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Association of Fellow Gynaecologists

In Memoriam...

the lost two years

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News letter of the
Association of Fellow Gynaecologists
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From the Editor's desk.....

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Dear AFGites,

Waiting to exhale.... and inhale without a mask. With bated breath, all of us are waiting for the pandemic to either end or co exist in a milder form. As we dare to hope for this, let us take a moment to remember the millions of fellow humans who lost their lives, before their time, in the past 2 years. The cover page of this issue reads' in Memoriam' to those who are no longer in our midst and to the lost two years of our lives.

Heartfelt Shraddhanjali

We will be having our first Physical Conference on 6th March, 2022 after a hiatus of two years.

Such a wonderful feeling to finally meet colleagues and friends.

Do enjoy this issue and contribute articles to
editorafgtimes@gmail.com

Keep pushing.....

DR. REKHA AMBEGAOKAR
Editor, AFG Times

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Facts and opinions expressed in the published articles are solely those of the Authors and do not necessarily reflect the opinion of the AFG.

From the President's desk.....

Dear AFGites,

Firstly, I want to thank the Association of Fellow Gynaecologists for allowing me to serve as President and be a part of an organization that not only works for spread of knowledge but allows friendship to be created.

The first challenge was how to spread knowledge. Thanks to the virtual platform, we have had virtual CME on important topics like Tuberculosis; hypertension in pregnancy, Antiphospholipid syndrome; mullerian anomalies; vulvovaginal disorders, anaesthesia in obstetric and Gyanec practice, conservative management of PPH & micro invasive CaCx. Recordings of these programmes could be saved on You Tube and link to these is available on AFG website.

RAN symposium, which is a teaching programme for students could be attended by students from outside Mumbai too. Here students had an opportunity to present cases as they would do for their examination. We have had symposia on SUI, Fistulae, Carcinoma endometrium, Screening in pregnancy, thyroid disorders, ectopic gestation & Recurrent pregnancy loss. The link to recordings of these programmes are available on AFG website.

We have associated with SOVSI for programme on Perineum, Isoparb for programme on PPH & Updates on Preeclampsia, OBGY Society of Mira Bhayander for Breast awareness programme during breast awareness week and FEPPA for multispeciality programme on Chronic Pelvic Pain.

On Menstrual Hygiene day, AFG released a video on "Menstrual Hygiene" prepared by compilation of short videos by all Managing Committee Members conveying different messages with a single theme to the public. This video is available and can be shared for social awareness.

We have been able to conduct 2 nurses training programmes attended by 350 and 200 nurses respectively.

Live operative workshop was planned on 23rd JAN at Shatabdi Hospital. Because of the 3rd wave of Covid, we changed it to virtual platform. Prerecorded video of the surgical procedure with description of the procedure and detailed discussion was held. This was a blessing in disguise as we could have faculty and participants from all over India. It was highly appreciated.

In this era, virtual meetings have been by default . Actual coming together is an experience we don't want to miss. Annual Scientific meeting on 6th March at Hotel Westin, is this opportunity.

Release of AFG Times at Annual Scientific Conference needs a special mention. It has been possible with special effort of the Editorial team under guidance of Dr Rekha Ambegaokar.

Launch of our new updated website is a special event associated with the Annual conference. We appreciate the efforts of Dr Saurabh Dani & Dr Rakesh Pandia . This will improve communication among members.

All this has been possible with whole hearted cooperation of all managing committee members, guidance from our senior members and the faith of AFG members who have attended the programmes.

Regards

DR. URMILA SUREKA

President 2021-22, AFG

Till we meet...

two cells meet
in a cosmic dance
nuclei divide
cytoplasms merge
into a pulsating
living throb
a heart beats
neurons fire
a comma appears
a faint silhouette
of flesh and blood
a form emerges
with nose
and lips
and fingers
and toes
a gently curving spine
and pumping fist
a swirl, a kick
a somersault

i am almost there
it says
sing to me
talk to me
love me
i am yours
made of genes
of ancestors
who roamed
this earth
aeons ago
i live
and breathe
and listen
to murmurs
words of love
caresses and kisses
i dance
in warm waters
and wait...
to meet you
finally

Dr. Rekha Ambegaokar

RHESUS ISO-IMMUNISATION

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Introduction

The Rhesus factor is a red blood cell surface antigen that was named after the monkeys in which it was first discovered. Rh incompatibility is a condition that occurs when a woman with Rh-negative blood is exposed to Rh-positive fetal blood cells, leading to the development of Rh antibodies. Erytroblastosis fetalis is a disease in which the red blood cells of the fetus and the newborn are hemolysed by maternal alloantibodies that have crossed the placenta resulting in fetal anemia.

The conquest of Rh isoimmunisation

The story of Rh disease began with the publication of the historic paper on hemolytic disease of the newborn infant by Philip Levine in 1939 in the city of New York. In the subsequent year Karl Landsteiner and Alexandar Weiner from the Rockefeller Institute in New York, USA, discovered the existence of Rhesus antigen isolated from the Rhesus monkey, hence the name (1). In 1941 Levine, Katrin and Burnham suggested Rhesus isoimmunisation as a cause for fetal erythroblastosis, a condition in which the fetus becomes edematous and often dies in utero from severe anemia and high output cardiac failure. Liley described the spectrophotometric findings of amniotic fluid in sensitized Rh pregnancies and developed the Liley's Chart in 1961. In 1957 Kleihauer, Betke and Braun introduced the Acid Elution test for fetomaternal hemorrhage.

Remarkably, only 20 years after the discovery of Rh incompatability in pregnancy, effective treatment became available. In 1963, Liley performed the first successful intrauterine intraperitoneal transfusion. The first direct intravascular intrauterine transfusion into the umbilical cord was first described by Charles Rodeck in 1981 where transfusion was done under fetoscopic guidance (2). The currently used technique of Ultrasound guided intravascular transfusion was performed by Fertinand Daffos in Paris and Jens Bang in Copenhagen directly into the umbilical vein (3). Intrahepatic portal vein transfusion was first described by Nicolini et al in 1990 (4).

In 1964 Kathryn became the first woman to be protected from Rh immunization when Vincent Freda and John Gorman of Columbia University first used gammaglobin for this purpose. By late 1960s, Rh alloimmunisation became preventable due to the availability of anti D immunoglobulin. In 1978, Bowman showed that 1% Rh negative women produce antibodies during pregnancy, thus prompting the need for prophylactic antenatal therapy.

Rhesus isoimmunisation is an example of a disease where pathogenesis, treatment and prophylaxis have all been elucidated in the past 50 years.

Rh blood group system and the Genetics

Rhesus factor is the largest and most complex protein based blood group system with over 49 different Rh antigens. Most of them are not clinically significant. The five antigens which are immunogenic and genetically significant are collectively called as Rhesus factor & comprise C,c,D,E,e. Most immunogenic is D antigen presence of which denotes the blood group as Rh positive and absence of D antigen as Rh negative. 97% of all cases of erythroblastosis fetalis are caused by maternal antibodies against RhD antigen on fetal red blood cells. Other rare atypical antigens systems that have potential to cause hemolytic disease are Kell, Duffy, Kidd and MNS.

According to the Fisher Race theory, the Rh antigens are encoded by 3 sets of allelic genes at closely linked loci on the short arm of chromosome 1. Every individual inherits one of each set of three from each parent as per the mendelian law of inheritance. If D is inherited from one parent, the individual is Rh positive but heterozygous (e.g. Cde/CDe) while if D is inherited from both parents, he is Rh positive homozygous (cDe/CDe). If both sets do not have D antigen the individual is Rh negative and they are always homozygous. 65% of all Rh positive individuals are heterozygous and 35% are

homozygous. The clinical implication of knowing the zygosity is that if the father is homozygous Rh positive all his offspring with be Rhpositive. If he is heterozygous Rh positive, the chance of the child inheriting paternal D gene and becoming Rh positive is 50%.

Incidence

The incidence of Rh negative individual varies by race. Caucasians have an incidence of 15% and African Americans of 7-8%. In Indian and Asians the frequency of Rh negative population is around 5%.

Pathophysiology

Transplacental feto-maternal hemorrhage is the most common cause of Rh isoimmunisation. Heterologous blood transfusion of Rh positive blood to Rh negative mother is the etiology in some. Placenta usually acts as a barrier to fetal blood entering the maternal circulation. Fetal cells can enter the maternal circulation through a 'break' in the 'placental barrier'.

During normal pregnancy small volumes of fetal red blood cells continually gain access into maternal circulation without a break in barrier. This fetomaternal bleed occurs in 3% cases in the first trimester, 12% in second, 46% in the third trimester and as a rule at the time of parturition (5). However only 10-15% of all Rh negative mothers with Rh positive fetuses will get sensitized at delivery and less than 1% during antenatal period. The major reason is that the fetomaternal bleed is too small to trigger an immune response. Coexistence of ABO incompatibility is protective for Rh isoimmunisation (4-5%). If mother is O negative with a fetus A,B or AB, maternal anti A and anti B antibodies will destroy the fetal RBCs that enter the maternal circulation before they can elicit an immune response to their Rh antigen. Few Rh negative individuals are genetically non- responders to Rh positive antigen and do not get sensitized.

When a fetomaternal bleed occurs in a Rh negative mother with a Rh positive fetus, maternal immune system is stimulated and B lymphocytes clones that recognize the RhD antigen are established. The initial primary immune response produces anti-rhesus IgM antibodies which are short lived and have a molecular weight too large to cross the placental barrier. This primary immune response is followed by synthesis of IgG antibodies which can cross the placenta and destroy fetal erythrocytes and cause fetal anemia. Fortunately this alloimmune response takes few weeks to months (6 weeks to 12 months) to occur and ensures that the first pregnancy is not at risk (Primary sensitized pregnancy). However the mother will remain sensitized and antibodies remain in maternal circulation throughout her life and in every subsequent pregnancy.

Once a sensitized mother has a second Rh positive fetus, Rh positive fetal D antigen will trigger an amnestic secondary immune response from a previously primed maternal immune system and produces IgG antibodies rapidly (usually measured in days). These IgG immunoglobulins cross the placenta and enter fetal circulation and attack fetal blood cells. In the event of an appropriate fetal antigen being present in the fetal blood, the maternal IgG antibodies bind to these antigens and cause an antigen antibody reaction leading to hemolysis of the fetal RBCs in the fetal reticuloendothelial system leading to fetal anemia (Figure 1). Depending upon the severity of hemolysis the clinical presentation may be congestive cardiac failure with hydrops fetalis, compensatory extramedullary hematopoeisis causing hepatosplenomegaly, placentomegaly, death in utero to hyperbilirubinemia / kernicterus (Icterus gravis neonatorum) or just mild congenital anemia of the newborn (Figure 2). Placentomegaly leads to pre- eclampsia like syndrome in the mother. Thus, both the mother and the fetus are edematous. Hence also called as "mirror syndrome".

