PUNE OB-GYN SOCIETY
ESTD. 1953

Womb & Wellness

POGS FOR HER
स्त्रिया: हिताय प्रवतमान:
Striving For The Betterment Of Woman

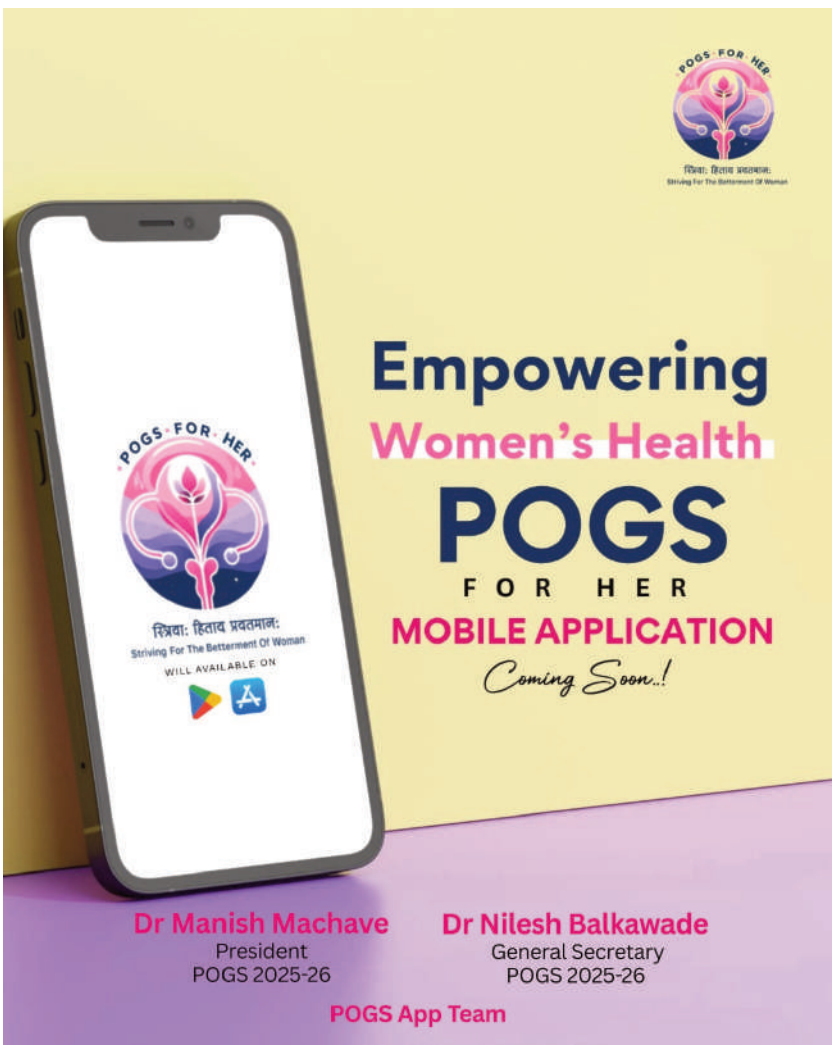
The POGS Chronicle ♦ Issue 1, April 2025

POGS App

On the auspicious occasion of Gudi Padwa, we are thrilled to announce the launch of the brand-new POGS App, set to debut at our 40th POGS Installation CME!
For the very first time, POGS is bringing you a state-of-the-art mobile application available on both Android and iOS. This app is designed to centralize all POGS-related information, making it easier than ever to stay connected and engaged.
Overview:
• Seamless New Member Registration: Join our community with just a few taps.
• Easy Conference Registration: Book your spots for upcoming events right at your fingertips.
• Monthly Quiz: Test your knowledge and win exciting prizes!
• Digital Library: Access monthly newsletters, a video library, and recordings of past conference lectures

Get ready to experience the convenience and innovation of the POGS App. Stay tuned and be prepared to take your POGS experience to the next level!

Dr Manish Machave
President POGS 2025-26
Dr Nilesh Balkawade
General Secretary
POGS 2025-26
POGS App Team
Dr Mahima Lalwani
Dr Mrinmayee Dharmadhikari



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Presidential Address

Dr Manish Machave
President, POGS

Respected seniors, past presidents, esteemed colleagues and dear members of POGS,
I begin with enriching words, "Ignorance is the curse of God; knowledge is the wing wherewith we fly to heaven." William Shakespeare.
And the best way to gain such wings is though the path shown by our teachers and gurus and the experience of scholars. It is with immense gratitude and humility that I accept the responsibility and honor of serving as the President of the Pune Obstetric and Gynecological Society. Standing here today, I am deeply aware of the legacy of excellence, innovation, and dedication that POGS represents. I am also reminded of the many stalwarts who have led this organization before me—each one leaving behind footprints of inspiration and huge loads of responsibility. I am here today not because of individual merit alone, but because of the collective support of mentors who guided me, colleagues who stood by me, and a family who walked every step of this journey with me and as a rock support no matter how hard the situations got.
POGS has always been more than an academic body. It is a vibrant, compassionate community committed to advancing women's health. As we move into this new year, my vision is to strengthen our scientific aspects, broaden our outreach, support young minds, and most importantly, continue to serve with empathy and excellence.Me n team POGS 2025-26 are completely committed towards safeguarding all our members and provide them legal, social and emotional support in any situation of crisis. Please feel free to contact me or any member of our team for any apprehension or problem you face.
Happy reading. We shall connect more. Striving for the betterment of women.
POGS FOR HER
Together, we shall! Teamwork is everything. Till Then,

ANDIO, NAMASKAR.

Dr Manish Machave

Dr Nilesh Balkawade
Secretary, POGS

Secretary's Address

Dear POGS Family,
उन्मेषःस्वप्नसंजात; तेजसा जीवनं स्पृशेत्।
संधानं नवलक्ष्ये, प्रेरणायाःसदा गतिः॥
Meaning: "Unmesh, born from dreams, touches life with brilliance.It aligns us with new goals and becomes the constant path of inspiration. Let's discover that within ourselves"
At the outset, I humbly seek your blessings and good wishes as we embark on another year of learning, service, and solidarity.
I'm delighted to invite you to our **Code Red Conference** on hemorrhage and hematological disorders in OBGYN—a critical gathering where we'll share cutting-edge insights and lifesaving protocols.
Standing Strong in Testing Times In recent days, when hospitals and doctors faced attacks, POGS stood unwaveringly by our colleagues—and we will continue to defend and support every member, just as we did in that incident.
Community & Social Programs Beyond academics, our year-round social programs—wellness workshops, public forums, and informal gatherings—are designed to nurture our bonds. Watch our social media for upcoming dates and venues.
2025 Conference Lineup
PG Con (June): Advanced training for postgraduates
BreastCon (August, Mahabaleshwar): Innovations in breast health
Fertility Carnival (October, Goa): Celebrating reproductive medicine
FOGSI Presidential Conference (November, Pune): National leadership forum
POGS Annual Conference (December): Our flagship scientific meeting
Exchange Conference (February): Collaboration and fun learning the lessons of life!
Cherry on the Cake Our "One Hall, One Faculty" masterclasses will bring you single-day, high-impact sessions with National and world-renowned experts—focused, immersive, and unmissable.
New POGS Initiatives
POGS App: Your personalized hub for programs, guidelines, and member networking
Dynamic Committee Chairperson Leadership: Fresh vision from our new Chairpersons
Vibrant Social Media: Daily case discussions, expert tips, and community spotlights
Member Connect: Enhanced platforms to share research and experiences
Let's unite—learning from one another, supporting each other through every challenge, and advancing women's healthcare together.
We for POGS!
Warmly,
Dr. Nilesh Balkawade

Editorial

Dr Kalyani Ingale
Editor

Warm greetings!!!

Welcome to the latest issue of POGS newsletter 'Womb & Wellness'!
This April, newsletter focuses on obstetric and gynaecological haemorrhage, a significant cause of maternal morbidity and mortality. Excessive haemorrhage is one of the commonest symptom when women consult an obstetricians and gynaecologist.
In most of the cases the cause is related to the primary obstetric and gynaecological pathology and is easily identified. Let's emphasize on early recognition, prevention, and effective management strategies to improve patient's outcomes. Through this newsletter, we the editorial team aim to equip practitioners with the knowledge and tools and the role of interdisciplinary teams necessary to prevent, diagnose, and effectively manage haemorrhage, ultimately improving patient outcomes and reducing maternal mortality.
Our seniors, teachers & colleagues have contributed to this newsletter to direct us in the right direction so that we all sail through this journey safely delivering best practices to the society. By focusing on these key areas, we hope to contribute to a safer and healthier future for mothers and their families.
We value your feedback and encourage you to share your thoughts and questions with us. We're always striving to improve our newsletter and make it more relevant to your needs.

Dr Kalyani Ingale, Clinical Secretary, POGS 2025 – 26

The Gift of Motherhood

Dr Laxmikant Behele
Chairperson, High Risk Obstetrics Committee, POGS

Motherhood is the most valuable gift that is bestowed upon our fellow women by the Almighty.
Hence, it is the dream of one and every lady to go through and enjoy this blissful experience of becoming a mother.
This gift comes at ease to a majority and at times with great pain to a few. It can be in the form of infertility to early pregnancy loss to few or an anomalous baby or IUFD at some time during pregnancy or an unexplained stillbirth to the remaining others.
It becomes our prime duty as fellow obstetricians to take care of the expectant mothers.
Also, we are equally blessed to work as obstetricians as it gives us a chance to serve the woman to sail smoothly through her journey of motherhood and help her fulfil the dream of becoming a mother.
In this endeavour, POGS has taken a step further to form a high-risk obstetrics committe.
The committee members shall conduct the programs/ CMEs or webinars to educate the doctors, nurses and the public in general.
Make them understand the importance of having a healthy diet, healthy lifestyle, Good haemoglobin, regular antenatal visits and of course a 100% hospital deliveries.
We do understand that this is a continuous process in order to achieve the goal of a two-digit figure of maternal mortality.

POGS MANAGING COMMITTEE

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President, POGS

Dr Nilesh Balkawade
Secretary, POGS

Dr Uma Wankhede
President Elect

Vice President
Dr Vaishali Chavan

Ex Vice President
Dr Vaishali Korde Nayak

Clinical Secretary
Dr Kalyani Ingale

Treasurer
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Jt Clinical Secretary
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Jt Treasurer
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Dr Amey Chugh

Dr Akash Thube

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Dr Madhu Juneja

Dr Charuta Jogalekar

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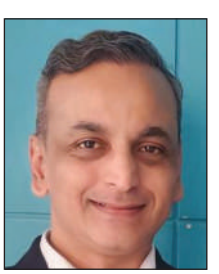

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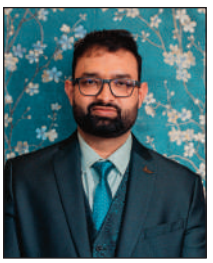
Dr Hemant Deshpande

Dr Manjiri Valsangkar

Dr Kanchan Durugkar

Dr Laxmikant Behele

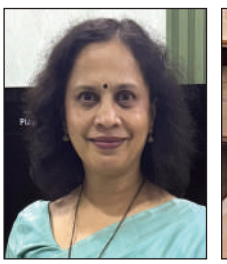


Dr Kunaal Shinde

Dr Satish Deshmukh

Dr Pandurang Burute

Dr Tanuja Joshi



Dr Vaishali Biniwale

Dr Sanjay Sharma

Zindagi ka Safar
POGS LANDMARK HISTORY

1. In 1953, a small group of 5 Gynaecologists gather at IMA Hall & POGS was born. Dr. Y. V. Pathak was the first president with Dr. A. R. Paranjpe as Secretary & Dr. Chaplabai Khadilikar as Treasure.
2. In 1956-Affiliation to FOGSI was obtained with a quorum of 12 members.
3. In Nov. 1963 the office was shifted to Dept. of Ob. Gyn., Sasoon Hospital where meetings were held at regular intervals
4. In 1976. Dr. A. G. Sathe & Dr. Nishikant Shrotri & the Managing Committee, trifurcated the post to the current posts of General Secretary, Clinical Secretary and Treasurer. Till thaen, the posts of General Secretary, Clinical Secretary and Treasurer were shouldered by the same person.
5. In 1976 the first constitution of the society was formed and the society was registered in 1976 by Dr. A. G. Sathe and Dr. Nishikant Shrotri with the help of managing committee and the senior members of the society.
6. Lit was in 1989 POGS started the CME. (Continuation of Medical Education)
7. In 1992 we launched the Annual Conference.
8. On 26th Jan. 2009, we shifted to our own office at Dr. Nitu Mandke IMA House on Tilak Road, Pune.
9. On 4th March 2019 the rules & regulations were amended & approved by the General Body
10. On 6th February 2010 POGS website was relaunched in new format.
11. 24th June 2014, FOGSI-FIGO Conference
12. AMOGS 2017, AMOGS Conference

BLESSINGS & GRATITUDE - Past Presidents Meeting

On 29 th January, incoming team of POGS of year 2025- 2026, under the leadership of President Elect, Dr Manish Machave, had organised a ' Blessings and Gratitude' meet at Hotel President. The honourable FOGSI president Dr Sunita Tandulwadkar graced the occasion with her presence. Dr Vaishali Chavan and Dr Vaishali Korde were the convenors of the programme. In an innovative effort , 13 past presidents of POGS were felicitated by Dr Manish Machave and incoming General secretary Dr Nilesh Balkawade, for their remarkable contributions to POGS. Alongwith them, senior teachers from various academic institutions and FOGSI chairpersons and office bearers were also felicitated. The whole team sought their blessings, expressed gratitude towards them and noted their inputs and insights . Everyone opined unanimously that collective efforts should be taken so that POGS excels more as an organisation.

The event ended with the group photograph of the incoming team and also with all the past presidents and senior members of POGS.

IFS Western Maharashtra Chapter Installation Ceremony with POGS - By Dr Meenu Agarwal

The CME with installation ceremony of Western Chapter of Maharashtra, Indian Fertility Society in association with Pune Obstetric and Gynecological Society held on 29th March at Hotel Crown plaza.
Total no of attendees - 76
Chief guest Prof Col Pankaj Talwar, President IFS

GOH Dr Arti Nimkar , President POGS
Dr Shweta Mittal Sec General IFS
Dr Kiran Kurtkoti President AMOGS
It was a well attended CME with good scientific content
2 CME points and 4 ICOG points, credits to good offices of POGS and ICOG.

POGS Committee Handover Ceremony

On 1st April 2025, team POGS 2025-26, under the leadership of President Dr Manish Machave, took charge from the outgoing team headed by Dr Aarti Nimkar, in the POGS office. It was indeed a proud moment for all of us. The event started with auspicious Ganesh Poojan. Dr Nilesh Balkawade, General secretary POGS, Dr Kalyani Ingale , Clinical secretary, Treasurer Dr Samidha Dalvi also took charge.
The outgoing team congratulated the incoming team and incoming team expressed their gratitude towards the outgoing team. We were extremely blessed by the presence of Dr Parag Biniwale, ICOG chair and Past President POGS and Dr Vaishali Biniwale for our handover ceremony.





Investigations and Management of Recurrent Pregnancy Loss

Dr Aswath Kumar
Professor and Unit Chief, Department of Obstetrics and Gynaecology, Jubilee Mission Medical College, Thrissur

Dr Lakshmi Nambiar
Assistant Professor, Department of Obstetrics and Gynaecology, Jubilee Mission Medical College, Thrissur

Recurrent miscarriage is defined as three or more consecutive pregnancy losses[1] and is found to affect 1% of couples.The American Society of Reproductive Medicine defines Recurrent Pregnancy Loss as 2 or more failed pregnancies.[2]Clinicians may use their clinical discretion to recommend extensive evaluation even after 2 pregnancy losses.It would be prudent to start investigating a couple with Recurrent miscarriage, after the 2nd loss if:

- Female partner > 35 years of age or male partner > 40 years
- Couple treated for Infertility
- Fetal heart activity observed in any of the concepts
- Normal karyotype of previous conceptus

How do we evaluate a case of RPL?
For a better understanding of the investigational approach to RPL, the etiologiesof RPL may be grouped as follows[3]

Undisputed causes	1. Parental chromosomal abnormalities 2. Anti Phospholipid antibody Syndrome	2-5% 15%
Established causes	1. Inherited thrombophilias 2. Cervical insufficiency 3. Fetal genetic abnormalities like heterotrisomy	8-12% 5-10% _
Other proposed causes	1. Anatomic causes 2. Endocrine causes 3. Infections 4. Immunological causes 5. Environmental causes 6. Unexplained RPL	15% 8-12% 0.5-5% _ _ 40-50%

The evaluation should include a comprehensive history from both partners, physical examination, pelvic examination and a diagnostic screening protocol. As per the RCOG Green Top Guidelines, the only evidence based investigations to be offered to a couple with Recurrent miscarriage are:

1. Karyotyping - of the conceptus of the third and subsequent consecutive miscarriages - of the parents ,if testing of the products of conception reports an unbalanced structural chromosomal abnormality.

2. Screening for Anti Phospholipid antibodies - in all women with recurrent first trimester miscarriage and all women with 1 or more second trimester miscarriage, to be done before pregnancy

3. Pelvic ultrasound - In all women with recurrent first trimester miscarriages and women with 1 or more second trimester miscarriages.

4. Screening for inherited thrombophilias - in women with 2nd trimester miscarriage. Screening for Factor V Leiden, Prothrombin gene mutation and Protein S deficiency.
In the Indian scenario, we have to consider the following investigations in a couple with RM :

ALL PATIENTS :
• Parental Karyotyping - for parental chromosomal abnormalities (like balanced reciprocal translocations or a Robertsonian translocation, inversion or X chromosome mosaicism like Klinefelter's Syndrome)
• Pelvic ultrasound - to assess uterine anatomy and anomalies. Suspected uterine anomalies would require further investigations to confirm the diagnosis, like hystero- laparoscopy, 3D ultrasound or MRI.
• AntiPL antibodies - Lupus Anticoagulant and Anticardiolipin antibodies IgG and IgM ,for Antiphospholipid antibody Syndrome.

