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# Orissa Medical Journal

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OBESITY - FACTS, MYTHS & CONTROVERSES

Obese comes from the latin word ‘Obesus’, past particulate of ‘Obedere’ (to devour) which is derived from ‘ob’ (meaning to) and ‘edere’ (meaning eat). Although for years world leading agencies and others are trying to mitigate the problems of starvation and hunger ironically enough more people now die of problems of obesity than from being overweight. In 1997 WHO formally recognised obesity as a global epidemic and in 2005 WHO estimated that 400 million adults (9.8%) were obese. In the later half of 20th century, when obesity was an upcoming problem in US, Americans were asked to look at the millions of Indians who were starving. No sooner a decade or two later than the epidemic seems to have struck India with 5% of the population suffering from obesity.

For obvious reason, life style changes and hormonal changes over a period of time leads to an increase incidence of obesity with age upto 50-60 years with more number of women being affected than men, yet CDCS third National Health and Nutrition Examination survey (NHANES) revealed that a higher percentage of children (14%) and adolescents (12%) were overweight.

Similarly when enough evidences are available about the negative health consequences of obesity, yet health outcomes in certain sub groups seem to be improved at an increased BMI, a phenomenon known as “obesity survival paradox”.

Obesity being a nutritional problem diet and exercise are the key to success in the weight losing programmes and believed by one and all. Yet hereditary influences and genetics account for 80% and 33% contribution to increased BMI respectively. Over a third of a century ago Neel at the University of Michigan proposed that the pressure of natural selection endowed our ancestors with a “thrifty genotype” which boosted the ability to store fat from each feast in order to sustain people through the next famine. Similarly single gene disorder like Bardet Biedl, Prader Willi, Ahlstrom and Cohen Syndrome can give rise to obesity.
With the whole world following the ‘No Tabacco Day’ in a desperate attempt to eradicate smoking as a silent killer, cessation of smoking is nevertheless associated with 4 to 5 Kg weight gain resulting in an increase in obesity.

Obesity is a cause of numerous life threatening disease like cardiovascular disease, non insulin dependent diabetes mellitus, certain form of cancer, gall stone, respiratory disorder, osteoarthritis with an overall increase in mortality. Most of these diseases are covered by insurances or reimbursement but ironically the insurance companies do not support treatment of obesity as such.

Obesity can cause devastating psychosocial problems. Overweight is connected to mental health, body image issues and suicidal tendencies but studies have shown that underweight people are more likely to deal with mental health problems. Professor Jorm from the centre for Mental Health Research at the Australian National University states that underweight people also have the advantage in that they have less physical disability and physical ill-health than obese people and that masks the underlying tendency to anxiety and depression, but when we extract out the physical ill-health component, we are left with this picture that it’s the underweight that have the worst mental health. However obesity still remains the cause of many life-threatening disease and is a cause of concern for the future generation.

(Rekha Das)
Editor
DO DOCTORS NEED PHARMACEUTICAL GIFTS OR SPONSORSHIP?

"Men who are occupied in the restoration of health to other men, by the joint exertion of skill and humanity, are above all the great of the earth. They even partake of divinity, since to preserve and renew is almost as noble as to create"

-- Voltaire

Anybody who receives a gift or complement, obviously has no necessity, neither choice nor demand for it. All of us have received gifts and also have surprised others with gifts. In the process of gifting the givers purposes are more defined than the takers. If anybody wants to stop the process, he has to warn the people who gift out rather than those at receiving end.

Everything is give and take in this world. We perform with a motive. Someone aims at achievements, someone at popularity; some expect money and others the recognition. We satisfy our personal ego in profession and in the process of helping and serving others.

Doctor community, being closely associated with life and death, sorrow and happiness, closely watch the laws of nature and mostly adapt at any circumstances. One never dreams to be a member of the profession because he is lured with attraction of gifts and sponsorship. He spends more years and put extra efforts to grab the professional skills the benefit of which is enjoyed by society at large. His knowledge, ability and performance add life to the years and years to the life of people. If artists like Amitabh Bachchan, leaders like Manmohan Singh and players like Sachin Tendulkar are performing that is because doctors have put them back in track. A doctor sacrifices his personal, familial and social life for professional challenges and in return what society gives him is negligible.

Which profession is flawless in rendering self less service to the people? From politicians to pilots, from businessmen to bureaucrats and builders, from merchants to media men, from advocates to accountants, from teachers to technocrats, from engineers to entrepreneurs, the so called god men and spiritual seers: if you screen around perhaps the medical professionals are the noblest.

In our country the administration has failed to utilise the benefits from the pharmaceutical industry for research and development in medical field. It has also failed to give doctors their due status and respect in the cadre of professionals. Warning them against receiving pharmaceutical gifts or sponsorship is another humiliating step against the saviours which only exemplifies inferiority complex of inability to become a member of this noble, healing profession and only time will answer to it.

Dr. Jayant Kumar Panda
Secretary
INTRODUCTION:

Target fixation for interventions in obesity has been a debate since obesity has been conceived as a disease. All treatments try to justify the action i.e. the principle that lay behind understanding the predominant etiology among many factors that cause obesity. Obesity has 4 different theories to explain it's etiology i.e. metabolic theory, genetic theory, neuro-endocrine theory, infective theory. Obesity treatments includes

a. Pharmaceutical treatments basically include reducing the appetite or reducing the amount absorbable fat. The pivot point of many currently prevailing pharmaco-therapeutic interventions is still based on the metabolic theory of obesity. According to the metabolic theory obesity is an imbalance of caloric homeostasis. Many new molecules to control or reduce obesity keep on coming to the market. Either they vanish without it's presence felt in the desired consumer community or the effectiveness is such that it is hard to believe that they really help reducing the problem. Further many drugs tax the pocket heavily. Finally most dangerous aspect is that it has side effects that are too dangerous to be not looked into for a disease which can have simple entry points of treatments such as diet & exercise. The dangers range from vitamin deficiency to suicidal tendency.

b. Surgical treatment is also primarily targeted to reducing the amount of food intake or reducing the intestinal absorptive surface. Hence surgery also keeps the metabolic theory at its center while making clinical decisions. The basis seems obsolete when high caloric density semi liquid or liquid diet can be ingested without being aware of the caloric value of the food. Apparently speaking surgery has a distinct advantage of reducing the risk associated with the obesity at a rapid pace. Weighing this modification of risk through surgery against other methods is a key question to be addressed. However, surgery still carries the disadvantage...
of procedural & iatrogenic mishaps, post-operative complications & recurrence.

c. The diet & exercise regimens are the bread & butter of many trained & untrained professionals. The diet & exercise regimen is also predominately based on metabolic theory of obesity. There is a high degree of uncertainty if the subject will lose weight or not, weight cycling (i.e. gaining the back the previous weight), commitment to lifestyle changes after weight loss. When the obesity etiologies are traced to genes, endocrine abnormalities & possibly infection then the metabolic perturbations accompanying the diet-exercise regimen recommendations seems fragile. However recent knowledge in diet & exercises is modified firstly by the fact that weight loss is attributed to sustained & continuous thermogenesis rather than the discrete diet & exercise caloric balancing acts. Secondly by gene-diet & gene-physical activity interactions and finally metabolic activity of brown fat.

THERMOGENESIS:

Thermic activity of cells & body as a whole is a preeminent activity for sustenance of human life form that is homoeothermically designed. To liberate & maintain heat cells require energy that come in the form of food. The heat liberating act is linked to sustained energy supply that is possible from all the sources including the labile proteins of the skeletal muscles. However to churn energy in the form of heat; stored carbohydrate in the form of glycogen needs to be converted into glucose which must enter the phosphorylating pathways to form ATP & later on ATP must be dispensed for biological activities producing heat. But, only portions of stored glycogen can be made labile by even the most exhaustive physical demand. That renders the thermal energy production dependant on the fat store. This kind of biological dependence on fat for supplying heat has an important evolutionary significance as fat almost represents unlimited energy supply (2200 calories in glycogen versus 1 lakh calories in fat stores of body) for homoeothermic characteristics. Scientists are trying to exploit this link to deplete excess fat in obese & overweight patients.

TYPES OF THERMOGENESIS IN DIET & EXERCISE:

1. Diet induced thermogenesis (DIT) or the specific dynamic action of food (SDA)

2. Exercise activity thermogenesis (EAT)

3. Non-exercise activity thermogenesis (NEAT) : energy expenditure except in sleeping, eating & sports like activities

Non-shivering thermogenesis was originally defined as a cold-induced
increase in heat production not associated with the muscle activity of shivering⁴. Thermogenesis discussed in points 1, 2 & 3 are in a combination can be called non-shivering thermogenesis. To some degree, all cells of endotherms give off heat, especially when body temperature is below a regulatory threshold. However, brown adipose tissue is highly specialized for this non-shivering thermogenesis. First, each cell has a higher number of mitochondria compared to more typical cells. Second, these mitochondria have a higher-than-normal concentration of a specific molecule i.e. thermogenin in the inner membrane. (Brown fat is described below)

A compartmental model of sustained thermogenesis to affect the fat stores in obesity: The author of this article proposes the scientific community to conceptually compartmentalize the thermogenesis acts of diet, exercise & non-exercise activities. He hopes by doing so the caloric expenditure calculations in thermogenic terms will be easy & later assimilation of all such calculations provides the overall thermic expenditure. Differentiating physical activities in the terms of EAT & NEAT are already a step in that direction.

THE BROWN ADIPOSE TISSUE (BAT):

The function of brown adipose tissue is to transfer energy from food into heat; physiologically, both the heat produced and the resulting decrease in metabolic efficiency can be of significance⁷.

It was believed that after infants grow up, most of the mitochondria (which are responsible for the brown color) in brown adipose tissue disappear, and the tissue becomes similar in function and appearance to white fat. However, more recent research has shown that brown fat is related not to white fat, but to skeletal muscle. Brown fat cells and muscle cells both come from the middle embryo layer. The mesoderm is the source of myocytes (muscle cells), adipocytes, and chondrocytes (cartilage cells). The adipocytes give rise to white fat cells and brown fat cells. Both the muscle & brown fat cells have the same marker on their surface (Myf5, myogenic factor), which white fat cells don't have. Furthermore, muscle cells that were cultured with the transcription factor PRDM16 were converted into brown fat cells, and brown fat cells without PRDM16 were converted into muscle cells⁸.

However, there may be two types of brown fat cells - with and without Myf5. The other type, without Myf5, may share the same origin as white fat cells. They both seem to be derived from pericytes, the cells which surround the blood vessels that run through white fat tissue⁹.

Contrary to common scientific belief PET scan studies of adult humans have shown that brown fat cells are remain to survive in adulthood at the upper chest and neck. The brown fat cells on PET scan become more visible with cold exposure, and less visible with adrenergic beta blocker administration before the scan. Brown adipose tissue grows and
regresses in accordance with the extent to which it is stimulated, both by cold and by diet. Further the capacity of the animal for cold-induced nonshivering thermogenesis and diet-induced thermogenesis increases or decreases accordingly\textsuperscript{5}.

**NEURAL REGULATION OF BAT:**

The neural regulation of thermogenesis in brown adipose tissue is thus not only part of the central control mechanisms involved in thermoregulation but also part of those involved in the regulation of energy balance\textsuperscript{5}.

The central neural components involved in control of thermoregulation are better understood than are those involved in the regulation of energy balance. Studies of animal with hypothalamic obesity indicate that the control of diet-induced thermogenesis in brown adipose tissue requires the participation of the ventromedial region of the hypothalamus whereas the control of cold-induced nonshivering thermogenesis does not\textsuperscript{5}.

**WEIGHT LOSS PROSPECTIVE VIA BAT**

Understanding of adult fat cell physiology could lead to a new method of weight loss, since brown fat takes calories from normal fat and burns it. Recent research shows it to be a metabolic process located primarily in brown adipose tissue and controlled by the activity of the sympathetic nervous supply of this tissue. Another stimulus to sympathetic nervous activity, the ingestion of food, promotes diet-induced thermogenesis in brown adipose tissue. Hence both the acute activity of the tissue, i.e., the heat production, and the recruitment process in the tissue (that results in a higher thermogenic capacity) are under the control of norepinephrine released from sympathetic nerves\textsuperscript{5,7}.

Heat production from brown adipose tissue is activated whenever the organism is in need of extra heat. Feeding as such also results in activation of brown adipose tissue; a series of diets, apparently all characterized by being low in protein, result in a leptin-dependent recruitment of the tissue; this metaboloregulatory thermogenesis is also under hypothalamic control. When the tissue is active, high amounts of lipids and glucose are combusted in the tissue\textsuperscript{7}.

**GENE-BEHAVIOR INTERACTIONS I.E. GENE-DIET & GENE-PHYSICAL ACTIVITY INTERACTIONS**

Though few researchers claim that the human genomics is decoded, in clinical terms many complex issues remain to be addressed and one of them is the genetics of human body fat content (obesity). In rat experiments, simultaneous genetic and physiological analysis points to critical molecules and cells in central nervous system and "peripheral" pathways that mediate the fine control over the defense of body fat\textsuperscript{3}. Despite many such
compelling evidences that genes play an important role in the development of obesity, many people argue that the increasing prevalence of obesity is simply due to an abundance of palatable food and a dearth of opportunities for physical exercise.

While activity and eating behaviors contribute substantially to the development of obesity, considering these to be the only etiologic factors is directly contradictory to what is now known about how eating and energy balance are regulated. According to a recently reviewed literature, genetic endowment influences a substantial percentage of responses to nutrition and exercise in both humans and animal models. Many of the genes participating in these pathways have reciprocal effects on both energy intake and expenditure, though different genes may have primary roles in respective responses to weight gain or loss. Such distinctions have important consequences for both research and treatment strategies. There is also a substantial amount of evidence that treatment responses to exercise and diet strategies may also be regulated by genes. Understanding gene-response relationships is the key to developing more efficacious intervention and prevention programs for obesity.

Gene-nutrient and gene-physical activity: is there a paradigm shift from metabolic theory to genetic theory of metabolism?

