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Academics : The backbone of activities of a technical association

Quality assurance............................

The expectations of patients, doctors, managers is that the healthcare should be delivered to the highest possible quality standards and should preferably be equal for all. The creation of good definition of quality is very difficult, though everybody is convinced that they know what quality is. Dictionaries give a literal definition which may be helpful to the doctor. The definition tends to be given in terms of the degree of excellence or as a measure of “how good or bad the Service or Product is” and it is always better to describe quality in terms of the degree of excellence for a specific purpose. Quality is therefore a relative concept.

The constant examination and improvement of quality is fundamental to clinical care. Quality should be viewed from the perception of the consumer and not the supplier. It must always be remembered however that to achieve excellence in quality depends on the interaction of many individuals and many processes and that the organization itself must be committed to change before change is possible. The different individuals will also posses different views on what they believe is quality. What is a high quality anaesthesia or surgical procedure or a procedure for a physician? The anaesthesiologist will put high on the list, issues such as no patient’s awareness during anaesthesia no postoperative nausea and vomiting, no post operative pain, no chance of adverse reaction to any drugs. The patient will put high on the list freedom form all risk in addition to those listed by the anaesthesiologist. The surgeon, a high quality anaesthetic produces ideal conditions for surgery and takes zero time to implement.

The problem is that the whole healthcare service was never designed to function in this way we are working within a cumbersome system that has been in existence for decades and are being asked to alter it by auditing and improving the process. This is laudable but well nigh impossible. It could be linked to huge old cargoship with no power steering wheel each in a different room and operated by a different person. The effort to turn the wheels is immense the amount by which the rudder has moved is not known and the inertia is such that it will have travelled many miles before any change in course can be detected. The ideal, which is impossible would be to start again with a new blank sheet of paper. There is an old joke which goes : Question: “Can you direct me to Liverpool?” Reply: “If I were going to Liverpool, I would not start from here”. “Quality should be designed into a system and not regulated as a late add-on option.

Quality, excellence and cost effectiveness are all interrelated factors and must be a part of everyday practice in medical science. It is clearly the duty of every doctor to provide high quality service. This aim is assisted by growing number of local, national and international guidelines for the medical practice. As the number of guidelines grow, maintaining current knowledge about them will become a major part in the global need to keep uptodate. Last, but by no means least, it is necessary to provide care within the available resources. Care must be as cost-effective as possible without there being any compromise on quality.

Prof. (Dr.) Deba Prasad Mohanty, Editor OMJ
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OMJ Secretary’s Message...

Needs and opportunities for research in undergraduates.

The culture of research in medicine introduced at undergraduate curriculum has become the call of the day. It has become a pure necessity to inculcate the attitude of research to find something new right at the beginning of the undergraduate curriculum. Once they know why it is necessary, and the experience of joy of having found out something new will pull them into the fold of research activities. Appreciation and reaching out to the medical community by way of publication is a step forward. When research is put into clinical practice for the benefit of patients: that is the ultimate. It’s just a matter of how you think. Research material abounds in India but unfortunately the maximum guidelines from research come from the western world. Looking at PM Modi’s skill India development, developing skill in medical research needs more opportunities and expanding horizons.

Comprehensive or systematic review of the existing state of knowledge about a topic in interest is the stepping stone and is now easily available. “UG experience fosters knowledge and skill needed to conduct other research and motivates graduates” UG research experience enhances employability. Research skill is a developmental skill for the rest of our life. Research and Academics are complimentary to each other and stimulate the interest in each other. UG research experience improves student-faculty relationship.

Few fundamental topics need to be addressed right at the commencement of the UG courses. They are:

- Appreciate principles of research.
- How is evidence derived?
- Critical appraisal which is required to make informed judgements, etc.

It is the cornerstone of effective and good evidence based practice and is associated with higher levels of student satisfaction and generic skill development. Research Skill Areas, Research Methods, Information gathering, literature search, critical analysis and review should be addressed. Independent thinking, data processing, statistics need to be made interesting. However, the most critical parts of the research process are those parts that are associated with thinking and not doing.

Research Question Analysis that need to be answered for undergraduates:

- Why is my work important to others? (Significance)
- How am I going to answer my question? (Methodology)
- What do I need to know and how is my topic different from what is already done in this area? (Literature search)

A recent study from south India on undergraduate medical research has shown that nearly 70% of undergraduate students are unaware of research. Though, every year about 35000 students join medical college, only a few motivated students- about 2-5% are interested in research. There is a huge lack of awareness among undergraduates about research methodology. And then comes funding. Apart from government, apex bodies are also encouraging undergraduate medical research. Things are improving as some of the Universities are allocating their budget to research.

Research Funds in India:

- Donated by the alumni of MMC from Batch of 1961
- MAA Research Grant
• ICMR Short Term Studentship (STS)
• The three national Science Academies offer two-month Summer Fellowships:
  o Indian Academy of Sciences, Bangalore
  o Indian National Science Academy, New Delhi
  o The National Academy of Sciences, Allahabad.
• Tata Institute of Fundamental Research Visiting Students’ Research Programme (VSRP2014)

Venues for research exchange and interaction: Presentation will boost their confidence and also provide chance to enhance their thinking in many ways. Emphasis should be laid to motivate student to present their work. It is very important in any research to make the presentation of the work and new findings.

Conferences for medical students: All conferences should encourage undergraduate students to present their research work. The regional/zonal medical conferences for ICMR and KVPY recipients organized by Moving Academy of Medical Sciences had got overwhelming response. MEDICON organised by Indian forum of medical research (INFORMER), Illuminati: undergraduate medical research conference Medsicon 2014: 4th Annual Medical Students International Conference, OSMECON 2014: 5th pan India Undergraduate Medical Conference at Osmania Medical College, KARMIC 2014, 3rd international conference for undergraduate medical students by the Indian Medical Students Association (IMSA) at KIMS Bangalore, Empower 2014: 10th International Medical Students’ Research Congress at Bangkok-Pattaya, Thailand, 35th annual conference of the Asian Medical Students’ Association (AMSA International) with international travel support to attend the international conferences,

ISCOMS at the University Medical Center of Groningen in Netherlands, International Student Congress of Medical Sciences (ISCOMS), etc. focus to encourage undergraduate students to present research work.

Without publication, students’ work will be unrecognized and their potential will go waste. Once the research is conducted, evidence is generated it needs dissemination through scientific publications. It exposes them to the art of writing; in addition to executing the work guided by the guide and presenting the work.

Students Medical Journal, PLOS Medicine, Student Lancet, Student BMJ, McGill Journal of Medicine, Journal of Young Investigators, International medical journals encourage the undergraduate medical students. Indian Journal of Medical Specialities (IJMS) gives preference to the articles submitted by undergraduate medical students; Indian Journal of Medical Research (IJMR) and Journal of Postgraduate Medicine has introduced students section. Having a publication in a peer reviewed journal and preferably Medline indexed journal increases the chance of getting international fellowships and other career opportunities.

THANK YOU
Long Live IMA !!!

Dr. Bhagyalaxmi Nayak
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Opportunistic Infections in PLHIV at A.R.T. Plus Centre, S.C.B. Medical College

Vijayalaxmi Nayak¹, Abhishek Biswal²

Introduction:
Human Immunodeficiency Virus (HIV) pandemic is among the greatest health crises ever faced by humanity. Globally, 36.7 million [30.8–42.9 million] people were living with HIV at the end of 2016 [1]. The total number of people living with HIV (PLHIV) in India is estimated at 21.17 lakhs (17.11 lakhs–26.49 lakhs) in 2015.
Despite the fact that different studies have been conducted on the prevalence of individual Opportunistic Infections (OIs) among HIV infected patients in various developing countries, information about the magnitude and spectrum of OIs is scarce in the study area. Therefore, this study was aimed to add updates to the existing data on extent of OIs and their immunological correlates among HIV-infected patients attending ART Plus Centre, S.C.B. Medical College.

Background:
Morbidity and mortality in HIV disease is due to immunosuppression leading to life-threatening opportunistic infections (OIs) during the natural course of the disease[2]. Opportunistic infections and neoplasms remain a major concern even in the current era of combination antiretroviral therapy. Some patients do not have adequate response to antiretroviral agents for various reasons including drug side effects, poor adherence, drug interactions, or acquisition of a drugresistant strain of HIV-1. Therefore OIs continue to cause substantial morbidity and mortality in patients with HIV infection despite use of ART[3].

This study was aimed to assess the prevalence of OIs & their CD4 correlation among HIV-infected patients attending ART Plus Centre, S.C.B. Medical College.

Materials & Method:
Study population and location - A cross sectional study was conducted on 340 adult HIV-infected patients attending ART Plus Centre, S.C.B. Medical College, Cuttack from August 2017 to October 2017. Patients’ OI was diagnosed through clinical examination and laboratory investigations. Sociodemographic and clinical data were obtained from interview and patients’ medical records.

Inclusion criteria - Adult HIV-infected patients attending the ART Plus Centre, S.C.B. Medical College, Cuttack.

Exclusion criteria – Patients with other significant comorbidities or concurrent medical conditions like DM, Heart disease etc. -Patients on steroids or other immunosuppressant.

Study Methodology - Study participants were selected through systematic random sampling method among HIV-infected patients visiting the ART clinic during the study period.

The diagnosis of the diseases was done through clinical examination and laboratory investigation as per NACO guidelines[4]. The patients who had 4 Symptoms(Current cough, fever, night sweat, weight loss) positive, were considered for the tuberculosis examination by CBNAAT. Diagnosis of Extra Pulmonary Tuberculosis (EPT) was done through physical examination followed by fine needle aspiration (FNAC) smear microscopy for acid fast bacilli (AFB) and cytological examination. For the confirmation of chronic diarrhoea, clinical presentations and stool routine examination was done. Oral candidiasis, skin fungal infections and other OIs were diagnosed by clinical presentations of patients[5,6].

Results
In this study, 340 HIV-infected patients were included; of whom (n = 187/340, 55%) were females. The overall prevalence of OIs was (n = 82/340, 24.1%).
Tuberculosis (n = 38/340, 11.17%) followed by candidiasis (n = 18/340, 5.29%) and diarrhea (n = 13/340, 3.8%) were the most frequently observed OIs. World Health Organization (WHO) clinical stage III and IV were found to have strong association with acquisition of OIs.

![Graph of OIs detected](image)

Figure – 1: Representation of common OIs detected

Analysis:

In this study, 340 adult HIV infected patients were included, out of which 153 were males and 187 were females. The number of opportunistic infections (OIs) clinically diagnosed among them was found to be 82, accounting for a prevalence of 24.11%.

Tuberculosis emerged as the most frequently detected OI in this spectrum. Out of 82 OIs, 38 cases were due to tuberculosis (n=38/340, 11.18%). While pulmonary TB accounted for 26 cases, extra pulmonary TB accounted for 12 cases.

38 year old female presented with cervical lymphadenopathy, which was later confirmed as extra pulmonary TB by FNAC and AFB staining.

44 year old female with multiple discharging TB sinuses.

Candidiasis was the second most frequent OI to be associated with HIV infection. Total number of cases detected was 18 (n = 18/340, 5.29%), out of which 16 were oral candidiasis cases and rest 2 were cases of esophageal candidiasis.

A 36 year old man presenting with oral candidiasis.
Diarrhoea was found to be the third most frequent condition with 13 cases in total (n=13/340, 3.82%).

Other specific OIs found to be associated with HIV infected patients were Seborrheic dermatitis (n=4/340), Tineacorporis (n=4/340), Scabies (n=3/340), Herpes zoster (n=2/340).

Discussion:

Although HIV is the initial causative agent for AIDS, most of the morbidity and mortality seen in immunocompromised patients results from OIs that take advantage of the lowered cellular and humoral defense of the patient.

In this spectrum, tuberculosis, candidiasis and diarrhea were concluded to be the most predominant OIs with a prevalence rate of 38, 18, and 13 respectively.

Conclusion:

Owing to the high mortality and morbidity attributable to the specific OIs encountered by people infected by HIV, it is imperative that immediate and appropriate strategies should be undertaken to prevent the incidence of OIs in HIV patients and if present, treat them at the earliest. Specific groups should be given priority like extremes of age group, poor clinical staging, low CD4 count, patients with family contacts etc. Early diagnosis and adherence to treatment will go a long way in diminishing the hazard posed by the multitude of OIs among the HIV infected population.

References:

INTRODUCTION:

Benign multiple diseases occurring in Ano rectal area in same patient is rarely encountered. Common diseases like Piles, Fissure and Fistulae may occur alone or in combination. Intolerable pain from one trivial diseases like a Fissure is so bad that it forces patient to attend surgeons. When 3 or 4 painful diseases are combined, the patient’s condition becomes horrible. Aim of this paper is to make doctors aware of conditions like this.

Total No. of Cases of Benign Ano-Rectal Diseases Treated in the above Period = 1152

Age Distribution:
# Below 20 Yrs. - 72 Nos.
# Above 20 Yrs. - 1080 Nos.

Sex Distribution:
# No. of Male - 720 Nos.
# No. of Female - 432 Nos.

Diseases Under Study:
- a) Piles
- b) Fissure
- c) Fistulae
- d) Polyp
- e) Prolapse
- f) Peri Anal Abscess
- g) MIPH Failure Piles
- h) Anal Stenosis

Distribution of Cases (Disease wise):
- # Piles with Fissure - 275
- # Fissure - 225
- # Piles - 211

# Piles with Fistulae - 180
# Fistulae - 135
# Polyp - 035
# Prolapse - 011
# Peri Anal Abscess - 074
# MIPH Failure Piles - 002
# Anal Stenosis - 004

Post up. Follow Up Study:
Two weeks to Three Years

<table>
<thead>
<tr>
<th>Sl.</th>
<th>Diseases</th>
<th>Success</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Piles with Fissure</td>
<td>95 %</td>
<td>05 %</td>
</tr>
<tr>
<td>2</td>
<td>Fissure</td>
<td>96 %</td>
<td>04 %</td>
</tr>
<tr>
<td>3</td>
<td>Piles</td>
<td>95 %</td>
<td>05 %</td>
</tr>
<tr>
<td>4</td>
<td>Piles &amp; Fistulae</td>
<td>90 %</td>
<td>10 %</td>
</tr>
<tr>
<td>5</td>
<td>Fistulae</td>
<td>92 %</td>
<td>08 %</td>
</tr>
<tr>
<td>6</td>
<td>Polyp</td>
<td>100 %</td>
<td>00 %</td>
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<tr>
<td>7</td>
<td>Prolapse</td>
<td>100 %</td>
<td>00 %</td>
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<tr>
<td>8</td>
<td>Peri Anal Abscess</td>
<td>80 %</td>
<td>20 %</td>
</tr>
<tr>
<td>9</td>
<td>MIPH Failure Piles</td>
<td>100 %</td>
<td>00 %</td>
</tr>
<tr>
<td>10</td>
<td>Anal Stenosis</td>
<td>100 %</td>
<td>00 %</td>
</tr>
</tbody>
</table>

Internal Anal Sphinterctomy:
- In 1839 - BRODIE was the 1st person to perform this.
- In 1963 - HILTON also suggested that treatment of Anal Ulcer can be done with this method.
- In 1864 - MILES gave the real credence for this method.
- In 1951 - EISON HAMMAR performed practical Internal Anal Sphinterotomy for treatment of Anal Fissure.

Fissurolasty:
The Anal canal is stretched exposing the Fissure, Sentinel Piles and the Papila Internally. With Electrocautery, the above mentioned structures are dissected and removed enmass exposing the Internal
Anal Sphintermuscle. The wound is closed, repairing mucosa and the skin with 2-0 Catgut.

HEMORRHHOIDS (PILES)

Piles is a common language all for a lump, pain, swelling, bleeding and protrusion etc. occurring in anal area.

Hemorrhoid complaints are one of the most common diseases of human civilization. It is a problem to occur at any age and in both sexes. It has been estimated that at least 50% of the individuals over the age of 50 years have at sometime experienced symptoms related to hemorrhoid. Prevalence rate of this disease is 4.5%. The following have been suggested as factors that contribute to the development of hemorrhoid.

# Heredity
# Anatomic features
# Nutrition
# Occupation
# Climate
# Psychological Problems
# Senility
# Endocrine changes
# Foods & drugs
# Infection
# Pregnancy
# Exercise
# Coughing
# Straining
# Vomiting
# Constrictive clothing
# Constipation

ETIOLOGY & ANATOMY:

In 1975 THOMSONS master thesis introduced the term vascular cushion. They are discontinuous cushions. Three main cushions are found in left lateral, right anterior & right posterior. Each cushion is rich with blood vessels, muscle fibers & muscularis submucosa formed with the conjoint longitudinal muscle attached to internal sphincter. They protect the anal canal from injury of hard stool. These anal cushions when slide down, lead to formation of hemorrhoids.

CLASSIFICATION:

Hemorrhoids are classified by location
- Internal Grade- I, II, Grade- I, II, III, IV
- External
- Mixed

External hemorrhoids with fissure
Grade IV Piles

Probe in Fistulae with Piles in 6 ‘O’ clock Position

Grade IV Piles, Fissure with Polyp

Thrombotic Piles

Grade IV Piles with multiple Fissures

Thrombotic Gangrenous Piles
Quackery Treatment by acid burn of Piles.

**Ambulatory Treatment:**

1) Injection Sclerotherapy

2) Rubber bandLigation (BLISDLL – 1954, BARRON-1962)

3) Infra RedCoagulation(1979 NEIGER)

4) Cryosurgery(LEWIS & COLLEAGUS)

5) Ultroyd – A devise for Ambulatory treatment of Monopolar low voltage instrument with a single used sterilised probe.

6) Bi-polar Diathermy – It is a method of local application of heat to induce tissue destruction, ulceration &fibrosis

7) Lords Dilatation – It is a simple anal dilatation (1968 LORD reported this method for treatment ofhaemorrhoid to reduce rectal pressure ,thereby causing amelioration of the condition).

**Operative Approach to the Treatment of Haemorrhoids:**

1) When Anorectal architecture has been severely compromised such as: External Compressio, Gangrene, Extensive Thrombosis, Hyper trophied Papillae or Anal Fissure.