SELECTED PATIENTS :
1. Thrombophilia screening:
1.1. Patients with a personal or family history of thrombophilias or thrombosis
1.2. Prior early onset (<34 weeks) severe Pre eclampsia or Fetal Growth Restriction
1.3. Women with a failed IVF
1.4. Women with SLE
2. 3D pelvic ultrasound/ HSG / Hysterocopy/ Laparoscopy - if uterine anomalies are suspected.
3. Early follicular phase LH, FSH, Testosterone- in PCOS , hyperandrogenism.
4. OGTT/ HbA1C - if clinical evidence of diabetes.

5. Thyroid function test - if clinical evidence of thyroid dysfunction.
6. Cervical length screening in pregnancy- if history suggestive of cervical insufficiency.
7. Vaginal swab for bacterial vaginosis- if history of Recurrent mid-trimester abortions or preterm labour.
8. Serology for Rubella - if negative, patient can be vaccinated.

SCREENING FOR ANTIPHOSPHOLIPID ANTIBODIES

In the etiology of pregnancy morbidity, the 2 most clinically important also are Lupus Anticoagulant (LAC) and Anticardiolipin Antibodies (aCLs) of IgG and IgM subclass.[4] Based on the International consensus statement on an update of the classification criteria for definite APS, diagnosis of APS requires one clinical and one laboratory manifestation[5].

Clinical:

• A documented episode of arterial, venous or small vessel thrombosis- other than superficial venous thrombosis, with no significant evidence of inflammation in the vessel wall, and/or
• 1 or more unexplained deaths of a morphological normal fetus at or beyond the 10th week of gestation, and /or
• 3 or more unexplained consecutive spontaneous abortions before the 10th week of gestation, and/or
• At least 1 premature birth of a morphologically normal neonates before the 34th week of gestation due to eclampsia or severe pre eclampsia or other features of placental insufficiency.

PLUS

Laboratory features

• Anti- cardiolipin antibodies (aCL) : IgG or IgM,positive in medium to high titre (i.e, > 40GPL or MPL), on 2 or more occasions, atleast 12 weeks apart (measured by standardized ELISA
• Anti beta2 glycoprotein 1 : IgG or IgM,positive in medium to high titre (i.e, > 40GPL or MPL), on 2 or more occasions, atleast 12 weeks apart (measured by standardized ELISA)
• Lupus Anticoagulant- detected on 2 occasions, at least 12 weeks apart, according to the guidelines of the International Society of Thrombosis and hemostasis.

- Women with one positive test result and a second negative result should have a third aPL test to confirm or refute a diagnosis of APS.
- Maternal aPLs may be down-regulated during pregnancy, hence the tests are best performed preceptionally.
- The dilute Russell viper venom test , together with platelet neutralisation procedure, is a more sensitive and specific test for detection of LA, than either activated Partial Thromboplastin Time or the Kaolin clotting time test.

Treatment options for recurrent pregnancy loss

1. Anti-phospholipid Antibody Syndrome:

All the standard guidelines recommend treatment with a combination of Low dose Aspirin plus Heparin in women with APS.However,there is no definite consensus on the type of Heparin to be used or about the timing of initiation of treatment.

A meta-analysis of RCTs[6] reported that Aspirin + Unfractionated Heparin combination was the only treatment that reduced the miscarriage rate by 54% and increased the live birth rate among women with APS. Multiple smaller prospective studies have reported no significant difference in the efficacy and safety between UFH and LMWH, when combined with Aspirin, in the treatment of women with RPL associated with Antiphospholipid antibodies.

LMWH is as safe as UFH, with the potential advantages of once daily injection owing to its longer half-life and increased bioavailability and also a lower risk of Heparin-induced Thrombocytopenia as well as osteoporosis[7].

As per the RCOG recommendations, there is no role for corticosteroids or Intravenous Immunoglobulins in the treatment of Recurrent miscarriage associated with APS.

Hence, in a woman with recurrent miscarriage associated with APS, it would be prudent to start the patient on Low dose Aspirin 75 mg/day ,as soon as the patient has a positive urine pregnancy test and subcutaneous LMWH 40 mg per day, as soon as an intrauterine pregnancy is confirmed by an ultrasound scan. If UFH is used,the dose should be 5000 IU twice daily.

Monitoring of anticoagulation

No routine monitoring of anticoagulation is required in women on LDA or LMWH. When UFH is prescribed, aPTT should be monitored weekly . LMWH in women with extremes of BMI requires monitoring of anticoagulation with anti-Xa activity (normal value of 0.6 to 1.0 U/ml, 4-6 hours after injection)

2. Genetic factors :

Couples detected to have an abnormal parental karyotyping in either of the partners, may be referred for genetic counseling, which offers them a prognosis for the risk of future pregnancies with an unbalanced chromo-

somal complement. Reproductive options in couples with chromosomal abnormalities include proceeding to a further natural pregnancy with or without a prenatal diagnostic test, gamete donation and adoption. IVF with pre-implantation genetic screening or diagnosis in couples with chromosomal abnormalities is associated with lower live birth rates(20-30%), compared to natural conceptions (50-70%) ,and hence is not recommended.

3. Anatomic factors :

- Congenital uterine anomalies:

Hysteroscopic resection of uterine septae, can have beneficial effects in women with RPL and a septate uterus.There are no surgical procedures recommended in unicornuate or didelphic uterus. Surgical correction should be considered in pregnancy loss associated with Asherman syndrome, uterine fibroid and polyps. In the event of irreparable anatomic uterine abnormalities and RPL, IVF with transfer of embryo to an appropriately selected surrogate has to be considered[8].

- Cervical insufficiency and cervical cerclage :

In women with a singleton pregnancy and a history of one 2nd trimester miscarriage, attributed to cervical factors, an ultrasound indicated cerclage should be offered if a cervical length of 25 mm or less is detected by transvaginal scan before 24 weeks of gestation [3]. A History indicated Cerclage may be beneficial in women with 3 or more spontaneous losses or preterm deliveries, according to the Medical Research Council/RCOG Trial 1993. Both the Mc Donald and the Mosified Shirodkar techniques of cervical cerclage have similar success rates when performed prophylactically. A transabdominal cerclage, with suture placed at the level of the uterine isthmus , is advocated in patients with prior failed vaginal cerclages or in very short and scarred cervix. Abdominal cerclages are associated with lower risks of preterm births and perinatal deaths, compared to vaginal cerclages. It may be done laparoscopically or by laparotomy,either precoceptionally, or during the early antenatal period (12-16weeks).

4. Inherited thrombophilias:

In women diagnosed with an inherited thrombophilia, with a personal/family history of a thromboembolic episode, the recommended regimen of anticoagulation is:

o UFH, 8-12 hourly subcutaneous, in doses sufficient to keep the mid-interval aPTT 1.5 times the control mean.

o LMWH (Enoxaparin 1mg/kg ,12th hourly)

Thromboprophylaxis is to be started 4-6 weeks ahead of the gestational age at which the previous episode of VTE during a prior pregnancy occurred, or in the late 2nd trimester, if the episode of VTE was unrelated to pregnancy. It has to be continued for 6-12 weeks post partum. We can switch over to Warfarin after 48 hours of delivery with the Heparin continued till the PT INR is between 2 and 2.59.

5. Endocrine factors :

o Diabetes,thyroid dysfunction:

Associated with sporadic miscarriage, but no evidence that these contribute to Recurrent miscarriage.

o Untreated hyperprolactinemia:

This can be a cause for RM and treatment with bromocriptine /cabergoline reduces the miscarriage rate4.

o Luteal phase defect :

Progesterone supplementation in women with early pregnancy losses or recurrent miscarriage can be done on an empirical basis, though there are no definite evidence based guidelines on the role of LPD and Progesterone supplementation in recurrent miscarriage.

o PCOS :

PCOS with insulin resistance is associated with higher rates of miscarriage than PCOS without insulin resistance, in women undergoing ovulation induction. Metformin treatment for women with PCOS, during ovulation induction and early pregnancy, may improve endometrial receptivity and implantation, thereby reducing the risk of miscarriage. But according to RCOG, there is insufficient evidence to show that Metformin supplementation or LH suppression in women with PCOS and RPL, improves the live birth rates[3].

6. Immunological factors:

No alloimmune mechanisms have been shown to cause recurrent miscarriage in humans. Hence, the current recommendation is that Immunotherapy or corticosteroids should not be offered to women with unexplained RPL.

7. Environmental factors :

Obesity has been recognized as a risk factor for Recurrent Miscarriage, in addition to being implicated in Infertility, sporadic miscarriage, late pregnancy complications and perinatal morbidity. Cigarette smoking, alcohol, caffeine consumption, use of NSAIDs and some anti- depressants in the peri-conceptual period are also other risk factors. Behavioral and lifestyle modifications to correct these environmental risk factors, have to be recommended to women with recurrent miscarriages.

8. Unexplained Recurrent miscarriage: 50%
The chances for a successful pregnancy with supportive care alone ,in women with unexplained RPL, is around 75%. Aspirin alone, or in combination with Heparin is widely being prescribed for women with unexplained Recurrent miscarriage. But no RCTs so far have documented an increase in live birth rates with either of these interventions. Hence, empirical treatment in women with unexplained RPL is unnecessary and should be restricted.

9. Infections:
• No association between TORCH infections and RPL
• Detection and treatment of Bacterial vaginosis early in pregnancy, may reduce the risk of preterm delivery in women with history of mid-trimester miscarriages or preterm births.

Further research and studies are required to establish the role of NK cells and cytokines in RPL, role of thromboprophylaxis in women with thrombophilias and RPL without a prior episode of VTE, Progesterone treatment in women with unexplained RPL and Metformin treatment in women with Insulin resistance and RPL.

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Prevention and Management of Eclampsia

Dr Charmila Ayyavoo
Director, Aditi hospital and Parvathy Ayyavoo fertility centre, Trichy. AP, Dhanalakshmi Srinivasan Medical College, Perambalur. Vice President FOGSI 2024

Introduction:
Eclampsia is the development of convulsions in a woman with hypertensive disorders in pregnancy (HDP) which cannot be attributed to other causes. Seizures are generalized & may appear before, during or after labor. The other conditions which need to be ruled out before a diagnosis of eclampsia can be made are epilepsy, ischemia of cerebral arterial circulation and infarction, intracerebral hemorrhage, or drugs. It is less likely to be eclampsia, if convulsions occur 2-3 days after delivery or convulsions occur during the usage of magnesium sulfate.

Imminent symptoms:
The occurrence of eclamptic convulsions is preceded by signs of cerebral irritation in many patients. The patients can have severe headaches, which may be persistent in the occipital and frontal areas. Headaches are due to an increased cerebral perfusion, cerebral oedema, and hypertensive encephalopathy. They can also manifest blurring of vision, photophobia, and altered mentation. Rarely, patients can have eclamptic convulsions without warning, imminent symptoms.
A group of patients who develop eclampsia need not have the diagnostic features of HDP like hypertension or proteinuria.

Prevention of eclampsia:
Eclampsia cannot be prevented in all pregnancies because of its atypical nature in a group of patients who may not develop hypertension, proteinuria, or imminent symptoms before convulsions occur. There need not be a linear progression from hypertension to preeclampsia without severe features to preeclampsia with severe features and eclampsia. Eclampsia can be prevented by timely delivery and the appropriate use of magnesium sulphate in patients with high-risk features.

Risk factors:
• Eclampsia
• Preeclampsia with severe features
• Gestational hypertension with severe features
The use of magnesium sulphate prevents the occurrence of further convulsions in a patient with eclampsia.

Magnesium sulphate:
Magnesium sulphate is more effective than phenytoin, diazepam, or nimodipine in reducing eclampsia and should be considered the drug of choice in the prevention of eclampsia in the intrapartum and postpartum periods. Benzodiazepines and phenytoin are justified only in the context of antiepileptic treatment or when magnesium sulphate is contraindicated or unavailable (myasthenia gravis, hypocalcemia, moderate-to-severe renal failure, cardiac ischemia, heart block, or myocarditis). For women requiring cesarean delivery (before onset of labor), the infusion should ideally begin before surgery and continue during surgery, as well as for 24 hours afterwards. For women who deliver vaginally, the infusion should continue for 24 hours after delivery.

Management of eclampsia:
When a pregnant patient presents with tonic-clonic convulsions,
• First diagnosis should be … eclampsia (unless proved otherwise)
• Quick history / Referral letter is taken.
• Blood pressure and urine albumin are measured.
• Confirmation of diagnosis is available within a minute
The management should ideally be done at a secondary or tertiary care centre. The aim should be to
• Abort the present convulsion.
• Prevent further convulsions.
• Control the hypertension.
• Timely and safe delivery.

The management is as follows:
• Help is called for.
• Airway is secured.
• Tongue bite and other injuries are avoided.
• Oxygen supplementation is given.
• Aspiration is reduced by lateral decubitus position, oral suction
• Bladder is catheterized with Foley's
• 18 /20 number intravenous cannula (IV) can-

nula are inserted preferably at two places.
• Through the inserted IV cannula, blood is collected for investigations: complete blood count, liver function tests, renal function tests, and platelet count.
• Without waiting for results, magnesium sulphate regimen must be started.
• Blood pressure is controlled.
• Delivery is planned.

The regimes of magnesium sulphate:
The Zuspan or Pritchard regimes are used for the prevention and management of eclampsia.

Zuspan's regime:
Loading
4 gm IV in 12 cc distilled water over 20 min
Maintenance
5 gm in 500ml RL at 1 gm/hour

Pritchard's regime:
Loading
4 gm IV in 12 cc distilled water over 20 min + 5 gm deep intramuscular (IM) in each buttock.
Maintenance
5 gm deep IM in alternate buttock 4 hourly

Refractory seizures:
• If convulsions recur, a further 2–4 grams of magnesium sulfate could be administered IV over 5 minutes.
• In cases refractory to magnesium sulfate (still seizing at 20 minutes after the bolus or more than two recurrences), sodium amobarbital (250 mg IV in 3 minutes), thiopental, or phenytoin (1,250 mg IV at a rate of 50 mg/minute) can be used
• Endotracheal intubation and assisted ventilation in the intensive care unit are appropriate in these circumstances.
• Head imaging is considered because most of cases refractory to magnesium sulfate therapy may prove to have abnormal findings on brain imaging.

Monitoring of magnesium sulphate therapy:
The maintenance dose of magnesium sulphate is given only after assuring that:
• Patellar reflex is present
• Respiration is not depressed. (RR > 16/min)
• Urine output during previous 4 hours- exceeded 100 mL or was 25ml/hr.
Absent patellar reflex
• If respiration is normal, further doses of MgSO4 are to be withheld until the reflexes return.
• MgSO4 can be restarted if considered necessary once reflexes have returned but at a reduced dose unless there have been further convulsions.
Urine output < 100 ml in 4 hours –
• If there are no other signs of magnesium toxicity, the next I/M dose of magnesium sulphate is to be reduced to 2.5 gm or the I/V infusion to 0.5 gm/hour.
• Particular attention is to be paid to fluid balance and blood loss.
Respiratory depression
• Magnesium sulphate therapy is to stopped.
• 1 gm calcium gluconate is given IV (10% Ca Gluconate 10 ml over 10 minutes with cardiac monitoring)
• Oxygen is given by mask.
• Airway is maintained.
• Patient is nursed in recumbent position.
Patients at risk of impending respiratory depression may require tracheal intubation and emergency correction with calcium gluconate along with furosemide intravenously to accelerate the rate of urinary excretion.
The most common side effect of magnesium sulphate is flushing. Less common side effects are nausea, vomiting, muscle weakness, thirst, headache, drowsiness, and confusion.

Control of hypertension:
The next step in the management of eclampsia is the control of hypertension with anti-hypertensive drugs.
• Severe hypertension should be treated with the first-line agents oral nifedipine, oral labetalol, intravenous (IV) labetalol, or IV hydralazine.
• Hydralazine is now only used when other drugs fail to control HTN, because of its increased perinatal adverse effects.

Investigations:
Hemogram, complete urine examination, liver function tests, renal function tests, platelet count, serum electrolytes, peripheral smear for evidence of hemolysis in the form of fragmented RBCS, spherocytes, and reticulocytosis, fundoscopy are done.

Delivery:
• Women with eclampsia should be delivered in a timely fashion.
• Eclampsia by itself is not an indication for cesarean delivery.
• Once the patient is stabilized, the method of delivery should depend on factors such as gestational age, fetal

presentation, and the findings of the cervical examination.
• During eclamptic seizures, there are usually prolonged fetal heart rate decelerations, even fetal bradycardia, and sometimes an increase in uterine contractility and baseline tone because of maternal hypoxia and hypercarbia.
• Only after maternal hemodynamic stabilization should one proceed with delivery.
• Maternal resuscitation is usually followed by normalization of the fetal tracing.

Anaesthesia in a patient with eclampsia:
• General anesthesia carries more risk to pregnant women than regional anesthesia does because of the risk of aspiration, failed intubation because of pharyngo- laryngeal edema, and stroke secondary to increased systemic and intracranial pressures during intubation and extubation.
• However, neuraxial anesthesia and analgesia are contraindicated in the presence of a coagulopathy because of the potential for hemorrhagic complications.

Magnesium sulphate during caesarean:
• Can cause significant anesthetic implications because it prolongs the duration of nondepolarizing muscle relaxants.
• But it should be continued during caesarean delivery.
• This recommendation is based on the observation that magnesium sulfate half-life is 5 hours and that discontinuation before cesarean delivery would only minimally reduce magnesium concentration at the time of delivery while possibly increasing the risk of seizure.
• Women with preeclampsia with severe features undergoing cesarean delivery remain at risk of developing eclampsia.
• The induction of general anesthesia and the stress of delivery may even reduce the seizure threshold and increase the likelihood of eclampsia in the immediate postpartum period if the infusion of magnesium sulfate is stopped during delivery.
Other anti-convulsant drugs:
• In the rare cases of an extremely agitated patient, IV clonazepam 1 mg, diazepam 10 mg, or midazolam may be used for sedation to facilitate the placement of the IV lines and Foley catheter, and the collection of blood specimens.
• These drugs should be used cautiously because they inhibit laryngeal reflexes, increasing the risk of aspiration and may depress the central respiratory centers leading to apnea.

