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Mailing Address
Prof. Satinder Gombar
Editor, Journal of Medical College, Chandigarh
Department of Anaesthesia & Intensive Care
Government Medical College & Hospital, Sector 32,
Chandigarh-160 030

Email: editor.jmcc@gmail.com

Webpage: http://gmch.gov.in/journalgmch/journal_main.htm

Tel: +91-172-2665253 Ext.: 1219
Fax: +91-172-2609360

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CONTENTS

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Tel: +91-172-2665253 Ext.: 1219

Fax: +91-172-2609360

Editorial

Comfortably Numb: Advance Directive and the Right to Die, Where do we stand?

Medical Science has made unforeseen strides in patient healthcare in last few decades; this along with advanced life support interventions has lead to improving outcomes in diseases and conditions which often left physicians exasperated earlier.

This however has created greed; greed amongst the healers, to prevent and postpone death in each and every patient. This modern "techno medicine" eventually resulted in prolongation of the process of dying through overzealous, burdensome and at times futile attempts to keep the patients alive. In our passion to safeguard the "*Right to Life,"* we, the health care providers forgot about the latter part of the sacrosanct, *"with dignity."* Hence, to grant more autonomy to the patients under care and save them from painful, heroic and futile attempts at protracting a debilitated life, the concept of "Advance Directive" was born.

Luis Kutner, an Illinois attorney in 1969 first proposed a way for an individual to predetermine his or her healthcare desires for a time when the individual is no longer able to express healthcare wishes. Since the will was applicable to a time when the individual was still alive, it was dubbed the "living will."[1] The United States of America first recognized advance directives in 1976 when the state of California passed the Natural Death Act,[2] while the Patient Self Determination Act (PSDA)[3] of 1991 directed healthcare providers in the United States of America to give patients information about their rights to make advance directives under state law.

Advance directive, a legal document essentially includes treatment decisions made in advance; in anticipation of some future casualties when the patient is incompetent or incapacitated to the extent that he/she cannot make or participate in such decisions himself/herself.[4,5] They can be further classified as:

- Living Will
- Durable power of attorney
- Do Not Resuscitate Order (DNR)

A Living will grants autonomy to the patient to not receive life-sustaining treatment in the event of an incurable, irreversible disease often listing specific interventions that patient does not want in the eventuality of him or her being incapacitated. Durable power of attorney is different from a Living will, as it allows the individual to appoint a representative in-charge of decision making while a DNR simply desists medical professionals from performing a Cardiopulmonary Resuscitation. Combination of the living will and the durable power of attorney for healthcare into one document is called an "advance health care directive".[6,7]

Lessening human suffering was the original moral impetus for advance directives; however, it poses a far more insidious moral and ethical question. Does the *"right to life"* include the *"right to die"*? The question itself is rather convoluted and vexing and implores us to tread rather finely between an individual's right to privacy and autonomy over the decisions involving the body on one hand , and the state's responsibility towards the individual as a guardian (*parens patriae)* on the other. In Indian scenario, this debate's 24 year long journey (starting with 1994 *P. Rathinam vs Union of India*[8] to a more recent *Aruna Ramchandra Shanbaug vs Union Of India & Ors,* 2011[9]) finally saw the Supreme Court of India, in a landmark judgment acknowledge, that the *right to live* with dignity includes, in certain circumstances, the *right to die* also.[10]

In a 538 pages long judgment, the five judge bench comprising of Chief Justice of India Dipak Misra, Justice AM Khanwilkar, Justice AK Sikri and Justice DY Chandrachud declared that a terminally-ill patient or a person in persistent vegetative state can execute an "advance medical directive" or a "living will" to refuse medical treatment. They recognized advance medical directives valid in matters of passive euthanasia taking human dignity, autonomy

and self determination. The bench held that right to live with dignity included "smoothening" the process of dying in a case of terminally ill patient, or a person in persistent vegetative state with no hope of recovery.[10,11]

The judgment also made distinctions between passive euthanasia, active euthanasia and physician assisted suicide. Active euthanasia comprises of administration of a lethal substance or force to kill a person, whereas in physician assisted suicide the patient is given choice of the drug and usually self administers it. Hence both types have an "act" and an intent to cause death (*mens rea)*. While passive euthanasia involves withholding or withdrawal of treatment necessary for continuance of life. The cause of death here is attributed to the patient's underlying condition rather than the withdrawal of support.[11]

With the government spending only 1.4% of its Gross Domestic Product on healthcare, the public health system in India is already in poor state. In a country which forms a little less than half of the people pushed into poverty due to out of pocket expenditure on healthcare (World Health Organisation and World Bank report 2017),[12] right to refuse treatment has far reaching implications. Living wills also provide a much needed moral and social reassurance to the family members that the decision to withdraw treatment was in accordance with patient's free will, thus absolving them of feelings of any guilt whatsoever.

Before we rejoice over the judgment however, there are certain limitations which need to be addressed. The living will or advance directive is a legal document, and not a medical order and is operational only if the patient is in a permanent vegetative state (PVS) and/ or afflicted with a terminal illness. What constitutes as terminal illness and how long the PVS lasts before the withdrawal of treatment, is a matter of clinical judgment. Also it must be realized that such documents are nothing but a piece of paper, which may be overlooked, lost, stashed in a forgotten drawer and treatments instituted before the attending physician stumbles upon it. Generally, the emergency medical personnel operate under standing orders to resuscitate, which can be only prevented by a DNR order. Then there is the matter of power of attorney (POA) and medical proxy. Under the provisions of the Indian Contract Act (ICA) of 1872,[13] POA is defined as "any instrument empowering a specified person to act for and in the name of the person executing it." However section 201 of the ICA specifies that such instruments and agencies would be *terminated* if the principal (the patient in this case) becomes incapacitated, hence defeating its very purpose. Finally, it must be acknowledged that India has a massive population with the majority of it lying in the nadir of the social strata. India shares 37% of the global burden of illiterate adults with 20.6% of the world's poor. The healthcare access is also, consequently, asymmetric. Chances are that poor won't even get an access to advanced life saving techniques and continuation of life therapies, let alone a chance to make an advance directive. In fact there is a real possibility of unethical harvesting of organs from people being declared 'brain-dead.'

There is no denying that this judgment on the matter of *Common Cause India vs Union of India*[14] is landmark in the advent of patient autonomy, and it opens up the medical fraternity to action rather than mere debates. The deficiencies however need to be addressed in a systematic and a swift manner. There is a long and arduous journey left for this judgment to be translated into a meaningful process. Meanwhile one's mind turns to the "Medical Litany" by Sir Robert Hutchinson of which, Hamilton Bailey and McNeil Love were particularly fond of:

"From inability to let well alone;
From too much zeal for the new and contempt for what is old;
From putting knowledge before wisdom, science before art and cleverness before common sense;
From treating patients as cases; and From making the cure of the disease more grievous than the endurance of the same, Good Lord, deliver us."

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Satinder Gombar[1], Pradeep Atter[2]
[1]Professor & Head, [2]Junior Resident,
Department of Anaesthesia & Intensive Care,
Government Medical College & Hospital, Chandigarh

Original article

Prevalence of Antimicrobial Resistance in *Staphylococcus Aureus* Isolates to Old and New Antibiotics- A Need for Continued Antimicrobial Surveillance

Varsha Gupta[1], Mandeep Kaur[2], Priya Datta[3], Jagdish Chander[1]

[1]Professor, [2]Demonstrator, [3]Assistant Professor

Department of Microbiology, Government Medical College Hospital, Sector 32, Chandigarh, India 160030

ABSTRACT

Background: Accurate antimicrobial susceptibility testing and screening for methicillin resistant *S. aureus* (MRSA) colonized and infected patients are important tools to limit the spread of this organism. The study evaluated the prevailing drug resistance among *S. aureus* and determined minimum inhibitory concentrations (MICs) for old and new antibiotics. *Methods:* One hundred *S. aureus* isolates from various clinical samples were included in the study. Antimicrobial drug susceptibility and MICs was determined as per Clinical Laboratory Standards Institute (CLSI) guidelines. *Results:* High level ciprofloxacin resistance was observed in 45% strains showing MIC values of ≥16 µg/ml. MRSA isolation was 10%. Low mean MICs were observed for most of the drugs except for vancomycin that although 100% susceptible showed raised MICs. *Conclusion:* Over the counter available drug use has increased the problem of antimicrobial resistance even in community. Therefore, continued antimicrobial surveillance is of utmost importance, in order to regularly revise the empirical therapies.

Keywords: *Staphylococcus aureus,* Minimum inhibitory concentration, Ciprofloxacin resistance, MRSA

INTRODUCTION

Staphylococcus aureus is an important cause of infectious diseases worldwide and is a frequently reported pathogen both from hospital and community-acquired infections. The infections caused by *S. aureus* were often fatal before the discovery of penicillin. The situation improved after the penicillin was used for the treatment of infections, but very soon, the penicillin degrading β lactamases (penicillinases) production by *S. aureus* made this antibiotic ineffective for use. In 1959, to overcome the penicillin drug resistance, methicillin, a penicillinase resistant β lactam was introduced in practice but within two years of its clinical use, the methicillin resistant *S. aureus* (MRSA) isolates started emerging. Until recently, MRSA was predominantly a nosocomial pathogen, but now MRSA strains are being increasingly isolated from community as well, thereby posing a greater challenge in treatment.[1]

Corresponding Author :

Dr. Mandeep Kaur, Demonstrator
Department of Microbiology,
Government Medical College Hospital,
Sector 32-B, Chandigarh, India 160030
Email: drmandeepkaur@gmail.com

Vancomycin (glycopeptide) is a drug of choice for managing serious MRSA infections, but the emergence of glycopeptide resistant *S. aureus* in Japan and the USA, and the heteroresistance to glycopeptides in MRSA have raised the concerns, further limiting the therapeutic options to treat these multidrug resistant (MDR) pathogens.[2] Such a scenario calls for development of newer antimicrobial options but there has always been a hesitation in their development and only few newer antimicrobials such as daptomycin (lipopeptide), linezolid (oxazolidinones), quinupristin/dalfopristin (streptogramin combination), telithromycin (ketolide), and tigecycline (glycylcycline) have been developed in the last ten years and these are also mainly derived from modifications of existing antibiotics.[3]

The frequently changing susceptibility profile of the pathogens calls for regular surveillance of the antibiotics so as to timely revise the empirical therapies against these deadly pathogens. The purpose of the present study was to find out the prevalence of antimicrobial drug resistance in various clinical isolates of *S. aureus* to both old and newer antimicrobials and also determination of MIC values of these antimicrobials which would help in formulation of empirical therapy for treating *S. aureus* infections.

MATERIAL & METHODS

Study setting and design: A hospital based descriptive study was conducted in the Department of Microbiology, Government Medical College Hospital, Chandigarh.

Study population and sample size determination: Both in-patient and out-patient patient's samples were included in the study. The patients admitted in intensive care units and admitted in other critical areas (Pediatric ICU, Cardiac care unit) were excluded from the study. Sample size was calculated taking the 45% prevalence of MRSA from a regional study and using Daniel's formula. The sample size required was calculated to be around one hundred.[4] During the study period, all non-repeat consecutive *S. aureus* isolates obtained from the clinical samples of the patients were collected and processed, until the sample size was achieved.

Sample processing and strain identification: All the samples were processed using standard microbiology techniques and final identification was done following recommended conventional test scheme.[5] Based on disc diffusion testing using cefoxitin disc (30 µg disc), *S. aureus* were classified as methicillin susceptible *S. aureus* (MSSA) and MRSA. Vancomycin resistance was detected by presence of growth on vancomycin (6 mg/l) screen agar and inducible clindamycin resistance was detected by D-test. For interpretation of drug susceptibility, Clinical Laboratory Standards Institute (CLSI) criteria were followed.[6] Furthermore, MICs were determined by performing E-test (Epsilometer) for all the antibiotics. All the culture media, antibiotics discs and standard strains of bacteria used in the study were procured from Hi Media Laboratories Pvt. Ltd. (Mumbai, India). *S. aureus* ATCC 25923 (MSSA standard strain), *S. aureus* ATCC 29213 (MRSA standard strain) and *Enterococcus faecalis* ATCC 51299 (vancomycin resistant *Enterococcus*) were used for quality control. E-test strips of oxacillin, tetracycline, ciprofloxacin, fosfomycin, clarithromycin, rifampicin, vancomycin, teicoplanin, tigecycline, daptomycin, linezolid, quinupristin/dalfopristin, doripenem and mupirocin were obtained from AB BioDisk, Solna, Sweden.

E-test for daptomycin was done on Mueller-Hinton agar supplemented with 50 mg/l calcium (Difco, USA) due to daptomycin's dependence on calcium. MIC values were interpreted according to the CLSI guidelines except for mupirocin and tigecycline for which European Committee on Antimicrobial Susceptibility Testing (EUCAST) criteria was followed.[7]

Statistical analysis

The antimicrobial susceptibility pattern of *S. aureus* strains was calculated in terms of percentages (percent

Table 1

Percentage susceptibility and distribution of MIC# values for various antibiotics in *Staphylococcus aureus*

Antimicrobial agent	% susceptibility	MIC# Range (µg/ml)	MIC50 (µg/ml)	MIC90 (µg/ml)	Geometric mean of MIC (µg/ml)
Chloramphenicol	100 %	0.75-6	3	6	3.1375
Clarithromycin	90%	0.094-256	1	3	26.54245
Clindamycin *	92%	0.016-0.064	0.032	0.047	0.03425
Ciprofloxacin	24%	0.064-32	12	32	15.6111
Daptomycin	99%	0.047-1.5	0.25	0.5	0.39385
Doripenem	100%	0.023-1	0.032	0.125	0.1097
Fosfomycin	100%	0.064-0.75	0.19	0.5	0.275
Linezolid	100%	0.38-2	1	1.5	1.069
Mupirocin	100%	0.25-1	0.5	0.75	0.54
Oxacillin	90%	0.125-8	0.25	1	0.7515
Quinupristin-dalfopristin	100%	0.25-0.75	0.38	0.5	0.446
Rifampicin	100%	0.002-0.008	0.002	0.006	0.0033
Tetracycline	95%	0.064-12	0.38	4	1.40105
Teicoplanin	100%	0.38-1	0.75	1	0.7505
Tigecycline	100%	0.047-0.25	0.125	0.25	0.1375
Vancomycin	100%	0.5-2	1.5	2	1.65

*8% inducible clindamycin resistance (iMLSB) was observed.
MIC-Mean inhibitory concentration

sensitivity) and the geometric mean of MICs, MIC 50 and MIC 90 for all the antibiotics against *S. aureus* isolates was calculated using Windows 7 Microsoft Excel software.

RESULTS

A total of 100 *S. aureus* isolates were obtained from various clinical samples namely pus samples (n-68), tissue biopsies (n-10), sputum samples (n-10), joint fluid aspirates (n-8) as well as blood cultures (n-4). Out of 100 *S. aureus*, ten isolates obtained from pus samples were methicillin resistant *S. aureus* (MRSA). One hundred percent susceptibility was observed for vancomycin when tested with agar dilution method and MIC range for vancomycin remained between 0.5-2 µg/ml with mean MIC of 1.65µg/ml. A high degree of fluoroquinolones resistance (76%) was observed in *S. aureus* strains where high level resistance to ciprofloxacin was seen in 45 strains showing MIC of ≥16µg/ml. Ten strains showed total resistance to clarithromycin (MIC ≥ 256µg/ml) while inducible clindamycin resistance (iMLSB) was observed in 8 strains. The distribution of MIC values to the range of antibiotics tested, along with MIC 50, MIC 90 and geometric mean of MICs for each antibiotic is given in Table 1. Overall low mean MICs were observed for most of the newer antibiotics except for a single MRSA strain isolated from pus that exhibited a very high MIC value of 1.5µg/ml for daptomycin.