The goal of conventional Hemorrhoidectomy is to excise completely all the hemorrhoidal tissue without post-operative complication. “John Golighar stated Anal Surgery is usually a matter of novice taught by the incompetent.” Many Surgeons have worked for surgical treatment of hemorrhoid. They are:

1) Buie
2) Fansler
3) Ferguson
4) Milligan
5) Morgan  
6) Parks  
7) Salmon  
8) White Head & their colleagues.

Closed (Fergusson Haemorrhoidectomy) is followed in this series.

2) Ligasure Haemorrhoidectomy:  
3) Laser Surgery:  
4) Stapled Haemorrhoidopexy:

**ANORECTAL ABSCESS**

Anorectal Abscess is an acute inflammatory process that often is the initial manifestation of an underlying Anal Fistula. An abscess in these areas may be consequence of other causes and association. These include:

1) Foreign intrusion  
2) Trauma  
3) Malignancy  
4) Radiation  
5) Immuno-compromised state (AIDS)  
6) Infectious dermatitis  
7) Tuberculosis  
8) Actinomycosis  
9) Crohns Diseases  
10) Anal Fissure  
11) Complication of Hemorrhoidectomy  
12) Internal Anal Sphincterotomy

The main cause of Anorectal Abscess is plugging of the anal ducts i.e Cryptogandular Theory (Chary-1878 & Herrmann & Desfosses- 1880)

KLOSTER HALFEN AND COLLEAGUES implied that plugging and infection of duct can result in an abscess. That can spread in several directions that may ultimately lead to the development of an Anal Fistula.

SHAFER AND COLLEAGUES opined that the Fistulae in Anois the result of a congenital anomaly of pre-disposition.

AGE AND SEX- Abscess and Fistulae occur more commonly in men than in women. MACELWN AND COLLEAGUES reported a ratio 3 men to one woman.

**TYPE OF ABSCESS:**

Four presentations of Anorectal Abscess are described.

1) Perianal  
2) Ischio rectal  
3) Intersphinteric  
4) Supralevator

**TREATMENT:**

**Acute Suppuration (Abscess):**

Should be drained in a timely manner. Lack of fluctuation is not a reason for delay in treatment.

**Synchronous Fistulectomy:**

In Many cases internal opening is identifiable with a probe and Fistulectomy is usually performed.

**Ischiorectal Abscess:**

This may be virtually un-apparent. The patient complains of only severe pain. This type of abscess is seen in 20-25% of patient.

**Technique of Drainage:**

Drainage of an Abscess requires some planning. It is better to create an external opening as close to the anal verge is possible.
Anal Fistulae is a condition that has been described virtually from the beginning of medical history. Hippocrates in about 430 BC suggested that the diseases was caused by contusion and tubercles caused by riding on horseback. He was the 1st person to advocate the use of Seton. Numerous papers and books on the subject are presented on Anal Fistulae for last 2000 years. SALMON (1796-1868) established a Hospital in London devoted to the treatment of Anal Fistulae.

It is said that reputation of more surgeons has been lost because of consequence of Fistulae operation than from any other operation procedure.

**SYMPTOM:**

a) Swelling  
b) Pain  
c) Discharge  
Fistulae have an antecedent history of abscess that drained spontaneously ;for which Surgical drainage had been performed.

**CLASSIFICATION:**

PARK & COLLEAGUES are credited with proposing the following somewhat complicated but extremely thorough classification.

**Classification of Fistulae-in-ANO:**

- Intersphinteric  
- Simple Low Track  
- High Blind Track  
- High Track with opening into rectum  
- High Fistulae without aperianal opening  
- High Fistulae with extrarectal or pelvic extension  
- Fistulae from pelvic disease  
- Transsphinteric  
- Uncomplicated  
- High blind track  
- Suprasphinteric  
- Uncomplicated  
- High blind track

**Extrasphinteric:**

- Secondary to transsphinteric fistulae  
- Secondary to trauma  
- Secondary to anorectal disease  
- Secondary to pelvic inflammation

**Combined:**

- Horseshoe  
- Intersphinteric  
- Trans-sphinteric  

**COMPLEX FISTULAE** is a variety of Intersphinteric and extraspincteric fistulae. They are more difficult to treat and with increased risk of recurrence and impairment of control.
Five types of Fistulae are generally described by most authors.
1) Submucosa
2) Intersphincteric
3) Transphincteric
4) Suprasphincteric
5) Extrasphincteric

IDENTIFICATION OF FISTULAE TRACT:
1) Goodsall’s rule
2) Careful physical examination
3) Probing of the tract
4) Injection and Radiologic Technique
5) Anal Endosonography
6) Magnetic Resonance imaging

PRINCIPLES OF SURGICAL TREATMENT:(Percivalpot- 1714-1788):
1. Fistulotomy
2. Fistulectomy with Sphinter repair
3. Dissection and Excision of Tract in complicated Fistulae by coring out Dissection with Electro Cautery.

ANAL FISSURE WITH SENTINEL PILES

It is a very common ano-rectal condition. It can be very troubling because of severity of patient discomfort & extent of disability far exceed that which would be expected from a trivial lesion.
Anal fissure is a cut or crack in the anal canal that may extend from the mucocutaneous junction to dented line. It can be acute or chronic. It may occur at any age, usually in young adults. Both sexes are affected equally. Anterior fissure occurs more in female.

**ETIOLOGY & PATHOGENESIS**

Constipation or straining at stool - the hard fecal bolus cracks the anal canal. Frequent defecation & diarrhea may cause this condition. Lockheart believed that muscle fibers in posterior anus may cause fissure. Lateral mucosa is best supported. The blood supply to the muscle in posterior part is less. So the ischemia is a factor causing anal fissure.

**SYMPTOMS**

A. PAIN: during defecation

B. Bleeding is minimal, but at times may be very severe requiring blood transfusion

**EXAMINATION**

Acute Fissure: Patient’s history is diagnostic. Gentle traction of the Peri-anal skin opens the wound often can be seen. Digital examination is painful & to be avoided. Appropriate treatment may be initiated without more specific confirmatory evidence.

**CHRONIC FISSURE**

After 2 months, the ulcer becomes chronic with clearly recognized, well circumscribed - with sentinel piles indurations & fibrosis. Hypertrophied anal papillae can be felt at the apex of the ulcer mistaken for a tumor. Anoscopy can be done.

**TREATMENT**

A. Medical treatment

Acute fissure: high fiber diet, sitz bath, laxative, suppository, oral analgesic, topical anesthetic cream

**Sclerotherapy:**

Antebi & colleagues treated by injection or sodium tetradecyl sulfate direct into the fissure with 80% free of pain.

**Solcoderm:**

Is a skin preparation used in treatment of anal fissure by CHEN & CO-WORKERS getting good results.

**Hyperbaric Oxygen:**

CUNDAL & COLLEAGUES used oxygen for treatment of acute fissure

**Glyceryl Trinitrate Ointment:**

Chemical sphinterotomy using nitric acid donor GTN has been the subject of numerous articles & considerable debate in medical literature. It causes reduction in internal anal sphinter pressure & healing of fissure.

Botulinum Toxin, Nifedipine & Diltiazem are used for treatment of fissure.

**SURGICAL TREATMENT:**

Sphincter Stretch: Originally described RECAMIAR IN 1838 for anal fissure as a ambulatory surgical facility & correct method.

**Result:**

It is a reasonable & effective procedure for the symptomatic relief of anal fissure but this procedure is abandoned in favor of lateral internal sphincterotomy.

**Internal Anal Sphinterotomy:**
CHRONIC ANAL FISSURE:

Pain is not as severe as in acute fissure. Bleeding is troublesome, mucus discharge, soiling of the under clothes & pruritus are troublesome.

SURGICAL TREATMENTS:

Excision & internal anal sphincterotomy in lateral position is commonly done. This removes the Eschar skin tag & papillae. The resulting wound reveals the internal sphincter at base. The wound is closed with 2-0 catgut; heals in 2 weeks period in our series. We name it Fissureplasty.

It is not an External Piles

Anal Fissure with Hemorrhoids:

Hemorrhoidectomy with Sphinterotomy should be performed concurrently. The sphincterotomy should be carried out laterally usually at the site of the left lateral piles

Chronic Anal Fissure with Stenosis:

Stenosis & Fissure may supervene. Conservative medical management with stool softener & dilatation is often advised, but Anoplasty as recommended by FERGUSON is preferred.

Observation:

1) Fissurectomy removes sentinel piles fibrous tissue base and the p Tissue base and the papillae en mass exposing the Internal Anal Sphinter muscle and wound is created. This wound is closed by 2-0 Catgut allowing the wound to heal by 2 weeks. It is called Fissuroplasty.

2) In this Series Grade-III & Gr.IV piles are treated by closed Hemorrhoidectomy. The rectal...
mucosaproximal ligation are treated by IRC. In almost all cases, it is observed that recurrence rate is very minimal or almost nil.

3) Anal stretch when performed with utmost gentleness relieves Fissure pain promptly and is a simple outdoor procedure.

IRREGULAR VISIT TO LATRINE INVITES PAINFUL DISEASES IN ANO RECTUM
TAKE CARE OF YOUR BOWEL HABIT.

SAVE YOUR ANUS
Endometrial Carcinoma : 7 Years Experience at Acharya Harihar Regional Cancer Centre
Ashok Kumar Padhy, SonzPaul, M Mohapatra, J Mohapatra, B L Nayak, J Parija, S K Giri, N Panda, S N Senapati

INTRODUCTION
Endometrial carcinoma ranks 3rd in India amongst the gynecological malignancies.[1] Endometrial cancer (EC) can be classified into two distinct groups – type I and type II, based on histology, which differs in molecular as well as in clinical and histopathological profiles.[2] Type II is nonestrogen dependent, nonendometrioid, with higher grade histologies, more aggressive and carries an adverse prognosis.[3,4] Although type II cancers contribute only about 10% of EC incidence, they present at advanced age and cause approximately 50% recurrence and deaths with a low 5-year, overall survival rate of 35%.[5,6]

Majority of patients with EC have early-stage (stages I-II) diseases at presentation when cancer is limited to the uterus.[8] Although tumors of these early stages can be completely resected, some clinical and surgicopathological features may be risk factors for failure after treatment. These features are, for example, older age, large tumor size, cervical or lower uterine segment involvement, aggressive histopathologic subtype or high-grade tumor, deep myometrial invasion, and lymphovascular space invasion.[7] The patients are generally categorized by the risk for recurrence into low, intermediate, and high risk.[7,11] Low-risk patients are those with stage IA grade 1 or 2. The intermediate-risk group includes stage IA grade 3 or stage IB grades 1 and 2, whereas the high-risk group includes patients with stage IB grade 3 tumors and stage II. All advanced stages III to IV diseases with tumors invading or spreading to the uterine serosa, adnexa, parametrium, vagina, retroperitoneal lymph node, or other distant organs are all considered as high-risk group. Adjuvant therapy is generally indicated for the patients in intermediate- or high-risk groups.

The adjuvant treatment for EC that has long been used for decades is external pelvic radiation therapy with or without brachytherapy.[10-12] Recent data reported that brachytherapy alone had similar efficacy in reduction in recurrence but with lesser adverse effects and better quality of life.[13] Despite a reduction in pelvic recurrence, radiation therapy does not yield any benefit on a reduction in distant failure or survival improvement.[14] Chemotherapy, which has previously been reserved for metastatic or recurrent EC, has also recently been investigated as adjuvant treatment with or without radiation therapy for patients with minimal or no gross residual diseases.[15] However, survival benefit is still inconclusive. Despite the potential benefit of adjuvant therapy, some patients who have risk for recurrence may decline or not be able to have additional treatment for many reasons such as suboptimal performance from the EC itself, other co-morbidities, or the myths about the “bad” effect of radiation therapy or chemotherapy.[16]

The aim of the Study is to evaluate the incidence of EC as per age, parity, stages, grades, clinical presentations, histological types, various correlations between grades, myo-invasion & Lymph node metastases &treatment modalities. Survival outcomes after treatment were also studied.

MATERIALS & METHODS
This study was a retrospective analytical study conducted in the Department of Gynecology Oncology, Acharya Harihar Regional Cancer Centre, Cuttack. With due permission of ethical committee all the required data were collected from the register of Endometrial carcinoma. The study includes 220 patients over a period of 7 years from January 2009 to December 2016. The following details are analysed thoroughly: age at diagnosis, parity, menopausal status, presenting symptoms, various preoperative investigations & their
features, surgical stagings, grades both nuclear & architecture.

Percentage of lymphnode positivity evaluated & correlation of grade, myo invasion & lymphnode positivity are studied. Requirement of post operative adjuvant therapy are studied in details as per categorizing patients into low, intermediate & high risk. The patients are followed up at scheduled interval. Though many a patients are lost for follow up, recurrences & residual diseases & death cases are searched & five year survival analysed.

**OBSERVATION & RESULTS**

Total no of 220 patients evaluated & analysed for the study.

**Table 1. Age Distribution (n = 220)**

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Number</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40</td>
<td>5</td>
<td>2.2</td>
</tr>
<tr>
<td>41-50</td>
<td>49</td>
<td>22.2</td>
</tr>
<tr>
<td>51-60</td>
<td>95</td>
<td>43.1</td>
</tr>
<tr>
<td>61-70</td>
<td>60</td>
<td>27.2</td>
</tr>
<tr>
<td>&gt; 70</td>
<td>11</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 1 analyses age distribution which shows about 43.1% patients present in the 5th decade of life. About 2.2% patients are of age 40 or younger & about 5% are of age 70 or above.

**Table 2. Incidence as per Parity (n = 220)**

<table>
<thead>
<tr>
<th>Parity</th>
<th>Number</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P0</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td>P1-P2</td>
<td>84</td>
<td>38.1</td>
</tr>
<tr>
<td>P3-P5</td>
<td>113</td>
<td>51.4</td>
</tr>
<tr>
<td>&gt;P5</td>
<td>12</td>
<td>5.4</td>
</tr>
</tbody>
</table>

Table 2 states that about 51.4% patients are of para 3 to 5 & about 5% are nulliparous in our study.

**Table 3. Histological Types of Endometrial Cancer**

<table>
<thead>
<tr>
<th>Histological Types</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endometrioid Adeno Ca.</td>
<td>177 (80.4)</td>
</tr>
<tr>
<td>Squamous Ca.</td>
<td>8 (3.6)</td>
</tr>
<tr>
<td>Clear Cell Ca.</td>
<td>11 (5)</td>
</tr>
<tr>
<td>Papillary Serous Ca. (UP SC)</td>
<td>21 (9.5)</td>
</tr>
<tr>
<td>Malignant Mixed Mullerian Tumor (MMMT)</td>
<td>3 (1.3)</td>
</tr>
</tbody>
</table>

Table 3 analyses various histological types of Endometrial cancer which indicates 80.4% are Endometrioid Adeno Carcinoma which constitutes the major group patients. Clear Cell Cancer constitutes 5% & Uterine Papillary serous (UPSC) 9.5%.

**Table 4. Surgical Staging (n=220)**

<table>
<thead>
<tr>
<th>STAGE</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>123</td>
<td>55.9</td>
</tr>
<tr>
<td>Stage II</td>
<td>54</td>
<td>24.5</td>
</tr>
<tr>
<td>Stage III</td>
<td>30</td>
<td>13.6</td>
</tr>
<tr>
<td>Stage IV</td>
<td>13</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Table 4 gives idea of analysis of comprehensive surgical staging which tells that about 55.9% patients present at an early stage i.e. at stage I. About 24.5% present at stage II & 13.6% present at stage III.

**Table 5. Nuclear Grading (As per HP, n=220)**

<table>
<thead>
<tr>
<th>GRADE</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>41</td>
<td>18.6</td>
</tr>
<tr>
<td>Grade 2</td>
<td>131</td>
<td>59.5</td>
</tr>
<tr>
<td>Grade 3</td>
<td>48</td>
<td>21.9</td>
</tr>
</tbody>
</table>

Table 5 reflects that maximum no of patients were moderately differentiated (G2) which constitutes about 59.5%. As Nuclear grade empowers the architectural grade by one grade, we have not taken architectural grade in to account in our study. About 18.6% patients are well differentiated (G1) & 21.9% are poorly differentiated.

**Table 6. Myometrial Invasion (n=220)**

<table>
<thead>
<tr>
<th>Myo invasion</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>177</td>
<td>80.5</td>
</tr>
<tr>
<td>&gt;50%</td>
<td>43</td>
<td>19.5</td>
</tr>
</tbody>
</table>

As per table 6, more than 50% myo invasion was present only in 19.5% of total patients.

**Table 7. Lymph node Status (n=220)**

<table>
<thead>
<tr>
<th>Lymph node status</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>No lymph nodes +</td>
<td>190</td>
<td>86.3</td>
</tr>
<tr>
<td>Pelvic LN+</td>
<td>23</td>
<td>10.4</td>
</tr>
<tr>
<td>Pelvic &amp; Para aortic LN+</td>
<td>7</td>
<td>3.1</td>
</tr>
</tbody>
</table>

Table 7 shows the lymph node status post CSS. Only 13% had positive LNs among which 10.4% was only pelvic and 3.1% had both pelvic & para aortic LNs. Isolated para aortic lymph nodes were not seen in any patient.
Fig 1. Comparison of Tumour grade and lymph node status

- 89.4% of Grade 2 tumours did not have lymph node metastasis (p=0.01). 31% of Grade 3 tumours had lymph node metastasis of which 16.5% were pelvic & 14.5% were para aortic lymph node metastasis. All cases with para aortic lymph node metastasis were grade 3 tumours.

Fig 2. Comparison of myo invasion and lymph node status

More than 50% myometrial invasion was found in 43 cases, of which 35% had lymph node metastasis (p=0.001). Out of this, 25% had pelvic lymph node metastasis & 10% had para aortic lymph node metastasis.

Table 8. Post-op Adjuvant Therapy (n=220)

<table>
<thead>
<tr>
<th>Adjuvant Therapy</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>No adjuvant therapy</td>
<td>29</td>
<td>13.2</td>
</tr>
<tr>
<td>Vault Brachy</td>
<td>94</td>
<td>42.7</td>
</tr>
<tr>
<td>EBRT + Brachy</td>
<td>54</td>
<td>24.5</td>
</tr>
<tr>
<td>RT→CT</td>
<td>30</td>
<td>13.6</td>
</tr>
<tr>
<td>CT</td>
<td>13</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Table 8 analyses requirement of post operative adjuvant therapy. About 13% cases did not require any adjuvant therapy. Maximum no of patients require both vault Brachy which is about 42.7%. About 13.6% cases require both Chemotherapy (CT) & radiotherapy (RT).