Complications of eclampsia:
• Significant cause of maternal death, particularly in low-resource settings.
• Seizures may lead to severe maternal hypoxia, trauma, and aspiration pneumonia.
• Although residual neurologic damage is rare, short-term, and long-term consequences such as impaired memory and cognitive function, especially after recurrent seizures or uncorrected severe hypertension leading to cytotoxic edema or infarction can occur.

Differential diagnosis of eclampsia:
• Cerebral venous thrombosis
• Intracerebral hemorrhage
• Brain tumors,
• Metabolic disorders such as uremia, hypoglycemia, and hyponatremia
• Infective diseases such as bacterial meningitis, acute viral hepatitis with fulminant hepatic failure, and cerebral malaria
• Neurocysticercosis
• Pituitary apoplexy and central serous retinopathy.

Principles of care during transfer of an eclamptic patient:
If the patient delivers in a primary care centre, she will need transfer to a centre which is level 2 or 3.
• The patient is referred after informing the concerned higher centre.
• All reports should be sent.
• Convulsions should be controlled with loading dose of magnesium sulphate before transfer.
• Hypertension should be controlled
• Ambulance with facilities and personnel for managing convulsions should be used for transfer.
Every place where deliveries are conducted should have an eclampsia kit ready and the eclampsia drill should be conducted periodically for the paramedical team.

Eclampsia kit contents:
• Airway
• IV Cannula, sticking tape, three-way cannula
• IV sets, blood set, 100 ml NS
• Magnesium Sulphate ampoules - at least 16
• Lignocaine 2%
• Foleys catheter
• Syringes - 5ml,10ml and 20 ml
• Bandage
• Gloves

Eclampsia drill:
The drill for eclampsia management is as follows:
• Call for assistance
• Designate team leader, checklist reader, primary RN
• Ensure side rails are up
• Protect airway + improve oxygenation
• Continuous foetal monitoring
• Place IV
• Draw blood for lab investigations
• Administer antihypertensive therapy if appropriate
• Develop delivery plan
• Debrief patient, family

Conclusion:
The initial steps in the management of eclampsia are basic supportive measures such as calling for help, prevention of maternal injury, placement in lateral decubitus position, prevention of aspiration, administration of oxygen, and monitoring vital signs including oxygen saturation. Only subsequently is attention directed to the administration of magnesium sulfate. Most eclamptic seizures are self-limited. Magnesium sulfate is not necessary to arrest the seizure but to prevent recurrent convulsions.

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MEDICAL MANAGEMENT OF PPH

Introduction

Postpartum hemorrhage (PPH) poses a significant risk to maternal health and is characterized by excessive blood loss after delivery. While uterine contractions and the coagulation cascade primarily regulate blood loss, PPH can lead to severe complications if untreated. Based on blood loss thresholds, the traditional definition of PPH has evolved to encompass broader criteria reflecting signs of hypovolemia. Traditionally, PPH is defined as more than 500 mL of estimated blood loss in a vaginal delivery or more than 1000 mL of estimated blood loss during Cesarean delivery. These parameters were redefined in 2017 by the American College of Obstetrics and Gynecology as a cumulative blood loss of more than 1000 mL with signs and symptoms of hypovolemia within 24 hours of the birth process, regardless of the mode of delivery. Though this change was made with the knowledge that blood loss at the time of delivery is routinely underestimated, more than 500 mL of blood loss at the time of vaginal delivery should be considered abnormal with the potential need for intervention.

• PPH is frequently subdivided based on symptom onset. Primary PPH is hemorrhage that occurs between the third stage of labor (ie, delivery of the placenta) and 24 hours after fetal delivery; secondary PPH occurs more than 24 hours after delivery—up to 12 weeks postpartum.

• Causes, summarized by the 4 "T's" (tone, trauma, tissue, thrombin), require prompt intervention. Management involves a multidisciplinary approach, emphasizing blood loss assessment, fluid replacement, and source control. Despite advancements, PPH prevention and prediction remain essential to maternal well-being. PPH poses a significant challenge in obstetrics, complicated by difficulties in accurately estimating blood loss, as cognitive biases can lead to delays in diagnosis and management. Though more accurate, quantitative blood loss measurement methods have not consistently improved clinical outcomes. Interprofessional approaches, including PPH bundles and perinatal quality collaboratives, reduce morbidity. Management involves a coordinated effort addressing uterine atony, genital tract lacerations, retained placental tissue, and coagulopathy, with surgical interventions like hysterectomy as a last resort. Prevention strategies include active management of the third stage of labor and prenatal identification of high-risk factors. Vigilance and prompt intervention remain crucial in mitigating maternal morbidity and mortality associated with

• Primary PPH is when you lose 500 ml (a pint) or more of blood within the first 24 hours after the birth of your baby. Primary PPH can be minor, where you lose 500–1000 ml (one or two pints), or major, where you lose more than 1000 ml (more than two pints).

• Secondary PPH occurs when you have abnormal or heavy vaginal bleeding between 24 hours and 12 weeks after the birth.

Etiology

The primary causes of PPH are the 4 “T’s”: tone (uterine atony), trauma (lacerations or uterine rupture), tissue (retained placenta or clots), and thrombin (coagulation deficiency). Uterine atony is the most common cause of PPH, accounting for approximately 70% of cases. A vast array of risk factors are associated with PPH, including advanced maternal age, nulliparity, and grand multiparty. However, most risk factors are related to underlying causes. Secondary PPH is associated with retained placentas, subinvolution of placental sites, inherited coagulopathies, and infectious etiologies. The following risk factors are associated with the 4 main etiologies:

• Uterine atony: Chorioamnionitis, magnesium sulfate therapy, prolonged labor or precipitous delivery, labor induction or augmentation, uterine fibroids, uterine inversion, or conditions resulting in uterine overdistention (eg, multiple gestation, fetal macrosomia, or polyhydramnios)

• Trauma: Cesarean delivery, instrument-assisted vaginal birth, midline episiotomy, precipitous delivery, and persistent occiput posterior position

• Tissue: Prior Cesarean birth, placenta accreta, placental abruption and associated hyperten-

sion, and uterine anomalies
• Coagulopathy: Severe preeclampsia and eclampsia, HELLP (hemolysis, elevated liver-enzyme level, and low platelet count) syndrome, intrauterine fetal death, placental abruption, amniotic fluid embolism, or inherited coagulopathies (eg, von Willebrand disease)

Etilogy and Risk Factors

Pregnancy itself is a risk factor for PPH; every pregnancy can result in PPH. It is important to identify the risk factors and prophylactically prevent PPH. The risk factors for PPH are provided by Hoveyda et al, which are modified to suit the Indian population in Table 2.[14]

Table 2. Risk factors for postpartum hemorrhage

Maternal issues	
• Teenage pregnancy	• Cardiac dysfunction
• Elderly primigravida	• Hypertensive disorders
• Multiparity and Grand multiparity (> 4)	• Thyroid dysfunction
• Inadequate prenatal visits	• ART pregnancy
• Low socioeconomic status	• Renal and liver disorders
• Previous postpartum hemorrhage	• Respiratory disorders
• Previous uterine surgeries	• Anticoagulant therapy
• Uterine malformations	• Viral infections, dengue
• Fibroid uterus	• Inherited and acquired coagulopathies
• Previous cesarean section	• Hemoglobinopathies
• Previous instrumental delivery	• Metabolic syndrome
• Anemia	• Post-bariatric surgery
• Thrombocytopenia	• Pregnancy after renal transplant
• Diabetes	• Multifetal gestation
Intrapartum	
• Induction and augmentation of labor	• Instrumental deliveries
• Precipitate labor and prolonged labor	• Cesarean section
• Obstructed labor	• In-coordinate uterine action (hypotonic & hypertonic)
• The arrest of labor in the second stage	• Prolonged rupture of the membrane (PROM/PPORM)
• Trial of labor after cesarean (TOLAC)/ vaginal birth after cesarean (VBAC)	• Chorioamnionitis
• Placenta previa	• Placenta abruption
• Placenta accreta syndrome	• Arteriovenous malformations
• Chorio angioma	
Postpartum	
• Genital tract trauma	• Uterine inversion, uterine rupture
• Retained placenta	• Subinvolution
• Retained placental tissues	• Puerperal sepsis
Fetal issues	
• Polyhydramnios	
• Large-for-gestational-age fetus	
• Fetal macrosomia (birth weight greater than 8 lb, 13 oz [4,000 g])	
• Congenitally malformed fetus	
Placental issues	
• Placenta previa	
• Placenta abruption	
• Placenta accreta	
• AV malformations	
• Chorio angioma	
• Placental abnormalities (battle door placenta, vasa previa etc)	

Epidemiology

PPH occurs in approximately 1% to 3% of all deliveries and is the leading cause of obstetric morbidity and mortality worldwide, accounting for approximately 8% of maternal deaths in developing countries and 20% of maternal deaths in developed countries. The United States has one of the highest maternal mortality rates at 11% and continues to rise, increasing from 8 to 40 cases per 10,000 deliveries. In Europe, PPH occurs in approximately 13% of deliveries. Uterine atony, the primary cause of PPH, accounts for 70% to 80% of all hemorrhages.

Pathophysiology

Physiologic changes during pregnancy, including an increase in uterine blood flow from approximately 100 mL/min in a nonpregnant uterus to 700 mL/min, coagulation changes resulting in a hypercoagulable state, and postpartum changes (eg, myometrial contraction and local decidual hemostatic factors) cause significant bleeding. Therefore, conditions that lead to a failure of these mechanisms can result in PPH. A loss of an approximate total blood volume of more than 1500 mL will typically cause clinical features of hypovolemia.

History and Physical Examination

Postpartum Hemorrhage Clinical Evaluation
An accurate clinical assessment of blood loss volume and evaluation of the underlying etiology is essential for promptly diagnosing PPH and initiating effective interventions. Initial patient evaluation should include a rapid clinical assessment and review of the patient's risk factors. Clinical features of significant blood loss, including tachycardia, tachypnea, and hypotension, may be masked initially, as pregnant women can have a blood loss of more than 1000 mL before signs of hypovolemia are apparent. Clinicians should remember that more than 25% of blood volume, or approximately 1500 mL or more, is likely lost when signs of hypovolemia are present. Prompt PPH recognition is difficult due to this delay in clinical features of hypovolemia. Continued assessment during delivery and postpartum is recommended, including visual estimation, weighing surgical sponges and drapes, and serial vital signs. Serial vital signs should include heart rate, blood pressure recordings every 15 min, respiratory rate, and peripheral oxygen saturation. During PPH evaluation, clinicians should also be aware of cognitive biases affecting their ability to diagnose PPH quickly. These biases include implicit biases, which are unconscious stereotypes that affect a clinician's response to or treatment of certain patients (eg, unconscious discrimination). Normalcy bias can cause clinicians to dismiss signs and symptoms of hypovolemia as normal or temporary (eg, anxiety). Anchoring biases are when clinicians only consider the first suspected diagnosis.

A rapid assessment of the entire lower genital tract, including vaginal walls, cervix, and labia, for lacerations, hematomas, or signs of uterine rupture (eg, regression of fetal presenting part and maternal abdominal tenderness), should be performed. The placenta should be examined to determine if the tissue remains intact, and a manual examination with extraction should be performed for any retained placental tissue; a bedside ultrasound assessment may be a part of the evaluation. A soft, "boggy" or noncontracted uterus is the common finding with uterine atony. Uterine inversion presents as a round bulge or mass with palpation of the fundal wall in the cervix or lower uterine segment, resulting from excessive umbilical cord traction or abnormally adherent placenta. Widespread bleeding, including from venipuncture sites, is a sign of disseminated intravascular coagulation. Patients may also present with acute vaginal bleeding peri- or postpartum. The patient may also have an increased heart rate, respiratory rate, and dizziness. As the patient loses blood, they may feel cold, have decreased blood pressure, and syncopal episodes. Patients may also have signs and symptoms of shock (eg, confusion, blurry vision, clammy skin, and weakness).

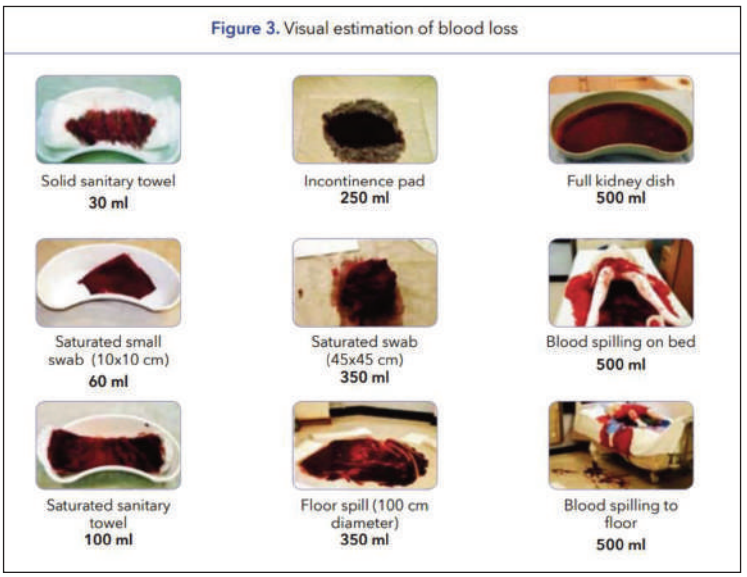
Table 6. Clinical signs and symptoms

	Stage 1	Stage 2	Stage 3	Stage 4
Blood loss (%)	<15	15–30	30–40	>40
Blood loss (cm^3)	<750	750–1500	1500–2000	>2000
Pulse rate	<100	>100	>120	>140
Respiratory rate	14–20	20–30	30–40	>35
Blood pressure	Normal	Decreased	Decreased	Decreased
Mental state	Normal/slightly anxious	Mild anxiety	Confusion and lathargy	Confusion

Blood Loss Quantification

Several studies have established that PPH blood loss totals are typically clinically underestimated, resulting in a misdiagnosis of excessive bleeding and, therefore, delayed interventions. Quantitative blood loss methods have been recommended for more accurate calculations, including graduated under-buttock drapes, laparotomy pads, sponge weighing, artificial intelligence-enabled technology, and irrigation canisters. These methods are more helpful than previous strategies involving awaiting hemoglobin changes and are meant to assist in the early recognition of PPH. However, clinical outcomes have not improved because clinicians may prioritize numeral parameters over the broad clinical picture. As such, waiting to begin PPH protocols until a specific blood loss threshold is met could lead to less favorable outcomes. Any amount of obstetric bleeding with clinical features of hypovolemia or a blood loss of more than 500 mL should precipitate PPH assessment and consideration of interventional protocols. Blood loss quantification should involve an interprofessional team of healthcare professionals, including physicians, nurses, and technicians, to ensure accurate estimations when active bleeding continues.

Figure 3. Visual estimation of blood loss



Modified shock index

The modified shock index is a bedside assessment defined as Heart rate (HR) to mean blood pressure (MAP), with a normal range of 0.5 to 0.7 in healthy adults.

Table 7. Shock index to mortality rates

	Shock index	Mortality rate	Blood products
No shock	<0.6	10.9% mortality	1 unit
Mild shock	≥0.6 to < 1.0		2.8 units
Moderate shock	≥1.0 to < 1.4		9.9 units
Severe shock	≥ 1.4	**39.8% motrality**	11.4 units

Method used to estimate the MAP: **MAP = DP + 1/3(SP – DP) or MAP = DP + 1/3(PP)**
*DP: diastolic blood pressure, MAP: mean blood pressure; PP: pulse pressure; SP: systolic blood pressure.

The common finding with uterine atony includes soft, boggy, or non-contracted uterus. Manual examination for any retained placental tissue should be followed by bedside ultrasound.
Uterine inversion presents as a mass with palpation of the fundal wall in the cervix or lower uterine segment. It is often linked to excessive traction on the umbilical cord or an abnormally adherent placenta.[19]

• A rapid assessment of the genital tract for hematomas, lacerations, or signs of uterine rupture should be performed.
• The amount of blood loss can be measured by using BRASS-V drape/or other blood collection receptacle. However, clinical conditions should always be kept in mind.

Evaluation
Laboratory studies are included as part of PPH protocols to help assess the degree of blood loss and guide interventions. PPH laboratory panels typically include complete blood count with platelet count, partial thromboplastin time, plasma thromboplastin, fibrinogen, comprehensive metabolic panel, ionized calcium, pH, and blood gases in addition to the routine prenatal laboratory studies that are obtained on admission (eg, blood type and antibody screening). However, some interventions should not be withheld pending the results of these studies. Complete blood count to assess hemoglobin, hematocrit, and platelets can be evaluated at intervals, although lab values lag behind the clinical presentation. Coagulation studies and fibrinogen are useful in patients with suspected disseminated intravascular coagulation, including patients with secondary PPH, placental abruption, or preeclampsia. An indwelling bladder catheter may monitor the patient's fluid status and assess urine output.

