Carcinoid Tumors: Anesthetic Considerations

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Objectives

- Describe the pathophysiology of carcinoid tumors and carcinoid syndrome.
- Describe signs and symptoms of carcinoid syndrome and carcinoid crisis.
- Discuss current recommended anesthetic management of patients with carcinoid disease.
- Describe how to prevent and treat a carcinoid crisis in the perioperative period.
Case Study

- 61 year old man with metastatic carcinoid disease admitted to teaching hospital for lethargy, sweats, & malaise.
- Extensive liver involvement
- Received his usual monthly dose of lanreotide 120 mg sq on time on admission
- For 2 days had over 20 abdominal exams by physician trainees.
- 2nd evening of hospital stay, patient developed STach, hypoxia, hypotension, hypoglycemia.

Morrisroe, et al., 2012
Case Study

- Patient was transferred to ICU where he continued to deteriorate
- Echo confirmed severe carcinoid heart disease
- Carcinoid Crisis was suggested as diagnosis
- Octreotide 100 mcg/hour IV and decadron started
- Resulted in rapid improvement
- Rapid response from octreotide treatment confirmed diagnosis of carcinoid crisis precipitated by repeated abdominal exams, despite receiving his lanreotide on time.

Morrisroe, et al., 2012
Case Study

- Excellent example of potential mortality associated with unrecognized crisis
- Crisis that occurred without surgical or procedural stimulation
- Crisis may still occur despite current somatostatin-analog treatment
Carcinoid Tumor Incidence

- Rare, but incidence of diagnosis is increasing
- Current incidence is between 0.2-10 in 100,000 people
- Autopsy incidental findings as high as 8%
- 75-80% of patients with carcinoid syndrome have small bowel tumors
- 5 year survival rate overall 80%
  - Survival rate decreases to 18-19% with distant metastasis
- 25% of carcinoid tumors produce carcinoid syndrome and <10% of people with tumor develop syndrome

Dierdorf, 2003; Quaedvlieg, 2001; Vaughn & Brunner, 1997; van der Leely & de Herder, 2005
Carcinoid Pathophysiology

- Neuroendocrine tumors that arise from GI tract or lungs
  - other rare sites
- Tumors synthesize, store, and release up to 40 bioactive mediators
  - Amount and effect to a specific symptom is unclear.
- Most prominent are serotonin, tachykinins, kallikrein, & prostaglandins
- Liver usually inactivates mediators secreted into the portal circulation
  - GI tumors usually only develop syndrome with distant metastasis

Melnyk, 1997; Modlin, et al., 2005
<table>
<thead>
<tr>
<th>Organ</th>
<th>Symptom</th>
<th>Frequency, percent</th>
<th>Putative mediator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Flushing</td>
<td>85</td>
<td>Kinins, histamine, kallikreins, other</td>
</tr>
<tr>
<td></td>
<td>Telangiectasia</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pellagra</td>
<td>7</td>
<td>Excess tryptophan metabolism</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>Diarrhea and cramping</td>
<td>75 to 85</td>
<td>Serotonin</td>
</tr>
<tr>
<td>Heart</td>
<td>Valvular lesions</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Right heart</td>
<td>40</td>
<td>Serotonin</td>
</tr>
<tr>
<td></td>
<td>Left heart</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Respiratory tract</td>
<td>Bronchoconstriction</td>
<td>19</td>
<td>Unknown</td>
</tr>
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</table>
## Products of carcinoid tumors

<table>
<thead>
<tr>
<th>Category</th>
<th>Substances</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amines</strong></td>
<td>Serotonin, 5-Hydroxytryptophan, Norepinephrine, Dopamine, Histamine</td>
</tr>
<tr>
<td><strong>Polypeptides</strong></td>
<td>Kallikrein, Pancreatic polypeptide, Bradykinin, Motilin, Somatostatin, Vasoactive intestinal peptide, Neuropeptide K, Substance P, Neurokinin A, Neurokinin B, Corticotropin (ACTH), Gastrin, Growth hormone, Peptide YY, Glucagon, Beta-endorphin, Neurotensin, Chromogranin A</td>
</tr>
<tr>
<td><strong>Prostaglandins</strong></td>
<td></td>
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Tryptophan Metabolism

