MORBID OBESITY - ANAESTHETIC MANAGEMENT

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Obesity means excessive body fat. The term obese, derived from Latin word means fattened by eating. The amount of fat tissue may increased to such an extent that mental and physical health is affected and life expectancy is reduced.

Body mass index (BMI) or Quetelet's index is calculated from subjects' height and weight. It is used to indicate obesity. Obesity is indicated by BMI $\geq$ 30.0 (M), $\geq$ 28.6 (F). Morbid obesity is when BMI is more than 40 kg/m2. Many clinical parameters depict women, as pear shaped, accumulating fat at bottom and is regarded as safe, fat is stored between skin and body wall. In men the classic beer belly look compared to a rounded apple, here the fat is stored to internal organs.

Prevalence and Epidemiology

There is a slow but steady increase in obesity. In UK, in 1980 - 6% males and 8% females were obese. In 1987 - 8% males and 12% females were regarded obese. Obesity in children is also on the rise. Prevalence varies with socioeconomic status. In developed countries poverty is associated with greater prevalence whereas in developing countries it is the affluent that carry higher prevalence. Obesity is complex, wherein the net energy intake exceeds the net energy expenditure over a prolonged period of time.

Pathophysiology

Brain controls appetite by means of signals triggered by dietary breakdown products and by autonomic signals produced by disturbance of the stomach and intestines. The multiple signals generated are processed by complex interactions between neuronal networks and neuro transmitters. Most important of them are cholecystokinin 8 (CCK 8), which act at gut and brain. It induces satiation, is released at beginning of a meal and promotes pancreatic secretion of insulin, which lowers blood sugar and increases appetite. Insulin crosses blood brain barrier and triggers hypothalamus, which in turn juggles many other signals. Therefore the complexity of weight maintenance is evident. 25-30% of human variations in BMI are genetic and rest is due to environmental factors. It is a complex picture how normal weight is maintained. The pathways regulating weight form series of redundant regulatory loops. If one loop is weakened or attenuated, the other can take over. It is this redundancy that regulates calorie storage, but at the same time the redundancy makes it hard to get a handle on how to prevent and treat obesity. It is an imbalance between food intake and energy expenditure. The balancing act involves neural and endocrinal signalling. These operate both by central and peripheral mechanism. Problems can occur any where in this complex system.

Genetic Predisposition

Tends to be familial. Children of two obese parents have a 70% chance to become obese. The genetic issue

plays 30% and linked to at least 6 genes. 70% is dependent on environmental factors. Genetic susceptibility may predispose and environmental issues may add on.

Ethnic Influences

In USA, the African Americans and Mexicans have higher rate of obesity than the Whites. Asian migrates have more central distribution of fat, which is associated with increased risk of diabetes and Coronary Artery Disease.

Medical Diseases

Cushing's disease, hypothyroidism, medications like steroids, antidepressants and antihistamines may also have a role to play in obesity.

Obesity Associated Medical Conditions

Diabetes, hypertension, hyperlipidemia, heart diseases, infertility, hepatobilary diseases, cerebrovascular disease, degenerative joint disease, chronic back pain, gallstones, increased rates of colon and breast cancer and asthma are also linked to rising levels of obesity. A BMI >29 kg/m2 increases the prevalence of pulmonary embolism. Risk of coronary artery diseases is doubled if BMI is > 25kg/m2. A BMI 35kg/m2 leads to a 40-fold increase in developing diabetes, respiratory diseases, sleep apnoea and osteoarthritis. Risk of death increases with body weight. Mortality rises exponentially with increasing body weight.

CHANGES IN VARIOUS SYSTEMS

Obesity and Respiratory System

5% of morbidly obese have Obstructive Sleep Apnoea (OSA) characterized by frequent episodes of apnoea or hypercapnia, snoring and daytime sleepiness. Recurrent apnoea leads to hypoxia, hypercapnia, pulmonary and systemic vascular hypertension, which in turn leads to right ventricle failure. Loss of pharyngeal muscle tone in obese person during sleep and significant narrowing of airway increases airflow turbulence resulting in OSA.

Risk Factors For OSA

Male gender, middle age, night sedation, evening alcohol can compound the problem. Other features which can help identify OSA are BMI > 30 kg/m2, hypertension, observed episodes of apnoea during sleep, hypoxemia, hypercapnia, changes in ECG and ECHO. Definite diagnosis is made by polysomnography in sleep laboratory. Such patients pose a great challenge to surgery, anaesthesia, obstetrics, trauma and in the ICU.