Human genetic studies provide evidence that body weight response to over- and underfeeding and to exercise is associated with specific genes & specific mechanisms. High-density genome-wide scans applicable to large numbers of human subjects have revolutionized the study of the genetics of obesity by generating substantial numbers of powerful linkage signals from smaller genetic intervals. Now whatever we know about many of the genes implicated will not have been previously related to energy homeostasis and will have relatively small effects on the associated phenotype(s).

Phenotypes related to energy intake and expenditure—which clearly are the major determinants of net adipose tissue storage—are not salient when individuals are in energy balance (weight stable); measurements obtained during weight perturbation studies are likely to provide more revealing phenotypes for genetic analysis. However for tools to study gene x gene interactions that must ultimately convey aggregate genetic effects on adiposity are not concretely developed. The body mass index (BMI) is a useful gross indicator of adiposity, but more refined measurements of body composition and energy homeostasis will be required to understand the functional consequences of allelic variation in genes of interest.

GENE-PHYSICAL ACTIVITY INTERACTIONS: OVERVIEW OF HUMAN STUDIES.

Physical activity level is an important component of the total daily energy
expenditure and as such contributes to body weight regulation. A body of data indicates that the level of physical activity plays a role in the risk of excessive weight gain, in weight loss programs, and particularly in the prevention of weight regain.

Our understanding of the molecular processes controlling eating behavior, in particular, has accelerated exponentially in the last 10 years, and this is one area in which obesity genetics has made great progress. Our challenge is to understand more fully how genetic variation may interact with behavioral factors to influence the regulation of body weight and adiposity. Although exercise and diet strategies are used routinely for obesity treatment, there is a huge variability in how individuals respond to these interventions\(^1\). Most studies dealing with potential gene-physical activity interaction effects use an exercise and fitness or performance model as opposed to an obesity-driven model. From these studies, it is clear that there are considerable individual differences in the response to an exercise regimen and that there is a substantial familial aggregation component to the observed heterogeneity\(^4\).

Few studies have focused on the role of specific genes in accounting for the highly prevalent gene-exercise interaction effects. Results for specific genes have been inconsistent with few exceptions. Progress is likely to come when studies will be designed to truly address gene-exercise or physical activity interaction issues and with sample sizes that will provide adequate statistical power\(^4\). Genetics has a substantial impact on responses to both diet and exercise. However, present day scenario does not allow individual diet and exercise recommendations on whatsoever knowledge we possess on this issue\(^2\).

REFERENCES:

INTRODUCTION:

Obesity has reached epidemic proportions in India in the 21st century with morbid obesity affecting 5% of the countries population. Although currently defined by the WHO (World Health Organisation) in terms of excess weight for a given height, obesity is best considered as being an increase in adiposity. It is interesting to note that along with obesity sleep disordered breathing (SDB) are on rise. Using data from 690 adults in the Wisconsin sleep cohort study Peppard et al. found that 10% weight loss was associated with 26% decrease in AHI. Thus same 10% weight gain was associated with a 6 fold increase in the odds of developing moderate to severe SDB. Because of this and other similar findings, there has been considerable interest in treating SDB using a range of obesity interventions such as dietary changes, behavioral modification, and for selected patients bariatric surgery. Unfortunately the success of these interventions has been inconsistent. This inconsistency triggers a thought, that SDB may be associated with alteration in energy metabolism that, in turn, lead to weight gain and complicate the treatment of these two disorders that often coexist.

OBESITY AND LEPTIN

According to the First Law of Thermodynamics, obesity results from an imbalance between energy expenditure and energy intake culminating in excessive accumulation of fat in adipose tissue, liver, muscle, pancreatic islets, and other organs involved in metabolism. Our knowledge of the neurobiology of feeding and energy homeostasis has benefited from the discovery of fat and gut derived hormones and their target in the hypothalamus. Fig-1.

Leptin is secreted by fat cells (adipocytes) and was originally thought to signal to the brain to inhibit food intake and decrease weight. This concept was partly driven by the observation that humans and rodents lacking in functional leptin protein or receptor manifested various feeding and obesity. As was predicted, leptin treatment particularly
direct injection of leptin into the cerebral ventricle or hypothalamus, profoundly inhibited food intake and decreased weight and fat in animals lacking leptin. However, the notion of leptin as an anti-obesity hormone was called into questions because obesity is typically associated with high leptin levels not leptin deficiency. Moreover, rodents and humans that become obese on a high fat (Western) diet do not respond to leptin. Studies have demonstrated that leptin is transported into the brain, binds to its receptor in the hypothalamus, and activates JAK-STAT3, leading to suppression of "orexigenic peptide" (e.g. neuropeptide Y and or goute-related protein which normally increase food intake), and increase in "anorexigenic peptides" (e.g proopiomelanocortin and corticotrophin-releasing hormone which normally decrease food intake).

**Sleep-disordered breathing and hormones with special reference to leptin.**

Sleep-disordered breathing (SDB) is an extremely common condition that compromises the vital functions of respiration and circulation. There is a myriad of adaptive physiological acid-base balance that are endangered. Therefore, SDB has wide spread systemic effects, which are unfortunately, rarely considered by medical professionals other than those specialized in diagnosing and treating this disorder. The many adaptive endocrine alterations associated with SDB are an example of how a seemingly local upper airway dysfunction induces systemic consequences affecting every cell of the organism. Conversely, manifestation of sleep apnoea is critically linked with control of breathing. All endocrine changes that increase the tendency for periodic breathing will also increase the episodes of sleep apnoea. The present review focuses on SDB secondary to leptin and its effect on obesity.

The concept of SDB has markedly evolved during the past decade. The episodes of sleep apnoea and hypopnoea result from periodic total or partial closure of the upper airway. These episodes are often accompanied by hypoxaemia and terminated with cortical electroencephalogram arousals. The severity of SDB is commonly expressed as the apnoea/hypopnoea index (AHI), which indicates the frequency of the apnoea/hypopnoea episodes per hour of sleep. Some authors also include the
respiratory effort-related arousals and express the severity of SDB as the respiratory disturbance index (RDI).

Sleep apnoea seems like an epidemic, which spreads rapidly with obesity, another major health problem in Western societies and Western aping society.

A number of hormones interact with sleep9 and breathing10. SDB affects hormones via a number of mechanisms. Conversely, hormones and endocrine states induce, aggravate or alleviate SDB. Finally, nasal continuous positive airway pressure (CPAP) therapy influences hormone secretion.

SDB and sleep disturbances may interact with hormones in several ways. Episodes of apnoea or hypopnoea cause sleep fragmentation and disturb sleep cycles and stages. Arousals may induce stress response resulting in increased levels of stress hormones11. Hypoxia may also have direct effects on central neurotransmitters12, which result in alterations in the hypothalamo-pituitary axis and in secretion of the peripheral endocrine glands13. Hypercapnia alone or combined with hypoxia may increase levels of renin, adrenocorticotrophic hormone, corticosteroids, aldosterone and vasopressin13,14. Finally, disorganization of sleep, sleep loss and naps disturb sleep-controlled endocrine rhythm resulting in endocrine and metabolic abnormalities.

Leptin, besides its best known function as a satiety hormones, leptin is also a powerful respiratory stimulant15. Plasma leptin levels are higher in sleep apnoeics than in controls matched for BMI16. Furthermore, hypercapnic patients with obstructive sleep apnoea syndrome (OSAS) have higher leptin levels than eucapnic BMI-matched controls with sleep apnoea17. Leptin secretion could provide an adaptive mechanism to enhance ventilation in patients with severe respiratory impair resistance at the level of the central nervous system. Elevated leptin levels are likely to contribute to comorbidity of OSAS because high leptin levels are associated with coronary heart disease18, insulin resistance19, impaired fibrinolysis20, development of obesity21 or type-2 diabetes, which are all highly prevalent in patients with OSAS.

CONCLUSION

Obesity and sleep disorder breathing has acquired the shape of epidemic in developing and developed countries. Though sleep disorder breathing is still under-diagnosed obesity, a physical change is diagnosed early. Contrary to the believe that obesity leads to SDB, increased leptin level in SDB which leads to leptin resistance which could be an isolated factor for obesity.

REFERENCE


BARIATRIC SURGERY

Sreejoy Patnaik
Consultant surgeon
Shanti hospital, Cuttack.

INTRODUCTION:

Obesity, once considered an "aristocratic" symbol of affluence, has now assumed the character of a life-threatening disease. It has become a serious epidemiologic health issue that is causing enormous psychosocial and economic problems in our consumer society.

While the growing problems of obesity have been well documented and explained in western history and their conservative treatment option are widely known, it is only in recent years that physicians have made significant progress toward mastering the challenging problem of bariatric surgery (obesity surgery, weight-loss surgery).

SURGICAL TREATMENT

Surgical procedure to help control obesity one generally divided in two categories

(1) Restrictive
(2) Restrictive as well and malabsorptive.

In the restrictive category two procedures are presently carried out

- Lap gastric banding
- Sleeve gastrectomy

In the restrictive as well as malabsorptive category:
- Laparoscopic Gastric bypass surgery is done.

LAP GASTRIC BANDING

The LAGB system has been used in Europe since 1934. It has been reported that laparoscopic placement of the band is less painful than open placement and is associated with reduced hospitalization and disability.

The LAP-BAND System is indicated for use in weight reduction for severely obese patients with a Body Mass index (BMI) of the least 40 or higher, or a BMI of at least 35 with one or more co-morbid conditions. It is indicated for use only in severely obese adult patients who have failed more conservative weight reduction alternatives such as supervised diet, exercise and behavior modification programs. Patients who elect to have this
surgery must make the commitment to accept significant and permanent eating habits.

Reversible procedure-stomach anatomy remains intact in addition to the above advantages, the benefits of a restrictive procedure over a malabsorptive procedure were retained:

No permanent cutting or bypassing of the intestinal tract

No vitamin deficiencies associated with malabsorption of nutrition

No "Dumping Syndrome" related to dietary intake restrictions

No permanent rearrangement of the intestinal tract

The LAP-BAND System is a long-term implantable medical device designed to induce weight loss in severely obese patients by restricting food consumption. The slip-through buckle design band is designed to ease laparoscopic placement around the stomach and form a small gastric pouch and stoma. No cutting or stapling of the stomach is required and there is no bypassing of portions of the stomach or intestines. The initial pouch and stoma size are determined during surgery and are defined by the use of a calibration tube (pouch size) and pressure sensitive sensor tip (stoma size).

After surgery, the stoma outlet form the small upper stomach pouch to the rest of the stomach can be adjusted percutaneously. The inner surface of the band is inflatable and connected by kink-resistant tubing to a subcutaneous access port. By injecting or withdrawing saline via the access port, the size of the stoma can be decreased or increased, respectively, enabling weight loss to be adjusted to individual requirements and preferences without the need for additional surgery.

Early experience with the LAP-BAND System in Europe led to a modification of the surgical technique resulting in a significant improvement in results2,3,4,5,6.

Dr. Belachew (22), who pioneered the introduction of the LAP-BAND System, has published the result of his series of 350 LAP-BAND System patients.

The LAP-BAND System depends on the success of the surgical procedure and the ability of the patient to change his or her diet and eating behaviour. After surgery, LAP-BAND System patients are encouraged to eat a balanced diet and to avoid the eating patterns of their pre-surgery lifestyle. Patients are particularly discouraged from consuming fatty or sweet liquids as these pass through the stoma without restriction. Likewise, drinking fluid while eating liquidates the food which then passes quickly through the stoma. By eating well-chewed solid food, the restrictive effect of the band produces a feeling of early satiety and long-lasting fullness and reinforces the patient's ability to eat smaller meals. After adequate weight loss, patients are encouraged to take up some exercise.

In summary, published clinical reports on the LAP-BAND System note several advantages over other surgical techniques
and many surgeons have adopted the LAP-BAND System over other obesity procedure\(^7\). The procedure is considered less traumatic than other forms of restrictive surgery as there is no cutting, crushing or stapling of the stomach or gastrointestinal tract and, should it be necessary to remove the band, normal stomach anatomy is restored.

Utilizing the LAP-BAND System, surgeons report that the average operating time is two hours and the patients is able to return home after two to three days\(^5,9,10,11,12,13\). Studies indicate a weight loss similar to other gastric restrictive surgical procedure but with an adjustability and reversibility not available in other types of obesity surgery.

The LAP-BAND System would appear to be a safe and efficacious surgical option.

**SLEEVE GASTRECTOMY**

Sleeve gastrectomy (SG) was first described in 1988 when Scopinaro's technique of biliopancreatic diversion (BPD) with distal gastrectomy and gastroileostomy was modified by Hess and simultaneously by Marceau\(^14,15,16\).

Due to its acceptably low complication rate and relatives ease of technical performance, SG was subsequently recommended as an initial step in the management of super obese patients or those with a high operative risk due to extensive comorbid conditions to minimize the associated morbidity and mortality.

As experience with the technique increased the role of LSG evolved and many began to consider it as a primary restrictive bariatric procedure. Early reports of prospective and retrospective studies were encouraging\(^17,18\). Mognot et al. reported their initial experience of 10 patients with a mean BMI of 64 kg/m\(^2\) (range 61-80) who underwent LSG. The mean operative time was 2 hours, all procedures were completed laparoscopically, and there were no postoperative complications or mortality. At 1 year the average EWL was 51\(^%\)\(^19\). A further retrospective analysis of 60 patients who under weight LSG demonstrated an EWL of 83\(^%\) at 1 year with a median weight loss of 25 kg. More importantly there was a significant improvement in comorbid conditions including diabetes, hypertension, dyslipidemia, and joint pain\(^20\).

The first part of duodenum, pylorus, antrum, lesser curvature and vagal nerve integrity are maintained creating moderate restriction while allowing a relatively normal eating behaviour.

Due to if acceptably how complication rate & relatively ease of technical performance, SG was subsequently recommended as an initial step in the management of super obese patients or those with a high operative risk due to extensive co-morbid conditions to minimize the associated morbidity & mortality. The average weight loss was 37kgs following SG a 11 months period.

In this procedure, a point on the curvature, approximately 4cm proximal to
the pylorus identified as the distal extent of the resection.

