

June 2015
Volume 4 : Issue 1

Obstetric NEWSLETTER M Medicine

SOCIETY OF
OBSTETRIC MEDICINE
INDIA

Editor

Hari Kishan Boorugu
Physician

Editorial Board

Ambuja C.
Obstetrician

Hemamalini V.
Physician

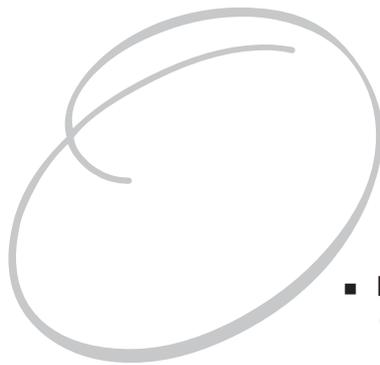
Nuzhat Aziz
Obstetrician

Praveen Nirmalan
Clinical Researcher

Shailesh Singi
Haematologist

Sunil T. Pandya
Obstetric Intensivist

CONTENTS



- From the Editor's Desk
 - Dengue Fever in Pregnancy
 - Swine Flu in Pregnancy
 - Malaria in Pregnancy
- Information for Registering in the Society of Obstetric Medicine, India (SOMI)
- Aims and Objectives of SOMI

Dear Members,

This issue deals with some of the common infections in our area. Pregnancy and Infections – there is paucity of literature on impact of most infections on mother and the fetus and effect of pregnancy on natural course of infections! Fever is a common manifestation in pregnancy and often treatment is empiric. There are a myriad of infections with overlapping clinical features and laboratory confirmation of many of them is not practical / cost effective or not available to most. Without identifying the specific pathogen, it is not possible to predict the effect of infection on pregnancy. Effect of infection on pregnancy is variable depending on the pathogen and host factors. For eg. We clearly know that malaria is associated with worse outcomes in pregnancy. Some infections like toxoplasmosis may have asymptomatic course in the gravida or may present as a non specific febrile illness which is difficult to suspect but it may have serious implications on neonatal health like choroiditis, retinitis and blindness. Out side pregnancy, diagnosis of mild toxoplasmosis may not have much importance but in pregnancy, it may have huge implications! Obstetricians need to be aware of various infections albeit rare, need to have high index of suspicion so as to diagnose, treat and prognosticate maternal and fetal outcomes as well.

This issue covers swine flu, dengue and malaria. Thanks to postgraduates and consultants of Durgabai Deshmukh Hospital, Hyderabad for contributing these articles and to Dr. Hemamalini V. and Dr. Usha of Fernandez hospital, Hyderabad for refining the articles. I request all SOMI members to contribute to future issues of our e-newsletter.

Yours infectiously,

Dr. Hari Kishan Boorugu
Editor

Introduction

Dengue infection is caused by dengue virus which is a single stranded RNA virus with an icosahedral nucleocapsid and covered by lipid envelope. The virus belongs to the family Flaviviridae. Dengue virus has four related but antigenically distinct serotypes : DENV1, DENV2, DENV3 and DENV4.

Each serotype is known to have several different genotypes. Viral genotype, serotype and sequence of infection with different serotypes determine disease severity. Symptomatic infections can present as mild febrile illness or as life threatening shock syndrome.

Classification

The WHO classification prior to 2009 classified the symptomatic infections into three categories : Undifferentiated fever, classic dengue fever and dengue hemorrhagic fever. The revised classification divides the disease into dengue and severe dengue. The non-severe dengue is divided into dengue with or without warning signs.

Course of Dengue Illness

Dengue is a systemic and dynamic illness. After the incubation period the disease starts suddenly and it is followed by three phases, febrile, critical and recovery. Following the febrile phase the patient enters the critical phase where he may suffer from plasma leak,

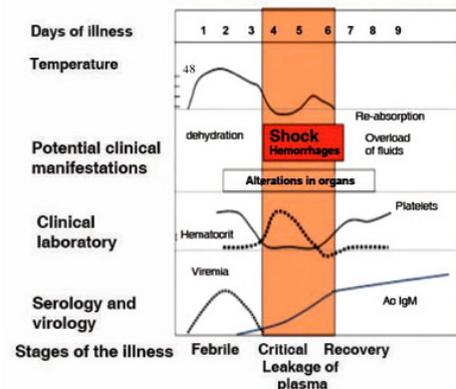
shock, hemorrhage or organ dysfunction. Unusual manifestations like encephalopathy and liver cell failure also may occur but death is usually due to profound shock especially if complicated by fluid overload.