Table 9. Follow up of Treated cases of Ca Endometrium

<table>
<thead>
<tr>
<th>Time after treatment completion</th>
<th>Recurrence or residual disease</th>
<th>No evidence of disease</th>
<th>Lost for follow up</th>
<th>Reported death</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 months</td>
<td>0</td>
<td>170</td>
<td>26</td>
<td>0</td>
</tr>
<tr>
<td>6 months</td>
<td>10</td>
<td>154</td>
<td>29</td>
<td>1</td>
</tr>
<tr>
<td>one year</td>
<td>16</td>
<td>126</td>
<td>45</td>
<td>5</td>
</tr>
<tr>
<td>3 years</td>
<td>25</td>
<td>88</td>
<td>54</td>
<td>8</td>
</tr>
<tr>
<td>5 years</td>
<td>42(19%)</td>
<td>59(26.8%)</td>
<td>65(29.5%)</td>
<td>15(6.8%)</td>
</tr>
</tbody>
</table>

Table 9 analyses follow up of all cases but 29.5% at 5 years. At 6 months follow up about 4% have residual disease & 1 case of death was reported. At 5 yrs follow up about 19% cases had recurrence & 6.8% cases died.

DISCUSSION

Table 10. Comparison of various Histological Types of Endometrial Ca (with other studies)

<table>
<thead>
<tr>
<th>Types</th>
<th>Present study</th>
<th>James Fanning</th>
<th>Christopherson</th>
<th>Underwood</th>
<th>GCRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endometrioid Adeno Ca.</td>
<td>177(80.4)</td>
<td>274(66)</td>
<td>50(61)</td>
<td>155(70)</td>
<td>88(81.4)</td>
</tr>
<tr>
<td>Squamous Ca.</td>
<td>8(3.6)</td>
<td>21(5)</td>
<td>69(6)</td>
<td>16(7)</td>
<td>2(1.8)</td>
</tr>
<tr>
<td>Clear Cell Ca.</td>
<td>11(5)</td>
<td>14(3)</td>
<td>43(5)</td>
<td>2(1)</td>
<td>2(1.8)</td>
</tr>
<tr>
<td>Uterine Papillary Serous Ca. (UPSC)</td>
<td>21(9.5)</td>
<td>4(3.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Mixed Mullerian Tumor (MMMT)</td>
<td>3(1.3)</td>
<td>5(4.6)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 10 compares various histological types of Endometrial carcinoma. In the present study most common histological type is Endometrioid adeno carcinoma which constitutes about 80%. Another Indian study at GCRI revealed 81% cases of Adeno carcinoma which are more than that of various western study by James Fanning, Christopherson & Underwood. In the present study Clear Cell & Uterine Papillary Serous Carcinoma constitutes 5% & 9.5% respectively. As per WHO ISGP study, Endometrioid Adeno Ca. constitutes 75-80%, UPSC 10% & Clear Cell 4%. In the present study three cases of Malignant Mixed Mullerian Tumor (MMMT) was found among one who had a prior history of Radiotherapy treatment for cancer cervix 18 years back.
In the present study maximum no. of patients are multipara with parity 3 to 5 & present at 5th decade of life. Though nulliparity is a risk factor for endometrial cancer, only 5% are nulliparous. As per study by Fanning et al, 23% of patients of adenocarcinoma are nulliparous.

Surgery is the primary modality of treatment in early stage endometrial cancer. In 1988, the inadequacies of pretreatment diagnostic assessment of endometrial cancer led to recommendations for routine surgical staging.[17] Surgicopathological staging not only provides information about the exact extent of the disease but classify the risk factors basing on which adjuvant treatment may or may not be given so predicts the treatment outcome. Surgical staging is a step wise procedure where careful exploration of all intrabdominal contents is done. The omentum, liver, peritoneal cul-de-sac and adenexal surfaces are palpated for possible metastases, followed by careful palpation of suspicious or enlarged nodes in aortic and pelvic areas. Therecommended procedure is an extrafascial total hysterectomy with bilateral salpingo-oophorectomy even if both adenexae appear normal, as they may contain micro metastases. The role of peritoneal fluid cytology is controversial as it rarely affects the prognosis as per some western studies[18]. FIGO staging 2008 also mentions that positive cytology has to be reported separately without changing the stage[19]. Vaginal cuff removal has neither surgical nor survival benefit. Parametrial tissue removal is not necessary in stage I and IIa. The decision for lymphadenectomy in the present study were decided basing on naked eye examination of specimen especially growth size, myoinvasion due to lack of frozen section.

Analysis of surgical staging of all patients revealed that maximum no. of patients belong to Stage I & about 5.9% belong to stage IV. Comprenhensive surgical staging of G1-2 disease with less than 50% myoinvasion without other risk factors is controversial. On contrary, as per SGO & ACOG guideline “Most women with endometrial cancer should undergo complete systematic surgical staging (including assessment of lymph nodes) to help determine appropriate management”. As per the study by Kilgore and associates, there is definite survival advantages in both low and high risk patients with more extended nodal dissection and recommended lymphadenectomy for all[20].

In the present study, 31% of Grade 3 tumours had lymph node metastasis of which 16.5% were pelvic & 14.5% were paraaortic lymph node metastasis. All cases with para aortic lymph node metastasis were grade 3 tumours. More than 50% myometrial invasion was found in 43 cases, of which 35% had lymph node metastasis.(p=0.001). Out of this, 25% had pelvic lymph node metastasis & 10% had para aortic lymph node metastasis. Large Study group needed to reach at conclusion. But this pattern of Node positivity undoubtedly reflects that tumour grade & depth of invasion are the factors which decides the node positivity so also responsible for need & type of adjuvant radiation. Myometrial invasion is a well recognized predictive factor for extra uterine spreading especially lymph node metastases. Patients with deep myometrial invasion were found more likely to have pelvic and para aortic lymphnode metastases and advanced stage disease compared to those with superficial myometrial invasion. Therefore it is important to assess the depth of myometrial invasion preoperatively and intraoperatively to determine the extent of lymphadenectomy[21].

As per a GOG study[22] by Creasman, Marrow et al pelvic Node Positivity is 3% for G1 Carcinoma confined to inner 1/3rd Myometrium & 34% for G3 Carcinoma confined to outer 1/3rd Myometrium. Grade I tumour with less than 2 cm diameter, limited to endometrium or only with superficial invasion can be included under low-risk category, as the risk of lymphnode metastases in such patients is up to 4%[23]. So omitting lymphadenectomy may be riskier in these 4% patients but at the same time proceeding for the procedure is unnecessary for the rest 96% increasing the operative morbidity. In the UK, the MRC ASTEC trial, which randomised women undergoing surgery for presumed stage I endometrial cancer to pelvic lymphadenectomy or no lymphadenectomy, showed no therapeutic benefit[24].

Table 8 analyses the need of adjuvant therapy. In the present study, about 25% patients received both External Beam Radio Therapy (EBRT) & Brachytherapy. 13.2% patients did not require any adjuvant as they belong to Low risk category (G1, G2
It is evident that most women with early stage and low risk disease did well without adjuvant radiation therapy. Intermediate risk patients (G1, G2 with >50% myoinvasion, No cervical extension, No LVSI) are at risk for loco regional relapse, and radiotherapy has been shown to effectively reduce this risk without significantly impacting overall survival. Most studies indicate that advanced age, grade 3 histology, deep myometrial invasions, cervical extension, adnexal spread, lymph node positivity, Clear Cell & UPSC histology type relate to a high risk of disease recurrence. Postoperative radiotherapy in such cases has demonstrated significant reduction in the 5-year vaginal and pelvic recurrence rate after RT, but without any overall survival benefit and higher rate of treatment related complications (PORTEC study), and endometrial carcinoma related death rates were 9% in RT group and 6% in control group [25]. A study by Denny L, Hecker NF et al. indicates adjuvant Radio therapy for high risk cases (G3 with >50% myoinvasion) is associated with 10% survival advantage [26]. Vaginal irradiation (IVRT) given in intermediate risk group improve local control [27]. In the present study 30% patients received both Chemo therapy & radiotherapy & most of them had distant metastases & systemic diseases.

Table 9 analyses the follow up of cases which reveals that about 4% had residual disease at 6 months, 19% cases had recurrence at 5 years. 70% cases had no evidence of disease at 6 months & 26.8% at 5 year. About 13% lost for follow up at 6 months & 30% at 5 year. 1 case of death at 6 month & 15 cases of death reported at 5 years. Due to maximum no. of cases of lost for follow up, exact figure of recurrence & 5 year survival could not be properly determined. Pelvic recurrence after Surgery only group are treated with RT except 2 cases where pelvic exenteration was done. Most cases of extra pelvic recurrences are the patients who had received both surgery & adjuvant RT earlier. For these patients chemotherapy with platinum and taxanes were administered.

CONCLUSION

Endometrioid Adeno Carcinoma is the most common type of Endometrial Cancer which usually present at 5th decade of Life. Though nulliparity is a risk factor mentioned in literature present study reveals that multiparous women belong to the major group. The most aggressive variety like Clear Cell Ca & UPSC usually present at later age than adenocarcinoma. Another interesting fact is the mean age of presentation of all histological types are higher than western counterpart. Though Significant progress has been made over the last two decades in understanding of prognostic factors in endometrial cancer, controversies still exist such as adoption of systemic surgical staging procedure for treatment and risk stratification. The present study reveals that most of patients with Ca Endometrium present in stage I & are moderately differentiated (G2). The present study reveals that Grades & Myo invasion are two important factors for Lymph node positivity thus for prognosis & need for adjuvant therapy but the results are not statistically significant. The current controversy of role of lymphadenectomy remains to be established. So larger study group needed to solve the issue & to reach at a conclusion. About 15% patients in the present study did not receive any adjuvant therapy as they belong to low risk group. But exact proportion of those patients who remained disease free & significant survival could not be evaluated as most of patients did not came for proper follow up. We emphasis that the standard recommendation from evidence-based data should be followed and recommended to the patients. This was especially applicable in patients with advanced staged diseases when the benefit of adjuvant therapy had been clearly demonstrated. Nevertheless, a number of patients with EC were elderly individuals with co-morbidities or had social and belief issues making postoperative treatment unsuitable. Furthermore, some patients with low-risk EC may have other coincidental pathology requiring treatment. Hence, tailoring the adjuvant therapy to each individual is crucial in real clinical practice. Issues concerning the patient population’s social and educational background should be considered before integration of new standards of treatment. Prospective randomized studies to explore these issues are needed to resolve current controversies.

REFERENCES


19. Meeting report the new FIGO Staging systems for Cancers of Vulva, Cervix, Endometrium & Sarcoma, Gynecol Oncol;115(2009) 325-328


Association of Human Papilloma Virus infection with Bacterial Vaginosis: An Ongoing Study at AHRCC, Cuttack

Pallavi Patnaik1, Bhagyalaxmi Nayak2, Janmejaya Mohapatra2, Manoranjan Mohapatra2, Dhananjaya Soren3, Priyadasini Patra4

Abstract: Bacterial vaginosis is one of the most prevalent causes of abnormal vaginal discharges, affecting women of reproductive age. This infestation is characterized by loss of indigenous lactobacillus predominant vaginal micro flora and a concurrent massive growth of anaerobic bacteria. The most common anaerobic organisms are Gardnerellavaginalis, Mobiluncus species, Prevotella, Mycoplasma and Atopobiumspecies. BV increases the risk of upper genital tract infection and adverse outcomes of pregnancy. Association between BV and cervical HPV has been inconsistent among studies conducted so far. The objective of this particular research is to clarify and summarize the co-relationship of human HPV infection with BV: In cases and controls respectively.

Keywords: Bacterial vaginosis, Human papilloma virus, anaerobic bacteria.

Introduction: Innate and cell mediated immunity are important for control and resolution of HPV infections. HPV can suppress or hide from protective immune responses. In addition to very low levels of antigen expression the keratinocyte is an immunologically privileged site for replication. The L1 protein of HPV is the viral attachment protein and initiates replication by binding to α6 integrins and heparan proteoglycans, the membranes of the lower basal keratinocytes and especially those of the transformation zone of the cervix on the cell surface. This is an area of immature epithelial cells that are especially hormone sensitive in women of reproductive age groups and thus are susceptible to infections and mutations. As basal cells differentiate and progress to the surface of the epithelium HPV DNA replicates and is transcribed and viral particles are assembled in the nucleus. Ultimately complete virions are released tightly associated with the remnants of the shed dead keratinocyte shell. Normal appearing epithelium may contain HPVDNA and the presence of residual DNA after the treatment of warts may lead to recurrent disease. In benign lesions caused by HPV, viral DNA is located extrachromosomally n the nuclei of infected cells however when HPV DNA is detected in high grade intraepithelial neoplasias and cancers, it is generally integrated. Integration of HPV DNA may occur at preferential sites in host cell chromosomes and it specifically disrupts the E2 ORF. Interruption of E2 probably plays a role in the pathogenesis of malignant disease because expression of this ORF normally leads to down regulation of E6 and E7. E6 and E7 genes of the high risk HPV types are by themselves true oncogenes since when they transfect normal keratinocytes in vitro, they can induce carcinogenesis: their products interact and inactivate tumor suppressor proteins, such as p53 and retinoblastoma protein with concomitant release of E2F transcription factor. As a consequence of the above, p16INK4A and telomerase expression is unregulated, cyclin inhibitors are inactivated and cyclins are activated and chromosomal instability and aneuploidy and apoptosis evasion occur. In warts or condylomata, viral replication is associated with proliferation of all epidermal layers except the basal layer. This leads to acanthosis, parakeratosis, hyperkeratosis and deepening of rete ridges, creating the typical papillomatuscylotarchitecture seen histologically. Some infected cells undergoing the characteristic transformation of koilocytosis. With histology, koilocytes are large usually polygonal, squamosalcells with a shrunken nucleus lodged inside...
a large cytoplasmic vacuole (Huh et al, 2009). Cytoplasmic keratin-hyalin inclusion bodies may also be observed. Excessive proliferation of the basal like cells with a high nuclear/cytoplasmic ratio, accompanied by a high number of mitoses, some abnormal s a feature of incipient and premalignant disease.

**Co-relation of BV with HPV infection and CIN.**

Mucolytic enzymes, musinases including those which degrade terminal carbohydrate residues or those which disrupt the mucinaprotein primary structure are produced by some bacteria associated with BV. Previous work has shown that the presence of glycosidases, including sialidase, is frequently associated with BV. Sialidases remove terminal sialic residues in various sialoglycoconjugate carbohydrate chains and as the negative charge in these molecules is lost this may affect visco-elasticity in mucins or other binding properties associated with sialoglycoconjugates. This may partially explain the thin nature of the vaginal discharge frequently associated with this condition. Till date sialidase is the only enzyme with mucinase activity suggested as a virulence determinant of BV associated micro-organisms, Prevotella and Bacteroides spp. Intensive production of hydrolytic enzymes in BV might lead to diminished mucosal barrier protection through disruption of secreted mucus visco-elasticity and deglycosylation of the apical cell surface glycocalyx. In addition key enzymes produced in BV may degrade the mucosal barrier, mediate bacterial adhesion to host surfaces, and provide energy sources for the bacteria, thus enhancing bacterial colonization of the mucosa. Another hypothesis proposes that mucin-degrading enzymes are increased in vaginal fluid of women with BV. These enzymes, like sialidases play a role in degradation of the gel layer coating the cervical epithelium, causing micro-abrasions or alterations of epithelial cells. Such enzymes may promote virulence through destroying the protective mucosa barrier and hence increase susceptibility to cervical HPV infection by facilitating adherence, invasion and eventually incorporation of HPV oncogenes into the genome of cells of the transformation zone. Both nitrites and aminases, the elements for nitrosamine synthesis are produced by the abnormal and predominant anaerobic vaginal flora of patients with BV. As abnormal microflora can also stimulate the release of cytokines, such as interleukin-1α, which has been suggested to be important in the development of cervical cancer. Pavic et al suggested that locally produced nitrosamines may act synergistically with other etiological agents in the development of cervical neoplasia. Carcinogenic nitrosamines increase the probability of DNA damage and an altered cytokine profile may reduce immune systems ability to eliminate HPV infection. Thus the changes may create a conducive environment for cancer development. In support of this, increased levels of IL-1α were seen in the cervical mucous of pregnant patients with BV. Furthermore, cervical mucus from these women with BV induced production of IL-6 from monocytic cells in-vitro. In another study a shift to TH2 cytokines was observed with increasing grades of cervical dysplasia. Therefore changes in the microbial flora, cytokine profile, or both may predispose to cervical dysplasia. (Pavicic et al 1984)

It has also been suggested that a raised vaginal pH may arrest squamous metaplasia in the post pubertal cervix and prolong the period in which the transformation zone is vulnerable to agents promoting dysplasia such as human HPV. Therefore, BV will interact with HPV infection with the consequence of higher risk of developing cervical cancer among those with BV and HPV-infection than those with a mono-infection. However other infections for instance Candida was not associated with the development of CIN or cervical carcinoma. (Warner et al, 2003)

**Materials and Methods**

Place of research: This prospective study was funded by DST, Odisha, and was conducted in the Department of Gynecologic Oncology, AHRCC, Cuttack in collaboration with the Departments of G and G, Microbiology, and Pathology SCBMCH, Cuttack along with the Virology Laboratory of RMRC, Bhubaneswar.

Study Population: Women aged between 19-49 years attending the gynecologic OPD of SCBMCH and AHRC, Cuttack were included in the study and consent was obtained from the women who were included in the prospective study.
Exclusion Criteria: The following women were excluded in the study such as:

a) Unmarried women  
b) Pregnant women  
c) Vaginal bleeding at the time of clinical examination  
d) Women who have undergone hysterectomy

Preparation of questionnaire: A research questionnaire was obtained from the participating women regarding their socioeconomic status, age of sexual debut, number of sexual partners during their lifetime, current contraception method, and history of STDs if any.