Acid Base Disturbances of OSA

Respiratory acidosis is limited to sleep, in the beginning. Longer the problem, alterations occur in breathing patterns, desensitization of respiratory center to hypercapnia leads to typeII respiratory failure. This leads to increased dependence on hypoxic drive for ventilation. Pickwickian syndrome is characterized by obesity, hypersomnolence, hypoxia, hypercapnia, right ventricular failure and polycythemia.

Airway

Perfect airway particularly upper is essential before any anaesthetic management. Difficulties are encountered for mask ventilation. Tracheal intubations may be difficult. The percentage of difficult intubation is quoted to be 13%. Excessive fat at upper airway, short neck, high anteriorly placed larynx, restricted cervical spine movements are a few problems.

Obesity and Lung Volume

Increased mass of abdominal and thoracic contents alters the lung volumes. Decrease in functional residual capacity (FRC) is seen exponentially with increasing BMI. Expiratory reserve volume and total lung capacity are decreased. FRC may be reduced in upright position to the extent that it falls within the range of closing capacity with subsequent small airway closure, ventilation perfusion mismatch, right to left shunting and arterial hypoxemia. The reduction of FRC impairs the capacity of obese patients to tolerate apnoea. They desaturate rapidly after induction of anaesthesia despite preoxygenation due to smaller O2 reservoir and increase in oxygen consumption. Residual volume remains normal or slightly increased due to increased gas trapping and coexisting obstructive airway disease.

O2 Consumption and CO2 Production

Both are increased in obese patients as a result of metabolic activity of excess fat and increased workload on supportive tissues. In exercise O2 consumption rises more sharply than in the non obese.

Gas Exchange

Only modest defect in gas exchange is noted in the obese patients with a reduction in PaO2, increase in AaO2 gradient with increase in shunt fraction. This is increased markedly with induction of anaesthesia, PEEP improves the PaO2, but at the expense of cardiac output and O2 delivery.

Compliance and Resistance

Increased BMI exponentially decreases compliance. As fat content increases, compliance decreases. This is due to increase in pulmonary blood volume, increased total respiratory resistance and shallow rapid breathing, which can limit maximum ventilatory capacity. These are more markedly observed in supine position.

Work of Breathing

30% increase is observed in work of breathing. If hypoventilation occurs in daytime the work of breathing may approach four times than predicted.

Cardiovascular System

Higher incidence of cardiovascular morbidity is associated with obesity. Mild to moderate hypertension is found in 60-70% and severe in 5-10%, with 3-4mmHg increase in systolic and 2mmHg increase in diastolic pressure for every 10kg increase of weight is noted. It is the commonest problem followed by ischaemic heart disease. An

expansion of extracellular volume resulting in hypervolaemia and increase in cardiac output is characteristic of obesity-induced hypertension. Exact mechanism is not known but interplay of genetic, hormonal, renal and haemodynamic factors are implicated. Hyperinsulinaemia activating sympathetic nervous system, causing sodium retention, increase in pressor norepinephrine and angiotensin II activity. Concentric hypertrophy of left ventricle leads to cardiac failure. Obesity is independent risk factors for Ischaemic Heart Disease (IHD) and is more common in individuals with central obesity. Blood volume is increased, most extra volume being distributed to fat organ. Splanchnic blood flow is increased by 20%, renal and cerebral blood flows are normal. Cardiac arrhythmias can be precipitated in obese by any number of factors, viz. hypoxia, hypercapnia, electrolyte imbalance, diuretic therapy, fatty infiltration of conducting tissue.

Cardiac Function

Obese are at risk of specific form of obesity induced cardiac dysfunction. Left ventricular systolic and diastolic functions are affected. Obesity induced cardiomyopathy is well-documented. Blood volume is increased and cardiac output increase by 20-30ml/kg of excess body fat. They tolerate exercise poorly. Any increase in cardiac output is by increase in heart rate.

Obesity and Diabetes

Obesity is an independent risk factor for Type II Diabetes. More than 10% of incidences of abnormal glucose tolerance are seen in patients undergoing bariatric surgery.

Thromboembolic Disease

Deep vein thrombosis appears twice as common in obese patients. It is the commonest complication of bariatric surgery with an incidence of 2.4% - 4.5% and is due to prolonged immobilization, leading to venous stasis, polycythemia, and increased abdominal pressure with increased pressure on deep vein. Decreased fibrinolytic activity with increased fibrinogen concentration could also be responsible.

Obesity and GI Disorders

Obesity is associated with increase in intra abdominal pressure, high volume and low pH of gastric contents, delayed gastric emptying, and increased incidence of gastro esophageal reflux. There is a high risk for aspiration of gastric content followed by pneumonia. Gastric volume is 75% higher than the normal individuals.