The Harmonic scalpel is used to divide along the greater curvature up to the angel of his. A 38fr is inserted trans-orally to the level of the distal stomach. Lineal cutting ENDO-GIA staplers are used to vertically transect the stomach creating an arrow gastric tube with an estimated capacity of 100 to 150cm. The stapled is then overseen with a running non-absorbable suture. The resected stomach is placed in a specimen bag & extracted. All patients have a routine methylene blue dye test to confirm any leak intra-operatively.

Laparoscopic SG is an effective primary restrictive procedure to achieve weight loss. It is a relatively straightforward procedure that can be performed laparoscopically with an extremely low conversion rate. Operative time is reasonable ranging from 1-2 hours & operative time for LSG was 80min. LSG is technically feasible in high risk patients with significant morbidity, and also in very obese patient with BMI >70kg/m².

CONCLUSION:

LSG can be performed with minor complications & low mortality. It has been demonstrated to be a safe and effective procedure in the short term. Prospective studies are required after LSG to determine the long term outcome, and efficacy of maintenance of weight loss & resolution of co-morbid conditions.

Surgical treatment is the most effective method to induce weight loss in morbidity obese patients. Gastric bypass is one of the most well-documented operations in obesity treatment. Mason and Griffen presented this operation already in the 1960s. Since then, and particularly due to the advent of laparoscopic gastric bypass, Gastric bypass is known to yield excellent long-term results regarding weight reduction with only few eating disturbances. However it is considered a large operation with the potential of severe complications as well as some metabolic disturbances, mainly Vitamin B12 with iron deficiency. The benefits of laparoscopy are undisputed and the procedure has shown to be also effective in the treatment of morbidly obese.

To gain access to the abdominal cavity can sometimes be difficult in morbidly obese patients. There are several ways described for access such as open Hasson technique at the umbilicus, use of the Verres needle, trocars with shielded tip, radial expandable trocars, optical trocars etc. In the majority of obese patients, the optical trocar provides a clear image of the various layers of the abdominal wall allowing for a safe access. Port placement involves the surgeon standing on the patients right side. If the surgeons uses a French position between the patients legs, the left-hand working port may have to be placed in a more lateral position to the right side of the patients. The camera port should be placed no more than a maximum of 20cm below the xiphoid process and slightly to the left of the midline in order to spare the ligamentum teres.
In purely restrictive procedure the pouch volume is of utmost importance. The preoperative decision-making, the size of the gastric pouch should be determined with the goal of minimizing the risk of the pouch dilatation, staple-line dehiscence, reflux esophagitis and marginal ulceration. The creation of a micro-pouch is capable of eliminating these problems without impairing the patient’s ability for reasonable food intake. On the other hand, pouch size seems to be not as critical in patients undergoing gastric bypass surgery.

Division of omentum provides an easy access to the ligament of Treitz and also shortens the distance between the proximal jejunum and the gastric pouch when using an antecolic route of the Roux-limb construction. Even though division of the omentum may not always be necessary on the routine basis, it adds very little to the complexity of the operation or time.

Several techniques to create a Gastroenterostoma have been described in the literature, including the original circular stapling, totally hand-sewn anastomosis or a combined technique involving linear stapling and anterior running suture of the stoma. With the laparoscopic approach the small intestine is placed in an antecolic antegastric position and the gastro-entero-anastomosis is accomplished using a linear stapler with blue cartridge. The anterior opening is then closed with a running suture using a stay suture at the opposite side. The proximal biliopancreatic limb of the jejunum is made only long enough to reach up to the pouch. The next step is to construct an omega-loop where the entero-entero-anastomosis is fashioned in a similar way, using a posterior linear stapler with white cartridge, and placing an anterior running suture with stay suture on the opposite lateral end of the opening. To avoid the risk of blue reflux, the jejunum is than divided between the entero-entero-anastomosis and the gastro-entero-anastomosis. Accordingly, the final construction will be an antecolic antegastric Roux-en-Y.

REFERENCE


INTRODUCTION:

Obesity is complex and multifactorial, and is defined as a condition of excessive fat accumulation in adipose tissue to the extent that health may be impaired. It occurs when the net energy intake exceeds the net energy expenditure. It is a multisystemic disorder, particularly involving the respiratory and cardiovascular systems, therefore, a multidisciplinary approach is required.

* In obese general surgical patients, there are increased technical problems with surgery, increased complications and, often, increased length of stay, but studies differs in their prediction of the effects of obesity on mortality.

CLINICAL IMPLICATIONS & OUTCOME

Patients with obesity are different by physical, physiological, psychological, and co-existing pathological factors. They also have altered pharmacokinetics.

* In ICU, studies have shown conflicting results as to whether obesity increases mortality, but length of stay is prolonged.

* Similarly, in cardiac surgery patients, obesity is associated with more postoperative complications and longer hospitalization, but not with an increased early or long-term mortality.

AIRWAY

Difficulties in airway management in obese patients arise from limitations in neck movement (especially when BMI >40) and excessive adiposity in front of neck, submental area and chest. Additionally they may have retropalatal redundant pharyngeal tissue narrowing the airway. Obstructive sleep apnea (5%) may co-exist.

RESPIRATORY SYSTEM

Obese patients produce excessive carbon dioxide ($VCO_2$) and have increased oxygen consumption ($VO_2$). This is related to an increased effort and work of breathing of up to 70%\(^9\,^{10}\). The excessive chest wall adiposity results in
atelectasis. The reduced functional residual capacity may be below the closing capacity. Morbidly obese patients can have alveolar closures even when upright. Such patients rapidly desaturate and may need intubation in a semi-reclining position. Splinting of the diaphragm due to pressure from abdominal contents in supine and Trendelenberg positions can cause ventilation perfusion mismatch. The co-existing pathologies include increased incidence of asthma, pulmonary hypertension and obesity hypoventilation syndrome (8%).

CARDIOVASCULAR SYSTEM

The circulatory volume and consequently the stroke volume are increased. The increased cardiac output reduces the ability for further increases if need arises. Patients may show a normal left ventricular function with diminished compliance. In extreme cases, ventricular dysfunction can become significant with associated pulmonary hypertension. Comorbidities include hypertension (mild to moderate 50-60%, severe 5-10%), atherosclerosis, coronary artery disease, cerebrovascular injury, and deep vein thrombosis (DVT) risk.

GASTROINTESTINAL SYSTEM

Fasting obese patients often have a gastric pH < 2.5 along with increased residual gastric volume. Co-existing gastroesophageal reflux and hiatus hernia can predispose to pulmonary aspiration. Hyperlipidemia and fatty liver have been associated increased risk of gastric cancer.

Others

* Endocrinal - diabetes mellitus, insulin resistance, hypothyroidism.
* Musculoskeletal - arthritis.
* Metabolic - gout, dyslipidemia.
* Psychological - depression, anxiety, low self-esteem, social problems.
PHARMACOKINETICS

Ideal Body Weight (IBW) = Height (cm) - x
(x = 100 for adult males and 105 for adult females)

* The bioavailability of oral drugs is unaffected in obese patients. Intramuscular route is unpredictable.

* Obese patients have increased circulating volume, hence watersoluble drugs have more volume of distribution (Vd). The Vd for fatsoluble drugs is significantly increased\textsuperscript{12,13}.

* Clearance (Cl) and termination of drugs remain similar to lean patients.

The loading dose of a drug is calculated based on Vd and infusion rates are calculated based on Cl. Loading dose of drugs with distribution restricted to lean tissues only are calculated based on IBW. Drugs which are equally distributed to lean and fat tissues should have loading dose calculations based on actual body weight (ABW).

If Cl of the drug increases with obesity, then the infusion rate is calculated based on ABW. Drugs whose Cl remains unaffected or decreased by obesity should have calculation of infusion rates based on IBW.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosing</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopental</td>
<td>IBW (somewhat increased)</td>
<td>Increased V\textsubscript{d}, increased blood volume, cardiac output, and muscle mass. Increased absolute dose. Prolonged duration of action. Longer elimination half-life. Adjust loading/induction dose accordingly.</td>
</tr>
<tr>
<td>Propofol</td>
<td>Induction: TBW (somewhat reduced)</td>
<td>Highly lipophilic. Total clearance and V\textsubscript{d} at steady state correlate well with TBW. Keep in mind negative cardiovascular effects. High affinity for well-perfused organs.</td>
</tr>
<tr>
<td></td>
<td>Maintenance: TBW</td>
<td></td>
</tr>
<tr>
<td>Midazolam</td>
<td>Loading dose: TBW (somewhat reduced)</td>
<td>Significant increase in V\textsubscript{d} and elimination half-life. Sedative effect correlates better with distribution than elimination/clearance. Prolonged sedation because higher loading dose is needed to achieve adequate serum concentration.</td>
</tr>
<tr>
<td></td>
<td>Maintenance infusion: IBW</td>
<td></td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>TBW</td>
<td>Larger extracellular fluid compartment in the obese. Pseudocholinesterase activity increases with increasing weight.</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>IBW</td>
<td>Faster onset and longer duration when dosed according to TBW. Pharmacokinetics and pharmacodynamics not altered in obese subjects.</td>
</tr>
<tr>
<td>Local Anesthetic</td>
<td>Distribution</td>
<td>Pharmacological Properties</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>IBW</td>
<td>Prolonged action when dosed according to TBW. Obesity does not alter distribution or elimination of the drug.</td>
</tr>
<tr>
<td>Atracurium</td>
<td>TBW</td>
<td>$V_d$, absolute clearance, and elimination half-life unchanged by obesity. Unchanged dose per unit body weight prolongation of recovery because of organ-independent elimination.</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>IBW</td>
<td>Pharmacokinetics similar to atracurium but prolonged duration of action when dosed according to TBW.</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Derived “pharmacokinetic (PK) mass”</td>
<td>Measured total body clearance has a nonlinear relationship to TBW. Fentanyl dosing based on pharmacokinetic mass correlates better with clearance. Dosing based on TBW overestimates does requirements in the obese.</td>
</tr>
<tr>
<td>Sufentanil</td>
<td>Loading dose: TBW</td>
<td>Increased $V_d$ and prolonged elimination half-life, which correlates with degree of obesity. Clearance similar in obese and nonobese. Overestimation of plasma concentration occurs in the morbidly obese range (BMI &gt;40 kg/m²).</td>
</tr>
<tr>
<td></td>
<td>Maintenance: IBW</td>
<td></td>
</tr>
<tr>
<td>Remifentanil</td>
<td>IBW</td>
<td>Pharmacokinetics similar in obese and non-obese subjects. Systemic clearance and $V_d$ corrected per kg of TBW is significantly smaller in the obese. Consider age and lean body mass for dosing.</td>
</tr>
<tr>
<td>Dexametomidine</td>
<td>TBW</td>
<td>Lacks significant effect on respiration. Ideally suitable as an analgesic adjuvant in morbidly obese subjects in whom opioid-induced respiratory depression may be catastrophic.</td>
</tr>
</tbody>
</table>

BMI, body mass index; IBW, ideal body weight; TBW, total body weight; $V_d$, volume of distribution.

The above-mentioned are broad guidelines. There is interpatient variability in response, hence case-by-case clinical assessment and titration of dose to effect remains central to the delivery of a safe anesthetic.
ANESTHETIC CONSIDERATIONS

Apart from general surgery, obese patients can present for restrictive/ malabsorptive bariatric surgery. An intragastric balloon sited via endoscopy under sedation in patients with BMI > 60 facilitates weight loss, enabling them to undergo more definitive procedure safely. Open abdominal procedures are increasingly being replaced by endoscopic and laparoscopic procedures. Patients are encouraged to lose weight prior to elective procedures by changing dietary habits and exercising.

PREOPERATIVE PHASE

* Obese patients are best assessed in a lying down position as this highlights physiological limitations and positioning problems.

* Mouth opening. Mallampatti score, neck extension, and circumference (collar size > 17.5 inches) should be noted in combination, these help to predict a potentially difficult airway.

* Preoperative investigation is directed by the history and examination findings of the patient and the nature of the proposed surgery. It may include full blood count (to exclude polycythemia), electrolyte, renal and liver function test, ECG, Echocardiography, chest X-ray, supine and upright blood gases and overnight oximetry. Spirometry as a preassessment screening tool does not make any difference in predicting postoperative pulmonary complications.

ECG changes in Obesity

Low voltage complexes
LV hypertrophy or strain
Prolonged QT/QTc
Inferolateral T wave abnormalities
Right axis deviation or RBBB
P pulmonale

* Preoperative nasal continuous positive airway pressure (CPAP) and bilevel-positive airway pressure (BIPAP) will benefit patients with obstructive sleep apnea.

* A preoperative discussion about the need for extubation when fully awake and in a sitting position may minimize patient anxiety and improve cooperation during recovery.

* Standard precautions such as preoperative fasting, H2 inhibitors, metoclopramide, and nonparticulate antacids are given to avoid aspiration injury.

* Morbidly obese patients often have difficulties lying flat. A wide electric tilting bed is useful since narrow trolleys cause cramping of the head and shoulders, compromising airway management.

POSITIONING

The anesthetist

* Standing at the head end, on a little
platform if needed, looking down the airway. This minimizes undue strain while attempting to safely bag, mask and secure the patient's airway, particularly when the patient is semi-reclined.

THE PATIENT

* Proper support ("ramping") with the head neutral or slightly flexed.

Head and neck positioning of patient to align the oral, laryngeal and pharyngeal axes. Alignment can be guided by being able to draw an imaginary horizontal line between tragus and sternum.

* Use of shorter handles and polio laryngoscope blade will optimize the chances of successful laryngoscopy. The efficacy of cricoid pressure in obese patients has been questioned. In extreme cases, awake fiberoptic intubation of the trachea is indicated.

* Special arm boards are used for proper positioning. Padded support along the whole length of the arms, with joints in slight flexion, will avoid the risk of brachial plexus, nerve compression and joint injuries. Nerves may also be vulnerable due to vitamin deficiencies secondary to inappropriate dieting.

Lumbar spine extension may be a problem due to buttocks fulcrumming the legs. Flexion of the hips by supporting the knees will minimize strain on lumbar spine.

MONITORING

* Breath sounds are distant and ETCO2 is very important. Endotracheal tubes tend to move in obese patients especially undergoing laparoscopic surgery. A visual confirmation using a fiberoptic scope may well be indicated.