Collection and Processing of Specimen:

a) 3 high vaginal swabs were collected from each patient by sterile cotton tip swabs from posterior vaginal fornix, the swabs were dipped in 0.5ml normal saline and the characteristics of vaginal discharge such as color, nature, consistency, odor etc were recorded. The material from the cervix was collected using Ayres spatula around the external os including squamocolumnar junction by 360% rotation with minimum pressure and uniformly spread by rotator smear technique on a single glass slide permanently marked by a diamond pencil. The slide was then immersed in a fixing solution and was allowed to remain in the fixative for at least half an hour to ensure adequate fixation and then stained by Papanicolaou technique and evaluated under Bethesda III system for specimen adequacy and general categorization. Additional endocervical cells were collected using a Dracon swab and was transferred into tubes containing PBS stored at 70% until further processing. Extraction of total DNA, was done using QIAamp DNA mini kit according to manufacturer’s instructions.

b) pH of vaginal discharge was recorded at the OPD using standard pH indicator paper with a range of 2-10.5.

c) The amine test was performed by adding a few drops of 10% KOH solution directly over the soaked swab to detect if there was any emission of amine like odor.

d) Clinico-microbiological diagnosis of BV was made on the basis of presence of any of the 3 criteria as described by Amsel et al 1984 such as:

1) A grey homogenous fishy smelling vaginal discharge.  
2) Vaginal pH e” 4.5.  
3) Presence of clue cells in wet mount preparation of vaginal discharge.  
4) The positive amine test in which a fishy odor is released after addition of 10% KOH of vaginal fluid.

e) The presence of clue cells was identified using Grams staining technique, and also for the identification of Gram negative or gram variable bacilli, budding yeast cells, polymorphs etc. A scoring of the vaginal flora pattern was done according to the scoring system of Nugent et al 1991.

f) The results of PAP smears were reported according to Bethesda III system (2001).

g) DNA extraction from cervicovaginal smear was done by manual extraction using QIAamp DNA Kits.

h) PCR amplification of the extracted DNA was performed using consensus degenerate primers(MY09 and MY11) derived from highly conserved L1 open reading frame of the HPV genome with an amplicon size of 450bp.

Results and Discussion

In the present study a total of 368 vaginal/cervical samples were processed and the results were analyzed. Out of 368, 7.06% samples revealed the presence of Candida species and 2.44% samples had presence of Trichomonas vaginalis (Table 1). Hence those samples were excluded from further study. Following Amsel’s microbiological criteria and taking any three positive criteria into consideration, presence of BV could be assessed in 30.93% out of 333 women (Table 2). By employing the widely accepted Gram stain scoring system of Nugent et al, out of 333 women, 26.12% women were diagnosed with BV, 202 women had an intermediate score and 44 women had a normal score (Table 3). In the present study findings with overall presence of BV is 30.93% (Table 4) which is
in agreement with most of the studies carried so far that showed different rates ranging from 17% - 37%. But higher rates of BV have been reported in some studies carried out in developing countries. In a study from Haryana BV was diagnosed in high percentages amongst rural women (Garg et al 2002). In the present study BV was found to be associated with older age groups. More than 75% of BV cases from the study varied between 25-45 years of age. (Table 5). This is in consistence with the study carried out by Madhavan et al. 2008. However it has been established by previous research that the condition is more common amongst younger women, while others have found that the risk of BV increases with age. (Moncla et al, 1990). Table 6 shows the association between socio demographic profile of the study population and prevalence of BV. All women in the study were reportedly married, with 10 women living with another partner apart from their spouse, four were widowed and four divorced. Almost 26% of the women were illiterate and did not receive any schooling. 74.17% of the women in the study population were either housewives or domestic maids. In addition to running a household, 20.12% of women worked as unskilled agricultural laborers and 5.7% of women reportedly were employed variously. None of the women in the study population were commercial sex workers. 60% of the target study cohort had made their sexual debut before 18 years of age. Condom use was almost 3% and reversible methods of contraception were rarely used. 56.45% of the participants had undergone tubal ligation. None of the women taken into consideration used multiple contraceptive methods. Cigarette smoking, douching and drug use were not reported by any woman in the study population. The highest prevalence of BV in this study was reported in urban slums (38.83%), and urban middle class community (29.12%). Although our study does not have adequate information on partner’s sexual behavior, studies suggest that the sexual habits and characteristics of the male partner may play a vital role in the development of BV. (Jones et al, 2007). There is however a limitation in our study. Since risk factors were self reported and it is possible that there may be under-reporting and misclassification of risk behaviors. This study involved collection of sensitive sexual behaviors as well as information on women’s sex partners so there is a possibility of measurement error that may lead to residual confounding effect obscuring the relationship between BV and risk factors.

Out of 103 cases of BV, the common symptom found was vaginal discharge in (84.4%), followed by lower abdominal pain (63.10%) and pruritus (27.18%)(Table 7). A number of studies explored the association of vaginal discharge with vaginal infections. With regards to clinical manifestations of vaginal infections among symptomatic women, all these studies found a variable degree of association between complaints of vaginal discharge and vaginal infections. (Bornstein et al, 2001). Clue cells in the wet mount were found to be significantly associated with BV as shown by its specificity(98.7%) and positive predictive value(78.27%)(Table 8). The findings on positive clue cells also support previous studies that found it to have the highest specificity of the individual clinical criteria for diagnosing BV. (Gutman et al, 2005). The present study has shown that the incidence of CIN was significantly higher in cases than in controls. (X= 18.44, p<0.001)(Table 9) but the presence of BV was not associated with the severity of CIN (p=0.76)(Table 10) and as per the co-relationship between BV and HPV infection, this study revealed a somewhat positive correlation between these two very common conditions, with an overall estimated odds ratio of 7.63. (95% confidence interval : 4.2 – 13.7)(table 11). These data are consistent with the results of four previous studies i.e.; 3 cross-sectional and I prospective that found an increased rate of HPV infection in women with BV.

However the question still remains whether BV and cervical HPV infection are simply related because there is a biological interaction between them or because both occur frequently in sexually active women. A positive co-relationship between BV and HPV might be explained by the fact that sexual risk behavior and promiscuity are found more often in women with BV than in comparison groups. A number of variables are contributing to the observed heterogeneity in previous studies. Various social habits and ethno-geographical risk factors may explain the wide BV prevalence range observed (3%- 47.2%). Technical biases such as collection of specimen, subjectivity, sensitivity, and specificity of diagnostic methods are also attributing to detected heterogeneity.
Complete STD screening was not performed for the present study which may have confounding effects to a certain degree on the results of the present study as well. Thus further research in this field is imperative.

The present prospective study comprises evaluation of 368 vaginal/cervical swab specimens collected from 368 women attending the gynaecology OPD of SCB & AHRCC Cuttack.

**TABLE: 1**

<table>
<thead>
<tr>
<th>Pathogens</th>
<th>Total no. Vaginal swabs n=368</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candida species</td>
<td>26 (7.06%)</td>
</tr>
<tr>
<td>Trichomonasvaginalis</td>
<td>9 (2.44%)</td>
</tr>
<tr>
<td>Others</td>
<td>333 (90.48%)</td>
</tr>
</tbody>
</table>

Out of the 368 numbers of vaginal swabs 26 were positive for Candida species and 9 revealed Trichomonasvaginalis. Hence these women were excluded from further study.

**TABLE: 2**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Total number of patients n=333</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homogeneous discharge</td>
<td>103 (30.93%)</td>
</tr>
<tr>
<td>pH &gt; 4.5</td>
<td>90 (27.02%)</td>
</tr>
<tr>
<td>Presence of clue cells</td>
<td>43 (12.91%)</td>
</tr>
<tr>
<td>Positive amine test</td>
<td>120 (36.03%)</td>
</tr>
<tr>
<td>Any 3 criteria positive</td>
<td>103 (30.93%)</td>
</tr>
</tbody>
</table>

Taking into consideration any of 3 positive criteria 103 out of 333 women were diagnosed with bacterial vaginosis according to Amsel’s criteria.

**TABLE: 3**

<table>
<thead>
<tr>
<th>Scoring of bacterial flora</th>
<th>No. of patients n=333</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 3 (normal score)</td>
<td>44 (13.21%)</td>
</tr>
<tr>
<td>4 – 6 (intermediate score)</td>
<td>202 (60.66%)</td>
</tr>
<tr>
<td>7 – 10 (BV score)</td>
<td>87 (26.12%)</td>
</tr>
</tbody>
</table>

According to Nugent’s criteria, Gram stains of 26.12% were positive for bacterial vaginosis, 13.21% had normal score and 60.66% had intermediate score.

**TABLE: 4**

<table>
<thead>
<tr>
<th>Total no. of women n= 333</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases 103 (30.9%)</td>
</tr>
<tr>
<td>Controls 230 (69.10%)</td>
</tr>
</tbody>
</table>

Taking Amsel’s criteria into consideration prevalence of bacterial vaginosis was found to be 30.9% in our study which were considered as Cases and the rest 69.10% were considered as Controls.

**TABLE: 5**

<table>
<thead>
<tr>
<th>Demographic data</th>
<th>Total no. n=333</th>
<th>Cases n=103</th>
<th>Controls n=230</th>
</tr>
</thead>
<tbody>
<tr>
<td>Religion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hindu</td>
<td>229 (68.76%)</td>
<td>52 (25.3)</td>
<td>177 (77.3)</td>
</tr>
<tr>
<td>Muslim</td>
<td>96 (28.82%)</td>
<td>10 (9.7)</td>
<td>86 (36.95)</td>
</tr>
<tr>
<td>Christian</td>
<td>9 (2.42%)</td>
<td>1 (1.0)</td>
<td>8 (3.45)</td>
</tr>
<tr>
<td>Literacy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Literate</td>
<td>246 (73.67%)</td>
<td>41 (39.8)</td>
<td>205 (88.56)</td>
</tr>
<tr>
<td>Illiterate</td>
<td>87 (26.33%)</td>
<td>62 (60.2)</td>
<td>47 (21.44)</td>
</tr>
<tr>
<td>Location</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban Slum</td>
<td>199 (59.75%)</td>
<td>46 (45.1)</td>
<td>153 (66.52)</td>
</tr>
<tr>
<td>Urban middle class</td>
<td>77 (23.12%)</td>
<td>18 (17.5)</td>
<td>59 (25.64)</td>
</tr>
<tr>
<td>Rural</td>
<td>73 (21.53%)</td>
<td>30 (29.1)</td>
<td>43 (18.86)</td>
</tr>
<tr>
<td>Age of 1st sexual contact</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Below 15</td>
<td>135 (39.6%)</td>
<td>37 (35.6)</td>
<td>98 (42.6)</td>
</tr>
<tr>
<td>Above 15</td>
<td>199 (59.75%)</td>
<td>52 (50.4)</td>
<td>147 (64.4)</td>
</tr>
<tr>
<td>No. of sexual partners</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>323 (96.9%)</td>
<td>99 (95.2)</td>
<td>224 (97.4)</td>
</tr>
<tr>
<td>More than 1</td>
<td>10 (3.1%)</td>
<td>4 (3.8)</td>
<td>6 (2.6)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hindu</td>
<td>229 (68.76%)</td>
<td>52 (25.3)</td>
<td>177 (77.3)</td>
</tr>
<tr>
<td>Muslim</td>
<td>96 (28.82%)</td>
<td>10 (9.7)</td>
<td>86 (36.95)</td>
</tr>
<tr>
<td>Christian</td>
<td>9 (2.42%)</td>
<td>1 (1.0)</td>
<td>8 (3.45)</td>
</tr>
<tr>
<td>Current contraception</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not using birth control</td>
<td>246 (73.67%)</td>
<td>41 (39.8)</td>
<td>205 (88.56)</td>
</tr>
<tr>
<td>Using IUD</td>
<td>86 (26.33%)</td>
<td>62 (60.2)</td>
<td>47 (21.44)</td>
</tr>
<tr>
<td>Oral contraceptive pill</td>
<td>146 (43.85%)</td>
<td>46 (45.1)</td>
<td>100 (43.5)</td>
</tr>
<tr>
<td>Condom</td>
<td>313 (93.6%)</td>
<td>18 (17.5)</td>
<td>125 (54.31)</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>30 (9.0)</td>
<td>30 (29.1)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Professions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>House wife</td>
<td>247 (74.27%)</td>
<td>85 (81.3)</td>
<td>162 (70.8)</td>
</tr>
<tr>
<td>Unmarried</td>
<td>76 (22.7)</td>
<td>32 (30.6)</td>
<td>44 (18.7)</td>
</tr>
<tr>
<td>Others</td>
<td>19 (5.7)</td>
<td>4 (3.8)</td>
<td>15 (6.5)</td>
</tr>
</tbody>
</table>

Prevalence of BV was highest between 25 – 40 years of age group and minimum in >40 years age group by both Amsel’s criteria and Nugent’s scoring system.

**TABLE: 6**

<table>
<thead>
<tr>
<th>Co-relation between bacterial vaginosis and socio demographic and risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographic data</td>
</tr>
<tr>
<td>Religion</td>
</tr>
<tr>
<td>Hindu</td>
</tr>
<tr>
<td>Muslim</td>
</tr>
<tr>
<td>Christian</td>
</tr>
<tr>
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<td>No. of sexual partners</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>More than 1</td>
</tr>
<tr>
<td>Race</td>
</tr>
<tr>
<td>Hindu</td>
</tr>
<tr>
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</tr>
<tr>
<td>Professions</td>
</tr>
<tr>
<td>House wife</td>
</tr>
<tr>
<td>Unmarried</td>
</tr>
<tr>
<td>Others</td>
</tr>
</tbody>
</table>
Out of the total 333 women included in the study, 199 belonged to urban slums while 77 and 57 belonged to urban middle class and rural communities respectively. The majority of women included in the study were not working (74.17%) and most of them were Hindus, literate and non-smokers. Women with BV were on the average Hindus, house wives had their sexual debut before 18 yrs of age and living with their partners for more than 10 years and belonged to urban slums. There was no significant difference in other base line demographic characteristics between the 2 groups i.e. Cases and Controls.

**TABLE:- 7**

Co-relation of symptoms with bacterial vaginosis

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Laboratory diagnosis</th>
<th>Cases (n=103)</th>
<th>Controls (n=230)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal vaginal discharge (%)</td>
<td>87 (84.4%)</td>
<td>16 (6.95%)</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Lower abdominal pain (%)</td>
<td>66 (63.10%)</td>
<td>24 (23.47%)</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Vaginal discharge and lower abdominal pain (%)</td>
<td>37 (35.92%)</td>
<td>10 (4.34%)</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Pruritus (%)</td>
<td>28 (27.18%)</td>
<td>35 (15.21%)</td>
<td>0.0001</td>
<td></td>
</tr>
</tbody>
</table>

Maximum number (84.4%) of cases presented with abnormal vaginal discharge followed by lower abdominal pain (63.1%) and Pruritus (27.18%). Among Controls, lower abdominal pain was the most predominant (23.47%) symptom.

**TABLE:- 8**

Statistical evaluation of clue cell and Amine test in diagnosis of BV.

<table>
<thead>
<tr>
<th>Test</th>
<th>TP</th>
<th>FP</th>
<th>TN</th>
<th>FN</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clue cell</td>
<td>40</td>
<td>3</td>
<td>227</td>
<td>63</td>
<td>38.83%</td>
<td>98.7%</td>
<td>93.02%</td>
<td>78.27%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Amine test</td>
<td>85</td>
<td>35</td>
<td>195</td>
<td>18</td>
<td>55.3%</td>
<td>90.4%</td>
<td>79.58%</td>
<td>&lt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

TP: True positive, FP: False positive, TN: True negative, FN: False negative, PPV: Positive predictive value, NPV: negative predictive value.

The sensitivity and specificity of clue cells was found to be 38.83% and 98.7% respectively whereas, Amine test had higher sensitivity (82.52%) but lower specificity (84.78%) but both the above criteria were significantly associated with BV.

**TABLE:- 9**

Co-relation between bacterial vaginosis with CIN and ASCUS

<table>
<thead>
<tr>
<th>CIN Present (%)</th>
<th>CIN Absent (%)</th>
<th>ASCUS (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases (n=103)</td>
<td>64 (62.1)</td>
<td>27 (26.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Controls (n=230)</td>
<td>59 (43.8)</td>
<td>157 (56.2)</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Among 103 women with BV (Cases), CIN was diagnosed in 64 women and among the 230 women without BV (Controls), CIN was diagnosed in 99 women. The incidence of CIN was significantly higher in Cases (p<0.001).

**TABLE:- 10**

Co-relation between bacterial vaginosis and severity of CIN

<table>
<thead>
<tr>
<th>CIN</th>
<th>Cases (n=103)</th>
<th>Controls (n=230)</th>
<th>Total</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low grade</td>
<td>25 (33.86%)</td>
<td>44 (44.11%)</td>
<td>69 (40.49%)</td>
<td>0.76</td>
</tr>
<tr>
<td>High grade</td>
<td>39 (60.64%)</td>
<td>38 (50.59%)</td>
<td>77 (59.51%)</td>
<td>0.76</td>
</tr>
<tr>
<td>Total</td>
<td>64 (100%)</td>
<td>82 (100%)</td>
<td>146 (100%)</td>
<td>0.76</td>
</tr>
</tbody>
</table>

When CIN patients were sub-divided into low grade CIN I group and high grade CIN II/III groups, lower grade was present in 40.49% women and high grade was present in 59.51% women, 39.06% No. of Cases had low grade CIN and 60.04% No. of Cases had high grade CIN, and the presence of BV was not associated with the severity of CIN (p=0.76).

