JABALPUR OBSTETRICS & GYNAECOLOGICAL SOCIETY

Jigyasa

The Curiosity..

UROGYNECOLOGY AND AESTHETIC GYNECOLOGY

PROF. DR. GITA GUIN
President - JOGS

DR. JIGYASA DENGRA
Editor- JOGS

DR. BHOOMIKA
Co Editor- JOGS

DR. SONAL SAHNI
Secretary- JOGS

J&GS

drjigyasadengra@gmail.com
www.jabalpurobgynsociety.org

JOGS TEAM 2021-22

Dr. Prof. Geeta Guin
President

Dr. Sonal Sahni
Secretary

Dr. Anuradha Dang
Imm. Past President

Dr. Prof. Kavita N Singh
Patron

Dr. Priyadarshini Tiwari
Vice President

Dr. Amita Saxena
Vice President

Dr. Archana Singh
Vice President

Dr. Kaveri Shaw
Vice President

Dr. Shweta Sirsikar
Treasurer

Dr. Ranu Jain
Jt. Treasurer

Dr. Jigyasa Dengra
Editor

Dr. Bhoomika Tantuway
Co-Editor

Dr. Deepti Gupta
Jt. Secretary

Dr. Roma S. Nag
Jt. Secretary

Dr. Richa Dhirawani
Jt. Secretary

Advisor Committee

Dr. Rama Shrivastava

Dr. Prof. Shashi Khare

Dr. Prof. A Lele

Dr. Prof. R. Chauhan

Dr. Ranjana Gupta

Dr. Pragya Dhirawani

Dr. Shirish Jamdar

Committee Co-Ordinating Executives

Oncology
Dr. Pushpa Pandey
Dr. Vineeta Ghanghoriya
Dr. Nidhi Jain
Dr. Kirti Patel

Family Welfare
Dr. G.K. Malhotra
Dr. Sharda Mishra
Dr. Neena Shrivastava
Dr. Bharti Sahu

Safe Motherhood
Dr. Nisha Sahu
Dr. Sangeeta Shrivastava
Dr. Rashmi Kurariya
Dr. Archana Thakur

Public Awareness
Dr. Chitra Jain
Dr. Bhavna Mishra
Dr. Sonal Richhariya
Dr. Dolly Maravi

Adolescent Health
Dr. Alka Agrawal
Dr. Sharda Mishra
Dr. Neena Shrivastava
Dr. Bharti Sahu

Infertility
Dr. D'Pankar Banerji
Dr. Archana Shrivastav
Dr. Rakhi Bajpai

The idea of having a unique logo for the JOGS tenure 2021-2022 materialized with active support and input from all friends, specially Dr Ranu Jain, Dr Sonal Sahni and Dr Shweta Sirsikar. Our multiple sketches was given the final artistic execution by Dr Akash Katara, my very darling intern.

It is our dream of giving wings to our women folk that we have tried to depict through the very beautiful, colourfuland meaningful logo.

The innermost circle depicts a mother cuddling her baby in her arms. Specially highlighted are her breasts. It aims to bring forth the importance of a mothers care including breast feeding in optimal development of the baby. The wings are coloured pink to mean it is eventually we women who can be the wings of our less fortunate women folk.

We chose blue for the inner circle to depict fluidity of a woman in adjusting to the multiple roles entrusted to her.

The outer circle is yellow, the colour of amalgamation, a colour that yields a different Shades when mixed with any other colour. So can a lady blend beautifully with every colour of the universe to create a beautiful world.

President JOGS
Dr. Gita Guin

PROF. DR. GITA GUIN
President
(JOGS 2021-22)

प्रिय एवम सम्मानीय JOGS बंधु

सर्वप्रथम मेरा हृदय आभार एवम शुभेच्छा स्वीकारें।

मैं नतमस्तक होकर इस भीषण कठिन माहामारी में, आपने मानवता के लिए किए गए निस्वार्थ सेवा का जो अप्रतिम उदाहरण पेश किया , उसके प्रति स्वयं के एवम JOGS के तरफ से पुनः आभार वयक्त करती हूं।

अतुलनीय चेष्टा, समर्पण की जो रूपरेखा मेरे सखिद्वय अनुराधा एवम कावेरी ने बांधी एवं असम्भावनाओं से सराबोर, हिचकते, सकुचाते मैंने जबलपुर स्त्री एवम प्रसूति रोग संघ का बागडोर संभाला। मन में अनिश्चितता के भाव यूँ उभरे जैसे समाज के भावों से डांवाडोल मेरा अंतर्मन भी कई बार उद्वेलित होता रहा, असुरक्षा के भाव उभरते रहे कि किस प्रकार मैं आप लोगो की अपेक्षाओं को पूरा कर पाऊंगी।

पर आपके सहयोग से आज मैं समाज के विभिन्न तबको तक अपनी सोच को पहुंचा पा रही हूं। साथ ही साथ, आप थक जरूर गए होंगें वेबीनार के मकड़जाल में फंस कर, पर हम चेष्टा कर रहें हैं कुछ थोड़ा बहुत सकारात्मक वैज्ञानिक गतिविधि बनाये रखने की।

बहुत सारी खामियां रहेंगी, कहानियां भी मस्त बनेंगी, पर जब आप साथ हैं, तो सोच को मैंने आपकी सोच पर भरोसा कर, अपनी अनिश्चितताओं को सोने भेज दिया है और हमसब किसी भी मुकाम को हासिल करने का हौसला लिए निकल पड़े हैं।

आइए, दिल से दिल मिलाएं, हाथ से हाथ बांधे हम स्वयं एवम समाज की बेहतरी की दिशा में एक कदम और बढ़ाते हैं। आशा है बहुत जल्द हम एक दूसरे से स्वछंद वातावरण में मिल पाएंगे एवं एक दूसरे के सानिध्य से इन विगत दिनों के एकाकीपन को मिटा देंगें।

ईश्वर से आपकी सार्वभौम प्रार्थना एवं शुभेच्छा संग

DR SONAL SAHNI
Secretary
(JOGS 2021-22)

Dear friends,

Greetings from Team JOGS 2021-22

“The beginning is the most important part of the work” -Plato

Foremost, a big applause to all fraternity members for their sheer dedication towards the service of patients amidst the Covid pandemic. As we assume office, and take upon this mammoth task of fulfilling our academic and social pursuits, teamwork and hardwork will be our motto.

We will weave our thoughts around women-welfareand execute our plans for strengthening the roots of women healthcare .We intend to reach out to the masses through our social connect campaigns encompassing vital issues like Safe Motherhood, Contraception, Save The Girl Child, Adolescent Health, Sexually Transmitted Diseases, Cancer Screening & Prevention, Save The Uterus ,Healthy Diet And Nutrition etc.

To quench our unending thirst for knowledge and pursuit of excellence, we plan to organise Continuous Medical Education programs throughout the tenure, utilizing both online and offline modes circumstantially. We intend to rope in eminent speakers from all corners to deliver valuable insights about recent advances and engage in fruitful discussions on burning topics.

Benchmarks have been set by our extremely efficient predecessors and we shall strive hard to prove ourselves worthy of the heritage. Looking forward to your gracious presence and participation in all our endeavors.

With heartfelt thanks

DR JIGYASA DENGRA
Editor
(JOGS 2021-22)

Hello all,

Hope and wish you all are doing well

"Abdominal Hysterectomy is the surgery;
Laparoscopic Hysterectomy is technology;
And Vaginal Hysterectomy is the ART."

Societal views about women's sexuality are negative and sexual problems are often considered to be part of normal ageing.

There is disparity between the number of women who experience bothersome symptoms and those who are treated. These women are often shy, unwilling or embarrassed to discuss the symptoms and with for health care professional to ask about their symptoms.

The problems are chronic and the treatment options available are not permanent. We have tried to focus on important aspects of these problems in this journal so as to treat them in better way

We have decided the theme of this edition of our quarterly journal, JIGYASA….the curiosity as 'UROGYNECOLOGY AND COSMETIC GYNECOLOGY' as there is the re emerging interest in no scar hysterectomy and people want to know and learn more about it.

I thank to all the contributors for sharing their knowledge with us.

I thank our president (JOGS) DR GEETA GUIN madam and secretary (JOGS) DR SONAL SAHNI madam who believed in me and gave me the opportunity.

I also am very much thankful DR BHOOMIKA, my co-editor who always stood with me.

Stress Urinary Incontinence: Practical Dilemmas

Dr Vineet Mishra
Director & HOD, Department of Obstetrics and Gynaecology
Institute of Kidney Diseases and Research Centre
Dr H L Trivedi Institute of Transplantation Sciences, Ahmedabad

ABSTRACT

Stress Urinary incontinence (SUI) is a significant cause of decrease in quality of life, especially among women. In addition to the significant social impact that SUI has on a woman's quality of life, this condition has a significant financial burden on individual and national healthcare. Treatment options for female stress urinary incontinence include pelvic floor muscle training, lifestyle interventions, bladder retraining, pharmacotherapy, anti-incontinence devices and eventually, surgery. There has been a lack of consensus in the uro-gynecologic community about which outcome measure to use to evaluate the efficacy of treatment for SUI. In treating a disorder that affects quality of life, the patient's perception of her quality of life and goal achievement appears to affect overall. This fact is important to recognize when treating a condition such as SUI, for which therapy is purely elective and for which some types of interventions could potentially worsen the existing symptoms and/or create new problems.

Keywords: Stress incontinence, Pelvic floor muscle training, Treatment, Quality of life, Patient satisfaction

INTRODUCTION

The International Continence Society (ICS) defines stress urinary incontinence (SUI) as a complaint of involuntary loss of urine on effort or physical exertion (e.g., sporting activities), or on sneezing or coughing.[1] The problem can occur at any age, but the prevalence and extent of urinary incontinence rise in women with increasing age. It has a profound negative impact on patient's quality of life and is considered a major barrier to social interests, physical and mental well-being. Depression and anxiety have been linked with incontinence.[2] In a hospital-based cross-sectional study done by Singh et al, of 3000 women in India, 21.8% women were found to be incontinent. Of the total women having incontinence, highest numbers were found to have stress incontinence (73.8%) followed by mixed (16.8%) and urge incontinence (9.5%).[3]

Each patient suspected of having SUI should undergo a thorough history taking, physical examination, and other studies. It is important to note the circumstances, frequency and severity of leaks of urine in women with SUI on initial assessment. This can be assessed by various methods, like urinary symptoms and quality of life questionnaires and a bladder diary. A 3-day bladder diary provides the means for assessing information on fluid intake (type and amount), voiding frequency and volume, together with the frequency of leaks. The extent of further diagnostic evaluation, especially urodynamics, can be tailored to the goals and desires of the patient. During the 2nd International Consultation on Incontinence (ICI), the panel recommended urodynamic studies for the investigation of incontinence symptoms in women in the following cases only: 1) voiding difficulty or neuropathy is suspected, 2) the patient has failed nonsurgical or surgical therapy, or 3) invasive or surgical treatments are being considered.[4]

Many factors should be considered when determining the optimal therapy for a patient with SUI. These include the etiology and type of SUI, bladder capacity, renal function, sexual function, severity of the leakage and degree of bother to the patient; the presence of associated conditions, such as vaginal prolapse, or concurrent abdominal or pelvic pathology requiring surgical correction; prior abdominal and/or pelvic surgery; and, finally, the patient's suitability for, and willingness to accept, the costs, risks, morbidity, and success (and failure) rates associated with each intervention. There are currently a wide

variety of treatment options for these patients, ranging from conservative treatment to surgical treatment. The European Association of Urology guidelines advocate a stepwise approach regarding management of SUI, which begins with addressing underlying medical or cognitive issues, progressing to lifestyle modifications, behavioral therapy, and mechanical devices. [5] Treatment should be dictated by the preferences of women. This is usually influenced by multiple extrinsic non-treatment-related factors such as the previous experience of family members and friends with treatment of SUI or the patient's own past experience with other medical disorders necessitating surgical intervention. Brubaker and Shull described the new concept of patient-centered outcomes using the EGGS acronym (E: expectations, G: goal setting, G: goal achievement, S: satisfaction) that is an important assessment before any operations for SUI. They recommended that the overall satisfaction of patients should be the most important measure to assess the postoperative outcome of anti-incontinence surgery since objective outcome measures may not reveal the full impact of a surgical procedure on a patient's physical well-being, social function, mental health, societal role, and sexual health.[6]

LIFESTYLE INTERVENTIONS AND BLADDER RETRAINING

Alterations in lifestyle factors associated with SUI, like obesity, fluid management, dietary factors (caffeine, alcohol, sweeteners), smoking, heavy work or high impact activity, constipation are frequently recommended. Bladder retraining aims at correcting faulty habit patterns of frequent urination, improving ability to control bladder urgency, prolonging voiding intervals, increasing bladder capacity, reducing incontinent episodes, and building patient confidence in controlling bladder function. Efficacy of bladder retraining for the treatment of stress incontinence has been suggested [7].

PELVIC FLOOR MUSCLE TRAINING (PFM)

The pelvic floor consists of several muscles that include the anal sphincter, the ischiocavernosus, the bulbospongiosus, the transverse perineal muscle, the striated urogenital sphincter and the levator ani (puborectalis, pubococcygeus and iliococcygeus muscles). The normal function of these muscles is to squeeze around the vaginal, urethral and anal openings and to lift inwards in a cranial direction. The contraction of these muscles is not visible but PFM are possibly contracted synergistically with abdominal, hip adductor and gluteal muscles [8].

The rationale for PFM training is based on two concepts: (1) improvement of urethral resistance and pelvic visceral support, including urethral support, by increasing the strength of the voluntary PFM and (2) voluntary contraction of the PFM before increase in intra-abdominal pressure [9]. The improvement of pelvic floor support will prevent decent of the bladder neck and urethra and closure of the urethra during abrupt increase in intra-abdominal pressure by PFM contraction. An increase in intra-abdominal pressure results in unconscious activation of PFM. Patients can learn to contract these muscles before activities that increase intra-abdominal pressure [10]. However, it is impossible for the patients to contract their PFM for a long time. Hence, the clinical improvement may be due to an automatic response level after the training program [11]. In patients with denervation or muscle detachments, the role of PFM training is limited. No change in urodynamic parameters was found after treatment in patients with clinical improvement. Only 49% of women were able to contract the PFM in a way that effectively closed the urethra. PFM training is significantly better than no treatment or placebo treatment based on self-reported cure/improvement and leakage episodes in women with stress incontinence. Women in the PFM training groups were 7.25 times more likely to be cured than women in no treatment groups and this increased to 23.04 times for combined cure/improvement [12].

In the treatment of SUI, first-line treatment tends to be PFMT. However, women may either have trouble identifying and controlling this group of muscles, or are just poorly compliant to the training and therefore other interventions need to be explored. Sets of graded weighted vaginal cones are the proposed solution to this dilemma. The cones provide progressive muscular overload. They are inserted into the vagina and the patient is instructed to maintain the heaviest cone possible within the vagina. Patients advance progressively to the use of heavier cones. This methodology is thought to allow for faster PFMT training.