* Pulse oximetry to monitor oxygenation is similar to the lean patient.

Noninvasive blood pressure (NIBP) monitoring of the upper arm is a problem because of the girth and the conical shape of the upper arm. NIBP
in the forearm is usually simple and reliable, but may show slightly wider pulse pressure.

* Invasive blood pressure and blood gas monitoring using an arterial line are not always indicated, but are useful in prolonged cases. They carry their own risks and challenges.

* Central venous pressure monitoring is used only if clinically indicated. Ultrasound guidance will minimize failure and avoidable risks. Intravenous cannulation may be a problem due to excessive subcutaneous tissue and might warrant central access.

* Monitoring neuromuscular function in the presence of NDMRs is important. Complete reversal is mandatory prior to attempting extubation.

**INTRAOPERATIVE PHASE**

* A standard asleep intubation is a practical and safe way to secure the airway.

* Difficult bag and mask ventilation can be overcome by a four handed technique and by the use of mechanical ventilator with the mask.

* Low oxygen saturations, high ETCO2 and high airway pressures are not uncommon. An initial high concentration of inspired oxygen followed by pulse oximetry/blood gas-guided titration is recommended. Ventilation and oxygenation can be improved by:

* Positive end expiratory pressure (PEEP);
* Reverse Trendelenberg position to offload the abdomen;
* Discussion with the surgeon regarding abdominal packing;
* Offloading the pressure by manually or mechanically lifting the abdominal pannus.

* Effective temperature maintenance is important by forced warm air over blankets are very effective along with fluid warmers.

* Use of short acting agents such as remifentanil, sevoflurane and desflurane should be used to minimize post operative hypoxemia and hypoventilation.

* Barotrauma is possible; consider "pressure" rather than "volume" controlled ventilation. Maintaining minute ventilation by increasing the respiratory rate while limiting the tidal volume may minimise barotrauma;

* Risk of aspiration during extubation can be reduced by extubating the obese patient while fully awake and in the sitting position. This facilitates increased tidal volumes as the abdominal contents gravitate away from the diaphragm.

**POSTOPERATIVE PHASE**

* Patients with preoperative hypoxia, those undergoing thoracic or upper abdominal surgery, and those with
significant co-morbidity should be nursed in high-care areas in the immediate postoperative period. Supplemental oxygen remains valuable.

* Post operative shivering, which increases oxygen consumption, prolongs the effect of some anesthetic agents and increase cardiovascular stress.

* Good pain relief is necessary to avoid postoperative complications, ideally using regional techniques. Nonopioid analgesics and local anesthetic blocks have a role in balanced analgesia. Opiates require careful titration, since overdosage can cause postoperative respiratory depression, while poor pain control can lead to difficulty in chest expansion with resultant atelectasis and respiratory infection. Reduced mobility can cause DVT.

* Nursing in a semi-reclined position helps to unload the diaphragm and improve ventilation and oxygenation.

* Catabolic response to surgery may require the use of insulin for maintenance of normoglycemia

* Low molecular weight heparin, stockings, and sequential compression devices as prophylaxis against DVT are important. Physiotherapy and early ambulation is helpful.

* The risk of wound infection is high. Meticulous attention to pressure points is essential.

* Patient is transferred to bed by Walter Henderson maneuver.

Illustration of the Walter Henderson maneuver. 1. Patient Transfer Device (PTD, also known as patient roller); 2. Patient tilted to slip roller under; 3. Roller slipped under patient; 4. Table tilted to roll patient downhill onto bed; 5. Patient rolled onto bed.

Regional anesthesia/analgesia

* Excessive somnolence and airway
obstruction is a risk with opioid analgesia. Regional blockade is a useful option and significantly reduces the risks, although it is technically challenging.

* Standard epidural needles can often be used, since the majority of obese patients have an epidural space less than 8 cm deep\(^2\). Obese patients have fatty infiltration of epidural space and increased blood volume secondary to increased abdominal pressure. This reduces the potential epidural space, leading to unpredictable spread of local anesthetic solution. Local anesthetic requirements for epidural and spinal are reduced up to 20-25% of normal in the morbidly obese.

* Epidural analgesia is very effective in obese patients though the maintainance of the epidural catheter has some practical problems like increased chance of displacement.

* Successful ultrasound-guided nerve blocks in obese patients have been reported.\(^3\)

**OBESO CHILD AND ANESTHESIA**

Obese children experience fewer medical complications than obese adults, although derangements of respiratory physiology are common across all age groups. Despite the relatively low prevalence of obesity-related comorbidity in children, they carry an increased likelihood of an anaesthetic critical incident, the risk rising with increasing BMI.

**BARIATRIC SURGERY AND ANESTHESIA**

Anesthesia for bariatric weight loss surgery poses several challenges for the anaesthesiologist. These are due to the problems of obesity itself, as well as to the co-morbid conditions often associated with obesity.

Certain medication used for obesity may interact with the anesthetic medications like MAO inhibitors, Serzone and herbal medicines. These drugs should be stopped at least 2 weeks prior to surgery. All the routine monitoring used in other surgeries in obese patients are used for bariatric surgery. All bariatric surgeries are performed under general anesthesia with endotrachial intubation. The endotracheal tube is placed either under topical anesthesia and intravenous sedation or after the induction of general surgery. Anesthesia is maintained during surgery by a combination of inhalation and intravenous agents. At the end of surgery the patient is allowed to awaken and breathe spontaneously. Endotracheal tube can be removed in operating room or left to be removed later when conditions are more optimal.

**OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS)**

Obstructive sleep apnea (OSAS)\(^4\) is characterized by recurrent episodes of upper airway collapse and obstruction during sleep. These episodes of obstruction are associated with recurrent oxyhemoglobin desaturations and arousals from sleep. In the perioperative
period the associated problems are difficult airway management during induction, hypertension, hypoxia, dysrhythmia, myocardial infarction, pulmonary edema, stroke & perioperative airway obstruction. These patients are more vulnerable to respiratory complications during intra operative period due to effect of positioning and drugs. nCPAP should be used in these patient in the post operative period.

REFERENCES:

1. Anaesthesia and morbid obesity
Sharmeem Lotia MBBS MRCP FRCA
Mark C Bellamy MBBS MA FRCA


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Premium Quality Anaesthetic Range

TROFENTYL-OTFC
Oral Transdermal Fentanyl Citrate 200 mcg

Troypofol
Propofol Injection 15 ml

ISOTROY
Isoflurane USP 100 / 250 ml

TROFENTYL
Fentanyl Citrate 50 mcg

TROYCURIUM
Atracurium inj. 2.5ml/5ml

BENZOSED
Midazolam HCl inj. 1 mg / 5 mg

Droperol
Droperidol inj. 2.5 mg/3ml.

BUPITROY
Bupivacaine HCl 0.25% 0.5% Heavy

PANURON
Pancuronium Bromide 1mg/ml

KETAMAX
Ketamine HCl Injection

MorphiTroy

+ Hospitroy
Quality First

Tronkin
Excellence built on basics

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INTRODUCTION:

The National Health & Nutritional Survey (NHANES-CYCLES I,II,III) has depicted that over past two decades in USA overweight & obesity has doubled among 6 -11 yrs & tripled among 12-17 yrs. According to WHO atleast 50% of adults & 20 % of children in USA & UK are overweight. In India standard data are not available. By school health survey in Chennai, Ramachandran et al. found overweight to be 22% & 4.5% in better off & lower income group school children respectively. Kapil U et al. detected overweight in 31 % & obesity in 7.5% among affluent school children in Delhi. Overall school based data depicts obesity in the range of 5.6% - 24% in children & adolescent in India. Nevertheless it is estimated that 80% of overweight adolescent become overweight adults.

SIGNIFICANCE AND PATHOPHYSIOLOGY:

Obesity in children becomes a chronic problem which tracks to adulthood and management is easier at childhood than adult life. Obese children have higher mortality (20 times) & morbidity than their peers. Pathophysiology of childhood obesity is poorly understood. Adipose tissue is the key player. Selective increase in size and number of fat cells and a cascade of secretion and regulation of neurohormones and chemical mediators with the chief action of leptin may explain the pathophysiology to some extent. Main mechanism of constitutional obesity is depicted in brief as follows:

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ETIOLOGY:

The etiology of human obesity is multi-factorial. There is complex interaction between genetic background, environmental stimuli & developmental process. The factors responsible are as follows:

A) Genetic & Familial:

- Poly genetic familial factors
- Mono genetic familial factors - Prader Willi, Laurence Moon Beidl, Alstrom, Cohen, Carpenter, Turner & Klinefelter syndromes.

B) Environmental:

- Increase calorie intake, Sedentary lifestyle, Decreased activity,
- Excess TV viewing, Video game & Computer use

C) Endocrine cause:

- Growth Hormone Deficiency, Hypothyroidism, Cushing syndrome,
- Hyper Insulinoma, Hyper androgenism

D) CNS cause:

- Post infectious (encephalitis, TBM), Trauma
- Tumor - Craniopharyngioma, Surgery/radiation

E) Miscellaneous:

- Psychiatric disturbances, Social & cultural pressure
- Steroids & anti epileptics, Leptin deficiency

CLINICAL EVALUATION:

Often obese children have obese parents and they might have decreased self esteem. Thus before interacting with them the physician should have a friendly, non-judgemental and sensitive approach to elicit detailed history of diet, life style and probe for other secondary causes of obesity. A simplified step-wise approach is as follows:

Step 1: Confirm the presence of obesity by BMI & skin fold thickness.

Step 2: Determine whether obesity is constitutional or pathological.

<table>
<thead>
<tr>
<th>FEATURES</th>
<th>Constitutional</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution</td>
<td>Generalised</td>
<td>Usually central</td>
</tr>
<tr>
<td>Ht. Age</td>
<td>Increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Bone Age</td>
<td>Advance</td>
<td>Retarded</td>
</tr>
<tr>
<td>Facial dysmorphism</td>
<td>Absent</td>
<td>May be present</td>
</tr>
<tr>
<td>Feature of endocrinopathy</td>
<td>Absent</td>
<td>May be present</td>
</tr>
</tbody>
</table>

Step 3: Meticulous History Taking

Birth wt, birth asphyxia, prematurity, Appetite, growth & development, Family history of obesity, hypertension, diabetes, hyperlipidemia, CAD, parent's BMI, race, mental retardation, H/o Child's diet and lifestyle, features of raised ICT, H/o CNS infection, trauma, surgery, radiation. Cushing syndrome/hypothyroidism/hypoglycemia, Family H/o DM-polyurea, polydypsia, nocturia, recent wt loss, H/o snoring, headache, waking up with headache, day time somnolence, sleep apnea
Step 4: Physical Examination

Dysmorphism, developmental assessment, sexual maturity assessment (SMR)-preoccocious/delayed puberty. Visual examination: retinitis pigmentosa - LMB, retinal degeneration - Carpenter syndrome, Alshtrum papilledema, pseudotumor cerebri, polydactyly - LMB, carpenter syndrome, blood pressure, Systolic or Diastolic BP > 90th percentile, stretched penile length/testicular volume, puberty status, hypogonadism, Prader Willi, Carpenter, LMB syndrome. Acanthosis nigricans - hyperinsulinism tonsillar hypertrophy - sleep apnea hip, knee, leg pain - orthopedics morbidity

Step 5: Investigations

FBS, PPBS, insulin, c-peptide, HbA1C, LFT, lipid profile, pulmonary function test, USG abdomen pelvis, X-ray hip, knee, chest free testosterone, LH, FSH, free cortisol, T4, TSH electrolytes, urea, creatinine, polysomnography, CSF exam, MRI, CT Scan

Step 6: Complications

Cardiovascular: hyperlipidemia, hypertension, premature CAD, Endocrine: insulin resistance, impaired glucose tolerance, frank DM type2, Syndrome X, hyperlipidemia, insulin resistance, obesity, ovarian dysfunction (polycystic ovary).
Respiratory: restrictive pulmonary disease, obstructive pulmonary disease; most imp is obesity, hypoventilation, Pickwickian syndrome, hepatic: fatty liver, cholelithiasis

Orthopaedics: slipped femoral epiphysis, flat feet, Blount disease (tibia vara), early onset osteo-arthritis.

Step 7: Management

Diet, activity & behavioural modification are cornerstones of obesity management. Intensive measure like drug therapy & surgery is reserved for morbid cases.

Diet: initial dietary measures for obesity includes mild calorie restriction (30-40%). Family dietary behaviour should be modified. Decreases consumption of junk food, carbonated drinks & sat fat. Increases consumption of dietary fibre, fruits & vegetables, less calorie densed fruit, food with low glycemic index like non starchy vegetables & whole grains.

Traffic light/stop light regimen

<table>
<thead>
<tr>
<th>Colour</th>
<th>Green light food</th>
<th>Yellow light food</th>
<th>Red light food</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quality</td>
<td>Low calorie, high fibre</td>
<td>Nutrient dense</td>
<td>High in calorie, sugar &amp; fat</td>
</tr>
<tr>
<td>Types of food</td>
<td>Fruits, vegetables</td>
<td>Lean meat, dairy products, starch, grains</td>
<td>Fatty meat, sugar &amp; fried food</td>
</tr>
<tr>
<td>Quantity</td>
<td>Unlimited</td>
<td>Limited</td>
<td>Infrequent/avoid</td>
</tr>
</tbody>
</table>

Weight maintenance rather than wt loss is essential for growth in children. Weight loss should be attempted in skeletally matured children & those with severe obesity & in this case also wt reduction is slow @ 0.5kg/week is ideal.

An initial 10% wt reduction is reasonable, after that the new wt should be maintained for 6 month, before further reduction.

A protein sparing modified fast
(PSMF) regimen in which there is severe calorie restriction (800kcal/day) is useful in severe obesity.

PSMF regimen with fortified micronutrients & vitamins leads to decrease body wt by 0.5kg/wk.

