venous line (Hartman sol, Haemacel, whole blood), external cardiac massage, injection atropine 1+1 mg, injection adrenaline 1+1 mg. Five units of whole blood pushed rapidly. Meantime surgeon ligated inferior venacava and haemostasis done. Patient was revived successfully. Transfusion continued. 1000 mg calcium gluconate given I.V. Three abdominal packs left in side

as there was oozing from raw surface after removing the tumour.

Postoperatively patient needed inotropic support for 48 hours due to hypotension. Another 06 units of blood transfused including 3 units of fresh blood as PFP and platelets were not available. Hb, Hct, Coagulation profile, cardiac enzymes, ECG, NIBP, Pulse oximetry, CVP, intake/output monitored postoperatively. Postoperative haemoglobin was 10.3 gm/dl (after 11 transfusions), cardiac enzymes, electrocardiography, Coagulation profile were within normal limits. After 48 hours patient was anaesthetized again to remove abdominal packs but patient was having fresh bleeding from lacerations of under surface of liver that was repaired through thoracotomy incision. Another two units of blood was transfused. Haemostasis was secured. Recovery after the second procedure was uneventful.

Patient was discharged on fourteenth postoperative day fully recovered without any residual effect. Retrospective analyses revealed preoperative history of occasional palpitations for last 01 year and electrocardiograph showing tachycardia. Diagnosis of Pheochromocytoma was confirmed on histopathology.

DISCUSSION

90% Pheochromocytoma tumours are located in adrenal medulla and 10% elsewhere extending from base of skull to anus.

This tumour has clinical effects by secreting excessive catecholamines (adrenaline, noradrenaline, dopamine). Tumour usually presents with classical triad of severe headache, diaphoresis and palpitations. 0.1% cases of hypertension are due to this tumour. Other symptoms may include weight loss, nausea, vomiting, diabetes like syndrome.

Pressor response to some of drugs like histamine, tyramine, droperidol, metoclopramide, cytotoxic drugs, phenothiazines, tricyclic antidepressants suggests presence of pheochromocytoma.

5% cases are inherited as autosomal dominant trait

either alone or part of neoplastic syndrome called multiple endocrine neoplasias. On clinical suspicion diagnosis is confirmed by 24 hours urine analysis for catecholamine, metanephrine and vanillylmandelic acid (VMA) and measurement of plasma catecholamines level. MRI and CT can confirm location of tumour.

Cornerstone of preoperative preparation is alpha-adrenergic blockers like phenoxybenzamine starting at 10-20 mg twice daily orally and phentolamine (5 mg) intravenously 2 hrs before surgery.

Beta blockers should only be started after alpha blockers. These patients are usually volume depleted due to marked vasoconstriction owing to excessive sympathetic activity and therefore need fluid preload. Preoperative sedation helps to reduce dose of anti hypertensive drugs.

During intraoperative management following drugs be avoided:- Histamine releasing drugs as morphine & atracurium, Catecholamines releasing drugs as ephedrine & ketamine. Vagolytic drugs as pancuronium & atropin, suxamethonium as fasciculations can release catecholamines from tumour. Halothane can provoke dysrhythmia.

Patient should be monitored with ECG, Pulse oximetry, invasive BP, CVP, urinary catheter. General anaesthesia, regional anaesthesia or combinations of both are acceptable. Sodium thiopentone is most commonly used induction agent. Administering 1.5 mg/Kg lidocaine 1-2 minutes before laryngoscopy can lessen the sympathetic response to laryngoscopy and intubation. Halothane is better avoided due to its arrhythmogenic effects. Isoflurane is most commonly used. Use of desflurane has also been found safe. Narcotics are also used concomitantly. When unrecognised or improperly managed perioperatively the discharge of excessive catecholamines can be disastrous.

A number of drugs are available for control of intraoperative hypertension and tachycardia which includes Nitroprusside infusion .5-1.5 micro gram/kg/min to maximum 8 micro gram/kg/min, beta blockers (propranolol, esmolol, metoprolol), alpha blocker

(phentolamine), labetalol (alpha + B blocker), Hydralazine, Magnesium sulfate I.V. Lidocain I.V should be considered for control of arrhythmias.

Postoperatively hypotension should always be taken into account that may need fluid and inotropic support. PEA(EMD) due to acute severe hypovolemia needs rapid restoration of blood volume in addition to CPR with external cardiac massage, and ventilation with 100% O₂ through ETT. During CPR inj epinephrine 1 mg is pushed IV and repeated every 3-5 minutes. If PEA rate is slow inj atropine 1 mg iv pushed, repeated every 3-5 minutes to a maximum dose of .04 mg/kg. Concomitantly effective oxygenation & ventilation is confirmed. For rapid restoration of blood volume in addition to wide bore peripheral line, central line is preferred.

Symptoms associated with acute blood loss generally appear after loss of 15-20 percent of blood volume. Haemorrhagic shock is associated with 35-40 percent acute blood loss. Treatment of patients who experience excessive blood loss usually involves transfusion of blood and blood components.

Initial treatment can be started with crystalloids, colloids or combination of both. Colloids need less volume but have high cost and can cause allergic reactions. Three main goals of therapy in haemorrhagic shock are volume restoration, adequate tissue perfusion and maintenance of normothermia.

Massive transfusion is replacement of one or more blood volumes within 24 hours. Whereas all transfusions have potential adverse reactions, massive transfusion of stored blood has some unique consequences.

The adverse consequences stem from the fact that refrigerated stored red blood cells and whole blood undergo changes during storage and in addition contain concentrations of anticoagulant/preservative solution. Compared with fresh blood stored blood is hypernatremic, hypoglycaemic, hyperammonemic hyperphosphatemic and hyperkalemic.

The dangers associated with massive transfusion are

mainly related to either quantity of blood transfusion (delusional coagulopathy) or rate of infusion (e.g. citrate toxicity, hyperkalemia, acid – base imbalance and hyperinsulinemia).

In massive transfusion citrate level may reach 100 mg/dl from normal 1 mg/dl. Although most of the patients who undergo massive transfusion do not require calcium supplementation. In some patients especially with impaired citrate metabolism e.g. liver dysfunction may need calcium supplementation as calcium chloride or calcium gluconate.

The dangers of delusional coagulopathy is high in patients who had severe prolonged hypotension or have been transfused two volumes of blood. Routine prophylactic use of platelets and coagulation factors is not advisable however patients with platelet count below $50 \times 10^9/L$ and intractable bleeding may benefit from administration of platelet concentrates. Transfusion of FFP is recommended in patients with transfusion volume in excess of two blood volumes who have also received large volume of crystalloid especially in patient with hepatic dysfunction. A suggested trigger point for FFP transfusion is when PT and PTTK exceeds 1.5 times the upper limit of normal with clinical evidence of coagulopathy.

Massive transfusion of cold blood has been associated with hypothermia and subsequent cardiac arrhythmia and cardiac arrest. The risk of transfusion-associated hypothermia can be reduced by the use of in line blood warmer. Other strategies to prevent hypothermia during massive transfusion can be helpful eg. Fluid warmer, increasing temperature of operation room.

CONCLUSION

Possibility of pheochromocytoma must be kept in mind in a patient with unexplained intraoperative hypertension especially on manipulation of tumour. Surgery in patients with undiagnosed pheochromocytoma has a mortality of 50%. In addition to undiagnosed pheochromocytoma our patient had multiple high risk complications i.e. acute massive blood loss and massive transfusion, cardiac arrest, postoperative bleeding, thoracotomy 48

hours after initial surgery involving cardiac arrest. Close intraoperative monitoring and prompt management resulted in uneventful recovery.

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