Investigations in PPH
• Haemoglobin and HCT –fall in Hb and hematocrit, (may not be initially low)
• Bed side tests: Bleeding time, clotting time, clot observation test
• Coagulation profile may be deranged in hemorrhage [10] » » » » » » »
✓ Bleeding time (BT) increased
✓ Clotting Time (CT) increased
✓ Platelet count (PC) decreased
✓ Prothrombin time (PT) increased
✓ International normalized ratio (INR) increased in coagulopathy
✓ Serum fibrinogen decreased or normal
✓ D-Dimers increased
Serum electrolytes may or may not be altered
• Renal parameters: Blood urea, serum creatinine elevated in renal failure, and hemolysis
• Serum lactate: Elevated in sepsis
• Serum calcium, magnesium and potassium: Can be low in hemorrhage
• Ultrasound: Retained placenta, adherent placenta, inversion, rupture
• Thromboelastogram (TEG): wherever available and feasible
• Arterial blood gas analysis and its interpretation
• Frequency of investigations: as per the clinical situation

Treatment / Management
General Postpartum Hemorrhage Management
The approach to treating PPH is focused on the patient's resuscitation when identifying and treating the underlying cause. Maintaining the patient's hemodynamic stability ensures continued perfusion to vital organs. As soon as PPH is identified, 2 large bore peripheral intravenous (IV) catheters (14- or 16G) should be placed, and maternal blood type and antibody screen should be confirmed in anticipation of aggressive corrective measures. Direct assessment of cumulative blood loss is essential, and a focus on early initiation of protocols for releasing blood products and massive transfusion protocols is necessary. Crystalloid and colloid IV fluids should be administered as indicated, and clinicians should also evaluate patients to identify the underlying cause and tailor treatments.

Uterine Atony Management
Uterine atony is the most common cause of PPH; therefore, clinicians should assess patients for this etiology. A soft uterus without physiologic contraction following delivery indicates uterine atony. Removal of clots and bimanual massage should be performed as the first step to mitigate excessive uterine bleeding. Medical management with uterotonic agents is typically the initial pharmacologic intervention if uterine atony is identified. While oxytocin is recommended routinely at delivery, additional uterotonic medications are necessary in up to 25% of patients with PPH. Furthermore, the use of multiple uterotonic agents can have a synergistic effect.

Pharmacologic agents used in PPH management include:
• Oxytocin: The International Federation of Gynecology and Obstetrics recommends giving oxytocin 10 international units (IU) IV or intramuscularly (IM) first-line for uterine atony if not administered prophylactically. The oxytocin hormone is naturally produced by the posterior pituitary and works rapidly, with an onset of action within 1 to 6 minutes, to cause uterine contraction following IV administration. Oxytocin has minimal adverse events and may be given during bimanual

massage in response to hemorrhage.
• Methylergonovine: Ergot alkaloids (eg, ergometrine, ergonovine, and methylergonovine) are serotonergic receptor agonists and partial α-adrenergic receptor agonists that cause sustained uterine contractions. The onset of action is approximately 1 to 3 minutes. Methylergonovine 200 µg IM or IV is typically recommended but is relatively contraindicated in patients with hypertension.
• Carboprost: As a 15-methyl prostaglandin F2-α analog, carboprost acts on prostaglandin receptors to stimulate uterine contractions. The recommended dosage is 250 µg IM or intramyometrially every 15 to 90 minutes for a maximum of 8 doses, with peak serum concentrations reached in approximately 15 minutes. Carboprost is contraindicated in severe hepatic, renal, and cardiovascular disease and may cause bronchospasm in patients with asthma.
• Misoprostol: Misoprostol is a prostaglandin E1 analog with a more prolonged onset of action than other uterotonics, depending on the administration route, which includes oral, sublingual, rectal, or buccal routes. The analog should be avoided in patients with anticoagulant therapy or cardiovascular disease; adverse effects may include nausea, diarrhea, and fever.
• Tranexamic acid: Tranexamic acid (TXA) is not uterotonic but inhibits fibrinolysis and is frequently used with uterotonic medications. The recommended dosage is 1 g of tranexamic acid IV over 10 minutes within 3 hours of delivery after a PPH diagnosis. TXA's onset of action is typically 5 minutes and is contraindicated in patients with a history of hypercoagulopathy.
• Carbetocin: Carbetocin is the carba analog that has prolonged activity and a long half-life due to deamination. It is a newer long-acting synthetic analogue of oxytocin with agonist properties. Carbetocin is available as room temperature formulation in India. which protects carbetocin from aminopeptidase cleavage, and its lipophilicity

If bimanual massage and uterotonic medications are insufficient to control hemorrhage, uterine tamponade may be considered. An intrauterine balloon tamponade system can be used, typically by filling an intrauterine balloon with 250 to 500 mL of normal saline. Uterine tamponade may be useful in those with lower uterine segment atony, where uterotonic agents may have a delayed onset of action. If an intrauterine balloon is not readily available, the uterus may be packed with gauze, or multiple large Foley catheters may be placed concurrently. An accurate count of what is placed in the uterus is critical to prevent retained foreign bodies.

Obstetrical Trauma
Rapid identification of the cause of PPH and the initiation of treatment should be simultaneous, including identifying genital lacerations. If lacerations are identified as the source of bleeding, they should be repaired quickly and bleeding should be reassessed. Transfer to an operating suite with anesthesia assistance may be indicated if uterine artery laceration is suspected. Genital tract hematomas may be suggested by clinical deterioration in the absence of other findings or genital pressure. Management of rapidly expanding hematomas includes packing, arterial embolization, or suturing.

Uterine Inversion
Uterine inversion, when the uterus protrudes through the introitus following delivery, is characterized by hypotension disproportionate to the amount of bleeding. Uterine inversion typically appears as a blue-gray mass protruding from the vagina. Immediate manual replacement of the uterus with the placenta in place (if not already detached) should be performed by pushing the protruding fundus with a steady pressure back through the vagina into the pelvis. However, if the uterus cannot be replaced, tocolytic agents (eg, nitroglycerin, terbutaline, magnesium sulfate, or halothane) should be administered to relax the uterus and cervix. If the uterus is still unable to be replaced, a laparotomy is performed, and the uterus can be replaced by gentle upward traction; the cervical ring may have to be incised to allow uterine replacement. Following uterine replacement, uterotonics should be administered to facilitate uterine contraction and control bleeding.

Retained Placenta
In patients with placenta accreta, a planned Cesarean delivery, with or without hysterectomy, is typically performed between 34 and 37 weeks gestation. Due to the complexity of the procedure, Cesarean hysterectomy involves interprofessional collaboration. Additionally, the ureters may be stented before the procedure to avoid injury. In patients suspected to have retained products of conception, manual intrauterine exploration or uterine ultrasonography is typically sufficient to diagnose retained products of conception (eg, succenturiate lobe). An ultrasound finding of an echogenic mass within the endometrial cavity immediately after delivery is consistent with a

retained placenta. For patients who have findings consistent with retained products of conception, manual removal or banjo curette, with or without ultrasound guidance, is performed. If the placenta is unable to be manually detached, the patient should be counseled on retained placenta complications and potential hysterectomy while being transferred to an operating room.

Coagulopathy

In patients with placental abruption or amniotic fluid embolus resulting in PPH, acute coagulopathy should be considered, as these conditions frequently cause a consumptive coagulopathy. Coagulopathies should also be considered in patients with secondary PPH. Blood transfusion and fluid replacement are required for most PPH caused by coagulopathies. Obstetrical transfusion protocols comprising packed red blood cells (RBCs), fresh-frozen plasma, and platelets in various ratios are utilized to correct coagulation deficits and maintain the hemoglobin level at more than 7 to 8 g/dL, the fibrinogen level greater than 2 g/L, and the platelet count at 50,000 to 75,000 µL.[10]

Hemodynamic Management

Implementing an effective blood transfusion protocol when the underlying etiology is being assessed and treated is critical to patient resuscitation and achieving hemodynamic stability. Severe hemorrhage leads to acidosis, hypothermia, and coagulopathy, which must be quickly corrected to avoid the risk of mortality. Therefore, other patient status indicators, including heart rate, respirations, peripheral oxygen saturation, temperature, and blood gases, should be monitored in addition to blood pressure. In patients with severe PPH, a massive transfusion protocol must frequently be initiated, which is standard in most institutions. Massive transfusion is defined as a transfusion of more than 10 units of packed RBCs within 24 hours or 4 RBC units in 1 hour with additional units anticipated. Most massive transfusion protocols recommend a combination of packed RBCs, fresh-frozen plasma, platelets, and cryoprecipitate in fixed ratios. The most common ratio utilized is 1:1:1 (1 unit of packed RBCs, 1 unit of fresh-frozen plasma, and 1 unit of platelets); other recommended protocols include 6:4:1 and 4:4:1. Lactate and electrolyte levels must be monitored throughout the transfusion to determine adequate vascular perfusion and correct metabolic abnormalities. Hyperkalemia, hypocalcemia, and hypomagnesemia commonly occur secondary to massive transfusion, and lactic acidosis indicates persistent tissue hypoperfusion. Complications associated with massive transfusions include transfusion-related lung injury, pulmonary edema, transfusion-associated circulatory overload, and blood transfusion reactions. Other adjunct strategies that may be considered include recombinant activated factor VII, and vasopressors (eg, phenylephrine or norepinephrine). Clinicians should discuss the need for blood transfusion with all patients who are pregnant during their prenatal visits. Following patient stabilization, decisions for additional transfusions should be based on clinical symptoms and laboratory studies.

Prevention of PPH: Active Management of the Third Stage of Labor (AMTSL)

After childbirth, blood loss and other clinical parameters should be closely monitored.[22] There is insufficient evidence to recommend quantification of blood loss over clinical estimation. Preventing PPH can reduce the number of women who die or suffer each year due to excessive bleeding associated with pregnancy. Most incidences of PPH are preventable. Several conditions can predispose a woman to hemorrhage. However, 90% of women have no risk factors. As a result, every woman must have access to a skilled birth attendant (SBA), who can manage labour and childbirth to minimize risk.[22] Routine active management is superior to expectant management in terms of blood loss, postpartum hemorrhage, and other serious complications of the third stage of labor. Active management should be the routine management of choice for women expecting to deliver a baby in a maternity hospital.[23]

Several Cochrane reviews have reported on the prophylaxis for the third stage of labor for women delivering vaginally.[24-27] According to these review studies, the risk of PPH can be reduced with both active management and the use of prophylactic uterotonics in the third stage of labor. The AMTSL involves the use of interventions (including the use of uterotonics, early clamping of the umbilical cord, and controlled cord traction) to expedite delivery of the placenta to reduce blood loss. In expectant management, signs of placental separation are awaited, and the placenta is delivered spontaneously. According to a Cochrane systematic review, for women at mixed levels of risk of bleeding, active management resulted in a reduction in the average risk of maternal primary hemorrhage at the time of birth (more than 1000 mL; average risk ratio [RR] 0.34, 95% CI 0.14–0.87) and maternal Hb less than

9 g/dL following birth (average RR 0.50, 95% CI 0.30–0.83).[28]

• All the three steps AMTSL should only be done by SBA/trained staff.

i. Administration of uterotonics after delivery of baby

ii. Delayed cord clamping

iii. Controlled cord traction

• AMTSL is a prophylactic intervention recommended by WHO. The prevention of PPH in AMTSL, over the years. consists of: 1) Giving uterotonic immediately after baby is born, 2) Controlled cord tension, and 3) uterine massage. The three procedures can be combined in one step of care with the administration of uterotonics, as it increases uterine contraction immediately after delivery.[29] All women giving birth should be offered uterotonics after cesarean or vaginal delivery of the baby for PPH prevention.

• Oxytocin IV injection has been the the uterotonic of choice for AMTSL over the years. Oxytocin (10 IU, IM) is the preferred uterotonic based on studies on the safety and effectiveness of uterotonics.

• If oxytocin is not available, room temperature stable carbetocin (100 mcg IM/IV), or methylergometrine (0.2 mg IV/IM), or misoprostol (800 to 1,000 mcg rectally or 600 to 800 mcg sublingually or orally) can be the first-line choices.

• Caution should be exercised when opting for ergot derivatives (methylergometrine) for the prevention of PPH as these drugs have clear contraindications in women with hypertensive disorders. Thus, it is probably safer to avoid the use of ergot derivatives in unscreened populations.

• If a skilled attendant is not present, and oxytocin is not available (such as at an unattended home birth), administer 600 mcg of oral misoprostol. Women delivering without a skilled attendant also need uterotonic for PPH prevention, so oral misoprostol should be given by a community health worker who is present.

• Delayed cord clamping (performed after 1 to 3 minutes after birth) is still recommended for all births to reduce newborn anemia while beginning essential newborn care at the same time.

• The uterus is palpated abdominally, and when it is contracted (this happens in 1 to 3 minutes of administration of uterotonics), controlled cord traction is done to deliver the placenta.

• Control Cord Traction (CCT) is not recommended in situations where SBA is not available.

• Suture any perineal or labial tears/ episiotomy quickly.

• Continue to palpate the uterus frequently to see that it stays firm (contracted).

• Help the mother to feed and care for her baby.

Overview of Uterotonics (Post-Delivery)

Drug	Dosage	Action	Side effects	Contraindication
Oxytocin	10U IM/IV	Onset: 1–3 mins Lasts: 10–15 mins	Minimal	• Allergic to oxytocin • Cardiac dysfunction (to minimize risk of volume overload) • Obstructed labour • Grand multiparity (relative contraindication)
Methylergometrine	0.2mg IV/IM	Onset: 2–7 mins Lasts: 2–4 hours	Nausea, vomiting, headache, hypertension	• Hypertension • Cardiac disease
Prostaglandin F2□	250mcg IM	Onset: 1–2 mins Lasts: 15–20 mins	Vomiting, diarrhea, bronchospasm	• Bronchial asthma
Misoprostol	800 to 1,000 mcg rectally or 600 to 800 mcg sublingually or orally	Onset: 3–5 mins Peak: 20–30 mins Lasts: <75 mins	Shivering, rise in temperature	• Pre-existing cardiovascular disease
Carbetocin room temperature stable	100 mcg IV/IM	• Rapid onset of action (within 2 minutes for both IV and IM administration) • Long half-life, and prolonged duration of action (60 min for a single IV injection, 120 min for an IM injection)	Generally well tolerated, Vomiting, abdominal pain, headache, tremor, dizziness, chest pain	• Serious cardiovascular disorders • In women with hepatic or renal disorders • Epilepsy • Hypersensitivity to carbetocin, oxytocin or any of the excipients according to the composition • Pregnancy and labour before the delivery of the infant • Must not be used in induction of labour

FIGO recommendations for the prevention and treatment of Postpartum Hemorrhage

Health workers at all levels of care (particularly in LMICs) need to have access to appropriate medications1 and training in PPH prevention and management procedures. All attempts should be made to reduce PPH using cost- effective, resource- appropriate interventions. At first, all should be done to avoid PPH and reduce the need for expensive, lifesaving surgical interventions. The routine use of active management of the third stage of labor by all attendants, regardless of where they practice, should be recommended. All birth attendants must know how to provide safe care (physiologic management) to prevent PPH in the absence of uterotonic drugs.

FIGO recommendations for prevention of postpar-

tum hemorrhage

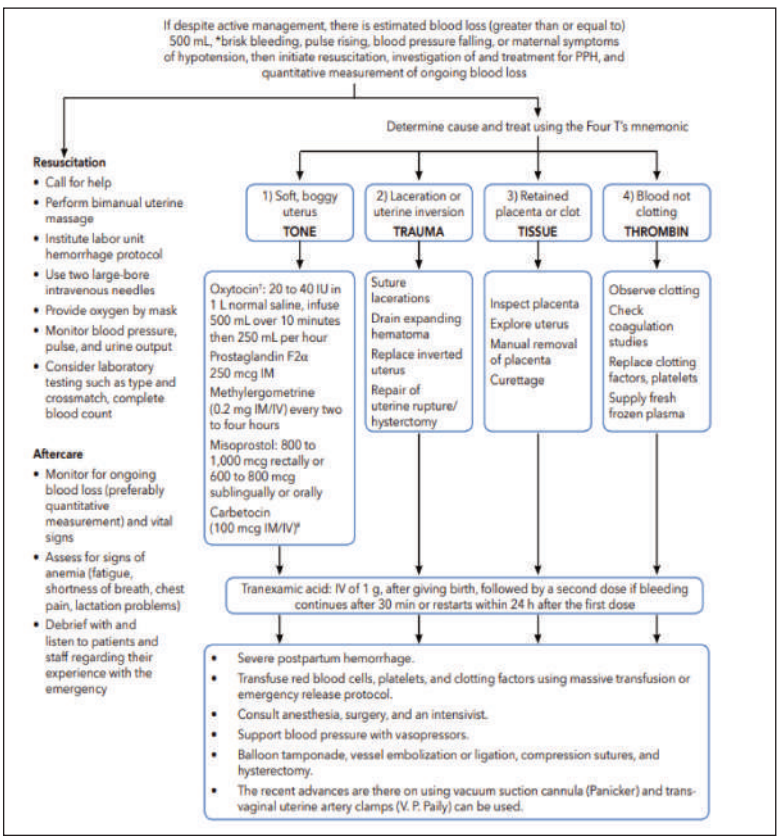
1. The use of uterotonics for prevention of PPH during the third stage of labor is recommended for all births. Oxytocin (10 IU intravenously/intramuscularly [IV/IM]) is recommended for the prevention of PPH for vaginal delivery and cesarean section.In settings where oxytocin is used, attention should be paid to the oxytocin cold chain.
2. In settings where oxytocin is unavailable or its quality cannot be guaranteed, the use of other injectable uterotonics (if appropriate ergometrine/methylergometrine 200 µg IM/IV; hypertensive disorders can be safely excluded prior to its use) or oral misoprostol (400–600 µg orally) or carbetocin 100 µg IM/IV is recommended for the prevention of PPH.
3. The combinations of ergometrine plus oxytocin or misoprostol plus oxytocin may be more effective uterotonic drug strategies for the prevention of PPH ≥500 ml compared with the current standard, oxytocin. This comes at the expense of a higher risk of adverse effects (vomiting and hypertension with ergometrine and fever with misoprostol)
4. In settings where skilled birth attendants are not present to administer injectable uterotonics and oxytocin is unavailable, the administration of misoprostol (400– 600 µg orally) by community healthcare workers and lay health workers is recommended for the prevention of PPH.
5. In settings where skilled birth attendants are unavailable, controlled cord traction (CCT) is not recommended.
6. Sustained uterine massage is not recommended as an intervention to prevent PPH in women who have received prophylactic oxytocin.
7. Postpartum abdominal uterine tonus assessment for early identification of uterine atony is recommended for all women.
8. Oxytocin (IV or IM) and CCT is the recommended method for removal of the placenta for the prevention of PPH in cesarean delivery.