Immunoprophylaxis

A major achievement in preventive obstetric was introduction of Rh D immunoglobulin derived from human plasma. Anti D immunoglobulin bind with Rh positive antigen sites on fetal cells in maternal circulation and neutralize them so that they are unavailable for sensitizing the maternal immune system. A single 300 µg dose of Anti D is adequate for 15ml of fetal cells or 30ml of whole blood, and is sufficient for preventing immunization in most patients.

Post- partum prophylaxis

Rh immunoglobulin administration came into general use as a 300 µg, single dose administered deep intramuscular within 72 hours of delivery to all Rh-negative mothers at risk of Rh sensitization. The beneficial effects may still be present with administration up to 28 days after delivery.

Antepartum Prophylaxis

Although sensitization occurs mostly after delivery, around 15% are caused by small fetomaternal hemorrhages (silent bleed) that occur between 20 to 40 weeks. The practice of routine antenatal anti D

prophylaxis (RAADP) after 28 weeks reduced the incidence of antenatal alloimmunisation from 2% to 0.1%.

Potential sensitizing events for antepartum Anti D prophylaxis (as per ACOG 2018, RCOG NICE 2002) (6,7)

- Threatened, inevitable and induced abortion
- Ectopic pregnancy
- Antepartum hemorrhage
- Invasive prenatal diagnostic procedures such as CVS, amniocentesis, fetal blood sampling
- External cephalic version
- Blunt trauma abdomen
- All nonimmunized Rh-negative pregnancies at approximately 28 weeks of gestation.

Recommended timing and dosage of Anti D

Uncomplicated Rh-negative pregnancies will be protected if they receive a single intramuscular dose of 300µg of Anti D at approximately 28 weeks of gestation, followed by another dose within 72 hours of birth of a Rh-positive baby. Anti D immunoglobulin has a half life of 24 days. Theoretically, based on the half-life, an anti D injection given at 28 weeks of gestation provides protection for 12 weeks or 84 days.

Serology to detect isoimmunisation

The presence of Anti D antibodies in the maternal serum by indirect coombs test is diagnostic of maternal alloimmunisation. If Indirect coombs test is positive, Rh antibody titres are quantified by double dilution of test serum that is able to produce agglutination with RBCs. The reciprocal of the highest dilution that causes agglutination is taken as the titre. Critical titre is the titre at or below which no fetal death or severe alloimmunisation occurs. Its value varies with different labs and the same lab should be used for subsequent titre analysis. For most labs the critical titre is ee 1:16 (Range >1:8- 1:32).

Tests for determining amount of feto-maternal bleed (Kleihauer- Betke Acid elution test)

Kleiuhauer Betke test is based on the fact that an acid solution elutes adult hemoglobin but not fetal hemoglobin from the red blood cells. A monolayer of blood film or smear of red cells are fixed using alcohol. An acid buffer is used to elute (remove) HbA as it is more soluble than HbF which is the predominant Hb in fetal red cells. This slide is then counterstained with eosin. Cells containing Hb F stain darkly whereas HbA cells appear as ghost cells (pale and unstained). This test can detect as little as 0.2 ml of fetal blood cells in 5 ml of maternal blood.

The KB test is required only when massive feto-maternal hemorrhage is suspected (0.1% of deliveries) as in abruption placenta, abdominal trauma, unexplained fetal demise, placenta previa, multifetal gestation, manual removal of placenta, etc. The dosage of anti D immunoglobulin would then need to be increased as per the amount of feto-maternal bleed.

Management

Rh negative nonimmunized pregnancy

All Rh negative pregnant women must have their partners checked for Rh grouping. If he is Rh negative, consider as normal pregnancy with no further alterations. If he is rh positive, mother should be screened for Rh antibodies by indirect coombs test. Detailed history of previous pregnancies and whether anti D was given or not must be elicited. Any previous still births, neonatal death, hydropic baby or severe jaundice in the neonatal period must be asked. Any prior blood transfusions must be documented as well.

Indirect coombs test is done at booking visit and repeated at 28 weeks and if negative antenatal Anti D prophylaxis of 300 mcg deep intramuscular is given followed by no further ICT testing. Post prophylaxis antibody titres will remain positive for varying duration but the titres should remain below 1:4 before delivery (Figure 3).

Precautions to minimize fetomaternal bleed:

- Precautions during delivery or cesarean section: Prevent blood spillage in the peritoneal cavity, immediate cord clamping after birth, placenta to be delivered by controlled cord traction rather than routine manual removal
- Avoidance of the use of ergometrine
- Minimum abdominal palpation during abruption
- Invasive testing only if required and avoiding transplacental passage of needle, giving Anti D post procedure
- Forcible attempt to external cephalic version avoided and anti D prophylaxis given if attempted
- Timely postpartum prophylaxis as early as possible within 72 hour of birth of an Rh positive fetus and direct coomb test is negative.

Cord Blood to be collected for ABO and Rh grouping, Direct coombs test, serum bilirubin, hemoglobin.

Management of Rh negative first sensitized

pregnancy (Indirect coombs test positive)

In the first immunized pregnancy, only about 10% of fetuses are affected by severe hemolytic anemia. Rh antibody titres correlate well with the severity of disease in the first immunized pregnancy.

If there is no history of an affected infant or fetal death and the indirect coombs test becomes positive during initial screening, monitor by Rh antibody titres every 4 weekly from 16-18 weeks till 28 weeks and subsequently every 2 weeks. If the titres remain below critical levels upto 36 weeks, deliver at term by elective induction between 38-40 weeks. If the titres cross the critical titres at any time of gestation or if there is a significant rise in the titers between 2 consecutive samples (> two tube dilutions), further assessment based on USG and MCA doppler is necessary to determine fetal anemia.

Paternal Rh genotype

Paternal zygosity can be evaluated by DNA testing using multiplex quantitative Polymerase chain reaction. Fetal Rhesus D status can also be now determined reliably by PCR analysis on cell free fetal DNA in maternal circulation without subjecting the pregnancy to invasive testing for fetal genotype.

Management of Rh negative isoimmunised pregnancy with a previously affected fetus

In females with a history of previous anemic /icteric newborn, the probability of subsequent affected Rh incompatible fetus is 80%. The antibody titres are not predictive of the severity of disease. The most powerful predictor of severity is the timing of fetal affection in previous pregnancy and the titres at which fetus was affected previously. Fetal anemia sets in 8-10 weeks earlier than in previous pregnancy and the assessment of the fetus for anemia should start 10 weeks prior to the gestational age of previous fetal affection.

When critical antibody titres are reached in a first sensitized pregnancy or in pregnancy with previously affected fetus, fetal anemia is monitored non invasively using Middle Cerebral artery peak systolic velocity (Figure 4).

Non invasive diagnosis of fetal anemia

Use of Middle cerebral artery peak systolic velocity as a noninvasive method for fetal anemia has been a practice changing discovery in the field of Rh isoimmunisation. The landmark study by Mari et al in 1995 showed that increased velocity in the middle cerebral artery above 1.5 MOM is extremely accurate method for diagnosing moderate to severe fetal anemia with a sensitivity of almost 100% and a false positive rate of 12% (8). This noninvasive monitoring has resulted in reduction of invasive procedures by 70% and is now the standard of care for diagnosis and management of fetal anemia.

Technique for MCA PSV measurement:

The middle cerebral artery was selected for use because of its ease of sampling while maintaining almost 0 degree angle for accurate and reproducible measurement of velocity of blood flow. Anemia causes a reduction in the viscosity of blood leading to increased velocity blood flow. It can be reliably measured between 18 weeks to 35 weeks of gestation.

Middle cerebral artery on ultrasound can be seen in an axial plane of the brain at the level of the sphenoid bone, thalami and cavum septum pellucidum (Figure 5). MCA of the cerebral hemisphere closer to the ultrasound beam is sampled at proximal portion as it originates from the Circle of Willis taking care that the angle of insonation is as close to 0 degree (Figure 6). The highest point on the peak is measured using electronic calipers and are plotted on Mari et al chart. The threshold for diagnosis of severe fetal anemia is 1.5 Multiples of Median for that gestational age. The measurements can be falsely increased if measurements are taken during fetal movement, uterine contraction or late in gestation after 35 weeks.

This non-invasive method has superseded the traditional technique of serial amniocentesis for the spectral analysis of amniotic fluid at 450 nm (delta OD450) first described by Liley in 1961. Liley's technique was used to measure bilirubin in amniotic fluid, an indirect measure of fetal haemolysis to determine time for intrauterine transfusion. These invasive techniques for determining fetal hemolysis have now been completely abandoned.

Other B mode ultrasound assessment for fetal anemia

a. Hepatomegaly occurs due to compensatory extramedullary hematopoiesis. Increase in the measurement of the abdominal circumference maybe seen (since liver occupies about two thirds of the fetal abdomen). Nomograms to compare fetal liver length with degrees of fetal anemia are available.

b. Splenomegaly : An increase in 3- 4 times the original size, due to extramedullary hematopoiesis.

c. Increased blood flow through the umbilical vein due

to hyperdynamic circulation leading to increase in the diameter of the umbilical vein and intrahepatic portal vein.

d. Presence of fluid collection in the various serous cavities. Pericardial space is the first site.

e. Ascites-First feature is clear delineation of the intestinal loops due to presence of free fluid in between.

f. Pleural effusion and diaphragmatic elevation due to hepatosplenomegaly and ascites may lead to pulmonary hypoplasia.

g. Skin edema- skin thickness > 5mm. Ideal site is the forehead while imaging the facial profile.

h. Changes in the fetal heart- The right atrium is the first chamber to dilate due to increased venous return due to increased flow in the umbilical and portal veins. Subsequently this leads to right ventricular dilatation and subsequent tricuspid regurgitation. Finally, the left heart also dilates leading to generalized cardiomegaly.

I. Placentomegaly – Thickness more than 4 cm.

j. Polyhydramnios

k. Hydrops presents as end stage sign of fetal anemia and is observed once the hemoglobin deficit is 7g/dl below the mean for the gestational age. Usually below a hemoglobin of 4-6 gm%.

Fetal blood sampling

It is a direct access to the umbilical cord vessels by ultrasound guided needle puncture and is done once the MCA PSV increases to a range requiring intrauterine transfusion so as to document the exact hematocrit. Due to the extreme accuracy of MCA PSV in predicting fetal anemia, most centres perform cordocentesis simultaneous with intrauterine transfusion. Sample prior to transfusion is collected for hemoglobin / hematocrit and blood group.

Treatment :

Intrauterine transfusion is the treatment of choice for fetal anemia. It is indicated if the MCA PS Velocity reaches above 1.5 MOM suggesting severe fetal anemia or development of immune hydrops or a fetal hematocrit of < 30% at cordocentesis and the fetus is not mature enough to be delivered (<35 weeks). This intervention is more effective if started prior to development of hydrops.