DISCUSSION

S. aureus is the leading cause of nosocomial infections, but now due to large number of patients being treated outside the hospital setting, it has become a cause of concern in the community as well.[8] It is one of the earliest bacteria to acquire drug resistance by various mechanisms ranging from production of β lactamase (penicillin resistance), altered binding protein (methicillin resistance), thickening of the cell wall (vancomycin resistance) and mutation in genes involved in protein synthesis (as in inducible clindamycin and macrolide resistance).[1] MRSA at present is the most commonly identified drug resistant pathogen in many parts of the world, including Europe, USA, North Africa, Middle East, and East Asia.[9] Our study found 10% *S. aureus* isolates to be MRSA while previous study results from the same institute and other Indian studies have reported a high prevalence i.e. between 25%-35%.[10,11] Also, a study by Indian Network for Surveillance of Antimicrobial Resistance (INSAR) group in 2013 had reported 41% MRSA prevalence that were mostly isolated from inpatients.[12] The increased acquisition of resistance genes due to selection pressure is more commonly seen among hospital circulating strains as compared to community strains. At our hospital, the institutional antibiotic policy was started two years back that could be the reason for such deceleration seen in antimicrobial drug resistance.

Vancomycin, the drug of choice in MRSA, nowadays is showing reduced susceptibility with high creeping MIC values. The clinical failures with vancomycin treatment have been observed where vancomycin was reported susceptible but had high MIC value.[13] Though all MRSA isolates were 100% susceptible to vancomycin, still a gradual shift in vancomycin MICs from 1 to 2 mg/l was observed during a five year study conducted at a premier institute in North India.[14] Our study too found 100% susceptibility for vancomycin but accelerated MICs (~2µg/ml) were also observed in 45 out of 100 *S. aureus* strains. Though vancomycin intermediate resistant *S. aureus* (MIC 4-8µg/ml) is being increasingly reported worldwide and from the Indian subcontinent, we did not detect Vancomycin intermediate *S. aureus* (VISA) or Vancomycin Resistant *S. aureus* (VRSA) in our study.[14] A study from South India had reported a VISA strain with MIC of 5 mg/l among MRSA isolates.[15] Western data on vancomycin susceptibility too showed almost similar patterns. In USA, a five year study done on MRSA blood culture isolates showed 100% vancomycin susceptibility but the percentage of isolates with higher MIC than the median MIC had significantly increased over the years. Vancomycin resistant *S. aureus* (VRSA) strains with vancomycin MIC ≥ 16 µg/ml are emerging but still scarcely reported.[16] Other commonly used antibiotics in treatment of *S. aureus* infections are macrolides and lincomycins (clindamycin). Macrolide resistance was seen in 10% isolates and inducible clindamycin resistance (iMLSB) was detected in 8 out of 10 clarithromycin resistant strains (MIC ≥256µg/ml). No cMLSB strains were observed in our study. In a study from north-east India also, inducible clindamycin resistance was detected in 10.7% and 39% macrolide resistance was observed among the tested strains.[17]

Ciprofloxacin which is now treated as a reserve drug in India and used as a last resort for the treatment is showing ineffectiveness in MRSA. Moreover, over the counter availability of this drug has further increased the resistance to this antibiotic against almost every pathogen. Our study too observed a high degree of ciprofloxacin resistance (76%) with high level resistance

(MIC of ≥16 µg/ml) observed in 45 *S. aureus* strains. The same scenario of ciprofloxacin resistance has been reported from almost all parts of country. A study from Bangalore, India, reported similar rates of ciprofloxacin resistance (70.6%) in both MSSA and MRSA strains[18] whereas other studies have reported resistance rates between 97-100% among MRSA strains.[19,20] Increased isolation of ciprofloxacin resistant strains among MRSA patients treated with ciprofloxacin can establish the association of fluoroquinolones exposure and MRSA.[19]

With older antimicrobials developing resistance, there is a need to explore newer options to treat. Daptomycin, a lipopeptide antibiotic, is one such new antimicrobial agent that is being used clinically for treating MRSA infections. The emergence of resistance in *S. aureus* to daptomycin is often associated with multiple genetic changes occurring during treatment.[21] Still not much resistance to this new agent has been observed worldwide and can be used in the setting of highly resistant *S. aureus* strain. In the present study also, only a single MRSA isolate out of all, showed high MIC value for daptomycin. Other newer agents like quinupristin-dalfopristin, doripenem, tigecycline, linezolid and fosfomycin were found to show 100% susceptibility with low mean MIC values. In most of the other studies also, Daptomycin, tigecycline and fusidic acid have been found to be 100% susceptible when tested on MRSA isolates.[22,23] Finally, at our institute, as almost all older group of antibiotics showed good susceptibility patterns with exception of ciprofloxacin, hence in suspected Staphylococcal infections, agents like cotrimoxazole, clindamycin, macrolides and tetracyclines could be started as empirical therapy. The only limitation of our study is the small sample size. But the reason for this is the limited infrastructure for research in our government aided institute; high cost of E-strips for determining the MIC values came as a major hindrance in increasing the sample size.

In conclusion, *S. aureus* is still showing good susceptibility for both conventional and newer agents with few exceptions. Increased MICs for vancomycin raise a concern as these usually result in clinical failures. Newer group of antibiotics showed promising results but more studies are needed from different regions of the world to give a global picture of the same. At the end, drug resistance in bacteria is difficult to handle in view of the increased armoury of microbes. Development of new and effective armament in fighting these powerful pathogens along with effective regular surveillance to bring out the actual burden of the problem is need of the hour.

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Ethical approval: The present study was not an interventional study involving either humans or animals, but was a laboratory work on bacterial strains. Hence ethical approval was not obtained.

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A Randomized Controlled Study to Compare Proseal Laryngeal Mask Airway with Classic Laryngeal Mask Airway in Anaesthetized Paralyzed Children

Anudeep Jafra[1], Satinder Gombar[2], Dheeraj Kapoor[3], Swati Jindal[4]

[1]Assistant Professor, [2]Professor, [3]Associate Professor, [4]Assistant Professor,

[1]Department of Anesthesia and Intensive Care, Post Graduate Institute of Medical Education & Research, Chandigarh.
[2-4] Government Medical College & Hospital, Sector 32, Chandigarh.

ABSTRACT

Background: The aim of the study was to compare Proseal Laryngeal mask airway (PLMA) and Classic Laryngeal mask airway (CLMA) in anaesthetized paralyzed children who were undergoing elective surgery. *Methods:* 100 ASA I-II children were included. They were randomly assigned to two groups. We assessed success rates as ease of insertion, oropharyngeal seal pressures (OSP), quality of initial airway, fibreoptic position, hemodynamic variables and frequency of mucosal trauma in either group. *Results:* The ease of insertion and the number of attempts at insertion were comparable in two groups while the OSP was significantly high in PLMA group (30.02 ± 3.21 cmH_2O) than CLMA group (21.62 ± 2.49 cmH_2O) (p value 0.001). Quality of initial airway was also better in PLMA group. The pharyngolaryngeal morbidity was comparable in two groups. *Conclusion* PLMA is a better alternative as compared to CLMA for positive pressure ventilation in anaesthetized paralyzed children.

Keywords: CLMA, PLMA, Paediatrics, Positive pressure ventilation, Oropharyngeal seal pressure

INTRODUCTION

The safety and efficacy of *Classic*™ Laryngeal mask airway (CLMA) for securing airway has been shown in various large scale studies. [1,2] However there are well known limitations of the CLMA in paediatric patients, in particular size 1 and 2.[3,4] The main concern is its low pressure seal which might be inadequate for positive pressure ventilation (PPV) with a risk of gas leakage into the stomach with subsequent risk of gastric distension and regurgitation. To overcome this problem modified Proseal™ LMA has been introduced, it has a large and deeper bowl, enlarged and soft cuff designed to create a higher sealing pressure and a parallel drain tube to prevent gastric aspiration, and insufflation and ensure adequate placement of PLMA. The paediatric versions of PLMA lack the adult dorsal cuff and have proportionately larger drain tube but these modifications do not interfere in its performance.[5] There is a paucity of data on the comparison of PLMA with CLMA in anaesthetized paralyzed children during positive pressure ventilation. In the present prospective, randomized and controlled study we compared the ease of insertion; oropharyngeal seal pressure (OSP), fibreoptic visualization and pharyngolaryngeal morbidity between the PLMA and the CLMA. The primary outcome studied was oropharyngeal seal pressure and secondary outcomes studied were time to insertion of device, number of attempts, quality of initial airway, ventilatory parameters (SpO_2 and End tidal CO_2), fiberoptic grading and pharyngolaryngeal morbidity.

Corresponding Author :

Dr Anudeep Jafra, Assistant Professor,
Department of Anesthesia and Intensive Care,
Post Graduate Institute of Medical Education & Research, Chandigarh, India
Email: anu_gmch@yahoo.co.in

MATERIAL AND METHODS

After obtaining institutional ethics committee approval, and written informed parental consent, 100 paediatric patients of American Society of Anaesthesiologists (ASA) physical status I /II of either sex, in the age group of 6 months - 10 years, weighing 6 to 30 kg who were scheduled to undergo elective genitourinary, general, or orthopaedic surgeries in supine position requiring general anaesthesia were included in this study. Patients with ASA status greater than II, upper respiratory tract infection, anticipated difficult airway, those at increased risk of aspiration, non fasting status and with significant cardiorespiratory or cerebrovascular disease were excluded from the study. Children were randomly allocated to either PLMA or CLMA group using computer generated random number table and coded sealed envelope method.

All the children were premedicated with 0.3 mg.kg^{-1} nasal midazolam (Midazolam Nasal Spray INSED Atomiser, SAMARTH Pharma Pvt. Ltd.), 30 min prior to surgery. Standard monitoring included pulse oximetry, non-invasive arterial blood pressure, electrocardiography and capnography (S/5^{TM} Datex Ohmeda USA). General anaesthesia was induced with sevoflurane (inspired concentration 6-8%) with 50% nitrous oxide in oxygen. Intravenous (IV) line was secured on the dorsum of hand. After checking for the ability to adequate mask ventilation, neuromuscular blockade was achieved with IV atracurium besylate 0.5 mg.kg^{-1}. After achieving complete relaxation, the airway device was placed (as per group allocation), while keeping the head and neck of the patient in neutral position. For intraoperative analgesia, IV fentanyl citrate 1-2 mcg.kg^{-1} was given and anaesthesia was maintained with sevoflurane (1- 2%) with 66% nitrous oxide in oxygen.

A size 1.5, 2, or 2.5 (depending on the weight), PLMA / CLMA was chosen as per group allocation of the patient. The cuff was fully deflated prior to insertion and posterior aspect of cuff was adequately lubricated with water soluble KY jelly. Insertion of device was as per manufacturer's guidelines, and a standard digital insertion technique was used for CLMA and introducer tool for PLMA.[6] The cuff was inflated to 60 cm H_2O using manometer. The device was then connected to the paediatric circle system of anesthesia machine (Aestiva 5^{TM} 7900, Datex Ohmeda, USA) and patient was ventilated. All the devices were inserted by the same anaesthetist with an experience of atleast 50 LMA insertions.

The number of insertion attempts to achieve effective ventilation (square wave capnogram) were recorded. If ventilation was not adequate or there was significant leak, the device was removed and reinsertion attempted. A maximum of three insertion attempts were allowed followed by securing airway using alternate device (endotracheal intubation). The time interval between picking up the device and obtaining square wave capnogram was recorded as insertion time. The quality of the initial airway was assessed during manual ventilation, with the pop-off valve set to limit peak airway pressure to 20 cm H_2O.[7] The airway was judged as: excellent = no audible leak; good = an audible leak with loss of air but sufficient ventilation ($EtCO_2$ <40 mm Hg); and poor = clinically relevant loss of air and insufficient ventilation, requiring repositioning or replacement of the device. The OSP was determined by closing the expiratory valve of the circle system at a fixed gas flow of 3 liters.min^{-1} and recording the airway pressure (maximum allowed 40 cm H_2O) at which equilibrium was reached.[8] The gas leak was determined at the mouth (audible), the stomach (epigastric auscultation) or the drainage tube (bubbling of lubricant placed on the proximal end of the drainage tube). The patient's lungs were ventilated with a tidal volume of 5-7 ml.kg^{-1}, the respiratory rate adjusted to maintain $EtCO_2$ of 35-40 mm of Hg with inspiratory: expiratory ratio of 1:2. For adequacy of ventilation, oxygen saturation and end tidal carbondioxide levels were documented preoperatively, after induction, at the time of device insertion and 5, 10, 15 mins after device insertion. Fibreoptic grading of the view through airway tube was carried out using flexible fibreoptic scope (Flexible Fiberoptic Scope; Pentax Medical Singapore PTE.LTD). The fibreoptic view was graded as: 1 = vocal cords not seen; 2= vocal cords and anterior epiglottis visible; 3= vocal cords & posterior epiglottis visible; 4= only vocal cords visible[9] The following complications were documented: device failure, intraoperative displacement, gastric insufflation, regurgitation/ aspiration, laryngospasm, bronchospasm and airway obstruction. After the surgery, residual neuromuscular blockade was reversed with neostigmine methyl sulfate 0.05 mg kg^{-1} and glycopyrrolate 0.01 mg kg^{-1}. The device was removed and any blood staining of the device, tongue-lip-dental trauma was recorded. Postoperatively, children were monitored in post anaesthesia care unit and subsequently for 24 hours for sore throat and hoarseness of voice.

STATISTICAL ANALYSIS

The primary variable tested was OSP in two devices. Considering a projected difference of 30% between the two groups for airway sealing pressures to be significant, at 95% confidence limits , a type 1 error of 0.05 and a power of 0.8, a total of 100 patients needed to be studied (50 each group). To account for possible drop outs, 120 patients were enrolled. Secondary variables tested were: ease of CLMA/PLMA insertion, fibreoptic view, quality of initial airway and any intra/post operative complications. The data was analysed using Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, version 15.0 for Windows). Kolmogorov Smirnov tests indicated normality of data. All quantitative variables were estimated using measures of central location (mean, median) and measures of dispersion (standard deviation and standard error). For normally distributed data, means were compared using Students t- test for two groups. For skewed data, Mann- Whitney U test was applied. Qualitative or categorical variables were described as frequencies and proportions. Proportions

were compared using Chi square or Fishers exact test whichever was applicable. All statistical tests were two-sided and performed at a significance level of $\alpha = 0.05$.

RESULTS

There were no differences between the two groups regarding patient demographic characteristics. First attempt success rates were similar between PLMA and CLMA (92% and 90%); four patients in group I and five in II required a second attempt. The difference in time taken for successful placement of PLMA (21.90 ± 4.93 secs) and CLMA (19.46 ± 5.99secs) was found to be statistically significant ($p< 0.05$). Though the difference was found to be significant but on comparing the time for insertion for different sizes, result was statistically insignificant ((1.5 size 20.22±1.09 secs in PLMA and 19.8±1.64 secs in CLMA), (2 size 23.64±3.79secs in PLMA and 21.58±4.44secs in CLMA) and 2.5 size 27.5±6.31secs in PLMA and 23.33±5.73secs in CLMA). Ease of insertion was similar between two devices. (Table 1,2) Though this difference was found to be significant, but when the time taken for inserting different sizes of LMA's were compared, we found no statistically significant differences.