**TABLE:- 11**

Co-relation of BV with HPV infection

<table>
<thead>
<tr>
<th>HPV Status</th>
<th>Cases (n=103)</th>
<th>Controls (n=230)</th>
<th>Total</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPV DNA negative</td>
<td>55.3%</td>
<td>90.4%</td>
<td>79.58%</td>
<td>0.05</td>
</tr>
<tr>
<td>HPV DNA positive</td>
<td>44.7%</td>
<td>9.6%</td>
<td>24.2%</td>
<td>0.05</td>
</tr>
</tbody>
</table>

(Odds ratio = 7.63; 95% Confidential interval 4.2 – 13.7)

Among the total 333 no. of women 20.42% were positive for HPV DNA, HPV DNA was present in 44.7% of Cases and in 9.6% of Controls. There may be a significant co-relation between BV and HPV infection (p=0.05).
Summary and Conclusion

The present study entitled “Association of Human Papilloma Virus infection with Bacterial Vaginosis, in Cases and Controls: An Ongoing Study at AHRCC, Cuttack” was funded by DST, Odisha, and was conducted in the Department of Gynecologic Oncology, AHRCC, Cuttack in collaboration with the Departments of Oand G, Microbiology, and Pathology SCBMCH, Cuttack along with the Virology Laboratory of RMRC, Bhubaneswar, with an objective to ascertain the following:

a) The prevalence of BV among the study population.

b) To determine the association of BV with development of CIN and acquisition of HPV infection.

A total of high vaginal/cervical swabs were evaluated. 7.06% were positive for Candida species and 9 revealed Trichomonas vaginalis. Hence these women were excluded from further study. The HVS were subjected to pH determination, Amine test, and Clue cell detection. All cervical swabs were also subjected to PAP smear and HPV DNA testing by conventional PCR method. Following the clinical microbiological criteria of Amsel et al, BV was diagnosed in 30.93% out of the total 333 women. Interpretation of Gram staining scoring by Nugent’s method revealed 26.12% to be positive for BV, 13.21% had normal score and 60.66% had an intermediate score. Taking Amsel’s criteria into consideration prevalence of BV was found to be 30.9% in our study which were considered as cases and the rest 69.10% were considered as controls. Maximum numbers of BV cases were from patients who complained of abnormal vaginal discharge (84.4%) and were detected in the age group of 25-40yrs (76.09%). Clue cells were found in 12.91% out of the total samples. The sensitivity and specificity of clue cell detection in relation to diagnosis of BV was 38.83% and 98.7% respectively. The positive and negative predictive values were 93.02% and 78.27% respectively. Clue cell detection was significantly associated with diagnosis of BV (pd”0.05). With respect to diagnosis of BV the Amine test had a sensitivity of 82.52% and a specificity of 84.78%. Positive predictive value of the test was found to be 70.83% and it was also significantly associated with BV.

The highest prevalence of BV was seen in urban slums (30.83%) followed by rural community (32.03%) and urban middle class community (29.12%). In this specific study more Hindu women had BV in comparison to women belonging to other religions. Other known socio-demographic factors such as douching and smoking were not associated with BV in the study population. Among 103 women with BV (cases), CIN was diagnosed in 64 women and in 230 women without BV (controls), CIN was diagnosed in 99 women. The incidence of CIN was significantly higher in cases (pd”0.0001), however the presence of BV was not associated with the severity of CIN (p=0.76). Among the total 333 women, 68 were positive for HPV DNA. HPV DNA was present in 46 cases and in 22 controls. Thus there appears to be a link between BV and HPV infection. Thus the present study suggests a somewhat significant association between BV, presence of CIN and cervical HPV infection.

However cervical screening remains of course a major preventive focus for the cancer control program. If BV is a risk factor for cervical HPV infection, it’s but natural that screening guidelines must adapt and implement a sensitive tool like HPV DNA testing in primary screening of BV positive women instead of cytological testing. If a longitudinal prospective study shows a cause effect model then it is clear that greater attention needs to be given to BV in the global fight against HPV infection and women with BV should be considered as a priority group for prophylactic vaccination. Regular follow-up of these patients should be considered. Restoring the vaginal micro flora should in that case be a promising answer to the high prevalence of HPV infections. Randomized clinical trials to effect of BV control measures on HPV acquisition may be worth considering. At present suitable vaccines targeting HPV types 16 and 18 accounting for 70% of cervical cancers worldwide opened up new avenues in prevention of this important public health problem. In addition the need to evaluate the potential of BV treatment to prevent HPV acquisition and transmission a better understanding of risk factors and determinants of recurrence is required.
Considering that these conditions are very common among women worldwide, further research in this field is imperative. More data from prospective studies are needed to accurately evaluate temporal sequence of acquisition of both conditions in any attempt to determine a causal relationship and to identify specific subpopulations with a stronger association between BV and HPV.

References


2) Boyle DC, SmithJR; Infection and cervical intraepithelial neoplasia; Int J Gynaecol Cancer; 1999; 9:177-186


4) Castellague X; Natural history and epidemiology of HPV infection and cervical cancer; Gynecol Oncol 2008; 110:54 – 7

5) Cherpes TL, Hillier SL, Meyn LA, Busch JL, Krohn MA; A delicate balance: risk factors for acquisition of bacterial vaginosis include sexual activity, absence of hydrogen peroxide producing lactobacilli, black race and positive herpes simplex type 2 serology; Sex TransmDis 2008 ; 35: 78- 83


8) Ka Hyun Nam, Yong Wook Jung, Eun Ji Nam, Young Tae Kim, Mi Kwung Lee; Association between bacterial vaginosis and cervical intraepithelial neoplasia ; Journal of Gynaeol Oncol 2009 ; 20(1): 39 – 43


16) Ugwumandu AM; Bacterial vaginosis in pregnancy; CurrOpin Obstet Gynaecol 2002 ; 14 : 115 – 118


Obstetric Hemorrhage: Quick Assessment & Management

Nibedita Pani¹, Devishree Das², Srimanta Haldar³

“The focus of resuscitation should be preservation of woman’s life rather than preservation of woman’s life rather than preservation of her uterus.”

INTRODUCTION

Bleeding has always been a dreaded complication in the field of obstetrics. It is one of the Common cause of maternal morbidity worldwide, accounts for 25-30% of maternal death. WHO estimates severe hemorrhages complicates 10.5% of live birth. But in India it is responsible for >60% of maternal morbidity.

Obstetric hemorrhage is challenging to anesthesia providers as it is usually sudden in onset & rapidly become life threatening. A multidisciplinary approach with a team consisting of obstetrician, anesthesiologist, neonatologist and hematologist is essential for management of peripartum bleeding.

HOW IS OBSTETRIC HEMORRHAGE DIFFERENT?

- Inability to recognize the risk factors
- Difficulty to exact blood loss estimation
- Difficulty in early diagnosis
- High uteroplacental blood flow

DEFINITION

- Sudden blood loss > 1500 ml (25% of blood volume)
- Blood loss > 3000 ml in less than 3 hours (50% of blood volume)
- Blood loss of 150 ml/minute in 20 minutes (>50% of blood volume)
- Requirement of acute transfusion of > 4 units of packed red blood cells.

ETIOLOGY

1. Antepartum haemorrhage
   a) Placental bleeding: Placenta previa & Abruptio placentae
   b) Unexplained or Indeterminate: Vasa previa, maternal coagulopathy, Circumvallate placenta & uterine rupture.
   c) Extraplacental causes: Cervical polyp, Cervical cancer, Varicose veins & Local trauma

2. Post partum haemorrhage: Atonic uterus, Uterine inversions, Retained placenta / Placenta accrete & Genital tract trauma

ANTEPARTUM HEMORRHAGE

- Bleeding from or into genital tract after 24 weeks gestation but before delivery of the baby.
- Complicates close to 3% of all pregnancies

Causes:

- Placenta previa
- Placental abruption

Fig 1: Types of Placenta Previa
POST PARTUM HAEMORRHAGE: Any amount of bleeding from or into genital tract following birth of baby upto the end of the puerperium.

Types:
- Primary haemorrhage occurs within first 24 hours
- Secondary haemorrhage occurs beyond 24 hours & within 6 wks of delivery
- PPH can be minor (500-1000 mls) or major (> 1000 mls).
- Major PPH could be divided to moderate (1000-2000 mls) or severe (>2000 mls).

The 4 ‘T’s pneumonic is useful to aid recall the major causes of primary PPH:
- Tone – uterine atony
- Tissue – retained products of conception
- Trauma – genital tract injury
- Thrombin – inherited or acquired coagulopathy

Massive obstetric haemorrhage (MOH):
- Defined as:-
  - Blood loss >1500ml
  - Decrease in Hb >4g/dl or
  - Acute transfusion requirements >4 units

ASSESSMENT AND MANAGEMENT OF HAEMORRHAGE:

Clinical evaluation:
1. Brief history to elicit cause of haemorrhage.
2. Vital signs:
   - Resting tachycardia: HR>120 beats/min
   - Hypotension
3. Orthostatic Vital Sign i.e Significant postural change defined as:
   - Increase in pulse rate by 30 beats/min
   - Decrease in SBP >20mm Hg or dizziness on standing
4. Urine output<0.5 ml/kg/min
5. Capillary refill <5 sec

Modified Early Obstetric Warning System (MEOWS)

1. Useful bedside tool for predicting morbidity patients and recommended in all obstetric patients. Also helpful to track maternal physiological parameters and to aid early recognition and treatment.
   - MEOWS includes looking for signs such as
     1. Tachycardia,
     2. Hypotension,
     3. Decreased urine output,
     4. Pallor,
     5. Lower abdominal pain
     6. Cold peripheries

“RULE OF 30”

- Patients systolic BP drops by 30 %,
- The heart rate rises by 30%,
- The respiratory rate increase to >30/min,
- The hemoglobin or hematocrit drops by 30%
- The urine output decreases to <30 ml/h,
- The patient is likely to have lost 30% of her blood volume

“SHOCK INDEX”

- Defined as the heart rate divided by systolic BP (normal up to 0.9 in obstetrics)
- Accurate indicator of compensatory changes in the chorionic villus sampling due to blood loss.
- Active periodic estimation improves the accuracy of estimation.
- According to one study, there was 16% underestimation at 300 ml blood loss which rose to 41% at 2000 ml loss.
MANAGEMENT OF MASSIVE OBSTETRIC HEMORRHAGES (MOH):

A) Anticipated MOH

- Cases at high risk of MOH can be predicted e.g. LSCS in a low lying placenta and previous uterine scar. These cases may be at a risk of placenta accreta and massive blood loss.
- 2 large bore iv cannulae
- Rapid infusion device or pressure bags in theatre
- Blood warmer & warming blanket
- Blood cross-matched & available
- Consider preoperative invasive monitoring
- Consider cell salvage
- Consider interventional radiological procedures if available

B) Unanticipated MOH

Communication ! resuscitation ! monitoring and investigation ! arresting the bleeding

A 2006 guideline from the British Committee for Standards in Haematology summarises the main therapeutic goals of management of massive blood loss is to maintain:

- Haemoglobin> 8g/dl
- Platelet count > 75 x 10^9/l
- Prothrombin < 1.5 x mean control
- activated prothrombin times < 1.5 x mean control
- Fibrinogen > 1.0 g/l

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose &amp; route</th>
<th>contraindications</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxytocin</td>
<td>20-60 mU/min Infusion</td>
<td>none</td>
<td>Hypotension with bolus IV doses Free water retention</td>
</tr>
<tr>
<td>Methylergonovine</td>
<td>0.2 mg IM</td>
<td>Hypertension Preclampsia Coronary artery disease</td>
<td>Severe nausea and vomiting Arterial constriction</td>
</tr>
<tr>
<td>15-methyl prostaglandin (carboprost)</td>
<td>250 mcg IM or IV</td>
<td>Reactive airway disease Pulmonary hypertension Hypotensive patients</td>
<td>Bronchomotor stimulation Shivering Temperature elevation Diarrhoea</td>
</tr>
<tr>
<td>Misoprostol</td>
<td>600-1000 mg per Rectum</td>
<td>none</td>
<td>Shivering Diarrhoea Nausea/vomiting</td>
</tr>
</tbody>
</table>

Fig 3: classification of hemorrhage

Fig 4: Pharmacotherapy

Invasive therapy:

- Balloon tamponade (Bakri/Rusch balloon, Foley’s/condom catheter, Sengstaken-Blakemore tube)
- Haemostatic brace suturing (B-Lynch)
- Bilateral ligation of internal iliac /hypogastric arteries
- Bilateral ligation of uterine arteries.
- Arterial embolisation or balloon occlusion radiologically
- Compression/ clamping Aorta to buy time

Fig 6: A Pictorial Reference Guide to Aid Visual Estimation of Blood Loss at Obstetrics Haemorrhage
Transfusion in obstetric haemorrhage: ASA Guidelines & ACOG recommendations

Although the physiological changes of pregnancy help to mitigate the parturient in response to haemorrhage and vital signs may not change until >1500ml of blood loss has occurred, patients should be transfused when there are signs of significant hypoperfusion.

PRBC administration is rarely indicated when haemoglobin concentration is >10g /dl but is almost always indicated when the hemoglobin level is <6g/dl.

Correction of electrolyte imbalance may be necessary; this may include hyperkalaemia (secondary to high concentrations of potassium in transfused blood) and hypocalcaemia (chelated by the citrate found in transfused FFP).

If base deficit is >15, in presence of significant and ongoing blood loss, then blood is administered.

O2 extraction of 50% can be used as an indication of transfusion of erythrocytes.

Hb should be maintained between 6-10g/dl.

Platelet count maintained over 50,000/dl.

INR to be maintained <1.5 by using FFP @ 10-15ml/kg body wt.

Cryoprecipitate should be used when fibrinogen levels fall below 100mg/dl @ 1u/5-10kg body wt.

Massive transfusion pack/ shock pack to be kept ready (PRBC:FFP:platelet::1:1:1).

In face of relentless bleeding empirical treatment with 1 liter of FFP and 10 units of cryoprecipitate (2 packs) can be given, while awaiting coagulation studies. Such empirical use of FFP and cryoprecipitate is in line with the recommendations in the british committee for standards in hematology guideline.

Recombinant factor VIIa: Used primarily as a treatment of uncontrolled hemorrhage in the trauma setting. It causes a thrombin burst, promoting clotting in open vessels. Effectiveness is markedly diminished by hypothermia and acidosis. RfVIIa will not work if there is no fibrinogen and effectiveness may also be suboptimal with severe thrombocytopenia (less than 20 x 10⁹/l).Fibrinogen should be above 1g/l and platelets greater than 20 x 10⁹/l before rfvia is given. The dose 90mcg/kg followed by a second identical dose if no response is seen after 20mins. Binds to exposed tissue factor & directly activates factor IX & X. Complications includes thrombosis, MI, PE & clotting of vascular access. Used as last resort when other strategies fail.

Vasopressin: Used in hemorrhagic shock unresponsive to conventional vasopressors. infusion rate-1-4mu/kg/min. It Has a potential to cause Myocardial Ischemia.

Autologous transfusion:

- Antepartum donation
- Intraoperative cell salvage
- Acute normovolemic hemodilution

Intraoperative Cell Salvage: Numerous reports shows the safe use of intraoperative cell salvage in parturient and it has been recommended in women who refuse traditional blood transfusions (jehovah witness) as well as in other major hemorrhage situations. Cell salvage is started after the majority of amniotic fluid has been suctioned to lessen the risk of amniotic fluid embolism. A leucocyte depletion filter should be used prior to re-infusion of the salvaged blood to remove additional contaminants. Remember that cell salvaged blood contains only red cells with no clotting factors or platelets.

For women who are rh-negative, to prevent sensitization, the standard dose of anti-D should be given and a kleihauer test taken 1 hour after cell salvage has finished, to determine whether further anti-D is required.

Antifibrinolytics: Tranexamic acid is a potentially useful drug that can decrease bleeding and reduce the need for further transfusion without many major side effects. The initial dose is a slow IV bolus of 1g followed by 1g 4 hours later.

Desmopressin (DDAVP): Treatment of VWD, management of persistent non surgical bleeding.

In a diagnosed case of VWD epidural anesthesia may be safely performed if VWF>50IU/dl.
Consider hemostatic agents like aprotonin & vitamin K.

RESPONSIBILITIES OF THE ANESTHESIOLOGIST ON ARRIVAL OF AN UNEXPECTED BLEEDING PARTURIENT:

- Assess vital signs like blood pressure, oxygen saturation, heart rate, capillary refill, peripheral and core temperature & level of consciousness
- Obtain brief history
- Assess airway, anticipate cannot intubate cannot ventilate condition
- Provide 100% oxygen by face mask
- Monitor vitals like BP, HR, SPO2 at regular interval
- Achieve proper venous access
- Compare vital signs with estimated blood loss & communicate discrepancies to obstetric team
- Review ongoing fluid resuscitation, pharmacotherapy and make sure that urine output is >0.5ml/kg/hr.
- Place arterial line early if significant hemorrhage present or anticipated
- Send initial lab sample (hemoglobin, coagulation status blood grouping & cross matching) if not already sent
- Order blood and blood products as needed

Induction:
- GA preferred technique with RSI
- Acid prophylaxis: nonparticulate antacid, metoclopramide & H2 blocker
- preoxygenate/denitrogenate for >3min or 4 vital capacity breath for 30sec with 100% oxygen
- Tell assistant to give cricoid pressure, give induction agent & muscle relaxant in rapid sequence, after 30-60 sec intubate by direct laryngoscopy
- Avoid propofol in hypovolemic patients
- In bleeding patient Ketamine (1.0 mg/kg) or Etomidate (0.3 mg/kg) is preferred whereas in severe hypovolemic shock only a muscle relaxant with BZD is advised

Maintenance:
- Maintain with 50% nitrous oxide with oxygen & 0.5-0.75 MAC of volatile agents
- Maintain EtCO2 30-32 mm hg, N2O is avoided in fetal distress
- Oxytocin (20 U/L) infusion started after fetal delivery
- If bleeding continues stop uterine relaxants & after delivery of fetus stop volatile agent

Emergence:
- Reversal of muscle relaxant and extubation when the patient is awake and responds to verbal commands

CONCLUSION:
The anesthesiologist plays a key role in the management of obstetric hemorrhage. As with almost all medical and surgical crises or emergencies, the principles of preparation and planning when possible, early notification when necessary, and good communication and teamwork at all times are the keys to successful outcomes.
BIBLIOGRAPHY:
3. Rajashree Chavan And M Y Latoo BJMP 2013;6(1):A604
11. Suzy Baldwin* And Matt Rucklidge; Update In Anaesthesia;2009/Dec
17. Obstetric Anaesthesia By Dr.Sunandagupta, Arya Publications
New Technology to Revolutionise Assessment of Ejection Fraction

Dipak Ranjan Das

Researchers have developed technology that can use a smartphone camera to measure left ventricular ejection fraction (LVEF) of the heart, potentially providing the same measurements generated with echocardiography, according to a study published in the journal Critical Care Medicine. (Crit Care Med, July 2017, Vol. 45:7, pp.1115-1120). The technology was developed by Mory Gharib, PhD, from the California Institute of Technology (Caltech), along with researchers from Huntington Medical Research Institutes and the University of Southern California.