Intraperitoneal transfusion :

Initially when high resolution ultrasound was not available clinicians used the peritoneal cavity for transfusions. In non-hydropic fetus, the rate of absorption is estimated to be 10-15% per 24 hours. Absorption is slower if hydrops is evident. Because of the erratic absorption, especially in hydropic fetuses intravascular fetal transfusion has largely replaced the intraperitoneal technique.

However intraperitoneal transfusion is still indicated if the need for transfusion is early in gestation <18 weeks or if the direct access to cord is not feasible.

The volume calculated for transfusion : (Weeks of gestation – 20) X 10.

Intravascular transfusion :

Two sites used for direct vascular access are intra hepatic portion of umbilical vein or the umbilical vein at the cord insertion site into the placenta (Figure 7). The procedure is done after detailed counseling of the couple regarding the technique, advantages and risk. Pre procedure tocolysis and antibiotic may be given. Operation theatre may be kept standby in case of need for emergency LSCS if bradycardia ensues. A paralyzing agent such as vecuronium / pancuronium may be given intramuscular in the fetal deltoid or intra vascular to minimize fetal movements during transfusion.

Under ultrasound guidance intrauterine transfusion is done of double packed (Hematocrit of 75-80%) fresh within 72 hours, O negative blood, leucodepleted, CMV negative, gamma irradiated, cross-matched with mothers blood into the intravascular (cord insertion site or hepatic portal vein) or intraperitoneal compartment. Volume of blood to be transfused is calculated depending on the initial fetal hematocrit, donor hematocrit and fetoplacental blood volume. A target hematocrit to be achieved at the end of procedure is 40-50%.

In experienced hands intrauterine transfusion is considered relatively safe with survival rates of about 90% for red cell alloimmunization (9). However the procedure related loss rate is 1-3% & includes preterm labour, premature rupture of membranes, cord hematoma, fetal bradycardia, cardiac overload, infection, fetomaternal bleed and fetal demise.

Post transfusion follow up is done using MCA PSV. Repeat transfusion is usually required in 1-2 weeks and is calculated by a 1% drop of hematocrit per day from the final post transfusion hematocrit or when MCA PSV reaches 1.5 MOM again.

Other Treatment options

1) Plasmapheresis

- There is a transient decline in antibodies after plasmapheresis and decreases the need for intrauterine transfusion by few weeks. It is costly

option, hence used only in early weeks of gestation < 20 weeks when IUT is difficult to perform and the father is homozygous for the offending antigen. It entails a risk of hepatitis, which should be considered seriously.

2) IVIg infusion :

- Maternal intravenous infusion of immunoglobulin (400-500 mg/kg maternal weight every 4 weeks) is proposed to decrease the severity of severe alloimmunisation.
- It is believed that the transfused immunoglobulins bind to the antigen binding sites on the Rh antibody thus rendering them unable to bind to Rh antigens on the fetal RBCs.
- Another option is to directly transfuse the IVIg into the fetal circulation.

Time of delivery

- Fetuses which do not need intrauterine transfusion, are mildly affected and one can wait till fetal maturity and deliver at 38-40 weeks
- Fetuses which require intrauterine transfusion are continued till almost 34 weeks, when the last transfusion is given, and the fetus is delivered at 36-37 weeks.
- Injectable steroids should be administered to hasten pulmonary maturity.

Route of delivery

- The obstetric philosophy need not be altered for the non-anemic transfused fetus.
- Fetuses which need to be delivered remote from term due to fetal anemia may benefit from caesarean section.
- If delivery is by caesarean section, placenta should be allowed to separate spontaneously because manual removal of placenta, increases the risk of fetomaternal hemorrhage
- If vaginal delivery is decided upon, intense intrapartum monitoring is required.
- Typical pattern seen on cardiotocography of a severely anemic fetus is the sinusoidal trace.
- Partogram should be charted, to diagnose and treat labour abnormalities at an earlier stage. Threshold for an operative delivery should be suitably low and prolonged trials are best avoided.
- Oxytocin should be best avoided due to the potential additive effect of oxytocin induced hyperbilirubinemia to that caused by early neonatal hemolysis.
- Second stage of labour should be cut short by applying outlet forceps.
- Cord should be clamped early & should not be milked.
- Cord blood should be collected for testing for Hb, HCT, Bilirubin, ABO Rh typing and DCT
- The cord should be kept long at the fetal end with aseptic precautions in anticipation of a neonatal exchange transfusion.

Care of the newborn

- Prevention of bilirubin encephalopathy- in the intrauterine period the fetal hyperbilirubinemia is cleared by the maternal system. However once delivered, the neonatal hepatic system is overwhelmed with the excessive bilirubin to be metabolized, which may lead to kernicterus due to binding of the free bilirubin to the basal nuclei and other central nervous system tissues.
- Phototherapy followed by double volume exchange transfusion forms the mainstay of treatment for hyperbilirubinemia due to the disease.
- The greater the number of intrauterine transfusions performed, the less severe the hyperbilirubinemia postnatally and the longer the suppression of erythropoeisis postnatally. Post-natal transfusion also plays a very important role in the postnatal management of these fetuses.

Figure 1 : Pathophysiology of isoimmunisation

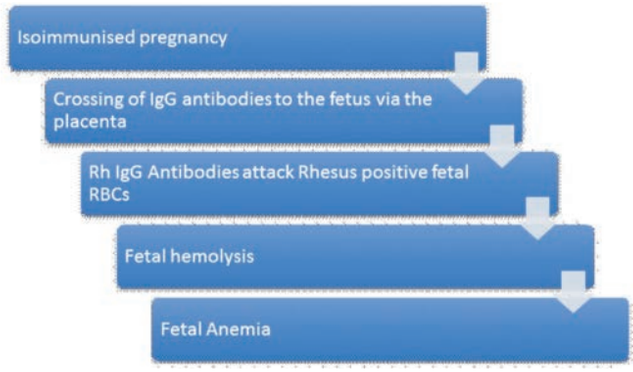


Figure 2 : Fetal & neonatal hemolytic disease clinical presentation

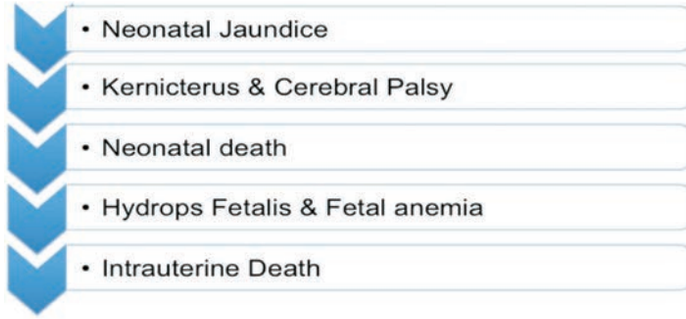


Figure 3 : Rh negative unimmunized pregnancy management

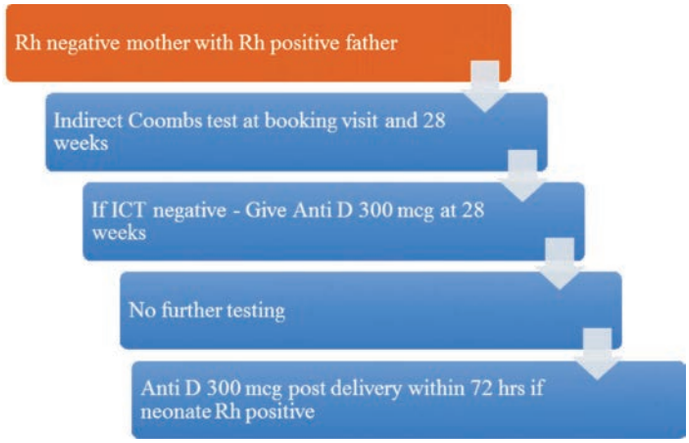


Figure 4: Rh negative immunized pregnancy management

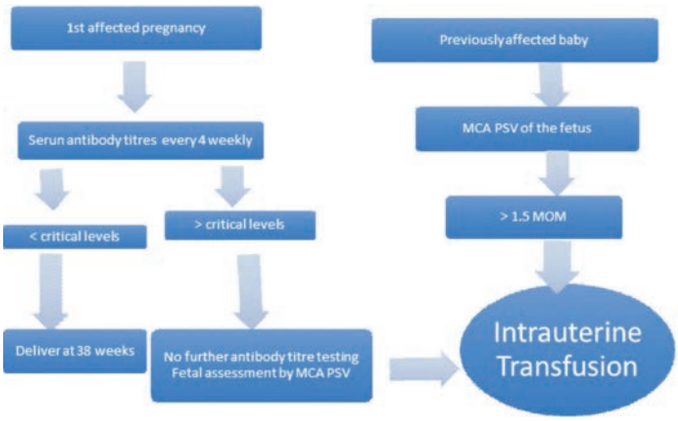


Figure 5: Middle cerebral artery and the Circle of Willis

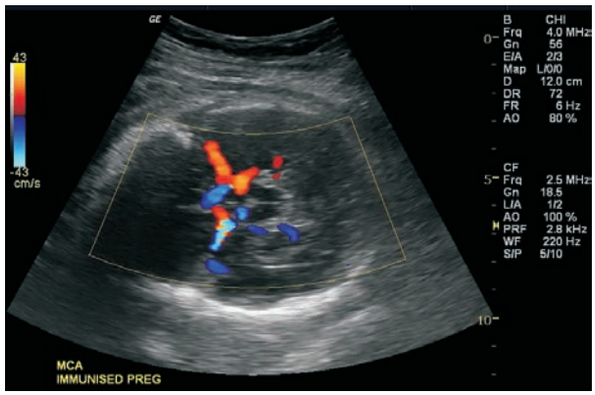


Figure 6: Middle cerebral artery peak systolic velocity

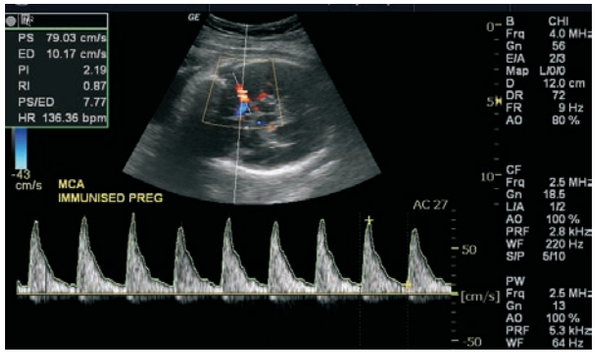
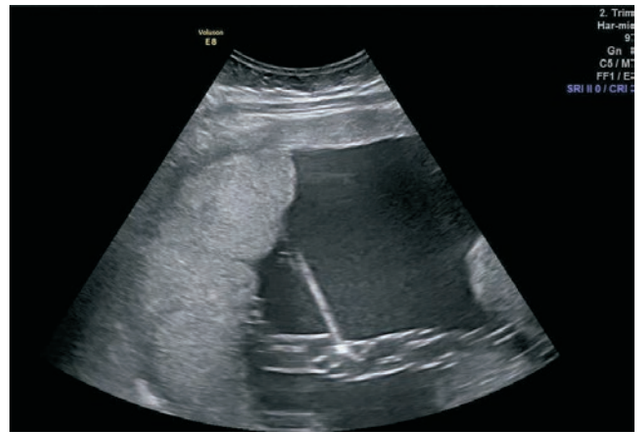