- 1% of dietary tryptophan is converted into serotonin
- In carcinoid syndrome, 70% of tryptophan is converted
- Serotonin is metabolized into 5-hydroxyindoleascetic acid (5-HIAA)
- Some foregut (gastric, bronchial) carcinoids lack aromatic amino acid decarboxylase
  - Produce 5-hydroxytryptophan and histamine instead
  - Hindgut (distal colon and rectum) rarely secrete serotonin or any other hormones and are unassociated with hormonal syndromes even when metastatic

Feldman, 1987; Swain, Tavill, & Neale, 1976
Tryptophan and serotonin metabolism

Pathways of tryptophan and serotonin metabolism in the carcinoid tumor cell. Patients with the carcinoid syndrome often have increased levels of 5-HIAA excretion in the urine and serotonin uptake by platelets; urinary serotonin excretion is either normal or slightly increased.
Tryptophan Deficiency

- The 70 fold increase in serotonin synthesis may result in tryptophan deficiency
  - Decreased protein synthesis
  - Muscle wasting
  - Hypoalbuminemia
  - Nicotinic acid deficiency
  - Pellagra
Pellagra

- Rough scaly skin
- Glossitis
- Angular stomatitis
- Mental confusion

Swain, Tavill, & Neale, 1976
Serotonin

- Most likely cause of diarrhea in carcinoid syndrome
  - Stimulates intestinal secretion, motility, and inhibits absorption
  - Stools up to 30/day, watery and non-bloody
  - Occurs in 80% of carcinoid syndrome patients
- Stimulates fibroblast growth and fibrogenesis
  - Can lead to peritoneal and cardiac valvular fibrosis
  - May be cause of severe abdominal cramping

Hendrix, et al., 1957; von der Ohe, et al., 1993; Lie, 1982
Signs and Symptoms
Flushed and flushing

- Sporadic flushing is the clinical hallmark of carcinoid syndrome
- Occurs in 85% of patients with carcinoid syndrome
- Begins suddenly, lasts 30 seconds to 30 minutes
- Involves face, neck, and chest: red to purple color
- Mild burning sensation
- Potential mediators are bradykinins, prostaglandins, tachykinins, substance P &/or histamine

Flushed

- Maybe accompanied by severe hypotension and tachycardia
- As disease progresses, flushing lasts longer, and may become more diffuse and cyanotic
- May be triggered by:
  - Certain foods & alcohol
  - Defecation
  - Emotional events
  - Mechanical stimulation
  - Anesthesia
    - Episodes triggered by anesthesia may last hours and present with severe hypotension

Wheezing

- 10-20% of patients with carcinoid syndrome have wheezing and dyspnea, often during flushing
- Should not be mistaken for bronchial asthma
- Treatment with beta agonists can trigger intense, prolonged vasodilation (controversial)

Warner, 1985
Venous Telangiectasia

- Purplish, vascular lesions
- Appear late in carcinoid syndrome, due to prolonged vasodilation
- Most often on nose, upper lips, and malar areas
Figure 1: A 72-year-old man with carcinoid syndrome, showing flushing, cyanosis and telangiectasia.
Atypical Carcinoid Syndrome

- Bronchial & carcinoids outside GI tract can release substances directly into systemic circulation & are not immediately cleared by the liver
  - syndrome can be present without mets
- Bronchial carcinoids exhibit atypical carcinoid syndrome
  - **flushing** and **diaphoresis**
- Other symptoms are tremor, periorbital edema, lacrimation, salivation, disorientation, hypotension, tachycardia, diarrhea, dyspnea, asthma, oliguria, and edema

Melmon, Sjoerdsma, & Mason, 1965
Atypical Carcinoid Flush

- Patchy, sharply demarcated, serpiginous, and cherry red
- Intense Pruritis
- Severe and prolonged, lasting days
- Also associated with primary gastric carcinoids that produce histamine
- May be treated with $H_1$ & $H_2$ antagonism

Vaughn & Brunner, 1997; Roberts, Marney, & Oates, 1979
Carcinoid Heart Disease

- Carcinoid syndrome can lead to carcinoid heart disease in up to 66% of patients
- Plaque-like, fibrous endocardial thickening involving usually the right side of the heart
  - Causes retraction and fixation of leaflets of the tricuspid and pulmonary valves
Carcinoid heart disease

A) Pulmonary valve (autopsy specimen, viewed from above). Diffuse carcinoid plaques have caused thickening and retraction of all three cusps and appreciable constriction of the annulus (valve ring). The result is a small fixed triangular orifice that was both stenotic and regurgitant clinically. B) Normal pulmonary valve, for comparison with (A) (autopsy specimen, viewed from above). All three cusps are thin and coapt (come together) centrally, and the annulus (valve ring) is neither constricted or dilated.