FIGO recommendations for treatment of postpartum hemorrhage

1. Intravenous oxytocin alone is the recommended first- line uterotonic drug for the treatment of PPH.
2. If intravenous oxytocin is unavailable, or if the bleeding does not respond to oxytocin, the use of intramuscular ergometrine, oxytocin– ergometrine fixed dose, or a prostaglandin drug (including sublingual misoprostol, 800 µg) is recommended.
3. There is no evidence about the safety and efficacy of an additional 800- µg dose of misoprostol for treatment of PPH when given to women who have already received 600 µg of prophylactic misoprostol orally.
4. The use of isotonic crystalloids is recommended in preference to the use of colloids for the initial intravenous fluid resuscitation of women with PPH.
5. Early use of intravenous tranexamic acid as soon as PPH is diagnosed but within 3 h of birth in addition to standard care is recommended for women with clinically diagnosed PPH following vaginal birth or cesarean delivery
6. Administration of 1 g (100 mg/ml) tranexamic acid intravenously at 1 ml/min (i.e. administered over 10 min), with a second dose of 1 g intravenously if bleeding continues after 30 min, or if bleeding restarts within 24 h of completing the first dose. Reducing maternal deaths due to bleeding through scaling up of tranexamic acid for PPH treatment could have a positive impact on health equity and improve outcomes among disadvantaged women, especially in LMICs.
7. Uterine massage is recommended for the treatment of PPH.
8. The use of bimanual uterine compression or external aortic compression for the treatment of PPH due to uterine atony after vaginal birth is recommended as a temporizing measure until appropriate care is available.
9. If women do not respond to treatment using uterotonics, or if uterotonics are unavailable, the use of uterine balloon tamponade is recommended as an effective nonsurgical technique that can potentially improve survival in women with PPH due to uterine atony after ruling out retained products of conception or uterine rupture as a contributing factor.
10. Use of the nonpneumatic antishock garment is recommended as a temporizing measure until appropriate care is available.
11. The use of uterine packing is not recommended for the treatment of PPH due to uterine atony after vaginal birth.
12. Uterine artery embolization can be another conservative management measure for PPH if technical conditions and skilled human resources are available for its use.
13. If bleeding does not stop despite treatment using uterotonics and other available conservative interventions (e.g. uterine massage, balloon tamponade), the use of surgical interventions is recommended. Surgical interventions include the use of compression suture techniques, uterine and hypogastric artery ligation, and hysterectomy.

14. The priority is to stop the bleeding before the patient develops coagulation problems and organ damage from under- perfusion. Conservative approaches should be tried first, rapidly moving to more invasive procedures if these do not work

Clinical and pharmacological management of PPH (modified flowchart)[40]
Management for Delayed PPH



Hemorrhage between 24 hours and 6 weeks postpartum is termed "delayed PPH." Common causes include retention of placental tissue and/or membranes and infection leading to endometritis, endomyometritis, and parametritis. Bleeding can be sudden and profound, resulting in rapid cardiovascular collapse.[30] Septic shock may also be present due to infection. The investigations in these cases are similar to atonic PPH; however, some additional investigations of septic foci to isolate organisms for culture and antibiotic sensitivity are mandatory. These cases may have early features of DIC, so a blood coagulation profile should be done earlier.

The main stay of management include:

• Resuscitation and fluid therapy.
• Broad spectrum intravenous antibiotic therapy (to cover gram positive, gram negative, and anaerobes) according to the hospital antibiotic policy. It can be changed according to the culture report and antibiotic sensitivity pattern.
• Evacuation of the uterus/surgical management for any septic foci.
• Individualized surgical procedure may be adopted depending on the case
• Uterotonics and tranexamic acid may be needed.
• Blood and blood products may be given depending on the haemoglobin and coagulation profile.

Prognosis
Post-partum hemorrhage is a leading cause of maternal and fetal morbidity however, correct and timely institution of treatment can vastly improve patient outcomes. Patients who had PPH in a previous delivery are at risk of having PPH in subsequent deliveries. Implementation of standardized PPH protocols and interprofessional simulation training has helped improve patient outcomes. Additionally, increased efforts to enhance coordination among healthcare team members in response to PPH have contributed to reduced maternal morbidity.

Complications
Blood loss occurs in PPH, putting the patient at risk of hypovolemic shock. When patients lose 20% of blood, they develop tachycardia, tachypnea, narrowed pulse pressure, and delayed capillary refill. This may lead to ischemic injury to the liver, brain, heart, and kidneys. Sheehan syndrome, or postpartum hypopituitarism, is also a complication of excessive blood loss seen in postpartum hemorrhage.

Complications related to PPH management include the following:
• Transfusion-related acute lung injury
• Infection
• Pulmonary edema
• Hemolytic transfusion reactions
• Intrauterine synechiae
• Preterm delivery
• Infertility

Consultations
An interprofessional approach is mandatory for the required outcomes in the management of PPH, including:

- Obstetrics and gynecology
- Maternal-fetal medicine
- General surgery
- Anesthesiology
- Hematology
- Urology
- Emergency medicine
- Laboratory personnel
- Interventional radiology

Enhancing Healthcare Team Outcomes

PPH necessitates a cohesive interprofessional team comprising physicians, technicians, nurses, pharmacists, laboratory personnel, and labor and delivery nurses. The primary focus is prompt resuscitation while pinpointing and addressing the underlying cause, often surgical. Maintaining patient hemodynamic stability is paramount for organ perfusion. The team ensures ample intravenous access and employs direct blood loss assessment, initiating protocols for blood products and massive transfusions. Simultaneously, rapid identification of the cause and treatment initiation occurs. Throughout this process, clear communication between team members and care coordination is critical to prevent missed diagnoses and delays in intervention. Resuscitation ideally unfolds in an operating department setting, allowing anesthesia assistance for complex laceration repairs, uterine inversion correction, analgesia provision, or surgical exploration if needed. Effective communication, clear delineation of responsibilities, and coordinated efforts among team members optimize patient-centered care, enhance outcomes, ensure patient safety, and elevate team performance in managing PPH.

SURGICAL MANAGEMENT OF PPH

Dr JP Rath
MD
Consultant Critical Care Obstetrics and Pelvic floor surgery

"Thou shall forever be wedded to the parturient uterus as an Obstetrician, for thee shall take decisions in the interest of thy patients to save her and her uterus, for then and only then, thine will be the Kingdom".

An Obstetrician is bound to face horrific situations, often unexpected, that may be "do or die"! Any and every case therefore should be handled with a high degree of care.
Maternal hemorrhage, defined as a cumulative blood loss of greater than or equal to 1,000 mL or blood loss accompanied by signs or symptoms of hypovolemia within 24 hours after the birth process, remains the leading cause of maternal mortality worldwide [(1)].
Anticipating, preparing and recognising the problem of PPH (post-partum haemorrhage), helps save lives. As increase in Caesarean section rates have inadvertently increased the risk to the mother for PPH.
Understanding the behaviour of the post-partum uterus becomes the cornerstone to the successful management, in countering PPH.
The uterus is a very vascular organ during late pregnancy, often drawing 15-20% of the blood volume, average of 600ml [(2)]. In a case of placenta previa, the supply increases substantially even up to 40%, making the pelvis a "bloody battlefield".
The first figure below depicts the blood flow in a normal gravid uterus. The lower uterine segment and vagina are highly vascular and show higher vascularity than the upper uterine segment.
The second figure depicts cases of central placenta previa with accrete where increased vascularity is seen extending laterally beyond the uterine walls.

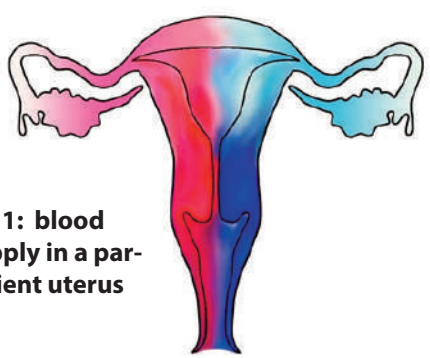


Fig 1: blood supply in a parturient uterus

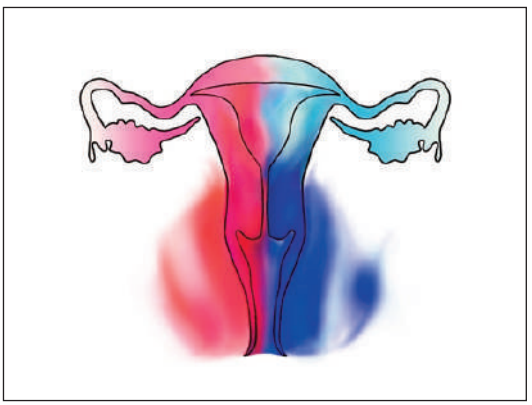


Fig 2: bloody battlefield in placenta previa accreta

In case of PPH following vaginal delivery, First Bundle Approach (Fig 3) should be immediately followed. Further, use of Bakri Balloon or tamponade with Condom catheter can be attempted in atonic PPH. Negative suction Tamponade has also shown encouraging results [(3)].
Uterotonics with proper dosing, fluid replacement and blood and blood product transfusion must be judiciously used.
In cases of well contacted uterus with ongoing PPH, inspection of the lower genital tract under appropriate anaesthesia and good visualization and repairing traumatized tissue in the tract should be the dictum.
On failing to control hemorrhage by the usual methods, laparotomy should be considered.

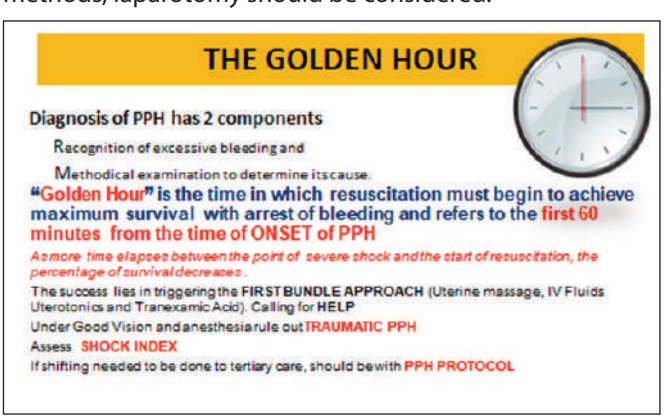


Fig 3: first bundle approach to PPH

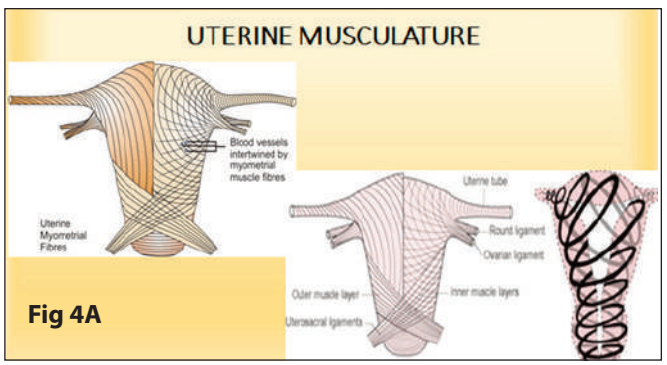


Fig 4A

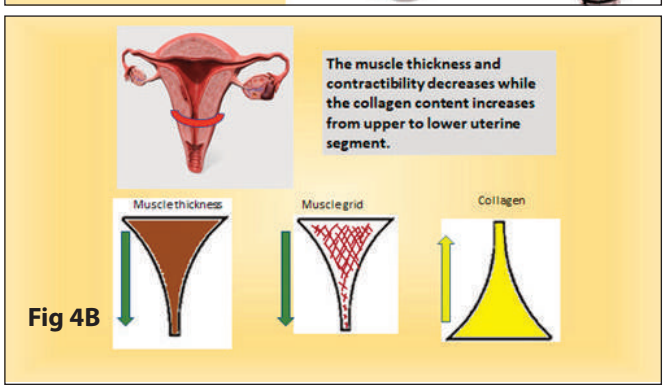


Fig 4B

Understanding the behaviour of the uterine musculature: fig 4A and 4B

The upper part of uterus (fundus and mid uterus) has a good amount of muscle fibres, which are strongly inter twined forming a formidable "muscle grid", which compresses the blood vessels effectively.
In the lower portion, the thickness as well as number of muscle fibres reduce and collagen increases as the uterus transitions from lower portion to the isthmus, and down to the cervix. This results in an increased risk of bleeding owing to the poor contractility of the tissue.
The vagina has fibro-muscular tissue which is congested in late pregnancy and can be subject to tears and hence torrential haemorrhage.
There is a preponderance of venous arcade in the lower part of uterus, cervix and vagina. Veins unlike artery are devoid of muscular layer and have a thin inelastic wall. They are often tortuous and bulged. They are therefore very prone to heavy bleeding.
The ideal way to deal with venous bleeding is to cut its arterial source, apply pressure and ultimately use of Fresh Frozen Plasma (FFP).
Fig (5) shows the distribution of blood in a parturient uterus, which gets exaggerated in Placenta previa. The table also shows the choice of material to be used, in an event of haemorrhage.

Finer calibre may be used for venous bunch or sinuses. The most commonly used suture material is polyglactin 910 (Vicryl) No. 1, however it has a poor knotting property and should be avoided.

Anatomical part	Blood supply- arterial and venous	Ideal suture material
Fundus	↑↑↑	Vicryl 1-0
Body	↑↑↑	Vicryl 1-0
Lower segment	↑↑↑↑ (↑)	Vicryl 1-0
Cervix	↑↑ (↑)	Catgut 1-0
vagina	↑↑↑↑	Catgut 1-0

Fig 5: distribution of blood in a parturient uterus and ideal suture material for achieving haemostasis

The Principles of controlling PPH during C sections are:
Devascularization of uterus – Uterine Artery Ligation, Ovarian Vessels Ligation
Compression of Uterus (Compression sutures)
Tamponade of Uterus – balloon, packs and also combining it with compression sutures
Internal Iliac Ligation
Obstetric Hysterectomy

USP size	Collagen (Catgut) Metric size	Sutures Diameter in mm	Polyglactin (Vicryl) Metric size	Sutures Diameter in mm	Quality and retention of knot
#2	6	0.6 to 0.699	5	0.5 to 0.599	Poor
#1	5	0.5 to 0.599	4	0.4 to 0.499	Average
# 0	4	0.4 to 0.499	3.5	0.350 to 0.399	Good
# 2-0	3.5	0.4 to 0.399	3	0.3 to 0.339	Excellent
#3-0	3	0.300 to 0.339	2	0.2 to 0.249	Weak

TABLE 1: Choice of Suture Material

The above table is a comparative study of the tensile strength and the diameter of the suture "number".
The best knotting is done by **2-0 vicryl** as highlighted in green. Then by 1-0 followed by number 1.
The knot integrity and stability is superior **2-0 vicryl**.
The above table shows tensile strength vs. diameter, and knot quality.
Knot quality is poorest with No.1 vicryl.
This is well demonstrated in Fig (6), wherein the suture material is mapped vis.-a-vis the tensile strength, knot integrity and knot stability.

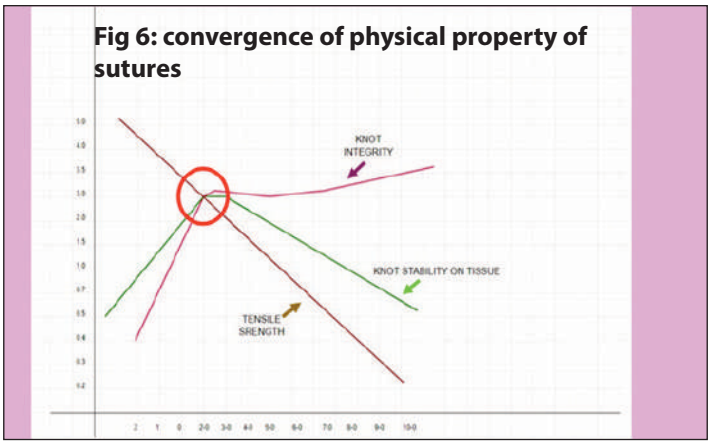


Fig 6: convergence of physical property of sutures

Devascularization of uterus – Uterine Artery Ligation

Uterine Artery is the principal source of blood to the gravid uterus (80%). The remainder is from the ovarian supply. In cases of Placenta previa – accreta syndrome however other large source of blood supply can be from the Superior Vesical A, Vaginal A as well as branches of Femoral A, Inferior Epigatric A and Circumflex Iliac A. These form an arterial arcade, making the whole pelvis a "lake of blood".

Prudent use of a Swab holder Fig (7) can immediately control bleeding as seen in Fig (6). By simply clamping the lower segment below the uterine incision, completely obliteration of blood supply from both uterine and ovarian vessels can be achieved, giving ample time and scope for the Obstetrician to make further decisions with ease.

Clamping of the Common Iliac Artery can also be done effectively and easily as shown in Fig (12).

Aortic Clamping by Debakey Clamp as espoused by Dr Vivek Joshi [4], is another method employed in cases of accreta.

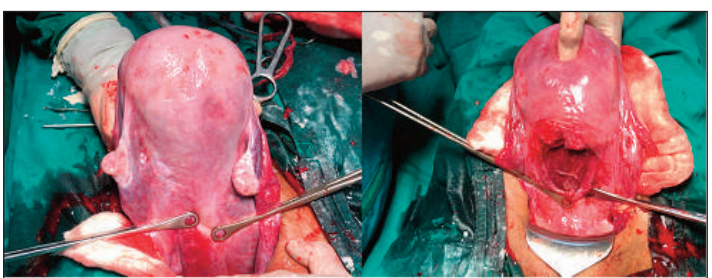


Fig 7: clamping of vessels supplying uterus by swab holder

Uterine Artery ligation (UAL) Fig (8A) can be easily approached posteriorly after exteriorizing the uterus, out of the incision. Inclusion of the entire bunch of uterine vessels along with musculature gives support to the vessels so that they do not lacerate. Use of 1-0 Vicryl sutures ensures an effective knot.