Figure 7 : Intravascular transfusion



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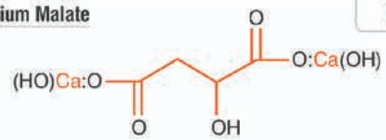
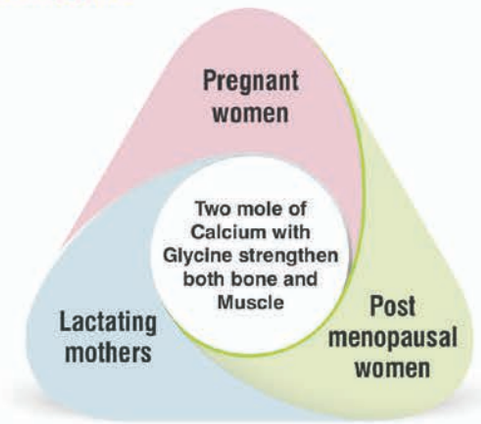


Two mole of Calcium with one mole of Malic acid



Senora
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MTP ACT AMENDMENTS : WHAT SHOULD GYNAECOLOGIST KNOW?

PROF DR. NIKHIL D DATAR (MD DNB FCPS FICOG DGO LLB DHA)
Consultant Gynaecologist Cloudnine Hospital,
Life wave Hospital, Yashada Hospital Mumbai
Visiting professor Maharashtra National Law University

Introduction:

After persistent judicial activism of the author through his case Dr. Nikhil Datar Vs GOI in the apex court for 12 years, the MTP Act has finally been amended. The bill was passed in both the houses and received assent from the president of India.

It must be noted that the amendment is now in force, however the rules have not yet been applied. Hence till such notification is not published by the government, gynaecologists should be careful when doing MTP between 20 to 24 weeks.

What has changed by the amendment?

- Change in the reasons/ indications for MTP

  The only major change in the indications is the removal of the word "married" in the clause that deals with "failure of contraception" as a clause for MTP. This means that gynaecologists can perform MTP even for unmarried women under this clause.

- Change in the limits of gestation for TOP

  The cut off of 20 weeks **remains unchanged for "failure of contraception".**

  MTP can be done upto 24 weeks if there is a serious fetal anomaly or in case of rape survivor. If a woman is widowed or divorced, MTP can be done upto 24 weeks. However, for single women who have not been sexually assaulted, the cut off for MTP remains to be 20 weeks only.

  For the indication "immediately to save the life of a woman", there was no upper limit in the law even in the previous law and that stays the same.

  For substantial anomalies, there will be no upper limit of gestation. But for these anomalies permission from the permanent medical board of the state will be necessary.

- Change in the number of RMPs certifying for MTP

  Till now, for MTP between 12- 20 weeks, certification from two RMPs was necessary. In the new law, this is done away with. Until 20 weeks, only one RMP has to certify. For TOP between 20 to 24 weeks, two RMPs are necessary.

- How will the board decide about the seriousness of the abnormality?

  Permanent medical boards are set up in all the states. There will be a gynaecologist, paediatrician, imaging specialist. Additional specialists may be invited. They will form their opinion and allow such termination.

- What has been the mechanism followed till date?

  The author has pro bono helped more than 200 women to file writ petitions in various high courts and obtained permission to conduct TOP in late gestation.

  The mechanism is set out by the GOI in the affidavit that it had filed in the case of Dr Nikhil Datar Vs GOI". This mechanism is as under:

  - The GOI has instructed all the state governments to establish permanent medical boards.
  - GOI has released a guidance note for the permanent medical boards.

  In this guidance note, the GOI has published an elaborate list of nearly 70 disorders that are classified as "serious abnormalities". This list works as guidance while taking decisions.

- How will TOP beyond 24 weeks be possible in cases of serious foetal anomalies? How to deal with the live birth in such cases?

  The guidance note has clarified that the medical boards are authorised to suggest the use of USG guided injection of intracardiac KCL while dealing with advanced gestation. It was expected that the rules and regulations will further clarify this. However the rules are completely silent on this.

Unresolved/ problematic issues:

- There is no clarity on the definition of the term "termination of pregnancy" in the amendment. The author considers that it is the intention that is a differentiating factor. Medical treatment done with the intent to safeguard the baby and the mother should be classified as "induction of labour". Whereas when the intent is to effect removal/ death of the foetus should be considered as termination of pregnancy.

  If the phrase termination of pregnancy is interpreted as "terminating the pregnant status of the woman" then inductions of labour/ elective caesareans will fall into this category and this would lead to absurdity.
- Arbitrary cut off of 24 weeks for women from special category: there is no clarity as to why there is another arbitrary cut off of 24 weeks. Even after the passage of this amendment, there have been three cases of minor rape survivors who had advanced gestational age and the author approached the court to seek permission. The permission was granted by the court.
- Increased penalty for violating the privacy of the woman:

  Under the amended law, violation of privacy is considered a serious offence for which imprisonment is prescribed. This is a contentious and unclear issue. Current mechanisms such as "Not writing name and identifying her by number" are superfluous and useless in this regard. There is a need to define "persons authorised by law" to whom the RMP can reveal the details.

The author is in the process of filling an elaborate report highlighting fallacies and corrective steps in the apex court and hopes that same are taken positively by the government. This will reduce autocracy, red-tapism and the rights of all stakeholders will be protected in future.

DIET IN PCOS

SHILPA JOSHI RD
Mumbai Diet and Health Centre
National Vice President: Indian Dietetic Association
Secretary: All India Association for Advancing Research in Obesity

PCOS is a condition in women of reproductive age. Usually the symptoms of this problem are irregular periods, signs of androgen excess like facial hair, acne (pimples), loss of hair on the crown of head (male pattern baldness) and polycystic ovaries as a report on ultrasound. Usually the underlying cause of this problem is higher body weight (high BMI) and insulin resistance. Insulin resistance is a condition where the body is less receptive to hormone insulin. Weight loss is usually recommended therapy in PCOS.

Weight loss as defined scientifically is loss of 5% to 10% of initial body weight which is maintained over one year. So, what does this mean in real life. It means that if your initial weight is 100kg, getting to about 90kg is what you need. More importantly once you become 90Kg, you have to maintain this lost weight for at least one year, preferably for a lifetime.

Due to this reason, any drastic weight loss strategies which include fad diets should be avoided. The maximum benefit is seen with slow and sustained weight loss with emphasis laid on maintaining the lost body weight.

Along with weight loss, it's important to see that your diet is nutritionally sufficient. Nutritionally sufficient diets have recommended a number of calories for weight loss along with enough protein to maintain lean muscle, enough good quality fats and rich in micronutrients like vitamins and minerals. It is important that although diets have limited calories, they are enriched with protective nutrients to ensure growth for a young girl with PCOS and good reproductive and metabolic health in older women.

The essence of weight loss is hypocaloric diets..... In simple terms, eat less. So, if you eat less, there will be less energy in your body and hence the body will use fat stored in the body as a source of energy, leading to weight loss. Strategy to eat less will be using a smaller plate to fill in food, using smaller vati/ katori/bowl to fill in foods and using smaller spoons for serving and eating. Another very useful strategy will be to avoid refilling your plate.

Carbohydrates are the most important part of Indian diets. Although it's recommended that only 45-50% calories of diet should come from carbohydrates, Indian diets contain about 60-65% calories from them. This worsens the insulin resistance, which is underlying PCOS. The crux of weight loss would be both choosing the right quantity and quality of carbohydrates. To overcome insulin resistance we need to choose complex carbohydrates like those coming from cereals like wheat , brown rice , millets, pulses , vegetables and fruit. The carbohydrates we need to avoid are those from simple forms like sugar, jaggery, honey, maida and products like bread , biscuits, namkeens, bakery and sabudana.

Strategy here would be to consciously consume whole grain products like home made rotis instead of nans and paratha, brown rice instead of white. Mid meal snacks need fruits instead of refined carbs like namkeens, crisp, bakery foods. While selecting complex carb, eating in limited quantities is very important.

Proteins are lacking in Indian diets is a well known fact. What have we done about it. Proteins are present in dals, pulses, milk and milk products like curd, paneer, nuts and of course all non veg foods. Proteins are our best friends on the weight loss journey! Why? Because they keep us full for a longer time. Besides this, protein helps to keep our muscle mass and in young girls also promotes growth.

Strategy to maintain protein intake would be to include protein in every meal you eat. Whenever you eat, ask a question “where is the protein?”. If you can’t find it on your plate, first get it and then start eating. Vegetarian proteins are lower in fat as compared to non vegetarian proteins, hence lower in calories. So choose vegetarian proteins more often.

Although fats are shown as bad guys, they form an essence of a balanced diet. We need some amount of fats in our diet as they lend food palatability and are also carriers of fat soluble vitamins. Besides that they also provide us with essential fatty acids which are required for proper functioning of the body. The way to use fat is to avoid trans fats which are present in dalda , Vanaspati, ready to eat snacks , bakery foods and saturated fats like those coming from butter, ghee, coconut ,coconut oil and choose oils coming from plants or seeds. Also, prevent overheating of oils which usually happens while deep fat frying and reheating of food repeatedly.

Strategy of good fat intake would be choosing the right kind of oil , cooking fat and using it in the right quantities. Good cooking fats would be groundnut oil, rice bran oil, soya oil, sesame oil., mustard oil and the right quantity would be about 4 teaspoon / person/day. Get even better quality fats by eating nuts like walnuts, almonds, pistachio, groundnuts etc.

Vegetables and fruits are sources of micronutrients and fibre in our diet. Choose about 3 servings of vegetables and 2 servings of fruits per day.

Strategy would be to include plenty of vegetables of different colours in different forms (raw and cooked) in each meal. Include fruit as a snack instead of eating fried foods and processed foods.

So, in short whenever you are eating, use a plate method of healthy eating : ¼ plate of raw veggies, ¼ plate of cooked veggies, ¼ plate of protein and only ¼ plate of carbohydrates. Also, when eating, enjoy the taste, look and aroma of the meal served rather than being busy on TV or phone. Enjoy what you eat!!

