AHMEDABAD OBSTETRICS AND GYNAECOLOGICAL SOCIETY

AOGS TIMES

2021-2022

OCTOBER 2021 VOLUME 7

THEME : IMPLEMENTATION OF EVIDENCE BASED CLINICAL CARE

MOTTO : SWEAT, SMILE & REPEAT

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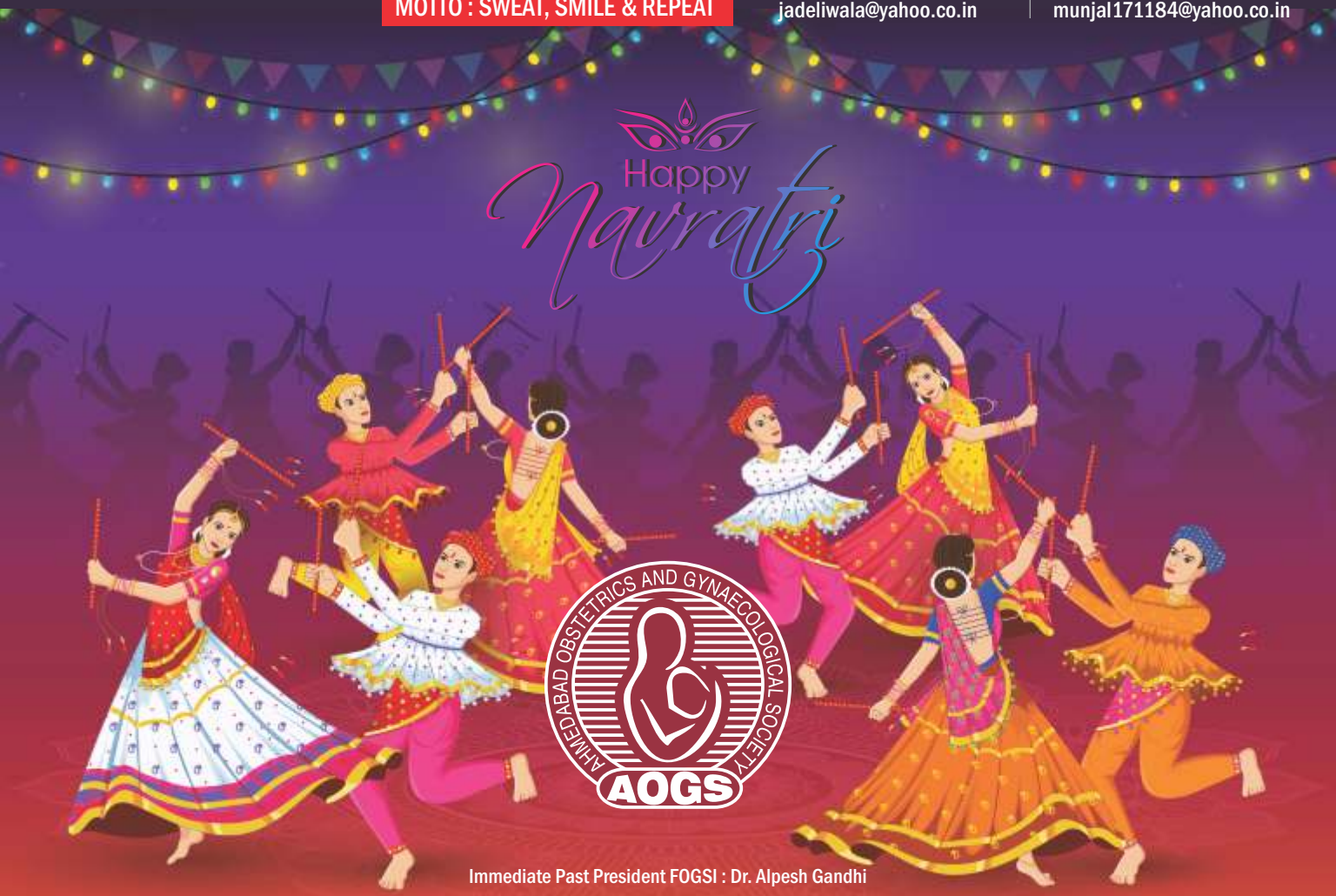

