CASE REPORTS

LEAKING ABDOMINAL AORTIC ANEURYSM REPAIR: AN ANAESTHETIC CHALLENGE

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INTRODUCTION

Abdominal aortic aneurysm (AAA) is a common degenerative condition affecting men three times more frequently than women [1]. The overall mortality rate from aneurysm rupture is in the order of 65-85% [2]. Anaesthetic management of these patients is extremely challenging because of the significant hemodynamic changes associated with the operation, frequent coexisting cardiovascular disease as well as the risk of spinal cord renal and mesenteric ischaemic injuries .The mortality rate for patients who survive to reach hospital and undergo emergency surgery is 36% compared with 6% for elective repair [3].

CASE REPORT

A 50 years old man was admitted in surgical ward of Combined Military Hospital Rawalpindi on 8th August 2008 with two days history of pain in right iliac fossa which later on spread to left iliac fossa and epigastric region. The pain was sudden in onset, colicky type with no aggrevating or relieving factors. It was not associated with any urinary or bowel complaint. The patient was known case of HTN and IHD for last 02 years and was taking anti hypertensive medication. He was incidentally diagnosed to be having abdominal aortic aneurysm two and half months back when he was admitted in Combined Military Hospital Kharian for headache & epigastric pain .Ultra sonography abdomen done at that time revealed abdominal aortic aneurysm for which he was referred to vascular surgeon Combined Military Hospital Rawalpindi. A contrast enhanced computerized tomography scan abdomen/chest was done which revealed large aneurysm of abdominal aorta measuring 7.6x 7.2cm arising below the origin of renal vessels up to the bifurcation. Along with pre op investigation, patient was sent to pre anaesthesia clinic for pre anaesthesia assessment. He was found to be having uncontrolled HTN and atrlial fibrillation (AF) for which consultation from medical specialist was sought and surgery was postponed. About one month later the patient presented to emergency department with sudden onset pain right iliac fossa spreading to whole of abdomen. Urgent ultra sound abdomen and computerized tomography scan abdomen revealed large abdominal aortic aneurysm with surrounding hematoma . On admission the investigations revealed a Haemoglobin =11.9g/dl, total leucocyte count =9.2x109/L with normal DLC, platelets=222x109/L, serum amylase = 53u/L, urea = 5.5 mmol/L creatinine = 87u mol/L urine-RE showed 4-6 RBCs / HPF. Electrocardio-graphy was normal. Prothrombin time =19/15 seconds, partial thromboplastin time= 34/33 seconds. Patient was kept nil per oral and I/V fluids were started. All his vital signs were monitored x2 hourly. 6 units red cell concentrate (RCC) and 10 units fresh frozen plasma (FFPs) were arranged. Informed written consent for high risk surgery was taken and operation theatre list for emergency aneurysmoraphy was sent.

The anaesthetic technique used for this patient was epidural combined with general anaesthesia. We used inj midazolam (0.1mg/kg) I/V ,inj maxolon (10mg) I/V and inj decadron (8mg) I/V as pre-medication .Epidural catheter was passed at L3/L4 space under sterile conditions. Right subclavain vein was cannulated with central venous pressuer (CVP) Line. Invasive B.P monitoring and large bore I/V access were secured. General anaesthesia was induced with thiopentone sodium (5mg/kg) and titrated doses of isoflurane with Nitrous oxide and Oxygen

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mixture. After confirming ventilation, intubation was done using Atracurium 0.5mg/kg with 8.0mm indewelling (ID) tracheal tube .Patient was monitored using cardiac monitor, pulse oximetry, invasive B.P, capnometer. Patient was also catheterized to measure urine output. The patient was in supine position.

Before aortic cross clamping (ACC), pulse, BP ,Urine output were measured.1gm/kg 20 % inj Mannitol was given for renal protection . B.P increased marginally by 15mm Hg to150/90mm Hg. Urine output was maintained at 1ml/kg/hr. Glycerl Trinitrate infusion was started to maintain B.P .

Blood loss during the surgery was around 4000ml which was replaced by judicious use of crystalloids, colloids, 4 units RCC , 6 units FFPs and 6 units platelets. Inj 10 % Calcium Gluconate 10ml was also given .When ACC was released BP fell to 60/40mmHg that returned to normal with fluid replacement and Norepinephrine infusion support. Core body temperature was maintained using warm I/V fluids.

Before closure of abdomen 10ml of plain Lignocaine was given in epidural space through already placed epidural catheter.

After completion of surgery, patient was reversed with Neostigmine 2.5mg+1mg Atropine. He was extubated once fully awake and later on shifted to Intensive Care Unit for post operative management.

On 1st post operative day, patient developed slight weakness of both lower limbs for which inj Decadron 8mg 8 hourly was started which was gradually tapered off over a period of 10 days. It started to resolve by itself and patient was able to stand and move his legs on 3rd post operative day. Chest physiotherapy was continued.

On 9th post operative day patient complained of infraumbilical swelling and pain abdomen. His peripheries were cold and clammy with pulse=100/min and BP=100/70mmHg. His bowel sounds were absent. He was immediately shifted to operation theatre for exploration of burst abdomen. Exploration was done under epidural anaesthesia. A loop of jejunum was protruding out of rent in linea alba. It was reduced and abdomen was closed with three tension sutures. He was discharged on 16th post operative day.

