Case Report

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Anesthetic Management for Organ Retrieval from a Brain-Dead Donor

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Abstract

With the improvement of intensive care facilities, brain dead organ donors have become a major source of transplant organs today. Optimal management of the donor, prior to organ retrieval is critical to the functioning of the graft. We report the anaesthetic management of a brain dead patient for organ harvesting. Patient was a hypertensive on treatment, and was otherwise healthy. Preoperatively patient was on mechanical ventilation and was on dopamine support. Intraoperative vasopressin and noradrenalin infusions titrated to blood pressure were given. Blood glucose was controlled with insulin. Liver harvesting was not done due to fatty infiltration. Both kidneys were harvested in good condition, after which ventilatory support was withdrawn and all infusions discontinued.

Keywords: Brain Dead Donor; Organ Retrieval; Directed Tissue Donation.

Introduction

Donation after brain death is a major source of organs for transplantation. It is important to properly manage the donor so that maximum number of organs can be retrieved in the best possible condition. We report the anesthetic management of a brain dead patient for organ harvesting.

Case Report

A 45 year old male suffered irreversible brain injury following a cerebrovascular accident and was declared brain dead on the day of hospital admission. The patient had history of hypertension on treatment with amlodipine 5mg twice daily. He was otherwise healthy, weighing about 65 kg and was on mechanical ventilation with pulse 68/min, BP 150/94 mmHg, Sp02 100%, normal heart sounds, clear chest, urine output 50 mL/hour. Blood investigations were: Haemoglobin 18 gm%, Urea 65 mg%, Creatinine 2.3 mg%, Na^+ 150 mEq/L, K^+ 3mEq/L, Lactate 1.9 mmol/L, Glucose 241 mg%. Arterial blood gas analysis revealed pH 7.13, $Pa0_2$ 332.4 mmHg, $PaCO_2$ 83.2 mm Hg, base excess of -10.7 mmol/L, HCO_3, 28.2 mmol/L, SO_2 99.8 %. His medications included I.V. dopamine 2 micg/kg/hr, I.V. fosphenytoin 150 mg q8h, I.V. mannitol 250 mL q8h, I.V. methylprednisolone 3 g daily, antibiotics, L-thyroxine 200 µg daily.

Patient was shifted to operation theatre with continuous monitoring, ventilation and medications. OT was warmed, and warm fluids and warming mattress were made available. Monitoring included ECG, $Sp0_2$, NIBP, $EtCO_2$ and urine output. Vasopressin infusion was started at the rate of 2.4 units/hr. Noradrenaline was titrated to maintain systolic BP around 120 mmHg. Pancuronium 4mg, morphine 7.5mg and fentanyl 100 µg were given intravenously, and ventilated with Oxygen and 1% isoflurane. Insulin infusion was titrated to keep the blood glucose below 200 mg % and CVP maintained at 8-10 mm of Hg. Right radial artery was cannulated for continuous blood pressure monitoring.

Surgery began with median laparotomy and inspection of abdominal organs. Liver was not harvested due to significant fatty changes. It was decided to harvest both the kidneys. Incision was extended to include a sternotomy. Mannitol 200 mL was infused I.V. over 20 min, followed by I.V. frusemide 40 mg. Once aorta was exposed, I.V. heparin 20,000 units was given, followed by aortic cannulation and cross-clamping. Organs were perfused with histidine-tryptophan-ketoglutarate (HTK) preservative solution. This was followed by asystole. Both the

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kidneys were removed. Ventilation and supports were discontinued.

Discussion

Brain death is associated with various pathophysiological changes. If not treated appropriately, these derangements lead to organ deterioration resulting in organ loss. The anesthesiologist has a critical role to play in maintaining the viability of organs prior to harvesting.

Pathophysiological Changes in Brain Death

Cardiovascular Changes

Initially there is catecholamine storm [1] (incidence 25-32% [2]) associated with hypertension, tachycardia and systemic vasoconstriction. This can produce myocardial injury and visceral ischemia. This is followed by neurogenic shock with loss of sympathetic tone resulting in vasodilatation and hypotension. Bradycardia resistant to atropine also occurs (incidence 81-97%).

Neuroendocrine Changes

Diabetes insipidus [3] occurs (incidence 46-78%) due to damage of hypothalamic-pituitary axis. Polyuria leading to hypovolemia and dyselectrolemia occurs (hypernatremia, hypokalemia, hypocalcemia, hypophosphatemia, hypomagnesemia). Insulin level falls resulting in hyperglycemia [1]. Sick euthyroid state, which is characterized by low serum levels of thyroid hormones in a patient with non-thyroid illness, similar to that also seen in critically ill patients without brain injury [4]. Thermoregulation is impaired with resultant hypothermia.