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TEAM AOGS MESSAGE

Dr. Jignesh Deliwala
President

Dr. Munjal Pandya
Hon. Secretary

નવરાત્રી- આસો મહિનામાં ઉજવાતો, માતાજીનો મહિષાસુર પરનો વિજય;
અનિષ્ટ પર સારાની જીત.
નવ દિવસ અને નવ રાત્રિ કરવામાં આવતી માતાજીના વિવિધ રૂપોની આરાધના.

દશેરા - સીતાજી અને શ્રીરામના ધર્મ અને ધીરજ; લક્ષ્મણ, હનુમાન અને અન્ય સર્વેના કર્તવ્યપાલનનો
અસુર સમ્રાટ રાવણના અભિમાન અને અધર્મ પર વિજય!

We had restrictions for celebrating Navaraatri, but we hope that everyone could celebrate it joyfully following proper guidelines and could instill happiness and new enthusiasm with life coming back on track...

Such festivities do instill ' જોશ' in monotonous routines. We welcome Diwali next month with all new hope, wishing everyone life full of health and happiness in forthcoming times!

We have prestigious Presidential Conference by Dr. Alpesh Gandhi with the whole team of ICCOB, in December 2021. The details are attached in one of the pages inside. Please do register at the earliest and get benefitted from Global as well as National and Our Own Society's Stalwarts!

We wish you all 'शुभ दीपावली' and 'नूतन वर्षाभिनंदन' in advance,
from Team AOGS!
May we all and our families,
Prosper and Progress with Health, Wealth and Happiness!

Dr. Jignesh Deliwala
President

Dr. Munjal Pandya
Hon. Secretary

PAST PROGRAMME

PAST PROGRAMME

ART FERTILITY CLINICS
Science Assisting Nature

ART Fertility Clinics in association with
Ahmedabad Obstetrics and Gynaecological Society (AOGS)
cordially invites you to an inaugural CME on Fertility Management

21st October 2021
7:30 pm onwards

Chairpersons:

Dr. Kamini Patel | Dr. Nisarg Dharaiya | Dr. Azadeh Patel

TOPIC	SPEAKER	TIME
Evidence Based Psychosocial Care in Medically Assisted Reproduction (MAR)	**Dr. Gurpreet Singh Kalra** Medical Director ART Fertility Clinics, India	8:00 pm - 8:20 pm
New Developments in Induction of Ovulation	**Prof. Dr. Human Fatemi** Group Medical Director ART Fertility Clinics	8:20 pm – 8:50 pm
PGT-A: Overcoming the Challenges	**Mr. Ibrahim ElKhatib** Chief Embryologist ART Fertility Clinics	8:50 pm – 9:10 pm
Interactive Session		9:10 pm – 9:25 pm
Dinner		9:25 pm Onwards

Venue: Radisson Blu Hotel
Near Panchvati Cross Road, Off C.G. Road, Ambawadi, Ahmedabad-380006

www.artfertilityclinics.com






FINAL RESULTS IN IMA NATIONAL OLYMPICS HELD AT NASHIK MAHARASHTRA

CONGRATULATIONS
Dr. Falgoon S. Parikh

WON TOTAL 12 SILVER MEDALS

at the end of the Mega Sports Event held on in November end 21
The Highest individual medal winner from Gujarat and also Nationally.
(Includes the whole event)

RESULTS

Freestyle	Breaststroke	Backstroke	Butterfly
50 , 100 , 200 metres	50 100 200 metres	50 100 200 metres	50 100 200

SILVER MEDALS IN ALL 12 EVENTS

State Champion in Khel mahakumbh Gujarat
National Bronze Medalist in open MASTERS SWIMMING COMPETITION 2018 at vishakhpatnam