The difference in OSP between two groups was found to be statistically significant ($p<0.001$), 38.85% difference was found between two groups with PLMA group exhibiting higher OSP (30.02 ± 3.21 cmH_2O) than CLMA group (21.62 ± 2.49 cmH_2O). When compared between different sizes of the devices (1.5,2,2.5) the pressures were found to be higher in PLMA as compared to CLMA. Size 1.5 of PLMA exhibited OSP of

Table I
Demographic Profile and Comparison of PLMA and CLMA

Serial number	Variables	PLMA (n=50)	CLMA(n=50)	p value
1.	Demographics			
	Age (years)	4.70±2.89	5.79±2.90	0.063
	Sex (female : male)n	16:34	16:34	NS
	Weight (kg)	15.92±5.90	18.18±5.91	0.059
2.	Time taken for PLMA/CLMA insertion (secs)	21.90±4.93	19.46±5.99	0.029
3.	Oropharyngeal seal pressure (cmH_2O)	30.02±3.21	21.62±2.49	0.001
4.	Quality of initial airway (excellent/good)	50/0	30/20	0.001
5.	Fibreoptic grading(4/3/2/1)	31/14/5/0	31/15/4/0	NS
6.	Adverse effects			
	Bronchospasm	0	0	NS
	Arterial desaturation	0	0	NS
	Lip/ dental trauma	0	0	NS
	Blood on device	2	3	NS
	Hoarseness/ sorethroat	0	0	NS

Data Expressed as Mean ± SD, N=number, NS= Not Significant

Table 2
Time taken to insert PLMA/CLMA different sizes (secs)

Size	PLMA	CLMA	p value
1.5	20.22±1.09	19.8±1.64	0.54
2	23.64±3.79	21.58±4.44	0.07
2.5	27.5±6.31	23.33±5.73	0.068

Table 3
Oropharyngeal seal pressure (OSP in cmH2O) in different sizes of PLMA and CLMA

Size	PLMA	CLMA	p value
1.5	31.5±3.17	22.46±2.94	<0.0001
2	30.96±3.65	23.0±3.19	<0.0001
2.5	30.4±2.45	22.33±3.45	<0.0001

31.5±3.17 cmH_2O and CLMA 22.46±2.94 cmH_2O, size 2 PLMA 30.96±3.65 cmH_2O and CLMA of 23.0±3.19cmH_2O and in size 2.5 PLMA had OSP of 30.40±2.45cmH_2O and CLMA 22.33±3.45 cmH_2O. (Table 3)

The mean values of heart rate, arterial oxygen saturation by pulse oximetry (SpO_2), and $EtCO_2$ preoperatively, after induction, at PLMA/CLMA insertion, 5,10 and 15 mins after PLMA/CLMA insertion was not significantly different between the two groups at specified times ($p>0.05$).

All patients in group I showed excellent quality of initial airway as compared to 60% (30) in group II, with 40% (20) patients having a good quality of initial airway in group II making it statistically significant.

The difference in fibreoptic grading of the view through the airway tube was found to be statistically insignificant revealing grade 4 in 31 patients in both group I and II. There was no case of airway obstruction, bronchospasm, gastric insufflation, dental or mucosal trauma. Blood was detected on mask in 10% of patients which was comparable in two groups. None of the patients had sore throat or hoarseness of voice in either group in immediate postoperative period and till 24 hours thereafter.

DISCUSSION

Our study suggested that significantly higher OSP was achieved with PLMA (30.02 ± 3.21 cmH_2O vs 21.62 ± 2.49 cmH_2O, difference of 38.85%, $p<0.001$) thus providing better quality of initial airway as compared to CLMA in paralyzed children. The improved seal of PLMA is due to its modified cuff which is proximally wider and distally larger enabling it to fit snugly in the proximal and distal pharynx and makes an efficient seal. Also, the parallel arrangement of the airway and drain tube causes the tongue to fall more efficiently over the wider proximal cuff. Our results are in agreement to the results of Kanthed et al[10] who found the OLP for PLMA and CLMA to be 18.72 ± 3.28 cmH_2O and 15.84 ± 2.94 cmH_2O respectively, revealing a significant difference. Similar results were found in study by Goldmann et al who reported OLP for size 1.5 PLMA to be significantly higher than that of CLMA in all positions of the head viz. neutral (26.7 ± 7.8 cmH_2O / 18.9 ± 4.8 cmH_2O), maximum flexion(35.6 ±5.4 cmH_2O and 28.2 ± 7.0 cmH_2O) and maximum extension (17.5 ± 7.5 cmH_2O and 15.2 ±5.5 cmH_2O).[7] Our results are in agreement with those of Goldmann et al and Lardner et al who compared size 2 PLMA/CLMA in children receiving general anaesthesia.[11,12].

The rate of successful placement of PLMA/CLMA was similar (100% in two attempts) in both the groups due to use of standardized insertion techniques.[6] Though the difference in time taken for successful placement of both devices was statistically significant, it was clinically not significant as it did not have much effect on the oxygenation status of the patients. This is in contrast to studies done by Lopez et al[13] and Kanthed et al,[10] where the time taken to obtain an effective airway was similar for the two devices. The difference in our results may be attributed to the fact that the total insertion time also included the time required for the removal of introducer tool of PLMA, hence slightly longer.

Ease of insertion of airway device was found to be similar in two groups. Similar results were obtained by a study conducted by Shimbori et al in children, who showed that ease of insertion of PLMA and CLMA was similar in two groups.[14] This is in contrast to study conducted in adult patients by Brimacombe et al which showed that PLMA placement is more difficult than CLMA, this is presumed to be due to larger cuff of PLMA which impedes its digital intraoral positioning and propulsion into pharynx.[15] Also the presence of dorsal cuff in adult PLMAs makes the cuff to fold over during insertion. On the contrary, in paediatric PLMA there is absence of dorsal cuff, hence when deflated there are no folds and parallel arrangement of airway and drain tube prevents its rotation during insertion, thus making its insertion easy.

Our study showed no significant difference between fibreoptic grading amongst 2 groups and none of the patients had inadequate ventilation suggesting that even if anatomic placement is not perfect it still provides satisfactory ventilation.[16,17] Similar results have been reported by other workers in children.[5,10,11,14]

There was no case of bronchospasm, desaturation and tongue, lip or dental trauma in our study. Absence of the above complications might have been due to the fact that strict selection criteria were followed before enrolling the children for study. An adequate depth of anaesthesia was ensured before attempting insertion, limiting the number of attempts used, and standardized techniques of insertion were followed.

None of the patient had cough, postoperative sore throat or hoarseness; it might be due to adequate lubrication of the device, lesser number of attempts required for successful placement of the device and limiting the cuff pressure to 60 cmH_2O.

There are certain limitations in our study; firstly the study is applicable to patients with a normal airway and

whether the same outcome can be extrapolated to patients with anticipated difficult airway needs evaluation. Secondly, the blinding was not possible which could cause biased interpretations. Also three different sizes of masks were not compared separately with each other.

CONCLUSION

Results of the present study showed that there is high reliability of greater OSP, adequate ventilation and oxygenation with lesser frequency of intraoperative complications associated with PLMA in anaesthetized paralysed children. It is as easy to insert as CLMA in the paediatric patients and provides better quality of airway. It might have important implications for the use of PLMA as a better alternative to CLMA for the use of PPV in children.

Authors report no conflicts of interest and no ethical issues involved

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The Green House Effects of Anaesthesia

Deepak Thapa
Professor,
Department of Anaesthesia and Intensive Care, Government Medical and Hospital, Sector 32, Chandigarh

ABSTRACT

In last few decades, use of inhalational anesthetic gases has increased significantly due to the introduction of newer inhalational agents with inherent benefits and safety. In recent years, there has been an increasing awareness about their role in the global climate change and the environment. As anaesthesiologists, we have a duty towards our future generations to minimize operation room pollution, by employing techniques which can minimize the untoward effects of these gases on the environment. Also, we should ensure adequate maintenance of anaesthesia delivery systems as well as scavenging systems which can promote a relatively safe and healthy environment.

Keywords: Inhalational anesthetics, Environmental pollution, Greenhouse effect.

INTRODUCTION

Health hazards in relation to climatic variations have become significant challenges in the present century. Global warming (GW) with greenhouse gases (GHG) is an example and industries (including the health industry) should strive for reducing GHG emissions and prevent rise in global temperature more than 2^0C.[1]

What are greenhouse gases?

GHG are those gases in atmosphere that have capacity to absorb infrared radiation (IR) and production of heat.[2] These include water vapours, carbon dioxide, nitrous oxide, halogenated fluorocarbons, ozone, and other fluorocarbons including perfluorocarbons.[2] All inhalational agents (IA) absorb IR.[3]

Extent of the problem

According to the World Health Organization, the climate-related mortality accounts for 0.3% of all annual deaths.[4] An increased incidence of asthma, heart diseases has been feared due to emission of gases from various industrial plants.[4]

Contribution from health industry

Total greenhouse effects produced by an individual, goods, activity or an arrangement is known as carbon foot printing.[5] The assessment of carbon footprints in healthcare industry is still in the initial stages.[1] Health industries in US contribute 8–10% of all GHG emissions.[1] Carbon footprint of surgery in UK, Canada and USA studied is approximately 9·7 million tonnes of CO_2 equivalents per year.[1] The energy consumption in operation theatres exhibits six times difference than anywhere in the hospital.[1] The largest contributions to greenhouse gas effects from operation theatres are from use of anaesthetic gases (Desflurane, isoflurane, isoflurane etc.) and from consumption of energy in lighting, ventilation, cooling, and heating. Increasing use of desflurane has been attributed as one of the significant contributors to greenhouse effects.[1]

Composition of atmosphere and mechanism of greenhouse warming

The earth atmosphere is made up of troposphere (up to 10,000 meters above earth), stratosphere (10,000 – 50,000 meters) and mesosphere beyond 50,000 meters.[5] The junction between troposphere and stratosphere is known as tropopause.[5] The tropopause is present at around 8000 meters from earth in polar regions and about 18000 meters at the equator. Around 80% of atmospheric ozone is present in Stratosphere while GHG are present in Tropopause (figure 1).[5]

The height of tropopause is determined with observed temperature changes with changes in height. The temperature changes with height are different in troposphere and stratosphere.[5] There is decrease in temperature with increasing height in troposphere whereas in stratosphere the temperature increases with increasing height. Whereas the temperature decreases

Corresponding Author :

Dr Deepak Thapa, Professor,
Department of Anaesthesia and Intensive Care,
Government Medical and Hospital, Chandigarh.
Email: dpkthapa@gmail.com

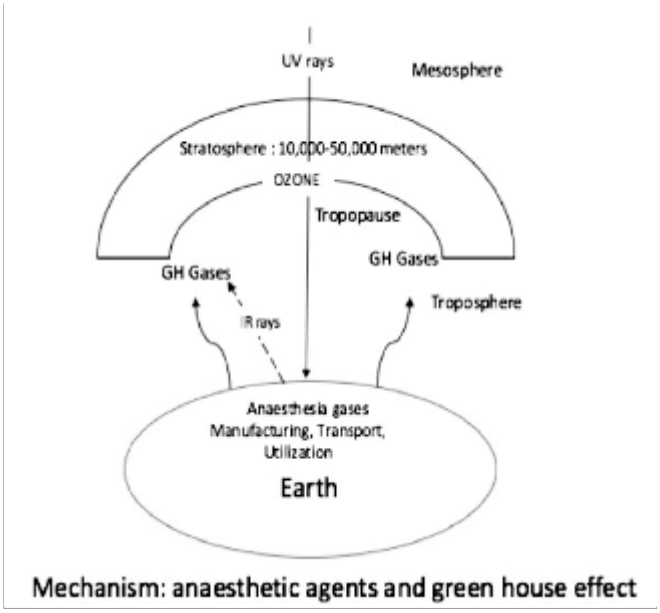

Fig.1: Mechanism: Anaesthetic agents and green house effects

with ascent through the troposphere, it increases further with ascent through the stratosphere.[5]

Mechanism of global warming

Greenhouse gases like carbon dioxide, inhaled anaesthetics, water vapours, fluorocarbons have the property of absorbing infrared radiation and generation of heat. The earth temperature has increased by nearly one degree centigrade over a century.[2] Normally, the ozone in stratosphere, filters sun radiations and prevent them from reaching the earth.[5] The incoming solar radiations are equally balanced and some are reflected back as reflected infrared radiations. Photolysis of chlorine containing compounds leads to generation of chloride atoms and each one of these have the potential to produce catalytic destruction of 100,000 molecules of ozone.[6]

Contribution of Anaesthesia to global warming

Gases used in general anaesthesia have the potential for global warming. There are primarily three sources of greenhouse emissions from operation rooms. These are anaesthetic gases, energy consumption as electricity and lastly, through the supply chain and waste disposal of material.[1] Inhalational anaesthetics are well recognized GHG. Inhaled anaesthetic agents absorb infrared radiations and thus may have GW potential.[6] Anaesthetic agents with longer life in atmosphere have more potential to reach up to stratosphere and destroy ozone.[5]

Sevoflurane has shortest life in atmosphere thus has the smallest global warming effects as compared to desflurane with longest lifetime with greatest global warming effect amongst inhalational agents. The lifetime of sevoflurane, desflurane and isoflurane in troposphere is approximately 1.1, 14 and 3.2 years respectively. One bottle of desflurane can have similar global warming effect as 886 kilogram of CO_2.[5]

Extent of problem related to global warming from use of inhalation anaesthetics

Globally, the warming effect of all inhalation agents per year has been calculated as an equivalent to one coal operated power station.[5] In other words, it has also been estimated as 0.01% of the CO_2 released from global consumption of fossil fuel.[5] The National Health Service in the United Kingdom attributed anaesthetic agents to be responsible for 5% of the CO_2e from some of the organisations.[5] Nitrous oxide is responsible for 90% of the effects. Two-thirds of these are from the use of Entonox which is used mostly in obstetrics.[5] Photolytic destruction of nitrous oxide (N_2O) can produce oxygen atoms which can react with ozone to generate oxygen molecules. Out of total N_2O emissions, the contribution of medical N_2O comprises <4%.[5] The global warming potential of agents like sevoflurane isoflurane and desflurane are much higher than CO_2.[6]

Another factor predisposing the anaesthetic agents to global warming is their minimal metabolism in body after their administration. Anaesthetic agents are minimally metabolized in body and are largely exhaled unchanged. Most anaesthetic vapours added to the anaesthesia circuit are finally released to the environment and contribute to global warming.[5]

Use of Entonox (including obstetric use) is the most common cause of hospital emission of N_2O.

Certain anesthetic events like leakage during mask ventilation, use of open circuits, wastage from inadvertently opened flowmeters, liquid IA spillage during filling, gas used for flushing anesthesia circuits, inappropriate use of airway devices can be source of environmental pollution and can be easily controlled.[7] Other anaesthetic sources of GHG include items for incineration like disposables and pharmaceutical wastages like propofol, industrial gas wastages and energy required for transportation with diesel trucks.[4]

Overcoming problems with GHG emissions

Montreal Protocol was a landmark judgement for the protection of stratosphere in 1987 in which the use of CFCs was universally banned.[5] Subsequently, Kyoto Protocol in 1997 also suggested reduction in emission of GHG. These restricted gases include CO_2, nitrous oxide, methane and sulpha hexafluoride, HFCs, and perfluorocarbons. Regulatory measures must be in place for ensuring reduced emission and scavenging of anesthetic gases in OR.[5]

It has been observed that use of open or semi-closed systems for anesthesia cause more theatre pollution and use of closed or circle systems with appropriate gas flow rates could be an attractive choice.[5] Similarly, ensuring effective seals with airway devices, preference for cuffed endotracheal tube intubations and avoidance of disconnections between anesthetic circuit and machine are supplemental measures to reduce theatre pollution.[5] Cutting down on the use of disposables, reusing of various devices (like LMA) and promoting recycling of products (avoiding incineration) could also help in protection of environmental from health industry. Steps taken for the prevention of wastages during production stage, transportation or during clinical use of anaesthetic gases could be rewarding to environmental sanity.[5]

Total intravenous anaesthesia has potential to avoid anaesthetic gas emission. However, propofol's use and disposal techniques (use of energy for driving syringe pumps, incineration of syringes and of the drug at 1000° C) may contribute to global warming.[5]

Role of regional techniques

Use of regional anaesthetic techniques can provide an attractive alternative to general anaesthesia by eliminating the use of anaesthetic gases and preventing subsequent atmospheric pollution. Use of regional anaesthesia is known to have 'MAC (Minimum Alveolar Concentration) sparing' effect when used in combination with general anaesthesia.[5]

Future measures

Anaesthetic gas condensers have the ability to capture and prevent emission of anaesthetic agents to atmosphere. The condensing devices may completely prevent leakage of anaesthetic agents to atmosphere.[5] Theoretically, various components can be separated by a process of fractional distillation for subsequent administration. Another way of preventing pollution of atmosphere from N_2O is to catalytically convert it into nitrogen and oxygen.[5]

In conclusion, though the contribution of anaesthesia to global warming is less but it is increasing. It is better to take steps to contain it rather than ignoring the extent of damage.

