Post-operative complications following bariatric surgery

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Extreme obesity affects every organ system and causes significant chronic medical co-morbidities. Most of the associated conditions are reversible following sustained weight loss. In 1991 the United States' National Institutes of Health Consensus Development Conference Panel recommended weight reduction (bariatric) surgery for patients with extreme obesity that cannot lose weight by diet and exercise alone. Since then the number of bariatric surgical procedures performed in the United States has risen and currently exceeds 200,000 annually. Most of the information we have on the perioperative management of the morbidly obese patient is based on experience with bariatric operations. This review considers the postoperative management of the morbidly obese patient. Most obese patients recover from anesthesia and surgery in the PACU. Admission to an intensive care unit is usually due to either severe underlying medical conditions, and/or complex surgical procedures.

PULMONARY CONCERNS

Obese surgical patients are at greater risk for hypoxemia in the postoperative period than normal weight patients undergoing similar operations. Their fatty chest and abdominal walls plus an increased pulmonary blood volume reduce pulmonary compliance. Functional residual capacity (FRC) is significantly reduced due to a decrease in expiratory reserve volume (ERV). These changes increase in direct proportion with increasing BMI.

General anesthesia results in a significant incidence of post-operative atelectasis in morbidly obese patients. If the patient is in the supine position, FRC is even further reduced. The semi-recumbent and/or reverse Trendelenburg positions maximize oxygenation by allowing the diaphragm to fall and FRC to increase. If hemodynamically stable, obese patients should have their airway extubated with their upper body elevated 30° - 45°. It is imperative that any residual effects of neuromuscular drugs be completely reversed.

They should be transferred from the operating room and recover in the PACU in a head-up position. Even in this position patients may become hypoxemic if supplemental O_2 is withheld. Restoration of normal pulmonary function after open abdominal surgery may take several days.

Despite these changes, postoperative mechanical ventilation is rarely needed. Factors that may necessitate ventilatory support include extremes of age, coexisting cardiac disease, carbon dioxide retention, fever or infection, and an uncooperative or extremely anxious patient. Occasionally, re-intubation of the trachea is necessary. The most important strategy for successful direct laryngoscopy is patient position. The head, upper body and shoulders should be elevated so that the ear is level with the sternum (head elevated laryngoscopy position, H.E.LP.). This position greatly improves the endoscopist's view during direct laryngoscopy in morbidly obese patients. A laryngeal mask airway (LMA) can serve as a 'bridge' until an endotracheal tube is placed when difficulty is encountered.

All obese patients should be continuously monitored by pulse oximetry in the postoperative period, even following a completely uneventful operation. Obesity is an important risk factor for Obstructive Sleep Apnea (OSA), a condition which is frequently undiagnosed in obese patients scheduled for surgery. A definitive diagnosis of OSA can only be confirmed by polysomnography in a sleep laboratory. OSA is characterized by frequent episodes of apnea (>10 sec cessation of airflow despite continuous respiratory effort against a closed airway) and hypopnea (50% reduction in airflow or reduction associated with a decrease of S_pO_2 > 4%).

OSA patients using nasal CPAP or bi-level positive airway pressure (BiPAP) devices at home should be instructed to bring their equipment for the hospital to use in the recovery room. These devices allow alveolar recruitment during inspiration and prevent alveolar collapse during expiration. In theory CPAP could distend the gastric pouch, but its use following bariatric surgery has not been associated with

anastomotic leaks. When a home CPAP device is not readily available, non-invasive ventilation with Boussignac CPAP will improve oxygenation compared to nasal oxygen alone, but it has no benefit in improving carbon dioxide elimination.

A small number of patients have the "obesity hypoventilation syndrome" (OHS), which is characterized by somnolence, cardiac enlargement, polycythemia, hypoxemia and hypercapnia. OHS patients tend to be older, super-obese (BMI > 50 kg/m^2), and have more restricted pulmonary function than other patients with OSA. Hypoventilation is central and independent of intrinsic lung disease. OHS is probably due to a progressive desensitization of the respiratory center to hypercapnia from nocturnal sleep disturbances. Its most severe form is called "Pickwickian Syndrome". OHS patients rely on a hypoxic ventilatory drive and may hypoventilate or even become apneic following emergence from general anesthesia after being given 100% O$_2$ to breathe.

CARDIOVASCULAR CONCERNS

Cardiac output rises proportionally with increasing weight. Stroke volume also increases since a greater total blood volume is needed to perfuse added body fat. Increased cardiac output combined with normal peripheral vascular resistance leads to systemic hypertension, a common condition in morbidly obese patients. With increasing age the increased left ventricular wall stress caused by increased stroke volume and the resultant ventricular dilation leads to cardiac hypertrophy. Some degree of left ventricular dysfunction may be present in young, asymptomatic patients. Even normotensive patients have increased pre-load and after-load, increased mean pulmonary artery pressure (PAP), and elevated right and left ventricular stroke work. Since many patients are not physically active, they may appear to be asymptomatic even in the presence of significant cardiovascular disease. Signs of pulmonary hypertension (exertional dyspnea, fatigue, syncope) should be sought preoperatively and trans-esophageal echocardiography (TEE) obtained in symptomatic patients. Right heart failure is common in older patients. A significant decrease in left ventricular function may occur in the immediate postoperative period. All patients must be closely monitored and inotropic agents given when indicated, especially if the patient is hypovolemic or is receiving local anesthetics via an epidural catheter.

