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CASE REPORT- Title Page

ANAESTHETIC MANAGEMENT OF A CASE POSTED FOR ELECTIVE ABDOMINAL AORTIC ANEURYSM RESECTION

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ABSTRACT:

Introduction: The term ‘aneurysm’ is derived from the Greek word *aneurysma*, meaning dilatation, or widening. The International Society for Cardiovascular Surgery/Society for Vascular Surgery states that an abdominal aortic aneurysm is any focal dilation of the aorta leading to a diameter of at least 50% larger than normal. The normal aorta diameter ranges from 17 to 24mm and is dependent on various factors such as such as age, gender, and body habitus. AAA occurs when the aortic diameter is more than 30 mm in size. An unruptured aortic aneurysms are incidentally detected and come with chronic vague complaints like back pain, abdomen pain, palpitations, dyspnoea on exertion etc. A ruptured AAA is an absolute emergency and require immediate surgical intervention.

Signs, symptoms, and Diagnosis: This is a case report of a 53-year-old female who presented with complaints of on and off chest discomfort associated with palpitations and dyspnoea on exertion for the past 2 years. CT aortography revealed infrarenal abdominal aortic aneurysm extending to the iliac arteries with maximum diameter of 67 mm. This review will mainly focus on the instructive significance for anaesthesia management, especially during clamping and unclamping of major vessels during abdominal aortic aneurysm resection surgery.

Conclusion: Patients with AAA undergoing resection surgery poses a challenge to the anaesthesiologist because among the risk factors for rupture, blood pressure is the only

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factor under his or her control during the operation. If blood loss can be kept to a minimum and hemodynamic stability achieved, AAA resection surgery can be carried out with minimal intra-op and post-op complications.

KEY WORDS: Abdominal Aortic Aneurysm; Vascular surgery; Focal dilatation; CT aortography; Resection surgery.

INTRODCUTION:

The incidence of AAA is 4.9–9.9% [1],[2]. The overall mortality for open repair of AAA surgery ranges between 1% and 6%. In comparison, the overall mortality after ruptured AAA 90% of which 75% of patients die before reaching the operative theatre and of those undergoing surgery, 40% die.

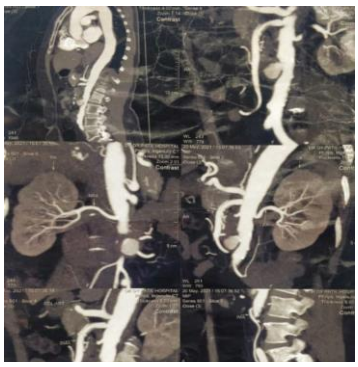
Smoking is the single most important modifiable risk factor in the formation, expansion, and rupture of abdominal aortic aneurysm (AAA). The incidence of AAA increases with age and hence patients who present with AAA are typically, elderly with significant co-morbidities [3]. Mortality rate after elective open AAA repair is significantly lower than that after emergency repair. Effective teamwork is of utmost importance, especially during aortic clamping and unclamping.

PATIENT INFORMATION WITH CLINICAL FINDINGS:

A 53-year-old female came with complaints of chest discomfort on and off associated with palpitations and dyspnoea on exertion for the past 2-3years with a Metabolic equivalent of task score (METS) of less than 4. Patient is a known hypertensive for twelve years and is currently on treatment with T.Nebivolol 2.5mg and T.Cilnidipine 5mg OD. Past surgeries include septoplasty, TAH and haemorrhoidectomy and was uneventful. She also gives history COVID-19 bronchopneumonia three months back for which she was hospitalised for a week for quarantine. There was no requirement for oxygen supplementation and was managed conservatively.

DIAGNOSTIC ASSESSMENT:

A CT angiography was performed, and it revealed an infrarenal abdominal aortic aneurysm extending to the iliac arteries with maximum diameter of 67 mm. Other investigative procedures included routine blood investigation along with C-Xray, USG Abdo-pelvis, cardiac studies (ECG, 2D ECHO) ad cardiac coronary angiography



THERAPEUTIC INTERVENTION:

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In view of low haemoglobin 2 pints of PCV were transfused pre-operatively. After a proper discussion with the operating surgeons, the patient was planned for AAA resection and grafting under general with epidural anaesthesia.