Ahmedabad Medical Association
Installation Ceremony - Year 2021 - 2022
President
Dr. Dilip Gadhavi

Ahmedabad Medical Association
Installation Ceremony - Year 2021 - 2022
President - Ladies Club
Dr. Praful Panagar

PAST PROGRAMME

AHMEDABAD OBSTETRICS & GYNAECOLOGICAL SOCIETY

Date : 31st October, 2021 - Sunday

Time : 10.00 am to 1.00 pm

Venue : Hotel Radisson Blu, Panchvati, Ahmedabad.

DR. JIGNESH DELIWALA
PRESIDENT, AOGS

DR. MUNJAL PANDYA
HON. SECRETARY, AOGS

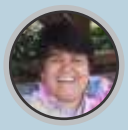
Co-Ordinator :

DR. JAYESH PATEL

Subject Experts :

DR. YAMINI TRIVEDI

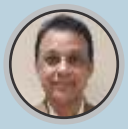
DR. MUKESH SAVALIYA



DR. DARSHANA THAKKAR

Speaker

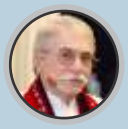
October : 'Down's Syndrome Awareness Month'



DR. BHADRESH SHAH

Speaker

Obstetric analgesia & anaesthesia



DR. M. C. PATEL

Speaker

Medico legal aspects in obstetrics haemorrhage

DR. JAYESH PATEL

Speaker

Carbetocin

Moderator :

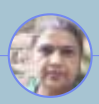
Panel Discussion :

Obstetric Haemorrhage

DR. ALPESH GANDHI

Panelists :

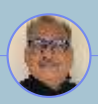
Dr. Ajit Rawal



Dr. Sapna Shah

Dr. Dipesh Dholakiya

Dr. Hetal Patoliya



Dr. Mahendra Soni

Dr. Yamini Trivedi

Dr. Mukesh Savaliya

Obesity and Pregnancy

Dr. Janki Pandya
Assistant Professor, AMC MET Medical College

Dr. Munjal Pandya
Assistant Professor, AMC MET Medical College

- WHO declared obesity as a Global epidemic (2000)
- NFHS-4 (2015–16): The national prevalence of obesity in pregnancy (12%) and in the postpartum period (13%)
- BMI: According to WHO & NICE; classification is based largely on association between BMI and mortality

Table 1 Classification of weight status according to BMI[1,2]

BMI (kg/m²)	Classification
<18.5	Underweight
18.5–24.9	Normal[1]/healthy[2]
25.0–29.9	Overweight
30.0–34.9	Obese I
35.0–39.9	Obese II
≥40	Obese III

BMI = body mass index

Limitations of BMI:

- Inconclusive about distribution of adipose tissue v/s absolute amount; Inconclusive about Abdominal obesity v/s Accumulation of fat around the hips and thighs; unable to distinguish between muscle and fat mass; Across different populations, a given BMI may not correspond to the same degree of 'fatness'
- Waist circumference : Better measure of visceral adiposity and its associated risk (Sattar N, 2001)

Ante-natal

- Nutritional Deficiency
- 40% deficient in Iron, 24% deficient in Folic Acid, 4% deficient in Vit B12
- Folic Acid, Calcium and Iron supplements
- Tendency towards straying away from fortified cereals, fruits and vegetables; and eating more of processed foods, high in calories, but low in nutritional value.
- Ideal diet: Healthy mix of fruits and vegetables, lean proteins, good quality carbohydrates
- **Weight gain during pregnancy:**
- GWG (Gestational Weight Gain): composite of products of conception, plasma volume expansion, extracellular fluid, maternal fat deposition, and imprecise estimate of increasing maternal (or fetal) adiposity.
- Excessive wt gain: very high risk of complications, including indicated preterm birth, CS, failed labour induction, LGA infants, infants with hypoglycemia.