Respiratory Changes

Pulmonary dysfunction can be due to multiple etiologies like aspiration, atelectasis, pneumonia and pulmonary oedema (incidence13-18% [5]).

Other Changes

A systemic inflammatory response occurs due to mediators released from damaged brain and generalized ischaemic reperfusion injury. Coagulopathy or DIC [6] (incidence 29-55%) may also ensue.

Optimization of a Brain-Dead Donor

Care of donor is a continuation of intensive care that was provided before brain death. Focus shifts from patient to better organ management. Invasive lines to monitor CVP, arterial BP and cardiac output should be sited if not already in place. Active warming techniques should be used to maintain body temperature more than 35^0C [1].

Hemodynamic goals [7] are to maintain systolic BP above 100mm Hg and CVP of 6-10 mm Hg. Initial catecholamine storm may require vasodilators like sodium nitroprusside or esmolol to reduce hypertension. Hypotension is treated with infusion of warm crystalloids/colloids to achieve euvolemia; packed red cells help to maintain haematocrit more than 30%. Vasopressors like dopamine, noradrenaline or dobutamine may be required to restore blood pressure. Dopamine (2-5mg/kg/h is the first choice in many centers) Vasopressin restores the vascular tone and treats diabetes insipidus. Use of a single vasopressor in low dose was found to be associated with high organ yield.

Respiratory management [8] uses lung protective ventilation–tidal volume 6-8 ml/kg with optimal positive end expiratory pressure so as to optimize oxygenation without compromising the hemodynamic status.

Diabetes insipidus should be treated with vasopressin. The aim is to keep urine output between 1-1.5 ml/kg/hr and serum Na^+<155meq/L. Vasopressin 1 unit slow I.V. followed by infusion of 2.4u/hr titrated to BP. Hypernatremia>155meq/L is treated using hypotonic solutions like 1/2NS, 5%D.

Hyperglycemia >200mg% is controlled using insulin infusion. Triiodothyronine (T3) 4 µg I.V. bolus followed by 3 µg/hr infusion may be given for correction of hypothyroid state. Methylprednisolone 15mg/kg/day helps to reduce systemic inflammatory response associated with brain death.

Adequate oxygenation (Pa02 >100mmHg) by adjusting $Fi0_2$ ensures preservation of organs. When lung retrieval is considered, it is important to maintain $Fi0_2$ <0.4 to minimize oxygen toxicity of lungs.

Preoperatively anesthesiologists should take a complete history, review the medical and surgical records, the condition and supportive measures of vital organs and the medications. Cardiac function

is assessed by means of hemodynamic profile, inotropic support, ECG and echocardiography. Pulmonary function is evaluated by degree of ventilatory support, chest X-ray and arterial blood gas study. Results of renal and hepatic function tests, hemogram, blood glucose, serum electrolytes and blood grouping should also be available. In addition, the validity of brain death certificate and consent of family for organ donation should be verified.

Transition from ICU to OT is crucial and the donor is continuously monitored, ventilated and supportive measures continued. OT should be kept warmed. In case of cardiac arrest, resuscitative measures should be resorted to in order to maintain organ perfusion.

Multiorgan retrieval requires an extensive midline incision from suprasternal notch to symphysis pubis and can produce significant blood loss and hemodynamic instability. Vasoactive drugs should be readily available. Surgical stimulation can produce sympathetic response and involuntary movements [9]. General anaesthetics and muscle relaxants are necessary to blunt these responses.

Isoflurane may provide additional benefit of ischemic preconditioning and reduction of reperfusion injury [10]. Opioids are the other choice. Muscle relaxation with NDMRs is essential. Bradycardia may require treatment with isoprenaline infusion.

Intraoperative goals [11] for vital parameters are the same as pre-operative goals. The "rule of 100" for intraoperative goals is stated as maintenance of systolic BP>100mmHg, PaO_2>100mmHg, urine output>100 mL/hr and hemoglobin concentration>100gm/L. Other goals include blood sugar<200mg%, CVP <10 mm Hg. Medications required include i.v. unfractionated heparin 300 Units/kg before aortic cannulation; i.v. mannitol 0.5 g/kg and frusemide 40mg are given to induce dieresis, before division of renal pedicle. Once preservative solution is perfused, aorta is cross-clamped, mechanical ventilation is discontinued and organs are removed quickly in the following sequence: heart, lungs, liver, pancreas, intestine and kidneys.

Conclusion

Several physiological changes follow brain death. The significant neurohormonal and inflammatory changes result in abnormal hemodynamic and metabolic milieus which are harmful to the potential donor organs. Active anaesthetic management of the donor from the time of brain death till organ retrieval is mandatory for better graft functioning.

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