BREASTFEEDING PRIMER FOR THE BUSY OBSTETRICIAN

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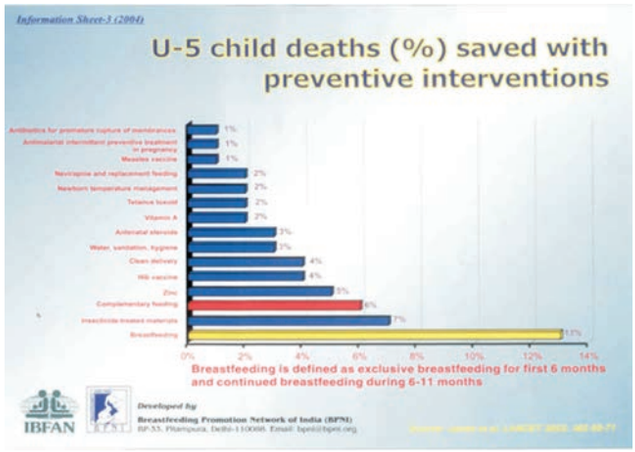
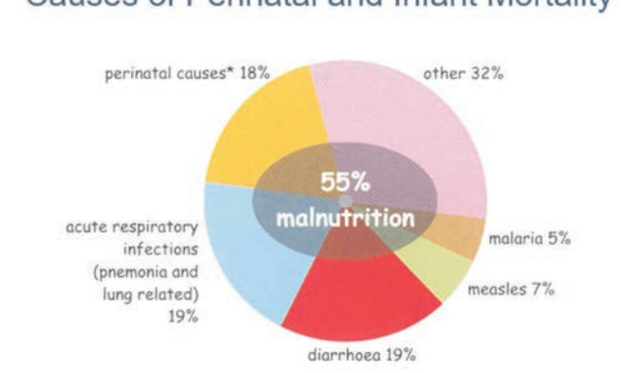
IBCLC
a Lactation Consultant practicing in Mumbai

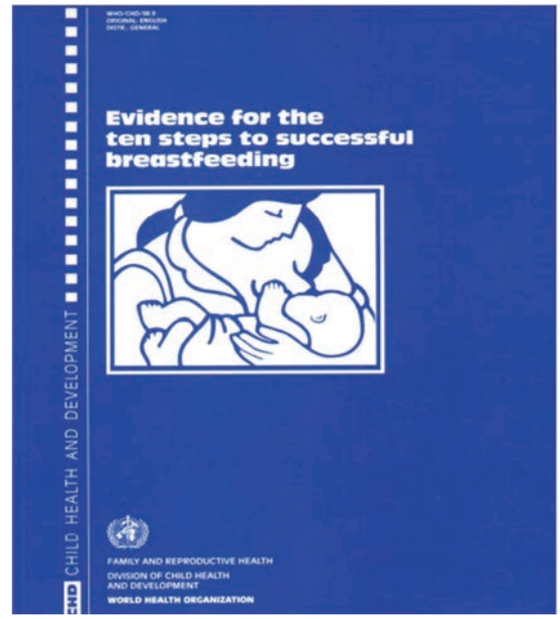
DR. KARTIKEYA BHAGAT
MD, FICOG
an Obstetrician running a Baby Friendly Hospital in Mumbai

1. Breast milk is the best milk for the baby. Breastmilk is species specific and mother's milk is made for human babies. Milk of every mammal has a different composition and the composition of human breastmilk changes with Age of the baby inside & out of the womb.
2. 19% infant deaths could be prevented just by Exclusive Breastfeeding and proper Complementary foods given to each child. Malnutrition is the underlying cause in 55% of all cases of infant mortality.





3. MoHFW, NIHFW, WHO and UNICEF advocate for every newborn
   a. Exclusive Breastfeeding for the first 6 months
   b. Proper home-made complementary foods to be introduced after the baby completes 6 months of age
   c. Continue breastfeeding till 2nd birthday
4. After extensive discussions, UNICEF and WHO came out with a document titled '10 steps to successful Breastfeeding' which summarizes a package of policies that facilities which provide maternity care could implement to support breastfeeding and pointed out the importance of the Maternity Services in promoting Breastfeeding.



5. WHO and UNICEF launched the Baby-friendly Hospital Initiative (BFHI) to motivate facilities providing maternity and new-born care to implement the Ten Steps to Successful Breastfeeding. Implementing each step is important but implementing all the steps has a far greater impact.
6. We now believe:
   a. It is the responsibility of the Maternity Service to look after the baby from the time the baby takes its 1st breath/ cry till lactation is established.
   b. Duration of the pregnancy is 15 months : 9 months in the womb & 6 months of Exclusive Breastfeeding
7. Three agencies are involved in helping the mother to Breastfeed: the **Doctors:** Obstetrician - Paediatrician,

the **Facility** providing Maternity Care and the **Society** (Family and the Government). Every mother will exclusively Breastfeed her child only if all the agencies work together to promote Breastfeeding and help the mother.

8. Role of the Obstetrician is pivotal in promoting Breastfeeding.
   a. He is the captain of the ship of Maternity Service and is in a position to not only implement baby-friendly changes in the functioning of the unit but also influence the mother in making the right choices for the baby.
   b. An Obstetrician will advocate Breastfeeding only when he/ she is convinced about the advantages. Only then will he introduce Baby-friendly practices in the Maternity service and will be able to convince and encourage the mother and the family to Breastfeed
   c. Who will convince the Obstetrician?

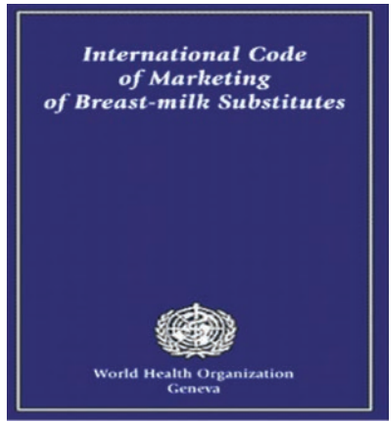
      His peer group: baby-friendly colleagues – Obstetricians and Paediatricians/ Neonatologists, scientific data/ literature, health agencies and workers/ zealots & of course, the patient/ mother

      His peer group: baby-friendly colleagues – Obstetricians/ Paediatricians/ Neonatologists, scientific data/literature, health agencies/ workers / zealots and, of course, the patient/ mother
   d. In the Antenatal Period

      This is the time when the expectant mother is most amenable to suggestions from her treating Physician. S/He is in a position to Educate and Influence not only the mother but the whole family – respecting their varied sensibilities; keeping in mind their educational background, social and religious customs, expectations and the feeding preferences


   e. In the Intranatal Period S/He can adopt Baby friendly practices in the unit
   f. In the Postnatal Period S/He can make arrangements to provide constant support to the mother for Exclusive Breastfeedingnot only Breastfeeding not only during the hospital stay but also after discharge from the facilityIn the Postnatal Period S/He can make arrangements to provide constant support to the mother for Exclusive Breastfeeding not only during the hospital stay but also after discharge from the facility

9. The government policies should be Baby Friendly and implemented in principle and letter. 'Ten Steps to Successful Breastfeeding' and the 'BFHI' are two criteria and the 'IMS Act' an important legislation to help the cause. Laws to enhance Maternity/ Paternity leave and opening of the 'Hirkani Kaksha' (breastfeeding rooms) in public places are welcome steps in this direction.



images –courtesy BPNI Maharashtra

10. Support from the family and endorsement from the society in the form of cultural practices and rituals go a long way to promote and sustain breastfeeding. Reinforcement from the AV media and the social media adds to the strength.
11. Facilities providing Maternity Care should have a baby friendly atmosphere.
    a. It should have a written breastfeeding policy that

is routinely communicated to all health care staff. Information about breastfeeding should be displayed in the common area. (Step 1)

All health care staff should be well trained in skills necessary to implement the policy (Step 2)

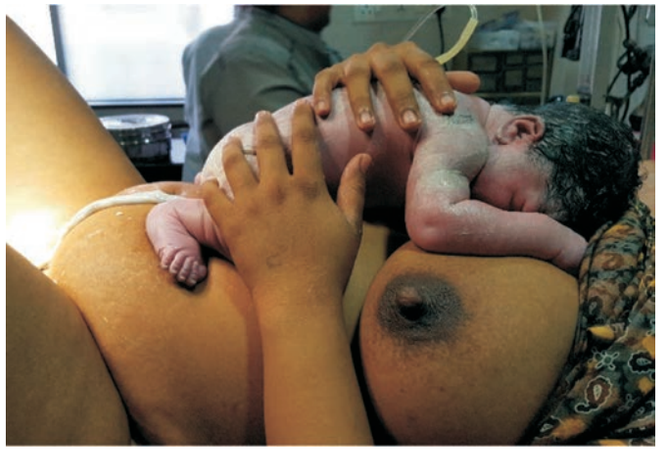
b. Inform all pregnant women about the benefits and management of breastfeeding. (Step 3)

c. Give new-born infants no food or drink other than breast milk, unless medically indicated. (Step 6)

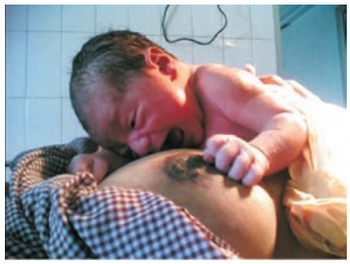
d. Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants. (Step 9)

e. Baby Friendly Birthing practices

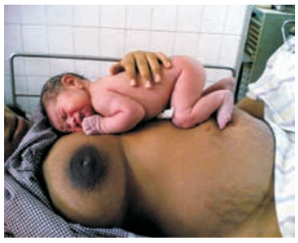
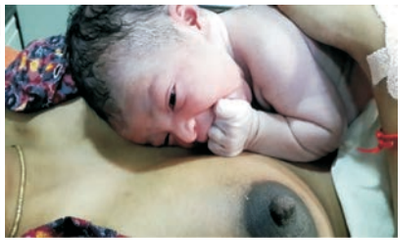
I. Delivering the baby on mother's abdomen, delayed cord clamping/ cutting



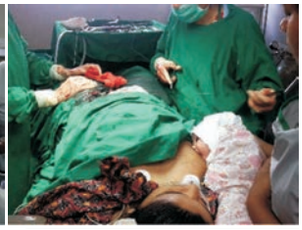
ii. Immediate cheek-to-cheek and skin-to-skin contact