IOM (Institute of Medicine)		Optimal wt gain (Cedergren MI, 2007)	
Underwt (BMI<18.5)	12.5-18 kg	Underwt	4-10 kg
Healthy (BMI 18.5-24.9)	11.5-16 kg	Normal wt	2-10 kg
Overwt (BMI 25-29.9)	7-11.5 kg	Overwt	<9 kg
Obese (BMI >30)	5-9 kg	Obese	<6 kg

- Obese women with less wt gain: reduced risk of pre-eclampsia, CS, instrumental delivery, LGA babies.
- Kiel DW, 2007: 4 outcomes were studied for pregnancy wt gain and obesity class: pre-eclampsia, CS, LGA and SGA)- 1st 3 outcomes decreased with reduced wt gain, but SGA increased. Conclusion was drawn that overall minimal risk for mother and baby should be taken at point of equal risk of LGA and SGA corresponding with wt gain of 4.5-9 kg for class 1 (BMI 30-34.9), 0-4 kg for class 2 (BMI 35-39.9) and class 3 (BMI ≥40)

Challenges with diagnostic tests:

- Excess body fat can make it challenging for USG examination. TVS can be considered in whom transabdominal USG can't conclude NT. Increased echogenicity of adipose tissue and increased absorption of ultrasonic sound beam by abdominal fat

- FHS: difficult to be heard by Auscultation
- BMI > 35 kg/m^2, are likely to have inaccurate SFH and should be referred for serial assessment of fetal size using USG
- Appropriate Sphygmomanometer cuff size, with its documentation

Maternal Complications associated with Obesity:

- **HDP, Pre-eclampsia:** Direct correlation between maternal BMI and risk of preeclampsia. *(O'Brien TE, 2003)*. Pre-pregnancy BMI >35 increased the risk of preeclampsia four-fold compared with women with a pre-pregnancy BMI of 19–27.
- Waist circumference: A non-pregnant waist circumference 80 cm has been associated with an OR for pregnancy-induced hypertension of 1.8 and for preeclampsia of 2.7 in a cohort of over 1000 unselected pregnancies. *(Sattar N, 2001)*
- **GDM:** Retrospective UK study of 287,213 pregnancies between 1989 and 1997: women with a BMI >30 were more likely to develop gestational diabetes than women with a BMI of 20.0–24.9 *(Sebire NJ, 2001)*. (GDM) increases the long-term risk of developing type 2 diabetes. *(Lauenborg J, 2004)*
- **Obstructive sleep apnea:** Sleep apnea can cause fatigue, increases risk of HTN, pre-eclampsia, heart and lung problems.
- **VTE:** 57% of Maternal Deaths from VTE in the UKwere obese. Denmark: Retrospective case-control study: A significant association between VTE and obesity *(Larsen TB, 2007)*. Associated problems of reduced mobility, co-morbid conditions that predispose to thrombosis, such as preeclampsia, and an increased frequency of operative delivery, higher levels of coagulation factors
- **Preterm birth:**
- Obesity is associated with lesser incidence of spontaneous preterm birth, but more of medically indicated induced preterm birth, as compared to non-obese counterparts.
- **Respiratory System:**
- Respiratory disease (asthma and obstructive sleep apnea) increases risk of non pulmonary pregnancy complications, such as CS and pre-eclampsia. Upto 30% experience an exacerbation of their asthma during pregnancy (1.5 times more than non obese). Wt loss b/w pregnancies reduces risk of SB, hypertensive complications and fetal macrosomia. Wt loss increases chances of successful VBAC.