In a Cochrane review by Herbison and Dean, 23 small clinical trials were analysed that compared

vaginal cones with traditional PFMT electrostimulation. The study was unable to identify if combination therapy with vaginal cones was better or worse than single modality. However, it was found that vaginal cones may be better than no active treatment and they may be a good conservative option as a method for PFMT [13].

ANTI-INCONTINENCE DEVICES

Vaginal support devices (Introl, Conveen, Ladycon, Femassist, Viva, Femsoft) can be considered depending upon the availability of product, patient acceptance, and cost and especially in younger patients who may be contemplating further pregnancies. Patient selection based on motivation, appropriate anatomy, and manual dexterity, in combination with efficacy and morbidity determines the overall satisfaction. The major morbidities are discomfort, urinary tract infections and haematuria. A recent Cochrane review looked at seven trials involving 787 women. Three small trials comparing mechanical devices (intravaginal such as pessary, sponge, or tampon-like device) with no treatment suggested that use of a mechanical device might be superior to no treatment; however, results were inconclusive [14].

PHARMACOTHERAPY

The stimulation of a1-adrenergic receptors in the bladder neck and the proximal urethra produces an increase in maximum urethral pressure and maximum urethral closure pressure. Women with stress incontinence have lower resting urethral pressures than age-matched continent women [15].

Estrogen supplementation in postmenopausal women has been advocated by several non-randomized clinical trials [16]. A critical analysis shows that the reported improvement was subjective with no change in the volume of urine lost and no change in maximum urethral pressure [17].

Propranolol, clenbuterol and methoxamine were reported to improve stress incontinence in studies with low level of evidence [18].

Duloxetine is a combined noradrenaline and serotonin reuptake inhibitor which increases the neural activity to the external urethral sphincter, and increase bladder capacity through central actions in the spinal cord [19]. Results from double-blind, placebo-controlled studies show that duloxetine (40 mg twice daily) decreases incontinence episode frequency (50% vs. 29% on placebo treatment) with comparable improvements in the more severely incontinent subgroup [20]. Duloxetine will be the first approved drug for the treatment of female stress incontinence with proven efficacy in randomized clinical trials. However, the discontinuation rate was higher with duloxetine (24% vs. 5% on placebo) with nausea being the most common reason for discontinuation.

Antimuscarinics and beta-adrenergic agonists are FDA-approved oral medications for urge UI. Antimuscarinics prevent recurrent spasm of the detrusor muscle, but side effects include tachycardia, edema, confusion, constipation, and blurry vision [21]. Selective antimuscarinic agents (darifenacin [Enablex], solifenacin [Vesicare]) are preferred over nonselective agents (oxybutynin, tolterodine [Detrol]) to reduce cognitive side effect [21]. Antimuscarinics are not recommended as first-line pharmacotherapy in older adults [22]. Mirabegron is a beta-adrenergic agonist that relaxes the detrusor muscle via beta-3 receptors [23]. Adverse effects include gastrointestinal upset, dizziness, headache, and increased blood pressure. Concurrent use with antimuscarinics increases the risk of urinary retention [21].

ELECTRICAL STIMULATION

Intravaginal pelvic floor electrostimulation devices are known for their low side-effect profile, which includes only burning or irritation at very high intensities. The mechanism of action for this modality relies on the electrical stimulation to induce hypertrophy of skeletal pelvic floor muscles via reflex contractions, while activating the detrusor inhibitory reflex arc. Prior studies have helped determine the optimal stimulation parameters for electrical stimulation, which include a stimulation frequency of 50 Hz, alternating or biphasic current, intermittent stimulation, and optimal stimulation intensity to allow for stimulation without pain.

Chêne and colleagues, identified 359 women with UI; of these women, 207 patients were identified with pure SUI. After treatment with pelvic floor muscle stimulation, the objective cure rate was found to be 65.7% in patients with pure SUI; failure rates for the group with SUI were found to be 19.8%. Measurements of levator ani muscle tones were also improved in these patients. Quality-of-life studies for all incontinence groups were improved after electrical stimulation in all groups (stress, urgency, and frequency). The overall patient satisfaction rate for this modality was found to be 83.6%, due to patients reporting satisfaction with ease, freedom, and rapidity of use, in addition to discretion [24].

INTRAVAGINAL LASER THERAPY

Stress urinary incontinence (SUI) is treated using intravaginal laser therapy.In a study by KUszka et al [25], Fifty-nine women, 32 with SUI I, 16 with SUI II, and 11 with SUI III were treated using an erbium-doped yttrium aluminium garnet (Er:YAG) laser following the IncontiLase® protocol. Therapy included five laser sessions with a 1-month interval between sessions. Objective (1-h pad test) and subjective data (International Consultation on Incontinence Questionnaire-Urinary Incontinence Short Form [ICIQ-UI SF], Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire [PISQ-12]) were assessed at baseline, after two and four laser sessions and 6 months and 2 years after the fifth laser session. They concluded intravaginal laser therapy led to cure/improvement for SUI I and SUI II, but not for severe SUI III. Outcome was better after four to five laser sessions than after two laser sessions.

According to the existing evidence, laser therapy may be a useful, minimally invasive approach for treating SUI. However, the methodological limitations of included studies render them prone to significant bias, limiting their scientific integrity [26].

SURGICAL MANAGEMENT

Surgical management of SUI includes many types of operations based on different principles with variable efficacy rates [Table 1]. The anterior colporrhaphy is associated with minimal morbidity but it is the least likely operation to be efficacious in the long term [27]. Open colpo-suspension (Burch) is a highly effective surgical treatment. Although efficacy declines with time, this method sustains a high cure rate in the long term. The Marshall–Marchetti– Kranz has also a high efficacy rate that sustains in time [28]. It is less popular than the open colposuspension due to an overall 22% complication rate (2.5% osteitis pubis. Slings with an autologous or synthetic material have high efficacy rates that sustain in time. Long-term studies using slings made of autologous or synthetic materials have indicated cure rates in excess of 80% and rates of improvement of greater than 90% [29]. Tension free vaginal tape (TVT) has efficacy rates similar to open colposuspension. Nilsson and colleague reported an excellent 5-year subjective and objective cure rate (84.7%) and a low failure rate (4.5%) using TVT, with no increase in the failure rate seen over a 5-year follow-up period [30]. The study comparing MUS and Burch coloposuspension confirms the superiority of MUS over Burch colposuspension [31]. The studies comparing insertion of RT-TVT and TO-TVT showed higher subjective and objective cure rates for the RP-TVT but at the cost of higher risks of some complications and voiding lower urinary tract symptoms [31]. Efficacy of inside-out and outside-in techniques of TO-TVT insertion was similar, although the risk of vaginal perforation was lower in the inside-to-out TO-TVT [31]. Efficacy rates of injectable bulking agents (PTFE, collagen, fat, Macroplastique, Durasphere) declines with time. Many women consider them an acceptable form of treatment because complications are rarely seen. There is a dearth of well-executed, published, peer-reviewed, randomized, placebo-controlled trials and comparator trials involving periurethral injectables [32].

Procedure	Short term results	Long term results
Anterior Colporrhaphy	63% cure at 1 year	37% cure at 5 years
Open Colposuspension	70-90% cure at 1 year	70% cure at 10–12 years
Laproscopic Colposuspension	80% cure at 1 year	77% cure at 5 years

Marshal-Marchetti -Kranz	90% cure at 1 year	75% cure at 15 years
Slings	80% cure at 1 year	80% cure at 15 years
Tension free Vaginal Tapes	80-90 % cure at 1 year	85% cure at 5 years
Peri urethral injection	60-90 % cure at 6 months	40–50% cure at 2 years
Artificial urinary sphincter	80 % cure	60% cure at 10–15 years

Castillo et al. [33] analyzed 91 surgical series of SUI and reported that 33% of these studies used only subjective measures, 4% only objective measures, and 63% both objective and subjective measures, for the definition of "cure." Not surprisingly, therefore, the cure rates of the same surgical procedure for SUI differed among these studies. A universal definition of cure is thus needed for accurate interpretation of cure rates when surgical treatment of SUI is contemplated.

DISCUSSION

Assessment of effectiveness of SUI treatment is a grey zone at best. In most clinical trials, the outcome measures are not comparable and there is no consensus on what is the most appropriate instrument to define efficacy. Efficacy is often expressed by means of cure (urodynamic diagnosis, no leakage episodes, < 2 g of leakage on pad test, women's report and questionnaires) or improvement based on dysfunction have been developed and validated. In most patients with uncomplicated SUI, the initial management involves a variety of non-invasive measures, including behavioral modification, pelvic floor exercises with or without biofeedback, and other accessory teaching aids. The rationale for conservative treatment is that UI is neither a life threatening or, necessarily, a progressive disease, and that conservative therapies can be effective, well tolerated, and safe. Furthermore, a moderate delay in surgical therapy does not make treatment more difficult or less effective [34]. Surgical correction of female SUI is directed toward one of the following 2 goals: 1) repositioning the urethra and/or creating a backboard of support or otherwise stabilizing the urethra and bladder neck in a well-supported retropubic (intraabdominal) position that is receptive to changes in intraabdominal pressure, or 2) creating coaptation and/or compression or otherwise augmenting the urethral resistance provided by the intrinsic sphincter unit, with (eg, sling) or without (eg, periurethral injectables) affecting urethral and bladder neck support or a combination of both. Surgeons tend to define the cure of SUI as the successful anatomical repair of defective tissue support of the urethra measured by objective improvement in urinary incontinence. However, these measures tend to underestimate the impact of lower urinary tract symptoms on social limitations and emotional well-being of the woman [35]. Patients may have completely different expectations of cure after surgery for SUI other than the anatomical cure, such as absence of odour, lack of disruption of routine activities, use of less pads, and/or improvement of emotional status. For patients, the improvement of these symptoms might be more important than the "objective" demonstration of leakage of urine. In fact, a number of patients who are considered as objectively cured from SUI might even feel worse following the operation due to surgery-induced negative outcomes such as de novo urge urinary incontinence or intermittent self-catheterization because of voiding dysfunction. Conversely, some patients who are considered not to be objectively cured by surgeons are satisfied with the outcome following the operation. This difference in perceptions of success of operative intervention between surgeons and patients may explain the discordant cure rates for surgical treatment of SUI reported in the literature [36]. The decision to treat symptomatic SUI with surgery should be made when the patient's degree of inconvenience and/or compromised lifestyle are great enough to warrant an elective operation and nonsurgical therapy is either not desired or has been previously ineffective.

Newer tools are evolving to evaluate prolapse, colorectal and sexual function such as the Pelvic Organ Prolapse-Urinary Incontinence Sexual Function Questionnaire in women with pelvic floor dysfunction. Such instruments have been primarily used to assess the impact of urinary incontinence such as the short and long forms of the Incontinence Impact Questionnaire, the Urogenital Distress Inventory and the Contilife [37]. Although there is a growing literature on the treatment of female stress incontinence,

many questions about efficacy of different treatment modalities are still unanswered. There is a need for high-quality randomized clinical trials that will share common methodology and endpoints.

CONCLUSION

There is no optimal therapy for all patients with SUI. However, the selection of an appropriate intervention for a properly motivated patient will most often result in an adequate improvement in symptoms. The patient's perspective concerning surgical intervention is the key point to be addressed when considering treatment of SUI. Patients most commonly list concerns of symptom relief and maintenance in activities of daily living. Achievement of

patient-selected goals should be the primary reason for selecting treatment. Studies attempting to bridge the gap between patient perception of surgical treatment and objective postoperative outcomes of SUI are awaited with interest. Until these data are available, there is an urgent need for a novel "gold standard" outcome measure that includes both the patient's expectations and surgeon's assessment and assigns them equal importance.

REFERENCES

1. Haylen BT, de Ridder D, Freeman RM. International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2010 Jan;29(1):4-20.
2. Rosenzweig BA, Hischke D, Thomas S et al (1991) Stress incontinence in women. Psychological status before and after treatment. J Reprod Med 36(12):835–838
3. Singh, Uma & Agarwal, Pragati & Verma, Manju & Dalela, Diwakar & Singh, Nisha & Sankhwar, Pushplata. (2013). Prevalence and risk factors of urinary incontinence in Indian women: A hospital-based survey. Indian journal of urology: IJU : journal of the Urological Society of India. 29. 31-6. 10.4103/0970-1591.109981.
4. Homma Y, Batista J, Bauer S, et al. Urodynamics. In: Abrams P, Cardozo L, Khoury S, Wein A, et al., editors. Incontinence. 2nd ed. Plymouth, UK: Health Publication Ltd; 2002. pp. 317–372.
5. Lucas MG, Bosch RJ, Burkhard FC, Cruz F, Madden TB, Nambiar AK, Neisius A, de Ridder DJ, Tubaro A, Turner WH, Pickard RS; European Association of Urology. EAU guidelines on assessment and nonsurgical management of urinary incontinence. Eur Urol. 2012 Dec;62(6):1130-42. doi: 10.1016/j.eururo.2012.08.047. Epub 2012 Aug 31. Erratum in: Eur Urol. 2013 Jul;64(1):e20. PMID: 22985745.
6. Brubaker L, Shull B. EGGS for patient-centered outcomes.
7. Caffeine and urinary incontinence in US women. Gleason JL, Richter HE, Redden DT, Goode PS, Burgio KL, Markland AD. Int Urogynecol J. 2013 Feb; 24(2):295-302
8. Bo K, Stien R (1994) Needle EMG registration of striated urethral wall and pelvic floor muscle activity patterns during cough, Valsalva, abdominal, hip adductor, and gluteal muscle contractions in nulliparous healthy females. Neurourol Urodyn 13(1):35–41
9. Gormley EA (2002) Biofeedback and behavioral therapy for the management of female urinary incontinence. Urol Clin North Am 29(3):551–557
10. Miller JM, Ashton-Miller JA, DeLancey JO (1998) A 639 pelvic muscle precontraction can reduce cough-re640 lated urine loss in selected women with mild SUI. J 641 Am Geriatr Soc 46(7):870–874
11. Bo K, Talseth T, Holme I (1999) Single blind, randomised controlled trial of pelvic floor exercises, electrical stimulation, vaginal cones, and no treatment in management of genuine stress incontinence in women. BMJ 318(7182):487–493
12. Blaivas JG, Appell RA, Fantl JA et al (1997) Standards of efficacy for evaluation of treatment outcomes in urinary incontinence: recommendations of the Urodynamic Society. Neurourol Urodyn 16(3):145–147

URINARY TRACT INFECTIONS IN PREGNANCY

Dr Riti Seth
Professor and HOD Department of Microbiology
NSCB medical college Jabalpur

Urinary tract infections (UTIs) is defined as "the establishment and multiplication of micro-organisms within the urinary tract," whereas bacteriuria is the detection of bacteria in a voided urine specimen. Significant bacteriuria is defined as greater than 1 x 106 of the same organism per milliliter of urine. Bacteriuria in pregnancy is often asymptomatic. Cystitis is defined as a symptomatic significant bacteriuria with associated bladder mucosal inflammation, whereas pyelonephritis is defined as symptomatic significant bacteriuria with associated inflammation of the renal parenchyma, calices, and pelvis. UTI are often seen to complicate pregnancy.

