ANAESTHESIA FOR ENDOCRINE DISEASE

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Introduction

This lecture will focus on the management of patients with diabetes mellitus, pheochromocytoma and adrenal insufficiency.
Diabetes Mellitus

• Adult normally secrete approximately 50 units of insulin each day from the B cells of islets of Langerhans in the pancreas.

• Insulin considered as the most important anabolic hormone, its lack is associated with catabolism and a negative nitrogen balance.
Clinical manifestations
DM is characterized by impairment of carbohydrate metabolism caused by deficiency of insulin activity, which lead to hyperglycemia and glycosuria.

The diagnosis is based on plasma glucose >140 mg/dl or blood glucose >126 mg/dl. Glucose tolerance test >200 mg/dl.
D M is classified to:

- Type I  absolute insulin deficiency secondary to immune–mediated or idiopathic.
- Type II adult onset secondary to resistance/relative deficiency.
- Type III specific types of D M secondary to genetic defect.
- Type IV Gestational.
Anaesthetic Management:

1- Preoperative Assessment

Diabetes causes diseases in many organ systems, the severity of which may be related to how long the disease has been present and how well it has been controlled.

Micro vascular, neuropathies and macro vascular complications of DM are of special concern for the anaesthetist.
Cardiovascular

Diabetics are prone to hypertension, ischeamic heart disease, cerebrovascular disease and myocardial infarction (MI) which may be silent.

A high suspicion for MI throughout the perioperative period, if unexplained hypotension, dysrhythmias, hypoxemia, or ECG changes develop.
Peripheral neuropathies are sensory symmetrical polyneuropathies in a glove and stocking distribution.

Preexisting neurological deficit is important to document.

Diabetics have increased susceptibility to perioperative nerve injury and soft tissue ischemia, thus, pressure points must be protected and patients positioned carefully.
Autonomic neuropathies are present in 40% of type I and 17% type II. and 50% if diabetic and hypertension.

It results in abnormal baroreceptor function, lack of heart beat variability (ventricular arrythmias).

Autonomic dysfunction worsened by:
old age, diabetes of >10 years, coronary artery disease, or beta adrenergic blockade.
Autonomic dysfunction: contributes to delayed gastric emptying (gastroparesis) leading to unexpected regurgitation and aspiration. Therefore evidence of autonomic neuropathy should be confirmed, a history of heartburn and acid reflux, if present, a rapid sequence induction with cricoid pressure even for elective procedures should be applied.
### Detecting autonomic neuropathy

<table>
<thead>
<tr>
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<th>Normal</th>
<th>Abnormal</th>
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<tr>
<td><strong>Sympathetic</strong></td>
<td>Measure BP in supine &amp; standing</td>
<td>&lt; 10mmhg</td>
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<tr>
<td><strong>Parasympathetic</strong></td>
<td>Pulse response to deep breath</td>
<td>Increase &gt;15</td>
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Airway
DM is associated with Stiff joint syndrome (nonenzymatic glycosylation of collagen and its deposition in joints) affecting the temporomandibular, atlanto-occipital and other cervical spine immobility and is a predictor of difficult endotracheal intubation.
(occur in 30% of type I diabetes).
Renal

DM is a leading cause of endstage renal failure, manifested first by proteinuria. Diabetics are at risk of acute RF postoperatively. Avoid nephrotoxic drugs. Provide adequate hydration.
Respiratory

Diabetics especially if obese and smokers are prone to chest infection.
Cataract and retinopathy are common. Prevent sudden rise in Bp and insure adequate depth of anaesthesia.
Infection

Diabetics are at increased risk of wound infection, because of compromised immune system thus strict aseptic techniques for any procedure undertaken.
Diabetic Ketoacidosis

Initially give IV fluids at a rate of 15-20 ml/kg/hr (1-2 L) during the 1st hour, then at a rate of 4-14ml/kg/hr (200-500 ml)
• About 1/3 of the estimated fluid deficit is corrected during the first 6-8 hours, and the remaining 2/3 over the next 24 hours.
• Following initial fluid replacement give regular insulin 0.1 U/Kg IV bolus followed by a maintenance rate of insulin infusion, where the calculated U/hr equals RBG divided by 150 or 100.
• The maximum rate of glucose decline is fairly constant 75-100mg/dl/h
• When serum glucose reaches 250mg/dl, add 5% dextrose at a rate of 50ml/hr.
• Rapid decline in serum K level occur, aggressive replacement therapy is required.
• Bicarbonate therapy is not recommended, but it may be used for pH less than 7.0. 1mEq/Kg over 1-2 hours.
• Rapid correction of acidosis with bicarbonate therapy may result in alterations in central nervous system function.
2- **Timing**
Diabetics should be placed first on list to minimize the fasting period.