Courtesy of Dr. William D. Edwards, Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, Minnesota.
Carcinoid Heart Disease

- Tricuspid regurgitation is most common
  - Tricuspid stenosis, pulmonary regurgitation & stenosis also occur
- Left-sided carcinoid heart disease occurs in less than 10% of patients and is usually associated with atrial right to left shunt (PFO) or primary bronchial carcinoid
- Patients should have thorough assessment to look for signs of cardiac dysfunction

van der Lely & de Herder, 2005; NCCN, 2011
Signs of Carcinoid Heart Disease

- Reduced exercise tolerance
- Orthopnea
- Paroxysmal dyspnea
- Peripheral edema
- Coronary artery spasm may occur with flushing episodes
Severe Carcinoid Heart Disease with Pulmonary Stenosis

- Creates high right-sided heart pressures
- Tricuspid Regurgitation
- Hepatomegaly
- Pulsatile liver
- Hepatic resection may be impossible without large blood loss due to high venous pressures and hepatic vein pressures
  - Seriously consider pulmonary valve surgery before hepatic resection in this patient population

Powell, Muhktar, & Mills, 2011
Carcinoid Crisis

- Results from overwhelming amount of bioactive mediators from tumor
- Predominant symptom is wide blood pressure fluctuations
  - Usually hypotension
  - Serotonin may cause vasoconstriction or vasodilation, therefore hypotension or hypertension may be seen
- Calcium and catecholamines may provoke release of mediators from tumor
  - Typical anesthesia management of hypotension may escalate the crisis

Vaughan & Brunner, 1997; Woodside, Townsend & Mark-Evers, 2004; Déry, 1971; Memom & Nelson, 1997
Carcinoid Crisis Case Report (Marsh, et. al., 1987)

- Pt had 5-HIAA confirmed carcinoid syndrome presenting for resection of primary tumor in terminal ileum.
- No mention of pretreatment with steroids, H₁ or H₂ antagonists, or other 5-HT₂ antagonist
- Induction of anesthesia uneventful
- 5 minutes after induction, the abdominal wall was exposed and vigorously scrubbed with betadine.
- Flushing occurred and BP unattainable by manual BP cuff, no peripheral pulses, HR 140.
- In 10 minutes, patient received 1000ml D5LR, 140 mcg phenylephrine, 1 gram CaCl, 1 mg epinephrine
VTach occurred after epinephrine was given
- Resolved to sinus rhythm after 50 mg lidocaine
- Radial cut-down site bloodless
- Patient was cyanotic
- Only carotid pulses palpable
Marsh, et al. Conclusion

- Permission was obtained for use of experimental drug, Sandoz 201-995
- 50 mcg IV was given twice
- Within 40 seconds, peripheral flow resumed, cut down site bled, arterial puncture was accomplished
- Systolic arterial pressure maintained throughout the remainder of lengthy surgery
- Sandoz 201-995 is known today as octreotide
Anesthetic Management of Carcinoid Tumors and Syndrome
Somatostatin-analogues

- Somatostatin is a 14-amino acid peptide that inhibits the secretion of a broad range of hormones.
  - Acts by binding to somatostatin receptors, which are expressed in the majority of carcinoid tumors
- Somatostatin analogs (octreotide and lanreotide) bind to somatostatin receptors on tumor cells
  - Highly effective at inhibiting the release of bioactive amines

Somatostatin-analogues

- Octreotide (also called sandostatin) is an octapeptide analog of somatostatin
  - Longer duration of action than endogenous somatostatin
- Flushing and diarrhea are significantly improved in >80% of carcinoid syndrome patients
- Increasing the dose of octreotide usually controls refractory symptoms
- Other somatostatin-analogues
  - Octreotide LAR (monthly injection)
  - Lanreotide (monthly injection also available)
  - Pasireotide (not yet available in US or other countries)