Left Ventricular Ejection Fraction (LVEF) is a key measure of heart health. Although the gold standard for measuring it is MRI, due to the modality’s high cost and limited availability, LVEF is most commonly assessed by using an ultrasound machine during a procedure known as echocardiography. Echocardiography, however, requires a trained technician, an expensive ultrasound machine, and up to 45 minutes of a patient’s time.

Researchers have shown that the camera on our smartphone can noninvasively provide critical inputs via an app by simply holding our phone up to our neck for a minute or two. Alternatively, this technology assesses LVEF by measuring the amount that the carotid artery displaces the skin of the neck as blood pumps through it, describing the movement as a waveform. This signifies the expansion and contraction of the artery walls. LVEF represents the amount of blood in the heart that is pumped out with each beat. In a normal heart, this LVEF ranges from 50 to 70 per cent. When the heart is weaker, less of the total amount of blood in the heart is pumped out with each beat, and the LVEF value is lower. LVEF is a key measure of heart health, one upon which physicians base diagnostic and therapeutic decisions. The app works because the walls of arteries are almost completely elastic - they expand and contract with each beat of the heart. That expanding and contracting can be measured and described as a waveform that encodes information about the heart. The study is based on previously reported mathematical analysis of arterial waveform that extracts hidden oscillations in the waveform that are called intrinsic frequencies.

To test the technique, Gharib’s group recruited 72 volunteers between the ages of 20 and 92 and conducted the study at an outpatient MR facility. The team held iPhone 5 smartphones running the application against volunteers’ necks for two minutes; then the volunteers underwent an MR exam. The measurements require no training to perform or interpret, no calibration, and can be repeated at the bedside to generate almost continuous analysis of left ventricular ejection fraction without arterial cannulation.

The researchers compared data from both tests and found that the measurements made by the smartphone had a margin of error of ± 19.1%, compared with those performed on an MRI. (In comparison, the margin of error for echocardiography is ± 20%.)

“What is exciting about this study is that it shows our technique is as accurate as echocardiography at estimating LVEF when both are compared to the gold standard of cardiac MRI,” Gharib said in a statement. “This has the potential to revolutionize how doctors and patients can screen for and monitor heart disease both in the U.S. and the developing world.”
Diabetes mellitus is a complex, chronic disease affecting multiple systems of the body. It is a condition characterised by an elevated level of glucose in the blood. Insulin, a hormone produced by the pancreas, controls the blood glucose level by regulating the production and storage of glucose. In diabetes, there may be a decrease in the body’s ability to respond to insulin or a decrease in the insulin produced by the pancreas which leads to abnormalities in the metabolism of carbohydrates, proteins and fats. The resulting hyperglycaemia may lead to acute metabolic complications including keto acidosis and in the long term contribute to chronic micro-vascular complications. Smeltzer & Bare (1992:1022). Phipps et al (1987:601) define diabetes mellitus as a complex, chronic disorder characterised by disruption of normal carbohydrates, fat and protein metabolism and the development over time of micro-vascular and macro-vascular complications and neuropathies and diabetic retinopathy.

EPIDEMIOLOGY

Diabetic retinopathy is a leading cause of vision loss globally. Of an estimated 410 million people with diabetes worldwide, approximately one third have signs of DR and of these, further one third have severe vision impairment due to diabetic macular edema.

Diabetic retinopathy is the resultant effect on retina caused by complications of diabetes mellitus, which can eventually lead to blindness. It is an ocular manifestation of systemic disease which affects up to 80% of all patients who have had diabetes for 10 years or more.

WHO ARE AT RISK?

1. Duration of diabetes is the most important determining factor
2. Poor metabolic control is less important than duration, but is nevertheless relevant to the development and progression of DR.
3. Heredity
4. Pregnancy may accelerate the changes of diabetic retinopathy.
5. Hypertension
6. Other risk factors include smoking, obesity and hyperlipidemia.

TYPES OF DIABETIC RETINOpathy

It can be classified in different ways, but there are three main types:

• Non-proliferative retinopathy
• Maculopathy
• Proliferative retinopathy.

A. Non-proliferative (or Background) diabetic retinopathy (NPDR)

It is primarily a disease of retinal blood vessels. It is the result of two major processes affecting the retinal blood vessels: vessel closure and abnormal vessel permeability.

1. Retinal Blood Vessel Closure

The earliest vessel closures in diabetic retinopathy are usually the capillaries. These small vessels are critical to the health of the retina, since they are needed to deliver oxygen and nutrients to the area and to carry away carbon dioxide and other waste products. The source of this capillary closure is not completely understood. Theories as to why these vessels close off include:

– Clumping of blood cells or other blood elements
– Abnormality or damage to the endothelium (the cells lining the inner wall of the capillary).
– Swelling of an abnormally permeable vessel wall.
– Compression of the capillary by surrounding retinal swelling.

Diabetics tend to have capillary closure causing areas of decreased oxygen supply to the small patches of retina corresponding to these capillaries. There is an associated dilation of the adjacent capillaries, probably in response to the decreased oxygen level in
that part of the retina. In addition, small focal saculations of the retinal capillaries called micro aneurysms can develop. These micro aneurysms are small sacs budding off from the vessel, often visible as tiny red dots on ophthalmologic examination. It is thought that micro aneurysms are the result of weakened capillary walls that allow a bulging outward of the endothelial lining of the capillary.

Localized closure of retinal capillaries (non-perfusion) is usually not significant visually, since the non-perfused area is so small. However, if the non-perfusion is in the central portion of the retina (the fovea), the vision can be significantly reduced. There is no known treatment for this visual loss due to foveal non-perfusion.

Retinal capillary closure can produce Proliferative diabetic retinopathy in more severe cases. When multiple areas of the retina have lost their blood supply, they start to send out chemical signals called neurogenic (VEGF) factors. These neurogenic factors are fragile and can cause bleeding and scar the retina. This is one way blindness associated with diabetes occurs.

2 Abnormal Vessel Permeability

Retinal blood vessels are different from vessels elsewhere in the body. Most blood vessels are fenestrated, meaning that they have tiny openings that allow fluid to pass through the vessel wall. The openings are small enough to prevent the egress of larger blood elements such as blood cells and large proteins, but large enough to allow water and small molecules such as ions to pass. Retinal blood vessels, on the other hand, have tight junctions between the cells of the blood vessel wall, so all fluid and molecules exiting the vessel have to pass through the cells. This lack of fenestration helps keep the retina relatively dehydrated, which is necessary for proper function.

In diabetic retinopathy, the vessels become more permeable. Water, blood cells, proteins, fats, and other large molecules may leak out into the surrounding retinal tissue. Accumulation of this fluid in the central region of the retina (the macula) is called macular edema or diabetic maculopathy. Diabetic Maculopathy is the most common cause of decreased vision in patients with background or non-proliferative diabetic retinopathy. It is visible on examination as a thickening and slight graying of the retina, and is often associated with exudates (yellow clumps or spots within the retina). Exudates are the result of fats and proteins leaking out of the permeable vessels along with water. The water can be quickly reabsorbed into the vessels or into the tissue under the retina, but the fatty material is absorbed only very slowly. These fatty exudates are left behind like a “bathtub ring”, often in a ring-like shape surrounding the leakage site.

B. Diabetic Maculopathy

In diabetic maculopathy, fluid rich in fat and cholesterol leaks out of damaged vessels. If the fluid accumulates near the center of the retina, there will be distortion of central vision. If too much fluid and cholesterol accumulates in the macula, it can cause permanent loss of central vision. CSME (clinically significant macular oedema) is the term given to describe water logging of the macular area. The area of the retina we use most is called the macula. It provides our central vision and is essential for clear, detailed vision. In maculopathy, the haemorrhages, exudates and swellings of the non-proliferative stage occur in the macula.

C. Proliferative Diabetic Retinopathy (PDR)

It refers to a severe stage of diabetic eye disease in which new blood vessels proliferate on the surface of the retina. Most patients with PDR have had Non-proliferative Diabetic Retinopathy for at least a few years prior to developing the proliferative form of the disease. The diagnosis of PDR requires the presence of new proliferating blood vessels (neovascularization) arising from the retina or optic disc and growing on the retinal surface or into the vitreous cavity. This diagnosis is made primarily by examination of the retina and sometimes by fluorescein angiography.

D. Diabetic Retinopathy End Disease – characterised by

Vitreous Haemorrhage, Neovascular Glaucoma and Tractional retinal detachment

SCREENING FOR DIABETES RETINOPATHY

- To prevent visual loss occurring from diabetic retinopathy a periodic follow-up is very important for a timely intervention. The recommendations for periodic fundus examination are as follows:
Every year, till there is no diabetic retinopathy or there is mild NPDR.

Every 6 months, in moderate NPDR.

Every 3 months, in severe NPDR.

Every 2 months, in PDR with no high risk characteristic.

**DIAGNOSIS**

Fasting blood sugar with at least 8 hours of fasting should be done followed by 2 hours of post-prandial blood sugar. Accordingly it gives the short term glycemic value whereas HBA1c (glycosylated haemoglobin) value which is an indicator of the long term glycemic control. All the patients who are known to be diabetic must have their lipid profiles and serum urea and creatinine monitored to avoid the other microvascular and comorbid conditions affecting multiple systems. Other special ophthalmic investigations like Flourescein Angiography, OCT and USG B Scan to know the integrity of vascular system and also posterior segment status in presence of hazy media due to cataract and vitreous haemorrhage.

**MANAGEMENT**

1. Control of systemic disease

2. Role of pharmacological modulation.

Pharmacological inhibition of certain biochemical pathways involved in the pathogenesis of retinal changes in diabetes is being evaluated. These include:

- Protein kinase C (PKC) inhibitors,
- Vascular endothelial growth factors (VEGF) inhibitors - Avastin, Lucentis
- Aldose reductase and ACE inhibitors, and

**Antioxidants such as vitamin E**

3. Role of intravitreal steroids in reducing diabetic macular oedema is also being stressed recently by following modes of administration:

- Flucinolone acetonide intravitreal implant
- Intravitreal injection of triamcinolone (2 to 4 mg).
- Photocoagulation.

It remains the mainstay in the treatment of diabetic retinopathy and maculopathy. Either argon or Green laser can be used. It converts hypoxic retina to anoxic retina, thus decreasing the release of VEGF and neovascularisation.

**Conclusion**

Diabetic retinopathy (DR) remains the commonest cause of blindness in the working age population of the developed world. Effective treatment is available if the condition is detected early, before visual symptoms occur. The need for a comprehensive DR screening programme has long been recognized and it is now feasible by the continuous efforts of Ophthalmologists. It is a silent disease and may only be recognized by the patient when the changes in the retina have progressed to a level, that treatment is complicated and nearly impossible. This disease can be prevented from developing into blindness if it is treated at an early stage and hence warrants continuous monitoring. Public awareness is thus an integral part of the prevention of deleterious effects of this silent yet deadly disorder.
NORA for Obese Patients
(NON-OPERATING ROOM ANAESTHESIA)
Basanta Kumar Pradhan¹, Akhilan S²

INTRODUCTION:

Out of operating room anaesthesia or NON OPERATING ROOM ANAESTHESIA provides anaesthetic care to the patient outside the traditional operating theatre. Such an anaesthetic care can be advantageous both to the patients and the physicians alike, but possesses lots of challenges to an anaesthesiologist. As to define NORA, it is administering anaesthesia care in remote locations where an anaesthesiologist is bound to provide anaesthesia or controlled sedation outside the operation theatre, within the hospital premises.

Such places include:

- Radiology suites e.g. cardiac angiography, interventional radiology, CT scan, MRI
- Endoscopy suites: gastro enterology procedures, bronchoscopy
- The dental clinic
- The burns unit
- Psychiatric unit for electroconvulsive therapy
- Renal unit for lithotripsy
- The gynaecology unit for in-vitro fertilisation.
- ICU for tracheostomy; percutaneous gastrostomy

Clinically obesity is defined as in increase in body fat content that leads to an array of obesity associated comorbidities and is most often quantified indirectly via the body mass index (BMI). A BMI of greater than 30kg/m² constitutes obesity and a BMI of more than 40kg/m² constitutes morbid obesity. More significantly, a BMI of more than 40kg/m² directly correlates with the mortality in the Peri-operative period. While providing anaesthesia in the operating theatre itself for an obese patient is challenging in its own respects, providing it outside the OT is even more concerning.

AIM OF THE ANESTHESIOLOGIST IN NORA:

- Guard the patient’s safety and welfare
- Minimise physical discomfort and pain
- Control anxiety, minimise psychological trauma and maximize the potential for amnesia
- Control movement to allow safe completion of the procedure
- Return the patient to a state in which safe discharge from medical supervision is possible

CONCERNS WHILE ADMINISTERING NORA IN THE MORBIDLY OBESE:

PATIENT SELECTION:

Patient selection takes into consideration the extent of surgical stress, anaesthetic procedure and the post-operative recovery. The ASA patient stratification should be taken into consideration while selecting the patient for the procedure. As a norm, ASA GRADE 1 and 2 are safe to undergo the procedure, ASA 3 is relatively safe to undergo the procedure, if the comorbidities are in the controlled limit. Ideally ASA 4 and 5 should undergo procedure only in a more traditional fully equipped operating theatre setting, but this is not always possible, as some of these procedures might be urgent or the equipment required for the procedure might be in remote location. In such patients it is important to make a quick assessment with regards to the safety of performing such a procedure bedside, its relative risks and benefits, requirement of an assistive airway and also an assessment of availability of the drugs to reverse potential over sedation.

ENVIRONMENT:

The basic requirement of the equipment and personnel resources as defined by the ASA practice guidelines for NORA must be met for every procedure.

¹Professor
²PGT
Anaesthesiology
SCBMCH Cuttack.
ASA GUIDELINES (last amended on 16th October 2013):

1. Reliable oxygen source with backup (a full E Cylinder)
2. A suction source
3. Waste gas scavenging
4. In each location there should be:
   a. A self-inflating hand resuscitator bag capable of providing 90% oxygen
   b. Adequate anaesthesia drugs supplies and equipment for the intended anaesthetic care
   c. Adequate monitoring equipment fulfilling the “Standards of Basic Anaesthetic Monitoring”.
5. Sufficient safe electrical outlets
6. Adequate patient and anaesthesia machine illumination with battery- powered backup
7. Sufficient space for the anaesthesia care team (equipment, personnel)
8. Emergency cart with a defibrillator
9. Adequate support staff trained enough to assist the anesthesiologist.
10. Compliance of the facility with all applicable safety and building codes
11. Appropriate post-anaesthesia management.

EQUIPMENT CHECK LIST FOR ANAESTHESIA OR SEDATION IN A REMOTE LOCATION AWAY FROM THE OPERATING THEATRE:

Remember the acronym SOAPME.

S (suction) – Appropriate size suction catheters and functioning suction apparatus.

O (oxygen) – Reliable oxygen sources with a functioning flow meter. At least one spare E-type oxygen cylinder.

A (airway) – appropriate airway equipments:
   ➢ Face mask
   ➢ Nasopharyngeal and oropharyngeal airways
   ➢ Laryngoscope blades
   ➢ ETT
   ➢ Stylets
   ➢ Bag-valve-mask or equivalent device.
   ➢ Supraglottic airway devices

P (pharmacy) – Basic drugs needed for life support during emergency:
   ➢ Epinephrine (adrenaline)
   ➢ Atropine
   ➢ Glucose
   ➢ Naloxone (reversal agent for opioid drugs)
   ➢ Flumazenil (reversal agent for benzodiazepines).

M (monitors):
   ➢ Pulse oximeter
   ➢ NIBP
   ➢ End-tidal CO2(capnography)
   ➢ Temperature
   ➢ ECG

E (equipment):
   ➢ Defibrillator with paddles
   ➢ Gas scavenging
   ➢ Safe electrical outlets (earthed)
   ➢ Adequate lighting (torch with battery backup)
   ➢ Means of reliable communication to main theatre site.

PRE OPERATIVE ASSESSMENT OF A MORBIDLY OBESE PATIENT:

Like any other scenario it is important to get a detailed history and do a focussed physical examination including the previous anaesthetic records. A detailed history must include an assessment of obstructive sleep apnea, cardiopulmonary and other common systemic diseases associated with morbid obesity.

<table>
<thead>
<tr>
<th>Cardiovascular</th>
<th>Congestive heart failure, ischemic heart disease, atrial fibrillation, dysrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine</td>
<td>Diabetes Type 2, metabolic syndrome, hypothyroidism</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Pulmonary hypertension, OSA</td>
</tr>
<tr>
<td>Cancer</td>
<td>Endometrial cancer, oesophageal adenocarcinoma</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Osteoarthritis, spondylolisthesis</td>
</tr>
<tr>
<td>Hepatic</td>
<td>NASH, cholelithias</td>
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<tr>
<td>Reproductive</td>
<td>Infertility</td>
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</tbody>
</table>
OBSTRUCTIVE SLEEP APNEA

OSA is a sleep related disorder characterized by repeated episodes of apnea and hypopnea. Morbid obesity has a direct correlation with obstructive sleep apnea, but the condition can often be undiagnosed. The diagnosis of OSA is made with the help of polysomnography by measuring the AHI (apnea – hypopnea index). Apnea is defined as complete cessation of airflow for more than 10 seconds whereas hypopnea is the airflow reduction of more than 50% for more than 10 seconds. OSA is considered to be mild if AHI is 5-15, moderate if AHI is between 15-30 and severe if AHI is more than 30. It is important to identify OSA in obese patients and also to assess its severity if present during pre-operative check-up.

The patient might give a history of fatigue, excessive daytime sleepiness, loud snoring and possibly witnessed apnea episodes by their family members. Screening of OSA can be done with one of the many questionnaires available; most commonly used being the STOPBANG questionnaire.