For patients with dense posterior adhesions or any difficult pathology, an anterior ligation may be done (Fig 8B). This can be replicated in subsequent surgery as anastomotic channels develop well. Anteriorly the Uterine A may be tied by isolating the bunch necessarily below the uterine incision with due precaution. [NOTE: the distance of ligation is more than 3 inches from ureter and hence the posterior approach is encouraged. Fig (9)]

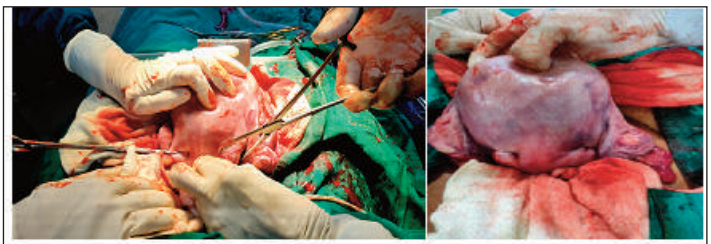


FIG 8A: Posterior approach to Uterine artery ligation

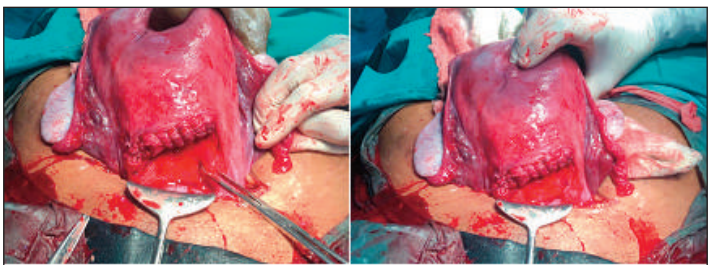


Fig 8B: Anterior approach to Uterine artery ligation

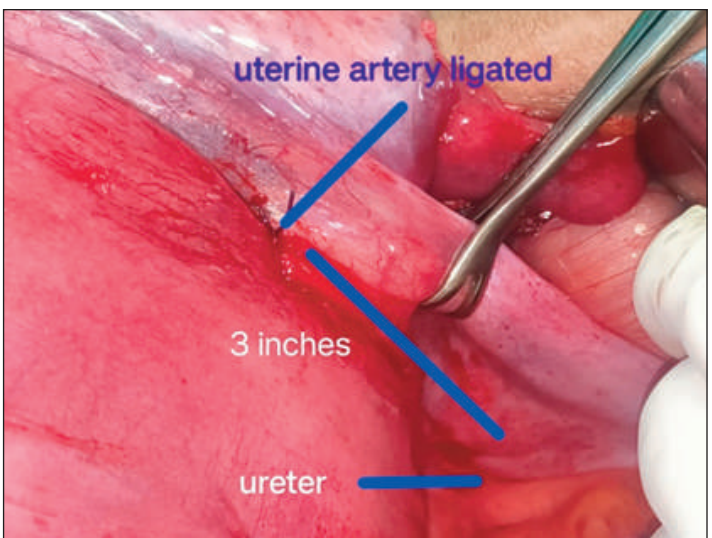


Fig 9: Uterine artery ligation and distance from the ureter

Internal Iliac Ligation (IIAL) is another effective way to prevent bleeding. The need for internal iliac ligation however is less as the previous methods arrest bleeding in most cases. During a massive pelvic hemorrhage or peripartum bleeding, bilateral ligation of the IIAL reduces the pelvic arterial blood flow by 49% and pulse pressure by 85% [5]. In the long term period, post bilateral ligation of IIA the collateral circulations maintain the re-functioning of the IIA.

Internal iliac artery ligation however is mandatory in cases of placenta accreta or previa where the congestion is intense, to reduces bleeding before proceeding with obstetric hysterectomy. Internal iliac ligation has also been described in many articles as a first line treatment for PPH.

During the ligation, care must be taken in removing the fascial sheath investing the vessels on the pelvic wall. After dissection, the Internal Iliac Artery can be seen.

The common Iliac divides and continues to the lower limb as External Iliac / Femoral A. Ureter is never at risk of injury, unless there is distortion of anatomy. While ligation the internal iliac artery ensure correct use of a non-traumatic Mixter's forceps passed lateral to medial with additional care to not injure the Internal Iliac Vein. The knot may be tied using 2-0 vicryl, linen 4-0 or by vascular clips. The IIAL is done after 1 inch of its bifurcation, after the posterior division is given out Fig (10). In an event of trauma to the vein, deep sustained pressure should be put for over 5 minutes, which suffices in controlling bleed due to a small rent. In case of larger avulsion, help from a Vascular Surgeon may be called for, using 6-0 Vicryl for its repair. Then an abdominal drain No 28 should be kept in the pelvis.

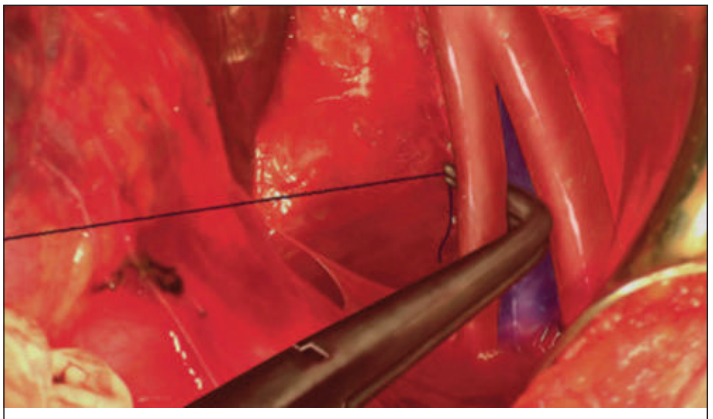


Fig 10: Internal iliac artery ligation (IIAL)

Modified B Lynch is essentially a compression suture devised by Christopher B. Lynch for atonic PPH, using Catgut. The Modification is use of Vicryl N0 1 (2347). Shouldering of the suture, folding of the fundus over the lower uterine segment is done which may be combined with Tamponade using Foleys catheter inflated with 50-60 ml of fluid in the uterine cavity. This is very effective in countering hemorrhage from predominantly venous origin, in the lower half of Uterus. Fig (11) show all the modifications mentioned.

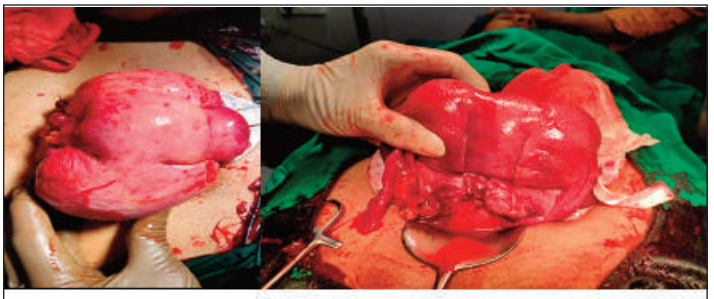


Fig 11: modified B Lynch suturing – top & front view

The compression is done in a manner to fold the uterus over the lower segment after shouldering (going through the fundus deep about an inch medial to the cornu) the uterine fundus, tightly. If the lower segment is oozy, a Foleys Tamponade may be used within the cavity. This is usually combined with Uterine Artery Ligation, and has proved to be very effective method.

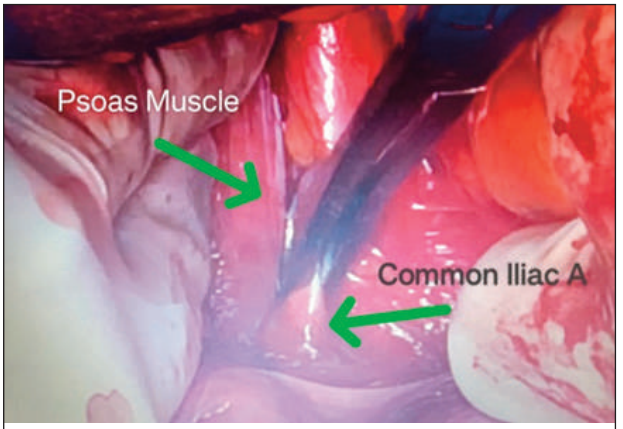


Fig 12: clamping of common iliac artery

Obstetric Hysterectomy: This is done as a last resort to save mother's life. The decision should be made wisely and in time, both haste and delay can prove difficult. The usual clamp, cut and ligate is done using Vicryl 1-0 (2346), and may require additional knotting with Vicryl 2-0. However using thicker Polyglactin su-

tures often causes oozing from bed.
Uterine atony and uterine rupture were formerly regarded as the commonest indications necessitating emergency hysterectomy. However, more recent reports have listed placenta accreta as the most common indication [6]. As we see an increasing number of Caesarean deliveries, we should also expect an increasing incidence of accreta syndrome. Always treat all previous section, or myomectomy with a low lying placenta, especially anteriorly placed, to be accreta with all preparation in advance. It is better done electively, and certainly has better outcomes. This should be done in tertiary care centre.
In the event of an 'on table surprise', after delivery of the baby through an upper segment uterine incision, exteriorization of uterus should be done without attempted removal of the placenta. Immediate clamping of major vessels should be done as explained before. It is prudent to do an Internal Iliac Artery Ligation before proceeding with obstetric hysterectomy.
Many authors believe in leaving the placenta behind for removal at a later time or spontaneous expulsion, however in these cases the blood loss is more. Subtotal hysterectomy may be insufficient, as adherent placenta may have additional vessels supplying blood from the Superior Vesical and Vaginal arteries.
The hazards are plenty which include massive haemorrhage as high as 3000ml [7], injuries to urinary system, DIC, shock, sepsis and unfortunate demise. Use of FFP and blood components should be liberal, keeping in mind the delicate fluid balance.
ICU care with Intensivist, Haematologist and other specialities may be required which helps improve outcome.

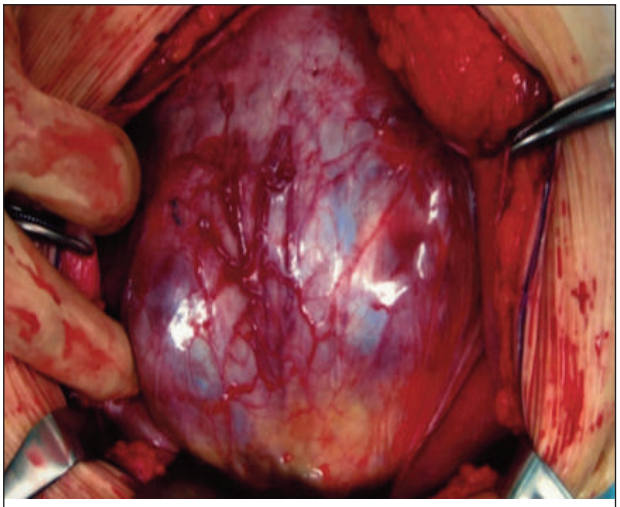


Fig 13 A: anterior placenta accreta

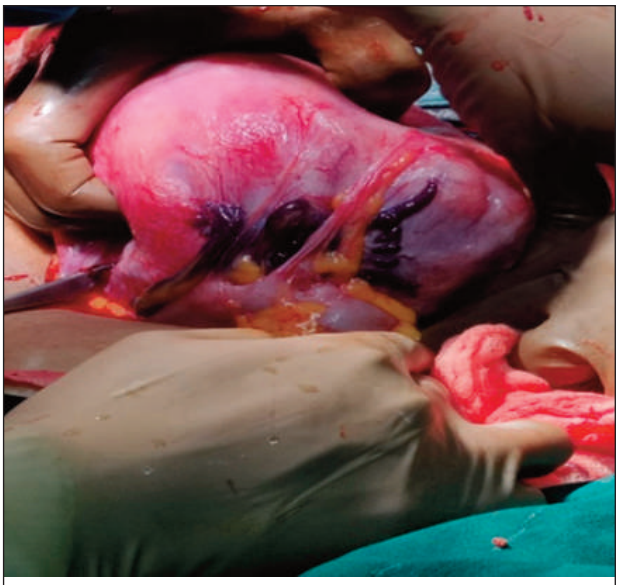


Fig 13B: posterior placenta accreta invading the rectum

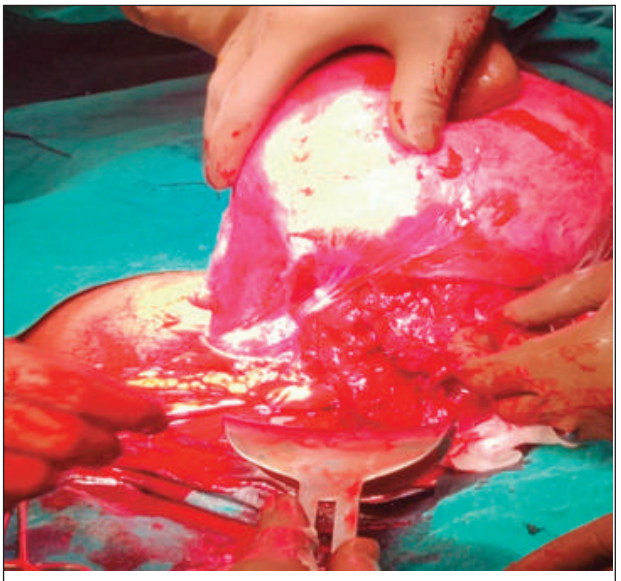


Fig 14: eventeration of uterus

In the above case, after eventeration of uterus, clamps were applied. Then placenta was removed, Internal Iliac ligation along with B Lynch suturing done. The uterus was conserved Fig (14).

The next Fig (15) is of an interesting case of Global Placenta increta which required hysterectomy and required massive transfusion. There was damage to bladder which was sutured in 2 layers. Healing and re

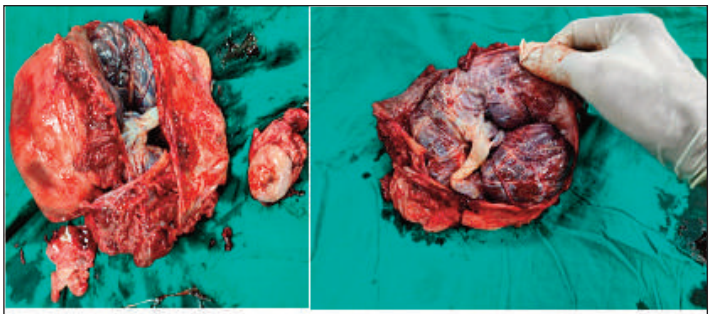


Fig 15: global placenta increta

covery was good.

Strategy:
1. Call for all hands to help (must include a senior Obstetrician experienced to handle such cases, 2 Anaesthesiologist, Neonatologist, well equipped OT, Facilities for Blood and components, preferably an Intensivist and Haematologist for guidance.
2. A large bore intravenous line, of either 16/18 gauge on both arms (Central Subclavian may be put) with rapid infusion of Crystalloids. Always have blood and components handy, begin with FFP followed by Packed Cells. Often Massive transfusion protocol may be initiated.
3. Clear counselling of relatives and written consent for the said procedures must be taken.
4. No delay in the decision making.
5. Serial clamping and devascularisation of uterus, tourniquet with Foley's catheter may be attempted to temporarily cease bleeding.
6. Tamponade with in dwelling Foley's catheter combining with Modified B Lynch Suturing.
7. Internal Iliac Ligation.
8. Obstetric Hysterectomy as the final choice with necessarily a wide bore drain and perhaps Subclavian cannulation and ICU care.
9. Judicious use of Crystalloids, colloids and blood products.
10. Multi-disciplinary approach.
11. Proper documentation.
12. Debrief and audit.
13. Tender Loving care of patient and relatives.

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IV FLUIDS AND BLOOD AND BLOOD COMPONENTS

The speciality of Obstetrics and gynaecology deals with a lot of blood. Obstetrics is also called "bloody business". Postpartum haemorrhage (PPH) is a leading cause of maternal mortality in developing countries. Each year 14 million women experience PPH resulting in about 70,000 maternal deaths globally. Ruptured ectopic pregnancy, abortion complications including septic abortion, antepartum haemorrhage also pose great threat to maternal life. Gynecological problems of heavy menstrual bleeding, gynaecological surgery are also closely related to blood loss. Large vessel injury is one of the major complications in gynaecologic oncologic surgery. Therefore, thorough knowledge of intravenous fluid resuscitation and blood component therapy is integral to a practicing obstetrician and gynaecologist.

Fluid resuscitation is defined as a rapid administration of intravenous fluids used to restore or maintain a patient's circulatory volume during severe hypovolemia or shock due to significant and sudden fluid or blood losses. The most common indications of fluid resuscitation in critically ill patients are severe hypovolaemia, sepsis, trauma, burn, and perioperative volume loss . The objective of fluid resuscitation is to quickly administer a large fluid volume to restore circulating volume, stabilize hemodynamics, and thereby restore tissue perfusion and oxygen delivery without causing harm due to fluid overload Identifying the cause of shock and treating it simultaneously is vital.

The complicated obstetric patient may have a number of additional factors that make fluid management more challenging. Cardiac, pulmonary, renal disease (pre-existing or acquired during pregnancy), sepsis, haemorrhage and hypertensive disorders of pregnancy all pose management dilemmas regarding the administration and optimization of fluid therapy. Injudicious or aggressive fluid therapy and volume overload have been associated with harm in a number of settings, including precipitation of pulmonary oedema.

CHOICE OF FLUID THERAPY

Volume resuscitation with crystalloids, colloids and blood products all have their own advantages and disadvantages. Crystalloids are isotonic and designed to replace losses within in the extracellular compartments therefore requiring large amounts when used to replace intravascular losses. Infusion of large amounts of crystalloids may induce acidosis, dilutional coagulopathy, formation of interstitial oedema and impairment of the microcirculation which can lead to the lethal triad of acidosis, hypothermia, and coagulopathy. Colloids such as hydroxyethyl starches designed to replace plasma deficits, may impair clot formation and lead to faster clot disintegration. Infusing a large amount of colloids can disrupt the haemostasis and as such actually increase the bleeding. Blood products will replace the lost coagulation factors, but are a scarce commodity and are not without risks.