Cardiac dysrhythmias are not unusual both preoperatively and postoperatively. The causes are many including chronic hypoxia (especially in patients with OSA), hypercapnia, increased circulating levels of catecholamines, electrolyte disturbances caused by diuretic therapy, fatty infiltration of the conduction system and ischemic heart disease.

The popular weight loss medication, sibutramine, works in the brain by inhibiting the reuptake of norepinephrine, serotonin and dopamine and been also implicated as a cause of dysrhythmias and hypertension.

INTRAVASCULAR FLUID

Intraoperative fluid requirements, even for laparoscopic procedures, are usually greater than would be anticipated in a normal weight patient. Although total body water is increased, morbidly obese patients have a relative decreased percentage of body water compared to normal patients.

Obese patients receiving 40 mL/kg of intravenous crystalloid had a faster recovery and fewer complications after cholecystectomy than patients receiving 15 mL/kg. These results can only be extrapolated to morbidly obese patients since to date no investigation of fluid requirements for laparoscopic bariatric surgery has been performed. It is essential that adequate amounts of intravenous fluid be given to reduce postoperative renal failure, and to avoid other rare but serious complications such as rhabdomyolysis.

TEMPERATURE MAINTENANCE

Even though adipose tissue is a thermal insulator, patients become poikilothermic during general anesthesia. Heat loss may be exaggerated by the CO$_2$ pneumoperitoneum and when cold irrigating fluids are used during laparoscopy. Hypothermia should be avoided because shivering may increase oxygen demand. Moderate hypothermia may prolong anesthetic effects and delay awakening.

Warming blankets and other devices should be employed both intra- and post-operatively, and warmed intravenous and irrigating fluid are occasionally needed if there is a significant drop in temperature.

VENOUS THROMBOEMBOLISM

Thrombo-embolism is a major cause of postoperative mortality. Complications can occur in the immediate postoperative period. Deep venous thrombosis (DVT) leading to pulmonary emboli may be as high as 1-2% of patients having bariatric surgery, and since many are undiagnosed, the true incidence may be higher. The estimated mortality in patients with DVTs is 20-30%.

The risk of thrombosis is increased because of greater blood volume and relative polycythemia in obese patients. Prolonged immobilization can lead to phlebothrombosis so early ambulation must be encouraged. Other risk factors include high fatty acid levels, hyper-cholesterolemia and diabetes and accelerated fibrin formation, fibrinogen-platelet interaction, and platelet function compared with controls.

The most common presentation for a pulmonary embolism is tachycardia and hypoxemia. Hyperventilation can also lead to hypocarbia. Patients experience anxiety, dyspnea and often a "feeling of doom". These signs and symptoms are similar to the presentation of a bowel leak, and leak must be considered in the differential diagnosis after bariatric operations.

Anticoagulation or other prophylaxis measures should always be considered in the postoperative period, even for patients with epidural catheters. A vena cava umbrella is occasionally placed preoperatively in older and high risk patients, and sequential compression boots are used during surgery.

Another popular diet medication, orlistat, blocks digestion and absorption of dietary fat by binding lipases in the gastrointestinal tract and can cause deficiencies in fat-soluble vitamins (A, D, E, K). A reduction in vitamin K levels can increase the anticoagulation effects of coumadin.

NAUSEA AND VOMITING

Obesity, per se, is not a risk factor for postoperative nausea and vomiting (PONV). However, many patients are at high risk (female, receiving opioids, emetogenic surgery) for PONV. Multi-modal intraoperative prophylaxis with several anti-emetic agents will reduce, but not eliminate PONV. We give dexamethasone (4-8 mg) as a routine part of the therapeutic regimen.

One retrospective study of, morbidly obese patients reported that those who received larger volumes of intravenous fluids intraoperatively complained of less nausea postoperatively following laparoscopic bariatric surgery.

ANALGESIA

Postoperative pain can lead to hypoventilation with hypoxemia, hypercapnea and atelectasis all of which can cause significant cardiac complications. Any medication that depresses ventilation will have an exaggerated effect in the morbidly obese patient, especially one with OSA who may be more sensitive to these effects. Epidural analgesia (with local anesthesia, with opioids, or combined) has long been favored after open abdominal and thoracic operations for obese patients.

Intravenous opioid patient-controlled analgesia (PCA) with drug dose based on ideal body weight is satisfactory. However, large amounts of opioids, especially longer acting opioids (morphine, demerol, hydromorphone), which can depress ventilation, and should be avoided.

Non-opioid analgesic adjuncts should be instituted early. Dexmedetomidine, which has no respiratory depressant effects, may become a useful alternative or supplement to opioids in obese patients. Dexmetetomidine, administered during surgery may reduce opioid requirements in the PACU. Non-steroidal anti-inflammatory drugs are helpful, but should be discontinued within a day or two to avoid the potential complication of gastric ulceration.