Drugs, Pharmcodynamics and Kinetics

Obesity leads to alteration in distribution, binding and elimination of many drugs. For drugs with narrow therapeutic indices like aminoglycosides and digoxin, toxic reactions can occur if patients are dosed according to actual body weight. Drug dose should be reduced keeping the ideal body weight in view; absorption of drugs given orally remains unchanged in obese patient.

Volume of Distribution (VD)

Apparent volume distribution of a drug in obese patient, depends on number of factors, which include the size of

fat organ, increase in lean body mass, increase in blood volume, and cardiac output, reduced total body water, alterations in plasma protein binding and lipophilicity of drug. High lipophilic drugs have increased volume distribution (Thiopentone). Increase in the volume distribution will reduce the elimination half-life unless the clearance is increased. Thiopentone, Benzodiazepines, and potent inhalation agents, may persist for longer time after discontinuation. Regarding protein binding, alteration may occur due to high levels of cholesterol, which inhibits protein binding, therefore more free drug is available. In contrast increased concentrations of a acid glycoprotein may increase the degree of protein binding of other drugs (e.g. local anaesthetics) so reducing the free plasma fraction.

Elimination

Clearance is mostly reduced in obese patients. Cardiac failure and decrease in liver blood flow may slow elimination of midazolam and lignocaine. Renal clearance increased is obesity because of the increased renal blood flow and GFR. If renal impairment is present elimination takes longer time. Hepatic metabolism is altered in obese patients for volatile agents. Reductive metabolism of halothane is more in obese patients. This may be important factor in liver injury. Nephrotoxicity can occur due to high fluoride concentrations with halothane and enflurane. Sevoflurane has 5% biotransformation but does not show adverse effects. Isoflurane does not increase fluoride concentration, and remains the agent of choice in obese.

ANAESTHETIC IMPLICATIONS

Preoperative: It is important to have a thorough clinical examination with excellent and relevant history looking for hypertension with appropriate sized cuff, signs of cardiac failure, viz. increase in jugular pulse, added heart sounds, pulmonary crackles, hepato jugular reflex and peripheral edema. These signs may be difficult to elicit in the morbidly obese. A thorough assessment of respiratory system for OSA is very essential.

Investigations

Besides routine investigations, ECG is mandatory, tachy arrythmias are common. Echo may be difficult but will provide valuable information regarding eccentric left ventricular hypertrophy. TEE may provide more insight. Cardiological evaluation is beneficial for further investigation. Optimization of blood pressure, treatment of cardiac failure, or if necessary, coronary angioplasty may be suggested. X-ray chest, lung functions, baseline arterial blood gases may be useful in morbidly obese. A thorough history regarding respiratory function, sleep apnoea are a must. Signs of right ventricular failure must be looked for.

Airway assessment

To plan the type of airway management is mandatory. A through examination can prevent the catastrophes, as it will enable to select the best technique for the patient. Preoperative evaluation of airway must include

1. Assessment of head and neck, flexion and extension and lateral rotation.
2. Assessment of jaw mobility and mouth opening.
3. Inspection of oropharynx.

4. Check the patency of nostrils

5. Inspection of previous anesthetic charts. If potential airway obstruction is suspected direct or indirect laryngoscopy, CT scan of soft tissues would be helpful.

Assessment of veins

Assessment of veins for placing infusion must be done in the pre operative visit. Examination of feet and back for any ulcers or sore is mandatory. Examination of calf muscles for any redness or tenderness gives a fairly good idea regarding deep vein thrombosis.

Preoperative medication

Avoid narcotics and sedatives. Avoid intramuscular and subcutaneous injections. If fibre optic intubation is planned, include an antisailagogue like glycopyrrolate. All morbidly obese must have acid prophylaxis. A combination of H2 blocker, e.g. ranitidine 150mg and prokinetic e.g. metaclopromide 10mg given orally 12 hrs and 2hrs before surgery will reduce risk of aspiration. Some anaesthetists prefer to give 30ml 0.3 M citrate before induction. Continue the normal medications on day of surgery. Stop ACE inhibitors the day before surgery. Dextrose-Insulin regimen should be followed in all diabetics, unless it is a very short surgical procedure. Insulin requirements may increase in postoperative period. Prophylactic antibiotics are given as per the hospital protocols, should have discussion with surgeon and microbiologist.

Position and transfer

Extra care is required in transferring the obese patient and special tables having adequate padding of pressure areas are used. Appropriate manpower to shift is mandatory. Compression of inferior vena cava is avoided by lateral tilt or a wedge. Transfer of obese patients is done in their own bed.