The choice of resuscitation fluid depends on the severity and etiology of hypovolemic shock. Three major categories of fluids used for resuscitation are:

1. Crystalloid fluids (normal saline, Ringer's lactate, and other chloride-restrictive balanced crystalloids such as PlasmaLyte)
2.Colloids (albumin, hydroxyethyl starch (HES), dextran, and gelatine)
3. Blood products (packed red blood cells).

Normal saline and Ringer's lactate are generally chosen as initial fluids for treating Hypovolemic shock: Crystalloid fluids such as normal saline and Ringer's lactate are sodium-rich electrolyte solutions and therefore are distributed only in the ECF compartment (25% in intravascular and 75% in interstitium). Infusion of 1 liter of these fluids will expand intravascular volume by about 250 ml, so the blood pressure rise will be much more rapid compared to 5%-dextrose.

Balanced crystalloid solutions (e.g., Ringer's

lactate, Plasma-Lyte) have progressively displaced normal saline as preferred initial resuscitation fluids. A meta-analysis by Williams et al. (2024) encompassing 12 studies with over 4,500 obstetric patients demonstrated that balanced solutions were associated with reduced incidence of hyperchloremic acidosis, lower rates of acute kidney injury, and improved maternal outcomes compared to normal saline Among the initial strategies for reanimation, the administration of crystalloids in small boluses of 500 ml is recommended.10 Scientific evidence recommends the use of balanced crystalloid solutions such as Ringer's lactate owing to the risk of hyperchloremic acidosis and the worsening of kidney function with chlorine- rich fluids (saline solution).7 This is particularly important for LMICs, where saline- based solutions are in abundance

Crystalloids vs Colloids

• Crystalloids and colloids are both effective, but evidence of comparative superiority and significant benefits of colloids are lacking; therefore, current trends and recommendations including the recent Surviving Sepsis Campaign guideline (2021) and the European Society of Intensive Care Medicine fluid therapy guideline (2024), favor crystalloids over colloids for the resuscitation of nonseptic and septic patients and no indications currently exist for the routine use of colloids over crystalloids.

• The potential benefit of colloids to provide better hemodynamic stability is due to their effectiveness in achieving greater, rapid, and prolonged intravascular volume expansion.

• Colloid solutions other than albumin (e.g., Hydroxyethyl starch dextran, gelatin) are not used routinely because of lack of benefits, safety, and potential adverse effects. Several studies have demonstrated increased risks, including tubular necrosis and acute kidney injury (AKI), associated with synthetic colloid treatment Isotonic crystalloids are used as first-line fluid resuscitation until appropriate blood and blood products become available in the haemorrhaging obstetric patient.

COMPLICATIONS OF FLUID THERAPY

Fluid therapy may be harmful if the incorrect fluids are given, if fluids are given in inadequate amounts, or if too much fluid is administered. starches have been associated with excess renal failure and mortality in ICU populations[2] and should be avoided.

Excess crystalloid fluid administration in women with postpartum hemorrhage may result in worsening anemia, shock and coagulopathy. Use of hypotonic (or less commonly hypertonic) fluids may lead to severe dysnatremias and other electrolyte abnormalities, with resultant potentially catastrophic neurological complications. Excessive administration of chloride-rich fluids may lead to a normal anion gap metabolic acidosis. Normal pregnancy is marked by an increase in the maternal circulating volume. Both pre-existing and superimposed conditions such as cardiac disease and renal dysfunction may exacerbate this, and hypertensive disorders of pregnancy may be associated with significant edema and varying degrees of volume state disturbance. Pre-eclamptic women are also at considerably increased risk of developing pulmonary edema, cerebral edema which has been associated with increased maternal mortality.

Specific Scenarios

1. Hypertensive disorders of pregnancy

The hypertensive disorders of pregnancy are a unique group of disorders and one among the leading causes of intensive care admission in the obstetric population. These gravidas have excessive extracellular fluid that has inappropriately extravasated from intravascular compartment in pre-eclampsia. Injudicious fluid management elevates the risk of pulmonary oedema and cerebral edema. Controlled conservative fluid administration is preferred in patients with severe preeclampsia. Crystalloid fluid is given at a rate between 60 and 125 ml/hr. NICE guideline recommends crystalloid fluid is given at a rate of 80 ml/hr. For labour analgesia with neuraxial analgesia, crystalloid solution are infused slowly in graded amount

2. Sepsis

Initial fluid resuscitation in sepsis is vital to restore circulating volume and prevent sepsis induced end organ dysfunction secondary to hypo perfusion. As per Surviving Sepsis Campaign recommendations (2021), in the resuscitation from sepsis-induced hypoperfusion, at least 30 mL/kg of IV crystalloid fluid should be given within the first 3 hours . An initial target of fluid resuscitation is to achieve a mean arterial pressure (MAP) of 65 mmHg . Following initial fluid resuscitation, additional fluid infusion is planned based on a frequent assessment of clinical parameters, hemodynamic status, and laboratory tests.

3.Trauma

The need to exclude fetomaternal haemorrhage, assess for foetal well-being and consider imminent delivery is emphasized. Fluid resuscitation for hypovolemia in trauma is generally suggested to follow that in the non-pregnant woman (with lateral displacement of the uterus to avoid aortocaval compression). Excessive use of crystalloid fluids should not occur, and blood and

blood products should be administered early.

4. Cardiac Disease

Fluid management poses a challenge in pregnant women with heart disease

Hypotension can be life threatening in Aortic stenosis, Pulmonary Arterial hypertension.

Patients with Mitral Stenosis are at risk of pulmonary oedema from fluid overload, so these women during labour are best to be managed on dry side.

5. Obstetric haemorrhage

Management of PPH consists of a combination of treatments with uterotonics, intervention surgery, coagulation support, and resuscitation with clear fluids and blood products. All treatments aim to resolve the cause of the bleeding, whilst keeping the parturient haemodynamically stable.

There are two strategies for fluid resuscitation in patients with hemorrhage:

1. Aggressive approach refers to the traditionally used strategy in which the key principle is restoring the effective circulating blood volume, and rapid normalizing of blood pressure with administration of large amounts of crystalloids.

Aggressive resuscitation may worsen coagulopathy and hemorrhage by increasing intravascular hydrostatic pressures, diluting coagulation factors, and inducing more hypothermia, which results in deterioration of the triad of death. Increased fluid administration leads to decreased concentrations of fibrinogen, hemoglobin, hematocrit, platelet count associated with prolonged prothrombin time, and partial thromboplastin time. The study also demonstrated that administration of >4 L of fluids is associated with subsequent bleeding and adverse maternal outcome.

2. Hypotensive resuscitation Approach, also called permissive hypotension, consists of restrictive crystalloid resuscitation during the early stages of a hemorrhagic shock to maintain lower than normal systolic or mean blood pressure, sustaining organ perfusion until control of the bleeding occurs. The concept of hypotensive resuscitation is because administering small crystalloid volumes reduces the risk of dilutional coagulopathy and maintaining a lower blood pressure is less likely to disintegrate the pre-formed blood clots. In contrast, hemostatic reanimation is based on early and aggressive blood product replacement, transfusing red blood cells(PRBC), fresh frozen plasma (FFP), and platelets (PLT) in the same proportion as found in circulating blood to correct coagulopathy. Hypotensive resuscitation and hemostatic reanimation are the fundamentals for Damage control resuscitation.

Intravenous fluids: Among the initial strategies for reanimation, the administration of crystalloids in small boluses of 500 ml is recommended. After the administration of each bolus, physicians must assess the clinical status of patients, looking for an improvement in signs and symptoms of shock resulting from blood loss.

Targeted blood pressure: The difference between aggressive and hypotensive resuscitation lies within targeted blood pressure management. Mean arterial pressure (MAP) represents the perfusion of the majority of organs, therefore providing the target for clinicians to guide fluid administration. The European guideline on management of major bleeding and coagulopathy following trauma recommends permissive hypotension with a systolic blood pressure target of 80–90 mm Hg (MAP 50–60 mm Hg) until major bleeding has been controlled urine output >0.5 mL/kg/hr, and normal mental status.

BLOOD COMPONENT THERAPY

Blood transfusion may be required during pregnancy in patients with anemia and acute haemorrhage. They are required in cases of massive haemorrhage (PPH, trauma, intraop loss)

During Antenatal period

- Pregnancy less than 34 weeks
  - o Hb less than 5 gm/dL (with/without signs of heart failure)
  - o Hb between 5–7 gm/dL (with signs of heart failure)
- Pregnancy more than 34 weeks
  - o Hb less than 7 gm/dL (without signs of heart failure)
  - o Severe anemia with heart failure
- Anemia due to acute haemorrhage
  - o Hb at or below 6 gm/dL
  - o Ongoing hemorrhage with hemodynamic instability (Hb estimation not needed)

BLOOD COMPONENTS

1. Packed Red Blood Cells (PRBC)

PRBC is prepared by the centrifugation of whole blood, achieving a hematocrit of 70–80%. Each bag raises Hb level by 0.5 g to 1 g/dL.

Practice Recommendations

PRBC tranfustion indication in Obstetrics:

- Severe anemia (Hb <7 g/dL) due to any cause
- Hemoglobinopathies
- Obstetric hemorrhage

The indications of irradiated PRBC transfusion:

- Allo/auto-HPC transplant recipient
- Intrauterine transfusion

• Highly immunosuppressed patients at risk for complication [graft-versus-host disease (GVHD)]
• Neonates/infants undergoing exchange transfusion or extracorporeal membrane oxygenation (ECMO)
• Cellular immune deficiency.

2. Fresh frozen plasma (FFP) contains the components of coagulation, fibrinolytic, and complement systems, particularly factors V and VIII, which gradually decline during the storage of blood.It provides a balanced source of all coagulant factors and volume expansion. Weight adjusted dose of fresh frozen plasma (FFP) of 15–20 mL/kg is recommended. ABO and Rh-specific plasma should be used. If not available, FFP of different group maybe used provided it does not possess high-titer of anti-A/anti-B antibody.

3. Cryoprecipitate: Cryoprecipitate contains factor VIII: C factor VIII vWF, fibrinogen, factor XIII, and fibronectin. A unit of cryoprecipitate contains 2 g fibrinogen for each 100 ml; thus, a unit of cryoprecipitate will increase serum fibrinogen by 10 mg/dl. The usual dose of cryoprecipitate is 10 units, which is estimated to raise serum fibrinogen by 100 mg/dl. Subsequent doses must be adjusted conforming to serum fibrinogen levels aiming for levels >1.5 g/L.

4. Platelets

About 50 ml. Stored at room temperature. One unit of RDP increases the platelet count by 5000/ µl. One unit of SDP increases the platelet count by 300,00 - 50,000/µl

Transfusion threshold & target

Maintain the platelet concentration 50 x 103/dL in actively bleeding patient

Transfusion trigger <75 x 103/dL in ongoing bleeding

Target >50 x 103/dL for anesthesia and delivery

Platelet transfusion, even without bleeding, if platelet counts <20 x 103/dL

BLOOD TRANSFUSION IN MAJOR OBSTETRIC HEMORRHAGE

Major hemorrhage remains an important cause of maternal mortality in India, leading to 38% of maternal mortality.

The cornerstones of resuscitation during postpartum hemorrhage (PPH) are the restoration of both blood volume and oxygen-carrying capacity. Volume replacement must be undertaken on the basis that blood loss is often underestimated.

Compatible blood to replace red cell loss should be transfused as soon as available. The clinical picture should be the main determinant of the need for blood transfusion and time should not be unnecessarily spent awaiting laboratory results.

Obstetricians should draw on the expertise of their colleagues in anesthesia, hematology, and transfusion medicine in determining the most appropriate combination of intravenous clear fluids, blood and blood products for continuing resuscitation.

The main therapeutic goals of the management of massive blood loss is maintaining:

• Hb greater than 8 gm/dL
• Platelet count greater than 50,000/dL
• Prothrombin time (PT) less than 1 .5 times normal
• Activated partial thromboplastin time (AP TT) less than 1.5 times normal fibrinogen greater than 2 g/L.
• If no haemostatic results are available and bleeding is continuing, then, after 4 units of RBCs, FFP should be infused at a dose of 12–15 mL/kg . If no haemostatic tests are available, early FFP should be considered for conditions with a suspected coagulopathy, such as placental abruption or amniotic fluid embolism, or where the detection of PPH has been delayed.

Fibrinogen supplementation should be given if fibrinogen concentrations fall below 1.5 g/L.

In patients with critical hypofibrinogenemia (<1 g/L), supplementation in the form of cryoprecipitate or fibrinogen concentrate should be offered.

Administer 2 pools of cryoprecipitate if hemorrhage is ongoing and fibrinogen less than 2 g/L. Cryoprecipitate is the standard concentrated source of fibrinogen. Two 5-donor pools may increase fibrinogen in an adult by ~1 g/L,

Fibrinogen concentrate may also be considered as an alternative for the management of bleeding in patients.

As a pragmatic approach in cases of major bleeding, it is suggested that platelet transfusion should be given to maintain the platelet count at >50,000/dL, although higher thresholds may be indicated in actively bleeding patients with failing platelet counts.

Administer 1 pool of platelets if hemorrhage is ongoing and platelet count less than 75,000/dL. Patients presenting with major bleeding may be on antiplatelet medications. Platelet transfusions are considered a safe and potentially effective intervention in major hemorrhage in these patients.

In hemostatic resuscitation, PRBC, FFP, and PLT are applied in a 1:1:1ratio due to the resemblance with whole blood and because a "high ratio" is related to fewer complications and better patient survival outcomes. If PRBC is not available, then whole blood can be used instead in case of massive hemorrhage. The PROPPR study demonstrated that patients transfused with a

1:1:1 ratio achieved hemostasis and suffered fewer deaths due to exsanguination at 24 h.

Massive transfusion protocols:

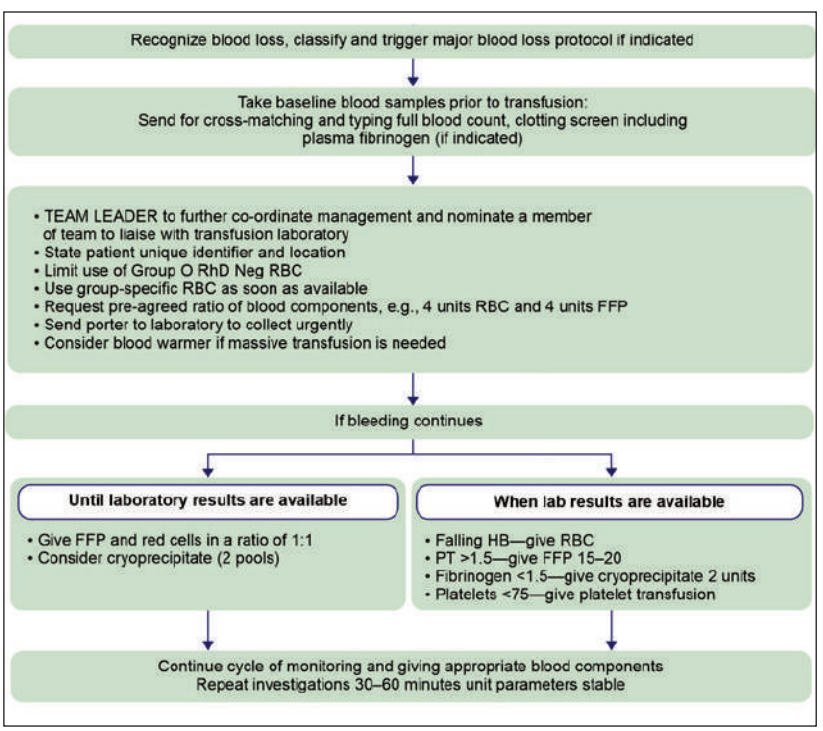
Massive transfusion means requirements of ≥4 PRBC units (some articles considered ≥10 PRBC within 24 h), replacement of total blood volume within 24 h, or replacement of 50% of blood volume within 3 h.The protocol for massive transfusion is specific at each institution.

ACOG recommends administration of blood products in 1:1:1 ratio, mimicking whole blood replacement.

TABLE 10 Massive transfusion protocol in obstetrics[a]

	PRBCs	FFP	Platelets	Cryoprecipitate
Round 1	6 U	6 U	6 U	10 U
Round 2	6 U	6 U	6 U	10 U
Round 3	Tranexamic acid 1 g intravenously over 10 min			
Round 4	6 U	6 U	6 U	

[a]Source: Pacheco et al. [7].



Once the massive transfusion protocol has been activated, the blood bank will send blood products in rounds to the operating or labor room. Each round has a specific number of PRBC, FFP, PLT and cryoprecipitate units according to the protocol established in the institution. Typical rounds consist of 6 units PRBC, 6 units FFP, 6 units PLT or 1 platelet apheresis, and 10 units of cryoprecipitate. Unless inactivated, the blood bank will prepare and send the products for rounds 2–4 successively, and if the patient continues bleeding the protocol will start again from round 1. It is very important to notify the blood bank as soon as the transfusion requirements decrease to stop the preparation of blood products.

Adverse Outcomes

Although early transfusions are lifesaving and in theory help to achieve hemostasis faster, thereby decreasing the number of blood products administered, the application of multiple units of blood products could be associated with a higher incidence of transfusion- related complications. These complications include hy perkalemia, hypocalcemia, citrate toxicity, transfusion- related im munomodulation, transfusion- related circulatory overload (TACO), transfusion- related kidney injury, transfusion- related acute lung injury (TRALI) (0.1 per 1000 units transfused), transfusion- related febrile nonhemolytic reactions (0.8 per 1000 units transfused), and acute hemolytic transfusion reaction (0.19 per 1000 units trans fused).22,24,25 Transfusion- related infectious diseases are uncommon (less than 1/100 000–1 000 000).