Various studies have established a 5% to 10% prevalence of asymptomatic bacteriuria (ASB) in pregnancy and the association of ASB with the development of symptomatic infections, including pyelonephritis. 25-30% of cases of ASB in pregnancy usually progress to symptomatic infection. This progression from asymptomatic to symptomatic infection is 3-4 times with pregnancy as compared with nonpregnant females. Pyelonephritis has been associated with maternal and fetal complications. Obstetric complications like preterm labor and low birth weight have been associated with bacteriuria.

PHYSIOLOGIC CHANGES OF THE URINARY TRACT IN PREGNANCY

Mechanical and hormonal factors contribute significantly to physiologic urinary tract changes typical for pregnancy.Usually, the ascending bacteria from contiguous sites are eliminated by voiding and local defense mechanisms but infection develops in some instances. In about 80% of pregnant women urinary tract is dilated combined with slight hydronephrosis, caused partly by a reduction in smooth muscle tone with slowing of ureteral peristalsis, and partly by urethral sphincter relaxation. This could be due to high levels of circulating progesterone. Simultaneously, the gravid uterus compresses the urinary bladder, thus increasing the pressure inside the bladder, which may lead to vesico-ureteral reflux and urinay retention in the bladder after micturition. Urinary stasis and impaired physiological anti-reflux mechanism create favorable conditions for bacterial growth and ascending infection. The other predisposing factors include pregnancy-specific biochemical changes in urine, with higher levelsof glucose, amino acids and hormone degradation products, which increase urinary pH.

TABLE 1: URINARY TRACT CHANGES IN PREGNANCY(structural and functional)

ANATOMICAL SITE	FACTOR
Kidney	Renal length is increased along with increase in glomerular filteration rate by 30%-50%
Collecting system	Peristalsis is decreased
ureters	Peristalsis is decreased due to muscle relaxing effects of progesterone Mechanical obstruction due to gravid uterus
bladder	Pushed anterosuperiorly Smooth muscle relaxation Increased capacity
Biochemical changes in urine	higher amounts of glucose, amino acids and hormone degradation products, which increase urinary pH

Data from Waltzer WC. The urinary tract in pregnancy. J Urol 1981;125(3):271–6.

Pregnancy-related hormonal factors that may increase susceptibility to UTIs include progesterone-induced ureteral and vesicular smooth muscle relaxation and estrogen mediated upper urinary tract bacterial infection (particularly with Escherichia coli).This hormonal effect may be related to the decreased ability of the kidneys to concentrate during pregnancy and a decrease in normal antibacterial activity of urine. Estrogens may also be associated with an increased incidence of bacteriuria, as seen in studies of women taking oral contraceptives. Other pregnancy-related changes include pregnancy-associated glucosuria and aminoaciduria, both of which contribute to bacteriuria by providing an excellent proliferation medium for bacteria.

EPIDEMIOLOGY AND MICROBIOLOGY

The urinary tract is sterile normally. Bacteriuria occurs because of ascending infection by fecal bacteria or vaginal/perineal skin colonisers. The risk factors contributing to bacteriuria and the organisms responsible are similar in pregnant and nonpregnant women. The relatively short female urethra, (3 to 4 cm in length), its proximity to the vagina and frequent colonization with organisms from the gastrointestinal tract are some of the important predisposing factors. As stated by Patterson and Andriole, "bacteriuria usually reflects prior colonization rather than acquisition during the pregnancy itself." Bacterial pathogens responsible for ASB, cystitis, and pyelonephritis are almost same, with E. coli accounting for the majority of UTIs (upto 80% of cases). Klebsiella and Entero-bacter,Proteus, Pseudomonas, and Citrobacter are also common pathogens. Among Gram-positive organisms, group B Streptococci (GBS), are common and lead to up to 10% of UTIs in pregnant women. Infection with other less common microorganisms, includes Mycoplasma hominis, Ureaplasma parvum, Gardnerella vaginalis, Chlamydia trachomatis, and lacto- bacilli.The significance of low colony counts (less than 105 organisms/mL) of gram positive organisms isolated from the urine of asymptomatic pregnant patients is not certain, although symptomatic UTIs have been reported with low colony counts of many gram positive isolates.

Normal host defence mechanisms against ascending colonization of the urethra by vaginal and distal urethral flora include micturition and a uroepithelial surface mucoprotein. These mechanisms are not always able to prevent, with upto 10% prevalence of ASB reported in pregnant women in various studies worldwide. Major Risk factors are sexual activity, increasing parity, low socioeconomic status,sickle cell disease or trait (with associated renal parenchymal involvement) diabetes mellitus, a history of UTIs, and functional/anatomic abnormalities, that are associated with an increasing frequency of UTIs in both the pregnant and nonpregnant phase. In a study, UTIs during the first 20 weeks of pregnancy and a history of UTIs before pregnancy were associated with at least a two times increased risk of symptomatic UTI during pregnancy.

SIGNIFICANCE OF BACTERIURIA DURING PREGNANCY

In the literature, the development of symptomatic UTIs in pregnancy with untreated ASB is well-reported.However the association of untreated ASB in pregnancy with risks of other maternal and fetal complication is not very clear. Norden and Kass reported that only 1% to 2% of pregnant women without bacteriuria in the first trimester developed symptomatic UTIs during pregnancy however untreated ASB may lead to development of acute pyelonephritis in 20% to 57% of cases. Nevertheless, other reserchers reported that acute pyelonephritis developed in 14% of pregnant patients with untreated ASB, also postpartum UTI was more common in patients with bacteriuria during pregnancy. The efficient treatment of ASB reduces the chances of subsequent symptomatic UTIs by 80% to 90%, with symptomatic infection more likely in patients in whom antibiotic therapy was not effective in clearance of bacteriuria. Untreated ASB also increases the risk of preterm labor and of delivering a low birth weight baby. The association of pyelonephritis with these complications is less clear especially in presence of confounding factors like socioeconomic status.

The mechanism of association of preterm labor and symptomatic UTIs is not very clear but is thought to be related to prostaglandin activation. As reported in a study, "pyelonephritis appears to be associated with preterm birth and low birth weight although the strength of the association is unclear." Other pregnancy-related complications like anemia and preeclampsias have also been reported to be associated with ASB, however, the strength of this association could not be established because of presence of confounding factors like socioeconomic status.

SCREENING FOR BACTERIURIA IN PREGNANCY

Lower urinary tract function and symptomatology do not correlate well in both the nonpregnant and pregnant. Relatively few studies have examined lower urinary tract symptoms in pregnancy. Frequency and urgency, which are indicators of the infection processes, are commonly experienced by many pregnant women even in the absence of a UTI. Pregnancy itself, in the absence of infection can cause most of the symptoms described by clinicians and patients in the nonpregnant state that direct towards diagnostic testing for infection. However Screening Studies In a meta-analysisfound 50% risk of preterm delivery and 65% risk of low birth weight infants in nonbacteriuric compared with bacteriuric patients. This difference in risk has led the policymakers to recommend screening for ASB in pregnancy. The major impact of such screening has been substantial decrease in the incidence of pyelonephritis over years, with studies reporting a decrease from 4% to 0.8% since the initiation of routine screening and treatment for ASB.

Although a catheterized urine specimen can detect ASB in 96% of cases, it is not recommended and may further lead to urinary tract colonization. The single clean-catch urine culture accurately detects ASB in 80% of cases, with two clean catch voided specimens getting the same accuracy as achieved by catheterization.

Most of these women having bacteriuria are culture-positive at the time of initial culture. The urine culture should be performed from a midstream sample after appropriate preparation of the external genitalia before passing the urine. Voided samples kept at room temperature for few hours before testing can have falsely increased colony forming units, hence samples should be sent to the laboratory at collection or refrigerated at approximately 4°C for upto 24 hours. Within the first 24 hours, a colony counting can be done; within 48 hours, the bacterial organism can be identified. The growth of multiple bacterial isolates or lactobacillus or both is mostly a result of contamination. Although urine cultures are expensive and labor intensive, they are the screening tool of choice because other screening methods are not accurate to that level. Screening methods such as microscopic urine examination and the use of urine reagent sticks do not have adequate sensitivity and specificity for antenatal screening. Microscopic urinalysis detects only 25% to 67% of infected samples however they have a good specificity ranging from 97% to 100%.8, 75 Gram staining may be used as an acceptable alternative, with a reported sensitivity of 90% and specificity of 80%, although it requires a trained and skilled person.Therefore, in light of the consequences of bacteriuria and pyelonephritis in pregnancy and because culture data are important to administer appropriate antimicrobial therapy in the era of increasing antimicrobial resistance, urine culture remains the recommended screening test.

CATEGORIZING URINARY TRACT INFECTIONS IN PREGNANCY

The three categories of UTIs include ASB, cystitis, and pyelonephritis. Kassestablished that a colony count of more than 105 bacteria from a midstream clean catch specimen differentiates contamination from significant bacteriuria.

ASYMPTOMATIC BACTERIURIA

Asymptomatic bacterial colonization of the urinary tract, occurs in 5% to 10% of pregnant women. Because the prevalence of ASB is comparable in the pregnant and nonpregnant states, pregnancy is not considered as a predisposing factor, however there are other factors like Sexual activity, increasing age, parity, lower socioeconomic status, a history of UTIs, sickle cell disease or trait, and diabetes

mellitus, which act as predisposing risk factors for ASB. The increased 20% to 30% risk for acute pyelonephritis in pregnancy needs treatment of ASB. This treatment decreases the risk of developing acute pyelonephritis by 80% and the rate of persistent bacteriuria from 86% to 11%. A study reported a small reduction, 15% to l0%, in the rate of low birth weight infants in women treated for ASB as compared with untreated ones. Various Randomized trials comparing different antibiotic regimens have shown that all are equally effective in clearing bacteriuria.Various single-dose treatment protocols have also been studied. Although no trials have shown lower rates of persistent bacteriuria or pyelonephritis with longer durations of therapy.No study has had adequate evidence to identify the optimal treatment regimen. Adverse effects of antibiotic treatment of ASB in pregnancy can lead to medical complications like allergic reactions including anaphylaxis to penicillins and cephalosporins, various fetal complications related to sulfonamides prescribed late in pregnancy, just prior to birth. Trimethoprim, an antifolate agent, is relatively contraindicated in the first trimester of pregnancy, and nitrofurantoin given to the patient or fetus with glucose-6-phosphate dehydrogenase deficiency may lead to hemolysis. Thus antibiotic treatment also has its own pros n cons. The pregnant patient with a positive urine culture needs to get a repeat urine culture done 1 week after therapy as a "test of cure." If the follow-up culture is negative, surveillance cultures at monthly interval are recommended until delivery. It is also recommended to consider nitrofurantoin prophylaxis, if persistence or reappearance of organisms on urine culture.

CYSTITIS

Cystitis leads to complications in 0.3% to 1.3% of pregnancies.Cystitis is defined as a symptomatic UTI withcolony count of more than 105 on a cleancatch, midstream voided urine. Antenatal screening programs aimed at identifying ASB have not decreased rates of cystitis during pregnancy. The effects of cystitis on preterm delivery, low birth weight, and pyelonephritis are not different from ASB. Treatment options are also similar to those for ASB. Because 18% of patients with acute cystitis have positive urine cultures later in pregnancy, routine repeat surveillance urine cultures throughout the pregnancy are recommended.

ACUTE PYELONEPHRITIS

The incidence of pyelonephritis, which complicates 1% to 2% of pregnancies, is dependent on the prevalence of ASB and whether ASB has been treated. The onset of acute pyelonephritis is typically indicated by fever, costovertebral angle tenderness, and symptoms suggestive of cystitis i.e. Frequency, urgency and burning micturition. Other associated symptoms may include nausea, vomiting, chills, and, in severe cases, sepsis and respiratory insufficiency. On laboratory investigation, presence of pus cells in urine is common. White blood cell casts confirm the diagnosis, and one to two bacteria per high power field on an uncentrifuged catheterized specimen (or greater than 20 bacteria per high power field on a centrifuged specimen) are seen. Blood cultures may be positive in up to 15% of cases. Bacterial endotoxin-mediated damage can include altered alveolar-capillary membrane permeability and can lead to pulmonary edema and respiratory insufficiency in up to 8% of patient. Other medical complications include altered renal function in 25% of pregnant patients with pyelonephritis (this is often a transient dysfunction, usually resolves on its own within a few days) and anemia, seen in 25% to 66% of pregnant patients with pyelonephritis. Endotoxin-mediated, lipopolysaccharide-induced damage of red blood cell membranes seems to be the mechanism behind this, which also resolves with treatment of the infection. As mentioned previously, pyelonephritis seems to be associated with preterm delivery and low birth weight, although the strength of this association has not been established. The treatment of pyelonephritis during pregnancy has conventionally been on indoor basis, however care constraints and other economic concerns have driven efforts to go for outpatient treatment. Outpatient pyelonephritis therapy can be used in selected nonbacteremic patients and it has been shown to be safe and effective. Similar rates of persistent or recurrent bacteriuria and recurrent pyelonephritis have been observed in selected pregnant patients treated as

inpatients versus outpatients. Antibiotics commonly used include cephalosporins or penicillins with an aminoglycoside since the approximate 30% resistance rate of urinary tract E. coli to ampicillin has been observed. More than 95% of patients respond markedly within 72 hours of therapy and patients who do not improve should undergo renal ultrasound to rule out a potentially obstructive uropathy secondary to nephrolithiasis. After the completion of a 10- to 14-day course of antibiotics, nightly prophylaxis with an agent such as nitrofurantoin is generally recommended for the duration of the pregnancy. It has been reported that antibiotic prophylaxis decreased the incidence of recurrent or persistent bacteriuria after pyelonephritis. Thus, antibiotic prophylaxis is recommended until delivery in the pregnant patient who sustains pyelonephritis to decrease the risk of recurrent infection.

RECURRENCE AND PREVENTION

In the academic community, two uncomplicated UTIs over a 6-month time period or three UTIs in a year are considered as minimum criteria for a diagnosis of recurrent UTI. The suffering associated with recurrent UTIs in women is significant, and these patients often pose challengesforclinicians. Recurrent UTI might be one of the most common problems. Treating UTI might not be difficult, but preventing UTI recurrence sometimes might be very troublesome for both patients and doctors. Recent studies have revealed many novel concepts in recurrent UTI, including the pathogenesis, risk factors, biomarkers, and prevention. Nowadays, recurrent UTI may be considered as a distinct entity, and patients with recurrent UTI should be managed proactively. Further studies are needed to get details in the pathogenesis, and to clarify the efficacy of the current management. There are many serum and urine biomarkers under study for diagnosis of recurrent UTI(table 2)

Table2: Possible biomarkers for recurrent urinary tract infection

Serum biomarker	**Urine biomarker**
Granulocyte colony-stimulating factor elevated	Nerve growth factor decreased
Prostate-specific antigen decreased	Neutrophil gelatinase -associated lipocalin decreased
Vitamin D decreased	IL-8 elevated
Macrophage colony-stimulating factor elevated	
IL-5 elevated	
IgG, IgM, and IgA elevated	

Ig: Immunoglobulin, IL: Interleukin,

There are various possible options for prophylaxis for recurrent urinary tract infection that includes behavioral modifications and avoidance of risk factors ,nonantimicrobial measures and antimicrobial prophylaxis as mentioned in table 3.