Pre-anaesthetic check-up was done meticulously. All systemic examinations and investigations were found to be within normal limits and Mallampatti score was 2. Patient was given fitness under ASA 3 with high risk, post-op SICU and ventilator consent. Patient was asked to continue her usual anti-hypertensives medication and T. Alprazolam 0.5mg was given on the night prior surgery.

The patient was counselled thoroughly on the day prior to surgery and was briefed about the procedure along the pros and cons of administering general and regional anaesthesia.

After conformation of informed consent, patient was shifted into the operating room the basic monitors were attached which included blood pressure, pulse rate, ECG, EtCO2 and SpO2. Her baseline heart rate was 75/minute, blood pressure was 110/80mmHg, ECG showed normal sinus rhythm and saturation was 98% on room air.

Two wide bore intravenous cannulas were secured and IV fluids -crystalloids were started. All anaesthetic equipment's, monitors and emergency drugs were prepared and checked.

Under all aseptic precautions, in sitting position, 18G thoracic epidural was secured at the level of T8 and T9 intervertebral space. Thoracic epidural space was identified at a depth of 3.5cm from the skin with loss of resistance to air technique and the epidural catheter was fixed at 8cm. Epidural catheter placement was verified by negative aspiration for CSF and blood, followed by an epidural test dose of 2% lignocaine-adrenaline (1:200000). The patient was then kept supine on the operating table and pre-oxygenation with 100% oxygen for general anaesthesia was given for 3 minutes. Inj.Midazolam 1mg+ Inj.Glycopyrrolate 0.2mg i.v was for induction. Inj.Scholine 100mg i.v was administered as the short acting muscle relaxant.

Patient was intubated using a size 3 Macintosh blade and 7.5mm endotracheal tube. Bilaterally equal air entry was confirmed and EtCO2 graph was established. Long-acting muscle relaxant Inj. Vecuronium 5mg was given as loading dose followed by top ups of 1mg every 35-45minutes. Patient was maintained on nitrous oxide with 1-1.5% of sevoflurane. The right IJV was canulated with 7Fr triple lumen central line for CVP monitoring and i.v access. Right radial artery was cannulated with 20G arterial cannula for invasive blood pressure monitoring. Intra op blood pressure was ranging between systolic 120-130mmHg and diastolic blood pressure between 70-90mmHg with pulse rate was fluctuating between 60-70/min. An Spo2 of 100% was maintained throughout the surgery.

Two approaches can be taken for the repair of AAA; open repair or endovascular repair. Open AAA repair is the recommended approach for para renal aneurysms as the graft placement is a long tedious process with high potential for failure with endoscopic approach. Hence a transperitoneal open approach was used in this patient. Due to the cross clamping and de-clamping of major vessels like the Internal Iliac and the Aorta, drastic fluctuation in hemodynamic parameters of the patient is to be expected and therefore the OT must be set adequately to manage any such events by keeping all the emergency drugs prepared and ready to go such as Noradrenaline, NTG, dexmedetomidine, sodium bicarbonate and vasopressin.

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It is of utmost importance that the pressor responses be attenuated to bare minimal levels during especially during laryngoscopy and intubation. Judicious use of analgesics such fentanyl and epidural top ups with 0.1% ropivacaine helped in tackling pressor responses. Patient was maintained on 1% sevoflurane which was adjusted accordingly with respect to vitals. Timely ABG analysis was done.

Three hours into the surgery, the Aorta was clamped following which there was a sudden rise in the patient's BP by 50mmHg of baseline systolic BP and more than 30mmHg rise of baseline diastolic BP (hence, increasing the afterload by increase in the systemic vascular resistance). Due to the sudden surge in blood pressure, NTG infusion was started at the rate of 3ml/hour. It was continued for approximately 60 mins and tapered according to the blood pressure levels. Epidural top ups and inhalational agents were increased to deepen patients plane of anaesthesia along with iv analgesics and restricted fluid administration to keep the patient normotensive.

Once the graft was placed both the vessels were de-clamped which caused a sudden fall in the blood pressure due to rushing of blood into the central vessels. Inotropes were started - Inj. Noradrenaline 0.16mg/ml at 2ml/hr and adjusted according to blood pressure. Fluids were rushed and inhalational agents were tapered, and finally blood pressure was stabilised. Total intra operative blood loss was 1200ml. Two pints of PCV was transfused and 3200 ml if crystalloids was given.