**Lifestyle Modifications**: Increase in physical activity with reduction in sedentary lifestyle is an important part of obesity management. For children atleast 1 hour exercise with sweating daily is recommended. Swimming, running, playing outdoor games to be encouraged. TV viewing, video games, computers which promote snacks eating should be restricted. Less than 2yrs avoid TV viewing & computers, 2-18 yrs max 2 hrs/day is allowed. TV should be removed from bedroom. Psychological & behavioural therapy is useful.

**Drugs**: Value of drug in treatment of children obesity is minimal & under experiment, however these are useful in severe obesity cases.

Sibutramine: Decreases appetite through Neuro transmitter.

Orlistat: Decreases absorption by inhibiting gastric lipase.

Octreotide: useful in childhood hypothalamic obesity.

**Surgery**: are indicated only in Morbid obesity (BMI > 40) & BMI >35 with complication.

Most acceptable permanent surgery is Roux en y gastric bypass & adjustable gastric banding.

**REFERENCES**:  
INFERIOR MEATAL ANTROSTOMY VS ENDOSCOPIC MIDDLE MEATAL ANTROSTOMY IN CHRONIC MAXILLARY SINUSITIS.

C. S. Ray, S. K. Behera

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ABSTRACT:

Fifty cases of either sex in the age group of 15-45 years suffering from chronic maxillary sinusitis as confirmed on CT scan paranasal sinus were selected for this study. In 25 cases, middle meatal antrostomy while in remaining 25, inferior meatal antrostomy was done. Endoscopic middle meatal antrostomy had better result than inferior meatal antrostomy. Patency rate after middle meatal antrostomy was 91.67% while that of inferior meatal antrostomy was 76% after 6 weeks.

Key Words: sinusitis, antrostomy.

INTRODUCTION:

Various treatment modalities available for chronic maxillary sinusitis are medical and surgical. Medical management includes antibiotics, mucolytic agents, decongestants and antihistaminics for appropriate duration. While surgical treatment involves: (i) Antral lavage, (ii) intranasal antrostomy via middle meatus or inferior meatus, and (iii) Caldwell Luc's operation. In tranasal inferior meatal antrostomy is based on the concept of dependent drainage which is changing with advent of nasal endoscopes. Middle meatal antrostomy, is based on the principles that mucociliary drainage of sinus in normal or pathological states is always towards the natural ostium only, even in the presence of inferior meatal antrostomy. Thus one should fashion on antrostomy in or near the natural opening. It not only provides ventilation and drainage but also restores the normal sinus mucosa, thereby preserving physiological ciliary activity. Spontaneous closure of the middle meatal antrostomy is less common though, technically it is more difficult to perform than inferior meatal antrostomy.

MATERIAL AND METHODS:

Fifty cases of either sex in the age group 15-45 years suffering from chronic maxillary sinusitis as confirmed on CT scan paranasal sinus were selected for this study. the patients were divided into 2 groups of 25 each:
Group I underwent unilateral or bilateral classical inferior meatal antrostomy.

Group II underwent unilateral or bilateral endoscopic middle meatal antrostomy using 4mm sinus endoscope and traditional endoscopic instruments by Karl Storz.

RESULTS AND DISCUSSION:

In our study, improvement in various symptoms was recorded after 6 weeks (Table 1). In Group I, improvement in headache was noted in 75%, postnasal drip in 73.91%, anterior nasal discharge in 75% and nasal obstruction in 86.67% of patients. Improvement in anterior nasal discharge occurred early while headache, postnasal drip and nasal obstruction improved with time. This could be because of edema and formation of crusts for few days post operatively which cause nasal obstruction and retention of secretions. Lund performed inferior meatal antrostomy of various sizes in their prospective study of 65 patients. Nasal obstruction got relieved in 75%, postnasal drip in 34%, rhinorrhea in 60%, facial pain in 49% and headache in 47% of her patients. Postnasal drip improved less dramatically.

In Group II in our study after 6 weeks, improvement of postnasal drip was noted in 84.21%, anterior nasal discharge in 85.71%, nasal obstruction in 93.75% patients and headache in 90%. Improvement in headache occurred early while improvement in postnasal drip, anterior nasal discharge and nasal obstruction took some time. Maximum improvement was noted in nasal obstruction (93.75%) and headache (90%). Salam and Cable performed 90 middle meatal antrostomies in 51 patients. In their series, 63% patients got cured of headache, 32% had improvement and only 5% reported no relief. Forty-seven percent got cured of nasal obstruction, 42% had improvement and 7% reported no relief. Thirty-three percent got cured of hyposmia, 50% had improvement and 17% got no relief. Subjective improvement was significant and maintained at follow-up of an average 26 months. This is different to the study carried out by Ames, et al. in which they showed that symptomatic improvement in both procedures was identical. However, in their study classical intranasal inferior meatal antrostomy was done on one side and endoscopic middle meatal antrostomy on the other side in the same patient.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Total no. of patients</th>
<th>Total improvement in Group I</th>
<th>Total no. of patients</th>
<th>Total improvement in Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>20</td>
<td>15</td>
<td>75</td>
<td>20</td>
</tr>
<tr>
<td>Postranal drip</td>
<td>23</td>
<td>17</td>
<td>73.91</td>
<td>19</td>
</tr>
<tr>
<td>Anterior nasal drip</td>
<td>12</td>
<td>9</td>
<td>75</td>
<td>14</td>
</tr>
<tr>
<td>Nasal obstruction</td>
<td>15</td>
<td>13</td>
<td>86.67</td>
<td>16</td>
</tr>
</tbody>
</table>

The patency of all 98 antrostomies was checked after 6 weeks. Out of 50 inferior meatal antrostomies in Group I, 38 (76%) were found patent on endoscopic
examination. Out of 48 middle meatal antrostomies in Group II, 44 (91.67%) were found patent after 6 weeks on endoscopic examination.

Lund\(^4\) in a retrospective study found that after initial circumferential healing within first within first few weeks, majority of antrostomies remained unchanged. It was concluded that an antrostomy of about 1.1 cm size was adequate to prevent spontaneous closure without jeopardizing related anatomy. Kamal\(^5\) carried out 94 endoscopic middle meatal antrostomies in 66 cases of chronic maxillary sinusitis. After follow up of 4-12 months endoscopically, 96.8% antrostomies were found patent. Better results observed in middle meatal antrostomy could be because of the fact that in case of middle meatal antrostomy, uncinate is done and natural ostium of maxillary sinus is widened, thereby leading to widening of osteomeatal complex and natural ostium. In our study, no major complication was noted in both the groups. Postoperative bleeding was noted in 3 (12%) patients of Group I and 1 (4%) of Group II. Also synchiae formed in 6 (12%) patients of Group I and 3 (6.25%) of Group II, which were managed successfully.

SUMMARY AND CONCLUSIONS:

Intranasal antrostomy (inferior meatal and middle meatal) is a surgical procedure for treatment of maxillary sinusitis. However, the concept of dependent drainage has changed with the use of nasal endoscopes. The mucociliary clearance of sinuses is always towards the natural ostium. Even in presence of patent inferior meatal antrostomy the tends to beat towards the natural ostium.

In our study not only the patency rate but also the improvement in symptoms was higher in middle meatal antrostomy. Thus, endoscopic middle meatal antrostomy should be accepted as the modality of treatment for chronic maxillary sinusitis.

REFERENCES:

1. Eichel B.S. Surgical management of chronic paranasal sinusitis. Laryngoscope 1973;83; 1195-1203.


POSTOPERATIVE ANALGESIA BY DEXAMETHASONE AS AN ADJUVANT TO MIXTURE OF LOCAL ANESTHETICS IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

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ABSTRACT:

Background: Local anesthetics alone for supraclavicular brachial plexus block though provide good operative condition but fails to produce adequate post operative analgesia due to shorter duration of action. Various adjuvants have been tried to prolong the duration of action of local anesthetics. Perineural injection of steroids is found to influence the post operative analgesia.

Patients and Methods: 60 adult patients undergoing various orthopedic surgeries of upper limb below shoulder joints were selected and randomly divided into 2 groups of 30 each. In group A (control) patients received 35 ml of a mixture of lignocaine 2% with adrenaline and bupivacaine 0.5% whereas in group B (dexamethasone) patients received 35 ml of same local anesthetic mixture along with 4mg of dexamethasone in it. The onset of action, peak effect and duration of analgesia in the 2 groups were compared and any complications were noted and treated accordingly. Statistical analysis was done using student’s unpaired t test.

Results: The demographic profiles of the 2 groups were comparable. The mean onset time of sensory and motor block was 280.55 ± 34.23 sec and 336.25 ± 28.63 sec in control group (group A) and 186.24 ± 24.54 sec and 218.78 ± 24.65 sec in dexamethasone group (group B) respectively. The mean peak effect time of sensory and motor block was 716.32 ± 52.54 sec and 772.53 ± 45.32 sec in group A and 540.76 ± 48.35 sec and 645.43 ± 34.84 sec in group B respectively. The difference between the 2 groups was statistically significant (p < 0.05). There was also markedly prolongation of duration of analgesia in group B (14 ± 2) hour compared to group A (5.5 ± 0.5) hour. The results between 2 groups was statistically highly significant (p < 0.001).

Conclusion: The addition of dexamethasone (4mg) as an adjuvant to mixture of local anesthetics in supraclavicular brachial plexus block results in significantly early onset & peak effect of sensory and motor blockade along with markedly prolonged duration of post operative analgesia without any significant side effect.

Key Words: Supraclavicular brachial plexus block, Analgesia, Local Anesthetics, Dexamethasone

INTRODUCTION:

Brachial plexus block is an easy & relatively safe procedure for upper limb surgeries. Though there are different approaches to it, supraclavicular approach is the most consistent & widely used method for surgical anesthesia and perioperative pain management in surgery below shoulder joint.
The provision of good quality of analgesia during intra-operative & post operative period is important not only to ease patients suffering and induce sense of wellbeing but also improve the ability to ambulate early and thereby reducing the post operative complications. But local anesthetics commonly used like lignocaine & bupivacaine provide analgesia for only 4-8 hours. So various adjuvants are tried till date like opioids¹, benzodiazepines, neostigmine², clonidine³, dexametomidine etc. to prolong the duration of local anesthetics. But the results are either inconclusive or associated with side effects.

Steroids have powerful anti-inflammatory as well as analgesic properties. Perineural injection of steroids is reported to influence post operative analgesia. They relieve pain by reducing the inflammation & blocking transmission of nociceptive c - fibres and by suppressing ectopic neural discharge.⁵

With this background data this study was carried out to evaluate the efficacy of dexamethasone as an adjuvant to mixtures of local anesthetics in supraclavicular brachial plexus block.

PATIENTS & METHODS

A prospective randomized double blind controlled study was carried out after obtaining institutional ethical committee approval & patients informed written consent among 60 ASA class I & II patients of either sex, aged between 20 - 60 years scheduled for elective or emergency orthopedic surgeries of upper limb below shoulder joint under supraclavicular brachial plexus block. Patients with history of peptic ulcer disease, uncontrolled diabetes mellitus, hepatorenal diseases, circulatory instability, pregnant women, those allergic to local anesthetics & on long term steroid therapy were excluded from the study.

Patients were randomly allocated into 2 groups of 30 each. In Group A (control) patients received 15 ml of lignocaine 2% with adrenaline (1: 200000) & 15 ml of bupivacaine 0.5%, while in Group B (dexamethasone) patients received 1 ml of injection dexamethasone (4 mg) in addition to above mixture. The total volume was made to 35 ml by adding normal saline in both the groups.

On arrival to the operating room standard monitoring was established (Pulse oximetry, ECG, NIBP) & the initial pulse rate, blood pressure, respiratory rate, SpO₂ were recorded as preblock values. An i.v line was secured with 18 G cannula in the peripheral vein of contralateral arm.

After appropriate patient positioning & aseptic precautions midclavicular point, external jugular vein & subclavian artery pulsation were identified. About 2 cm above midclavicular point just lateral to subclavian artery pulsation a 24 G 1.5 inch needle was introduced & directed caudally and medially until paresthesia was encountered. Then 35 ml of local anesthetic mixture with or without dexamethasone was injected in this area.
with repeated negative pressure aspiration after every 5-6 ml to avoid intravascular injection.

Sensory & motor blockade of radial, median, musculocutaneous and ulnar nerve were recorded at regular intervals (0, 2, 4, 6, 8, 10, 12, 15 & 20 minutes) after drug injection. Sensory blockade of each nerve was assessed by pinprick and compared with the same stimulation on the contralateral hand. Sensory blockade was graded as:- Grade 0 - No sensation felt, Grade 1 - Dull sensation felt, Grade 2 - Sharp pain felt

Onset time is defined as the time from injection of brachial plexus block to feeling of dull sensation along any of the nerve distribution.

Peak effect time is defined as the time from injection of brachial plexus block to complete loss of sensation along all the nerve distribution.

Motor blockade was evaluated by thumb abduction (radial nerve), thumb adduction (ulnar nerve), flexion of elbow (musculocutaneous nerve) and thumb apposition (median nerve). The motor blockade was graded as:- Grade 0 - Complete paralysis, Grade 1 - Paresis, Grade 2 - Normal muscle force

Onset time is defined as the time from injection of brachial plexus block to the time of feeling of heaviness on abduction of arm at shoulder.

Peak effect time is defined as the time from injection of brachial plexus block to the time when patient is unable to perform any of the maneuvers as defined above for each individual nerve.

Block was considered incomplete when any of the segments supplied by the nerves did not have analgesia even after 20 minutes of drug injection and failure when more than one nerve remains unaffected. Injection Midazolam 1mg was given i.v after 20 minutes when the assessment of block was complete. In failed block general anesthesia was given. Intra operatively patients were monitored for vital parameters like pulse rate, blood pressure, respiratory rate, and SpO₂ at regular intervals. Patients were observed for any side effects in intra and post operative period and treated accordingly. Post operative vital parameters were monitored every 3 hourly and analgesia was assessed using visual analogue scale.

Duration of analgesia is defined as the time from injection of brachial plexus block to a VAS score of 0-4 when rescue analgesia in form of injection Diclofenac Sodium was given.

Statistical analysis was performed in SPSS 17 programme. The data was analyzed with chi square test for qualitative data & student's unpaired 't' test for quantitative data. The results were considered significant if p value < 0.05 and highly significant if p value < 0.001.