Table3: Possible prophylaxis options for recurrent urinary tract infection

Prophylaxis option	methods
behavioral modifications and avoidance of risk factors	Avoiding use of diaphragm,spermicide for contraception Voiding soon after intercourse Increasing volume of fluid intake when UTI symptomatology noted
nonantimicrobial measures	cranberry juice probiotics hormonal replacement prophylaxis Immunoactive agent prophylaxis
antimicrobial prophylaxis	Various antibiotics may be administered as continuous or postcoital prophylaxis

SUMMARY

Although pregnancy does not increase the prevalence of ASB in women, it does enhance the progression rate from asymptomatic to symptomatic disease. Furthermore, ASB is associated with preterm delivery. Considering that identification and treatment of ASB in pregnant women can reduce the chances of developing pyelonephritis and prevent preterm delivery, every pregnant lady should be systematically screened for ASB and appropriately treated. In the majority of studies, a first-trimester urine culture remains the screening test of choice; reliance on symptoms to prompt screening is inadequate because the state of pregnancy itself can provoke frequency and nocturia. Multiple antibiotic regimens for ASB are safe during pregnancy and effective.However one should also remember that antibiotic resistance of bacteria may differ depending on geographic area, hospital and even hospital ward, and the information on this topic may be crucial when making therapeutic decisions.

REFERENCES

1. Alexander P. G, Anthony J. S . Urinary Tract Infection and Bacteriuria in Pregnancy:Urol Clin N Am 42 (2015) 547–560
2. Flavia G, Amelia H, Kath R:Urinary tract infections and antibiotic use in pregnancy - qualitative analysis of online forum content :BMC Pregnancy and Childbirth (2019) ;19:289
3. Jack D. S:Pathogenesis of urinary tract infection, Role of Host Defenses Infect Dis Clin North Am:1997; 11 , (3) : 531-549
4. Joanna MR, Jolanta M, Monika W.Urinary tract infections in pregnancy: old and new unresolved diagnostic and therapeutic problems;Arch Med Sci 2015; 11, 1: 67–77
5. Kass EH: Bacteriuria and the diagnosis of infection of the urinary tract. Arch Intern Med 100:709-714, 1957
6. Macejko AM, Schaeffer AJ. Asymptomatic bacteriuria and symptomatic urinary tract infections during pregnancy. Urol Clin North Am 2007;34:35–42
7. Nicole MG; Valerie P. O, Scott H; George M; Warren G. L; Amanda L. L:Urinary Tract Infection as a Preventable Cause of Pregnancy Complications: Opportunities, Challenges, and a Global Call to Action:Global Adv Health Med. 2013;2(5):59-69. DOI: 10.7453/gahmj.2013.061
8. Patterson TF, Andriole VT. Detection, significance, and therapy of bacteriuria in pregnancy. Update in the managed health care era. Infect Dis Clin North Am 1997;11(3):593–608.
9. Schieve LA, Handler A, Hershow R, et al. Urinary tract infection during pregnancy: its association with maternal morbidity and perinatal outcome. Am J Public Health 1994;84(3):405–10.
10. Waltzer WC. The urinary tract in pregnancy. J Urol 1981;3:271–6.

GENITOURINARY SYNDROME; THE AGONY OF MENOPAUSE

Dr Jigyasa Dengra, Dr (Prof.) Gita Guin
Director & Consultant
Mahi IVF Jabalpur

INTRODUCTION

Genitourinary syndrome of menopause (GSM) is the new term for vulvovaginal atrophy (VVA). Genitourinary syndrome of menopause describes the symptoms and signs resulting from the effect of estrogen deficiency on the female genitourinary tract, including the vagina, labia, urethra, and bladder. This syndrome includes genital symptoms of dryness, burning, and irritation; urinary symptoms and conditions of dysuria, urgency, and recurrent urinary tract infections (UTIs); and sexual symptoms of pain and dryness. Physical changes and signs are varied. Women may experience some or all of the symptoms and signs, which must be bothersome for a diagnosis of the syndrome. Other causes of similar signs and symptoms must be ruled out, including vulvovaginal dermatoses, infection, or cancer.

Vulvovaginal atrophy is a component of GSM. Although VVA was the commonly used term in the past to describe the genitourinary changes of menopause, it has limitations. Vulvovaginal atrophy describes the appearance of the genital tissues but not the associated symptoms. It does not include urinary tract changes related to estrogen deficiency, and the term atrophy has negative associations for women. The term genitourinary syndrome of menopause was developed during a consensus conference of experts and subsequently was accepted as the preferred term by many medical societies.

It is bothersome in more than 50% of women, having an adverse impact on quality of life, social activity and sexual relationships. GSM is a chronic and progressive syndrome that is under diagnosed and undertreated. In contrast to vasomotor symptoms that usually improve over time, GSM is generally progressive if appropriate treatment is not given.

PATHOPHYSIOLOGY AND ANATOMY

The genital and lower urinary tract share a common embryologic origin in women, with the urethra, bladder trigone, vulvar vestibule, and the upper vagina all derived from the same estrogen receptor (ER)-rich primitive urogenital sinus tissue. The vulva is also derived from the urogenital sinus, but the epithelium of the labia majora is of ectodermal origin.

The vagina is composed of an inner stratified squamous epithelium, a middle muscular layer, and an outer fibrous layer. In the presence of endogenous estrogen after puberty and before menopause, the lining of the vagina is characterized by a thickened, rugated surface that is well vascularized and lubricated in most women. Estrogen is a dominant regulator of vaginal and lower urinary tract physiology. Estrogen receptor-a is present in the vaginal tissues of both premenopausal and postmenopausal women, whereas ER-b appears to have no or low expression in postmenopausal vaginal tissue. Estrogen therapy (ET) does not appear to affect the presence of ER-b. Estrogen receptor density is highest in the vagina, with decreasing density across the external genitalia to the skin. The density of the androgen receptor is the reverse. There are low levels in the vagina and higher levels in the external genitalia. Progesterone receptors are found in the vagina and the transitional epithelium of the vulvovaginal junction.Estrogen receptors have also been found on autonomic and sensory neurons in the vagina and vulva. Estrogen therapy has been reported to decrease the density of sensory nociceptor neurons in the vagina. This function may serve to decrease the discomfort

associated with GSM. With respect to the lower urinary tract, estrogen and progesterone receptors have been identified in the urethra, bladder, and pelvic floor muscles. The changing physiology of the vaginal epithelium after menopause is not completely understood.

On the basis of a cell-culture model that used vaginal-cervical epithelial cells, diminished estrogen levels and aging were found to be independent factors in decreasing vaginal-cervical paracellular permeability, a change potentially related to vaginal dryness. With atrophy, wet-mount microscopy shows more than one white blood cell per epithelial cell and immature vaginal epithelial cells with relatively large nuclei (parabasal cells). Cytology shows an increase in parabasal and intermediate cells, and superficial cells decrease or are absent.Immune cell populations seem to be similar or slightly decreased in number, with similar cytolytic capacity as before menopause. However, some studies show differences in inflammatory markers in the vaginal fluid of postmenopausal women compared with premenopausal women.

Hormone changes throughout the life cycle influence the vaginal microbiome from birth through postmenopause.During the reproductive years, the presence of a microbial community dominated by Lactobacillus species is associated with a lower pH and lower risk for bacterial vaginosis (BV), sexually transmitted infections, UTIs, and HIV infection.After menopause, women are less likely to have a Lactobacillus-dominant vaginal bacterial community and less likely to have a low vaginal pH.Although cultivation-based studies show a significantly lower quantity of vaginal Lactobacillus in postmenopausal women, several newer sequencing studies observe that close to half have a high proportion of lactobacilli. In one study, a higher proportion of Lactobacillus correlated inversely with examiner-reported dryness in postmenopausal women, but in another study, there was no association between Lactobacillus dominance and the severity of patient-reported symptoms.The vaginal bacteria community of postmenopausal women has many similarities with that of reproductive-aged women with BV: high pH, higher diversity, and an abnormal Nugent score. In many women with GSM, however, these abnormalities reflect a decline in lactobacilli rather than an increase in the prevalence of pathogens. Treatment with systemic or topical estrogen is associated with an increase in detection of vaginal lactobacilli. This suggests that for many postmenopausal women, the best approach to promoting a healthy vaginal microbial community is not antibiotic therapy (as though treating BV) but rather vaginal estrogen therapy

Box 1. Anatomical and functional changes in the genitourinary tissues

- Loss of labial and vulval fullness
- Contraction of labia majora and clitoral hood
- Narrowing and stenosis of the introitus
- Loss of hymenal remnants or reduced elasticity
- Vaginal shortening and narrowing
- Prolapse
- Pelvic floor weakening
- Vaginal epithelium dry and thin with petechiae
- Loss of superficial cells and increase in parabasal cells
- Loss of vaginal rugae
- Inflamed vaginal tissues
- Alkaline pH changes the vaginal microbiome with loss of Lactobacilli (vaginal pH >4.5)
- Persistent or recurrent discharge with odour
- Urethral meatal prominence and prolapse with thinning of the urethral epithelium
- Touch perception altered either hypersensitive or decreased feeling
- Loss of clitoral stimulation

DIAGNOSIS:

The diagnosis of GSM requires the presence of both characteristic examination findings and bothersome symptoms.

The most commonly reported symptoms include irritation of the vulva, inadequate vaginal lubrication, burning, dysuria, dyspareunia, and vaginal discharge. Symptoms adversely affecting sexual function are often the most distressing.

Signs of GSM include labial atrophy, vaginal dryness, introital stenosis, clitoral atrophy, and phimosis of the prepuce. Severe GSM can result in a vaginal surface that is friable and hypopigmented, with petechiae, ulcerations, and tears, as well as urethral findings such as caruncles, prolapse, or polyps. Bleeding may occur from minimal trauma, such as speculum insertion. Genitourinary atrophic changes increase the likelihood of trauma, pain, recurrent UTIs, bleeding with or after sex, and absence of sexual activity. The genitourinary syndrome of menopause most commonly develops in the setting of hypoestrogenism associated with natural menopause. Hypoestrogenic states also may occur in the setting of primary ovarian insufficiency (POI), surgical menopause (bilateral oophorectomy with or without hysterectomy), hypothalamic amenorrhea, the postpartum state and breastfeeding, use of gonadotropin-releasing hormone agonists or aromatase inhibitors (AIs), and cancer treatments such as surgery, pelvic radiation therapy, or chemotherapy that render ovaries inactive, either temporarily or permanently. Several studies suggest that early estrogen deficiency caused by premature menopause or POI is associated with more severe sexual dysfunction compared with age-matched controls.Younger women with vaginal atrophy and dyspareunia may be especially distressed by changes in sexual function. Women with surgical menopause often present with a more severe GSM symptom profile than do women with natural menopause, likely because of the concomitant, abrupt, and persistent 50% decline in circulating androgen levels that occurs in addition to the loss of estradiol. Genitourinary syndrome of menopause that develops in the setting of chemotherapy-induced menopause has been associated in some studies with greater sexual dysfunction and distress and with poorer QOL outcomes. Younger women with GSM related to induced menopause from cancer treatment may be especially distressed by changes in sexual function.The stress, fatigue, and mood changes that accompany a cancer diagnosis and its treatment also contribute to sexual problems. Aromatase inhibitors reduce breast cancer recurrence by blocking conversion of androgens to estrogens and inducing a profound estrogen-deficiency state. The magnitude and duration of estrogen deficiency induced by A Is result in the development of severe GSM in most survivors, particularly given that extended duration therapy is now typical. Compared with tamoxifen, A Is result in a greater incidence of vaginal dryness and dyspareunia, causing a large percentage of AI users to express dissatisfaction with their sex lives.

Societal views about women's sexuality at older ages are essentially negative, and sexual problems are often considered to be part of normal ageing leading to many women not seeking help for their symptoms.

There is a disparity between the number of women who experience bothersome symptoms and those who are treated. First, women are unwilling, shy or embarrassed to discuss their symptoms with their healthcare professional, especially if:

- the healthcare professional is young and male
- the patient has had a previous negative experience with a healthcare professional
- the patient regards her symptoms as a natural part of ageing that she should 'put up with'.

Women often wait for their healthcare professional to ask the questions.

Second, the healthcare professional may be embarrassed or reluctant to ask appropriate questions (especially about sexual function), dismiss the symptoms as part of normal ageing, or feel pressured for

time. Healthcare professionals may also be unaware of available treatments or their recommended doses, and may treat inadequately and in the short term.

Box 2. Risk factors for genitourinary syndrome of menopause

- Menopause
- Bilateral oophorectomy
- Premature ovarian failure
- Smoking
- Alcohol abuse
- Decreased sexual frequency or abstinence
- Lack of a vaginal birth
- Other causes of low estrogen (e.g. postpartum period, hypothalamic amenorrhea)

· Cancer treatments, including pelvic irradiation, chemotherapy and endocrine therapyThe pelvic examination helps to exclude other vulvovaginal conditions that can cause similar symptoms. As GSM progresses, examination of the external genitalia often reveals reduced mons pubis and labia majora bulk, reduced labia minora tissue and pigmentation, and prominence (telescoping) and erythema of the urethral meatus. Urethral caruncle, a benign outgrowth of inflammatory tissue arising from the posterior urethral meatus, is common in postmenopausal women and likely related to hypoestrogenism. The clitoris may recede and in some cases become completely flush with the surrounding tissue. The vestibular tissue may become pale. If the introitus is noted to be narrow, use of a narrow pediatric vaginal speculum with lubricant is appropriate. The vaginal mucosa may appear smooth (loss of rugation), shiny, and dry. Minimal blunt trauma from the speculum may result in petechiae (reflecting mucosal thinning) or bleeding (friability). With progression of GSM, attenuation of the vaginal fornices may be apparent, and the cervix may appear flush with the vaginal apex. With atrophic vaginitis, brown or yellow (sometimes malodorous) discharge may be present. With severe GSM, there may be such shortening of the vaginal vault and narrowing of the introitus that speculum insertion and visual inspection of the vaginal vault as well as cervix may not be possible. Although the vaginal maturation index (VMI) and vaginal pH are routinely assessed in clinical trials, they are not essential to make a diagnosis of GSM in clinical practice. With GSM, vaginal pH is typically greater than 5.0. Wetmount microscopy shows more than one white blood cell per epithelial cell, immature vaginal epithelial cells with relatively large nuclei (parabasal cells), and reduced or absent lactobacilli. Repopulation with diverse flora can occur including enteric organisms commonly associated with UTIs.67 The appearance of the wet mount in severe GSM may be difficult to distinguish from that of desquamative inflammatory vaginitis or vaginal erosive lichen planus.68 A culture or vulvovaginal biopsy should be considered if there are atypical findings or if the vulvovaginal symptoms fail to resolve after a trial of vaginal estrogens or DHEA. A woman's symptoms do not always correlate with physical findings. For example, a woman who is not sexually active may have few symptoms, despite signs of advanced genitourinary atrophy on examination. In contrast, a woman with an active sex life may complain of dryness and discomfort with sex, whereas the pelvic examination suggests only mild atrophy. Of note, women who are not sexually active also may be bothered by symptoms related to GSM, including discomfort with exercise or dysuria and benefit from treatment. Thus, both history and examination are essential to make the correct diagnosis.