Preoperative Assessment

• Search for preoperative comorbidities:
  • Obstruction
  • Malnutrition
  • Dehydration
  • Anemia
  • Electrolyte imbalance
  • Diarrhea
  • Flushing and other symptoms of carcinoid syndrome
  • Cardiovascular history upmost importance due to higher risk of right-sided & biventricular heart disease

Powell, Muhktar, & Mills, 2011
Communication

- Successful anesthetic management dependant on pre-op optimization
- Good communication between endocrinologist, anesthesia provider, and surgeon
- Seek advice from centers with more expertise in managing carcinoid syndrome
- Patients should be viewed as having multi-system disease
- Require thorough pre-planning and post-op managements in ICU environment, by clinicians familiar with carcinoid management

Powell, Mukhtar, & Mills, 2011; Mancuso, et al., 2011
Perioperative Risk

• Kinney, et al. (2001) published a retrospective study of 119 carcinoid syndrome patients that underwent elective resection by same surgeon in same institution over 14 years
• Perioperative deaths or complications occurred in 15 (12%) patients
  • Flushing, sustained hypotension, bronchospasm, acidosis, VT
  • Higher frequency of carcinoid heart disease than in those who did not have complications (53% vs 15%, \( P = 0.002 \))
  • Urinary 5-HIAA output was significantly higher in this group
    • All 119 patients had elevated 5-HIAA
    • 3 patients died >10 days post op d/t comorbidities or sepsis
      • 2 had severe carcinoid heart disease

Kinney, et al., 2001
Preoperative Assessment

- Carcinoid Syndrome symptoms should be controlled with necessary somatostatin analogs before treatment directed at tumor debulking in order to prevent crisis
  - Recheck urinary 5-HIAA levels after control of symptoms before proceeding to surgery
- Multiple current articles advocate for pre-op echo in all patients with carcinoid syndrome for correct management of cardiac disease

Vaughan, Howard, & Brunner, 2000; Yates, 2010; Powell, Muhktar, & Mills, 2011
Preoperative Assessment

- Kinney, et al. (2001) concluded that presence of carcinoid heart disease and high pre-op urinary 5-HIAA were significant risk factors for perioperative complications, including death.

- Veall, et al. (1994) reviewed 22 records of patients with carcinoid syndrome undergoing laparotomy and found no association between high 5-HIAA levels and perioperative morbidity.
  - Poor predictor of cardiovascular stability.
  - One patient had lower abnormal 5-HIAA level, but very severe hypotension.
Preoperative Assessment

• Mancuso, et al. (2011) provide a current article on anesthetic considerations for carcinoid syndrome based on literature review and unpublished data from LSU

• Concludes that the severity of carcinoid syndrome does not predict the severity of perioperative complications
  • Urinary 5-HIAA provides an indicator of disease progression, not risk of severity of intraoperative response to tumor manipulation

• Chemistry, urinary 5-HIAA, CBC, glucose, EKG, low threshold for cardiology consult
Carcinoid Crisis Prophylaxis

- Even if carcinoid tumor patient lacks carcinoid syndrome, prophylaxis with octreotide is essential because stimulus that may release vasoactive mediators is much greater than daily living stimuli.
- Tumor activity must be minimized at least the day before surgery with octreotide.
- Multiple articles recommend starting a continuous infusion at 50 mcg/hour at least 12 hours prior to surgery.
- Increase octreotide dose until symptoms of carcinoid syndrome are abated before proceeding with surgery.

Powell, Mukhar, & Mills, 2011; Yates, 2010; Ramage, et al., 2005
Carcinoid Crisis Prophylaxis

• In their case study, Yazbek-Karam (2009) state pre-op SQ doses of octreotide used did not prevent crisis during RFA, & recommend pre-op octreotide infusions of 100-300 mcg/hr before manipulation of liver mets.

• Yates (2010) recommends starting 12 hours before surgery and continuing for a minimum of 48 hours postoperatively.

