MSA 631
Research Project

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Case scenario

- 76 y/o male, 156 lbs, 69 inches
- PMH: 4 cm Infrarenal AAA, Aortic Stenosis, Intracranial mass
- PSH: Cholecystectomy 1986
- Current medications: Metoprolol, Lisinopril, Zofran
- Procedure: Craniotomy for removal of tumor

Intracranial Mass

- Classification:
  - Primary tumors
    - those arising from the brain and it's coverings
  - Metastatic tumor
    - Most often from primary sites in lungs or breasts
    - Likely diagnosed with tests indicating the presence of more than one lesion

- Causes
  - Ionization radiation is an unequivocal risk factor that has been identified for glial and meningieal neoplasms
- Alleged Causes with conflicting data
  - cellular phones
  - exposure to high tension wires
  - use of hair dyes
  - head trauma
  - Dietary exposure to N-nitrosourea compounds

- Sign and Symptoms
  - Headache –considered classic symptom
  - Nausea/Vomiting
  - Mental changes
  - Visual disturbances
  - Decreased pupil reactivity
  - Systemic hypertension
  - Seizure

- Diagnosis
  - CT
  - MRI
    - Best diagnostic

- Treatment
  - Usually started on high dose steroid therapy to lessen edema
  - Surgery
  - Radiation
    - Increased neurological deficits during radiation are probably due to cerebral edema
    - Treat with corticosteroids

Aortic Stenosis

- Occurs as a congenital lesion but more commonly as an acquired disease. Stenosis may develop on a previously normal valve following Rheumatic Fever or from progressive calcification.
- AS represents a chronic systolic pressure load on the LV. The ventricle undergoes parallel duplication of muscle fibers in an attempt to compensate for the increase in tension. This results in increased wall thickness or concentric hypertrophy and some decreases in radius thereby normalizing wall stress.
- Contractility is preserved and EF is maintained at a normal range until late in the disease process.

- The enlarged muscle mass has increased basal myocardial oxygen requirements, while the demand per beat rises because of the elevated intraventricular systolic pressure.
- Simultaneously, with a capillary density often inadequate for the hypertrophic muscle, supply may further be compromised and total vasodilator may be impaired.
- This situation is compounded in the presence of coronary obstruction.
- Often, patients with aortic stenosis will present in heart failure

Aortic Stenosis: Signs & Symptoms

- The triad: angina, syncope, and congestive heart failure- which represent the progression of symptoms.
- Angina:
- ✓Results from both increased demand for and a decrease in the supply of coronary blood flow.
  ✓The increased muscle mass from LVH and high energy requirements generate increased systolic pressure combine to increase demands for CBF.
  ✓In addition, insufficient supply secondary to decreased perfusion gradients and a decrease in coronary vasculature in relation to myocardium= decrease myocardial blood supply.

•Therefore, up to 1/3 of patients with AS can have angina in the absence of significant CAD

Preoperative Evaluation and Preparation

- The role of premedication is to allay the anxiety of the impending surgical procedure thereby controlling the sympathetic outflow that may accompany the stress response.
- However, acute changes in HR, venous return, and systemic resistance can have particularly profound effects on patients with valvular disease
- Avoid decreases in SVR: because SV is relatively fixed, need to maintain SVR to maintain BP
- Maintain HR: avoid bradycardia (fixed SV), and avoid tachycardia (myocardial ischemia & reduced LV filling)
- Patients with AS may benefit from premedication by preventing unnecessary increases in HR. concern must be taken to ensure adequate venous return and preservation of sinus mechanism.
- Premedication includes: supplemental oxygen, and light doses of premedication titrated intravenously (while patient is under close monitoring)
- Antibiotics for endocarditis prophylaxis
- Patients with AS have a high propensity for arrhythmias
- Pts with AS have increased risk of perioperative cardiac complications >>> the risk is greatest in major surgery associated with significant hemorrhage or fluid shifts.