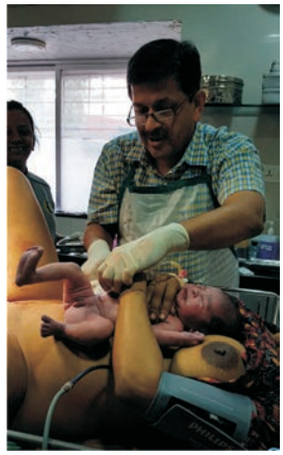
iii. Breast Crawl

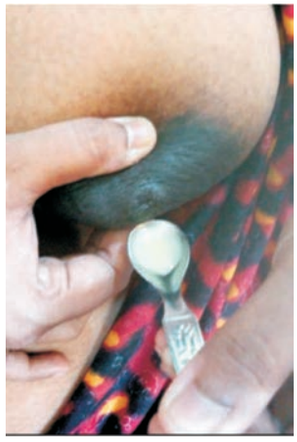
iv. Not breaking the skin-to-skin contact while suturing episiotomy & during Caesarean Section

v. Neonatologist can examine the baby on mother's abdomen without breaking the skin-to-skin contact



vi. If the baby cannot be brought to the mother for the 1st breastfeed, colostrum could be expressed, collected and fed to the baby with a sterile spoon or a syringe




vii. Delay Injection Vit K and other routines

viii. No baby bath or oil massage

ix. Help mothers initiate breastfeeding within one hour of birth. (Step 4)

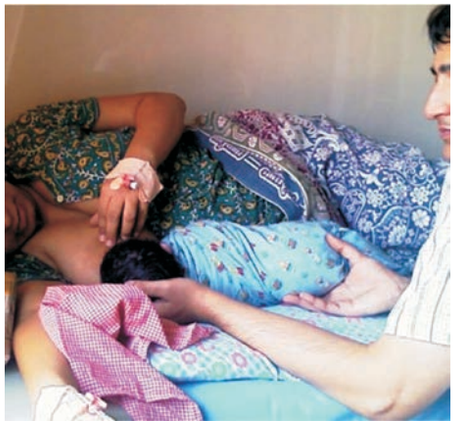
f. Baby Friendly practices in the wards

i. 'Bedding in' rather than 'Rooming in' (Step 7)

ii. Kangaroo care

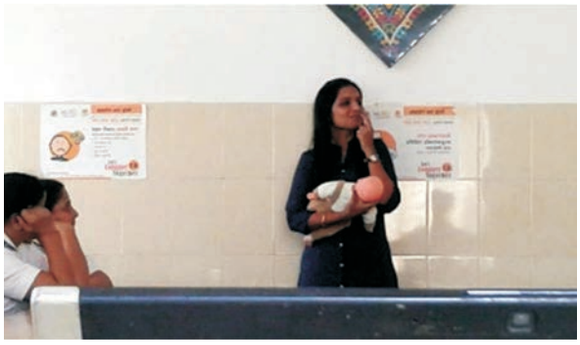
iii. Proactive frequent feeding initially followed by breastfeeding on demand (Step 8), encourage non-nutritive suckling

iv. Involvement of the husband



v. Minimise visitors: a honeymoon period for the family!

vi. Show mothers how to breastfeed, and how to maintain lactation even if they should be separated from their infants. (Step 5)



g. After discharge from the Maternity unit

i. The baby is to be followed up to check weight gain, urine output and color (neonatal jaundice) regularly till breasfeeding is established. Neonatology reference as required. Maintain Growth Charts.

ii. Address breastfeeding problems in the mother – reinforce knowledge/ technique for correct position/ attachment of the baby and expression of engorged breast

iii. Take help of Lactation Counsellors and Consultants (bpnimaharashtra.org) and use the shishuposhan app

iv. Introduction to the Mother Support Group

12. Setting up a babyfriendly maternity service is very much possible if the Obstetrician is convinced about the advantages and is helped/ backed by an equally enthusiastic and sincere Paediatrician. The project can't take off without the involvement of the support staff (nurses and ayahbais). Once the staff nurses understand the importance of exclusive breastfeeding, their support is assured.

13. We dream of a world where all maternity services are babyfriendly and every mother will exclusively breastfeed her baby which will help her achieve her optimal potential.

14. All we need to do is look at the animal world and learn:

Animals are sensible..... **Are we??????**

PROGESTERONE IN PREVENTION OF PRETERM BIRTH - AN OVERVIEW

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Masters In ART (UK)
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Preterm birth is defined as the delivery before completion of 37 weeks gestation. Preterm deliveries usually culminate in one of the following complications-neonatal mortality and morbidities or long term sequelae such as cerebral palsy and developmental disability. Hence a preventive approach must be adopted.

Preventive approach includes timely identification of causative factors in at risk patients.

Preterm births are multifactorial in origin and the main risk factors include previous history of PTB and short cervical length. Relatively less predictive risk factors include – multiple pregnancy, smoking, uterine anomaly and history of curettage and cervical conization. Once the risk factors have been identified, the treatment includes timely administration both a specific approach as well as supportive treatment

Among the supportive treatments, Progesterone therapy is one of the few proven effective methods to prevent PTB in high risk women.

Progesterone is known to act in three ways One of the major mechanisms of progesterone action in maintaining pregnancy is inhibition of the contractions of the myometrium: research has demonstrated the relaxant effect of progesterone on myometrial strips in vitro[1] Similarly in the endometrium it reduces the production of local PG , with a resultant reduction in uterine irritability . It also helps to maintain the cervical length and its firm consistency.

In 2003, 2 randomized, double-blind, placebo-controlled trials demonstrated that progesterone supplement therapy can prevent PTB in women with past history of PTB. Many following studies were carried out to add evidences about prevention of PTB through progesterone supplement therapy, and now the American Congress of Obstetricians and Gynecologists (ACOG) and the Society for Maternal-Fetal Medicine (SMFM) recommend the usage of progesterone to prevent PTB in certain pregnant women - those with history of spontaneous PTB, such as preterm labour and premature rupture of membranes, and those with short CL during the mid-trimester[2].

The most recent Cochrane review on progesterone for the prevention of preterm birth (published in 2013) examined evidence on the use of any progestogen for women at risk of preterm birth either because of a previous preterm birth or because of a short cervix.[7] Although the two risk categories were examined separately, all progestogens were considered together. For women with a previous preterm birth, the review suggested that progestogens reduce the risk of preterm birth before 34 gestational weeks or women with cervical shortening the review found that, among the above outcomes, only preterm birth before 34 weeks was reduced by progestogen.[3]

Type, routes, dose, and interval of administration

Type	Route	Dose	Interval
17 alpha hydroxy progesterone caproate	Intramuscular	250mg	weekly
Natural micronized progesterone	Vaginal suppository	100,200, 400mg	Daily
	Vaginal gel	90mg	Daily
	Oral capsule	100,200, 400mg	Daily

It has not been fully elucidated whether which progesterone therapy is better with regard to the efficacy of preventing PTB, cost-effectiveness, or side effects. Choice of drug would depend upon the availability, affordability, practitioner's preference and patient's choice. However as far as route of administration is concerned, Vaginal progesterone

decreases the risk of preterm birth and improves perinatal outcomes in singleton gestations with amid trimester sonographic short cervix, without any demonstrable deleterious effects on childhood neurodevelopment.[4]

Indications for Usage

Pregnancies likely to benefit from progesterone supplementation

- In women who have had a previous spontaneous preterm singleton birth and
- In women with a short cervix on ultrasound examination in the current pregnancy.

Pregnancies where the benefit of progesterone supplementation is unclear

The benefit of progesterone supplementation in women at high risk of preterm birth, but without a short cervix or a prior history of singleton spontaneous preterm birth, is not supported by strong evidence.

- **Singleton pregnancy with spontaneous twin preterm birth in prior pregnancy**

  A prior spontaneous preterm birth is a risk factor for a subsequent spontaneous preterm birth whether the initial preterm delivery was a singleton or a twin pregnancy [5,6]. No study has specifically evaluated whether progesterone supplementation decreases the risk of a preterm birth of a singleton after a previous spontaneous preterm birth of twins, but a benefit is plausible.

- **After cerclage**

  In women with short cervix without prior history of spontaneous preterm, if cerclage is performed for short cervix, adjuvant treatment with progesterone has not shown any significant benefit. However, in practise many obstetricians do prefer to supplement these women with vaginal progesterone.

- **After preterm prelabour rupture of membranes**

  Beginning progesterone supplementation is not beneficial in women who develop preterm prelabour rupture of membranes (PPROM) in the current pregnancy.

  By contrast, women with a history of preterm birth due to PPROM appear to benefit from progesterone supplementation in subsequent pregnancies (7)

- **Maintenance therapy after threatened preterm labour**

  Use of progesterone in women who remain undelivered after an episode of threatened preterm labour is investigational, although most practitioners routinely prescribe progesterone supplementation for maintenance tocolysis(8)

- **Multiple gestation**

  Progesterone is not effective in **unselected** multiple gestations. One reason may be that the pathogenesis of preterm labour and delivery in multiples is different from that in singletons and less impacted by changes in progesterone.(9)

- **Uterine anomaly or assisted reproductive technology**

  Women with some uterine anomalies and those who conceive with assisted reproductive technology appear to be at increased risk of preterm birth. The effectiveness of progesterone therapy for prevention of spontaneous preterm birth in these women is unknown

Recommendations for progesterone supplementation to prevent preterm birth [10]:

INDICATION	Progesterone supplementation indicated?	Management
Singleton pregnancy Prior spontaneous PTB Normal cervical length	Yes	Hydroxyprogesterone caproate 250 mg intramuscularly weekly beginning between 16 and 20 weeks of gestation and continuing through 36 weeks of gestation or until delivery and monitor cervical length. Natural micronized progesterone administered vaginally is a reasonable alternative. Short (≤ 25 mm) cervix = consider performing cerclage

INDICATION	Progesterone supplementation indicated?	Management
Singleton pregnancy, prior spontaneous twin PTB Normal cervical length	Possibly	Hydroxyprogesterone caproate 250 mg intramuscularly weekly beginning between 16 and 20 weeks of gestation and continuing through 36 weeks of gestation or until delivery and monitor cervical length. Natural micronised progesterone administered vaginally is a reasonable alternative. Short (≤ 25 mm) cervix = consider performing cerclage
Singleton pregnancy No prior spontaneous PTB Short cervix (≤ 20 mm)	Yes	Micronised Progesterone 200 mg vaginally each night from time of diagnosis through 36 weeks of gestation. Other options include 8 percent vaginal gel containing 90 mg micronized progesterone per dose.
Multiple pregnancy (twins or triplets) without prior PTB Normal cervical length	No	No Progesterone , No Cerclage
Twins, prior PTB	Possibly	Hydroxyprogesterone caproate 250 mg intramuscularly weekly beginning between 16 and 20 weeks of gestation and continuing through 36 weeks of gestation or until delivery and monitor cervical length. Natural micronized progesterone administered vaginally is a reasonable alternative
Twins, short cervix	Possibly	Vaginal progesterone, no cerclage
Preterm premature rupture of membranes	No	
Undelivered after an episode of preterm labour	No	

Summary and recommendations:

- Progesterone supplement therapy is effective in prevention of PTB. However, its efficacy varies depending on the indication and type, administration route, and dose of progesterone.
- For singleton pregnant women with history of spontaneous PTB, including preterm labour and premature rupture of membranes, weekly injection of 250 mg of 17α-OHPC, as well as daily administration of vaginal micronized progesterone suppository (100 or 200 mg) are effective in preventing recurrent PTB, but the preventative effects of vaginal progesterone gel or oral progesterone capsules currently lack evidence.
- For singleton pregnant women with CL <25 mm during mid trimester, daily administration of vaginal micronized progesterone suppository (100 or 200 mg) or gel (90 mg every day) is effective in preventing PTB,

but the preventative effect of 17α-OHPC therapy lack evidence.