### STOP

| STOP | 
| --- | --- |
| Do you SNORE loudly (louder than talking or loud enough to be heard through closed doors)? | yes | No |
| Do you often feel TIRED, fatigued, or sleepy during daytime? | yes | No |
| Has anyone OBSERVED you stop breathing during your sleep? | yes | No |
| Do you have or are you being treated for high blood PRESSURE? | yes | No |

### BANG

| BANG | 
| --- | --- |
| BMI more than 35kg/m2? | yes | No |
| AGE over 50 years old? | yes | No |
| NECK circumference > 16 inches (40cm)? | yes | No |
| GENDER: Male? | yes | No |

- high risk of OSA: Yes 5 - 8
- Intermediate risk of OSA: Yes 3 - 4
- Low risk of OSA: Yes 0 – 2.

If the patient is using CPAP in diagnosed cases. The settings can be made use of in the post-operative period, if CPAP support becomes essential. If the patient is having OSA or is suspicious of having OSA, then there are additional criteria that must be met prior to discharge. The ASA guidelines for OSA patients include the following:

- Monitored for 3 hours longer compared to normal healthy adults
- Patient should not have hypoxemic episodes or airway obstruction when left alone
- The patient should return to their baseline oxygen saturation level on room air
- Monitored for an additional 7 hours after an episode of airway obstruction or hypoxemia while breathing room air.

### POSITIONING AND ITS RELEVANCE IN MORBDILY OBESE PATIENTS

Positioning becomes relevant in these patients because they have a lower threshold of desaturation owing to their dramatic changes in the physiological reserves. Ideally in all situations where it is possible, there should 30 degrees head end elevation. If the procedure demands the patient to be in supine position, then constructing a ramp causing elevation of chest should be done. It will also account for better alignment of the oropharyngeal and laryngeal axis. This is accomplished when the external auditory meatus becomes parallel to the chest. Lateral positions or prone positions are more advantageous as these are seldom associated with airway obstruction.

### INTRAVENOUS ACCESS IN MORBDILY OBESE PATIENTS

Direct visualisation of peripheral veins can be difficult in obese patients, because of the extra adipose tissue, also palpation of veins can be difficult. The method of applying warm towel over a vessel when doubtful about its position can be helpful because of its vasodilatory properties. Ultrasound guided peripheral intravenous access can also be tried but success rate depends on user proficiency. Remote locations for peripheral access can also be attempted. If peripheral line could not be obtained, central intravenous access should be obtained prior to the procedure.

### AIRWAY

Airway assessment should focus on both the predictors of difficult mask ventilation and intubation. Morbidly obese patients by definition are one of the
predetermined risk factors for difficult mask ventilation. Other risk factors leading to mask ventilation should also be taken into consideration.

The predictors of a challenging airway include:
- Mallampati 3-4
- Short thyromental distance
- Inter-incisor gap of less than 3 cm
- Limited atlanto occipital extension
- Limited mandibular protrusion
- Prominent incisors
- High arched palate

**GENERAL ANAESTHESIA**

Preoxygenation is of utmost importance in these patients, as these patients might be having drastic reduction in their functional residual capacity. Preoxygenation should be attempted in Reverse Trendelenburg position, as it reduces the basal atelectasis further. Combining a low pressure CPAP along with Preoxygenation is also recommended as it improves the FRC and also decreases the incidence of desaturation in airway manipulative procedures. Routine airway devices like oropharyngeal airways and difficult intubation cart should be ready by the side of the anaesthesiologist in all cases of morbid obesity.

**SEDATING AN OBESE PATIENT OUTSIDE THE OPERATING ROOM**

Three problems come together when sedating obese patients in remote locations. First the obesity, second the sedation of the patient and third the location being remote. The combination of these three factors makes this act very dangerous and difficult. Therefore the choice for a light general anaesthesia with an artificial airway, LMA or better an endotracheal tube is in such a situation frequently the best option.

Obesity measured by BMI is not enough to exclude a patient for sedation. The high BMI combined with central obesity is dangerous. The sedation is further more difficult if the patient has a higher degree of OSA. The patient position and the access to the airway during the procedure have to be questioned. The immobilisation level that is required and hence the sedation level will determine if sedation is possible in the obese.

**The level of sedation should be assessed:**

**Minimal Sedation (anxiolysis):** a drug induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired ventilatory and cardiovascular functions are unaffected.

**Moderate sedation (conscious sedation):** a drug induced depression of consciousness during which the patient respond to verbal commands either alone or accompanied with light tactile stimulation. No interventions are required to maintain a patent airway and spontaneous ventilation is adequate. Cardiovascular functions are also maintained.

**Deep sedation:** a drug induced depression of consciousness during which the patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway. Cardiovascular function will be maintained.

**General anaesthesia:** a drug induced state of unconsciousness during which the patients are not aroused even by painful stimulation. The patient require airway assistance and the cardiovascular function may be impaired.

Sedation in obese is difficult because of obstructive breathing. When breathing assistance is required we also encounter difficult mask ventilation. An experienced anaesthesiologist recognizes obstructive breathing and can handle this. However the post procedure breathing is a larger problem when the patient wakes up slowly after opioids and benzodiazepines. Therefore we should avoid all opioids and benzodiazepines if possible for sedation of obese patients in remote locations, or antagonize always with flumazenil and Naloxone. Opioids even in a low dose weaken the pharyngeal muscles giving obstructive breathing and blocking the respiratory centre reducing breathing depth and frequency.

**Before planning the sedation you have 4 questions to answer.**

First, is the procedure painful during and after the procedure? If painful during the procedure, deep sedation to an anaesthesia level is required. Adding opioids are what normally is done but should be avoided.
in obese patients. Therefore the best option is to use analgesics with minimal impact on respiration. If infiltration with local anaesthetics is possible this is ideal, if a loco-regional block is possible one should evaluate the invasiveness versus the procedure. Non-opioid analgesics are a good option starting with paracetamol and NSAID if not contraindicated.

Second question is the requirement for immobilisation during the procedure. No movement at all requires anaesthesia and ventilation. NMB is then the safest option instead of deep hypnosis or high dose opioids. If movement is not critical but needed then the choice is deep sedation or full conscious sedation with patient request to stay quiet. Benzodiazepines gives amnesia making it difficult to explain the patient repetitively what is happening.

The third question is the need for amnesia. Deep sedation and general anaesthesia will prevent the patient from remembering what happened but this anaesthesia depth requires artificial airway and ventilation support if used in obese patients. Benzodiazepines are the simplest to create amnesia but should be avoided or reversed with Flumazenil.

The last question is the duration of the procedure. If the procedure is very short only Propofol bolus or inhalation is able to provide sedation and rapid recovery. Procedures between 15 min and 1 hour in obese patients can get only a loading dose of dexmedetomidine or an infusion of Propofol. Longer procedures require dexmedetomidine loading and infusion sometimes combined with a Propofol infusion.

 Conscious sedation requires a full conscious patient who stays awake with open eyes and full cooperative. You talk with the patient and explain what he/she will feel or let him/her listen to music. The classical method without an anaesthesiologist is using a combination of midazolam and an opioid, both drugs that should be avoided in obese patients. Sedation in obese and OSAS patients should therefore only be done by an anaesthesiologist. Sedation without midazolam or opioid is possible when using Propofol.

Propofol has the benefit of rapid onset and offset of effect, but does not provide analgesia or sympathetic block. Propofol is therefore only useful for pain free procedures and patients who can control their stress reaction or sympathetic output. If Propofol is used in higher dose respiration becomes depressed. This is again what should be avoided in obese patients. “Patient controlled sedation”, uses Propofol boluses of 25-50 ìg/kg and is becoming popular in adults. Propofol administered by anaesthesiologists as a continuous infusion of 2 – 4 ìg/kg/h I.V. is extremely useful in non-painful sedation procedures where a quick wake up is desirable. Its antiemetic effect makes it particularly useful for outpatient procedures.

The best method in obese patients is using a Dexmedetomididine 0.2 – 0.5 ìg/kg loading dose over 10 min. It provides sedation and some analgesia with minimal respiratory depression or obstructive breathing. If the procedure is longer than 15 minutes, a repeat bolus of 0.1 ìg/kg can be given after 30 minutes or an infusion can be started from the beginning at 0.2 – 0.5 ìg/kg/h. There is no place for midazolam or ketamine. N20 or low dose Sevoflurane below 0.3 MAC is an option with an open mask or nasal cannula.

Deep sedation requires a deep sedated patient who reacts only on pain stimuli. This level is too deep for obese patients except if an artificial airway is in place and ventilation is supported. The safest airway in the obese is an intubation and therefore this level is an anaesthesia level. A total opioid free anaesthesia method using Dexmedetomidine and Propofol combined with low dose Ketamine, Lidocaine and Magnesium allows using each drug far below its maximum while keeping hemodynamic stability and no pain on awakening. Be aware that immobilisation without any sudden awakening and using no opioids requires neuromuscular blockers.

Monitoring in obese patients can be challenging. Open capnography on the nasal cannula or non-invasive tidal volume monitoring is helpful as pulse oximeter alone is not sufficient to validate the spontaneous breathing quality on obese patients. Be aware also that the level of sedation and the measured BIS level depend not only on the drug dosage but also on the procedural stimuli. Some patients fall one level of sedation deeper during a no stimuli phase while others jumps up during the procedure to a full awake state when extra stimuli are given.
COMPLICATIONS REGARDING PATIENT MANAGEMENT:

RESPIRATORY COMPLICATIONS:

Respiratory depression is more frequent during sedation or anaesthesia outside the OT, so all must be familiar with the difficult airway algorithm. The most common cause of difficult intubation was reported amongst 10% anaesthesiologist who had no backup plan. The most specific damaging event reported in the NORA claims where inadequate oxygenation, which is preventable by better monitoring with pulse oximetry and capnography. Techniques in reducing deoxygenation can be prevented with adequate pre-oxygenation of the patients who are vulnerable especially the elderly and maintaining an appropriate seal between the mask and the face is also important.

HYPOTHERMIA:

Hypothermia commonly occurs in paediatric patients. It can be reduced by using warmed blankets, administrating heated fluids and proper warming. According to NORA settings, mild hypothermia increases surgical blood loss and increases the risk of transfusion.

ASPIRATION:

It occurs because of blunting of protective airway reflexes, so preoperative fasting is important in elective procedures.

HYPOVOLEMIA:

Patients with fluid restriction are at a risk of dehydration and hypovolemia, so by pre-hydration and slow injection of drugs we may prevent adverse events.

POSTOPERATIVE NAUSEA AND VOMITING

CONCLUSION

The proceduralist and anaesthesiologist will have a general idea of the important aspects that should be considered while performing NORA to decrease complications with the procedure and anaesthesia. However, because each procedure is different, additional standards according to specific procedures should be implemented. When performing NORA, the Anaesthesiologist must ensure that participating staff are adequately trained to assist in anaesthesia as well as CPR. Anaesthesiologists must acknowledge malpractice or negligence in the past and cooperate to develop guidelines for future safe practice of anaesthesia outside the operating room. This does not necessarily require remodelling diagnostic or interventional rooms or GI suites or purchasing new and expensive monitoring machines and equipments. Above all, involvement of a dedicated and vigilant anaesthesiologist is the best way of preventing adverse events.

REFERENCES:
1. Statement on non-operating room anaesthesia by ASA (October 2013).
3. Metzner j Domino kb. Risks of anaesthesia or sedation outside the operating room.
6. Obstructive sleep Apnoea- British Sleep Society.
7. Dripps rd, Lamont a, Eckenhoff JE. The role of anaesthesia in surgical mortality.
“Hospice – Care In India” – Present Scenario & Future Objectives

Sumita Mohanty

What is Hospice Care?

The word “Hospice” is derived from the Latin Word Hospitium meaning a guesthouse/host. It focuses on creating a natural and comfortable end of life experience for those confronted with terminal condition as was started in India in 1952 (1). In today’s scenario Hospice Care is used to indicate a philosophy of care rather than a place and is used synonymously with palliative care. The concept of Hospice is well known in the west but in India where palliative care is so desperately needed it is almost unheard. In India, it seems there is no definitive eligibility criteria described for admission to Hospice. Hospice care is typically provided as an outpatient home care or through in patient care. The goal is to provide comfort and QOL as well as to avoid aggressive futile care at the end of life(2).

What is Palliative Care?

1. It can be at the discretion of the Physician at anytime, any state of illness, from diagnosis to end stage.
2. It is supportive care with or without a curative intent.
3. From office visit to prescription charges it is paid for by insurance and/or self pay
4. It typically takes place in a hospital.

What is Hospice care?

1. It begins when two physicians certify that the patient has < 6m to live of the disease Follows its usual course.
2. It is comfort care without curative intent Patient is no longer responding to curative treatment.
3. All expenses related to the terminal diagnosis is are covered by medicine, Medicate and most private insurances.
4. It is delivered wherever the patient calls home.

What does Hospice Care provide?

1. It gives pain & symptom control, psychosocial and spiritual care.
2. Co-ordinates and supervises all care 7 days a week, 24 hrs a day & family meetings.
3. Respite care provides short term break for care givers for 5 days with the goal of preventing care giver burnout. A study is currently underway at TMH, Mumbai evaluating the effectiveness and applicability of Respite model of palliative care (Salin’s et al). The Cancer Centre has partnered with a charitable trust to form a Respite care facility, acting as a bridge between hospital and home based palliative care thereby empowering the caregivers and ensuring continuity of care for the patient (4).
4. Bereavement care for about a year after the patient’s death through visits, phone calls and other contact, as well as through support groups.

HISTORY

Damecicely Mary Sounders is the founder of modern Hospice movement, who revolutionised palliative care in India and helped people to die with dignity free from fear and pain 5. Hospice were originally the places of rest for the travellers in 11th century. In 17th century a religious order established, Hospices for the dying poor, where they offered food, clothing, shelter as well as minimal medical care. Modern Hospice is a relatively recent concept that originated and gained momentum in UK after the foundation of St, Christophers Hospice in London in 1967 6. In 1986 Prof. D’ souza opened the first Hospice in India “Shanti-avedna Ashram”, in Mumbai. “Jeevodaya” the second Hospice in India started at Chennai in 1990, South India’s first Hospice.
Who provides Hospice Care?

An inter disciplinary health care team consisting of dedicated volunteers Social Workers, Spiritual advisers, Nurses and doctors provide “holistic care” to terminally ill cancer patients. They are typically kind and caring, communicate well, are good listeners and understand that each patient and his needs are unique.

Hospice care can be given in the patient’s home, at hospital, nursing home or private Hospice facility. Although home hospice programme are staffed by Nurses and Doctors and other professionals, the main care giver is a family member or friend who is responsible for round the clock supervision of the patient. Thus family member is trained to provide much of hands on care. To handle round the clock needs or crises, they have an on-call nurse who answers phone calls day and night, makes home visits or sends out the team members may between scheduled visit.

Need for Hospice in India

1. Late Diagnosis and inadequate pain relief: India has one million new cancer cases each year. 80% of them are diagnosed when the disease is already advanced and two third of them are in need of palliative care. In India every hour more than 60 cancer patients die agonizing and undignified death without medicine, help and support. Total no. of Cancer cases in India is likely to go up from 979,786 cases in the year 2010 to 1,148,757 cases in the year 2020. Less than 3% of cancer patients in India have access to adequate pain relief. To make matter worse, there are very few cancer facilities in India and therefore little time or space in overburdened hospital for the care of the terminally ill.

2. Lack of Palliative Care facilities: In India the coverage of PC services is extremely patchy. Hospice are very rare in India and only 16 of India’s 28 states and 7 Union territories (i.e less than 45%) have any palliative care services. 19 states in India have absolutely, no medical facilities that prescribe Morphine. Currently there are over 908 palliative care centres in India, which are accessible to a mere 1% of a population of over 1.2 billion. Out of 908 Centres in India Kerala is having 841 centres which has 3% of India’s population. Lack of funding, lack of awareness and interest by the medical profession in general, lack of government support and lack of morphine are some of the major difficulties facing development of Hospice and palliative care in India.

3. Poor quality of Death Index - The Indian Reality: The economist Intelligence unit has given India the lowest ranking in end-of-life care across the world among 40 countries. In India there is very little awareness about palliative and end-of life care which is complicated by the perception that “Hospice care” is often associated with “giving up”.

4. Lack of medical infrastructure – The majority of the urban poor have to rely on the Govt run hospitals, which are overcrowded. Palliative care is the least of their priorities. It is natural that they would devote their limited resources to patients who can be cured. In the rural areas, the doctors and hospitals are few and far between. The vast distance and poor transportation facility prevent these patients from getting medical relief.

Time line of Palliative Care Development in India:

Palliative care in India is a relatively new concept having developed only over past 30 years compared to 50 years in developed nations. In 1984 it was started as a part of National Cancer Control Programme (NCCP) to make pain relief one of the basic services to be provided at the primary health care level. In 1986 a first Hospice facility in India based on traditional western model of an inpatient hospice, the Shanti...
Avedana Ashram was opened in Mumbai. The pain palliative Care society (PPCS) was formed in 1993 in Calicut, Kerala and functioned purely on the basis of volunteer organisation. It eventually developed an outpatient service and a home visit program with the help of WHO. Indian Association of Palliative Care (IAPC) in 1994 was formed with the help of WHO at Ahmadabad. In 2001 Neighbourhood Network of Palliative Care (NNPC) was developed in 14 districts of Kerala. In 2003 Pallium India was formed by Prof. M.R Rajagopal, Calicut. Pallium India also undertook the initiative for developing curricula for undergraduate medical and nursing education, with a scheme of incorporating palliative care education into current curricula. Action on this is yet to be taken by the Medical Council of India and the Indian nursing council. In terms of medical education, there are two postgraduate training programme for MD in palliative Medicine per year and only a handful of certificate programmes and fellowships ranging from 4 weeks to 1 year. In 2008 Kerala State Palliative Care was approved. In 2013 National Program of Palliative Care (NPPC) was approved. Over the next few years, in the latter part of 1990s, several new palliative care initiatives were started such as the Guwahati pain palliative care society in Assam, the Jivodaya hospice in Chennai, Can support in Delhi, the Laxmi palliative care trust in Chennai and the Karunasraya hospice in Bangalore. Some Regional cancer centres like Trivandrum, Bangalore and Delhi which already had pain management programmes, also included palliative care in their service. Though every year a few centres were added, the growth was limited considering the economy of Indian population. Recently in Uttarakhand, Gangaprem Hospice has opened, whose activities are aimed at helping create cancer awareness in the state as well as providing free consultation and care.