RESULTS

The demographic data and duration of surgery were comparable in both the
groups (Table - 1).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
<th>Group B</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>30</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Age (years) Mean ±SD</td>
<td>34.56 ± 6.5</td>
<td>32.33 ± 7.93</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>20:10</td>
<td>18:12</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Weight (Kgs)</td>
<td>56.35 ± 6.05</td>
<td>56.05 ± 6.43</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Height(cm)(Mean±SD)</td>
<td>158.52 ± 5.42</td>
<td>159.24 ± 5.34</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>ASA I</td>
<td>18 (60%)</td>
<td>19 (64%)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>ASA II</td>
<td>12 (40%)</td>
<td>11 (36%)</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

The mean onset time of sensory & motor blockade was 280.55 ± 34.23 seconds and 336.25 ± 28.63 seconds in group A & 186.24 ± 24.54 seconds and 218.78 ± 24.65 seconds in group B respectively. The mean peak effect time of sensory & motor blockade was 716.32 ± 52.54 seconds and 772.53 ± 45.32 in group A & 540.76 ± 48.35 seconds and 645.43 ± 34.84 seconds in group B respectively. The difference was statistically significant with p value < 0.05. (Table 2) There was no change in vital parameters in both the groups on intra or intergroup comparison.

The mean VAS score (Table 3) was 5.8 ± 0.5 hours at the end of 6 hours in group A and 5.2 ± 1.4 at the end of 14 hours in group B. The difference was statistically highly significant with p value < 0.001. Thus in group B, patients had significantly longer duration of analgesia of 12-16 hours whereas in group A, patients had analgesia of only 5-6 hours. There was no incidence of failed block in this study. Except for nausea & vomiting in 1 patient in both the groups, no other significant complications were observed during our study.

<table>
<thead>
<tr>
<th>Onset time (Mean ± SD)</th>
<th>Group A</th>
<th>Group B</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory</td>
<td>280.55 ± 34.23</td>
<td>186.24 ± 24.54</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Motor</td>
<td>336.25 ± 28.63</td>
<td>218.78 ± 24.65</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Peak effect time (Mean ± SD)</th>
<th>Group A</th>
<th>Group B</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory</td>
<td>716.32 ± 52.54</td>
<td>540.76 ± 48.35</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Motor</td>
<td>772.53 ± 45.32</td>
<td>645.43 ± 34.84</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time in hours when VAS &gt; 4 (Mean ± SD)</th>
<th>Group A</th>
<th>Group B</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5 ± 0.5</td>
<td>14 ± 2</td>
<td>&lt; 0.001</td>
<td></td>
</tr>
</tbody>
</table>

DISCUSSION

Steroids are one of the adjuvants to local anesthetics for prolongation of duration of analgesia. Steroids are very potent anti-inflammatory & immunosuppressive agents. Perineural injection of steroid is reported to influence post operative analgesia. Epidural steroids were used for treatment of backache & sciatica. Various steroids has been used for this purpose but dexamethasone, a 9 α - derivative synthetic glucocorticoid is preferred due to its highly potent anti-inflammatory property without any mineralocorticoid activity thus devoid of potential side effects.

Corticosteroids are capable of reducing prostaglandin synthesis by inhibiting phospholipase A2 through production of calcium dependent phospholipid binding protein called annexins & by the inhibition of cyclooxygenase - 2 during inflammation. Preoperative administration of dexamethasone by oral & i.v routes has been shown to reduce overall pain scores
& analgesic requirements in the post operative period without any adverse effect in various general surgical procedures. The possible mechanism of analgesic action are due to anti-inflammatory property of dexamethasone.6,7

The exact mechanism of action of dexamethasone in prolonging the duration of analgesic action of local anesthetics is not clearly understood. But it may be due to its local action on nerves rather than systemic action as believed by some authors. The question of whether these results were attributable to local or systemic effects warrants further investigations. But when steroids alone were used in regional blocks the blockade is not produced corroborating to its local action on nerve fibres. The local effects on nerve fibres might be mediated via glucocorticoid receptors. Steroid might bring about this effect by altering the function of potassium channels in excitable cells.8

It has been demonstrated that addition of dexamethasone to bupivacaine microcapsules prolonged the duration of blockade of peripheral nerves.9,10 Few earlier studies already reported the adjuvant action of dexamethasone in prolonging the duration of analgesia of local anesthetics. A study on axillary plexus block suggest that dexamethasone when added to lignocaine significantly prolonged the duration of analgesia without any change in onset of action.11 Another study in supraclavicular block reported that dexamethasone when added as adjuvant to mixture of local anesthetics resulted in significantly early onset & longer duration of analgesia.12 In another study by Golwala M P etal, they noticed significantly early onset of sensory and motor block & prolonged duration of analgesia with addition of 8 mg of dexamethasone to a mixture of lignocaine with adrenaline and bupivacaine.13

In our study we noticed significantly earlier onset of sensory & motor block in group B patients with 4mg dexamethasone added to local anesthetic mixture in comparison to the control group A. The early onset of action might be due to synergistic action with local anesthetics on blockade of nerve fibres. The duration of pain relief was markedly prolonged in group B up to about 12 -16 hours while it was only upto 5 - 6 hours in group A. Our results are comparable to study reports of Golwala M P etal, who reported onset of action of 245 - 305 seconds for local anesthetic group & 170 - 222 seconds for group with local anesthetic & dexamethasone. The duration of analgesia was 4 - 6 hours in local anesthetic group while it was about 12 -18 hours in local anesthetic with dexamethasone group of patients. The less duration of analgesia in our study than that of Golwala M P etal may be due to smaller dose of dexamethasone. But we have used the smaller dose of dexamethasone (4mg) as a safety precaution. In all previous studies dexamethasone was used safely in a dose of 4 - 8 mg without any adverse effects. But the exact dose of dexamethasone to be used in peripheral nerve block has not been described yet,
which requires large numbers of study to optimize the dose of dexamethasone as an adjuvant to local anesthetics.

CONCLUSION

In conclusion, the addition of dexamethasone (4 mg) as an adjuvant to mixture of local anesthetics in supraclavicular brachial plexus block results in significantly early onset & peak effect of sensory and motor blockade along with markedly prolonged duration of post operative analgesia. But further studies are needed to evaluate the optimal dose of dexamethasone to be used in brachial plexus block as well as the mechanism of its prolonged analgesic effect.

REFERENCES


Case Report

PILOMATRIXOMA OF PAROTID REGION

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ABSTRACT:

Pilomatrixoma is a benign adnexal tumor with differentiation toward hair cells. It usually manifests as a solitary, asymptomatic, firm nodule. It has long been considered a rare tumor, but it may be more common than previously realized. It is more common in children, but occurrence in adults is increasingly being recognized. Recommended treatment is surgical excision. Pilomatrix carcinoma is a rare condition.

Key Words: Pilomatrixoma, Parotid gland

INTRODUCTION:

Originally described as a calcifying epithelioma by Milharbe and Chelantis in their original description in 1880, it is a benign neoplasm of hair follicle matrix. Affected individuals are mainly in the age group of 8 to 13 years and more prevalence is found in females. It is firm, solitary, slow growing and painless lesion of dermis, which most of the times extend to subcutaneous tissue. It often develops into capsulated form, size ranging from 0.5 to 5 cm.

This is the case report of Pilomatrixoma of left parotid region in a 13 years old female, who presented with a mass in the parotid region. Fine needle aspiration cytology and radiological investigations confirmed the diagnosis of Pilomatrixoma. Complete excision of the tumor was done through superficial parotidectomy incision. Since the overlying skin was also involved, it was also excised in continuity and facial reconstruction was done by microvascular anastomosis using free flap from thigh.

CASE HISTORY:

A 13 year old female presented to us with a mass in the left parotid region since 2 months. It started as a tiny, painless swelling below the angle of the jaw 8 months ago. Since then the nodule enlarged progressively to involve parotid region with the overlying skin. Pt. didn't have any history suggestive of sialolithiasis or chronic granulomatous disease.
On examination, there was a multinodular mass, measuring 5cm X 5cm, in the left parotid area, extending into left infra auricular region pushing left ear lobe upwards. Overlying skin was bluish in color. It was firm in consistency, mobile, nontender. There were no palpable nodes in the neck.

CT scan was suggestive of tumor of superficial lobe of parotid gland with extension into the subcutaneous tissue. The diagnosis was confirmed with fine needle aspiration cytology, which was suggestive of Pilomatrixoma.

Complete excision of the tumor was done through superficial parotidectomy incision. Since the overlying skin was also involved, it was also excised in continuity with the tumor mass. Facial tissue defect was corrected with free microvascular flap from thigh, which was done by the plastic surgeons.

Histopathology of the mass confirmed the diagnosis as Pilomatrixoma.

**DISCUSSION:**

Pilomatrixoma most commonly occurs in head and neck region; between 56 and 72% of all cases appear in this area especially the cheek, preauricular area, eyelids, forehead, scalp and lateral and posterior neck. The next most common site is the upper extremities. Together, these two sites host the vast majority of these tumors.

Most reported cases have occurred in white persons. Most studies report a slight preponderance in females. In one retrospective study of 209 cases, the female-to-male ratio was 1.5:1. Most reported cases have occurred in children. Lesions are often discovered in the first 2 years of life; however, in a recent 1998 retrospective study of 209 cases, investigators found the age of presentation having a bimodal pattern, with the first peak being 5-15 years and the second being 50-65 years.

Most lesions measure 0.5-3 cm,
but, rarely, giant lesions up to 15 cm are reported. Patients usually have a single, firm, stony, hard nodule. Lesions are usually of the color of the normal skin, but reddish-purple lesions have been observed (probably resulting from hemorrhage). Stretching of the overlying skin can give the lesion a multifaceted, angulated appearance known as the "tent sign," likely due to calcification in the lesion.

It is difficult to diagnose Pilomatrixoma, as differential diagnoses include dermoid and inclusion cysts, preauricular sinuses, hemangiomas and malignant soft-tissue tumors. But presence of such a nodule on the head, neck or upper extremity, especially in a younger patient, should raise the clinician's suspicion of Pilomatrixoma.

Diagnosis is always confirmed with histopathological examination. Fine-needle aspiration of these masses can yield cytologic results, that are consistent with Pilomatrixoma. It is rare to have malignant Pilomatrixoma, but some cases have been reported. This exceedingly rare malignant variant of Pilomatrixoma is histologically characterized by prominent nucleoli, focal areas of necrosis and multiple mitotic figures. The lesion is usually found in the lower dermis and subcutaneous fat. It is sharply demarcated and is usually surrounded by a connective tissue capsule. Irregularly shaped islands of epithelial cells are seen; they can be recognized as either basophilic cells or shadow cells. Basophilic cells are usually arranged either on one side or along the periphery of the tumor islands. The shadow cells have a central unstained area, corresponding to the lost nucleus. As the lesion ages, the number of basophilic cells decreases. Calcium deposits are seen in 75% of lesions with von Kossa staining. Evidence of vascular invasion and perineural or perichondrial infiltration support a diagnosis of a more aggressive type of Pilomatrixoma that has the potential for malignant degeneration.

Various imaging methods for evaluating Pilomatrixoma have been reported. Plain radiographs of suspicious lesions have limited utility, but they can detect foci of calcification. Preoperative CT scan was done in our patient, which was suggestive of Pilomatrixoma. 4Computed tomography or magnetic resonance imaging might be considered for those patients, who have larger or more unusual tumors. Ultrasonography has been described as a relatively fast and noninvasive investigative technique for estimating the depth of larger masses. The clinically superficial location of most of these tumors makes routine radiographic evaluation unnecessary. An accurate histopathology-logic evaluation is the most important tool for confirming the diagnosis.

Treatment consists of surgical excision.5 Recurrences are rare, but
when they do occur, the physician should suspect a malignant Pilomatrixoma variant. The surgical approach can be modified depending on the site of involvement, to ensure the total removal of the lesion without injury to vital neural or vascular structures.

In our case modified Blair incision was used to remove the preauricular tumor. The preoperative assessments were consistent with small, superficial neoplasms, that were easily removed and did not require facial nerve dissection. Complete surgical excision of the tumor is the recommended treatment. In our case, as it was large mass, we have to sacrifice the skin over the mass, which resulted into skin and tissue defect over face. It was corrected by taking free microvascular flap from thigh, which was done by plastic surgeons.

REFERENCES:
SQUAMOUS CELL CARCINOMA OF SCROTUM OVER LONG STANDING ELEPHANTIASIS NODULE

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ABSTRACT:

Lymphatic Filariasis is caused by Wuchereria bancrofti and Brugia malayi. It is most commonly seen in tropics and subtropics. Even in tropical countries it is unusual to find microfilaria in routine cytological smears. Filariasis of the scrotum is a clinical entity wherein evolution of various pathological stages occurs over a span of 20 to 30 years. A stage of inflammation is followed by a stage of lymphedema and finally a stage of nodule formation or elephantiasis occurs. To the best of our knowledge squamous cell carcinoma over long standing elephantiasis nodule is not reported in previous literature. Here we report a case of squamous cell carcinoma over a long standing elephantiasis nodule of 40 years.

Key Words: Filariasis, Elephantiasis nodule, Squamous cell carcinoma

INTRODUCTION:

Humans are the only definitive hosts for the parasite Wuchereria Bancrofti and Brugia malayi, which are the causative agents of lymphatic filariasis.¹ This disease is most commonly found in Asia, Africa, South America and Caribbean basin.² Single or small number of case reports have described detection of microfilaria in smears such as bone marrow, breast, thyroid, bronchial aspirate, cervicovaginal smears, pleuropericardial and hydrocele fluid.²³ Most of the infections encountered in India are by Wuchereria bancrofti.⁴ Long term infection in the lower limb and scrotum usually leads to elephantiasis with multiple sister nodules. No previous literature has reported malignant transformation in such nodules. Here we are presenting a rare case of malignant transformation in a long standing filariasis nodule of the scrotum.