DHF is defined by the following four WHO criteria :

- Fever or recent history of fever lasting 2 – 7 days
- Any hemorrhagic manifestation
- Thrombocytopenia (platelet count of < 1,00,000/ cumm)
- Evidence of increased vascular permeability

DSS is any case that meets the four criteria for DHF and has evidence of circulatory failure manifested by :

- Rapid weak pulse and narrow pulse pressure (< 20 mm of Hg)
- Hypotension
- Restlessness and cold, clammy skin.



DIAGNOSIS	METHOD	INTERPRETATION	SAMPLE CHARACTERISTIC
Confirmed Dengue Infection	Viral isolation	Viral isolation	Serum (collected at 1 - 5 days of fever)
	Genome detection	Positive RT-PCR	
	Antigen detection	Positive NS1Ag	Necropsy tissues
		Positive immunohistochemical	Necropsy tissues
	IgM seroconversion	from negative IgM to positive IgM in paired sera	Acute serum (1 - 5 days) and convalescent serum (after 15 - 21 days of first serum)
IgG seroconversion	From negative IgG to positive IgG in paired sera		
Probable Dengue Infection	Positive IgM	Positive IgM	Single serum collected after 5 days
	High IgG levels	High IgG levels by ELISA	

ELISA = enzyme-linked immunosorbent assay; IgG = immunoglobulin G; IgM = immunoglobulin M; NS1 Ag = non-structural protein 1 antigen; RT-PCR = reverse transcriptase polymerase chain reaction

Management of Dengue : General Principles

A stepwise management of the disease involves overall assessment, assessment of disease phase and severity and initiation of treatment. For a disease that is complex in manifestations, management is simple, effective and lifesaving so long as correct and timely interventions are instituted. The key is early recognition and understanding of the clinical problems during different phases of the disease leading to rational approach to case management.

What to look for when patient is being evaluated for dengue fever

Look for

Evidence of bleeding on skin or other sites

Evidence of capillary permeability (pleural effusion, ascitis, hemoconcentration)

Evaluate

Heart rate, Capillary refill, Skin color, Temperature, Peripheral pulse volume, Pulse pressure, Blood pressure. A drop in systolic pressure is usually the last sign and appears only when the patient is clinically critical.

Patients who are not critically ill and tolerate oral fluids should be encouraged to take fluids orally. Antipyretics are to be given when indicated. Aspirin and NSAIDs are to be avoided as they increase the risk of bleeding. Patient's hydration has to be monitored during the febrile phase of illness. Hemodynamic status has to be frequently assessed by checking patient's heart rate, capillary refill, pulse pressure, blood pressure and urine output. Intravenous fluids have to be started based on these parameters. Hematocrit and platelet counts need to be serially monitored. The patient has to be monitored in defervescence when the critical phase begins and lasts for 24 – 48 hours.

Dengue in Pregnancy

In the recent decade more cases of dengue are being reported in pregnancy. The clinical manifestations, treatment and outcome of dengue in pregnant women are similar to those of general population but with some important differences.^{1,2}

Impact of Dengue on Pregnancy:

- **Adverse pregnancy outcome**²⁻⁶

It is still uncertain whether dengue is a significant factor for adverse pregnancy outcomes such as preterm birth, low-birth weight and caesarean deliveries, as most of the published data were based on hospitalized patients.

- **Risk of vertical transmission**^{2, 6-10}

The risk of vertical transmission is well established among women with dengue during the perinatal period .

- **Significant impact of dengue at parturition**^{5, 11}

Severe bleeding may complicate delivery and/or surgical procedures performed on pregnant patients with dengue during the critical phase, i.e. the period coinciding with marked thrombocytopenia with or without coagulopathy and vasculopathy.

Management of Dengue during Pregnancy

- Early admission for close monitoring is recommended, especially for women close to full-term/labour.
- Conservative medical and obstetrical management is the treatment of choice.¹²

Challenges in Recognition of Dengue Disease and Plasma Leakage in Pregnancy

- Symptoms of hyperemesis during the first trimester of pregnancy resemble the warning signs of severe dengue and this may delay the recognition of severe dengue.

After the second trimester of pregnancy it is normal to see an increase in circulating blood volume with generalized vasodilatation, resulting in an increased baseline heart rate and lower baseline BP, as well as a lower baseline haematocrit. This can confuse the diagnosis of dengue and therefore clinicians need to be alert to the following:

- The lower BP and tachycardia of normal pregnancy could be misinterpreted as hypotensive shock.
- The lower baseline haematocrit after the second trimester of pregnancy should be noted. Establishing the baseline haematocrit during the first 2-3 days of fever is essential for early recognition of plasma leakage.
- Clinical signs of plasma leakage such as pleural effusion and ascites could be difficult to elicit in the presence of a gravid uterus.