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ANTEPARTUM HEMORRHAGE

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APH is defined as bleeding from or into the genital tract after 20 weeks of gestation and prior to delivery of the baby.
Quantification
Minor Blood loss of <50 ml,includes spotting
Major Blood loss of 50-1000ml
Massive-Blood loss of >1000 ml

Causes of APH
• Placenta praevia
• Placental abruption
• Vasa praevia
• Unclassified

Placenta praevia
It is defined as a placenta that is implanted over or adjacent to the internal os.At the time of delivery the placenta precedes the baby.

Risk factors
• Previous LSCS
• Previous placenta praevia
• Multiparity
• Multifetal gestation
• Prior curettage/uterine surgery

Current classification
• True placenta praevia Internal cervical os is covered by placenta partially or completely
• Low lying placenta Placenta is in the lower segment ,the edge of the placenta is within 2 cm of the internal os.
Incidental diagnosis during antenatal USG
The majority of placenta (90%)diagnosed in early pregnancy resolve by term.This is called placental migration.Earlier the gestational age at diagnosis greater the migration.Placenta covering the os are less likely to migrate.Placenta on the anterior wall are less likely to migrate than posterior wall.

Clinical features of placenta praevia
• Painless vaginal bleeding
• May occur in second trimester, in third trimester or in labour
• Bleeding may be mild,moderate, profuse and recurrent
• May be associated with preterm uterine contractions,malpresentations

Complications
• Maternal
Hemorrhagic shock
Preterm labour
PPH
LSCS
Placenta accreta
• Fetal
Prematurity
Malpresentations
IUGR

Diagnosis
In most women diagnosed in ANC by USG.
Painless vaginal bleeding is the most important symptom.
Pallor,uterine height will correspond to period of gestation.Uterus will be relaxed. Malpresentations may be present
Transabdominal and transvaginal USG are diagnostic.Colour doppler should be performed to rule out adherent placenta.MRI is also useful but not routinely done.

Management
If diagnosed on routine USG,advise to avoid strenuous activity,report immediately if bleeding occurs.Repeat USG at 32 weeks for placental migration
If mild bleeding and preterm woman should be hospitalized.Woman should be offered rest,her hempglobin should be improved,Steroid can be given to improve lung maturity.If bleeding recurs and gestational age>34 weeks deliver.if no further bleeding delivery should be planned at 36-37 weeks.
LSCS is mode of delivery in almost all cases.
If initial bleeding is profuse,LSCS is indicated iresspective of gestational age.

Placental abruption
Premature separation of a normally implanted placenta after 20 weeks.

Classification
• Grade 0-Small RP clot seen after placental delivery
• Grade I-Vaginal bleeding mild,pain mild, no maternal/fetal complications
• Grade II-Vaginal bleeding moderate,pain moderate,uterus tense and tender,fetal distress
• Grade III-Vaginal bleeding severe or concealed,pain severe, marked uterine tender-

ness,fetal death,maternal shock/DIC/renal failure

Pathogenesis

In abruptio placenta bleeding occurs at the decidua basalis. This causes a retroplacental clot that expands and leads to total or near total placental separation.The blood seeps between membranes and uterine wall and escapes out of vagina as revealed abruption.Sometimes the blood is trapped behind placenta causing concealed abruptio.Most often revealed and concealed abruption coexist as mixed abruption.Retroplacental hemorrhage interferes with blood supply to fetus causing fetal hypoxia.Blood can seep into the uterine wall causing a Couvelaire uterus.

Risk factors

• Past history of abruption
• Vasospasm due to preeclampsia,thrombophilias
• Rapid uterine decompression like polyhydramnios,multifetal gestation,PPROM
• Trauma

Clinical features

• Vaginal bleeding which is mild or profuse
• Abdominal pain which is mild or severe
• Uterine tenderness
• Overdistended uterus
• Fetal heart rate abnormalities

Complications

Maternal
• Hypovolemic shock
• ARF
• DIC
• Preterm labour
• LSCS
• PPH

Fetal
• Fetal distress
• Fetal death
• Prematurity
• IUGR

Diagnosis

History
Clinical examination
PV should be done carefully if definite signs of abruption present.The presence of clots in vagina,dilatation and effacement of cervix,presentation,station,drainage of blood stained amniotic fluid should be noted.
USG This is more helpful to rule out placenta praevia.

Management

Stabilise the general condition.Send blood for cross-matching, coagulation profile
Transfuse bood and blood products as required
Perform USG if available
Immediate delivery is required in most women as bleeding is moderate to severe and maternal complicationa and/or fetal distress is usually present
LSCS is done if severe bleeding,fetal distress or maternal complications
Vaginal delivery can be allowed if bleeding mild,fetal heart normal or if fetus dead but mother stable.ARM should be done .Oxytocin augmentation can be done.If fetal distress or poor progress of labour,LSCS should be done
PPH should be aggressively managed.Couvelaire uterus is not an indication for obstetric hysterectomy.

Vasa praevia

Presence of umbilical vessels running through the membranes across the internal os below presenting part before reaching the placenta
Bleeding is from the fetus
If known antenatally planned LSCS
If diagnosed intrapartum emergency LSCS
Unclassified hemorrhage
Due to marginal sinus rupture,exaggerated show, trauma,infection or tumors of cervix. Diagnosed on speculum examination. If fullterm deliver.

CESAREAN HYSTERECTOMY IN OBSTETRIC CARE : A case review and lessons learnt

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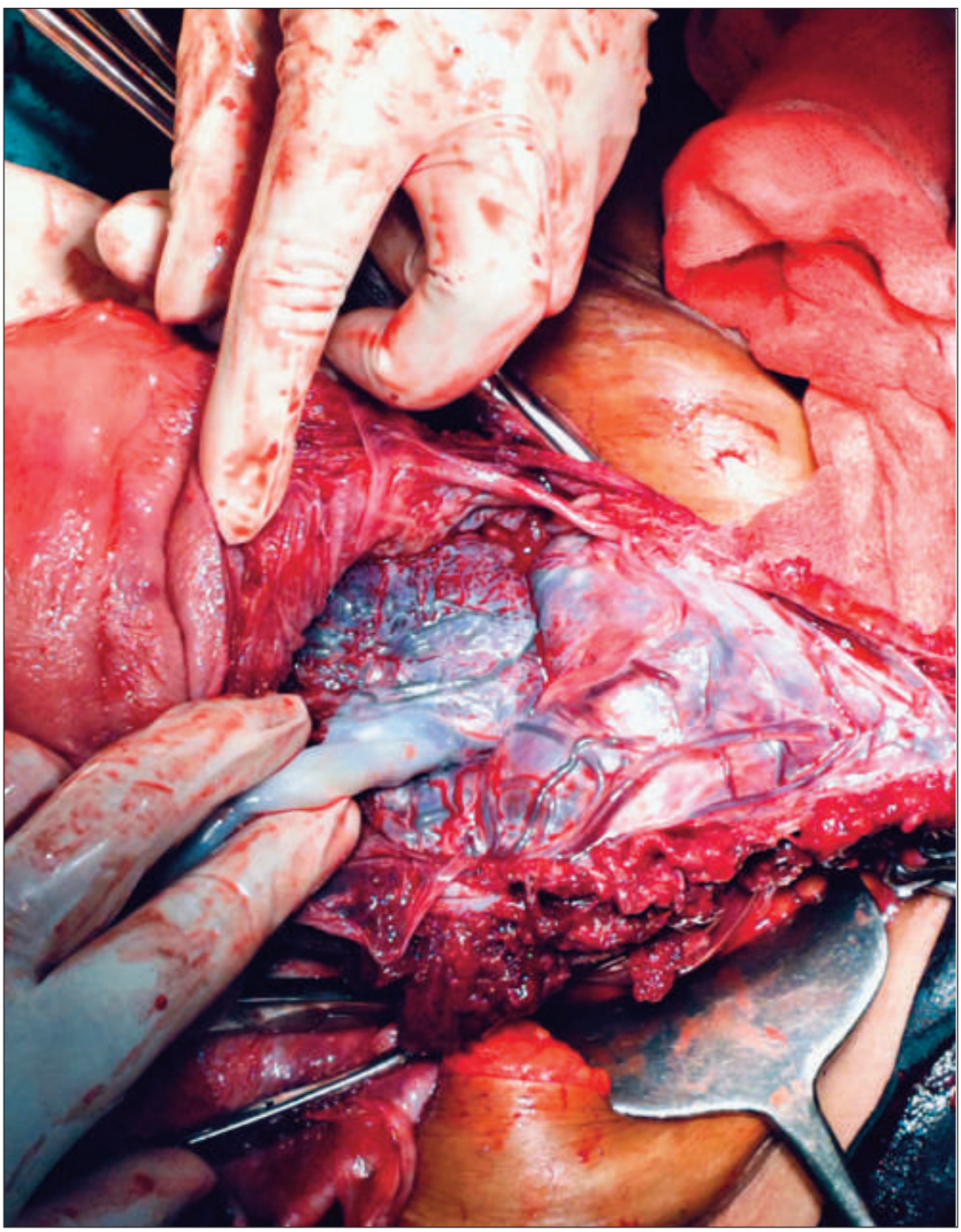

As an Obstetrics and Gynaecology resident in a tertiary care center like Sassoon hospital, Pune, obstetric emergencies were an everyday affair. However, patients with previous LSCS with or without history of multiple D and E's were always alarming even in between all the labour room chaos. Reason being – the ever-unnerving Placenta Accreta spectrum! Enough for entire unit to be on their toes for days together.

One such case that we encountered in March 2025 was of a 29-year-old G6P2L1D1A3 with previous 2 LSCS and 3 induced abortions with D and E with 29 weeks of gestation, with a known case of seizure disorder. Patient was a resident of Jejuri and was referred to Sassoon for further management. She was admitted and stabilized. Patient was not compliant with her anti-epileptic drugs and convulsed the first time that she came to us. With the help of the Neurology team, her anti-epileptic medications were stepped up and compliance was ensured. Her hemoglobin was 11 g/dl and rest labs were within normal limits. Ultrasonography done at Sassoon was suggestive of grade 4 placenta previa with multiple patchy areas of loss of myometrium interface noted with bulging of placental bed seen along anterior uterine wall. Few vessels were seen reaching upto mucosa of urinary bladder suggesting placenta increta/percreta. MRI was done which reported a grade 4 placenta previa with a total score of 7/8 in the Placenta Accreta Scoring System suggesting a high possibility of Placenta Increta/Accreta. After an episode of PV bleed, patient was posted electively for a Cesarean section with Obstetric hysterectomy by a team led by our unit head and professor Dr Shilpa Naik, general surgery team headed by Dr Sarfaraz Pathan, associate professor and unit head. Urosurgery team was intimated about this case and their intra operative assistance was sought. Interventional radiology team headed by Dr Ibrahim Ansari, professor and head of the department along with Dr Kiran Naiknavare was versed with this case for an intra-aortic balloon placement for reducing intra operative bleeding just prior to the surgery.

Our PPH trays were stocked up, blood and blood products were sanctioned not just at our institute but also in the nearby blood banks, residents and interns were allotted roles for the day of surgery, steroid cover was ensured, NICU team informed, ICU bed reserved for the patient and high risk consent explained.

On the day of scheduled OT the checklist was complete, we stuck to our assigned roles and the patient was swiftly wheeled in the OT after an intra-aortic balloon placement by the IR team. Along with the general surgeon a vertical incision was taken on the abdomen, engorged vessels were seen on the lower uterine segment. A 1.3 kg male child was delivered by vertex through a transverse incision on the upper segment. Anterior wall of uterus was adhered to the posterior wall of bladder. The case which was reported to be placenta accreta on MRI was actually placenta percreta intra operatively. Bilateral uterine arteries were ligated swiftly and obstetric hysterectomy was performed by clamp down technique. While separating the anterior uterine wall a rent of 5 cms was observed on the bladder which was repaired by the surgeon. The specimen was out within 40 minutes of incision. The intra-aortic balloon was intermittently inflated and deflated which wonderfully aided in controlling hemorrhage. Haemostasis was achieved. Abdomen was closed in layers. A total of 1.5 litres of blood loss was noted. 3 PCV, 2 units of cryoprecipitate, and 2 units of double strength FFP was transfused intra operatively. Patient was extubated successfully post operatively and was shifted to ICU for monitoring. She was shifted to ward on POD – 2 and had an uneventful post operative period.

Through this case it was yet again reiterated that a high-risk patient is never a one man's job. It requires a multidisciplinary approach as showcased here and thorough preparedness keeping always the worst outcome in mind. While on table complications are expected for such patients, we as tertiary center health care providers and experts can always over prepare ourselves to give the best possible outcome to the mother and her child – the only reason why thousands of poor patients traverse across kilometers hopeful to go home healthy and happy in lesser cost of treatment – a joyful reminder of why all of this is worth it.

Social initiative by POGS members

Dr Vaishali Biniwale
Chairperson, Public awareness committee, POGS

Nurses are the backbone of healthcare. They are responsible for providing direct patient care. Pune Obstetric & Gynaecological society is planning nurses training program under Surakshit Matrutva Abhiyan on 22/04/2025 at MG Auditorium, BJGMC, Pune.
The aim is to ensure high quality patient care during pregnancy & delivery. We will have deliberations on Antenatal Care, labour guide & postpartum period. There will be special demonstrations on Neonatal & Adult resuscitation. The program is arranged by Public awareness committee along with High Risk Pregnancy committee of POGS.

Upcoming Events


COMING SOON...
FERTILITY
CARNIVAL 2025
October 3rd, 4th & 5th | Goa
Intricacies, Advances & Research
in Reproductive Medicine
Coming soon for details...

POGS MASTERCLASS OF THE MILLENNIUM

ONE HALL
ONE FACULTY
ONE THEME

12 Materclasses Throughout The Year With POGS

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Masterclass	Date	Venue
Medicolegal Masterclass	18th May 2025	IMA Hall Pune
Infertility Masterclass with Dr Jatin Shah	3rd August 2025	Pune

Dr Manish Machave
President

Dr Nilesh Balkawade
General Secretary

Dr Kalyani Ingale
Clinical Secretary

POGS Calendar 2025 - 26

April 2025	Code RED POGS Installation CME April 18th - 20th, Boat club and Sheraton Grand Sassoon Nurses programme, POGS with AMOGS April 22nd, BJGMC Auditorium
May 2025	A-Z Masterclass - Medicolegal issues May 18th, IMA Nitu Mandke Hall
June 2025	Saand Ki Aankh PG Conference June 14th & 15th, Yashada Hall A-Z Masterclass - Internal Iliac Artery ligation & Cadaveric dissection June 28th, AFMC
July 2025	A-Z Masterclass on Colposcopy July 6th, SKN Medical College A-Z Masterclass Endoscopy with IAGE - EAGLE Project July 10th & 11th, MIMER Medical College
Aug 2025	A-Z Masterclass - ART August 3rd Nurture & Bloom - Breastcon Conference Aug 23rd & 24th, Mahabaleshwar
Sep 2025	A-Z Masterclass EndoART Sep 13th & 14th, Sheraton Grand A-Z Masterclass - USG in Infertility Sept 15th

POGS Calendar 2025 - 26

Oct 2025	Fertility Carnival Conference 3rd - 5th Oct, Goa
Nov 2025	FOGSI Presidential Conference - Theme Endoscopy, ART, Oncology, Surgical Skills 21st, 22nd & 23rd November
Dec 2025	A-Z Masterclass - Male Infertility Dec 4th POGS Annual Conference with A-Z Masterclass - Fetal Medicine Dec 13th & 14th, Symbiosis Vishwabhavan
Jan 2026	A-Z Masterclass - Fetal monitoring Jan 18th, BVMC
Feb 2026	Exchange Conference with Akluj Society in Association with Baramati, Pandharpur. 7th & 8th Feb Pune Labour Conference Feb 20th - 22nd, DMH
Mar 2026	POGS Cultural Programme March 8th A-Z Masterclass - Urogynaecology March 22nd

POGS CORE TEAM

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President, POGS

Dr Nilesh Balkawade
Secretary, POGS

Dr Uma Wankhede
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Vice President
Dr Vaishali Chavan

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Dr Vaishali Korde Nayak

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℞ In Subchorionic Hematoma

Eggtorr™

Alpha-lipoic acid 200mg +Leucine 800mg

Science-Backed Support for Subchorionic Hematoma

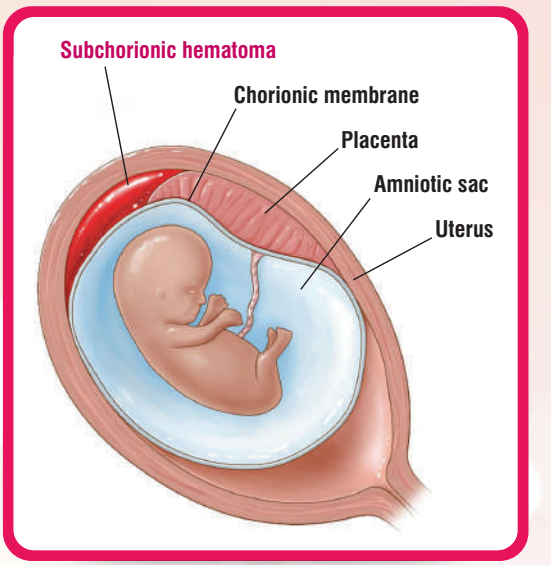
Subchorionic Hematoma (SCH) ?

SCH- Accumulation of blood between the chorion and the uterine wall during early pregnancy.

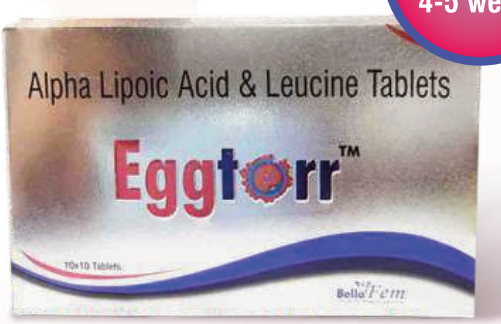
Occurrence: 10-20 weeks. gestation

How Common?

Incidence: ~30.5% of vaginal bleeds



1 Tablet BID for 4-5 weeks



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