Fetal complications during pregnancy:

- Higher incidence of **first trimester miscarriage**(OR 1.2) *(Lashen H, 2004)*
- **Birth defects:** increased risk of heart defects and NTD. 3-fold risk of spina bifida, omphalocele and heart defects in babies of obese mothers. Confirmed association between maternal obesity and spina bifida, heart defects, anorectal atresia, hypospadias, limb reduction defects, diaphragmatic hernia, omphalocele. *(Watkins ML, 2003; Cedergren MI, 2003; Waller DK,2007)*
- **Macrosomia:** high risk of birth injuries; high chances of CS. Nearly a fifth of women with a BMI ≥30 had fetal macrosomia; independent of whether the mother also had pre-existing or gestational diabetes. *(Jolly MC, 2003)*. Macrosomia is a risk factor for operative delivery, a low Apgar score at one minute and a low umbilical arterial pH level, as well as shoulder dystocia and significant injuries to the baby, including fractures and nerve palsies.
- **Preterm birth:** Medically indicated preterm birth.
- **Stillbirth:** Higher BMI, greater risk of SB. Women with a BMI >30 had a stillbirth rate of 6.9/ 1000 total births compared with 4/1000 total births in women with a BMI of 20 –25 (adjusted OR 1.40)
- **Cerebral Palsy:** The risk of developing cerebral palsy increases with increasing BMI, risk being 22%, 28%, 54%, 202% for Overweight, Obesity class 1, Obesity class 2, Obesity class 3 respectively.
- **Neonatal Death**

Intra-partum:

- Difficulties with **venous access**
- *Vahratian* A, 2004: Women with a BMI ≥30 were more likely than women with a BMI ≤26 to have their **labour induced** and to receive oxytocin. Labour progression from four to 10 cm was slower in obese women (7.9 versus 6.2 median hours). These data suggest that obesity is associated with inefficient uterine activity in labour.
- Primary emergency **caesarean section** rates were higher for obese women compared with women with a healthy BMI (27% versus 19%, P , 0.04), with the majority of the deliveries occurring during the first stage of labour for failure to progress in labour and fetal distress
- **Anesthesia:** Increased requirements, higher risk of anaesthesia-related morbidity, higher epidural failure rate *(Dresner M, 2006)*. Increased risk of aspiration under general anaesthesia, Difficult endotracheal intubation, Difficulty in achieving regional analgesia/anaesthesia, Postoperative hypoxaemia and atelectasis. Co-morbidites

such as hypertension, ischaemic heart disease and heart failure, adding to the risks associated with anaesthesia

- **C.S.:**Preoperative skin cleansing before CS. Vaginal cleansing using povidone-iodine/Chlorhexidine gluconate before CS in laboring pts and those with ruptured membranes may be considered. *(ACOG, 2018)*. Surgical access to uterus : Transverse incision was found to be better than vertical incision*(RCOG GTG, 2018)*. Class 3 obesity is associated with increased rates of uterine rupture during trial of labour and neonatal injury. Emergency CS is associated with increased risk of serious maternal morbidity because of anesthetic and operative difficulties
- Suturing of subcutaneous fat: More than 2 cm depth of subcutaneous fat layer will require suturing of the layer. Negative suction drain in that layer has not found to be of additional advantage.

Post Partum:

- **Hemorrhage and Infections:** PPH, Genital tract infection, UTI, wound infection.
- Increased risk of **VTE** after both CS and ND. Postpartum infection with obesity may increase chances of VTE.
- **Breastfeeding:**BF rates are poor among obese *(Amir LH, 2007)*. Possible reasons: difficulty with correct positioning of baby, psychological issues, or endocrine issues such as prolactin response to suckling. *(Rasmussen KM, 2004)*.
- Challenges for BF: takes longer for the milk to come in, lower production (breast size has nothing to do with amount of milk produced). Indicated preterm and admission in NICU means prolonged separation of mother from baby. Plus higher rates of maternal complications along with CS reduce the rate further.
- BMI > 40 kg/m^2 is a risk factor for developing **pressure sores;** immobility being one more risk factor
- **Mental health problems:** high prevalence of depression symptoms. High antenatal anxiety, postpartum anxiety, eating disorders, antenatal serious mental illness.
- **Long term implications in childhood:** Intra-uterine exposure to obesity is associated with increased risk of developing obesity and metabolic disorders in childhood. *(Barker hypothesis, 1990)*
- **HAPO Study:** Amongst obese women: Raised maternal blood glucose, Raised tryglyceride and fatty acids, and fetal insulin concentrations, contributing to fat accretion in offspring. Maternal and cord blood leptin conc is elevated, with evidence of low grade inflammatory state in mother with high levels of CRP and IL 6; linked to insulin resistance.