- In women with twin pregnancy, an injection of 17α-OHPC nor an administration of vaginal micronized progesterone suppository or gel could prevent PTB. Yet, for twin pregnant women with short CL, vaginal progesterone supplement therapy may be effective for reducing the rate of PTB and improving the neonatal outcome.
- As a maintenance therapy after the inhibition of preterm labour, 17α-OHPC cannot prevent PTB but can extend the gestational age and increase the birth weight. Both vaginal and oral micronized progesterone treatment can prevent PTB <37 weeks of gestation, extend the gestational age, and increase the birth weight. Yet the exact role of progesterone as a maintenance therapy after the inhibition of preterm labour remains much to be discovered.
- In cases of premature rupture of membranes, there lacks evidence on the effect of progesterone supplement therapy in preventing PTB.
- The progesterone supplement therapy generally begins at 16 to 24 weeks of gestation and ends at 34 to 36 weeks of gestation.
- No evidence currently exists on which progesterone supplement therapy can maximize the preventative effects while minimizing the side effects

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CARBETOCIN : An Oxytocin Analogue

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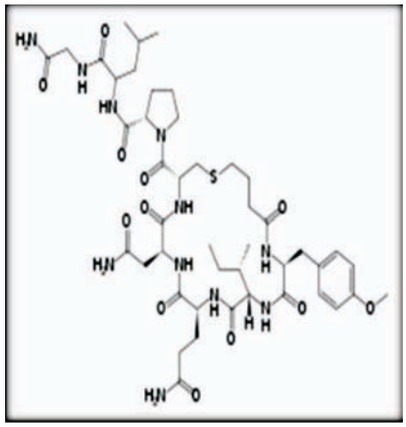
- Consultant at Ashish Maternity and Nursing Home, Vile Parle East
- IVF Consultant at ProFert IVF Clinic, Kandivali (East)
- Shan S Ratnam Young Gynecologist Awardee (YGA) 2019- AOFOG
- MOGS Youth Council Member



- First described in 1974
- Carbetocin is a long-acting synthetic oxytocin analogue 1-deamino-1-monocarbo-(2-O-Methyltyrosine)-oxytocin
- The changes made to the molecule extend the pharmacological action of carbetocin by:[1]
  - Reducing enzymatic degradation
  - Prolonging the half-life of the peptide
- While half-life of both, endogenous and synthetic oxytocin is 3-5 minutes, carbetocin is 10-15 times longer acting than oxytocin-Half-life:85-100 minutes[1]

Indication :

- Prevention of uterine haemorrhage due to postpartum uterine Atony.
- Probable scope of use in Myomectomy

Dosage :

- 100 g Carbetocin I.V slowly over 1 min.
- Alternatively 100 µg may also be administered I.M

Compared with oxytocin, carbetocin:

- Significantly reduces the need for uterotonic interventions in caesarean section[2-4]
- Significantly increases the time before further uterotonics are required during caesarean section2
- Significantly reduces the need for uterine massage in caesarean and vaginal deliveries3,5
- Is associated with a higher incidence of blood loss ≤ 500 mL3

Carbetocin is available in a room temperature-stable formulation and convenient vial preparation

- Oxytocin and ergot alkaloids should be kept in a refrigerator to avoid degradation. Heat-stable formulation of oxytocin is currently not available. Previous formulation of carbetocin were to be stored between 2^0C and 8°C[5]. Resource-poor countries do not always have cold-chain transport and storage capabilities[6]
- New formulation of carbetocin is stable at room temperature. It has shelf-life of 24 months at 30oC and 75% humidity. Avoids need for cold-chain storage and logistics and is provided in a vial.

Mechanism Of Action

- Agonistic Action at Peripheral oxytocin receptors, particularly in the myometrium
- Carbetocin has much longer lasting effect than oxytocin, thus necessitating a single dose
- Oxytocin displays high structural similarity to vasopression6, leading to water retention, water intoxication & hyponatremia 7
- In contrast, carbetocin has a minor antidiuretic effect8 (vasopressor activity: <0.025 I.E./vial)
- Thus, reducing the chances of water intoxication and hyponatremia

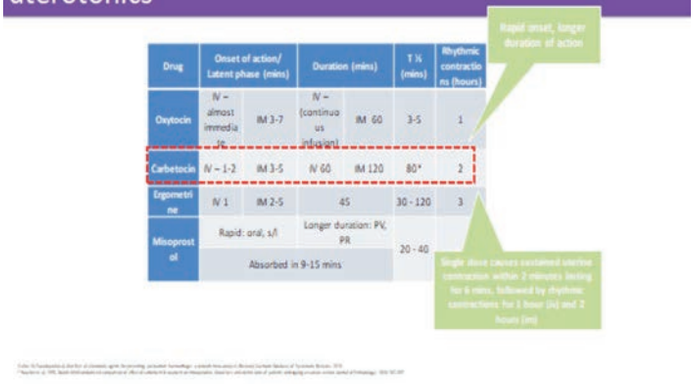
Approved role of carbetocin

Indication: Carbetocin is currently approved for "Prevention of PPH" due to uterine atony, in vaginal delivery or cesarean section

- Dose: 100 mcg in 1 ml solution (1 vial) i.m. or i.v. administered slowly over 1 minute
- Carbetocin must only be administered after delivery of the infant, and as soon as possible after delivery, preferably before the delivery of the placenta
- Carbetocin is intended for Single administration only.

- Heat-stable carbetocin will now be available for use in heat-prone countries like India to overcome drawbacks such as loss of efficacy, as with other traditional uterotonics
- Being a longer acting agonist, it is **not** to be used in pregnancy & in labour before delivery of infant

Pharmacokinetics of Carbetocin compared to other uterotonics



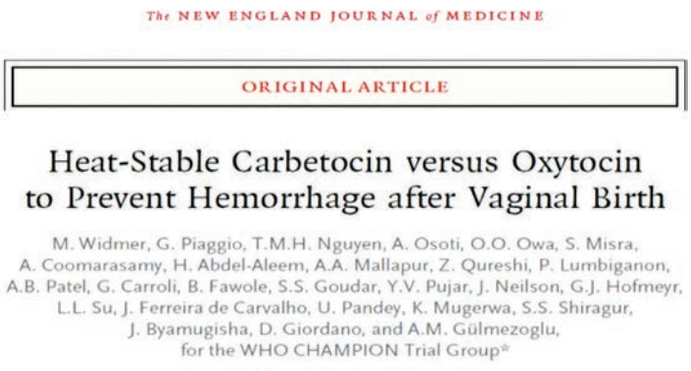
Drug	Onset of action/ Latent phase (mins)		Duration (mins)		T ½ (mins)	Rhythmic contractions (hours)
Oxytocin	IV – almost immediate	IM 3-7	IV – (continuous infusion)	IM 60	3-5	1
Carbetocin	IV – 1-2	IM 3-5	IV 60	IM 120	80*	2
Ergometrine	IV 1	IM 2-5	45		30 - 120	3
Misoprostol	Rapid: oral, s/l		Longer duration: PV, PR		20 - 40	
	Absorbed in 9-15 mins					

Rapid onset, longer duration of action

Single dose causes sustained uterine contraction within 2 minutes lasting for 6 mins, followed by rhythmic contractions for 1 hour (IV) and 2 hours (IM)

CHAMPION Trial

Carbetocin HAemorrhage PreventION Trial



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Heat-Stable Carbetocin versus Oxytocin to Prevent Hemorrhage after Vaginal Birth

M. Widmer, G. Piaggio, T.M.H. Nguyen, A. Osoti, O.O. Owa, S. Misra, A. Coomarasamy, H. Abdel-Aleem, A.A. Mallapur, Z. Qureshi, P. Lumbiganon, A.B. Patel, G. Carroli, B. Fawole, S.S. Goudar, Y.V. Pujar, J. Neilson, G.J. Hofmeyr, L.L. Su, J. Ferreira de Carvalho, U. Pandey, K. Mugerwa, S.S. Shiragur, J. Byamugisha, D. Giordano, and A.M. Gülmezoglu, for the WHO CHAMPION Trial Group*

- Largest Double-Blind RCT: 29,645 women from 10 countries enrolled (>7000 from India)
- In collaboration with WHO, heat-stable version of carbetocin (100 mcg I.M) was compared with oxytocin (10 IU I.M) for prevention of PPH after vaginal birth
- The frequency of blood loss of atleast 500 ml or the use of additional uterotonic agent was 14.5% in Carbetocin group and 14.5% in oxytocin group
- Conclusion : Heat stable carbetocin is non inferior to oxytocin for prevention of blood loss of at least 500 ml.

However...

- Oxytocin being heat-labile, both drugs in the trial were kept in cold storage (2 to 8°C) to maintain double-blinding and to retain the efficacy of oxytocin
- Evidence in real-world may differ due to proven reduction in efficacy of heat-labile oxytocin as compared to heat-stable carbetocin in transportation and clinical settings
- Practically observed efficacy of heat-stable carbetocin may, therefore, be better!

Key Take-home message

- Carbetocin is a longer acting analogue of oxytocin indicated for prevention of PPH
- Efficacy & side-effect profile similar to oxytocin with key advantages of,
  - Less activity at Vasopressin receptors, therefore, less chances of water intoxication
  - Reduced need for additional uterotonics and additional interventions
- Amongst top 3 uterotonics for,
  - Prevention of minor & major PPH
  - Reduced uterotonics, reduced blood loss & change in Hb
- Heat-stable carbetocin holds promise for middle-income countries like India where lacunae in maintenance of cold-chain have been demonstrated, both, in transportation & storage

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BUTTERFLIES & BUTTERLY GARDENING

LORNA OZA
Butterfly Farming

Butterflies are some of our most loved and most recognisable insects. Many people create flower gardens specifically to attract the colourful butterflies. Butterflies are great for your garden as they are attracted to bright flowers and need to feed on them for nectar. When they do this, their bodies collect pollen and carry it to other plants. This helps pollination in fruits, vegetables and flowers to produce new seeds.

Butterfly gardening is a way to create, improve, and maintain habitat for butterflies. Butterflies are flying insects with large scaly wings. Like all insects, they have six jointed legs and three body parts: the head, the thorax and the abdomen. The wings are attached to the thorax and they also have a pair of antennae, compound eyes and an exoskeleton. Butterflies have four distinct life stages—egg, larva, chrysalis, and adult.