  • especially important in patients with carcinoid heart disease that may not be able to compensate for severe hemodynamic changes.
Carcinoid Crisis Prophylaxis

- Veall, et al. (1994) found that after octreotide was given, hypotension less severe and more responsive to additional boluses of octreotide.
- Vaughan, Howard, & Brunner (2000) recommend additional prophylaxis of chlorpheniramine & ranitidine for $H_1$ & $H_2$ antagonism to prevent any histamine effects from tumor manipulation.
Avoiding Sympathomimetics

- Epinephrine and norepinephrine could be hazardous
  - Norepinephrine shown to activate kallikrein in the tumor and lead to synthesis and release of bradykinin paradoxically worsening vasodilation and hypotension
    - However hypertensive responses have been seen
- Powell, Muhktar, & Mills (2011) recommend cautious small doses of phenylephrine
- Vaughan & Brunner (1997) feel phenylephrine or other adrenergic agonist are dangerous as they may trigger further peptide release
  - In other case studies, phenylephrine had no effect on hypotension
- In Zimmer, et al. (2003), most extreme hypotensive and tachycardic event occurred after administration of atropine, phenylephrine & theoadrenaline-caffedrine

Powell, Muhkar, & Mills, 2011; Cortinez, 2000
Somatostatin Does Not Prevent Serotonin Release and Flushing during Chemoembolization of Carcinoid Liver Metastases

Zimmer, Christian; Kienbaum, Peter; Wiesemes, Richard; Peters, Jürgen
doi:

Fig. 1. Hemodynamic measurements and serotonin (5-HT) concentrations in whole blood and plasma before and during chemoembolization of carcinoid liver metastases. Flushing episodes and pharmacologic interventions are marked by arrows, and chemoembolizations are marked by bars. Several flushing episodes with either hypertension or hypotension occurred, mostly during or shortly after embolization. Whereas 5-HT whole blood concentrations were stable, 5-HT plasma concentrations increased after the first embolization and markedly fluctuated. Atropin was given at the request of the radiologists for a better embolization result with higher heart rate.
Anesthesia Technique

- Anesthetic goal is to prevent mediator release that may be caused from stress, induction of anesthesia, intubation, and tumor manipulation
  - Stable, controlled conditions
  - Avoid BP variation
  - Stable induction
  - Adequate depth of anesthesia prior to intubation
  - Maintenance of anesthesia and analgesia
  - Hypothermia may trigger release as well
- Invasive monitoring should be used for quick detection of cardiac instability

Mancuso, et al., 2011; Powell, Muhktar, & Mills, 2011
Anesthesia Technique

- GA usually plan of choice to prevent sympathectomy that accompanies neuroaxial anesthesia
  - Epidural and subarachnoid blocks have been successful in patients with carcinoid syndrome
- SAB has been successful with preoperative fluids, octreotide, and low dose spinal anesthetic supplemented with low dose intrathecal opioids
- Hypotension must be avoided to prevent release of tumor mediators and treatment with sympathomimetic agents that may further release tumor mediators

Orback-Zinger, Lombroso, & Eidelman, 2002; Vaughan & Brunner, 1997; Mancuso, et al., 2011
Anesthetic Technique

- Choice of technique should be made based on familiarity and preference
- TIVA and inhalational techniques have both been successful
- Morphine and atracurium should be avoided due to histamine release
- Abdominal defasciculations with succinlycholine may cause release of peptides from tumors
- Remifentanil (0.05-0.2 mcg/kg/min) may be ideal for optimized intubation, titratable analgesia, and intraoperative BP control
  - Benefits must be balanced by risk of hypotension & bradycardia

Powell, Muhkar, & Mills, 2011
Epidural Anesthesia

- Thoracic epidural insertion before induction of GA recommended for stable analgesia to reduce risk of carcinoid crisis triggered by pain or stress intra-op and post-op
- Drug volumes and concentrations should be titrated cautiously to prevent hypotension that would need to be treated with sympathomimetics
  - Hypotension and sympathomimetics both may lead to mediator release

Powell, Muhkar, & Mills 2011
Hepatic Resection

- Carries the most significant risk for carcinoid patients
- May be especially useful in hepatic resection to maintain low CVP during clamping of hepatic artery and portal vein to avoid backflow into liver and venous bleeding, which would further exacerbate hypotension
Anesthetic Equipment

- Large bore IV access
- Fluid warmers
- Rapid infusion system
- Invasive monitoring depending on local resources & cardiac involvement
  - A-line
  - CVP
  - TEE

Powell, Muhkar, & Mills, 2011; Vaughn, Howard, & Brunner, 2000
Intraoperative Octreotide