Use of regional anaesthetic techniques could see an enhanced role for the protection of environment in the future. For reduction of green-house effects arising out of using various products in operation theatre in relation to anaesthesia the dictum “reduce, recycle and reuse” still holds true.

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Persistent Post-Cholecystectomy Syndrome Secondary to Cystic Duct Stump Calculus - A Case Report

Sahajdeep Singh Sohal[1], Sushma Devi Bhardwaj[2], Simrandeep Singh[2], Robin Kaushik[3]

[1]Postgraduate Studentt, [2]Assistant Professor, [3]Professor

Department of General Surgery, Government Medical College and Hospital, Chandigarh, India

ABSTRACT

Cholecystectomy is the preferred surgical treatment for cholelithiasis. However occasionally, patients continue to have symptoms even after cholecystectomy. This is now a well-defined clinical entity that is labelled as 'post-cholecystectomy syndrome' (PCS). We report the case of a 42 year old female who underwent open cholecystectomy 17 years ago, and presented to us with dyspepsia and upper abdominal discomfort, especially after meals (her symptoms started within six months of cholecystectomy). Investigations revealed a calculus within a dilated cystic duct stump for which she underwent surgery and excision. The patient remains well, nearly 6 month after her operation.

Keywords: cholecystectomy, choledocholithiasis, duodenal ulcer

INTRODUCTION

Presence of any symptoms at any time after cholecystectomy is called Post-cholecystectomy syndrome (PCS) The wide spectrum of symptoms as well as an unlimited timing thus assigns a very high incidence to PCS.[1] In existing literature, the incidence of PCS ranges from as low as 5% to as high as 63%; it has been documented to occur even 25 years after cholecystectomy.[2] The symptoms can be any, but most commonly include nausea, vomiting, gas bloating, jaundice, diarrhoea, constipation and / or abdominal pain; these can be secondary to a wide range of diseases, not necessarily limited to the biliary tract.[3] It is common to divide the etiology of PCS into biliary and non-biliary causes, which occur with almost equal frequency - the commonest biliary cause is ductal calculi, whereas the commonest non-biliary cause is gastroduodenal ulcers.[4]

Cystic duct stump calculi are an extremely uncommon cause of PCS, with a reported incidence of only 2.5%.[1] Most patients with cystic stump calculi present with recurrence of symptoms within 2 years of surgery, and once diagnosed, have relief after excision of the cystic duct stump containing the calculus.[3] We report one such case of PCS due to cystic duct stump calculus who presented to our hospital after 17 years, and underwent surgery uneventfully.

Corresponding Author :

Dr. Robin Kaushik,
Department of General Surgery
Government Medical College and Hospital
Sector 32, Chandigarh (160 030) India
Email: robinkaushik@yahoo.com

CASE REPORT

A 42 years old female presented to our outpatient department with complaints of pain in the right hypochondrium and dyspepsia for more than one and a half decade. She had undergone open cholecystectomy 17 years back, and had had recurrence of symptoms within six months of surgery. She had learnt to live with the symptoms, but since there had been multiple episodes of pain in the right hypochondrium within the past year, she had again started seeking opinion for her condition. An ultrasound of the abdomen done outside revealed a dilated common bile duct (CBD) with a cystic structure in the gallbladder fossa. Based on this report, we carried out a magnetic resonance cholangio-pancreaticography (MRCP) which clearly revealed a calculus within a long, dilated cystic stump; though the CBD was dilated, there were no stones within it. Her routine investigations (including liver function tests) were normal, and she was planned for elective surgery.

Surgery was performed through previous right subcostal incision. There were adhesions of the omentum, colon and duodenum to the gallbladder fossa which were cleared using a combination of blunt and sharp

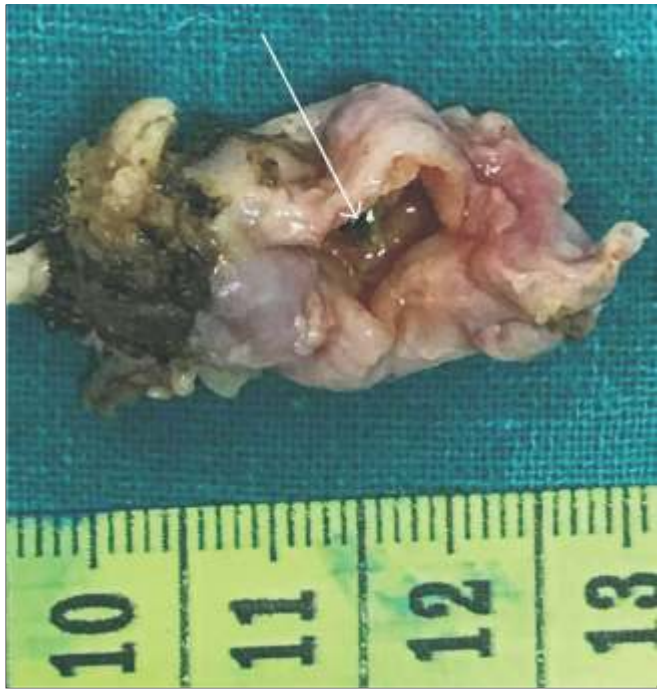

Fig. 1: Resected cystic duct stump showing thick walls and stone within it (white arrow)

dissection until the CBD was visualised; this was followed upwards to where the cystic duct joined it; the cystic duct was elongated, distended at its end, with thick walls and a palpable stone within it. Excision of this was done after ligating the cystic duct just a few millimetres away from the CBD (Figure 1).

The post-operative period was uneventful and the patient is well on a follow up of almost 6 months, with no recurrence of symptoms. The histopathology report of the excised tissue revealed multiple concretions, mild inflammatory infiltrate, as well as multiple foci of previous suture material surrounded by dense fibrosis.

DISCUSSION

A long cystic duct stump is defined as a duct remnant more than 1 cm long, with or without calculi, a long cystic stump is common after laparoscopic cholecystectomy because of its inherent safety mechanism to keep dissection away from the common bile duct and to stay as close to the gall bladder–cystic duct junction as possible.[5] Other causes may be low long parallel insertion of the CBD or in patients with cirrhosis, portal hypertension and difficult gallbladders, with a frozen Calot's triangle anatomy where partial cholecystectomy may be resorted to by preserving a thin rim of the Hartman's pouch that may be sutured close after removing any stones and cauterising its mucosa.[5]

The natural history of remnant cystic duct is unknown; patients may remain asymptomatic throughout life, or may become symptomatic at any period following surgery. In normal circumstances, the terminal end of the cystic duct resembles a 'leaf bud' on ultrasound, but is described as a 'flashlight bulb' when it is dilated pathologically; dilatation of the stump more than 5 mm is also considered abnormal, and such remnants may be associated with infection, calculi and chronic inflammation.[6] Malignancy of the stump is extremely uncommon.

The diagnosis of cystic stump stone is difficult, but can be made by abdominal ultrasonography, computerised tomography scan (CT), endoscopic retrograde cholangiopancreaticography (ERCP) or MRCP.[2] It is often difficult to differentiate between remnant gall bladder stump and long dilated residual cystic duct, and these investigations may show a long, dilated cystic stump with calculi, gravel, microliths or sludge within it, wall thickening, enhancement or adjacent fat stranding suggesting the diagnosis. MRCP is considered to be more accurate than ultrasound, and it also has the advantage of providing better delineation of biliary anatomy.[2]

Once diagnosed, stones within the cystic duct remnant need to be tackled in order to relieve symptoms and avoid potential complications such as recurrent obstructive jaundice, pancreatitis, cholangitis and the risk of cancer.[7,8] Although a few authors have reported ERCP as the first line of treatment, cystic duct stones are usually difficult to remove by ERCP either due to size, difficult position and impaction of stone, diameter of cystic duct, angle of insertion of cystic duct into CBD as well as patency of the cystic duct; however, ERCP is useful in clearing the CBD prior to surgery.[2] Completion cholecystectomy, in which the cystic duct and residual gallbladder are removed, (either by open surgery or laparoscopically) is often the final resort for these patients.[2,5,8] The residual gall bladder or the cystic stump are usually surrounded by dense inflammatory tissue making dissection difficult, and care must be taken during redo surgery to avoid injury to the CBD, portal vein, hepatic artery, duodenum and other surrounding structures. Successful surgery is followed by resolution of symptoms.

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Nonrecurrent Laryngeal Nerve: An Important Clinical Entity

Manish Gupta[1], Ginni Datta[2]

[1]Professor, [2]Associate Professor

Department of ENT, Maharishi Markandeshwar Institute of Medical Sciences & Research, MMU, Ambala.

ABSTRACT

Thyroid gland related nonrecurrent laryngeal nerve is encountered as an uncommon anatomical exception to its commoner cousin- the recurrent laryngeal nerve. It is crucial that the head and neck surgeon be familiar with it in order to avert postoperative morbidity. We hereby report a case of right sided nonrecurrent laryngeal nerve, an uncommon incidental finding on routine hemithyroidectomy for benign thyroid disease.

Keywords: Nonrecurrent laryngeal nerve; thyroid surgery; hemithyroidectomy; vagus nerve

INTRODUCTION

Thyroid surgery for both benign and malignant pathology is frequent in India and southern region of Asia.[1] Hemorrhage, hypoparathyroidism and recurrent laryngeal nerve (RLN) injury are collectively responsible for roughly 50% of all the complications of thyroid surgery.[2] RLN injury during thyroidectomy, even though rare, can threaten the patient's quality of life.[3] With unilateral RLN damage, hoarseness occurs, while bilateral injury results in severe dyspnea due to glottal obstruction.[4] Only way to ascertain the integrity of RLN is by recognize it and preserve it during all surgical manipulations on thyroid.

The RLN normally branches from vagus nerve looping underneath the subclavian artery on right side and below ligamentum arteriosum on the left side, before finally entering the larynx and supplying all the intrinsic muscles of larynx excluding the cricothyroid muscle.[5] Besides anatomy, the precise understanding of common variations of the nerve especially the nonrecurrent variant is indispensable to reduce the threat of injury during intraoperative period. As the nonrecurrent laryngeal nerve (NRLN) is more challenging to identify, it is more prone to injury.[6]

Corresponding Author :

Dr. Manish Gupta
1156-C, Govt. Medical College & Hospital Campus,
Sector 32-B, Chandigarh- 160030, India.
Mobile : 09915025819
Email: manishgupta1217@gmail.com

We hereby report a case of colloid nodule of right thyroid gland that was taken up for right hemithyroidectomy and we encountered the right NRLN on operating table.

CASE REPORT

A 30 year old female presented with a 5cm x 4cm sized painless, progressive and cystic swelling in the anterior lower neck for last 3 years. The swelling was moving with deglutition. Indirect laryngoscopy was normal with bilateral mobile vocal cords. Oropharyngeal examination and routine blood investigations were within normal limits. The patient was euthyroid on

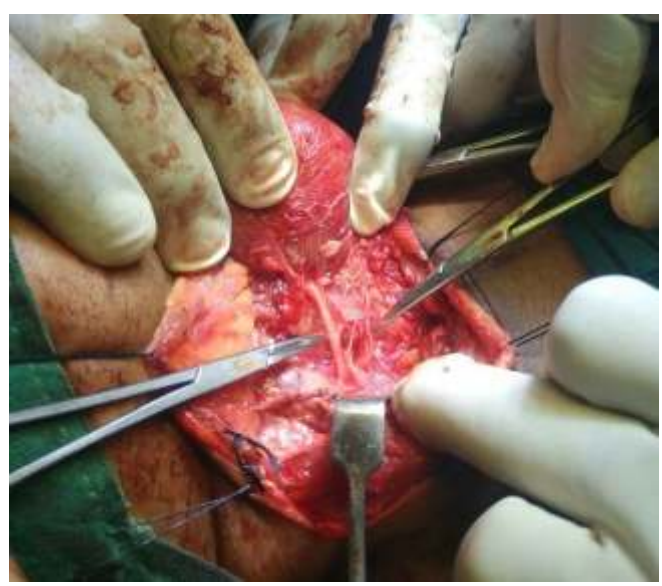

Fig.1: Intraoperative clinical picture showing right thyroid lobe being lifted, over trachea and left side artery forceps indicating right nonrecurrent laryngeal nerve lying parallel and cranial to inferior thyroid artery, marked by right side artery forceps.

thyroid function tests. Ultrasound revealed cystic degeneration of thyroid nodule and fine needle aspiration cytology from the nodule confirmed it to be a case of colloid goiter.

The patient was taken up for right hemithyroidectomy under general anaesthesia. Middle thyroid vein and superior thyroid pedicle were identified and ligated after opening the paracarotid gutter. While working near the inferior pedicle and trying to delineate inferior thyroid artery and RLN, it was found that there was no nerve identifiable running lateral to medial in the tracheoesophageal groove, or forming one of the borders of Beahr's triangle. On further dissection, there was direct branch from vagus, running parallel and cranial to inferior thyroid artery at the level of isthmus, running medially into cricothyroid membrane, i.e NRLN (Fig 1). It was graded as NRLN type IB.

DISCUSSION

A NRLN is a rare anatomical variant. Steadman was the earliest to demonstrate a NRLN in a cadaver in 1823, who also noted a simultaneous anomaly of the right subclavian artery in its origin and course.[7] Henry et al described an occurrence of 0.54% on the right side (17 cases in 3098) and 0.07% on the left (2 cases in 2846).[8]

The general agreement on this anomaly is the existence of a vascular defect named arteria lusoria where the fourth right aortic arch gets absorbed so that the vessel fails to pull the right RLN caudally during the process of cardiac descent and neck elongation through embryonic growth.[6] Preoperative diagnosis of a NRLN is particularly tricky without contrast enhanced computed tomogram (CECT) of the chest. The suspicion of NRLN arises on CECT, which reveals nonexistence of the brachicephalic trunk and an aberrant (largely retroesophageal) path of the right subclavian artery originating from the aortic arch.[9] Left NRLN is either related with situs inversus or loss of ductus arteriosus.[10] However, we did not subject the patient to CECT chest either preoperatively or postoperatively due to monetary constraints.

NRLN can assume either one of 3 variations described. In type IA, the nerve runs a direct course corresponding to superior thyroid pedicle. In type IB, which is by far the most common, NRLN paths transversely at the level of isthmus. In type II, NRLN initially descends and then makes an upward loop reaching tracheo-esophageal groove.[11] Ours was Type IB variant.

RLN or NRLN injury may present as hoarseness post operatively. This complication is completely preventable provided the surgeon carefully identifies and traces the nerve path intra-operatively. The nerve may get damaged in discrete manner like partial transaction, traction, rough handling, contusion, crushing, burned with diathermy, clamping and mislaid ligature or compromised blood supply.[12]

Existence of NRLN must be suspected once the nerve is not in its expected location, i.e. below the trunk and branches of the inferior thyroid artery. In these instances, any transversely running structure above the inferior thyroid artery, between carotid sheath and larynx must not be incised without methodical dissection knowing well the anatomical landmarks.[10] Further, in cases with NRLN, vagus trunk is located medial to the common carotid artery, because anatomically, NRLN has a shorter course than RLN and traction on the laryngotracheal axis during thyroidectomy causes medialisation of the vagus.[13]

Laryngeal nerve injuries are higher during thyroidectomies for carcinoma, toxic or recurrent goiter as identification of nerve and hemostasis may become complex, thus need of extra caution in these cases.[14] Ultimately, meticulous dissection is still the most important element in thyroid surgery as even the usage of nerve monitoring device that detects vocal cord movement on stimulation of laryngeal nerve has not decreased the RLN injury rates.[14]

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Septic Pulmonary Embolism Revisited. A Case Report and Points to Ponder

Deepak Aggarwal[1], Jeet Ram Kashyap[2], Varinder Saini[3], Arjun Kumar[4]

[1,2] Associate Professor [3] Professor & Head, [4] PG Resident

[1,3,4] Department of Pulmonary Medicine, [2] Department of Cardiology
Government Medical College & Hospital, Sector-32, Chandigarh, India

ABSTRACT

Septic pulmonary embolism is a common but under-diagnosed condition that mimics bacterial pneumonia and tuberculosis. We describe the successful management of a case of septic pulmonary embolism who was misdiagnosed as tuberculosis. Early suspicion and echocardiography helped to clinch the diagnosis. The case is supported by a concise review highlighting key points regarding its management.