A Major victory for PC in India came in 2014 with amendment of NDPS act which enables Registered Medical Institute (RMI) to procure morphine by obtaining a single license from the state drug controller rather than 5. In February 2016 23rd International Conference of Palliative Care held at Pune, has issued a “Pune Declaration” to Govt. of India for adequate funding for timely and effective implementation of NPPC, NDPS Act as well as the inclusion of Palliative care into Undergraduate Medical and Nursing curricula.

Current facilities and provisions

India has a huge burden of suffering from life limiting diseases. MC Dermott et al. identified 138 organisations, currently providing hospice and palliative care services in 16 states or union territories. These services are usually concentrated in big cities and regional cancer centre with the exception of Kerala, where services are more widespread. It is estimated that 5.4 million people a year are in need of palliative care in India. As of 2014 in Kerala more than 170 institutions stock and dispense morphine. Kerala network is often cited as a “beacon of hope” contributing to two third of India’s palliative care services and one of the largest palliative care network in the world. This programme trains volunteers from the local community to identify the symptoms and psychosocial issues of chronically ill members of their areas and to intervene effectively with the help of a network of trained professionals. The Calicut model (PPCS) has become a WHO demonstration project serving as an example of low cost, high quality and flexible PC delivery in the developing world illustrating sound principles of co-operation between Govt, NGO and community. This grew into present Institute of Palliative Medicine, which now has over 100 Palliative Care Centres in Kerala as well as several out side the state.

Future Objectives:

1. In other countries there are defined guidelines for Health Care providers to refer to when discussing Hospice eligibility. India doesn’t seem to have this yet. The question remains how do we impress access to Hospice and palliative care in India? Using a model that has been established in Kerala by enlisting the help of lay people in the community in addition to providing education to key providers is a good start. Palliative Care model created should not be an imitation from other countries but should be based on care at home and empowering families. The co-existence of multiple systems of medicine (Modern medicine, Ayurveda, Homeopathy and Naturopathy) should be used as complimentary.
2. The innovation, enthusiasm and commitment of volunteers, families, activists and palliative care practitioner are clearly the driving force of Indian Palliative care but implementation of created policies and law still requires massive efforts by both the government system and non government organisations. The non government organisation do have the commitment but would need support to effectively facilitate government activity. Co-ordination & partnership with Hospice programmes is a major feature as PC continues across the trajectory of diseases.

3. Another important step would be to set up a outpatient palliative care centre so that burden on inpatient palliative care established in Cancer hospitals can be reduced as recommended by ASCO. The ASCO recommends increased integration of palliative care into the Oncology setting to achieve high quality comprehensive cancer care by 2020. In an article entitled “Outpatient clinics are a new frontier for palliative care,” by Meier and Beresford describes this field as an essential link in the continuity of care with inpatient Palliative care services. By providing this link, Outpatient palliative care in oncology may prevent or shorten hospitalizations, improve quality of life (QOL) and mood, and prolong life.

4. Research and Advocacy for Palliative Care need to be strengthened at different levels. Hence building evidence for support in Palliative Care is needed. The recent declaration by the World Health Assembly asking all member states to integrate Palliative Care with Routine health Care comes as a major tool in advocacy and hopefully will boost the current efforts .

5. Though the national programme in palliative was created in 2012, due to lack of budget allocation, only a tiny part of the programme has been implemented. Even for the part that is funded considerable catalytic work is needed with the state government to ensure that proper plans are made and implemented.

6. Developing the understanding about the care needs among the community groups through awareness programs among public will help mobilize the involvement of various stakeholders in PC. Education of Professionals and public awareness are now seen to be the greatest needs for improving Palliative Care in India.

References


A Rare Case of Thoracic Actinomycosis
Nalini Prava Das1, Kalyani Prava Gouda2, Gopal Krushna Sahu3, Priyanka Das3, Jyoti Patnaik4

ABSTRACT
Actinomycetes are branching gram positive anaerobic bacteria belonging to Actinomycetaceae family and are commensals in human oropharynx, gastrointestinal tract and female genitalia. It is a rare infection that in the past has been reported to occur in 3 lakhs people per year.

CASE REPORT
A 32 years young female presented with low grade irregular fever. Her cough was for 6 months, non-postural with scanty expectoration. She had two bouts of scanty hemoptysis with no loss of weight. She had taken several courses of antibiotics and anti-tubercular drugs with no improvement in the symptoms.

On clinical examination she revealed multiple cervical glands which was 2cm x 2cm with absent breath sounds in infraclavicular, infra-scapular area.

Chest Xray PA view revealed a large heterogeneous mass in the right upper and midzone with complete erosion of clavicle and loss of lung volume on the right side.  

CECT thorax showed a lytic mass in right clavicular region extending to bilateral sternoclavicular joints along anterior and posterior surface of sternum from manubrium to level of sternocostal junction with central necrosis and lytic destruction of whole of right clavicle, anterior ends of bilateral first to fourth ribs and mediastinal ends of left clavicle.

A lobulated mass 60mm x 50mm extending to right apical region and extra pleural space. Multiple necrotic lymph nodes were seen in right supraclavicular, retrosternal, paratracheal, aortopulmonary, subcarinal and anterior mediastinal stations of left innominate vein is severely compressed. Right lung shows fibronodular opacities and traction bronchiectasis in right upper lobe and pleural thickening along right major fissure. Mild pleural effusion is seen.

A diagnosis of Tuberculosis / Malignancy was made.

CECT thorax showed minimal pleural effusion.

A Rare Case of Thoracic Actinomycosis

USG OF ABDOMEN & PELVIS was normal except for mild splenomegaly and minimal ascites.

PLEURAL FLUID ANALYSIS showed:

- Protein: 3.5 gm/dl
- ADA: 17.5 U/L
- Cytology total count: 2500/dl, lymphocytes: 80%, mesothelial cells: 20%

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FNAC OF MASS - pus was aspirated.
Cytology - non-specific inflammatory lesion
- AFB - negative
- gram stain - gram positive bacili
- culture negative

BRONCHOSCOPY - was normal
A repeat FNAC of mass was performed and special emphasis on histopathology revealed sulphur granules against an inflammatory background.

DISCUSSION

Pulmonary actinomycosis is a rare but important and challenging diagnosis to make. Even when the clinical suspicion is high, the disease is commonly confused always with other chronic suppurrative lung diseases and with malignancy.

Any early, accurate diagnosis will prevent the considerable psychological and physical morbidity, including unwarranted surgery, associated with delayed diagnosis.

An increased awareness of the infection may expedite diagnosis and prevent undesirable complications, including unwarranted surgery, in patients under investigation for persistent pulmonary shadowing.

Thoracic actinomycosis is third most common type after cervico-facial and abdomino-pelvic form. Thoracic actinomycosis accounts for 15% of total burden of the disease. It is slightly more common in males than in females and children are rarely affected.

It is common in patients with underlying respiratory disorders such as emphysema, chronic bronchitis, bronchiectasis and any other condition that are destroyed by previous infection. Alcoholism, poor dental hygiene, facial or dental trauma are important risk factors. Unlike other granulomatous diseases, the infection rate is not increased in immunocompromised patients.

Detection of sulphur granules, or histoligis staining is the principal method of detection of Actinomycetes.

Histopathology reveals an acute inflammation surrounded by fibrosing granulation tissue.

CULTURE AND STAINING CHARACTERISTICS

Actinomyces are fastidious bacteria that are difficult to culture. Bacterial confirmation of a clinicopathological diagnosis is usually obtained in <50% of cases.

Characteristically, colonies of Actinomyces appear as "molar-tooth" or "bread-crumb" colonies in broth media after 3-7 days of incubation. Fluoroscein-conjugated antibody typing is now available for species differentiation in some centres.

Sulphur granules are the pathological hallmark of the disease. These are round or oval basophilic masses with a radiating arrangement of eosinophilic clubs on the surface; they sometimes can be seen even with a magnifying glass.

The average duration of illness before definitive diagnosis was ~6 months, a consistent figure in most series.

In general, an attempt at establishing diagnosis by percutaneous biopsy with fine needle aspiration or core biopsy is now made before "blind" thoracotomy. When guided by ultrasound or CT, this has proven to be a simple, safe and effective diagnostic technique and reduced the number of unnecessary resections.

BIBLIOGRAPHY

2. Weese WC, Smith IM. A study of 57 cases of actinomycosis over a 36 year period. Arch Intern Med 1975; 135;1562-1568.
**Introduction**: Rhabdomyosarcomas are the most common soft tissue sarcomas in children and Parameningeal RMSs (PM-RMS) are about half of all Head and Neck RMS in the pediatric population. However, the lesion is not always clinically visible and an IHC becomes mandatory many a times. The state of the art treatment includes chemotherapy followed by skillful and judicious use of conformal radiotherapy. We present a PM-RMS in a young boy treated successfully in the Department of Radiation Oncology, AH Regional Cancer Centre, Cuttack.

**Case**: A 14 years old boy presented in the Department of Radiation Oncology, AH Regional Cancer Centre, Cuttack with an enlarged mass in the left nasal area for the last 3 months. The growth was large about 8X6 cm around the left nasal region and the maxilla, infiltrative, diffuse and firm in consistency. There was no visible intranasal extension and no epistaxis. Regional neck nodes were not palpable. Abdomen was soft, nontender with no hepatosplenomegaly or ascites. FNAC from the mass showed small round cell tumor. Incisinal Biopsy from the mass was done and histopathology revealed stratified squamous epithelium with underlying tissue and desmoplastic stroma. Tumor cells were arranged in alveolar pattern, solid sheets and clusters with attachment of tumor cells in the fibrous septa. The cells were small in size, round to oval in shape with scanty eosinophilic cytoplasm and coarse clumped chromatin and indistinct nucleoli consistent with Alveolar Rhabdomyosarcoma or PNET. Histopathology Review in the Department of Oncopathology, AHRCC revealed the same. Immunohistochemistry was strongly positive for S100 and MyoD1 suggestive of Rhabdomyosarcoma. This was followed by a metastatic work up.

Bone Marrow Aspiration and Cytology showed absence of marrow infiltration. Whole body Bone Scan revealed no scintigraphic evidence of osteoblastic skeletal metastases. A CECT Thorax showed bilateral normal lung parenchyma and no evidence of lung metastases. A CECT of the PNS (paranasal sinuses) showed nasal septum deviated to the left and an ill-defined soft tissue density lesion of size 27X49 mm in the upper lip. The lesion extended upto the upper gingivobuccal sulcus and caused scalloping of the alveolar process of the maxilla. With a diagnosis of Parameningeal Rhabdomyosarcoma the patient was planned for Chemotherapy: Inj VAC (Vincristine, Adriamycin and Cyclophosphamide). He received 4 cycles of Inj VAC in the Department of Medical Oncology, AHRCC with good response and was referred to the Department of Head and Neck Oncology for evaluation. It was opined as inoperable and then referred to the Department of Radiotherapy. He was planned for Radiation using IMRT (Intensity Modulated Radiation Therapy) to a dose of 60 Gy in 30 Fractions over a period of 6 weeks. During Radiotherapy the patient is tolerating treatment well, is asymptomatic without development of any acute toxicities. A weekly review is done every Wednesday to evaluate the clinical status and biochemical parameters of the patient which are in the normal range.

**Discussion**: Rhabdomyosarcoma (RMS) is the most common soft tissue sarcoma in children, constituting 3–5% of childhood malignancies. (1) It originates from the primitive mesenchymal tissue, the head and neck region representing about 35% of RMS (2). Parameningeal rhabdomyosarcomas (PM RMS) are tumors arising from sites adjacent to the meninges (nasopharynx, middle ear, paranasal sinuses, infratemporal and pterygopalatine fossa) and constitute more than half of all head and neck RMS cases. (3) However, PM RMS is less visible than superficial head and neck sites with a tendency of invasion of critical anatomic structures and therefore represents a major
diagnostic and therapeutic problem. The present case was thought to be either a rhabdomyosarcoma or a PNET. However, IHC confirmed the diagnosis and provided the lineage of the tumor.

Histological categorization of PM RMS is based on pediatric international classification of rhabdomyosarcoma. The definition of parameningeal sites include:

1. Middle ear: Medial to or extends through the tympanic membrane and appears to be arising in the ear canal.
3. Nasopharynx: Bounded anteriorly by the back of the nasal septum, superiorly by the sphenoid sinus, inferiorly by a level corresponding to the soft palate, and laterally and posteriorly by the pharyngeal walls.
4. Infratemporal fossa/pterygopalatine and parapharyngeal area: Tissues bounded laterally by the medial lobe of the parotid gland and medially by the pharynx that may extend through the parotid gland and present as a mass in the cheek. The superior boundary of this tissue volume is the base of the skull just under the temporal lobe, hence the term “infratemporal”.

The treatment of PM RMS includes Chemotherapy according to the IRS (International Rhabdomyosarcoma Study Group IV). It consists of alternating doses of multi-agent chemotherapy VAC every 21 days with weekly vincristine. Doses of chemotherapy are: Vincristine (VCR); 1.5 mg/m² IV push, Actinomycin (DACT); 1.35 mg/m² IV push. Cyclophosphamide (CTX); 1.5 mg/m² at weeks 0 and 3 to be increased to 1.8 gm/m² if tolerated, given IV infusion over 2 h with MESNA and IV fluids. In our case the patient was treated with 4 cycles of Inj VAC and assessed for response.

Radiotherapy in PM-RMS should ideally be done using highly conformal techniques as in our case. The tumor is in close proximity to intracranial structures and conformal radiotherapy like IMRT helps to maximise the dose to the target (tumor) with minimum dose to the surrounding normal structures.
the CTV to form the PTV (Planning Target Volume). The PTV margin accounts for set up uncertainties on a daily basis and also for any geometric variations due to respiratory movement. Volume-based (three-dimensional) planning was applied leading to a homogenous dose to PTV with a minimal dose to the surrounding organs at risk, not exceeding the tolerance level of any of them and treatment was delivered using Intensity modulated radiation therapy (IMRT). Treatment verification was done on Day 1 of the treatment using Kilovoltage Images in XVI and treatment was done using high energy Linear Accelerator in the Department of Radiation Oncology, AH regional Cancer, Cuttack. The Response Evaluation as per guidelines is as follows:-

Complete response (CR): Complete disappearance of the tumor confirmed at >4 weeks.

Partial response (PR): At least 64% decrease in volume compared to the baseline.

Progressive disease (PD): At least 40% increase in tumor volume compared to the smallest measurement obtained since the beginning of the therapy.

Stable Disease (SD): Neither sufficient shrinkage to qualify for PR nor sufficient increase to qualify for PD taking as reference the smallest disease measurement since the treatment started.

Conclusion: The present case is undergoing Radiation therapy, is asymptomatic and tolerating well as seen in weekly reviews. The present case highlights both the diagnosis as well as treatment in a difficult case of parameningeal rhabdomyosarcoma. It brings to light the importance of Immunohistochemistry in reaching a conclusive diagnosis and confirming the lineage after histopathology. Finally it stresses upon the use of Conformal Radiation Techniques like IMRT in treating such tumors which are close to vital structures. More number of cases and longer duration of follow up is necessary for definitive conclusion.
Gynaecologists in the past used to be concerned that a fibroid may grow quickly due to the rising oestrogen levels in pregnancy and cause major problems. Recent medical research has shown that most fibroids do not actually become larger during pregnancy, and those that do often return to their pre-pregnancy size afterwards.

However, there are several ways that a fibroid may affect a pregnancy.

A uterine fibroid can cause uncomfortable feelings of discomfort, pressure, heaviness or even pain if they grow larger and press on surrounding organs or pelvic structures. A fibroid may lead to sharp pain in the lower back (lumbar region) and legs if there is pressure on a nerve.

Red degeneration of a fibroid in pregnancy is a rare complication of a fibroid during pregnancy. This problem is known medically as carneous degeneration. If red or carneous degeneration occurs there is haemorrhage within the centre of the fibroid. This usually happens in the middle trimester of pregnancy and is thought to result from the leiomyoma growing rapidly and outgrowing its blood supply. Red degeneration can be very painful, with severe lower abdominal pain, usually requires treatment with strong painkillers, but nearly always settles down without causing serious problems or needing specific treatment.

With regard to falling pregnant, fibroids are thought to account for about two to three per cent of all infertility problems. If a fibroid or fibroids develop just under the endometrium this may affect the way in which a fertilised egg attaches or implants in the endometrium of the womb. A fibroid therefore, may cause recurrent early miscarriage, which is often so early that a woman is not even aware that she has been pregnant.

More rarely a fibroid may obstruct the canal of the cervix or the opening of the fallopian tubes into the womb. Sometimes later in pregnancy, fibroids may also disrupt the normal development and growth of the uterus, leading to premature labour and childbirth. Extremely rarely, fibroids may lead to a miscarriage before 23 weeks of pregnancy.

Even though they are becoming more common, the exact nature and reasons for leiomyomas causing infertility and problems when a woman is pregnant are not fully understood.
Manuscript submission: Check List for Contributors

1. Letter of submission.
2. Copyright statement signed by all the authors.
3. One copy of manuscript with copies of illustrations attached to each.
4. Title page.
   Title of manuscript.
   Full name(s) and affiliations of author(s); institution(s) and city(ies) from which work originated.
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5. Structured abstract (objectives, methods, results, conclusion) along with title, and key words.
6. Article proper (double spaced on A/4 size paper). Maximum word limit for Original or Review Article is 5,000 and Case Report is 2,000.
7. Acknowledgements (separate sheet).
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9. Maximum number of references for Original articles - 10, Short articles - 5,
   Case reports - 5, Documentation - 3, Correspondence - 3.
10. Each table on separate sheet.
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14. Covering letter signed by all authors confirming that they have read and approved the contents and also confirming that the manuscript is not submitted or published elsewhere.
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– Hon Editor.