DIFFERENTIAL DIAGNOSIS

Symptoms similar to GSM result from many other conditions. The differential diagnosis includes allergic or inflammatory conditions (e.g., lichen sclerosus, erosive lichen planus, desquamative inflammatory vaginitis, contact dermatitis, and cicatricial pemphigoid), vulvovaginal candidiasis and other infections, trauma, foreign bodies, malignancy, vulvodynia, vestibulodynia, chronic pelvic pain, provoked pelvic floor hypertonia (previously known as vaginismus), and other medical conditions (eg, diabetes, lupus erythematosus) or psychological disorders. An alternate etiology is more likely in women with chronic or

recurrent vulvovaginal symptoms that were present before menopause.

The aim of management and treatment of GSM is to provide symptom relief. However, the consultation is also an appropriate time to discuss lifestyle, diet and exercise, smoking cessation and appropriate alcohol consumption. Also use the time to perform routine cervical screening and STI tests, if indicated.

INVESTIGATIONS

Investigations are not routinely performed, but are ordered depending on specific findings and possible differential diagnoses (Box 3). Investigation of any postmenopausal bleeding to exclude gynecological malignancy is imperative.

A vaginal or vulval swab may be taken if there is a vaginal discharge or a vulvitis to exclude infection. A vulval biopsy may be necessary if the vulval findings are suspicious or do not respond to the recommended treatment. Urine investigations may be required if symptoms are related to bladder and urethra.

Box 3. Differential diagnosis

- Dermatological conditions of the vulva (eg lichen sclerosus or planus, eczema, dermatitis, chronic vulvovaginitis)
- Vulvodynia, vaginismus
- Autoimmune disorders
- Malignancy
- Chronic pelvic pain
- Trauma, foreign bodies
- Diabetes
- Lupus

TREATMENT

Treatment will depend on the symptoms and signs, and the degree of severity.

Non-hormonal therapies include personal lubricants, vaginal moisturizers and vaginal laser (long-term safety and efficacy have not been established). Hormonal therapies include vaginal estriol cream or pessaries, vaginal estradiol tablets, or systemic hormone therapy (menopause hormone therapy [MHT]).

PERSONAL LUBRICANTS AND VAGINAL MOISTURIZERS

Lubricants and vaginal moisturizers are effective in relieving discomfort, friction and pain with penetrative sex. Lubricants are used at the time of intercourse, whereas vaginal moisturizers provide longer term relief. Lubricants can be water-based or silicone-based. Water-based lubricants are non-staining and have fewer side effects than silicone-based lubricants. However, the efficacy of lubricants depends on the osmolality, pH and additives of each individual product. High osmolality, >1200 mOsm/kg, is associated with irritation, contact dermatitis and cytotoxicity. Oils, such as olive or sweet almond oil, are alternatives.

Moisturizers rehydrate dry tissues by changing the fluid content in the vaginal epithelium, absorbing and adhering to it, mimicking vaginal secretions, and lowering the pH. The effect lasts about three days. Moisturizers contain polymers for adherence and other additives that effect osmolality and pH.

HORMONAL THERAPIES

Estrogen vaginal preparations reduce symptoms and reverse the atrophic changes in pelvic tissues,

and improve blood flow and the thickness of the epithelium in the vagina, bladder and urethra. There is minimal systemic absorption, with an initial peak, then almost no further absorption. Vaginal estriol preparations of cream and pessaries provide a human estrogen. Estriol is the weakest estrogen and has one-tenth of the potency of estradiol. There is minimal absorption systemically and estriol cannot be metabolized to estradiol or estrone. Low-dose vaginal estradiol tablets are also very effective in relieving atrophic symptoms. The individual dose is 10 µg, and studies have found that the annual absorption of estradiol is only 1.14 mg.

There are no studies on the long-term risks of vaginal estrogen preparations, but absorption is negligible once the atrophic changes are reversed. Added progestogens are not needed to prevent endometrial stimulation. The safety in breast cancer survivors is not established, especially with aromatase inhibitors, because of the possible risk of recurrence. In women with breast cancer, vaginal estriol preparations are prescribed on an individual basis, in consultation with the woman and her breast physicians, depending on symptoms and their impact on quality of life.

POTENTIAL CONTRAINDICATIONS TO VAGINAL ESTROGEN THERAPY

Although most women with GSM are candidates for low dose vaginal ET, use is contraindicated in women with undiagnosed vaginal/uterine bleeding and should be used with caution in women with estrogen-dependent neoplasia. Management of GSM in women with nonhormone-dependent cancers is similar to that for women without a cancer history.

Low-dose vaginal ET has not been studied in women at increased risk of thrombosis, but may be used with caution given minimal systemic absorption, the absence of a hepatic first-pass effect, and minimal, if any, effect on prothrombotic factors. Of note, in large observational studies, neither vaginal estrogen nor systemic transdermal formulations of ET have been associated with an increased risk of VTE. Although circulating estrogen concentrations generally remain within the menopause range with low-dose vaginal ET, the package insert for these products includes the same boxed warning regarding risk of endometrial cancer, breast cancer, cardiovascular disorders, and probable dementia that accompanies systemic HT products. Women must be educated about the differences between low-dose vaginal and systemic ET and be prepared for the boxed warning, or else they may not initiate prescribed treatment.

Systemic hormone therapy (i.e. MHT) will improve the vasomotor symptoms of menopause and may improve genitourinary symptoms; however, in some women, a vaginal estrogen may also be needed. Individualizing the vaginal estrogen therapy, discussing which preparation the woman would prefer and instructing her in how to use it, will increase the woman's adherence to therapy. The recommended therapy is daily use (at night) for two weeks, then a maintenance dose of two to three times per week. After improvement is noted in the woman's symptoms, it may be possible to reduce the frequency to the lowest effective dose.

SEXUAL DYSFUNCTION

Management of a woman with sexual dysfunction, including loss of libido, dyspareunia due to vulvovaginal atrophy and pelvic floor tension, requires a more complex and multidisciplinary approach. The severity of the symptoms will determine the therapies required. Lubricants and moisturizers may be recommended initially for dryness and loss of lubrication with intercourse. Vaginal estrogens are prescribed when severe atrophic changes are present.

If there is pelvic floor dysfunction, pelvic pain or urinary symptoms, referral to a pelvic floor physiotherapist for pelvic floor training and relaxation will help to reduce symptoms. Sometimes, vaginal trainers will help dilate the vaginal introitus.

Consider changing regular medications that affect sexual function (e.g. antidepressants), and referral to a sexual therapist and/or couple counseling may be necessary.

NEWER OR OTHER TREATMENTS

Newer treatments are becoming available. Vaginal laser therapy is being trialed for vaginal dryness, but long-term data are not available.

A number of preparations that improve atrophic symptoms, but are currently not available in India, are ospemifeme, an oral selective estrogen receptor modulator (SERM), and a vaginal gel of dehydroepiandrosterone (DHEA). Research is ongoing into new and improved vaginal estrogen preparations.

FOLLOW UP

Follow-up visits should be regular to review the woman's response to treatment and her ongoing needs. The frequency will depend on whether the management strategies are successful. Improvement in GSM symptoms typically occurs within a few weeks of starting therapy; however, treatment for 12 weeks may be needed for maximum benefit. In the absence of contraindications, therapy should be continued as long as needed for symptom management as symptoms will recur upon discontinuation. Clinical trial safety data are limited to 1 year, but observational studies demonstrate safety with long-term use. Based on available limited safety data, use of a progestogen and routine endometrial surveillance are not recommended in low-risk women using low-dose vaginal ET. Women at increased risk of endometrial cancer because of obesity or diabetes may warrant endometrial surveillance. Because uterine bleeding is generally a sign of endometrial proliferation, any spotting or bleeding requires a thorough evaluation that may include transvaginal ultrasound (TVU) and/or endometrial biopsy.

CONCLUSION

GSM is a chronic complex syndrome of multiple changes in the genitourinary tissues in response to the loss of estrogen with menopause. The experience of atrophic symptoms varies and ranges from mild to debilitating, with effects on genitourinary function, sexual function, relationships and quality of life. Treatments, ranging from simple measures (egg lubricants, moisturizers) to vaginal estrogen preparations and hormone replacement therapy, are available to reduce symptoms. A multidisciplinary approach may be necessary where there are complex problems, including sexual dysfunction.

The healthcare professional is in a unique position to sensitively discuss symptoms, such as incontinence, sexual pain, prolapse, vaginal irritation and dryness, and to advise, educate and manage accordingly, providing long-term follow-up.

KEY POINTS

GSM is very common and does not improve with time after menopause.

- Symptoms can have a severe impact on quality of life, sexual function and relationships.
- Women are often embarrassed or reluctant to inform healthcare professionals.
- Direct questioning, initially with open-ended questions, is needed.
- Examination of the vulva and vagina is necessary to provide appropriate treatments.
- Non-hormonal and hormonal therapies are available.
- A multidisciplinary team improves outcomes.
- Use the consultation to discuss lifestyle and health.

RESOURCES

- www.menopause.org.au
- https://jeanhailes.org.au/contents/documents/Resources/Booklets/The_vulva.pdf
- https://jeanhailes.org.au/health-a-z/vulva-vagina-ovaries-uterus/vulva-vaginal-irritation
- www.menopause.org

- www.menopause.org/docs/default-source/2013/vva-position-statement.pdf?sfvrsn=0
- www.imsociety.org
- www.dermnetnz.org

REFERENCES

1. Portman DJ, Gass ML, Vulvovaginal Atrophy Terminology Consensus Conference Panel. Genitourinary syndrome of the menopause: New terminology for vulvovaginal atrophy from the International Society for the Study of Women's Sexual Health and The North American Menopause Society. Climacteric 2014;17(5):557−63. Search PubMed
2. Gandhi J, Chen A, Dagur G, et al. Genitourinary syndrome of menopause: An overview of clinical manifestations, pathophysiology, etiology, evaluation, and management. Am J Obstet Gynecol 2016;215(6):704–11. Search PubMed
3. Dennerstein L, Dudley E, Hopper J, Guthrie J, Burger H. A prospective population-based study of menopause symptoms. Obstet Gynecol 2000;96(3):351–58. Search PubMed
4. Nappi RE, Kokot-Kierepa M. Vaginal Health: Insights, views and attitudes (VIVA): Results from an international survey. Climacteric 2012;15(1):36–44. Search PubMed
5. Archer DF, Labrie F, Bouchard C, et al. Treatment of pain at sexual activity (dyspareunia) with intravaginal dehydroepiandrosterone (prasterone). Menopause 2015;22(9):950–63. Search PubMed
6. Nappi, M. Kokot-Kierepa. Women's voices in the menopause: Results from an international survey on vaginal atrophy. Maturitas 2010;67(3): 233–38. Search PubMed
7. Archer DF. Efficacy and tolerability of local estrogen therapy for urogenital atrophy. Menopause 2010;17:194–203 Search PubMed
8. Edwards D, Panay N. Treating vulvovaginal atrophy/genitourinary syndrome of menopause: How important is vaginal lubricant and moisturizer composition? Climacteric 2016;19(2):151–61. Search PubMed
9. Simon J, Nachtigall L, Ulrich LG, Eugster-Hausmann M, Gut R. Endometrial safety of ultra-low-dose estradiol vaginal tablets. Obstet Gynecol 2010;116:876–83. Search PubMed
10. Moegele M, Buchholz S, Seitz S, Ortmann O. Vaginal estrogen therapy in postmenopausal breast cancer patients treated with aromatase inhibitors. Arch Gynecol Obstet 2012; 285(5):1397–402. Search Pub Med

AKI IN PREGNANCY

Dr Neeraj Jain
Associate Professor Department Of Medicine
NSCB Medical College Jablapur

Acute kidney injury (AKI) is an important cause of morbidity and mortality of mother and fetus in pregnancy. Incidence of AKI has decreased in recent past due to better health care in pregnancy but still continue to play an important role in feto-maternal health.

There are physiological changes that occur in the urinary tract system during normal pregnancy like kidneys increase in size by about 1–1.5 cm due to renal vascular and interstitial space volume expansion. The physiological hydronephrosis of pregnancy characterized by a dilation of the calyces, renal pelvis, and ureter occurs in over 90% of pregnant women. This anatomical abnormality may be present until the 16th postpartum week and promotes urinary stasis in the ureter, leading to the development of urinary tract infection. The dilatation of the urinary system is due to the hormonal effects of progesterone, external compression by the gravid uterus, and morphological changes in the ureteral wall. Renal plasma flow can increase up to 85% in the second trimester of pregnancy. .(1) Gfr increases in tune of 40%-50%, so as proteinuria and urinary protein excretion of 300 mg/day is considered physiological in normal pregnancy. Serum bicarbonate may decrease by 3 to 4 mmol/l to compensate for respiratory alkalosis secondary to respiratory stimulation by progesterone.

During the first trimester of gestation, AKI develops most often due to septic abortion or hyperemesis gravidarum. Prevention of unwanted pregnancy and avoidance of septic abortion are keys to eliminate abortion associated AKI in early pregnancy. AKI secondary to hyperemesis gravidarum is rare now days. It is defined as severe and persistent nausea and vomiting, leading to weight loss, exceeding 5% of the pre-pregnancy body weight, and ketonuria. Patients present in the first trimester of pregnancy with AKI associated with hypokalemic metabolic alkalosis. Symptomatic treatment with intravenous fluids and antiemetics usually corrects the renal function. In the third trimester, the differential diagnosis of AKI in association with pregnancy associated syndromes namely preeclampsia/HELLP syndrome, acute fatty liver of pregnancy and thrombotic microangiopathies of pregnancy (TMA), is more challenging. Sepsis also contributes to AKI significantly in postpartum females. Less commonly hypotension secondary to torrential bleed may also lead to AKI. Urinary stasis due to physiological dilatation may predispose the patient for acute pyelonephritis.

BRIEF OVERVIEW OF SPECIFIC DISORDERS

PREECLAMPSIA (PE): PE, a multisystem disorder unique to human pregnancy, is characterized by new-onset hypertension (blood pressure >140/90 mmHg) and proteinuria (>300 mg/dl) after 20 weeks of gestation. The presence of hypertension combined with systemic involvement (such as thrombocytopenia, elevated levels of liver transaminases, renal insufficiency, pulmonary edema, and visual or cerebral disturbances) favors diagnosis of PE in the absence of proteinuria.The incidence is higher in primigravid females and females with certain risk factors such as diabetes mellitus, hypertension, and chronic kidney disease (CKD).