Keywords: septic pulmonary embolism, tuberculosis, pneumonia

INTRODUCTION

Septic pulmonary embolism (SPE) is an uncommon syndrome characterized by embolization of infected thrombi from a primary infectious site into the venous circulation and subsequent implantation into pulmonary vasculature resulting in necrotizing infection of lungs.[1,2] The clinical presentation varies from an insidious illness mimicking lower respiratory tract infection to life threatening sepsis. The present case depicts a subacute presentation of SPE which was misdiagnosed as tuberculosis. In view of fulminant and disseminated nature of SPE, early identification and aggressive therapy is cornerstone to successful outcome.

CASE REPORT

A 59 year old non-diabetic male presented with history of fever with evening rise of temperature for the last 1 month, progressive breathlessness and cough for last 1 week. He was a non-smoker but a chronic abuser of intravenous opioid drugs. He had taken prior antibiotics from local hospital and was later put on anti-tubercular drugs 1 week back. On examination patient was febrile, tachypnoic with decreased breath sounds on right hemithorax. Routine blood examination revealed leucocytosis (TLC 17×10^9/L). Chest radiograph showed bilateral nodular infiltrates with cavity formation and blunting of right costophrenic angle (Fig 1). Diagnostic pleural aspiration revealed exudative, neutrophilic (75%), low ADA (13.1 U/L) picture. Blood, urine, pleural fluid and sputum cultures were sterile and sputum was negative for acid fast bacilli by microscopy. Patient was started on intravenous antibiotics piperacillin plus tazobactum and amikacin and was further evaluated for confirmation of diagnosis. Contrast enhanced computed tomography of thorax showed bilateral, multiple nodules and patchy consolidation some of which showed cavitation along with right pleural effusion and mediastinal lymphadenopathy, suggestive of infective etiology, possibly tuberculosis or septic pulmonary embolism (Fig 2 & 3). With 7 days of treatment, TLC decreased to normal levels but fever persisted.

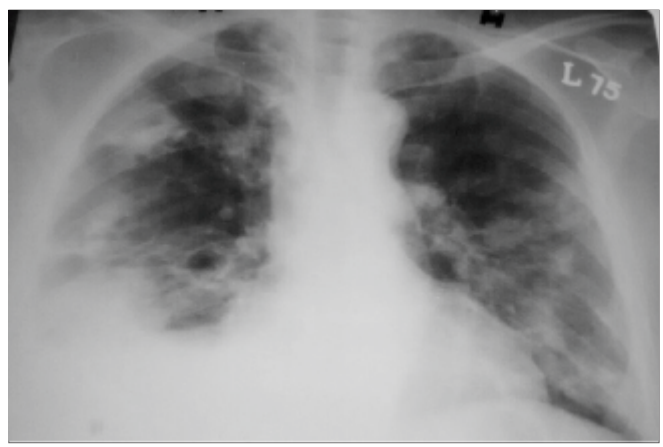
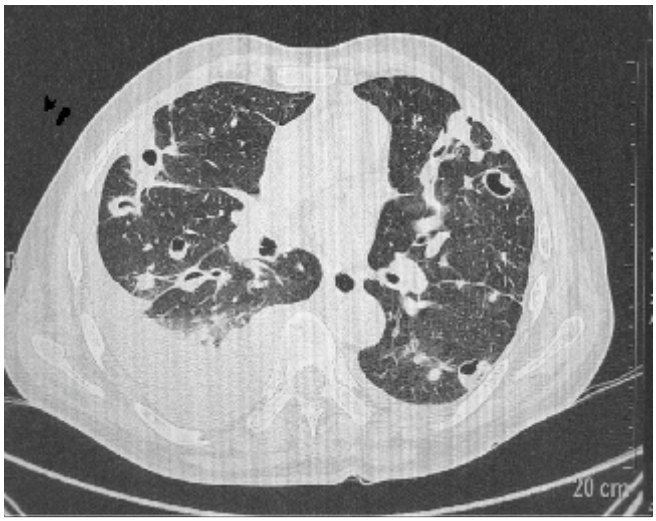


Fig. 1: Plain X-ray P-A view shows bilateral ill-defined cavitating nodules and homogenous opacity in right lower zone with obliteration of the right costophrenic angle

Corresponding Author :

Deepak Aggarwal, Associate Professor,
Dept. of Pulmonary Medicine, Block-D, Level-5,
Government Medical College & Hospital,
Sector-32, Chandigarh. Pin-160030, India
E mail: drdeepak @hotmail.com.
Mobile: 09646121584



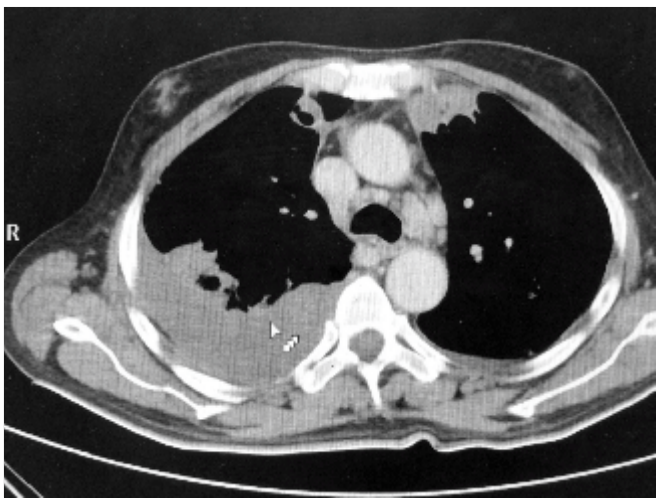


Fig. 2 & 3: Contrast enhanced computed tomography of thorax showing bilateral cavitating nodules, few abutting the chest wall along with right pleural effusion and mediastinal lymphadenopathy

Culture of bronchoalveolar lavage didn't yield any organisms. With a suspicion of septic pulmonary embolism, echocardiography was performed which revealed tricuspid valve vegetations on both anterior and septal leaflets along with dilatation of the right atrium and ventricle (Fig 4). Final diagnosis of right sided infective endocarditis with septic pulmonary embolism was made and patient was started on intravenous vancomycin and gentamycin. Over 10 days, patient's fever gradually improved. The antibiotics were continued for 3 weeks and were later substituted with oral linezolid and cloxacillin. Patient completed 6 weeks of antibiotic therapy with complete recovery.

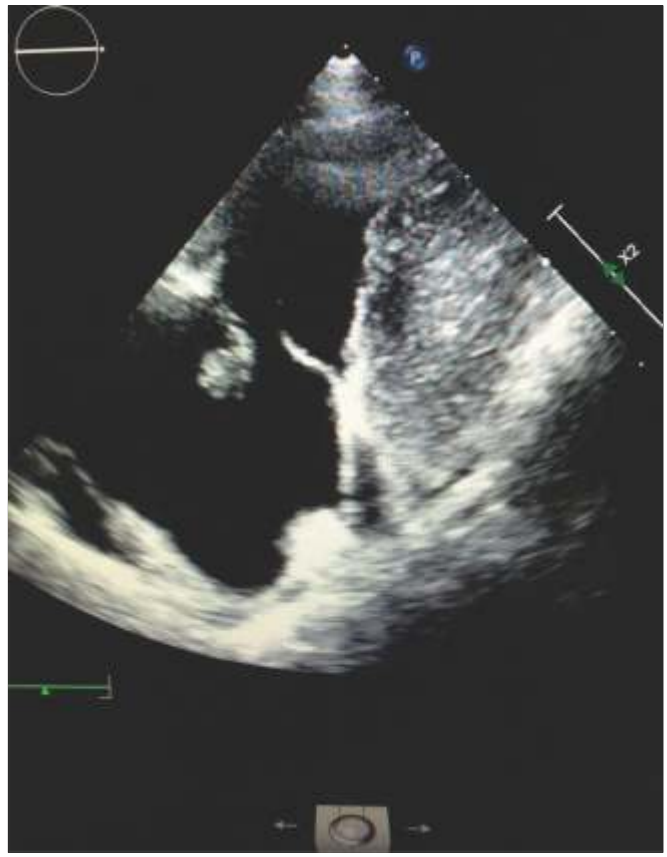


Fig. 4: 2D-Echocardiography showing Tricuspid valve vegetations

DISCUSSION

Septic pulmonary embolism is characterized by multi-focal parenchymal infection caused by embolization of infected thrombi into the pulmonary vasculature. Even though the syndrome is recognized since decades, exact burden is not known. Due to non-specific presentation, varied etiologies and lack of suspicion, it often goes undetected and is treated as tuberculosis or community acquired pneumonia.[2]

The primary focus of infection in septic embolism can be highly variable. Classically, the disease has been seen to occur in intra-venous (IV) drug abusers with right-sided infective endocarditis, in patients with pelvic thrombophlebitis and suppurative infections of head and neck.[1,3-5] Our patient was a known IV drug abuser which was an important diagnostic clue, even though, given their immunocompromised status, other infections are also common in such patients. In the recent times, SPE has become uncommon in IV drug abusers, presumably

due to greater awareness of needle hygiene. Contiguous skin and soft tissue infections, infected indwelling catheters and cardiac devices have become frequent sources of infection.[2,6]

Clinically, the disease has highly protean presentations ranging from insidious onset of cough, fever, chest pain and breathlessness to life threatening condition like septic shock and multi-organ failure.[6-8] Our patient had a sub-acute presentation with evening rise of temperature, cough, worsening breathlessness and right sided chest pain for which he was put on anti-tuberculosis treatment. With lack of suspicion, this non-specific presentation can easily be attributed to tuberculosis in an endemic country. Symptoms pertaining to the extra-pulmonary infection can help in arising the suspicion for SPE. However, our patient was totally asymptomatic from cardiac side, though, he was subjected to echocardiography due to high index of suspicion.

Definitive criteria for the diagnosis of SPE are difficult to formulate since histopathological confirmation is infrequently done in clinical practice. Diagnosis of SPE relies on the typical radiological features supported by evidence of active focus of extra-pulmonary infection and positive blood cultures.[6] Contrast enhanced computed tomography of the chest is more sensitive and specific than plain chest radiograph in detecting the lesions. Typically, SPE presents as multiple, poorly marginated parenchymal nodules with peripheral distribution and a tendency for cavitation, on CT scan.[9,10] However the picture can mimic bacterial pneumonia, fungal pneumonia, tuberculosis and cavitating carcinoma. Microbiological confirmation of infection should be done by blood and sputum cultures as well as culture of sample from extra-pulmonary infectious focus. *Staphylococcal aureus* remains the most likely bacterial pathogen in different studies.[2,6,8] Candida is seen frequently in patients with catheter related infection especially who are immunocompromised.[2] Preceding antibiotic therapy may hamper the microbiological diagnosis as was seen in our patient. Identifying the nidus of infection using echocardiography or other relevant investigations is highly recommended for accurate diagnosis as well as timely treatment.

Early empiric antibiotic therapy is the main stay of treatment. It may be modified in light of culture results and continued for 4-9 weeks.[2,6] The management should invariably include amelioration of the infective source or device. Surgical management in the form of thoracocentesis or chest tube placement may be required in cases presenting with empyema or pyopneumothorax.

The condition develops due to two insults to the lung tissue i.e. infection and infarction. Hence it usually carries a poor prognosis than other pulmonary infections. The present case highlights need of high index of suspicion for timely detection of SPE. Pertinent clinical features and imaging results should always be evaluated further by echocardiography and/or other investigations for identification of potential sources of infection. Early diagnosis, appropriate anti-microbial therapy and control of infectious source are hallmark of successful outcome.

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Delayed Onset of Sevoflurane Induced Malignant Hyperthermia in a child with Osteogenesis Imperfecta – An unusual entity

Puja Saxena[1], Uma Rathi[1], Satinder Gombar[2], Parmanand Gupta[3], Chandrika Azad[4]

[1]Assistant Professor, [2,3]Professor, [4]Associate Professor

Department of Anaesthesiology and Intensive Care[1,2], Orthopedics[3], and Pediatrics[4]
Government Medical College and Hospital, Sector 32, Chandigarh -160030, India

ABSTRACT

Malignant hyperthermia (MH) is a rare pharmacogenetic disorder of skeletal muscle calcium regulation, consisting of hypermetabolic response to volatile anaesthetic agents (like halothane, sevoflurane, and desflurane), and succinylcholine. It is seen more commonly in males than females (2:1). The classical signs of malignant hyperthermia are marked rise in body temperature, tachycardia, tachypnea, hypercarbia, increased oxygen consumption, acidosis, muscle rigidity and rhabdomyolysis. In this case report, we present a case of rarely-seen delayed onset malignant hyperthermia-like reaction after the second exposure to sevoflurane in a 2 year old male child with Osteogenesis imperfecta (OI), scheduled to undergo corrective osteotomy under general anaesthesia. Malignant hyperthermia was probably induced by sevoflurane, an inhalation agent of low-inducing probability, despite no prior complication to previous exposure. By virtue of the same, we wish to emphasize the importance of detailed pre-anaesthetic evaluation and preparation before anaesthetizing such patients despite prior uneventful exposure. The anaesthesiologist should be able to recognize the possibility of an atypical MH and be alert for the possible occurrence of MH in susceptible patients.

Keywords : Malignant Hyperthermia, Osteogenesis Imperfecta, Inhalational agent, Sevoflurane

INTRODUCTION

Malignant hyperthermia (MH) is an inherited condition manifested during general anaesthesia as a progressive increase in body temperature, associated with hypoxaemia, hypercapnia, metabolic acidosis, muscle rigidity and rhabdomyolysis. MH is characterized by a hypermetabolic response to various triggering agents like halothane, sevoflurane, and desflurane.[1] In this case report, we present delayed onset malignant hyperthermia- like reaction after the second exposure to sevoflurane in a 2 year old boy of Osteogenesis Imperfecta (OI).

CASE REPORT

A 2 year old male child weighing 13 kilograms, a diagnosed case of OI, with underlying abnormalities like multiple contractures, dimorphic facial features, moderate atrial septal defect (ASD), hypermobile joints and ambiguous genitalia was scheduled to undergo revision corrective osteotomy of bilateral lower limbs under general anaesthesia. Patient had been investigated for other associated congenital disorders and was cleared by the cardiologist under low to moderate risk in view of moderate ASD for which no intervention was done. As per history given by the parents, the antenatal and immediate postnatal period was uneventful. There was no history of delay in milestones. The patient underwent similar surgery 6 months back under general anaesthesia which was uneventful. Preoperative systemic examination and laboratory work up was unremarkable. Airway assessment was within normal limits. The previous medical records of the patient revealed that prior anaesthetic exposure to inhalational agents (sevoflurane) was without any complications. Anaesthesia was induced with fentanyl 2µg/kg, and propofol 2 mg/kg intravenous (IV). After checking for ability to mask ventilate, atracurium 0.5 mg/kg IV was administered followed by endotracheal intubation.

Corresponding Author :

Dr Puja Saxena, Assistant Professor,
Department of Anaesthesia and Intensive Care
Government Medical College and Hospital, Sector 32
Chandigarh -160030, India
Email : puja.saxena36@gmail.com

Anaesthesia was maintained with sevoflurane and nitrous oxide in oxygen. The intra-operative course was uneventful. After completion of surgery, trachea was extubated uneventfully and the patient was observed in Post Anesthesia Care Unit for one hour and subsequently shifted to the ward in a stable condition.