3- **Hydration**
Check for dehydration and start IV fluids. Avoid Lactated Ringer's Solution because lactate may lead to metabolic acidosis, also infusing large volumes of normal saline can lead to hyperchloremic acidosis.
4- medication

The preoperative aim is to maintain blood sugar levels at 110-180mg/dl and in critically ill at 80-120mg/dl.
Methods of achieving glycemic control

• Patient undergoing minor surgery (patient is expected to eat or drink within 4 hours of surgery and random blood glucose (RBG) on admission <180 mg/dl)

• **Type I:**
  - Omit morning insulin.
  - Check RBG 1 hour preop and hourly perioperatively then 2 hourly postoperative until eating then 4 hourly.
  - Restart subcutaneous (SC) insulin regimen with first meal.

• **Type II:**
  - Omit morning oral hypoglycemic except metformin which should be omitted the night before because it can precipitate lactic acidosis.
  - Check RBG as above.
  - Restart oral hypoglycemics with the first meal.
• **Patient undergoing major surgery** (Include patients not falling into the above category, emergency surgery and poorly controlled)

• Preoperatively check electrolytes especially potassium (K) to allow sufficient time to correct any abnormality prior surgery.

• Omit morning oral hypoglycemics or short acting insulin. Long acting insulin should be stopped 24 hours prior surgery.

• Check RBG and K 1 hour preop then 2 hourly from start of infusion, hourly perioperatively and hourly postoperatively for 4 hours or until blood glucose is stable then 2 hourly.
Insulin infusion regimens

• 1 – Alberti Regimen (If no infusion pump available):
  • Start intravenous infusion of 10% dextrose 500 ml plus 10 mmol KCL at rate of 125 ml/hr add regular insulin according to blood glucose level: if
    
    < 90 mg/dl add 5 U;
    90 – 180 mg/dl add 10 U;
    180 – 270 mg/dl add 15 U;
    360 mg/dl add 20 U.
2 – Tight Control Regimen (If infusion pump available):

- Start intravenous infusion of 5% dextrose at a rate of 50 ml/hr to dextrose insulin infusion 50 U in 50 ml infusion pump. Set insulin infusion rate U/hr = RBG divided by 150 (or 100 if patient is on corticosteroids or obese).

- Postoperative: Type II: Stop infusion and restart oral hypoglycemics once eating or drinking.

**Type I:** Stop infusion once eating or drinking.

Calculate total daily dose (units) of insulin in the last 24 hours and divide into 3 daily doses and give SC regular insulin, adjust the dose until blood glucose is stable. Once stable to their normal insulin regimen.
Regional anaesthesia:

- less regurgitation and difficult intubation.
- early detection of hypoglycemic episodes and rapid return of oral intake.
- Attention to avoid hypotension by ensuring adequate hydration.
- Patient with autonomic neuropathy may not be able to keep BP within normal range.
- For medico-legal; preexisting nerve damage must be documented prior to block.
• General anaesthesia:
There are no contraindications to standard anaesthetic induction or inhalational agents.
If gastric stasis is suspected rapid sequence induction is performed.
Hypotension worsens after Intravenous induction in patient with autonomic dysfunction.
The Adrenal Gland

The adrenal gland is divided into two parts

- The adrenal cortex secretes androgens, mineralcorticoid (e.g., aldosterone), and glucocorticoids (e.g., cortisol).

- The adrenal Medulla secretes catecholamines (e.g., adrenaline, noradrenaline, and dopamine).
Adrenal medullary Sympathetic Excess

Phaeochromocytoma are catecholamine–producing tumors derived from chromaffin cells. 90% are found in the adrenal medulla and the remainder in other sites including paraganglionic cells of sympathetic nervous system.
Methylation of noradrenaline to adrenaline only occurs in the adrenal medulla and depends on cortisol delivered by intact portocapillary circulation which can be disrupted by the tumor, thus most tumor mainly produce noradrenaline.
• Phaeochromocytomas are referred to as the 10% tumor as 10% are familial, 10% bilateral, 10% extra-adrenal and 10% malignant.