- 0/45 patients receiving octreotide experienced significant intra-op complications compared with 8/73 pts who did NOT receive octreotide.
- 41 (38%) patients received intra-op octreotide
  - Median dose 350 mcg
  - Range 30-4000 mcg
- Analysis demonstrated that patients receiving octreotide intra-op experienced significantly fewer intra-op complications
Intraoperative Octreotide

- Drug of choice to treat hypotension in carcinoid crisis in addition to fluid boluses
  - Should be ordered, available, and administered in the OR prior to induction
- Treats hemodynamic instability in 15 seconds to 10 minutes
- 10 mcg SQ/IV until effect minimum published dose
- 500 mcg IV until effect in other publications
- At LSU, drips up to 500 mcg/hour have been given in over 300 major resection cases without crisis or adverse effect
  - Also up to 1 mg IV boluses for crisis

Mancuso, et al., 2011; Koopmans, et al., 2005
Octreotide Adverse Effects

- Only serious side effect is prolonged QT interval, therefore avoid other medications with same effect (e.g. ondansetron)
Treating Hypotension

- If Hypotension is refractory to high-dose octreotide and fluids, decrease anesthetic depth, and have surgeon halt tumor stimulation (Mancuso, et al. 2011)
  - Fluids if cardiac function is good
- Vasopressin have been used with limited success.
- Use Aprotinin (kallikrein inhibitor) as back up when octreotide fails to treat hypotension during tumor manipulation
  - Also with limited success

Treating Hypertension

- Octreotide
- Deepen anesthesia
- Beta blockers
- Ketanserin (serotonin receptor antagonist)
- Labetalol infusions
- Alpha blockade

Mancuso, et al., 2011; Powell, Muhkar, & Mills, 2011
Treating Bronchoconstriction

- If refractory to octreotide, antihistamines and nebulized iprotropium should be used instead of beta agonists to prevent mediator release.

Mancuso, et al., 2011
Postoperative Management

- Should be monitored for mediator release due to delayed recovery from anesthesia from high serotonin levels
- Undetected metastases may still secrete peptides
- Preoperative drug therapy with octreotide should be continued and possibly reduced slowly over the first week post op
- Fluids and electrolytes should be monitored closely due to large fluid shifts
- Analgesia to prevent excess sympathetic activity and stress prevents post-op crisis
- Fentanyl and epidural analgesia has been used with success

Mancuso, et al., 2011
Postoperative Management

- Powell, Muhktar, & Mills (2011) also recommend ICU care with invasive monitoring
- IV and then SQ octreotide for at least 48 hours to minimize post-op crisis
Further Study Needed

- Further study is needed to compare prophylaxis regimens.
- Is SQ dosing every 8 hours as effective as continuous IV administration?
- Is octreotide still the most effective compared to other newer somatostatin analogues?
Conclusions

- Anesthesia providers need to be aware of carcinoid syndrome symptoms for patients presenting for resection of tumors with unknown pathology.
- Providers need to know clinical picture of carcinoid crisis to appropriately manage it.
- Patients with carcinoid syndrome should have symptoms well controlled with somatostatin-analogue prior to surgery to prevent complications.
- Thorough preoperative assessment including cardiology consult when indicated is essential.
Conclusions

- Octreotide infusion 50-500 mcg/hr infusion should be initiated prior to any anesthetic in patients with known carcinoid tumors.
- Continue octreotide for at least 48 hours postoperatively in ICU setting.
- Invasive Monitoring with minimum of A-line.
- Smooth induction and maintenance of anesthesia with steady BP control.
- Anxiolysis and analgesia are important to prevent crisis not only in OR, but post op as well.
Conclusions

- Neuroaxial anesthesia may be used with octreotide and BP control
  - Epidural is preferred for post op analgesia and less sympathectomy
- Hemodynamic instability, flushing, and bronchospasm should be rapidly treated with IV octreotide and fluid boluses to effect
  - Octreotide up to 1 mg IV bolus (each dose)
Conclusions

- If hypotension continues despite octreotide and fluid, consider aprotinin, vasopressin, and small cautious doses of phenylephrine
  - Sympathomimeticss should be avoided
- Hypertension refractory to octreotide should be treated with deepening anesthesia, beta blockers, labetolol, alpha antagonists, or 5-HT\textsubscript{2} antagonist such as Ketanserin
References


References


References


References


National Comprehensive Cancer Network (NCCN) guidelines available online at www.nccn.org


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