After almost 4 hours of postoperative period, the nursing personnel notified a sudden rise in body temperature ~38ºC, which did not respond to IV paracetamol, as well as aggressive hydrotherapy. Patient had persistent rise in body temperature (~40ºC) along with tachycardia (pulse rate-150 -160/min) and tachypnoea (respiratory rate- 40/min). Blood gas analysis revealed hypoxemia (SpO_2 84%), respiratory acidosis ($PaCO_2$ 70mmHg) and metabolic acidosis (HCO_3 12mEq/L). In view of the ensuing respiratory distress, child was immediately intubated and shifted to Paediatric Intensive Care Unit for further ventilatory management. Aggressive intravenous hydration was also started. Suspecting it to be a delayed manifestation of MH, symptomatic treatment including positive pressure ventilation with 100% oxygen, hydrotherapy along with intravenous hydration was continued. Unfortunately dantrolene could not be administered due to its non availability in our setup. Patient continued to deteriorate and developed refractory hypotension (unresponsive to vasopressors and inotropes), persistent high grade fever (~40.2 ^{O}C), hemodynamic instability, severe metabolic acidosis, cola coloured urine secondary to rhabdomyolysis and ultimately expired despite aggressive resuscitation measures.

DISCUSSION

Malignant hyperthermia can have a variable clinical presentation and delayed onset, making a definitive diagnosis challenging particularly in patients having OI. The triggering agents cause an increase in calcium release in the muscles leading to contracture of the skeletal muscles.[2,3]

In the typical presentation of MH, elevations of temperature can be delayed, therefore the earliest signs of MH include tachycardia, tachypnoea and increased end-tidal carbon dioxide levels.[1] However, in the present case, many of these early signs of MH were not apparent. Although gradual elevation in temperature manifested in this case, but other signs like intra-operative tachycardia, tachypnoea or increased end-tidal CO_2 levels were not evident. Moreover the immediate postoperative period was also uneventful.

Porsberg et al[4] reported that the hypermetabolic state and hyperthermia that develop in patients with OI is different from typical MH. Many cases of hyperthermia in OI are not of the malignant type but the result of hypermetabolic state, the pathogenesis of which is unknown. It has been suggested that hyperthermia in patients with OI is the result of either central nervous temperature dysregulation or abnormal cellular energy metabolism. The difference is the negative contracture test on muscle biopsy in these patients. Diagnosis in the present case could not be confirmed as the parents were not willing for an autopsy. Secondly, the hyperthermia and hypermetabolism in these patients is self limiting, the mechanism of which is still not known and there is no associated generalized rigidity. Brownell et al has also reported that the occurrence of MH in OI is coincidental.[5]

Chen et al[6] have reported malignant hyperthermia in a 5-years old boy after second exposure to sevoflurane. This patient had general anesthesia with sevoflurane for 2 times with an interval of 2 days, however our patient had second exposure after 6 months. Probably volatile agents cause delayed onset of MH due to the latent effect on the skeletal muscles. Certainly, there are several lessons learnt. A high index of suspicion for MH should be kept in patients with associated musculoskeletal deformities. Standard ASA monitoring including central core temperature monitoring and arterial monitoring should be employed. Sevoflurane is thought to be a less potent triggering agent of MH; however, in literature review, the onset of MH after exposure to sevoflurane is associated with delayed calcium release from the sarcoplasmic reticulum leading to delayed manifestation of MH. Changing the anaesthetic plan to non-triggering agents should be considered especially in patients coming for repeated surgeries. Use of Total Intravenous Anaesthesia (TIVA) should become a common practice.[7]

If at all, during anaesthesia, malignant hyperthermia is suspected the anaesthetic is often terminated before severe signs become manifest and provision of specific treatment with IV dantrolene should be put into practice.[4] The diagnosis must then be confirmed at a later date by contracture test of a muscle biopsy. The gold standard for diagnosis of susceptibility to malignant hyperthermia is

caffeine-halothane contracture test. [8] However, as this test is not widely available, the diagnosis of malignant hyperthermia can only be made by clinical presentation. Dantrolene sodium is a specific antagonist of the pathophysiologic changes of malignant hyperthermia and when given early, as per MH protocol, it is lifesaving.[1] Gopalakrishnan et al[9] pointed out the concerns regarding the availability of dantrolene in India. Dantrolene is not marketed in India, probably because of lack of demand from the anaesthesiologists who generally believe that MH is so uncommon in our patient population that the cost cannot be justified. Therefore the anaesthesiologist should remain vigilant in recognising possibility of an atypical MH during routine anaesthetic practice.

CONCLUSION

The unusual clinical appearance of malignant hyperthermia demonstrates the variability of the disease and has heightened the awareness of atypical presentation in our set up. In the preoperative assessment, stress should be laid on obtaining careful personal and family history. Avoidance of triggering agents, use of TIVA, temperature monitoring, cooling devices and availability of intravenous dantrolene are the precautions to be used intra-operatively. Any such episodes should be aggressively dealt with according to malignant hyperthermia protocol until proven otherwise.

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Purpura Fulminans in Granulomatosis with Polyangitis

Saurabh Gaba [1], Monica Gupta[2], Rajpal Singh Punia[3]

[1]Postgraduate student, [2,3] Professor

[1,2]Department of General Medicine, [3] Department of Pathology,

Governmental Medical College and Hospital, Chandigarh, India

ABSTRACT

Purpura fulminans is a catastrophic cutaneous manifestation of systemic disorders of varied etiologies and is generally associated with high fatality rates. We are reporting a case of 36 year old female who presented with purpura fulminans and was found to have multisystem involvement. After detailed work up, a diagnosis of granulomatosis with polyangitis (GPA) was made, which is a very rare cause of pupura fulminans.

Keywords: purpura fulminans, granulomatosis with polyangitis, Wegener's granulomatosis

INTRODUCTION

Purpura fulminans is haemorrhagic necrosis of skin resulting from thrombotic occlusion of small cutaneous blood vessels. The rash, which begins as a rapidly progressive necrotic lesion, grows incessantly and is associated with systemic features of sepsis. It carries a high potential for mortality and prompt recognition of underlying etiology is imperative. Timely treatment directed at the core pathology can improve the outcome. The most common causes include disseminated intravascular coagulation, sepsis, thrombotic thrombocytopenic purpura and vasculitis.[1,2] The patient presented with purpura fulminans and peculiar systemic features which raised suspicion of granulomatosis with polyangitis (GPA). The diagnosis was established after an extensive array of investigations which were also aimed at excluding other possible causes.

CASE REPORT

The patient, a 36 year old female, hailed from a rural background and was engaged in farming. She presented with progressive bluish discolouration of skin on dorsal surface of right foot, and flexor and extensor surface of both arms for last 10 days. The non-pruritic rash, which was initially macular, evolved to involve more extensive areas along with formation of bullae and areas of necrosis (Fig 1). She had been experiencing persistent nasal discharge, cough and dyspnea on exertion for past 4 months. There was no history of hemoptysis, arthralgia, hematuria or oliguria. She had had three uneventful pregnancies, the last one being 5 years back. She had hemoglobin of 9.1 gm/dl (12-16 gm/dl), platelet count of 1.34 lacs/uL (1.5-4.0 lacs/ul), total leucocyte count (TLC) of 4300/uL (4000-12000/uL) with 88% neutrophils and 11% lymphocytes. The erythrocyte sedimentation rate was 88 mm in 1st hour (<20 mm in 1st hour). C- reactive protein was elevated to 132 mg/dL (<10 mg/dl). Serum electrolytes were within the reference range and creatinine was 2.3 mg/dl.

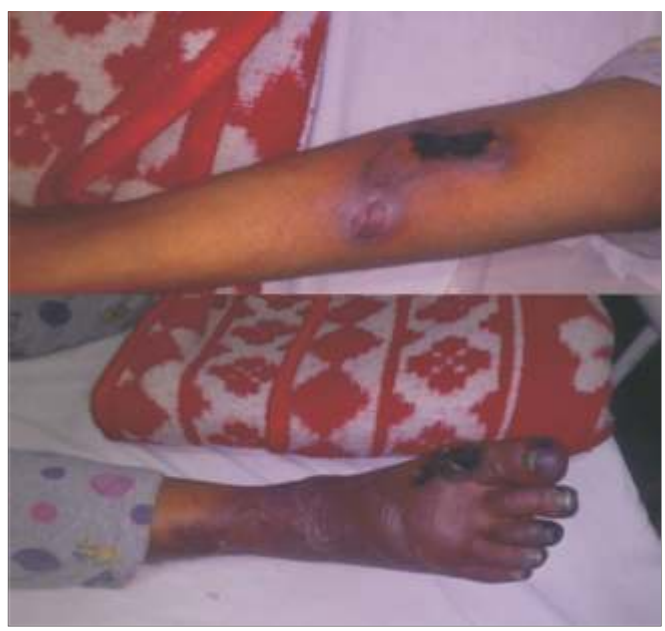

Fig. 1 : Purpura fulminans on arm and foot

Corresponding Author :

Dr Monica Gupta, Professor
Department of General Medicine,
Governmental Medical College and Hospital,
Sector 32, Chandigarh. Email: drmg1156@gmail.com

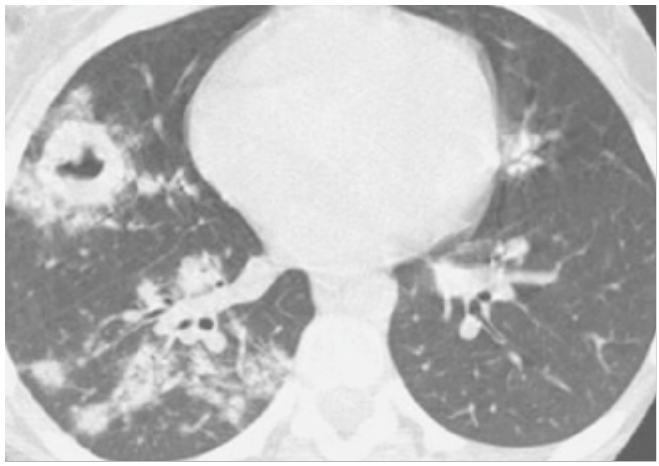

Fig. 2 : Chest CT : Multiple nodules with one showing cavitation on right side.

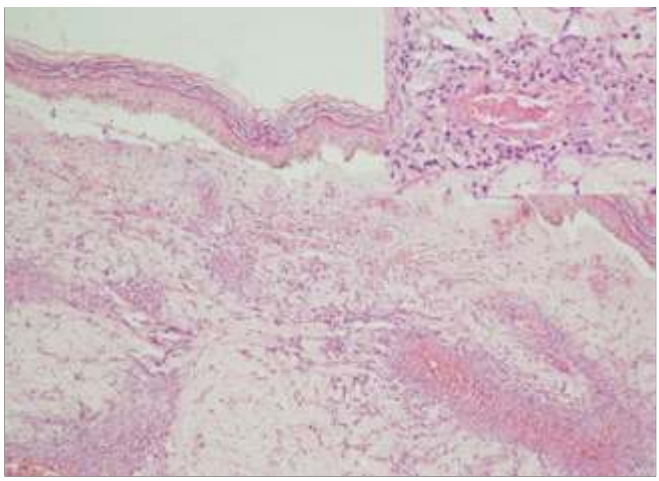

Fig. 3 : Skin Biopsy : Dermoepidermal separation with collection of RBCs and neutrophilic infiltrate destroying vessel wall and reaching deep dermis (H&E x 40) Inset shows vasculitis and fibrinoid necrosis of vessel wall (H&E x 200)

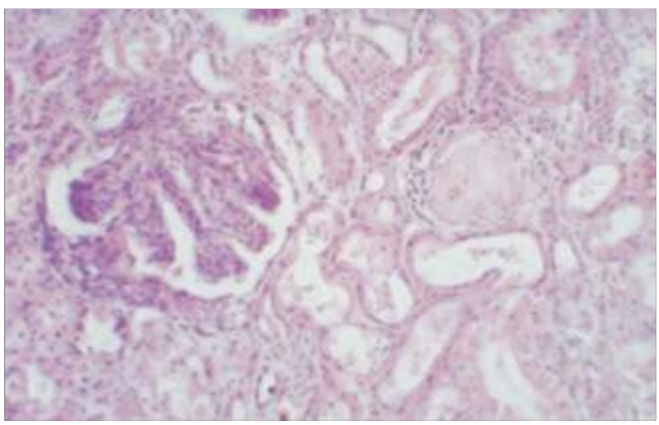

Fig. 4 : Kidney Biopsy : Glomerulus on left showing fibrous crescent

Prothrombin time was 2 seconds above control. Serum aminotransferases were raised to two times the upper limit and albumin was 3.1 mg/dl (3.5-5.5 mg/dl). Urine examination revealed 20-30 red blood cells/high power field and 1+ albumin on dipstick. 24-hour urine protein was found to be 700mg. Serum procalcitonin (PCT) was 0.03 ng/ml (<0.05 ng/ml). On ultrasound, the kidneys were normal sized but had raised cortical echogenicity. Chest CT revealed bilateral nodular lesions, more on right side (Fig 2). No growth was obtained on culture of bronchoalveolar lavage fluid. The serum cytoplasmic antineutrophil cytoplasmic antibodies (**c**-ANCAs, or PR3-ANCA) were strongly positive – 131 U/L (laboratory reference < 20 U/L). Skin biopsy done from a lesion on foot confirmed vasculitic pathology (Fig 3). Renal biopsy revealed presence of crescentic glomerulonephritis (Fig 4). Diagnosis of GPA was made and 500 mg of cyclophosphamide was administered. Intravenous methylprednisolone pulse (1 gm daily) was given for first three days, followed by oral prednisolone 50 mg/day. After 5 days, her creatinine was 2.7 mg/dl and the skin lesions had continued to progress. She developed fever on 7th day and had evidence of sepsis in the form of hypotension and PCT of 8 ng/ml. The TLC was 4800/uL. Parenteral piperacillin-tazobactam and linezolid were started empirically with renal dose modification but the patient continued to deteriorate with progression of skin lesions, renal dysfunction and septic shock. Inotropic support and peritoneal dialysis were initiated, however cardio-pulmonary arrest led to her death on 11th day.

DISCUSSION

The ANCA associated vasculitides include granulomatosis with polyangitis (previously called Wegener's Granulomatosis), eosinophilic granulomatosis with polyangitis (Churg Strauss vasculitis) and microscopic polyangitis.

GPA can occur at any age and does not have predilection for any sex.[3] Presentation is variable, ranging from mild upper respiratory tract symptoms to an explosive, multisystemic illness as in our case. GPA has antibodies directed against proteinase 3 (PR3 or cANCA) in 70% and against myeloperoxidase (p ANCA) in 20% of the cases. 10% are ANCA negative.[4] It is characterized by formation of non caseating granulomas, typically in upper and lower respiratory tract, and necrotizing vasculitis of small vessels. Pauci-immune glomerulone--phritis can take form of rapidly progressive

glomerulonephritis with crescent formation in glomeruli that can lead to end stage renal disease within weeks of onset. The cutaneous manifestations include papules and vesicles, subcutaneous nodules, petechiae, pyoderma gangrenosum and Raynaud's phenomenon.[5] Purpura fulminans is a rare manifestation and portends a bad outcome. Upper respiratory tract involvement occurs in the form of nasal crusting, chronic rhinosinusitis, and nasal discharge, septal perforation leading to saddle deformity and subglottic stenosis. Lower respiratory afflictions manifest as interstitial lung disease, pulmonary haemorrhage and pulmonary nodulosis.[6]

In the present case, excluding an infective etiology was of paramount importance. Inappropriate immuno-suppression leads to flaring up of any infection, while a delay in commencing it, when actually indicated, denies a patient of life saving therapy. Timely treatment was instituted, however, disease progression and a newly acquired infection led to septic shock and patient's demise.

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Ocular Charring from Molten Metal

Rohan Bir Singh[1], Parul Ichhpujani[2], Suresh Kumar[3]

[1]Massachusetts Eye and Ear Infirmary, [2]Associate Professor, [3]Professor

[1]Department of Ophthalmology, Harvard Medical School, Boston, MA 02114

[2,3]Department of Ophthalmology, Government Medical College & Hospital, Chandigarh

ABSTRACT

We report a case of a patient with severe ocular burns due to molten iron while working in a factory. The heat-retaining capacity of the heavy metal, heat of the molten metal at impact and the duration of exposure determine the outcome. Our patient had to undergo an enucleation due to extensive charring of the globe.