Pathogenesis of PE is not fully understood but involves defects in placentation. placentation requires an extensive angiogenesis for the development of appropriate and adequate vascular network for fetal needs. The placenta produces a wide variety of pro-angiogenic proteins (vascular endothelial growth

factor [VEGF] and placental growth factor [PIGF]) and anti-angiogenic mediators such as soluble fms-like tyrosine kinase 1 (sFlt-1) and soluble endoglin (sEng), balance of both determines the normal placental development. The circulating concentration of antiangiogenic factors sFlt-1 and sEng are increased in PE. It is hypothesized that increased sFlt-1 effectively reduces the concentration/activity of VEGF, resulting in endothelial dysfunction, hypertension, and proteinuria. Glomerular endotheliosis is the histological change seen in the patient with preeclampsia characteristised by swelling and detachment of glomerular endothelial cells. This is often associated with altered hemodynamic abnormalities such as and renal vasoconstriction leading to decreased renal plasma flow and reduction in GFR in tune of 30%–40%. Renal tubular secretion of uric acid is impaired in PE, resulting in elevated concentration of uric acid in the blood. AKI mostly seen in antepartum, but development of AKI in the early postpartum was reported. AKI most often develops in the setting of complication of PE such as placental abruption, disseminated intravascular coagulation (DIC), sepsis, postpartum bleeding, or intrauterine fetal death. Renal cortical necrosis has been reported in preeclamptic women with AKI. (2).

Sometimes,PE can lead to several adverse outcomes on long-term follow-up. They include increased risk for CKD and higher risk of cardiovascular disease (chronic hypertension, ischemic heart disease, and stroke). Treatment is to lower blood pressure (labetalol is most commonly used drug) and delivery of baby in severe cases is the most effective.

HELLP syndrome: Haemolysis, low platelet count and low platelet count (HELLP) syndrome is widely considered a variant of PE and occurs only in pregnant women. The pathogenesis of HELLP syndrome is not fully known. HELLP syndrome seems to be a TMA-like disorder because of several clinical similarities between these two disorders, mechanical hemolysis, thrombocytopenia, and AKI. The link between HELLP syndrome and complement dysregulation has been suspected in recent studies.

HELLP syndrome occurs typically in the third trimester and sometimes in the second trimester or in the postpartum period. The clinical features are variable. The most common symptoms are epigastric/right upper quadrant pain, nausea, vomiting, and headache, but it may manifest by its complications such as DIC, placental abruption, AKI (7%–36%), pulmonary edema, hepatic capsular hematoma, and retinal detachment.

THE DIAGNOSTIC CRITERIA FOR HELLP SYNDROME IS

(a) Microangiopathic hemolytic anemia (MAHA), Schistocytes in peripheral smear, Serum bilirubin ≥1.2 mg/dl, lactate dehydrogenase (LDH) > 600 U/L; (b) Increased liver transaminases (2 times the upper limit of normal) (c) Platelet count < 100 × 103 cells/mm3.

Majority (more than 90%)of patients with HELLP syndrome-related AKI have near complete recovery of renal function, even dialysis dependent AKI has excellent prognosis with complete reversal of renal function in almost all cases. The progression to CKD is reported to occur in < 10 % of patients who developed AKI on preexisting renal disease and/or hypertension. Thus, prognosis of AKI in patients with HELLP syndrome is favorable. (3)

ACUTE FATTY LIVER OF PREGNANCY (AFLP)

AFLP is an emergency that, if not treated, may progresses to fulminant liver failure. It usually affects women in the third trimester of pregnancy and is characterized by sudden onset of acute liver failure with coagulopathy. It is an autosomal recessive disorder leading to defective function of long-chain 3-hydroxyacyl-CoA dehydrogenase causing defective beta oxidation in fetal mitochondria, there is an excessive accumulation of fetal fatty acid, which in turn is released into the maternal circulation. The increased load of long-chain fatty acid is deposited in the liver tissue which leads to impaired hepatic function in the mother. Histological features are lipid microvesicles infiltration of the hepatocytes, without inflammation or necrosis. Symptoms of AFLP are generally nonspecific with malaise, fever, nausea, vomiting, and abdominal pain. With further progression of disease jaundice and hepatic

encephalopathy ensues, the low fibrinogen level, prolonged prothrombin time, depressed antithrombin III levels, and thrombocytopenia are the main laboratory features of disease. Hyperbilirubinemia, increased hepatic transaminases, hypoglycemia, and leukocytosis are the other laboratory abnormalities in patients with AFLP. The close mimic is viral hepatitis.The presence of peripheral edema, thrombocytopenia abd thrombocytopenia favor diagnosis of PE/HELLP syndrome over AFLP. AKI is seen in 20%–100% of cases and etiology of AKI is multifactorial. AKI in patients with AFLP is mostly mild and without need for dialysis support. In most women, there is complete liver and kidney recovery after delivery. Renal insufficiency in AFLP is usually non-oliguric although oliguria and ATN can occur in the setting of hemorrhage-induced hypovolemia. The various factors that may contribute for AKI in AFLP include hypovolemia, coexisting PE, coagulopathy, hepatic failure, and intra-abdominal hemorrhage. Treatment is the urgent delivery of fetus and is associated with favorable maternal and fetal prognosis. (4)

TMA (THROMBOTIC MICROANGIOPATHY)

TMAs in pregnancy is a rare disorder characterized by thrombi of fibrin and/or platelet in the microvasculature of various organs. TMA may occur anytime during pregnancy or at puerperium, in previously healthy women. Depending on the clinical presentation, two clinical forms of syndrome have been described, TTP and HUS. Thrombotic thrombocytopenic purpura (TTP) and hemolytic-uremic syndrome (HUS) may occur de novo with pregnancy, may relapse and/or may recur with subsequent pregnancies. TMA can be classified into three groups based on underlying pathogenic mechanism: (1) complement alternative pathway (CAP) dysregulation related (2) ADAMTS-13 deficiency related, and (3) unknown mechanism. The combination of severe thrombocytopenia, MAHA (microangiopathic haemolytic anaemia), fever and predominant neurologic symptoms (disorientation, ataxia, headache, focal deficits, seizures) are the presenting features of TTP. The treatment of TTP includes fresh frozen plasma infusion and plasmapheresis to correct ADAMTS-13 deficiency and remove the ADAMTS-13 circulating antibodies. In suspected case of autoimmunethe regulatory protein, steroids are recommended as initial immunosuppressive therapy. The B-cell depleting antibody rituximab may be considered as second-line therapy for cases not showing adequate response to plasma exchange or fresh frozen plasma (FFP) infusion. Only mild renal dysfunction occurs in such patients.

Hemolytic-uremic syndrome (HUS): HUS- TMA associated with complement activation pathway (CAP) dysregulation clinically presents as atypical HUS (aHUS). Clinical features of aHUS are similar to TTP, but renal involvement is more severe and the neurological manifestation is rare. aHUS occurs mainly in the first 6 months postpartum. Pregnancy and delivery are usually uneventful in majority of the patients. TMA caused by CAP dysregulation occurred mainly (80% of cases) during the postpartum period. Infections and bleeding, which frequently complicate the postpartum period, may trigger complement activation leading to TMA (aHUS). Eighty percent of Pregnancy associated Hemolytic uremic syndrome (P-aHUS) had CAP dysregulation with factor H (FH) and factor (FI) encoding gene (CFH and CFI, respectively) mutation, and majority of patients (80%) reaches end-stage renal disease during the first aHUS episode. Treatment of aHUS also includes plasma infusion and plasma exchange to restore the missing mediators (factor H and I) and remove anti-factor H. The criteria use to define aHUS resistant to plasma exchange therapy include (i) the absence of platelet count increase (to > 1 lac) (ii) absence of LDH level decrease, and (iii) absence of significant decrease (>25%) of serum creatinine, despite 3–5 plasma exchange. The use of potent inhibitor of CAP activation is indicated for cases resistant to plasma therapy. The optimal first line of the current treatment for patients with aHUS is eculizumab (monoclonal humanized IgG antibody) regardless of evidence of underlying CAP dysregulation. Eculizumab (very costly, not usually available) is a powerful inhibitor of C5 and thus prevents the generation of C5a and C5b.(5)

TREATMENT OF AKI IN PREGNANCY IN GENERAL

Important general measures to minimize renal injury (such as treatment of etiology, avoidance of

nephrotoxic drugs or treatment of an infectious disease) should be started as soon as possible.Second step is administration of intravenous fluids (if patient is not in CCF/fluid overload state) to restore or maintain renal perfusion. Pharmacological therapy of AKI and its known complications such as hypertension, hyperkalemia, metabolic acidosis, and anemia.

Dialysis : The indications for dialysis in AKI are similar to the general population, and they include uremic symptoms (encephalopathy, pericarditis, or neuropathy), volume overload, hyperkalemia, and/or metabolic acidosis unresponsive to initial medical treatment. It is essential to keep certain aspects in mind when dialysis is prescribed in pregnant women with AKI. They include (i) increased dialysis dose (daily dialysis with duration of more than 20 h/week if pregnancy is continued). This will improve the uremic milieu, a high risk factor for pregnancy related complications like prematurity and polyhydramnios, . It is advised to maintain certain biochemical parameters in narrow range in pregnant women, such as bicarbonate to 22- 25 mEq/L , sodium to 135-145 mEq/L and urea < 100 mg%. Both conventional and low molecular weight heparin are safe to use in pregnancy during hemodialysis treatment because they do not cross the placental barrier.

CONCLUSION

The management of AKI in pregnancy is a major clinical challenge because it poses a risk to mother and fetus. It is mostly a preventable complication in pregnancy if basic cause are addressed in time. Pregnancy hypertensive complications, notably PE/HELLP syndrome, are the leading cause of AKI worldwide. In underdeveloped and developing nation puerperal sepsis and obstetrical hemorrhage (APH and PPH) still account for majority of AKI in the peripartum period in addition to PE/HELLP syndrome. Any use of indigenous drugs (may be nephrotoxic) should always be considered in unexplained AKI and obstructive uroapthy with ultrasound should always be ruled out. In case of no recovery patient may be considered for contrast CT-KUB to rule out acute cortical necrosis and renal biopsy to know the histologic changes guiding the path to the specific renal issues. Fortunately, overall, incidence of AKI has probably decreased in our country with improved maternal and fetal outcome.

REFERENCES

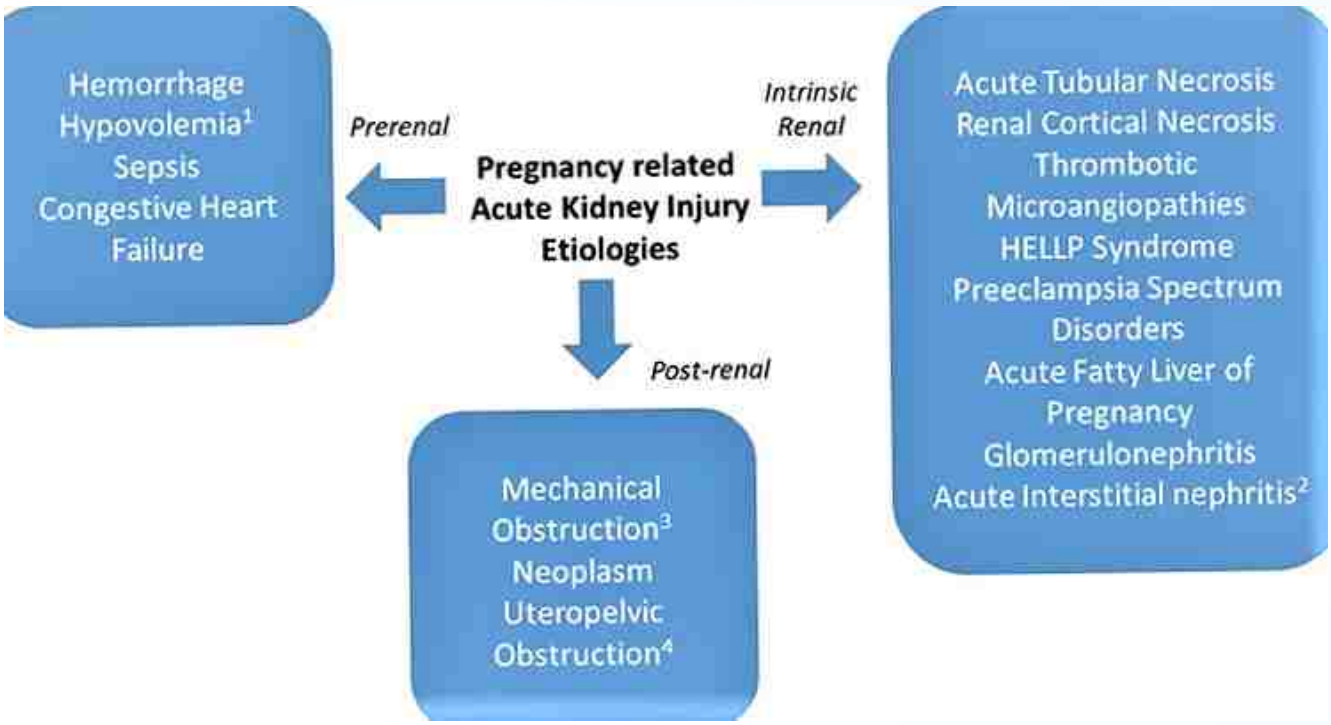
(1)Rasmussen PE, Nielsen FR. Hydronephrosis during pregnancy: A literature survey. Eur J Obstet Gynecol Reprod Biol. 1988;27:249–59. [PubMed]

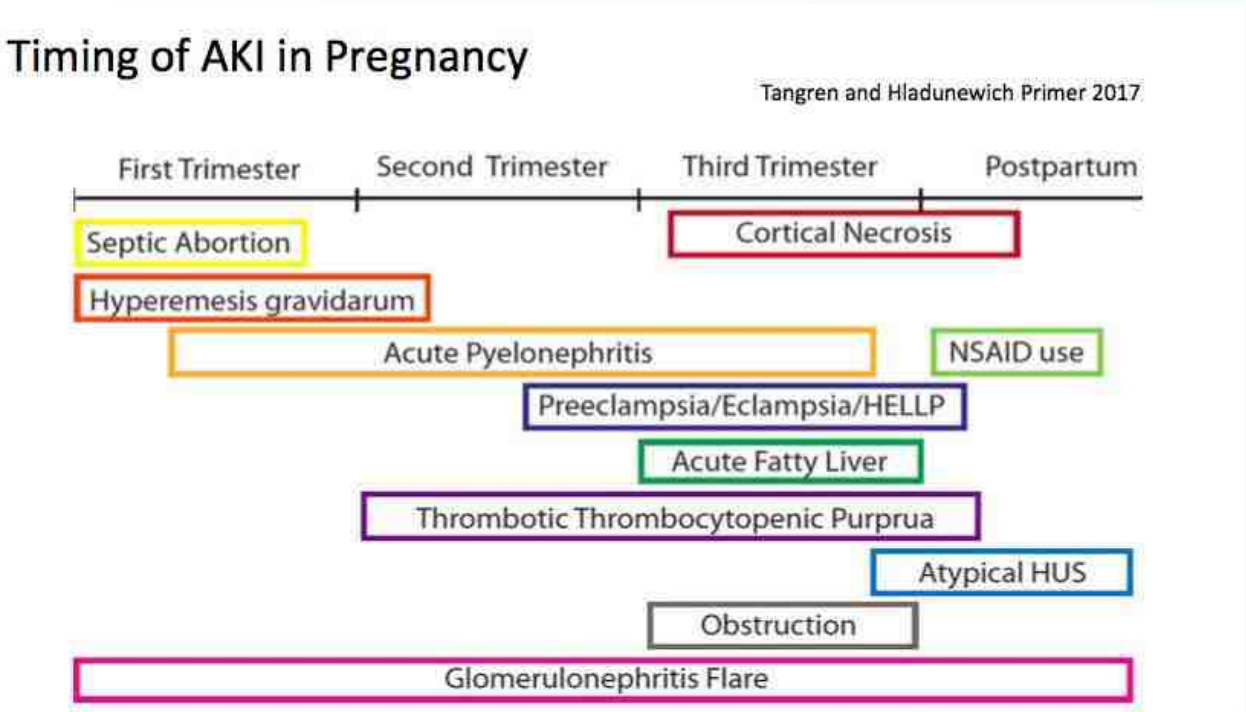
(2) American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol. 2013;122:1122–31. [PubMed]