CASE HISTORY:

A 70 year old male presented with a non healing ulcer over a nodule of preexisting elephantiasis of the scrotum for the last one year. The ulcer started as a small sore of about 0.5 cm X 0.5 cm over an elephantiasis nodule of the scrotum one year back. Inspite of some
local medication, the ulcer refused to heal and produced foul smelling discharge which soiled the undergarments and gradually increased to 2X 2 cm over a period of 1 year. He was a known case of filariasis of the scrotum for the last 40 years which went through the stages of inflammation, lymphedema and nodule formation over the scrotum and both the lower limbs. During the last 30 years, the nodules grew in size and branched into sister nodules. He was not a known case of diabetes mellitus and history did not reveal any chemical application to the scrotum. He was then referred to Regional Cancer Centre for further treatment. Scrape cytology from the scrotum revealed squamous cells with features of malignancy. USG of the scrotum showed bilateral atrophic testes with thick degenerated fluids in both the sacs of tunica vaginalis of the testes. As it was long standing elephantiasis of the scrotum with bilateral atrophic testes, a total scrotectomy was done with bilateral orhidectomy with valid consent of the patient. The postoperative histopathology of the filariasis nodule showed hyper chromatic pleomorphic neoplastic squamous epithelial cells infiltrating the sub epithelial stroma with formation of keratin pearls at places. Marked fibroblastic proliferation was seen in the stroma. Dense inflammatory cell infiltration rich in eosinophils and lymphocytes was a prominent feature. The final histopathology opinion was well differentiated squamous cell carcinoma with evidence of squamous carcinoma cells amidst chronic granulomatous inflammatory cells.

**DISCUSSION:**

Squamous cell carcinoma arising in chronic lymphedema is rare; only 9 cases have been reported arising from the leg but not a single case has reported malignant transformation in an elephantiasis nodule of the scrotum. Furukawa et al. described the evolution of squamous cell carcinoma in chronic lymphedema. They also strongly
suggested that lymphedema itself is one of the carcinogenic factors for both angiosarcoma and squamous cell carcinoma. Epstein et al. reported a case of congenital lymphedema who developed squamous cell carcinoma on his affected foot. He suggested the role of viral oncogenes and ulceration as a promoter for malignancy. Lister et al. reported 2 patients of chronic lymphedema of the lower limbs who developed squamous cell carcinoma. Fergusson et al. reported a case of metastatic malignant fibrous histiocytoma originating in the lower limbs of a patient with Milroy’s disease.

CONCLUSION:

Elephantiasis of the scrotum is a very common manifestation of filariasis and is endemic in eastern part of India. Lymphatic obstruction and subsequent fibrosis in the lymph logged tissue causes this condition. The disease evolves through stages of filarial fever, lymphangitis, lymph stagnation and finally elephantiasis. The lymph logged tissue provides suitable nutrition for growth of fibroblasts. Thickening occurs at the most dependent part of the scrotum and gradually extends upwards. The testes usually become atrophic due to lack of nutrition and pressure in long standing cases. A malignant change over such a nodule has not been previously reported. This will probably be the first reported case of elephantiasis of the scrotum turning into malignancy after a period of 40 years.

REFERENCES:

Case Report

A CASE OF FOETUS PAPYRACEUS

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ABSTRACT:

Foetus papyraceus is a rare occurrence and both fetuses presenting at the os during labour is a still rarer phenomena. We present the case of a twenty year old primigravida who presented with the diagnosis of pregnancy at 38 weeks of gestation in labour. She was diagnosed as a case of compound presentation and emergency LSCS was undertaken only to disprove that it was a case of normal foetus and a foetus papyraceous presenting simultaneously.

Key words: foetus papyraceous, compound presentation

INTRODUCTION:

Foetus papyraceous is a variant of a vanishing twin. Vanishing twin is a fetus in a multigestation pregnancy which dies in utero and is then partially or completely reabsorbed by the mother or twin.¹ It is also otherwise known as twin embolisation syndrome or vanishing twin syndrome (VTS) since the 1980s when twin pregnancies were made visible by ultrasonography. Occasionally the dead foetus will be compressed by its growing twin to a flattened parchment like status, that resembles a scrap of parchment paper. Hence the name foetus papyraceous.²

mummification of the fetal parts, will cause a fetus papyraceous (Potter, 1962).³

The cause of such foetal demise may be a poorly implanted placenta, developmental anomaly, tight cord round the neck, etc. Death usually occurs during the second trimester and the crown rump length is a good guide to the stage of pregnancy when death occurred, but obviously unless sufficient time has elapsed for mummification, the dead fetus will be delivered as a macerated stillbirth rather than a fetus papyraceus. Therefore delivery of a fetus papyraceus indicates that death must have occurred at least 10 weeks prior to delivery. (Saier et al., 1975).⁴

They usually emerge during the process of labour and do not pose a risk to the living baby.

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CASE REPORT:

Mrs. S. J. an unbooked primigravida, 20 years old, at term attended the labour room with complaints of pain abdomen and leaking for two days. On examination her general condition was good, vitals stable and heart lungs NAD. She was mildly pale. On per abdomen examination uterus was of term size with cephalic presentation and mild contractions. The fetal heart rate was 112/minute, regular. On per vaginal examination cervix was fully effaced and os 3 cm dilated. Presenting part was vertex along with another soft part beside the vertex which was of wax like consistency. There was thick meconium stained liquor with small caput. She was posted for an emergency LSCS with the presumptive diagnosis of foetal distress with compound presentation.

At caesarean section a healthy living male baby weighing 3.5 kg with Apgar score 6/10 was delivered followed by a shrunken wax like foetus which occupied the lower pole completely. This was followed by delivery of placenta which clearly showed a small contracted portion attached by a thin umbilical cord to the foetus papyraceous. The healthy portion of the placenta with the normal cord was found attached to the healthy foetus. The foetus papyraceous was 35 cm long and weighed 510 gms. It was antero posteriorly compressed, yellowish with wax like consistency. Membrane was adherent to the foetus. The patient had a normal recovery and mother and baby were discharged on the 7th post operative day.

DISCUSSION:

Foetus papyraceous is a very rare occurrence with a frequency of 1 in 12,000 live births & 1 in 184 -200 twin pregnancies.\(^5\)

In a study by E Daw, four of the 11 cases were established as monozygotic and one as dizygotic, with the zygosity in doubt in remaining cases which suggests monozygotic multiple pregnancies may be prone to fetus papyraceus formation. Fetus papyraceus with congenital anomalies in the 2nd twin is rare\(^6\) though congenital intestinal atresia, gastroschisis, aplasia cutis, congenital and cardiac anomalies have been reported. One of the 11 cases had a cardiac anomaly in the survivor similar to the case of Baker and Doering. Antepartum diagnosis of fetus papyraceus is infrequent and usually it is a chance finding during investigation of some other pregnancy problem. More recently an elevated alpha feto protein in amniotic fluid along with a positive acetyl choline esterase band in the absence of sonographic evidence of neural tube defect in the fetus is highly suspicious of a vanishing twin.\(^7\)

The following clinical signs are, however, very suggestive:

(i) Rapid enlargement between 12 and 24 weeks gestation, followed by a normal or slowed growth period and

(ii) The sudden appearance or subsidence of toxaemia of pregnancy.

(iii) Unexplained bouts of vaginal bleeding and
Amniotic fluid leakage which suddenly ceases. 4

Maternal health is rarely affected, polyhydramnios and toxaemia of pregnancy may suddenly disappear, though complications have been reported—premature labour, obstructed labour necessitating Caesarean section, infection and postpartum haemorrhage (due to retention of the fetus papyraceus). Consumptive coagulopathy following fetal death in a triplet pregnancy has been reported. 8

Velamentous insertion of the cord is increased in twin pregnancy, but rare in fetus papyraceus. 4 Four of the 11 cases studied by E.Daw had a velamentous insertion of the cord and another fetus papyraceus had the cord tightly around its neck. The study suggests that cord complications may, in fact, increase the chances of intrauterine death and formation of a fetus papyraceus.

There has been no correlation to maternal age, parity or gravidity.

REFERENCE:
6. Azubike JC. Multiple births in Igbo women, Br J Obstet Gynaecol 1982; 89: 77-79
CASE REPORT OF MULLERIAN ANOMALIES A VARIANT OF UTERUS DIDEPhYS

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Consultant Gynaecologist
Mumbai

ABSTRACT:

The case report is of a patient who initially presented as a case of primary infertility and upon work up and investigation was found to be a case of Double Uterus or Uterus DidePhys. Uterus DidePhys or Double Uterus results from failure of the midline fusion of the two mullerian ducts; Mullerian Anomalies arise due to defects in fusion of the two mullerian ducts during intrauterine life. Most cases go unreported as they are asymptomatic and do not cause any menstrual, coital or reproductive problems. The one odd cases presenting thus end up getting investigated thoroughly and are unforgettable learning experiences worth reporting and sharing. The case below is reported for its rarity and as testimony to the fact that despite all advances in our understanding of human embryology, some questions still remain unanswered.

INTRODUCTION:

Mullerian Anomalies result from defective fusion or absorption of the Mullerian ducts during embryonic life. Their incidence is difficult to estimate as majority of these anomalies are minor or insignificant and do not affect menstrual and reproductive function and thus remain asymptomatic. Patients with symptomatic anomalies usually have symptoms of obstruction or reproductive failure. Diagnostic methods for evaluation of the exact nature of the anomalies have evolved immensely over the past years. The advent of ultrasound, hysteroscopy and magnetic resonance imaging has facilitated rapid and accurate diagnosis. The increased availability and utilization of these diagnostic techniques yields complete information about the anomaly under investigation and has resulted in higher reported incidence of various anomalies.

CASE HISTORY

The patient - Mrs. VM - a 26 year old housewife presented in the out patient clinic for investigation of primary infertility. She had been married for 08 yrs and had been living with her husband since then. She gave history of regular coitus two to three times a week with no dyspareunia. Her menstrual history was normal. She had attained menarche at the age of 13 years and had regular cycles since then with moderate flow for three to four days and no dysmenorrhoea. There was no
history of any addictions or drug intake and no significant past medical or surgical history.

Her husband was 30 years old and working as a motor mechanic. He was a non-smoker and non-alcoholic and had no addictions. There was no positive medical or surgical history in his case too.

The patient gave history of being investigated for primary infertility on two occasions in the past, however, no reports were available from the first instance and only films of the hysterosalpingogram from the second.

EXAMINATION

The general and systemic examination was normal. Her vitals were normal and there was no pallor. There was no thyroid swelling or galactorrhoea and no evidence of any signs suggestive of hyperandrogenism.

Gynaecological examination

Per abdomen (P/A) examination was normal.

Per speculum (P/S) examination revealed a complete longitudinal vaginal septum with a cervix visualised in each of the two vaginal cavities (Figures 1 and 2).

Per vaginum (P/V) examination was performed through both the vaginal cavities and revealed a single uterus which was antverted, normal in size, freely mobile and both the fornices were free.

INVESTIGATIONS

The Hysterosalpingogram (HSG) plates available from one of the previous investigations revealed the presence of a septate uterus with two horns. The fallopian tubes were not visualised. There was no spill of contrast into the peritoneal cavity. It was thus decided not to repeat the investigation. (Figures 3 and 4)

The other tests (hemogram, urine analysis, x-ray chest and biochemistry) were normal and were performed with a view to and prepare the patient for examination under anaesthesia (EUA) with diagnostic laparoscopy and dilatation and curettage (D&C).

Husband's semen analysis (HSA) showed evidence of oligoasthenospermia. A urological examination was requested in view of the above and was reported as normal.

Ultrasonography (USG) of the pelvis of the patient (trans abdominal and trans vaginal) confirmed presence of a vaginal septum and a septate uterus with visualization of two separate horns of the uterus. Rest of the pelvic anatomy was unremarkable. (Figures 5 and 6).

USG - Kidney Ureters & Bladder was normal.

Provisional diagnosis - Uterus Dideiphys with bicollis with bicolpos and patient was posted for EUA with diagnostic laparoscopy and D & C under general anaesthesia.

EUA revealed a complete longitudinal vaginal septum with a cervix in each vaginal cavity. The left cervix was not communicating with the uterus while the right one was patent. The upper cervical length was one and half inches. The per vaginum examination was performed through both vaginal cavities and a single
uterus was found to be present. The uterus was anteverted, normal sized, freely mobile and both fornices were free.

**Laparoscopy** revealed presence of a single uterus which appeared normal in shape and size. The tubes and ovaries were unremarkable. A corpus luteum was visualised in the right ovary.

**Chromopertubation** was attempted but was not possible through the left cervix. The same was successfully performed through the right cervix. There was moderate resistance to the passage of dye with only bilateral staining of tubes but no free spill into the peritoneal cavity.

**D & C** was not possible through the left cervix. It was performed through the right cervix and revealed a single uterine cavity with moderate endometrium obtained on curettage which was sent for histopathology examination (HPE).

The HPE report revealed endometrium in late secretory phase.

**Final Diagnosis** - Incomplete Uterus Didelphys with bicornis with bicolpos.

**Treatment** - Patient was advised surgical correction with In Vitro Fertilisation with Embryo Transfer but could not proceed with the same in view of the financial implications of the treatment.

**DISCUSSION**

Uterus Didelphys is a Class III category of malformation as per the American Fertility Society’s Classification of uterine malformations\(^1\). In such cases the Mullerian tract fails to fuse along all or most of its length. There may be complete duplication of the vagina, cervix and uterus and the two halves maybe divided by a ligament of connective tissue.

**EMBRYOLOGY**

In a female foetus, the uterus starts out as two small tubes - the Mullerian ducts. As the development occurs, the tubes normally join to create one larger, hollow organ - the uterus. Sometimes, however, the tubes do not join completely. Instead, each one develops into a separate cavity. Lack of fusion of the two Mullerian ducts results in duplication of corpus and cervix. This condition is called Double Uterus or Uterus Didelphys\(^2\).

Each cavity in a double uterus often leads to its own cervix. Some women with a double uterus also have a duplicate or divided vagina. A vaginal septum is present in about 25% of patients with uterus didelphys\(^1\). Double uterus is rare - and sometimes not even diagnosed as these patients usually have no difficulties with menstruation and coitus\(^3\).