Keywords: Ocular Trauma, Eye Injury, Thermal injury

INTRODUCTION

Approximately 34% of all visits to an ophthalmology emergency department are due to traumatic etiologies. The management and visual prognosis depends upon the etiology of the injury, the extent, and the tissue affected by the trauma. The ocular injuries are typically divided into, traumatic or exposure related. The traumatic injuries include ocular surface abrasions and foreign bodies while exposure-related injuries include thermal and chemical burns, and radiation injuries.[1]

Thermal injuries are catastrophic for the eye and require prompt intervention to maintain the anatomical integrity of the globe. The management for such injuries is aimed at suppressing inflammation, accelerating re-epithelialization, and prevent tissues from melting further. Thermal injuries are caused due to exposure of the eye to steam, boiling water, fireworks explosion, and molten metal. The severity of the thermal injury depends upon the agent structure, duration of contact, and contact surface area. Extensive prolonged exposure to the source may result in complete destruction of limbal stem cells, cause conjunctivalization of the cornea, formation of symblepharon, palperbral deformities, and perforation of the cornea and/or sclera.

We report a case of a patient with severe ocular surface, and eyelid burns due to molten iron. The patient's clinical presentation, and management are discussed in detail.

Corresponding Author :

Dr Parul Ichhpujani, Associate Professor,
Department of Opthalmology
Government Medical College & Hospital, Chandigarh
Email: itsdrparul@gmail.com

CASE REPORT

A 48-year-old male, who worked in a steel mill, presented to the emergency room with the complaint of severe ocular pain, and sudden loss of vision in the right eye. The patient reported sudden, painful and severe burning sensation immediately after a “foreign body” landed in his affected eye. The patient's eye was immediately irrigated with saline extensively for about 30 minutes. On initial examination, there was no light perception in his right eye as compared with 20/40 in his left eye. The adnexal examination of the right eye showed excessive palpebral congestion, chemosis and singed eyelashes. The anterior segment examination of the right eye revealed thick, black charred sclera and

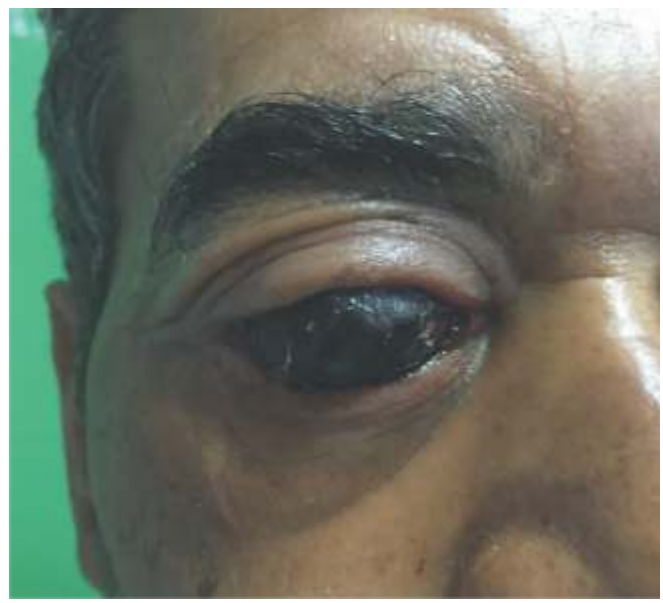

Fig. 1: Scleral and Corneal Eschar following Thermal injury

cornea or eschar; no other details could be made out (Fig 1). Anterior segment and posterior segment examination of left eye was essentially unremarkable.

Since the affected eye was extensively damaged from the thermal burn, and there was no potential of vision restoration, the patient's eye was enucleated.

DISCUSSION

Traumatic eye injuries are the reason for about 20-40% of the visits to an ophthalmology emergency room. Usually the thermal ocular injuries are mild burns, which heal within few days to weeks after adequate treatment with topical antibiotics, steroids and artificial tears. However, moderate and severe thermal burns can lead to necrosis of cornea, conjunctiva, sclera, iris, and even ciliary body due to ischemia, after extensively breaking down the ocular surface epithelium. The eyelid damage results in severe periocular edema, may cause necrosis, and compromise the mechanical barrier for the ocular surface.

In mild to moderate thermal burns, inflammatory mediators released in the ocular surface microenvironment after the injury may lead to subsequent leukocyte and fibroblast invasion, causing corneal opacification. Direct thermal damage to the cornea causes collagen shrinkage, with prominent stress lines radiating away from the area of greatest area of contact with the thermal agent. This shrinkage may cause corneal distortion and opacification, leading to steepening of the axis. Collagen damage may be so severe as to produce a rapidly excavating corneal ulcer originating from liquefactive necrosis. Not much has been reported in literature about post injury phase of thermal ocular injuries.

Goldblatt et al conducted a study to examine the effect of a heat conductor on rabbit's ocular surface.[6] They observed no changes on exposing the eyes to 45°C for 15 minutes. However, increasing the temperature to 59°C for 15 minutes caused diffuse stromal edema, and loss of endothelial cells. Our patient's ocular surface was exposed to molten iron at a very high temperature, approximately 1500°C. The prolonged exposure to such an extremely high temperature caused charring of the sclera, perforated the cornea, thus breaking down the anatomical integrity of the globe.

The lifetime prevalence of self-reported workplace eye injury has been reported to be significantly higher among men (13.5%).[7] This catastrophic outcome could have been averted by the patient, if he was wearing adequate protective eye wear at work. Our case brings forth the need of compliance of safety guidelines by employees at workplaces, and strict enforcement about wearing proper protective eye wear by the employers is required to be implemented to avoid repeat of such incidents.

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Submandibular abscess in a child : Anaesthetic Considerations

Swati Jindal[1], Kompal Jain[2] Satinder Gombar[3] , Tanvi Khera[4]

[1]Assistant Professor, [2]Senior Resident, [3]Professor, [4]Postgraduate Student

Department of Anesthesia and Intensive Care, Government Medical College and Hospital, Chandigarh, India

ABSTRACT

Children with neck abscesses may present in an emergency setting thus catching the physician off guard, wherein the management would depend primarily on the anesthesiologist's experience and available resources. We wish to report the anaesthetic management of an eighteen month old child with submandibular abscess who was scheduled for incision and drainage in emergency.

Keywords: Submandibular abscess, Paediatric anaesthesia

INTRODUCTION

Neck abscess can be an anticipated difficult airway situation, which is further complicated when the patient is a child. Owing to already limited reserves, and the added problems associated with the abscess pus/blood spilling into the airway, the anesthesiologist must be aware of the airway management options before-hand and be prepared to manage worst situations.

CASE REPORT

An 18 months old child, weighing 10 kg came to emergency department of our hospital with a large abscess in the neck. The child had upper respiratory tract infection for two weeks. There was swelling and redness in the submandibular region (Fig 1) with history of decreased oral intake, excessive crying and high grade fever. However there was no stridor or difficulty in breathing. The child was seen by an Otorhinolaryngologist who advised for incision and drainage. Anteroposterior and lateral views of X- ray neck were done which showed a soft tissue swelling in the mandibular area. A plan for an emergency incision and drainage of the abscess was made in view of impending rupture, stridor or risk of aspiration. Child was kept fasting according to the ASA fasting guidelines. The child was not premedicated in view of the anticipated difficult airway. In the operation room, the child was awake, febrile and crying with a toxic look. His heart rate was 150/minute, respiratory rate 30/minute and SpO_2 was 98% on room air. There was no nasal flaring or signs of respiratory distress. On physical examination, the angle of mandible could not be palpated due to extensive swelling, with possible anticipated difficult mask ventilation and intubation. Difficult airway cart was thus prepared preoperatively which included oropharyngeal airways (size 0,1), smaller size endotracheal tubes, LMA (size 1,1.5 both classic and Proseal). C-Mac Videolaryngoscope with small blade and pediatric fiberscope was not available in emergency setting, so emergency surgical tracheostomy was kept as a last resort.

After topical EMLA cream application on dorsum of hand, an intravenous line was secured before induction of anesthesia. Child received 20mg/kg of intravenous paracetamol followed by 1mcg/kg of fentanyl. After preoxygenation for 5 minutes, inhalational induction with sevoflurane was started in increments of 0.5%. Slow increments upto 2% resulted in grunting sounds. But when respiration was assisted, the child could be mask ventilated. A further dose of 10 mcg fentanyl was given. A check laryngoscopy was done which revealed Cormack Lehane grade 3. In view of the possibility of mask ventilation, it was decided to give succinylcholine 1.5 mg/kg IV. After 1 minute, trachea was intubated with 4mmID uncuffed endotracheal tube, and fixed at 13 cm. Bilateral air entry was checked and was equal. Surgery lasted 15 minutes. Trachea was extubated uneventfully.

Corresponding Author :

Dr Swati Jindal, Assistant Professor
Department of Anesthesia and Intensive Care
Government College and Hospital, Chandigarh
India-160030 Email: swatirohit604@gmail.com

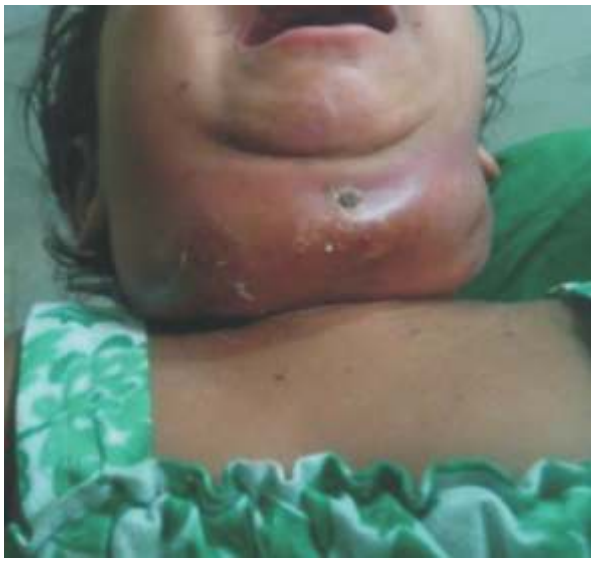

Fig. 1: 18 month child with submandibular abscess sized 74x69mm

Child cried on extubation. Patient was observed in post anesthesia care unit for 3 hours and then sent to ward.

DISCUSSION

Neck abscess, a fulminant cellulitis may initiate even after a simple tooth extraction. It may spread to the submental, sublingual and submandibular spaces and may be associated with fever, dysphagia and edema of the neck which may further complicate the situation by causing asphyxia.

The anesthesiologist may follow the various difficult airway guidelines to aid management. The problems associated may include limited/ painful mouth opening, difficult laryngoscopy, aspiration of blood, pus or mucous. Sometimes, induction of anesthesia and use of muscle relaxants may make mouth opening and thus intubation easier.[2] However, this cannot be relied upon, therefore, check ventilation and check laryngoscopy prior to administration of muscle relaxant is of paramount importance and it may be life saving in these situations.

Backup plan must always be thought of in the form of difficult airway cart and equipment for establishing front of neck access. Alternately, one may use other methods to secure airway like awake fibre-optic intubation and C Mac Videolaryngoscope if available. In an emergency airway management, the ability to ventilate and oxygenate the patient till a definitive airway is established is the only safeguard that might be there.

Ethics- No ethical issues involved

Funding-None

Disclosures- No conflict of interest

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Urinary Myiasis by Pericoma Species: A Case Report

Nidhi Singla,[1] Jagdish Chander,[2] Sarabmeet Singh Lehl[3]

[1]*Associate Professor,* [2,3] *Professor*

[1,2] *Department of Microbiology,* [3] *Department of General Medicine, Government Medical College & Hospital- 32, Chandigarh*

ABSTRACT

Myiasis is infestation of humans by larvae (maggots) of Dipterous family. Occasionally such types of patients are encountered in any of the hospital setting. An unusual case report of urinary myiasis in 12-year-old boy is presented here. The larvae were diagnosed to be Pericoma species belonging to family Psychodidae.

Keywords: Myiasis, Urine, Pericoma, Psychodidae, Larva

INTRODUCTION

Myiasis constitutes a parasitic disease invariably secondary to presence of larvae of certain insects, in skin wounds (cutaneous myiasis) or in the body (deep myiasis) of man and other vertebrates. Larvae of maggots are able to invade natural cavities and induce myiasis. They may invade the nasal fossae (nosomyiasis), eyes (opthalmomyiasis), ear (otomyiasis), urinary tract (cystomyiasis) and sometimes even vaginal and gastrointestinal tract. They may also invade the scalp or complicate surgical wounds causing surgical myiasis. There are usually three main families: Oestridae (botflies), Calliphoridae (blowflies) and Sarcophagidae (fleshflies), which are causative agents for economically important myiasis in livestock and occasionally in humans[1]. We report probably the first known case of urinary myiasis by Psychodidae (mothflies) of Pericoma species from our area.

CASE REPORT

A 12-year-old boy, belonging to lower socioeconomic strata and residing in the suburban areas came to the outpatient department of our hospital with the chief complaint of regular passage of worms in the urine for last 2 months. It was associated with increased frequency of micturition but with constant feeling of incomplete emptying of urinary bladder every time. There was also history of mild pain in right lumbar region for the last one month, relieved on its own without taking any antispasmodic drugs. In addition there was history of loss of weight despite increased appetite. There was no history of fever, haematuria, burning or painful micturition. No history of any skin disease, rash, pruritis, allergy or infection could be elicited. General physical examination was unremarkable. The patient was advised to undergo an ultrasound of abdomen, which was normal, with the only finding of mild urinary bladder distention. All routine laboratory investigations were within normal limits.

The urine sample of the patient was collected in sterile container. On examination: 8-10 mm motile, slender, brownish-black larvae having a flattened body divided into segments were seen. Integument seemed to be covered with short hairs or bristles (Fig 1 and 2). The

Fig.1: Macroscopic appearance of larvae

Corresponding Author :

Dr. Jagdish Chander, Professor & Head,
Department of Microbiology,
Government Medical College Hospital,
Sector 32, Chandigarh, India - 160030.
E-mail: jchander@hotmail.com

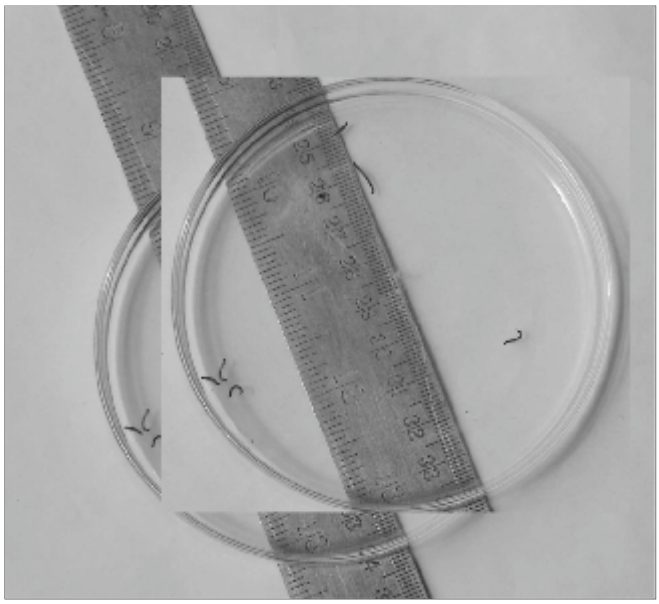

Fig. 2: Approximate size estimation of larvae

larvae were preserved in 10% formal saline and the specimen was sent to Department of Helminthology, Mahidol University, Thailand, where it was identified as larvae of mothfly i.e. *Pericoma* species, belonging to family Psychodidae. Patient was advised broad spectrum antibiotics but he did not comply and was later lost to follow up.