(3) Abraham KA, Kennelly M, Dorman AM, Walshe JJ. Pathogenesis of acute renal failure associated with the HELLP syndrome: A case report and review of the literature. Eur J Obstet Gynecol Reprod Biol. 2003;108:99–102. [PubMed

(4) Guntupalli SR, Steingrub J. Hepatic disease and pregnancy: An overview of diagnosis and management. Crit Care Med. 2005;33(10 Suppl):S332–9. [PubMed]

(5) Fakhouri F, Roumenina L, Provot F, Sallée M, Caillard S, Couzi L, et al. Pregnancy-associated hemolytic uremic syndrome revisited in the era of complement gene mutations. J Am Soc Nephrol. 2010;21:859–67. [PMC free article] [PubMed]


Hemorrhage
Hypovolemia[1]
Sepsis
Congestive Heart Failure
Prerenal
Pregnancy related Acute Kidney Injury Etiologies
Intrinsic Renal
Acute Tubular Necrosis
Renal Cortical Necrosis
Thrombotic Microangiopathies
HELLP Syndrome
Preeclampsia Spectrum Disorders
Acute Fatty Liver of Pregnancy
Glomerulonephritis
Acute Interstitial nephritis[2]
Post-renal
Mechanical Obstruction[3]
Neoplasm
Uteropelvic Obstruction[4]


Timing of AKI in Pregnancy
Tangren and Hladunewich Primer 2017
First Trimester
Second Trimester
Third Trimester
Postpartum
Septic Abortion
Cortical Necrosis
Hyperemesis gravidarum
Acute Pyelonephritis
NSAID use
Preeclampsia/Eclampsia/HELLP
Acute Fatty Liver
Thrombotic Thrombocytopenic Purprua
Atypical HUS
Obstruction
Glomerulonephritis Flare

OVERACTIVE BLADDER REVIEW OF EVALUATION AND MANAGEMENT

Dr Anurag Dubey, Dr Fanindra Solanki
Assistant Professor
Department of Urology Superspeciality Hospital
NSCB medical college Jabalpur

ABSTRACT

Overactive bladder (OAB) syndrome is bothersome condition which affects quality of life of significant amount of the population. Estimated prevalence is 16.5% of OAB. Many general practitioner and even gynaecologists are not familiar with this condition. Aim of our article is to review important aspect of this bothersome condition and suggest tools for assessment and management strategies.

INTRODUCTION

Overactive bladder (OAB) syndrome is a chronic condition which is having poor negative effect over quality of life [1]. OAB affects performance of daily activities and social activities like work, traveling, physical exercise, sleep, and sexual function. The definition of OAB updated in 2010 by the International Continence Society is: A condition with characteristic symptoms of "urinary urgency, usually accompanied by frequency and nocturia, with or without urgency incontinence, in the absence of urinary tract infection or other obvious pathology" [2].

Urgency is main feature which leads us to diagnose overactive bladder. Other features associated to this disease are frequency of urination in day time and urge incontinence. Nocturia is reported as the most bothersome symptom [3]. Nocturia was found to be directly related to decreased sleep quality, decreased health-related quality of life, and depression in the elderly population [3].

Only a third of OAB patients show urge incontinence also called wet OAB. Some patients may have both OAB and urinary stress incontinence symptoms and are diagnosed as having mixed urinary symptoms. [1, 5]

To diagnose OAB there should no urinary tract infection, any disorder affecting urine output like diabetes mellitus, diabetes insipidus, any metabolic disorders or stress incontinence. Female gender is associated with a higher prevalence of OAB, particularly in younger people. A small influence of racial factors is also present; for men, prevalence among African-Americans is 20%, Hispanics 18%, and whites 15%, with the figures for women being 32%, 29%, and 29%, respectively [4]

OAB can occur in any age group but it is over all more common at middle age .[6]. As being a bothersome condition usually patient seeks attention early but is challenging since risk factors unknown most of time idiopathic. This review addresses various aspects of diagnosis and clinical management of the OAB syndrome.

PATHOPHYSIOLOGY OF THE OAB SYNDROME

Various factors may be involved in OAB and the major cause may vary from individual to individual. The etiology of OAB is still under investigation and is not well understood. However, 4 theories have been proposed to explain the pathophysiology of OAB:

1. The neurogenic theory: reduction in the inhibitory neural impulses and increase in the afferent impulses from the bladder trigger the voiding reflex [7].
2. The myogenic theory: the detrusor muscle becomes more sensitive to cholinergic stimulation leading to increased spontaneous activity [8].
3. The autonomous bladder theory: alteration or exacerbation of phasic activity is generated by

muscarinic stimulation [9].

4. Integrative hypothesis: A range of triggers can generate localized detrusor contractions, which can spread in the bladder wall through various routes of propagation. Consequently, urgency is a result of distortions in the bladder wall, and it is associated with detrusor overactivity if the contractions spread to a sufficient proportion of the bladder wall [9]

All these theories attempt to explain what is referred to as “detrusor overactivity”. The micturition reflex is activated when the detrusor muscle is stretched, while control of the bladder is achieved through a complex of interactions between the central and the peripheral nervous systems. OAB syndrome's pathological conditions affect the bladder's sensory pathway and contribute to the urge to urinate at a low bladder volume.

A pathological partial denervation of the detrusor may induce muscle contractions leading to an urgency sensation and possible urge of urinary incontinence. Anatomically and functionally the detrusor muscle phasic activity is also controlled by the autonomous nervous system and any imbalance in the excitation or inhibition of smooth muscle modulators may also result in detrusor overactivity [10]

EVALUATION OF PATIENTS WITH THE OAB SYNDROME

In OAB signs are usually absent so careful history utmost important. History to rule out common urological conditions which can cause bladder irritation like UTI, bladder stone , bladder pain syndrome, bladder tumours are important. Systemic conditions that can cause bladder symptoms like diabetes mellitus, diabetes insipidus, spinal cord injury, Parkinson's disease, multiple sclerosis, pelvic surgery, dementia, and psychiatric disease.

Table 1

Assessment for OAB

Key topics	Keynotes to follow and comments
Patient characteristics	gender, age, presenting symptoms, frequency, better, worse, impediments to life style, voiding diary
Current drugs taken	diuretics aggravate symptoms, alpha-agonists may lead to overflow incontinence
Past medical history	heart failure, poorly controlled diabetes, strokes, neurological diseases
Previous surgeries	transurethral resection, colposuspention, midurethral slings
Physical examination	general, gynecological, neurological
Laboratory and urology tests	blood test for HbA1c, creatinine levels, urine analysis and culture of residual urine and flowmetry, urodynamics

Certain medications may contribute to urinary symptoms through the following mechanisms:

neuroleptics, benzodiazepines, α-adrenergic blockers usually by bladder neck and urethra dialatation

diuretics by producing excess urine β-blockers, anti-Parkinson agents by relaxing bladder lithium cause excess fluid retention

Radiotherapy for pelvic organ cancer can also lead to radiation cystitis, leading to decreased bladder compliance and capacity.

Thorough pelvic and gynaecological examination is required to rule out cystocele and rectocele. These conditions can mask incontinence specially stress urinary incontinence.

A bladder diary is simple and important tool which gives a lots of information about drinking habit and voiding pattern of patient.[16].Bladder diary should be filled for three days at least.

Routine urological investigations like urine analysis, urinary culture, complete blood count and renal

function test should done to rule out diseases causing bladder symptoms.

Ultrasound abdomen and pelvis is very useful and handy tool to diagnose various diseases associated to OAB. Special concern should be over post void residual urine.

Uroflowmetry gives objective assessment of voiding pattern; normal values are maximum urinary flow should greater than 15 ml/s, with at least 150 ml voided. Values of < 150 ml may not accurately reflect the patient's true maximum flow [11, 12]

MANAGEMENT STRATEGIES

True treatment of OAB includes non-pharmacological, pharmacological and interventional category

NON-PHARMACOLOGICAL TREATMENT

Non pharmacological treatment mainly involves life style changes which are mean to increase understanding about disease and help them to manage urge and urge incontinence. Life style changes mainly include cessation of smoking, weight reduction, reduce intake of caffeine, acidic foods, and alcohol, bowel regulation, and exercise are helpful[13, 14].

Double and triple voiding is helpful for bladder retraining. Initially the voiding intervals may be as short as 30 minutes and training may bring a gradual increase of voiding intervals. This procedure may lead to a slow increase of bladder capacity.

Pelvic floor muscle training (with or without biofeedback) is a treatment aimed at reducing detrusor contractions through inhibitory reflexation of the pelvic floor, thus reducing episodes of urgency and urge incontinence [15]. Behavioral therapy was found to be most effective when combined with oral drug therapy [16]. Table Table22 summaries the options for non-pharmacological treatment.

Table 2

Non-pharmacological treatment of OAB

Classfication	Treatment
Life style changes	weight loss and exercise dietary and fluid intake changes (restriction of fluids) bowel regulation cessation of smoking bladder training (habit-training schedules)
Pelvic floor exercise	Kegel exercises
	vaginal weight training
	pelvic floor exercise with biofeedback
	pelvic-floor electrical stimulation

PHARMACOLOGICAL TREATMENT

Previously main drug treatment for OAB was anticholinergic drugs. Main effect of these drugs are relaxation of the bladder muscle and consequently to improve patient's symptoms. This drug family improves the OAB symptoms by 2 mechanisms. The first mechanism of action works at the level of the neuromuscular junction on cholinergic-muscarinic receptors producing a competitive inhibition of the process through which parasympathetic stimulation leads to detrusor muscle contractions. In addition, a second mechanism of action may work on urothelial sensory receptors inhibiting afferent nerve activity. Several anticholinergic drugs used today are available and prescribed worldwide and are recommended by the International Consultation on Incontinence [17].

Second drug which got much interest is β adrenergic drug (Mirabegron). It has shown enough benefit in OAB. Mirabegron is a first-in-class β3-adrenoceptor agonist licensed for the treatment of OAB and has shown to be well tolerated and effective in the treatment of OAB symptoms. Mirabegron 50 mg had similar efficacy to most antimuscarinics with a lower incidence of dry mouth and constipation.

MANAGEMENT OF RESILIENT OAB

Once one or two bladder relaxant and combinations have been tried and patients are not responding then better to refer to patients to urologist or urogynaecologist. After pharmacological treatments fails next treatment strategy involves botulinum toxin injection, posterior tibial nerve neuromodulation, and sacral neuromodulation.

Botulin toxin, comes as name Botox is used by direct cystoscopic multiple injections of the detrusor muscle. It mainly acts by blocking presynaptic release of acetylcholine from nerve endings. Which ultimately result in decrease contractility, and muscular atrophy. This can be done OPD bases in local anaesthesia [18]. In 2013 the FDA expanded the approved use of Botox (onabotulinumtoxin A) to treat adults with OAB who cannot use or do not adequately respond to anticholinergics [19].

Another approach to resilient OAB is the use of neuromodulation to regulate bladder and pelvic floor function. There is peripheral tibial nerve stimulation and sacral neuromodulation.

An external electrical signal is sent through the tibial nerve retrograde to the sacral plexus, through a small needle inserted into the lower leg near the ankle[20]. This treatment which consists of repeated 30 minutes sessions for 3 months was associated with no serious adverse effects.

In more severe cases, S3 nerve root stimulation by an implanted electrical pulse generator can be used. Surgical implantation of a pulse generator is performed with electrical probes lying in close proximity to the nerves and provides continuous stimulation [21]

In refractory cases, augmentation cystoplasty can be done as an last resort. [22]. However, this procedure is requires long term followup , bowel resection, electrolyte imbalance, patient may require long term catheterisation and clean intermittent catheterisation.

Among other investigational therapies, neurokinin receptor antagonists, alpha-adrenoceptor antagonists, nerve growth factor inhibitors, gene therapy, and stem cell-based therapies are of considerable interest. The future development of new modalities in OAB treatment appears promising [23,24,25,26].

CONCLUSION

There is a significant influence of OAB on health-related quality of life. The importance of diagnosis and proper treatment cannot be overemphasized, especially in elderly patients. Practitioners can easily overlook urinary complains if they not directly queried. We would like to encourage practitioners to give more attention to this issue. In our opinion, familiarity with this condition and basic knowledge about the diagnosis and treatment options can contribute to the general health.

REFERENCES

1. Stewart WF, Van Rooyen JB, Cundiff GW, Abrams P, Herzog AR, Corey R, Hunt TL, Wein AJ. Prevalence and burden of over-active bladder in the United States. World J Urol. 2003;20:327–336. [PubMed] [Google Scholar]
2. Haylen BT, de Ridder D, Freeman RM, Swift SE, Berghmans B, Lee J, Monga A, Petri E, Rizk DE, Sand PK, Schaer GN. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2010;29:4–20. [PubMed] [Google Scholar]
3. Van Dijk MM, Wijkstra H, Debruyne FM, De La Rosette JJ, Michel MC. The role of nocturia in the quality of life of men with lower urinary tract symptoms. BJU Int. 2010;105:1141–1146. [PubMed] [Google Scholar]
4. 4Middelkoop HA, Smilde-van den Doel DA, Neven AK, Kamphuisen HA, Springer CP. Subjective sleep characteristics of 1,485 males and females aged 50–93: effects of sex and age, and factors related to self-evaluated quality of sleep. J Gerontol A Biol Sci Med Sci. 1996;51:M108–115. [PubMed] [Google Scholar]

5. Marinkovic SP, Rovner ES, Moldwin RM, Stanton SL, Gillen LM, Marinkovic CM. The management of overactive bladder syndrome. BMJ. 2012;344:e2365. [PubMed] [Google Scholar]
6. Milsom I, Abrams P, Cardozo L, Roberts RG, Thuroff J, Wein AJ. How widespread are the symptoms of an overactive bladder and how are they managed? A population-based prevalence study. BJU Int. 2001;87:760–766. [PubMed] [Google Scholar]
9. de Groat WC. A neurologic basis for the overactive bladder. Urology. 1997;50((6A suppl)):36–52. [PubMed] [Google Scholar]
10. Wein AJ, Rackley RR. Overactive bladder: a better understanding of pathophysiology, diagnosis and management. J Urol. 2006;175:S5–10. [PubMed] [Google Scholar]
11. Haylen BT, Ashby D, Sutherst JR, Frazer MI, West CR. Maximum and average urine flow rates in normal male and female. populations – the Liverpool nomograms. Br J Urol. 1989;64:30–38. [PubMed] [Google Scholar]
12. Al Afraa T, Mahfouz W, Campeau L, Corcos J. Normal lower urinary tract assessment in women: I. Uroflowmetry and post-void residual, pad tests, and bladder diaries. Int Urogynecol J. 2012;23:681–685. [PubMed] [Google Scholar]
13. Hashim H, Abrams P. How should patients with an overactive bladder manipulate their fluid intake? BJU Int. 2008;102:62–66. [PubMed] [Google Scholar]
14. Lohsiriwat S, Hirunsai M, Chaiyaprasithi B. Effect of caffeine on bladder function in patients with overactive bladder symptoms. Urol Ann. 2011;3:14–18. [PMC free article] [PubMed] [Google Scholar]

COSMETIC GYNECOLOGY
The FUTURE of Women's Health

Dr. Richa Dhirawani, Dr Amita Saxena
Consultant
Jabalpur Cosmetic Gynecology Centre

INTRODUCTION

"Childbearing, trauma, aging, and inherited irregularities affect not only the aesthetic look of the external female organs but also create feelings of being different or inadequate. It can affect intimate relationships and women's emotions."