Post-op patient was reversed using i.v Neostigmine 0.05mg/kg and Glycopyrrolate 0.008mg/kg extubated on table after achieving stable vitals and adequate tone and was shifted to S-ICU for observation.

FOLLOW UP AND OUTCOMES:

In the post operative period care was taken to provide adequate pain relief and timely monitoring of the vitals were done. As the cross-clamping time was less than 60 minutes, transient early post operative hypertension which is a common complication was avoided. Appropriate anti-thrombotic prophylaxis was given. Total duration of ICU stay was for 7days. Patient was discharged on post-op day 23 with stable vitals and is on regular follow up visits.

DISCUSSION:

Reperfusion following the aortic clamp removal is responsible for the paradoxical deleterious events that take place after the insult of ischemia that may have exceeded the original ischemic injury [4]. Primary hemodynamic responses to aortic unclamping include marked hypotension as a result of reduced afterload, hypoxia-led peripheral vasodilation, and a significant increase in the vasodilation and myocardial-depressant metabolites from areas that are located distally to the clamp [5]. Release of oxygen radicals and polymorphonuclear neutrophils causes platelet aggregation along with white cell activation that lead to microthrombi formation causing organ dysfunction [6],[7]. Moreover, activation of complement pathway causes tissue oedema of the hypo perfused organ.

Intestines are one of the most affected organs due to labile cells that cause bowel infarction, short-bowel syndrome, acute respiratory distress syndrome, systemic inflammatory response

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syndrome, and MOF [8],[9]. Damaged endothelium of the vasculature causes hemodynamic fluctuations and a decrement in organ perfusion. Post de-clamp GFR and renal blood flow can remain low for a brief period causing Acute renal failure [5].

Cardiac complications are detrimental especially in those with previous events of coronary artery disease. The enhanced inflammatory response causing an increase in cytokines, worsens cardiac function due to increase of local concentration of NO, causing an impaired adrenergic and cholinergic stimulation [10]. This in turn cause a decrease in the ventricular compliance and hence decrease the cardiac output and coronary perfusion [11],[12],[13]. During reperfusion, damaged endothelial cells and myocytes generate oxygen radicals and hastens the membrane deterioration process and thus aggravating endothelial injury and increased vascular permeability and finally causing raised microvascular filtration and myocardial oedema [14]. This in turn increases the stiffness of the cardiac chamber.

After the aorta is unclamped, there may also be an increase in the pulmonary vascular resistance and pulmonary arterial pressure, due the release of micro emboli into the pulmonary vascular system and the resultant increase in the vasoconstriction causing agents such as histamine and cytokines from the lungs promoting chemotaxis, local inflammation, and ARDS [5],[15].

Spinal cord hypoperfusion is an unpredictable event, especially in shock causing sensory and motor deficits associated with bladder or rectal incontinence with conservation of vibratory and proprioceptive sensation.

In case of AAA surgery, balanced anaesthesia is the appropriate choice. Protection from spinal cord hypoperfusion from cross clamping is very crucial. 30 minutes of maximum cross clamping time is said to be safe. Intra-op hypothermia of up to 32-34 degrees Celsius is recommended for neuroprotection.

CONCLUSION:

Surgery should be considered when the operative morbidity and mortality is less than the predicted aneurysmal rupture risk. Urgent referral to a surgical unit is indicated in aneurysms of 55mm diameter (or if symptomatic). Ischemia-reperfusion injury causes widespread changes in the human body following AAA repair. Open repair of AAA is a major high-risk surgical procedure undertaken in patients with significant co-morbidities and poor physiological reserve. To achieve good outcomes, risk factors should be optimized, the surgical intervention planned, pathophysiology understood, and organ protection strategies used. Effective communication and teamwork are essential.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

1. Ethical Approval: There is no requirement for ethical committee approval since the case report is on the anesthetic management of a patient posted for surgery. Consent for administering Anesthesia was taken from the parents prior to surgery.
2. Human and Animal Guidelines: Helsinki Declaration was strictly followed for involving human subjects in the study.

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3. Consent for Publication: Consent for Publication was obtained for this study.

CONFLICT OF INTEREST

The author reports no conflicts of interest in this work.

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DISCLOSURE

The author reports no conflicts of interest in this work.

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