• These tumors are biochemically very active, producing and secreting biogenic amines mainly noradrenaline and adrenaline.
• Noradrenaline secreting tumors tend to present with severe refractory hypertension, headaches and glucose intolerance.

• Adrenaline secreting tumors tend to cause palpitations, anxiety and panic attacks, sweating, hypoglycaemia, tachycardia, tachyarrhythmias.
25 – 50% of mortalities occur during induction of anaesthesia or during operative procedures for other causes.
Diagnosis:

- Urinary vanillylmandelic acid, elevated level of urinary normetanephrine and metanephrine
- Elevation of total plasma catecholamine concentration.
- CT scan without intravenous contrast media. MRI, or uptake of $[^{131}\text{I}]$ metaiodylbenzylguanidinne by gamma camera.
Anaesthetic Management
Preoperative Preparation

Catecholamine-induced vasoconstriction and hypovolemia should be controlled by α-blockade prior to surgery. Oral phenoxybenzamine (non-competitive, non-selective) 10mg twice daily increasing daily to 60–200mg until blood pressure is controlled. 10–14 days to stabilize blood pressure. Prazosin and doxazosin (competitive, alpha1-selective), but the non-competitive are preferable.
• Reflex tachycardia, treated by selective β1–blocker, oral atenolol 100 mg daily.

• Never commence β – blockers prior α blockade as this will precipitate a hypertensive crisis which is exacerbated by the negative inotropic effects of β – blockade, this includes labetalol which has greater β than α effect 7:1. Phenoxybenzamine dissipates over 36 hours, postoperatively patients remain sleepy due to persistent central α2 – blockade and may require large volumes of IV fluids until blockade has worn off, thus patients better stop the drug 24 – 48 hours prior surgery.
The alpha methyl p-tyrosine, competitively inhibits tyrosine hydroxylase, which is a rate – limiting ability of phaeochromocytoma to react to stimulation. It can be used for patients with poor left ventricular function. This drug may cause severe side effects including diarrhea, fatigue, and depression.
Roizen et al have recommended the following criteria for optimal preoperative control:

1 – Blood pressure consistently < 160 / 90 mmHg
2 – Orthostatic hypotension not < 80 / 45 mm Hg,
3 – Absence of ST – T changes in ECG for at least 1 week
4 – No more than one ventricular ectopic every 5 minute.
• **Premedication**: Benzodiazepine
• If bilateral adrenalectomy is possible coverage with glucocorticoids and mineralocorticoids should be planed
• **Surgical approach**: can be performed using open lateral retroperitoneal approach or, laparascopic.
Techniques and monitoring:

Large caliber IV cannulae, arterial catheter, central venous catheter, but pulmonary artery catheter is indicated only in the presence of significantly impaired myocardium.

Epidural catheter is placed for perioperative analgesia (T9-L1)

Urinary catheter.
Anaesthesia
general anaesthesia, Sevoflurane and isoflurane have been used extensively. Halothane can result in severe arrhythmias. Desflurane causes significant sympathetic stimulation and best avoided. Propofol, thiopental and etomidate accepted. Indirect sympathomimetics as ketamine, pancuronium and ephedrine, should be avoided. Drugs that cause histamine release as morphine and atracurium have infrequently been reported to trigger crisis. Isoflurane – remifentanil.
Tumor manipulation can produce increases in plasma catecholamine up to 200 times normal.

Phentolamine, a competitive α1, weak α2 adrenoceptor, 1 – 2 mg IV boluses (slow onset, long duration, with tachyphylaxis)
• Sodium nitroprusside 0.5-10 Mig/kg/min (rapid onset, short duration, cyanide toxicity in high doses > 0.5mg/kg/hour) or nitroglycerin.
Magnesium sulphate (antiarrhythmic properties due to calcium channel blockade, regulation of intracellular potassium and ATP activation).

It causes vasodilation by blocking the adrenoceptor response to noradrenaline and angiotensin II.

It also inhibits catecholamine release from adrenal medulla and peripheral adrenergic nerve terminals.

The effective plasma concentration is 2 – 4 mmol/litre, IV loading 40 – 60 mg/Kg followed by infusion 2 g/hour.

Side effects potentiation of neuromuscular blockade, inhibition of platelet activity, narrow therapeutic window.
• Adrenaline induced tachyarrhythmias are controlled with esmolol or labetalol.

• Nicardipine
Hypotension  sudden fall in blood pressure after clamping vessels.

ensure euvoilaemia.

vasopressors and glucose should be available.
**Postoperative management:**

Patients should be managed in ICU. Postoperative complications include hypertension, hypotension and hypoglycemia.