DISCUSSION

Aortic aneurysm usually occurs in sixth to seventh decade of life. It affects men more than women. About 50% of patients with abdominal aortic aneurysm and 70% of patients with thoraco abdominal aortic aneurysms are hypertensive. Following rupture of an AAA, only an estimated 38-64% of patients reach hospital alive [4]. AAA rupture can result in hemodynamic collapse and death before the patient reach hospital. In slow ongoing contained leaks, the patient present with abdominal mass, falling blood pressure and hemoglobin. Ruptured AAA is a surgical emergency and a brief and targeted preoperative assessment should be made. Proper premedication and sedation in from of opioids by intra muscular route and oral benzodiazepine may be administered to prevent anxiety, tachycardia and hypertension. Prophylactic antibiotics administered preoperatively results in a lower rate of post operative infections.

Induction of anaesthesia should be slow and controlled without hypertension because stress on the aneurysm can cause rupture. Heart rate should be maintained near baseline because myocardial ischaemia is often rate related. Balanced anesthetic technique for aneurysm induction usually involves a combination of an opioid ,low dose potent inhalational agent, a benzodiazepine and a long acting muscle relaxant. Standard monitoring should include pulse oximetry, capnography, body temperature and echocardiograpy. Foley`s catheter for urinary output is a must. Lead II is extremely helpful for diagnosing dysrhythmia and V-5 lead in detection of myocardial ischaemia. Continuous intra arterial monitoring of blood pressure should be done. Intra operatively, we also carried out serial arterial blood gases (ABG`s) and accordingly corrected the acidosis with sodabicarb.

Infra renal aortic cross clamping is associated with increased vascular resistance and a 30 % reduction in blood flow [4].During this period intra vascular volume should be maintained and replenished using crystalloids, colloids and if necessary blood transfusion. With clamp application, vasodilation therapy may be started as required. Cross clamping results in lowered total body oxidative metabolism and oxygen consumption as well as conversion to anaerobic metabolism by the ischaemic body mass distal to the clamp.Lactate concentration promptly rises during clamp period. A continuous infusion of sodium bicarbonate at the rate of 0.05meq/kg/min is recommended throughout the period of clamp. Bolus administration immediately after unclamping may further increase Pa CO2 and worsening of intracellular acidosis.[5] During aortic cross clamp there may be an increase in the CVP [4] however in our case it was not much because since the ACC was distal to coeliac artery, volume from the distal venous vasculature was redistributed into the splanchnic vasculature without any increase in the pre-load [6].

Factors detrimental to spinal cord function are reduced spinal blood flow, rate of neuronal metabolism, post ischaemia reperfusion. Maintaining a high proximal blood pressure, keeping the cross –clamp time <30min, avoidance of hyperglycemia and pharmacological agents such as lV thiopental Na and lV mannitol can minimize this [4]. Since hypothermia [7], hyperglycemia [7] and decrease in end tidal CO_2 greater than 15% during aortic cross clamping [8] are associated with a poor outcome these parameters were monitored and kept within normal limits. Prior to unclamping, vasodilators should be stopped. Ventilation should be increased in anticipation of increased acid load from the distal circulation .Severe hypotension occurs after aortic unclamping .Vasoconstrictor /inotropic support may be required [9].

Renal protection is of prime importance during aneurysm surgery .Clamping of the aorta for more than 1 hour carries high risk of acute renal failure. Thoraco abdominal aneurysm surgeries in which renal arteries and kidneys are exposed, the kidneys can be perfused with cold crystalloid solution or subjected to surface cooling .Pharmacologic renal protection includes use of diuretic drugs such as mannitol or furosemide.

Significant hemodynamic changes associated with aortic cross clamping and unclamping, major fluid shifts, spinal cord and renal protection were some of the challenges of the case. Anticipation of these changes, rigorous monitoring and control of BP, CVP, urine output and prompt correction of blood loss are key to proper case management.

REFERENCES

1. Mac Sweeney STM, O` Meara M, Alexander C, O` Malley MK, Powell JT, Greenhalgh RM.High prevalence of unsuspected AAA in patients with confirmed symptomatic peripheral or ceorebral arterial disease . Br J surg 1993; 80:582-4
2. Kniemeyer . HW, Kessler T Reber PU, Ris HB, Hakki H, Widmer MK. Treatment of ruptured aortic aneurysm a permanent challenge or a waste of resources? Prediction of outcome using multi-organ dysfunction score.Eur J Vasc Endovasc Surg 2000;19:190-6
3. AAA: A service in need of surgery? National confidential Enquiry into patient outcome and Death , 2005. Available from http:/www.ncepod.org.uk/2005report2/downloads/ AAA-report.pdf.
4. Robert Stoelting, Stephen Dierdofy; Anaesthesia and Co existing diseases 4th edition ;Aneurysms of the thoracic and Abdominal aorta; 146
5. Saleh SA, Crawford ES, Bamberger RA. Intra operative acid base management for the resection of thoraco abdominal aneurysms : A comparison of continuous infusion of Sodium bicarbonate versus bolus. Anaesthia Analg 1982; 61:213. (abstract)
6. Prabhat Sinha, Praveen Neema ; Abdominal Aortic Aneurysms Anaesthetic considerations ;Indian Journal of Anaesthesia ; Oct 2005;376-388
7. Millers Anaesthesia 6th edition 2070-2081
8. Boccara G, Jaber S, Eliet J, Mann C,Colson P.,Monitoring of end – tidal carbon dioxide partial pressure changes during infrarenal aortic cross clamping ; a non – invasive method to predict unclamping hypotension; Acta Anaesthesiol Scand .2001Feb; 45(2):188-93
9. Barash PG, Cullen BF.Stoelting RK, eds. Clinical Anaesthesia, 5th ed. Philadelphia: Lippincott Williams and Wilkins, 2006:1405-40.