- Dr. Adam Ostrzenswski

Cosmetic Gynecology may seem like an alien term to you but in this article, we will walk you through the basics of cosmetic gynecology and why you may need it. Without further ado, let's begin.

Cosmetic Gynecology can be used to increase the way the female genital organs look or function. Sometimes, both goals are achieved simultaneously. After this procedure, the body appears younger, sexual intercourse becomes pleasurable, and it also adds to the aesthetic appeal of your body. It repairs the vagina and labia that may have degraded due to aging or childbirth.

Cosmetic Gynecology was discovered rather unexpectedly on a Mediterranean cruise in 1973 when a Belgian, Italian, and French dermatologist met on a holiday. It was during this time that these genius minds discussed and dwelled upon the concept of "aesthetic gynecology" and came up with cosmetic gynecology.

FEMININE INDICATIONS

- **Sexual Dysfunction -** Persistent, recurrent problems with sexual response, desire, orgasm or pain that distress you or strain your relationship with your partner are known medically as sexual dysfunction
- **Urinary incontinence -** Urinary incontinence is usually caused by problems with the muscles and nerves that help the bladder hold or pass urine.
- **Vaginal relaxation syndrome -** As women age, relaxing of the vaginal wall can lead to vaginal relaxation syndrome (VRS), which is exacerbated by childbirth, especially multiple pregnancies and deliveries, and the vaginal atrophy associated with menopause-related hormonal changes.
- **GSM - Genitourinary** syndrome of menopause (GSM) is the new term for vulvovaginal atrophy (VVA). Estrogen deficiency symptoms in the genitourinary tract are bothersome in more than 50% of women, having an adverse impact on quality of life, social activity and sexual relationships.
- **Recurrent vulvovaginitis -** 2 Vulvovaginal candidiasis is considered recurrent when at least four discrete episodes occur in one year or at least three episodes occur inoneyear and are not related to antibiotic therapy. Recurrent vulvovaginal candidiasis is distinguished from persistent infection by the presence of a symptom-free interval.
- **Pelvic organ prolapse -** Pelvic organ prolapse (POP), the herniation of the pelvic organs to or beyond the vaginal walls, occurs in up to 50 percent of parous women and causes a variety of pelvic, urinary, bowel, and sexual symptoms
- **Vulvodynia -** Vulvodynia is chronic pain in the vulva, the area on the outside of a woman's

genitals. It is usually described as a sensation of burning, stinging, itching or rawness. It is defined as pain that lasts more than three months and doesn't have a clear identifiable cause, such as an infection or a skin disorder.

- **Stretch Marks -** A stretch mark is **a** type of scar that develops when our skin stretches or shrinks quickly. The abrupt change causes the collagen and elastin, which support our skin, to rupture. As the skin heals, stretch marks may appear. Not everyone develops these narrow bands on their skin.
- **Saggy Breasts -** Ligaments in your breasts, called Cooper's ligaments, lift and support your breasts. Over time, these ligaments can stretch out and cause the breasts to sag. Loose skin or the loss of skin's elasticity can also lead to droopy, deflated breasts.
- **Hyperpigmented Genitals -** The private parts turning dark due to various reasons including friction etc.

VAGINAL LASER TREATMENT

There may be a lot of confusion surrounding the topic of cosmetic surgery. We will list down all the procedures that one can undergo. If you feel relevant with any of the options, you can undergo that procedure. There are various surgical procedures involved in cosmetic gynecology.

Different energy based devices are used for vaginal rejuvenation; they make vaginal tissue functionally better.

FDA cleared the first cosmetic laser to reduce unwanted hair and wrinkles in 1990.Today medical Aesthetic has grown to include more than just lasers ,infrared light,RF energy and ultrasound technology and different lasers is being used to reduce the appearance to cellulite, improve skin texture and tighten sagging skin addressing many functional disorders like; **SUI**,**GSM**, not only this aesthetic treatment are done with injectable also along with EBDs,**BOTOX**, **DERMAL FILLERS**,**FAT**,**PRP**(Platelet-rich Plasma) ,and stem cells are being used to address vulvo vaginal rejuvenation.

Today's women need to address her intimate health problems. New technologies like LASERS **"AN AESTHETIC UPLIFT CAN LEAD TO A PSYCHOLOGICAL UPLIFT ".**Different Lasers,**RF**(Radio Frequency) ,**HIFU**(High-intensity focused Ultrasound) and adjuvant regenerative therapies has been introduced in the field of vaginal cosmetology to treat disorders like **SUI** (Stress Urinary Incontinence) ,**GSM** (Genitourinary syndrome of menopause) ,mild prolapse and medical conditions like vulvodynia , Lichen sclerosis and vaginismus.

OTHER PROCEDURES OF COSMETIC GYNECOLOGY

- **Vaginoplasty**

  Vaginoplasty is one of the most popular cosmetic surgeries in the world. It is used to reduce the width of the vagina. The permanent stretching of the vagina can degrade the sexual experience in females. It can be caused due to multiple childbirths.

  With this procedure, the damaged tissues of the vaginal cavity are reconstructed and repaired to restore the elasticity of the vagina. The vagina becomes tighter which results in enhanced coitus experience later.

- **Labiaplasty**

  A vagina consists of two types of labial skin: the upper lips and inner lips. The variation of the size in these two tissues may vary for females globally.

  Sometimes it may not be in symmetry or there may be the presence of excess skin on both sides of the labia. Due to these difficulties, females may find it difficult to enjoy their intimacy. They also feel discomfort in wearing tight-fitting pants or shorts. The excess skin can be removed by laser treatment. This builds confidence, comfort aids, and increases sexual pleasure.

- **Hymenoplasty**

  With hymenoplasty, the cosmetic surgeon restores the hymen of your vagina that was broken during sexual intercourse, horseback riding, cycling, use of tampons, or birth defects. This is a controversial procedure due to the cultural and social beliefs associated with the concept of purification. Many cultures view virginity as a sign of purity in women.

  However, it can be performed on anyone who has lost their hymen due to other causes. The hymen already has an opening that allows menstrual blood to flow through it. It widens after coitus or other activities.

- **Hoodectomy or Clitorial Unhooding**

  Through this procedure, you can get rid of the excess skin that surrounds your clitoris. Clitoris is said to be the most sexually aroused organ in a female body and it is often compared to a penis in terms of that. Thus, it is clear that the clitoris plays a major role in sexual arousal and pleasure in females.

  Moreover, women with a larger clitoral hood will find it extremely uncomfortable to wear snug or tight clothes as the constant rubbing may induce pain down there.

- **Monsplasty**

  This procedure helps in removing the excess fatty tissues that surround the mons pubis. Due to the presence of excess fat, the bulge in the tissues may cause embarrassment and discomfort in wearing tight clothes, revealing dresses, or a swimsuit.

WHY IS COSMETIC GYNECOLOGY IMPORTANT?

With the enhancement in technology around the world, there is a remarkable surge in cosmetic surgeries globally. A specific reason has not been found for this surge but with the growing love for social media, more and more women want to look and feel good around the camera and even behind it. There's also a special focus on athleticism that also drives the cosmetic gynecology industry.

The sociological standard of beauty has driven more women towards cosmetic gynecology now. Cosmetic gynecology helps in empowering women to make them feel comfortable in their skin and enhance their sexual life as well. It also builds their confidence and sense of self-worth so that they can face the world without any regrets or embarrassment.

More and more women are now being vocal about their desires and needs. In a study of 2008; it was found that 32% of women underwent this surgery to repair a functional impairment, 31% for functional impairment as well as aesthetic reasons, and 37% for aesthetic appeal only. Overall, this procedure helps in providing a solution to physiological or genetic changes that have affected a woman negatively.

Cosmetic Gynecology is the need of the hour. It has bridged the gap between cosmetic surgery and gynecology and opened new doors for women all around the globe.

CONCLUSION

People must understand that sexual satisfaction in life also enhances the quality of life. Everyone, including women, has the right to do things that make them happy. To be able to love their body is a big step towards body positivity and cosmetic gynecology helps establish the same.

EDITORS CORNER

Prof Dr Gita Guin
Professor, Dept of Obs & Gynae,
Govt. NSCB Medical College Jabalpur (MP)
East Zone Coordinator, Ethics and
Medico-legal Committee, FOGSI

Editor

Co-Editor

When it comes to treatment options infield of urogynecology we have very limited options and sometime symptoms continue to recur in spite of best possible treatment offered. When treating these patients as a doctor we have to face two sides of sword, we have to decrease patient's sufferings and along with it we have to save ourselves from medico legal implications also.

We had so many questions in our mind, so, also we had best person to answer, our medico legal expert DR GEETA GUIN MADAM. Here is what madam has to say

Majority medicolegal issues in genito-urinary syndromes relate to their

1. Chronicity: The urogenital changes around the menopausal years makes these problems long standing. Often, patient satisfaction is difficult to achieve and recurrence is a challenge. Under such circumstances, the mainstay to not only improve patient satisfaction but also to avoid medicolegal issues is OPTIMAL COUNSELLING and ADEQUATE AND LOGICAL DOCUMENTATION, with consent where ever needed.

To say optimal counselling means, it should be a good and prudent balance of what best can be achieved in the background of available resources and physician competence as well as patients' socioeconomic status. Whenever feasible, every effort should be made to refer a willing patient to where ever any better treatment is possible. Special care should be taken to not make any false promises and godly claims. These diseases have their own problems of treatment and the patient should be well aware and consent for same.

2. Limitations of treatment success which essentially depends on the nature of the problem, available treatments, whether drugs or interventional, available expertise and available resources and patients willingness for the same.

Obviously, the need for an indemnity policy and valid consent is undisputed and paramount.

बेघर पंछी

Dr Alka Agrawal
Director
Vimal Nursing Home

सुबह कुछ जल्दी ही जाग गया,
टक – टक की आवाज़ आ रही थी द्य
दरवाज़ा खोलकर देखा
कोई दिखा नहीं ! .....
आवाज़ की दिशा में देखा
खिड़की पर दिखा एक पक्षी
अपनी चोंच से खिड़की पर
उकेर रहा था नक्काशी .....

मैंने पूछा –
भाई क्या बात है
बोला ष क्या किराये से मिलेगा
कोई पेड़ घोंसला बनाने के लिए ?

अकेला तो कहीं भी रह लेता,
परन्तु घर चाहिए चूज़ों के लिए

तुम्हारे ही भाइयों ने लूट लिया है,
हमारा जंगल पूरा ही काट दिया है
बेघर तो कर ही दिया है,
दाने–पानी के लिए भी तरसा दिया है

पुण्य कमाने के लिए रख देते हैं ,
छत पर थोड़ा दाना थोड़ा पानी,
पर रहने के लिए छत भी तो चाहिए
यह तो कोई सोचता भी नहीं

पेट तो भरना ही है,
भीख ही सही, थोड़ा खा–पी लेते हैं
थोड़ा घर भी ले जाते हैं
भले ही सर पर छत न हो
पेट में भूख तो है ना?

कभी –कभी सोचता हूँ
आत्मघात कर लूँ
बिजली के तारों पर बैठ जाऊँ ,
या पटक दूँ सर मोबाइल के ऊँचे टावर पर

जैसे सरकार दे देती है कुछ
फाँसी लगाने वाले किसान को
वैसे ही मिल जाएगा कोई पेड़
मेरे चूज़ों के घोंसले के लिए
सुनकर मैं सुन्न हो गया
इतना कुछ तो सोचा न था ?
जंगल काटकर घर उजाड़ दिया
इन बेचारों का विचार नहीं किया !
मैंने हाथ जोड़कर उससे कहा
सबकी ओर से मैं माफी माँगता हूँ,

आत्मघात का विचार त्याग दो, यह दिल से निवेदन है मेरा
अभी तो इस गमले के पौधे पर
अपना वन रूम किचन का घर बसा लो
थोड़ी अड़चन तो होगी परंतु अभी इसी से काम चला लो
उसने कहा
बड़ा उपकार होगा
परंतु किराया क्या होगा ?
और कैसे चुकाऊँगा
मैंने कहा–
तीनों पहर मंगल कलरव सुनूँगा, और कुछ नही माँगूँगा
वह बोला मुझे तो आप मिल गए पर मेरे भाई बंधुओ का क्या?
उन्हें भी तो घर चाहिए ,कहाँ रहेंगे वे सब ?
मैंने कहा अरे ! अब लोग जाग रहे हैं,
बड़, पीपल, नीम, गूलर रोप रहे हैं
धीरे –धीरे बदलाव आ रहा है

किसी को आत्मघात करने की आवश्यकता अब नहीं है
सुनकर पक्षी उड़ गया,
घोंसले का सामान लाने के लिए
और मैंने मोबाइल उठाया
आपको बताने के लिए
पक्षी की टक –टक से
मेरे मन का द्वार खुल गया
आप भी एक पेड तो रोपेंगे !
आंगन में... या गमले में ही सही.....

Save Girl Child Da & Awareness Programme

Breastfeeding Awareness











JOGS Installation Ceremon












Anaemia Awareness at Indrana PHC B Dr Shaline Agrawal

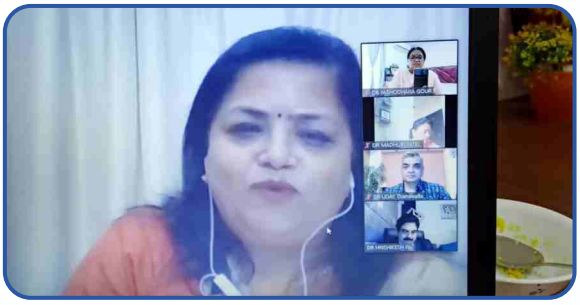
Covid Vaccination for Pregnant women at Ranjhi Civil Hospital Jabalpur

FOGSI FRIENDSHIP DAY

Cancer Awareness & Health Checkup

Rangoli Slogan

Onco fogsi committe



Webinar on Fetal Medicine

RTM on Newer Conception Oral Contraceptive

CME ON PRETERM LABOUR & RPL