Management:

- **Folate** Supplementation: 400 microgram daily
- **Wt loss:** loss of 4.5 kg b/w 2 pregnancies reduced risk of developing GDM by upto 40%. *(Glazer NL,2004)*
- 10% wt loss over 6 months is ideal, safe and possible to sustain in long term.
- Wt loss during first trimester may increase NTD; but prior to pregnancy does not appear to carry this risk. *(Carmichael SL, 2003)*
- **Following Bariatric Surgery:** Maternal and perinatal complications were less
- Dumping syndrome: To avoid same, OGTT isn't recommended for women who had bariatric surgery; instead, Home based glucose monitoring for at least 1 wk is advisable. *(Ukleja A. 2005)*
- RCOG GTG, 2018 & ACOG: Min waiting period of 12-18 months after bariatric surgery is recommended before attempting pregnancy
- RCOG GTG (2018): women with more than one moderate risk factor (BMI of 35 kg/m^2 or greater, first pregnancy, maternal age of more than 40 years, F/H of pre-eclampsia and multiple pregnancy) may benefit from taking 150 mg **Aspirin** daily from 12 wks of gestation until birth of baby.
- **Regular Moderate intensity physical activity:** RCOG (2006) &Cochrane review suggested regular aerobic exercise during pregnancy improved maternal fitness, beneficial effects of fetal growth. *(Kramer M, 2008)*
- Anti-obesity or wt loss drugs are NOT recommended for use in pregnancy: Orlistat- No major malformation risk. Phentermine/Topiramate- Topiramate during pregnancy- oral clefts; both are excreted in breast milk, and carries unknown risks to infant. *(RCOG GTG 2018)*
- Metformin: for overwt/obese pts without DM; addition of metformin to diet and lifestyle changes, starting at 10-20 wks DID NOT have improved pregnancy/birth outcome. *(Dodd JM et al. 2019)*
- Type 2 DM steep increase within first 5 years

Future Prospects:

- Prospective RCT are needed in obese pregnant women to assess
- Effects of diet, physical activity, lifestyle changes on maternal, fetal and neonatal outcomes.
- ? Optimal wt gain is needed
- ? Optimal methods of assessing body fat in women
- ? Optimal gestation of screening obese women for GDM and whether early detection and management improves outcomes

Antihypertensive Treatment- Newer Insights

Dr. Sanjay Gupte

Dr. Arati Gupte Shah

The treatment of pregnancy hypertension has always been controversial for various reasons.
First is the classification as it stands. Gestational Hypertension, Chronic Hypertension, Preeclampsia and Superimposed Preeclampsia , though now defined categorically, still lackspecificity.
Traditionally the level of 140/90 mm of Hg is considered the minimum level for diagnosis.From 140 to 159 mm of Hg systolic and 90 to 109 mm of Hg diastolic is considered mild hypertension.Some guidelines divide this between mild and moderate.
The only non controversial figure is 160/110 mm of Hg which is taken as severe hypertension.
Second point of difference is: traditionally in contrast to hypertension guidelines in adults, which emphasizes the importance of systolic BP, much of the obstetric literature focuses on diastolic rather than systolic BP; in part, because of the lack of clinical trials to support one approach versus another.
During pregnancy, antihypertensive agents are mainly used to prevent and treat severe hypertension; to prolong pregnancy for as long as safely possible, to maximize the gestational age of the fetus; and to minimize fetal exposure to medications. So the challenge is in deciding when to use antihypertensive medications and what level of BP to target.
The choice of antihypertensive agents is less complex, because only a small proportion of currently available drugs have been adequately evaluated in pregnant women, and many others are contraindicated.