Intravenous lines

Peripheral lines may be difficult. Establish central line in the beginning to avoid calamities. Even these are difficult in morbidly obese. Doppler or ultrasound guided placements could reduce complications.

Monitoring

Intraarterial blood pressure measurement is advocated for all but most minor procedures, ECG, Pulse oximetry, Capnography, neuromuscular monitors are essential. Central catheters are essential and PA Catheter where indicated.

Regional anaesthesia

Where ever it is possible and feasible, regional anaesthesia should be administered. The advantages are that one can reduce the use of opioids, inhalational agents, reduce postoperative complications, and prevent loss of airway and prevention of aspiration. Excellent postoperative analgesia can be given by placing a epidural catheters in sitting position, using ultrasound for identification of the space is helpful. Local anaesthetic

requirements are reduced in morbidly obese. Higher blocks are common. Blocks extending above T5 can cause cardiorespiratory collapse. All resuscitation equipment should be handy.

Systemic opioids

These are hazardous in obese patients, and intra muscular route is not recommended. If intravenous route is preferred, patient controlled analgesia should be the best option. Oral analgesics like paracetamol and cox2 inhibitors may be appropriate. Postoperative analgesia with local anaesthetics and opioids via the epidural catheter will provide ideal pain relief with minimal complications.

Obstetrics and morbidly obese

All attending complications are compounded. Regional anaesthesia is a better choice; avoid general anaesthesia as far as possible. Sitting an epidural catheter during labor is a better option. Local anesthetic requirement may be reduced by up to 25% in obese pregnant state.

The ideal anesthetic agents, which can be used in an obese individual, can be summarized as propofol for induction, atracurium for relaxation, remifentanyl for opioids and iso or sevoflurane as inhalation agents. All ideal drugs may not be available or may not be cost effective. Therefore the anaesthesiologists should, with their understanding of pathophysiology, use the drugs and techniques to the best of their ability to cause least morbidity.

Obesity and Trauma

The out come in obese patients with trauma is poor. The obese patients have more blunt trauma, chest trauma is more when compared to head injury. Investigations are more difficult to handle and interpret. They may require earlier respiratory support and higher oxygen concentration.

Obese patient in ICU

It is generally believed that the outcome of obese patients is poor. Postoperative pulmonary events are more in obese patients. Morbidly obese may present in emergency room and intensive care staff with formidable challenges. Better understanding of the pathophysiology and complications may improve the outcome. Anaesthesiologist has a major role to play as the perioperative physician at every level.

BARIATRIC SURGERY *(weight reductive surgery)*

The indications for bariatric are as follows

BMI >40kg/m2

BMI >35kg/m2 with co morbidities

Should show that dietary attempts have been ineffective

It addresses both perioperative care and long-term management. The patients must have a clear understanding of risk, benefit, and complications, and may require life long management strategies. Bariatric surgery comprises two types namely Restrictive and Malabsorptive. The common operations include Rouxen Y grade bypass, vertical banded gastroplasty, pancreaticobiliary diversion and its variations, various gastric banding procedures, gastric bypass procedures and laproscopic procedures of the above-mentioned operations. The preoperative evaluation consists of obesity evaluation, behavioural evaluation, medical evaluation, surgical evaluation, which includes education & potential risk, and anaesthetic evaluation.

Complications

The complications associated with bariatric surgery are : nutritional deficiencies, cardiovascular disease and sudden death, pulmonary embolism, which is the leading cause of death, bleeding and spleenic injury, gastrointestinal leaks - leading to sepsis, wound infections 1-3%, wound dehiscence, vomiting and diarrhea and stomal dilatation. The morbidity and mortality has come down for these procedures ranging from 10% and less than 19%.

As we enter the new millennium severe obesity remains an incurable disease. The consequences and cost to society are significant. Though etiology is becoming more clear, non-surgical treatments are still inadequate for achieving sustained and significant weight loss. Surgical procedures have evolved into safe and effective options. Newer technologies such as laparoscopy should add further advances to the field. For appropriately selected patients, surgery can achieve weight loss necessary to prevent the development of significant medical conditions and improve the quality of life. Unfortunately dietary indiscrimination and malabsorptive eating behaviour can result in weight loss failure despite an excellent surgical result. Therefore preoperative evaluation and education is a corner stone for long-term success.

CONCLUSIONS

Morbidly obese patients are encountered commonly. With changing lifestyles, high incidence of diabetes and coronary disease in the younger individuals, one may encounter them in the practice of anaesthesia for different types of surgical procedures, bariatric surgeries, investigations, trauma and in ICU setup. They do pose tremendous challenges. Understanding the pathophysiology, anticipating the problem and preventing calamities by a systematic approach will certainly bring down the complications rate.

References:

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