Anesthetic Technique

- For the patient undergoing cardiopulmonary bypass (CPB) and aortic valve replacement (AVR), GETA is the obvious choice
- Regional can be used if hemodynamic goals are met. Induction of GA is a time of particular risk
- Both narcotics and inhalation anesthetics can be safely administered- narcotic based anesthesia is usually well tolerated
- Some muscle relaxants can alter hemodynamics both from effects of histamine release including vasodilation and bronchospasm and through effects on rhythm
    - If high-dose narcotic anesthesia is used consider Pancuronium.
    - Hemodynamically ‘neutral’ relaxants such as vecuronium, rocuronium, and cis-atracuriumare safe

Intraoperative Management

- Patients with AS need the left ventricular filling obtained through a well-timed atrial contraction.
- Similarly, LVH renders the ventricle stiff and adequate preload is required.
- Reducing vascular tone will do little to relieve the fixed afterload increases from a stenotic valve but rather lower diastlic coronary perfusiongradients and should be AVOIDED.
- Patients with AS experiencing angina may require the administration of an alpha agonist (such as phenylephrine) to increase coronary perfusion pressure.
- Hemodynamic Goals:
    - HR= 60-70bpm,maintain NSR which is essential to CO. AVOID tachydysrrhythmias
    - Preload= Full
    - Afterload= maintain
    - Optimize contribution of atrial contraction to left ventricular filling
- Monitoring:
    - Standard ASA recommended monitors

Arterial blood pressure monitoring is recommended

PA catheter insertion-

Following induction of anesthesia and endotracheal intubation, a TEE probe should be inserted to confirm the valvular pathology and to assess ventricular function

Intraoperative Management

- Critical to management of patient with AS is avoidance of hypotension
- Low BP can initiate a cascade of events leading to cardiac arrest
- Patients with AS are particularly dependent on their atrial kick for adequate ventricular filling volume and can rapidly become hypotensive and ischemic following the onset of SVT or atrial fibrillation.
- Use caution in administration of vasodilators for ischemia because may reduce preload
- Treat ischemia by increasing coronary perfusion pressure and decreasing oxygen consumption
- Be prepared to treat arrythmias rapidly

Postoperatively

- Obtain ECG to look for ischemic changes
- Monitor for worsening s/s of aortic stenosis
- Analgesia as directed by procedure performed, which will help prevent tachycardia
- Consider whether ICU monitoring is warranted based on intraop course and type of surgery

Abdominal Aortic Aneurysm

- Dilation of the aorta > 50% of baseline located below the diaphram due to a pathological weakness in the vascular wall
- Pathophysiology: increased lipids, thrombosis, calcification, ulcerations damage tunica intima leading to adventitial inflammatory infiltrate causing a degradation of the tunica media by proteolytic processes
- Most often involves all layers of aortic wall
- AAA most common form of aortic aneurysms due to less elastin and less vasa vasorum than thoracic aortic. Wall tension is also higher.
- Can be suprarenal, infrarenal, and pararenal. Infrarenal is most common with approx 90% being below renal arteries.
- More common in men who smoke and are between the ages of 65-75
- Law of Laplace: wall tension (T) is proportional to pressure (P) x radius (r).

Classifications of AAA

- Two classifications: The Debakey (I-III) and The Standford (A or B)
- Standard: classifies aortic dissetions as type A or B
  - type A: includes all cases in which ascending aorta is involved
  - type B: includes all cases in which descending thoracic aorta involved
- Debakey: classifies aortic dissections as types I-III
  - type I: intimal tear usually in the proximal ascending aorta involving aorta, arch, and variable lengths of the descending and abdominal aorta
  - type II: Dissection is confined to the ascending Aorta
  - type III: may be confined to descending thoracic Aorta (IIIa) and may extend into the abdominal aorta and iliac arteries (IIIb)

Diagnosis of AAA

- Observation of pulsating mass in the absence of other causes
- Bruit heard over abdomen
- Abdominal Ultrasonography detects AAA with a sensitivity of close to 100%
- CT is a very accurate diagnostic tool which can be more accurate than ultrasound in estimating the size
- MRI superior to ultrasound and CT in the accuracy of measurements without exposing pt to radiation or contrast

Preoperative Evaluation

- Assess cardiac function: stents, EF, cath report, echo, and exercise tolerance
- Assess for abdominal pain as this may be indication of tearing or rupture
- CAD is a common co-existing disease, 30-40%.
- HTN is single most important risk factor for AAA disection - be aware of medications and pre-op BP
- Assess renal function: BUN, Cr, GFR - due to placement of aortic aneurysm
- Assess H and H for active bleeding
- Other co-existing diseases: COPD, DM, and Elderly
- Other predisposing factors: cystic medial degeneration of aorta, pregnancy, disorders of connective disorder (Marfan syndrome, Ehlers-Danlos syndrome)