According to one estimate, double uterus occurs in 2 percent to 4 percent of women who have normal pregnancies. The percentage may be higher in women with a history of miscarriage or premature birth\(^4\).

It is not known as to what exactly causes a double uterus. Congenital anomalies of the Mullerian ducts are frequently associated with abnormalities in the urinary tract, which suggests that something may influence the development of these related tubes before birth\(^3\). The renal abnormality is usually present on the same side as the Mullerian defect. Normal
vaginal development is subsequent to the canalization and fusion of the two Mullerian ducts once vacuolization has occurred, but not their union, hence, a duplication of different degree will be the consequence.

INVESTIGATION & DIAGNOSIS

A double uterus is often diagnosed during a routine pelvic exam when the doctor observes a vaginal septum, double cervix or an unusually shaped uterus. It may also be a finding in cases presenting for investigation of primary infertility or after repeated reproductive failures. If the doctor suspects an abnormality, he or she may recommend any of the following tests to help confirm the diagnosis.

- Magnetic resonance imaging (MRI).
- Ultrasound.
- Hysterosalpingography.
- Hysteroscopy.
- Laparoscopy.

Treatment for this anomaly is basically surgical and is needed only if a double uterus causes symptoms or complications, such as pelvic pain or repeated miscarriages. The reproductive outcome for patients with uterus didelphys is similar to that of patients with unicornuate uteri. In view of the acceptable rates of foetal salvage and technical difficulties of uteroplasty, surgical correction is rarely performed. The various surgical modalities that have been suggested for this anomaly include a modified Strassmann procedure and cervical circlage. Strassmann's unification procedure is the operation of choice although hysteroscopic metroplasty with resection of the dividing wall and preservation of both the cervices is also a possibility.

PROGNOSIS

Many women with a double uterus have normal sex lives, pregnancies and deliveries. In fact, the more complete the duplication, the lesser the chances of likely complications. Sometimes a double uterus leads to infertility or miscarriage. A double uterus may also cause premature birth or malpresentations of the baby.

REFERENCES

1. Kenneth J Ryan, Ross R Berkowitz, Robert L Berbeiri - Kistner's gynecology - Principle And Practice - Sixth Edition Pg 144
2. www.mayoclinic.com/health/double-uterus/DS00821-26k
3. Leon Speroff, Robert H Glass, Nathan G Kase - Clinical Gynaecologic Endocrinology And Infertility - Fifth Edition Pg 127(A),130(B).
4. Mulleriananomalies.blogsot.com
5. Linkinghub.elsevier.com/retrieve/pii/50020729203004272
6. Adam Ostrzenski - Gynecology: Integrating Conventional, Complementary, and Natural Alternative Therapy / Genetic Disorders and congenital Malformation in Gynaecology - Pg 36-37

Other References & Internet search modules

A. www.medicyclopedia.com
B. www.ultrasoundmed.org
C. www.emedicine.com
D. Vaclav Insler, Bruno Lunenfeld - Infertility: Male and Female
ACKNOWLEDGING AND EVALUATING PAIN IN ELDERLY CANCER PATIENT

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ABSTRACT:
Pain is now considered the "fifth" vital sign after pulse, BP, temperature and respiration. The WHO estimates that approximately six million people are diagnosed as having cancer in each year.

Our priority should be to improve pain relief in cancer patients but the review articles regularly published on the subject rarely focus on the elderly. More specifically, they seldom concentrate on the communication problems encountered by care teams trying to assess the potential pain of elderly patients, in whom classic methods for self assessment of pain often can't be used. This article outlines an overall assessment of pain in elderly cancer patients, especially those who cannot communicate.

Key words: Cancer Pain, Elderly, VAS Scale, Doloplus-2 scale.

INTRODUCTION:

High prevalence of pain in the elderly patient is a reality. Almost half of those die of cancer are over 75 years of age. In 1994, the New England Journal of Medicine published a survey on the treatment of pain in 1308 cancer sufferers which revealed that first indicator of poor pain management is being an age over 70 years. Hence relief of pain in these patient groups needs to be addressed.

Various authors agree that there is a co-relation between age and the prevalence of persistent pain, that 47-80% of elderly outpatients do not receive suitable treatment for their pain and that many patients suffering from some form of dementia receive no pain relief at all, despite the presence of a concomitants potentiality painful illness. These alarming figures certainly reflect, at least to some extent, they have difficulties encountered by palliative care teams and doctors in assessing pain in elderly patients. The diagnosis of pain is, therefore the first indispensable step in treatment.

ASSESSING PAIN IN ELDERLY:

While assessing pain a distinction needs to be drawn between two different groups of elderly subjects. First groups are those whose intellectual factors are intact and who are therefore able to use different tool (VAS, simple verbal, numerical) that...
exist for self assessment of pain. Second group, afflicted by some form of dementia such as Alzheimer's disease who are very often unable to use such tools.

PATIENT WHO CAN COMMUNICATE:

The evaluation of pain is not as simple in the elderly population class and as being able to communicate normally as it is in younger adults. Older people often tend to minimize their pain, believing that it is inevitable at their age and with their illness. They also have greater difficulty using the tools for self assessment of pain, be it due to a change in their sensory ability. (Lower visual acuity, deafness) or to a poor understanding of how the tools work.

PATIENT WHO CAN NOT COMMUNICATE:

It has also been demonstrated that elderly people presenting with cognitive disorder problem receive analgesics less frequently than patients with intact cognitive function even though their illness are equally painful. This is no doubt explained by the difficulty in diagnosing pain in these patients. These patients do need to be questioned directly but in many carers will get no response and will find themselves left to their own devices in assessing the patient's pain. These difficulties are compounded still further by the possibility of an 'atypical' expression of pain in these subjects. Withdrawal into one self or the manifestations of behavioural problems such as agitation or aggression could be caused by pain. The hetero evaluation of pain therefore becomes necessary to help carers with their observations and to facilitate an assessment as possible.

DOLOPLUS-2 AS AN ASSESSMENT TOOL:

In 1993 Wary et al. put forward a scale known as DOLOPLUS-2 scale (Table-1) validated by a group of geriatricians from various centres in France. Doloplus- 2 scale is simple to use. It is based on observations of patient behaviour on ten different situations that could potentially reveal pain. This behaviour is classified into three groups covering the somatic, psychomotor or psychosocial impact of pain. Each of the ten areas of behavioural studied can be ascribed one of four different levels rated from zero to three, representing increasing intensity of pain. The scores allocated in each category are added together to produce a total score of between zero and 30. Pain is confirmed by a score greater than or equal to five out of 30.

WHO SHOULD USE THE SCALE?

This applies to carers who are already aware of the problems of pain control and or those who have undergone specific training. A systematic recourse to DOLOPLUS-2 by a care team already trained in palliative care and pain evaluation meant that the diagnosis or treatment of pain was rectified in 20% of
<table>
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<th>Table -1. DOLOPLUS-2 SCALE</th>
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| **1. Somatic complaints** | - No complaints  
- Complaints expressed upon enquiry only  
- Occasional involuntary complaints  
- Continuous involuntary complaints  |
| 1 | 2 | 3 |
| **2. Protective body postures adopted at rest** | - No protective body posture  
- The patient occasionally avoids certain positions  
- Protective postures continuously and effectively sought  
- Protective postures continuously sought without stress.  |
| 0 | 1 | 2 | 3 |
| **3. Protection of sore areas** | - No protective action taken  
- Protective actions attempted without interfering against any investigation or nursing  
- Protective actions against any investigation or nursing.  
- Protective actions taken at rest, even when not approached.  |
| 0 | 1 | 2 | 3 |
| **4. Expression** | - Usual expression  
- Expression showing pain when approached  
- Expression showing pain even without being approached  
- Permanent and unusually blank look (voiceless, staring, blank looks).  |
| 0 | 1 | 2 | 3 |
| **5. Sleep pattern** | - Normal sleep  
- Difficult to get to sleep  
- Frequent waking (restlessness)  
- Insomnia affecting waking times.  |
| 0 | 1 | 2 | 3 |
| **Psychomotor reactions** | - Usual abilities unaffected  
- Usual abilities slightly affected (careful but thorough)  
- Usual abilities highly impaired, washing and/or dressing is laborious and incomplete  
- Washing and/or dressing rendered impossible as the patient resists any attempts.  |
| 0 | 1 | 2 | 3 |
| **6. Washing and/or dressing** | - Usual abilities remain unaffected.  
- Usual abilities are reduced (the patient avoids certain movements and reduces his/her walking distance  
- Usual activities and abilities reduced(even with help, patient cuts down his/her movement)  
- Any movement is impossible, the patient resists all persuasion  |
| 0 | 1 | 2 | 3 |
| **Psychosocial reactions** | - Unchanged  
- Heightened (the patient demands attention in an unusual manner  
- Lessened (the patient cuts himself/herself off)  
- Absence or refusal of any form of communication  |
| 0 | 1 | 2 | 3 |
| **8. Communication** | - Participates normally in every activity (meals, entertainment; therapeutic workshop)  
- Participates in activities only when asked to do so  
- Sometimes refuses to participate in any activity  
- Refuses to participate in anything  |
| 0 | 1 | 2 | 3 |
| **9. Social life** | - Normal behaviour  
- Problems of repetitive reactive behaviour  
- Permanent repetitive reactive behaviour  
- Permanent behavioural problems(without any external stimuli)  |
| 0 | 1 | 2 | 3 |
elderly patients suffering from dementia.  

CONCLUSION:

The consequences of cancer pain influence the functional capability and psychological well being of the patient as the disease progresses. Unrelieved chronic pain leads to complications such as depression, withdrawal from society, loss of independence, sleep disorder and loss of appetite and the more systematic use of DOLOPHAN-2 should lead to a real improvement in the quality of life of elderly patients who all, too often receive inadequate pain relief.

REFERENCES:

1. Melding PS. Is there such a thing as Geriatric pain? Pain 1991; 46: 119-121
CONGRATULATIONS

1. Awardees of National President Appreciation Awards 2009
   - Dr. Abhoya Kumar Kar, President IMA Orissa State Branch as Best State President.
   - Dr. Sreejoy Patnaik, State Secretary, IMA Orissa State Branch as Best State Secretary.
   - Dr. R.K. Satpathy, President, IMA Berhampur Branch as Best Local Branch President.
   - Dr. Braja Kishore Dash, Secretary, IMA Cuttack Branch as Best Local Branch Secretary.

2. Dr. Sarat Kumar Mohapatra (Cuttack) - President Elect IMA Orissa State Branch for 2010-11.
   Dr. E.Jayanti Rao (Berhampur) - Vice-President Elect (South Zone) for 2010-11.
   Dr. L.N.Prasad (Talcher-Angul) - Vice-President Elect (East Zone) for 2010-11.

3. Dr. Jayanta Sahu & Dr. Janmejaya Mohapatra of Cuttack as State Ambassadors on Stop Sex Selection declared by National IMA.

4. Dr. Jayant Kumar Panda & Dr. Pravat Kumar Thatoi of Cuttack as State Resource Persons on Swine Flu.

5. Dr. Kamalakanta Panigrahi of Berhampur, Dr. Mrutyunjaya Mohapatra of Cuttack & Dr. Jitendra Kumar Panda of Bhubaneswar as State Resource Persons on Contraceptive update & Safe Abortion Techniques.

6. Dr. Niranjan Rout and Dr. Shivram Prasad Singh have been honoured by IMA College of GP as Honorary Professor of IMA College of GP during its annual convocation on 4th October 2009.

7. Talcher-Angul Branch of IMA has been awarded by Red Cross Blood Bank, Angul on 15th September, 2009 "in appreciation of the invaluable service to humanity rendered".

8. Dr. Satya Ranjan Panigrahi as Master Trainer & Dr. Sanjit Patnaik of as Unit Coordinator of IMA Orissa State Branch on RNTCP.
OBITUARY: 2009-2010

The IMA Orissa State Branch & members is deeply mourned on the sad demise of its members and pray almighty "May the departed souls rest in peace".

BERHAMPUR BRANCH

1. Dr. M.C. Dandapat
2. Dr. Prabhash Ch. Rath
3. Dr. Ananda Pati Patro.
4. Dr. Manju Patanaiik.
5. Dr. Panchanana Moharana.

SAMBALPUR BRANCH

1. Dr. Lal Anant Saideo

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1. Dr. Jogendra Mohan Senapati
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1. Dr. Arun Ranjan Pal

ROURKELA BRANCH

1. Dr. Dhobesh Chandra Mishra
2. Dr. K. P. Sinha
3. Dr. M. C. Bhatacharya
IMA ORISSA STATE BRANCH HONOURS
THE FELLOWSHIP CONFERED ON
DR. NIRANJAN ROUT
BY
IMA COLLEGE OF GENERAL PRACTITIONERS

IMA COLLEGE OF GENERAL PRACTITIONERS
HEAD QUARTERS, CHENNAI.

Honorary Professor Ship

In recognition of Academic excellence,
Social Contributions, altruistic attitude and aptitude
for teaching, it is declared and known
on this day of 4th October 2009.

Dr. Niranjan Rout MBBS., MD (Pathology)
303, Majestic Tower,
Ring Road,
Mangalabag, Cuttack - 753 001

is designated as
HONORARY PROFESSOR
of IMA College of General practitioners,
and privileged to teach and contribute for the promotion
of Family Medicine Speciality in India.
IMA ORISSA STATE BRANCH HONOURS
THE FELLOWSHIP CONFERRED ON
DR. SIVRAM PRASAD SINGH
BY
IMA COLLEGE OF GENERAL PRACTITIONERS

IMA COLLEGE OF GENERAL PRACTITIONERS
HEAD QUARTERS, CHENNAI.

Honorary Professor Ship
In recognition of Academic excellence,
Social Contributions, altruistic attitude and aptitude
for teaching, it is declared and known
on this day of 4th October 2009,

Dr. Sivram Prasad Singh, MBBS, MD, DM
1362/B, Sector-6, CDA,
Abhinava Bidanasi, Cuttack - 753 014

is designated as
HONORARY PROFESSOR
of IMA College of General practitioners,
and privileged to teach and contribute for the promotion
of Family Medicine Speciality in India.
With Best Compliments From:

Bharat Serum