For laparoscopic bariatric procedures, local anesthetic is infiltrated into the trocar sites during the procedure, so incisional pain in the immediate recovery period is less than after laparotomy. The insufflated CO_2 used during laparoscopy causes pain, from irritation or stretching of the peritoneum, and that discomfort is not alleviated by analgesics. Despite attempts at warming and humidifying the CO_2, postoperative discomfort in the PACU is common following laparoscopic procedures. When the patient complains of discomfort, the PACU nurse may be tempted to administer opioids. Unfortunately, additional opioids will not relieve this and may depress ventilation.

RHABDOMYOLYSIS

A potentially fatal postoperative complication in morbidly obese surgical patients is rhabdomyolysis (RML). RML results from injury to skeletal muscle. Damaged skeletal muscle releases myoglobin into the systemic circulation where it is filtered out of the bloodstream by the kidneys. When very high concentrations of myoglobin are released, the kidneys can be overwhelmed and acute renal failure (ARF) can develop. Disruption of the skeletal muscle membrane integrity also allows an influx of electrolytes and extra-cellular fluid into the damaged muscle. Large volumes of intravascular fluid (as much as 12 liters) can leave the circulation and become sequestered in the edematous muscle. This fluid shift can result in intravascular hypovolemia with hemodynamic instability, which further compromises renal function.

Prevention of RML begins with careful intra-operative padding of all pressure points and close attention to patient positioning. Minimizing operative time, adequate perioperative hydration, and close postoperative monitoring are obviously essential.

Postoperative RML is believed to be due to pressure injury to muscle after being placed in a non-physiologic position on the operating table. The majority of cases have followed surgery in the supine or lithotomy positions. Long duration surgery is the major risk factor, but other factors including super-obesity (BMI > 50 kg/m^2), male patients, co-existing hypertension, diabetes and/or peripheral vascular disease also contribute to RML.

Local signs and symptoms of RML include muscle pain, tenderness, swelling, bruising and weakness. Complaints of numbness and muscular pain are almost always present. Aggressive pain management, especially epidural analgesia, may mask symptoms and delay diagnosis.

The primary diagnostic indicator of RML is elevated serum creatine phosphokinase (CPK) levels. A CPK value > 5 times normal (1,000 IU/L) is diagnostic of RML. Elevated CPK levels occur in as many as 25% of morbidly obese surgical patients. Any morbidly obese patient who complains of buttock, hip, or shoulder pain in the postoperative period should have their CPK level measured. Early diagnosis of RML leads to earlier treatment and a better prognosis. Treatment should be instituted once CPK levels increase > 5,000 IU/L. Therapy focuses on the prevention of ARF and the management of the life-threatening metabolic complications.

Myoglobinuria (brown or "tea colored" urine), particularly in the absence of haemoglobinemia and hematuria, is also diagnostic of RML. Other biochemical alterations include hyperkalemia, hypo-calcemia, hyperphosphatemia, hyperuricemia, and raised levels of other muscle enzymes. Metabolic acidosis may result from release of phosphate, sulphate, uric acid, and lactic acid from the muscle cell.

Aggressive hydration with large volumes of intravenous fluids will flush myoglobin from the kidneys. Diuretic such as mannitol or furosemide should also be instituted once the diagnosis of RML is made. Mannitol mobilizes interstitial fluid and increases renal tubular flow but can also depletes circulating volume. Urine should be alkalinized by infusion of sodium bicarbonate with an objective of achieving a urinary pH > 7 to increase the solubility of myoglobin. Acetazolamide can be used if arterial pH is > 7.45. The target for aggressive hydration and diuresis is a urine output of at least 1.5 mL/kg/h. Persistent oliguria or anuria may require dialysis.

Any patient suspected of developing RML should be admitted to a critical care unit for intense monitoring and treatment. Although RML usually presents in the recovery room immediately following surgery, late presentation (or perhaps more accurately *late diagnosis*) is not uncommon, especially if pain management masks muscle or nerve complaints.

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Postoperative rhabdomyolysis

Risk factors
 Preoperative
 Male
 Age > 40 years
 BMI > 55 kg/m^2
 History of hypertension, diabetes mellitus, or peripheral vascular disease
 History of statin use
 Elevated preoperative serum CPK level
 Intraoperative
 Operation duration and anesthesia time > 5 hours
 Inadequate padding of pressure areas
 Inadequate hydration
 Urine output < 1.5 mL/kg/h
 Bleeding and/or hypotension
 Use of propofol and/or succinylcholine

Postoperative
 Complaints of muscle pain and weakness
 Delayed ambulation
 Urine output < 1.5 mL/kg/h
 Serum CPK > 1,000 IU/L
 Urine myoglobin > 250 ng/L
Treatment
 Immediate
 Intravenous hydration
 Diureses (mannitol, furosemide)
 Alkalinization (bicarbonate, acetazolamide)
 Correct electrolyte abnormalities
 Lower uric acid (allopurinol)
 Late
 Dialysis for renal failure
 Treat disseminated intravascular coagulopathy
 Decompress "compartment syndrome"