Catecholamine secretion from the contralateral adrenal gland will be down regulated for some time.

Hypoglycemia may occur due to abrupt cessation of $\alpha_2$-mediated pancreatic $\beta$ cell inhibition. Persistent $\beta$-blockade may mask hypoglycemic symptoms. Glucose monitoring is recommended.
Adrenocortical Malfunction

The adrenal cortex is responsible for the secretion of three classes of steroids: glucocorticoids, mineralocorticoids and androgens.
Hyperaldosteronism

Primary hyperaldosteronism (Conn's dyndrome), there is excess secretion of aldosterone from an adrenal adenoma (60%), bilateral hyperplasia (30%) or, rarely, carcinoma.

Secondary hyperaldosteronism high plasma concentration of rennin and aldosterone. (congestive cardiac failure and liver cirrhosis and nephrotic syndrome.)
Hypertension
Increase extracellular fluid volume.
malignant hypertension (centrally or direct effect on vascular endothelium via aldosterone-induced vasoconstriction.
Hypokalaemia, exacerbated by treatment of hypertension with diuretics.

Metabolic alkalosis due to hydrogen ion loss
Treatment

Spironolactone 400 mg day,
Manipulation of the adrenal gland may cause secretion of catecholamines
Replacement corticosteroid and mineralocorticoid therapy is required for all patients undergoing bilateral adrenalectomy and occasionally in those undergoing unilateral.
Cushing's syndrome

- Cushing's syndrome is caused by excessive levels of glucocorticoids.
- Intrinsic hyperfunction of adrenal cortex (adrenocortical adenoma), ACTH production by a non-pituatery tumor, or hypersecretion by a pituatery adenoma (cushing disease).
- Exogenous administration.
poorly controlled hypertension
obesity; sleep apnoea, suspect difficult intubation.
Impaired glucose tolerance and D.M.
Hypokalaemia due to sodium retention and potassium loss.

• Gastrooesophageal reflux
• Extra care should be taken with positioning because there is susceptibility to pressure sores and fractures because of fragile skin and osteoporosis
• If the Cushing's syndrome is iatrogenic, an additional dose of steroids may be required preoperatively.
Addison's disease

Primary adrenal insufficiency is characterized by reduced or absent secretion of glucocorticoids, associated with deficient mineralocorticoid activity.

Destruction of the adrenal cortex by autoantibodies is the cause in 80% of cases; other causes include tuberculosis, metastatic carcinoma, bilateral adrenalectomy and haemorrhage (e.g. meningococcal sepsis).
Patient clinical manifestation are due to aldosterone deficiency (hyponatremia, hypovolemia, hyperkalemia and metabolic acidosis) cortisol deficiency (weakness, fatigue, hypoglycemia, hypotension, and weight loss)

Increased pigmentation of palmar areases and buccal mucosa due to high plasma levels of ACTH that mimic melanocyte – stimulating hormone.
Etomidate suppresses adrenal function by inhibiting enzymes that are essential for the production of corticosteroid hormones. Etomidate therapy can lead to glucocorticoid deficiency.
Secondary hypoadrenalism results from prolonged corticosteroid therapy and hypopituitarism.

Serum potassium and glucose check regularly.

For major surgery, blood glucose should be checked every 4 h and electrolytes should be measured daily.
The adequate steroid coverage is controversial, while adults normally secrete 20 mg of cortisol daily; this may increase to be 300 mg under maximal stress.
All patients who have received potentially suppressive doses of steroids equivalent to 5 mg of prednisolone by any route (topical, inhalation, or oral) for a period of more than 2 weeks any time in the previous 12 months should be considered unable to respond appropriately to surgical stress.
• For minor surgery I.V. hydrocortisone 25 mg should be given at induction. is sufficient.
• For intermediate surgery, such as hysterectomy, 100 mg infusion or four doses of i.v. hydrocortisone 25 mg should be given over the first 24 h after surgery.
• For major surgery, 200 mg of i.v. hydrocortisone should be administered each 24 h until the patient can be re- started on maintenance therapy.
Acute Addisonian crisis

Any stressor can precipitate an adrenal crisis in patients with adrenal suppression, surgery, trauma, and sepsis, long-term corticosteroid therapy discontinuation.

Treatment is of 200mg is followed by 100 mg every 6 h until oral supplements can be taken. Fluid resuscitation is required, and dextrose may be necessary to treat hypoglycaemia.