DISCUSSION

Myiasis is an animal or human disease caused by the larvae of more than 50 species of flies belonging to Order: Diptera. It may be accidental - eating eggs or larvae, which are not destroyed in the intestine, facultative - eggs laid on malodorous or festering wounds or obligate - necessary for development of fly.[2] Myiasis is invariably a self limiting and relatively harmless clinical entity but it could be potentially lethal requiring removal of the larvae and in some cases reconstructive surgery if there is secondary infection or aural and nasopharyngeal involvement. Occasional cases of myiasis have been reported worldwide[3,4] but it is much more common in tropical countries like India.[5-8]

In the present case of urinary myiasis, the infestation was due to larvae of Pericoma species belonging to Psychodidae family (Moth flies). These flies develop by complete metamorphosis with entire life cycle ranging from 8-24 days. Mature larvae are 4-10 mm long, elongate, cylindrical or somewhat flattened. Body segments are frequently divided to form annuli. Head is sclerotized, rounded and clearly separate from the thorax. The prolegs are absent. The larvae feed on the decaying material that collects in drains; therefore, moth flies are also called sewer flies, drain flies or filter flies. They are commonly seen in the moist, shaded habitats like homes, locker rooms, rest rooms and also bathrooms.[9] Here, they prefer areas with high organic content such as dung and rotten vegetables, sink drains, moist mops and sewage treatment facilities. The larvae get attracted to the draining infections, to the clothing soiled with urine and faeces or the body orifices emanating a bad odour. Individuals especially children, living in poor sanitary conditions and with poor hygiene in rural areas, are more susceptible. The other predisposing factors may be urinary obstruction, urinary retention or decreased general health of the patient.[3] However, the exact cause of urinary myiasis is not clear. Probably, the larvae gain access to urinary bladder through urethral orifice by patient's own fingers due to bad habits and poor hygiene, lodge and multiply there and then, subsequently get excreted in the urine. Use of unsanitary toilets or sleeping in open without a covering can be some of the causes leading to infestation. The condition can be serious as continuous and consistent presence of these larvae can lead to urinary tract pathology due to inflammatory toxins, microorganisms and viruses secreted by the larvae. There can be progressive and continuous necrosis of bladder wall due to larval migration.[10]

This case report emphasizes the importance of good hygiene and sanitation practices to avoid such cases in the population at large. There is a need to diagnose the condition early and correctly to avoid potential destruction of the area involved. The clinician should not ignore the complaints of the patient suggesting such an uncommon presentation.

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Perioperative Management of Obese Patient with a large Thyroid Swelling: A Case Report

Vanita Ahuja[1], Savita Rana[2], Ravneet Kaur[3]

[1]Associate Professor, [2]Junior Resident, [3]Senior Resident,
Department of Anaesthesia and Intensive Care, Government Medical College and Hospital, Chandigarh

ABSTRACT

Anaesthetic management of patients with large thyroid swelling is always challenging. Moreover, if patient is obese it makes the airway management more difficult. In addition, these patients tend to desaturate very fast due to decreased functional residual capacity. So, the primary goal is to secure airway in these patients with minimum airway complications. We are describing the case of a 62 year old female with body mass index (BMI) of 38 kg/m^2 who presented with a large thyroid swelling. Patient was planned for total thyroidectomy. Airway examination revealed short neck, modified Mallampati grade III, normal neck movements and receding mandible. Since patient had anticipated difficult intubation with no tracheal compression, our plan was to intubate trachea with video-laryngoscope in Rapid Airway Management Position (RAMP) with emergency airway cart standby. Anaesthesia was maintained with isoflurane and nitrous oxide. After thyroidectomy, trachea was extubated and she had an uneventful postoperative period.

Keywords: Thyroid swelling, Obesity, Difficult airway, Rapid Airway Management Position

INTRODUCTION

Difficult airway management has always been a major concern for the anaesthesiologist. Large thyroid swelling and obesity, both are associated with anticipated difficult airway even if present independently. There is a greater chance of failed intubation in these patients. So anaesthetic management of these patients is a formidable challenge.[1] In this case report, we are describing perioperative management of a morbidly obese patient with large thyroid swelling with no tracheal compressive symptoms.

CASE REPORT

A 62 year old female, (height 155 cm and 92 kg, BMI 38.2 kg/m^2) presented with anterior neck swelling which was progressively increasing since 4 months. The swelling was painless, and not associated with dysphagia, cough, dyspnoea, noisy breathing, hoarseness, even when position of patient changed from sitting to supine. There was no history of sleep apnoea syndrome and daytime somnolence. Patient was a known case of hyperthyroidism since 5 years and was receiving carbimazole 5mg once daily and was presently euthyroid. She was a known case of diabetes mellitus type 2 and was on oral hypoglycaemic drugs since 6 years. She was diagnosed with hypertension 2 years back and received telmisartan 20 mg once daily. Patient had no other co-morbidity. In the past, she was operated for hysterectomy 26 years back and open cholecystectomy 2 years back. General physical and systemic examination was normal. On inspection neck was short and an irregular thyroid swelling of approximately 10 x 7 cm was present. On palpation, two discrete swellings of approximately 5 x 4 cm were present on either sides of the neck extending anteriorly from chin to suprasternal notch and posteriorly up to the posterior border of sternocleidomastoid muscle. The swellings moved on deglutition and Pemberton's sign was negative. The swellings were not tender to touch, there was no rise in temperature and no bruit. Airway examination showed normal neck movements with adequate mouth opening, modified Mallampati grade (MPG) of III, receding mandible and sternomental distance of 12 cm. Indirect laryngoscopy reported normal vocal cord movements. Blood investigations and arterial blood gas reports were normal. Thyroid function test showed a T_3 1.50 ng/dl, T_4 7.6 ug/dl and TSH 3.6 IU/ml. Chest X Ray showed bilateral perihilar infiltration. Contrast enhanced

Corresponding Author :

Dr Vanita Ahuja, Associate Professor,
Department of Anaesthesia and Intensive Care
Government Medical College and Hospital,
Sector 32, Chandigarh -160030, India
Email: vanitaanupam@yahoo.co.in

computed tomography (CECT) chest reported bilateral prominent perihilar vascular markings, bronchitis and bilateral apical paratracheal enlarged node. Pulmonary function test showed FVC = 2.49, FEV_1 = 1.84, FEV_1/FVC = 87.78. Computed tomography (CT) neck showed grossly enlarged thyroid gland with multiple heterogeneously enhancing hypodense nodules, largest measuring 41 x 39 mm in left thyroid lobe suggestive of multinodular goitre. Major neck vessels were normal. Ultrasonography (USG) guided FNAC of thyroid swelling showed benign colloid goitre.

Patient was planned for total thyroidectomy under general anaesthesia. Premedication instructions included tablet alprazolam 0.25 mg per oral, ranitidine 150 mg per oral and nebulisation with short acting bronchodilators, anticholinergic and steroid at night and on the morning of surgery. Patient was kept nil per oral overnight. In operation theatre standard anaesthesia monitoring (pulse-oximeter, noninvasive blood pressure monitor, electrocardiography, capnography) was started. A RAMP (rapid airway management position) position was made in view of anticipated difficult intubation. Pillows were placed under the head and torso of the patient so to align external auditory meatus with sternal notch in one horizontal plane and face of the patient parallel to ceiling. Preoxygenation was done with 100% oxygen for 3 minutes. Intravenous (IV) glycopyrrolate 100µg, midazolam 2 mg and fentanyl 160µg was administered. IV propofol 120 mg was administered with titration to dose required for patient to sleep. After ability to ventilate the lung with bag and mask ventilation, IV vecuronium 6 mg was given. Trachea was intubated with 7.0 mm cuffed endotracheal tube (ETT) using CMAC videolaryngoscope. Anaesthesia was maintained with oxygen, nitrous oxide (40:60) and 1% isoflurane. A large size thyroid was removed intraoperatively (Fig 1). The size of thyroid was approximately 12 x 13 cm (Fig 2). Intraoperative period was uneventful; thyroid gland was removed with minimal blood loss. At the end of surgery, vocal cord movements were assessed with videolaryngoscope which were normal. Neuromuscular block was reversed with IV neostigmine 3.5mg and 0.6 mg glycopyrrolate. A nasopharyngeal airway of 7.0 mm size was inserted in the right nostril after lubrication to maintain a patent airway and this was followed by tracheal extubation. Patient had an uneventful postoperative period. Postoperative care included analgesia, nebulisation with short acting bronchodilators and anticholinergic drugs and antibiotics.

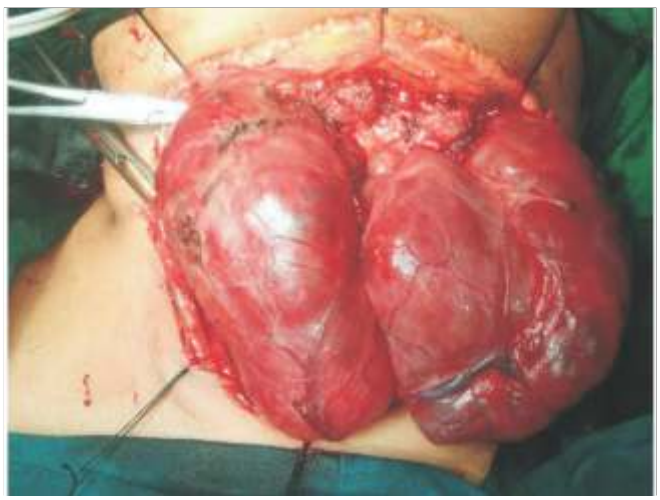

Fig. 1: Intraoperative total thyroidectomy surgery

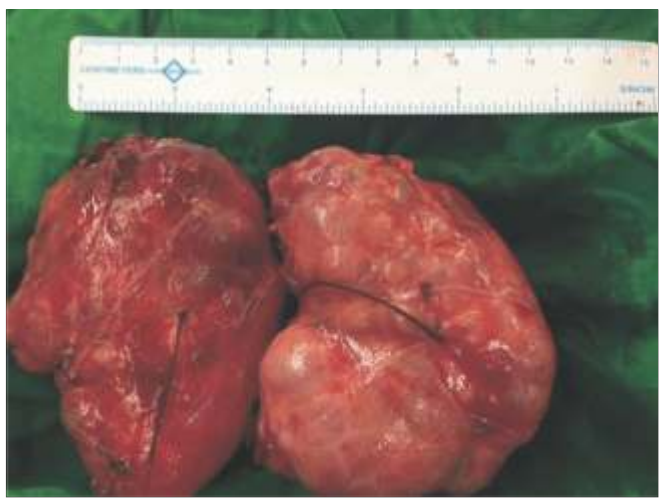

Fig. 2: Thyroid gland specimen after removal

DISCUSSION

Airway management in thyroid surgery is always considered challenging for both the surgeon and the anesthesiologist. The airway is challenging in the present patient due to grade 2 obesity, short neck and large thyroid swelling. Amathieu et al reported the overall incidence of difficult intubation as 11.1% in thyroid surgery.[1] The authors concluded that the usual preoperative criteria like MPG, limited neck movements and thyromental distance, were reliable predictors of difficult airway and there is no specific predictor for the difficult intubation in thyroid swelling.[1] On the contrary, Bouaggad et al reported that the presence of cancerous goitre was a major factor for predicting difficult airway and not the greater size of goitre.[2] Large thyroid swelling

leads to compression symptoms like dyspnoea, dysphagia, change in voice and tracheal deviation. All these are considered as predictors of anticipated difficult airway. Other predictors include increased BMI, MPG III or IV, thyromental distance of less than 6 cm, restricted neck movements and history of obstructive sleep apnoea.[3] Obesity is associated with a 30% greater chance of difficult or failed intubation.

In the perioperative period the emphasis should be on obtaining RAMP position in obese patients. RAMP position maximize the chances of successful direct laryngoscopy and intubation as it aligns the external auditory meatus and sternal notch in the same horizontal plane aligning the laryngeal and pharyngeal axis. Hence, improving airway patency and respiratory mechanics, facilitates passive oxygenation during apnoea and successful tracheal intubation.[4,5]

Management of difficult airway includes difficult airway cart including fibreoptic laryngoscope, video-laryngoscope, and surgical airway. Each technique has its own advantages and disadvantages. A case has been reported where complete airway obstruction occurred during awake FOI with the use of local anaesthetic, it lead to acute loss of the airway. So urgent surgical intervention was required.[6] Hence, airway management in large thyroid swelling requires experience and preparation.[7]

In our case, patient had large thyroid swelling without tracheal compression symptoms, short neck, MPG III, receding mandible and BMI of 38 kg/m^2. Trachea was intubated by an experienced anaesthesiologist with CMAC videolaryngoscopy with patient in RAMP position and difficult airway cart ready which lead to successful outcome.

It has been observed that most dreaded complications related to thyroid surgery manifest in the postoperative period.[8] Hemorrhage is the most common postoperative complication. It can lead to acute airway obstruction by compressing the neck structures. Patient should be shifted to operation room and sutures should be removed immediately. Patient's airway should be secured by using LMA or ETT. Laryngeal oedema is another dreaded scenario caused by multiple attempts of laryngoscopy due to difficult intubation or due to venous obstruction caused by enlarging hematoma. If the oedema causes stridor, intubation with ETT is mandatory.[9] Recurrent laryngeal nerve (RLN) palsy can also result because of traction, transaction, entrapment or ischemia and it can be temporary or permanent. If unilateral RLN is involved then the patient presents with difficulty in breathing, hoarseness of voice, difficulty in vocalization. Bilateral RLN palsy results in complete adduction of vocal cords leading to severe stridor which can be treated by tracheal intubation or tracheostomy.[10,11] External branch of superior Laryngeal nerve is the most commonly injured nerve, leading to paralysis of cricothyroid muscle resulting in alteration of quality of voice as vocal cords fail to tense. If internal branch get injured, then patient can develop dysphagia due to deranged swallowing reflex as sensory supply of supraglottic mucosa is absent.

A large goitre compressing over tracheal structures for a long period can lead to pressure atrophy and erosion of cartilaginous tracheal rings ultimately ending in tracheomalacia. After thyroidectomy, tracheal wall loses the surrounding support and can collapse which can lead to respiratory obstruction. In such cases reintubation and ventilatory support may be required till the strength of tracheal wall returns to normal as it is self-limiting.

Injury to the parathyroid glands is one of the operative complications which can lead to acute hypocalcemia in approximately 20% of patients. Clinical features include perioral tingling, mental confusion, muscular twitching, tetany, seizures.[12] Cardiorespiratory manifestation include cardiac irritability, prolongation of QT interval, arrhythmias and laryngospasm. It can be treated with oral supplements of calcium if Ca+ levels are >2 mmol/l, and if the levels are below 2 mmol/l calcium gluconate intravenously needs to be given.

The drug titration in obese patients is based on lean body weight (LBW) and depth of anaesthesia. The present patient was of obese type 2.[13] The opioids and neuro-muscular blockers were calculated based on lean body weight. Lean body weight excludes fat mass which is the cause of majority of weight in obese patients. There are several formulas to calculate LBW. Regardless of total body weight, LBW generally never exceeds 100 kg in men and 70 kg in women.[13]

In conclusion, perioperative airway management an obese patient with large thyroid swelling requires proper preoperative preparation and an expert anaesthesiologist for a successful outcome.

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Discussion: Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence. What this study adds to the available evidence, effects on patient care and health policy, controversies raised by this study; an future research directions.

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References

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I. Standard journal article

If less than six authors, list all the authors.

Halpern SD, Ubel PA, Caplan AL. Solid-organ transplantation in HIV- infected patients. N Engl J Med. 2002;347:284-7.

If more than six authors, list the first six authors followed by et al.

Rose ME, Huerbin MB, Melick 1. Marion DW, Palmer AM, Schiding JK, et al. Regulation of interstitial excitatory amino acid concentrations after cortical contusion injury. Brain Res. 2002;935: 40-6.

2. Books and other monographs

Personal author(s)

Murray PR, Rosenthal KS, Kobayashi GS, Pfaller MA. Medical Microbiology. 4th ed. SI. Louis: Mosby; 2002.

Chapter in a book

Meltzer PS, Kallioniemi A, Trent JM. Chromosome alterations in human solid tumors. In: Vogelstein B, Kinzler KW, editors. The genetic basis of human cancer. New York: McGraw-Hill; 2002. p. 93-113.

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