Treatment of AAA

- Surgery is usually recommended for all AAA > 5cm with a low mortality rate in elective surgeries
- Studies have shown that 25%-41% of AAA >5 cm rupture within 5 yrs
- Smaller anuerysms have a decrease chance of rupture
- Alternative to surgery include an endovascular placement of a stent via ateriotomoy through the leg
- Medical therapy involves tight control of BP at normal or low normal ranges

S/S of Rupture AAA

- Classic triad : hypotension, back pain, and pulsating abdominal mass
- Triad only presents in 1/2 of patients with rupture AAA
- S/S may easily be confused as other diseases like renal colic, diverticulitis, or GI bleed
- Most rupture into left retroperitoneum and do NOT exsanguinate due to clotting and the tamponade effect
- If rupture then must be surgically repaired. Be careful in fluid resuscitation, can exsanguinate

Anesthetic Considerations

- Control fluid volume
- No single anesthetic drug or technique is ideal
- Consider Etomidate if CAD present
- Maintain BP at normal to low levels depending on current therapy
- Consider BP control with beta blockers or nitrates
- Consider beta blocker and opiods prior to DVL
- BP control post op is of great concern
- Avoid hypothermia as shivering causing vasoconstriction leading to HTN
- Assess all cardiac, pulmonary, and renal test thoroughly

Metoprolol

- Cardioselective B1 blocker
- Onset: immediate
- DOA:2-4 hrs
- Half life: 3-4 hrs
- IV 2.5-5mg q 5 min up to 15 mg
- PO 25-100 mg
- Metabolized: liver
- Prevents inotropic and chronotropic responses to beta-adrenergic stimulation. (prevents remodeling)
- Bronchodilator, vasodilator, and metabolic effects of B 2 receptors remain intact
- At large doses metoprolol can become non cardioselective and antagonize B2 receptors

Lisinopril

- Mechanism of Action: inhibits angiotensin converting enzyme, interfering with conversion of angiotensin I to angiotensin II
- Dose: 10mg
- Onset: 60 min
- Peak: 2-4 hrs
- DOA: 18-30 hrs
- Excreted 100% unchanged by kidneys
- Instruct patient to hold preoperatively in order to decrease intraoperative HOTN

Odansetron (Zofran)

- Serotonin 5HT3 receptor antagonist
- Uses:
  - Prophylaxis and treatment of chemo and radiation-induced N/V
  - Given intraoperatively to prevent PONV
  - Not effective in treatment of motion-induced N/V
  - Not effective in treatment in postop N/V owing to vestibular stimulation
  - Treatment of pruritus associated with neuroaxial morphine

- Side Effects
  - Headache
  - Diarrhea
  - Slight prolongation of QTc interval, but not to the same level of concern as droperidol
- Dose
  - Adult 4-8 mg PO or IV
  - Pediatric
    - Oral 0.15 mg/kg
    - IV 0.05-0.15 mg/kg (Max 4 mg)

Craniotomy

- Preop testing
- Preop labs
- Preop medications
- Preop complications
- Type of anesthesia
- Airway
- Induction
- Positioning
- Maintenance of anesthesia
- Intraoperative medications
- Fluid Therapy, hemodynamics
- Intraop monitoring

Preop Testing

- CT- evaluate for midline shift
- Monitor for signs/symptoms of increased ICP

Preop Labs

Preop Medications

- Avoid opiods
  - Ventilatory depression increases CO_2 resulting in increased ICP
  - CNS depressant can mask alterations in LOC
- Oral Benzodiazepines in alert adults relieves anxiety without ventilatory depression
- Anticholinergics and H_2 receptor antagonists administration is not influenced by ICP

Preoperative Complications

- Edema formation leads to increased ICP
- Herniation of brain stem
  - Caused by increased ICP
  - ‘Cushings Triad’- HTN, bradycardia, and respiratory irregularity

Type of Anesthesia

- General anesthesia is almost always used
- MAC is used on rare occasions
  - When surgeon needs to assess motor or sensory function during resection of tumor adjacent to critical motor or speech areas

Airway

- If in stereotactic frame or difficult intubation is anticipated use awake fiber optic intubation