Treatment of Severe Hypertension

There is consensus that severe hypertension in pregnancy, defined as 160/110 mm Hg, requires treatment, because these women are at an increased risk of intracerebral hemorrhage, and that, treatment decreases the risk of maternal death. [1,2]
In treating severe hypertension, it is important to avoid hypotension, because the degree to which placental blood flow is autoregulated is not established, and aggressive lowering may cause fetal distress.

Protocol for management of severe hypertension

BP more than 160/110 with emergent presentation

IV Labetalol	5 mg. 1st dose, double every 15mins. To Max.300mg.
Nifedipine	10mg. Oral every 20mins.Max. 90mg.
IV Hydralazine	10mg. IV every /15 mins. Max. 300mg.
Nitroprusside	0.25micro g./kg/min. Max. 10micro g./kg/min.

Use of Nicardipine

Nicardipine acts more selectively on the blood vessels than on the myocardium, with less reflex tachycardia. In a multiple-center placebo-controlled study, intravenous nicardipine was shown to be both effective and safe in patients with severe hypertension.
Onset of action is in 10 mins and results in adequate reduction of BP within 20 mins (duration of action 4–6 hrs).The most common adverse reaction was headache.

Comparison of labetalol and nicardipine in severe hypertension

Sixty consecutive pregnant women admitted beyond the 24th wk of pregnancy with severe hypertension[3].Patients were randomly assigned to receive intravenously for 1 h either labetalol (n=30) or nicardipine (n=30). Treatment was titrated to achieve a 20% lowering of blood pressure (BP).
Labetalol and nicardipine achieved the 20% lowering in BP in the same proportion (63% and 70% success rates, respectively). Overall nicardipine caused a significantly greater decrease in systolic and diastolic BP. No patient had any episode of hypotension.
The length of time to achieve the BP goal was also similar (12 vs. 11 min, respectively). Both drugs were well tolerated except for a moderate tachycardia observed with the use of nicardipine.

Treatment of mild to moderate hypertension.

This is still a controversial issue for many.

In favour of treatment is the fact that blood pressure may be extremely labile in preeclampsia and treatment at lower blood pressure levels will prevent or attenuate acute and severe rises in blood pressure. In addition, it is possible that pharmacologic arteriolar vasodilation may help improve organ perfusion.

Arguments against treatment include that there is little risk to the mother in having relatively mild hypertension for a short time (usually only a few days or at the most weeks), that fetal perfusion is dependent upon adequate maternal blood pressure and that, lowering blood pressure suppresses an important sign of the severity or progression of preeclampsia.

Principles for Treatment of Mild-to-Moderate Hypertension in Pregnancy

The benefits of antihypertensive therapy for mild-to moderately elevated BP in pregnancy (160/110 mm Hg), either chronic or pregnancy induced, have not been proved in clinical trials.

Recent reviews, including a Cochrane meta-analysis in 2007[4], concluded that there are insufficient data to determine the benefits and risks of antihypertensive therapy for mild-to-moderate hypertension (defined as 140 to 169 mm Hg systolic BP and 90 to 109 mm Hg diastolic BP).

A retrospective review of 28 patients who suffered stroke in the setting of preeclampsia demonstrated that the cause of stroke was usually arterial hemorrhage, that the average BP before stroke was 159 to 198 mm Hg systolic and 81 to 133 mm Hg diastolic, and that 54% of women died.[5]

Of note, systolic hypertension (155 to 160 mm Hg) was more prevalent than diastolic hypertension (most women did not reach a diastolic BP of 110 mm Hg) in women who suffered strokes. This case series underscores the need for clinical trials and evidence-based guidelines for antihypertensive treatment in pregnant women.

A 2014 Cochrane systematic review of 49 trials (4,723 women) concluded that treatment of mild-to moderate hypertension reduced the risk of developing severe hypertension but had no effect on the incidence of preeclampsia, preterm birth, fetal death, fetal growth restriction, or any other measured outcome[6].