Eggs

Caterpillers

Chrysalis

Picture below shows lifecycle of Common Mormon Butterfly.

Adult

The life cycle starts with the egg. When the caterpillar comes out of the egg, it first eats the egg shell. Then it eats the leaves of the host plant and keeps on growing bigger in size and then enters Pupa or Chrysalis stage. Finally, an adult butterfly emerges from the chrysalis. It then hangs on to the empty chrysalis or the plant branch to dry its wings for some time and then flies away. The life span of the butterflies is very short, couple of weeks to a few months.

Host plants are plant that adult butterflies depend upon to raise their larval young. Female butterflies lay their eggs directly onto their host plant of choice & the caterpillars feed on the plant leaves. Host plants are species specific. Host plants are to be planted in accordance with the butterfly

species existing in the particular geographic location. This butterfly availability can be checked by capturing pictures of butterflies visiting your locality, in parks, nurseries or wherever you spot them on your morning walk and identifying them. Various forums on the web are available to get the butterflies identified. Most host and nectar plants are available in local nurseries.

With any ecosystem, diversity is key. The more complex and diverse the garden landscape is, the more likely beneficial insects will call it home. Any garden has lizards, frogs, wasps, bugs and a lot of spiders, all beneficial to the garden. They keep the population of unwanted pests and butterflies in control. Butterûies and other pollinators including bees, moths, birds, and bats pollinate over 75% of the world's ûowering plants. We depend on what these pollinators do to help plants reproduce which helps strengthen ecosystems and maintain life sustaining biological diversity in nature. With fall in host plants numbers, the Butterflies tend to migrate to other areas in search of host plants. All Gardeners should include at least some Butterfly host and nectar plants in their gardens. This will help in increasing the Butterfly population considerably.

Initially a butterfly garden can be started by planting limited number of host and nectar plants. Plants can be added gradually as per availability of space and traffic of visiting Butterfly species. Below is a list of host plants for some common species of butterflies found in Mumbai.

Host Plants:

Host plants can be planted in semi-shade or shady areas too, in the near vicinity of the nectar plants. Frequent trimming of host plants is required, which enhances growth of new leaves on which the butterflies lay their eggs. Trimming also makes the plant dense. Monitoring of Butterfly lifecycles also becomes easy if the height of the plant is kept short. Host plants should be planted or placed close to each other as seen in Miyawaki method. The Butterflies love a dense forest type habitat.

List of host plants & corresponding butterfly name:

1. Bael. Blue Mormon/Common Mormon/Lime Butterfly.
2. Passiflora vine. Tawny Coster
3. Turmeric . Grass Demon
4. Ixora. Monkey Puzzle
5. Bahava. Common Emigrant.
6. Badak Vel. (Aristolochia)Common Rose.
7. Son Chafa. Common Jay
8. Mussaenda. Commander
9. Bryophyllum. Red Pierrot
10. Kalaonche. Red Pierrot
11. Plumbago. Zebra Blue.
12. Lemon. Common Mormon & lime
13. Kadipatta. Common Mormon & lime
14. Powder Puff. Common Grass Yellow and Three Spot Grass Yellow Butterflies.
15. Oleander khaner. Common Crow.
16. Sankasur. Common Grass Yellow.
17. Mango. Baron, Monkey Puzzle
18. Dates. Palm fly / Indian Palmbob
19. Ginger lillie (Son takka) Grass Demon
20. Spider Lily. Spider Lily Moth.
21. Crotalaria. The Tiger and Crows Butterflies congregate on this plant.
22. Senna Occidentalis, Coffee senna.
23. Cinnamomum verum. Common Mime, Tailed Jay and Narrow-Banded Bluebottle.

Nectar plants:

They have to be planted in sunny locations, where maximum sunlight is available most times of the day. A cluster of single coloured flowers, attracts more attention of Butterflies. e.g. a patch of 4 X 8 feet of yellow colour lantana flower will attract a lot of Butterflies throughout the day. Similarly a large patch of Stachytarpheta plants will attract a large number of Butterflies, bees, waps and sunbirds. Subsequently more nectar plants can be added as per availability of space. The Pagoda and Ixora plants are a good choice as they have multiple flower bunches. Large butterflies get engaged nectaring on these flowers for a long amount of time.

List of Nectar Plants.

1. Ixora
2. Bleeding heart.
3. Mussaenda.
4. Wedelia Trilobata.
5. Lantana Camara.
6. Penta.
7. Allamenda.
8. Tecoma.
9. Rangoon Creeper
10. Powder Puff.
11. Pagoda plant.
12. Hamelia Patterns
13. Mirabilis (four o'clock plant)
14. Scarlett Milkweed.
15. Jethropa.
16. Ruillia.
17. Yellow Alder.
18. Periwinkle
19. Bougainvillea
20. Ravenia Spectibillis.
21. Jamaican Spike.

Some Nectar Plants.

Below is a list of common butterflies in the city of Mumbai whose lifecycles are fun to watch and record.

1. Common Rose. Aristolochia littoralis.
2. Common Crow. Asclepias curassavica / Nerium Oleander.
3. Grass Demon. Ginger lily/ Turmeric.
4. Commander. Mussaenda.
5. Mottled Emigrant. Bahava / Senna Occidentalis.
6. Lime Blue. Lemon plant.
7. Indian Palmbob. Dates / Cocos nucifera.
8. Common Castor. Castor plant.
9. Tawny Coster. Passiflora.
10. Lime Swallowtail. Butterfly. Lemon / Curry leaves.
11. Common Palmfly. Cocos nucifera.
12. Plain Tiger. Asclepias curassavica.
13. Common Mormon. Lemon / Curry leaves / Bael.
14. Red Pierrot. Kalanchoe / Bryophyllum.
15. Rice Swift. Garden grass.
16. Common Grass Yellow. Pithecellobium dulce.

Butterfly fruit bait:

Butterfly fruit bait or Butterfly fruit feeder.

Most Butterflies live on flower nectar. Some species of Butterflies feed exclusively on overripe rotten fruits alone. Butterflies are particularly fond of rotten Banana, Papaya and Pineapple. Adding a Butterfly fruit feeder to your garden would be a great idea, and would increase the Butterfly traffic. You can expect the Baron, Blue Oakleaf, Castor, Great Eggfly, Gray Pansy, Indian Palmfly, Common Evening Brown, Black Rajahs, Nawabs etc. to visit the feeder frequently. I have made a butterfly food bait container. Made out of a steel perforated pen stand and a rubber funnel as cover. 14 SWG GI wire used to anchor the bait box. I have also made one more feeder using a plastic pen stand. Containers are filled with rotten overripe banana. A lot of Butterflies used to keep visiting the bait throughout the day last year. Planning to include the bait, in the garden again. Seen in the collage are visiting butterflies and the two fruit baits installed in my garden.

Ecosystem value :

Butterflies are indicators of a healthy environment and healthy ecosystems . They indicate presence of a wide range of other invertebrates, which comprise over two -thirds of all s pecies. Areas rich in butterflies are rich in other invertebrates. These collectively provide a w ide range of environmental benefits, including pollination and natural pest control. Butterflies are an important element of the food chain and are prey for birds, bats and other insectivorous animals. Butterflies support a range of other predators and parasites, many of which are specific to individual species, or groups of species. Butterflies have been w idely used by ecologists as model organisms to study the impact of habitat loss and fragmentation, and climate change.

Health value :

People enjoy seeing butterflies both around their homes and in the countryside.

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OOCYTE BANKING – A quick recap

Shreya Agrawal Jain
(M Sc. Clinical Embryology – Manipal University)
Chief Embryologist at Aarush IVF centre, Malad and Mira road.
Merck and ASSET trainer for ICSI, Vitrification
-Thawing and Advanced Andrology

Cryopreservation of oocytes is a process wherein the oocytes are extracted, frozen and stored for later use when they have to be thawed, fertilized and transferred back into the uterus. To get good number of oocytes, woman has to undergo controlled ovarian hyper stimulation and ovum pick up procedure is done as in any ART cycle.

CLINICAL APLICATIONS:

1) **Oocyte donation programme** is offered primarily with fresh oocytes with disadvantages of
   - Complexity in synchronization between donor and recipient
   - No quarantine period (HIV and other infectious agents)

With cryopreserved donor oocyte bank we can provide wide selection of donors with desired phenotypic characteristics from catalogue, availability of rare donor, convenience of starting the IVF (since no synchronization would be needed), ability to repeat infection screen on the egg donor after 6 months (quarantine period) making oocyte donation programme safer and easier.

2) **Fertility preservation in Cancer patients:**

Recently attention has been focused on preservation of fertility and reproductive needs of cancer survivors. Toxicity of treatment modalities in cancer patients are as follows:

- Chemotherapy has mutagenic effects which are drug and dose dependant .The gonadotoxic effects also depend on the patient's age and ovarian reserve.
- Radiotherapy induced ovarian damage depends on Irradiation area (total) and direction of beam (abdominal, pelvic), dose, patient's age & ovarian reserve (AMH) at time of exposure.
- Combination chemo-radiotherapy leads to 100% premature ovarian failure (POF).

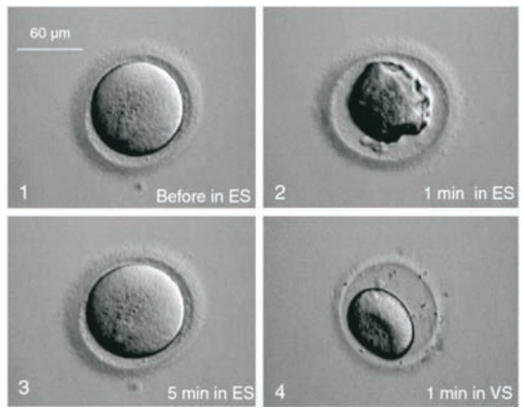
3) Healthy women without medical indications:

Oocyte banking has social reasons (career, military combat roles, extra eggs not intended to be inseminated with present partner as embryos are jointly owned by both partners). Legal and ethical issues regarding embryo and oocyte freezing and banking can be considered.

4) Different clinical situations in ART:

- Oocyte and embryo cryopreservation may increase the flexibility of the options offered by current ART strategies especially in cases where the delaying in embryo transfer is mandatory due to the risk of OHSS, low response to gonadotropins, or failure to obtain sperm on the day of OPU.
- Women at risk of losing reproductive function due to premature ovarian failure can resort to oocyte banking to preserve fertility.

Oocytes are frozen by the process of vitrification – ultra rapid cooling. Cryopreserved (vitrified) oocytes are stored in liquid nitrogen at -196° C. ICSI is performed for all frozen thawed oocytes.



The morphological changes of human oocytes in equilibration solution (ES) and vitrification solution (VS) during the process of vitrification

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- Improve oocyte & embryo quality[2,3,4,9]
- Improve ovarian function in PCOS patients[1]

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