Induction

- Adequate depth of anesthesia and profound skeletal muscle paralysis is necessary
- Drugs that produce reliable onset of unconsciousness with minimal effects on CBF
  - Thiopental (3-5 mg/kg)
  - Etomidate (0.2-0.4 mg/kg)
  - Propofol (2-3 mg/kg)
- Large doses of NDMB to facilitate intubation
  - 3 times ED95
- Succinycholine may be associated with modest, transient increases in ICP
- Lidocaine 1.5 mg/kg IV one minute before DL
- Short acting opioids, such as Remifentanil (2-3 mcg/kg)

Positioning

- Depends on location of lesion
- Supine- elevate head 10-15 degrees
- Avoid excessive flexion or rotation of head
- Sitting positing- offers excellent surgical exposure and decreased blood loss, but has risks involved
- Lateral and prone as alternatives

Maintenance of Anesthesia

- Often combo of drugs
  - Nitrous oxide (<50%)
  - Volatile anesthetics (0.6 MAC)
  - Opioids (Remifentanil infusion 0.05-0.1 mcg/kg/min)
  - Propofol (75-100 mcg/kg/min)
  - Pancuronium/ Rocuronium (10 mcg/kg/min)

Fluid Therapy

- Goal to minimize the risk of adversely influencing the ICP
- hypertonic salt solution such as 5% glucose in lactated Ringer's solution is appropriate
- Rate should not exceed 1-3 mL/kg/hr perioperatively
- Blood loss should be corrected with PRBC, whole blood, or colloid solutions NOT with large volumes of crystalloids

Hemodynamics / Control ICP

- Peripheral vasodilating drugs may increase ICP and CBF despite accompanying decreases in systemic BP
  - Nitroprusside
  - NTG
- Mechanical hyperventilation used with goal of decreasing $PaCO_2$ to near 30 mmHg
- Avoid hypotension
- Avoid positive end-expiratory pressure
- Mannitol 0.5-1 g/kg $\pm$ furosemide 0.3 mg/kg
- Lumbar CSF drain

Intraop Monitoring

- Arterial Line
- ICP
- Capnography
- Foley Catheter
- CVP
- Doppler transducer
- ECG

Intraop Monitoring cont…

- Brain stem auditory evoked responses for acoustic neuromas
- Visual evoked responses for parasellar tumors
- Somatosensory evoked responses for parenchymal and brain stem lesions

Intraop Complications

- Venous Air Embolism
  - Whenever operative site is above the level of the heart
  - Pressures in the veins are subatmospheric

Venous Air Embolism

- Intracranial surgery are at increased risk
  - Operative site above level of heart
  - Veins of skull may not collapse due to their attachment to bone or dura
- Common sites of entry
  - Cut edge of bone
  - Burr holes
  - Where veins are held open by bone

VEA Pathophysiology

- Air enters right atrium
- Interference with blood flow to pulmonary artery
- Death usually secondary to acute cor pulmonale, CV collapse, arterial hypoxemia
- Systemic air through Right-to-Left shunt
- Neurological damage with air embolism to brain
- MI and VF from air obstruction to coronary arteries

VAE Detection

- Doppler trandducer- most sensitive
- Sudden decrease in ET CO_2
- Increased RA and PA pressures- reflect size
- Sudden attempts by patient to spontaneously breath during controlled ventilation
- Hypotension, tachycardia, cardiac dysrhythmias, cyanosis- late signs
- Millwheel murmer- late sign

VAE Treatment

- Surgeon identify and occlude sites of air entry
- Turn on Left side
- Aspirate air through Right Atrial cathether
- Discontinue Nitrous Oxide
- Use Oxygen and PEEP to increase venous pressure
- Transfer to hyperbaric chamber

Emergence

- Prevent increased ICP !!!
  - Titrate Beta blockers or vasodilators
  - Ensure full NDMB reversal
  - Prophylactic antiemetics (Zofran 4 mg) 30 min prior to extubation
  - Extubation
    - Lidocaine 0.5-1.5 mg/kg to attenuate response to ETT as awakens
    - If alert preop, possible nasal airway and deep extubation
  - Leave intubated if hypothermic (<34^{o} C)
  - HOB up 30^{o} for transport

Postop Care

- ICU or close observation unit for 1-3 days
- May need antihypertensives to control BP
- Meperidine minimizes postop shivering
- Monitor for postop complications
  - Seizure
  - Neurological deficits
  - Edema
  - Increased ICP
  - Tension Pnuemocephalus
  - Hemorrage

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