The theoretical concern of fetal harm (primarily growth restriction) resulting from possible impairment of the uteroplacental blood flow was not found in this systematic review.

The CHIPS trial[7] (Control of Hypertension in Pregnancy Study) has provided evidence that antihypertensive treatment of nonsevere hypertension in pregnancy is of benefit to the mother, without associated perinatal risk.

This was an open international multicenter trial with 937 women with nonsevere, non proteinuric pre-existing or gestational hypertension. The patients were randomized into 2 groups- tight control group and less tight control group. Analysis showed that within the same population, less tight control is associated with more severe hypertension, that severe hypertension is a risk marker for adverse maternal and perinatal outcomes, and that the risks associated with severe hypertension are over and above those associated with the co-occurrence of preeclampsia.

CHIPS data also indicates that severe hypertension is an outcome worthy of avoidance to minimize maternal and perinatal risk. As such, we should move from detection and prompt treatment of severe hypertension to prevention.

This can be achieved with antihypertensive therapy to normalize maternal BP, as practiced in the tight BP control arm of the CHIPS trial, aiming for a modest dBP of 85 mm Hg. Future work should focus on whether one antihypertensive agent offers advantages over another.

Which Drug should be used?The Indian context

The choices that we have today :

Alpha- methyldopa : time tested

Nifedepine : fast and effective

Labetolol : oral as well as injectable

New addition -- Amlodipine

Amlodipine is among the most widely prescribed antihypertensive medications for nonpregnant individuals; however, initially, only limited data (<50 cases) regarding safety in pregnancy were available.[8,9]Mito and colleagues recently (2019) reported important new information about amlodipine exposure in the first trimester of pregnancy.[10]Theyconcluded that, based on this admittedly small sample of 231 patients, amlodipine exposure in early pregnancy does not appear to be associated with an increased rate of fetal malformations compared with other antihypertensive medications or maternal hypertension without treatment.

Would there be any reasons to preferentially use amlodipine rather than Nifedipine SR in pregnancy, provided sufficient safety data were available?

Both are dihydropyridine CCBs, and lower BP similarly by preventing the entry of calcium through L-type calcium channels in the vasculature. [11,12] The half-lives of the various dihydropyridines differ, which may affect BP control over a 24-hr period.

Amlodipine is one of the longest acting dihydropyridine CCBs. Amlodipine is tolerated well. It can be given in single dose. No adverse side effects are noted like Sudden Hypotension, Tachycardia, FGR.

Nifedipine SR is designed to provide BP control at a constant rate over 24 hours and has a shorter half-life than amlodipine (≥44 hours), [11] but in most patients it provides adequate BP control over a 24-hour period, provided the dose is adjusted appropriately.

Conclusions

The development of severe hypertension raises concern about elevated stroke risk, but the CHIPS data demonstrate that the risk of other adverse perinatal and maternal outcomes, including serious maternal complications, is also increased, independently of the co-occurrence of preeclampsia.

Hence it is wise to start controlling mild hypertension to prevent severe hypertension.So tight control seems to be the way forward.

References:

1. Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. *Am J Obstet Gynecol.* 2000;183:S1–S22.
2. Rey E, et al Report of the Canadian Hypertension Society Consensus Conference: 3. Pharmacologic treatment of hypertensive disorders in pregnancy. *CMAJ.* 1997; 157:1245–1254
3. Elatrous F et al. Short-term treatment of severe hypertension of pregnancy: prospective comparison of nicardipine and labetalol. Intensive Care Med. 2002 Sep;28(9):1281-6. doi: 10.1007/s00134-002-1406-3. Epub 2002 Jul 26. PMID: 12209278.
4. Abalos E, Duley L, Steyn D, Henderson-Smart D. Antihypertensive drug therapy for mild to moderate hypertension during pregnancy. *Cochrane Database Syst Rev.* 2007;CD002252.
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