Challenges in Monitoring and Management

- Close observation and monitoring, prompt, adequate and appropriate fluid replacement therapy during

the pre, intra and post-delivery periods are essential.

- Failure to recognize plasma leakage and/or shock early will lead to prolonged shock and eventually massive bleeding and multi-organ failure.
- There is no difference in fluid therapy compared with the non-pregnant state. However it is important to note that the growing gravid uterus may result in narrower tolerance of fluid accumulation in the peritoneal and pleural cavity from plasma leakage. Hence excessive fluid replacement should be avoided.
- The increased baseline heart rate and a lower baseline BP are normal physiological changes in late pregnancy. Targeting an inappropriate heart rate and "normal" levels of BP could result in fluid overload and respiratory distress.
- The presence of wounds or trauma during the critical phase of dengue with marked thrombocytopenia, coagulopathy and vasculopathy creates a substantial risk of severe haemorrhage.
- If severe haemorrhage occurs, replacement with transfusion of fresh whole blood/fresh packed red cells should be promptly instituted.
- Prophylactic platelet transfusion is not recommended unless obstetrically indicated.
- Delivery should take place in a hospital where blood/blood components and a team of skilled obstetricians and a neonatologist are available.
- Tocolytic agents and measures to postpone labour to a suitable time may be considered during the critical phase of dengue illness. However there is currently a lack of evidence on this practice.

Inevitable Delivery during Critical Phase

- If delivery is inevitable, bleeding should be anticipated and closely monitored.
- Blood and blood products should be cross-matched and saved in preparation for delivery.
- Trauma or injury should be kept to the minimum if possible.
- It is essential to check for complete removal of the placenta after delivery.
- Transfusion of platelet concentrates should be initiated during or at delivery but not too far ahead of delivery, as the platelet count is sustained by platelet transfusion for only a few hours during the critical phase.

- Fresh whole blood/fresh packed red cells transfusion should be administered as soon as possible if significant bleeding occurs. If blood loss can be quantified, it should be replaced immediately without waiting for the haematocrit to decrease to low levels.
- Ergotamine and or oxytocin infusion as per standard obstetrical practice should be commenced to contract the uterus after delivery to prevent postpartum haemorrhage.

Post-delivery

- Newborns of mothers who had dengue just before or at delivery, should be closely monitored in hospital after birth in view of the risk of vertical transmission.^{7,8}
- At or near-term/delivery, severe foetal or neonatal dengue illness and death may occur when there is insufficient time for the production of protective maternal antibodies.
- Clinicians should be aware that presentation in either maternal or neonatal disease may be atypical and confound diagnosis.
- Congenital infection could eventually be suspected on clinical grounds and then confirmed in the laboratory.

Conclusion

Dengue is the most rapidly spreading mosquito borne viral disease. Early diagnosis and appropriate management are essential for a good maternal and fetal outcome. Educating patients regarding preventive aspects of the disease is important.

References

1. Waduge R et al. Dengue infections during pregnancy: a case series from Sri Lanka and review of literature. *Journal of Clinical Virology*, 2006, 37(1):27-33.
2. Tan PC et al., Dengue infection in pregnancy: prevalence, vertical transmission, and pregnancy outcome. *Obstetrics & Gynecology*, 2008, 111(5):1111-1117.
3. Carles G et al., Dengue fever in pregnancy. A study of 38 cases in French Guiana. *European Journal of Obstetrics & Gynecology and Reproductive Biology* (Paris), 2000, 29(8):758-762.
4. Seneviratne SL, Perera J, Wijeyaratne C. Dengue

infections and pregnancy: caution in interpreting high rates of premature deliveries and maternal mortality. *Southeast Asian Journal of Tropical Medicine and Public Health*, 2007, 38(1):195-196.

5. Basurko C et al., Maternal and fetal consequences of dengue fever during pregnancy. *European Journal of Obstetric Gynecology and Reproductive Biology*, 2009, 147 (1):29-32.
6. Pouliot SH et al., Maternal dengue and pregnancy outcomes: a systematic review. *Obstetrical & Gynecological Survey*, 2010, 65(2):107-118.
7. Perret C et al., Dengue infection during pregnancy and transplacental antibody transfer in Thai mothers. *The Journal of Infection*, 2005, 51(4):287-293.
8. Sirinavin S et al., Vertical dengue infection: case reports and review. *The Paediatric Infectious Disease Journal*, 2004, 23(11):1042-1047.
9. Kerdpanich A et al., Perinatal dengue infection. *The Southeast Asian Journal of Tropical Medicine and Public Health*, 2001, 32(3):488-493.
10. Fernandez R et al., Study of the relationship dengue-pregnancy in a group of Cuban-mothers. *Revista Cubana de Medicina Tropical*, 1994, 46:76-78.
11. Thaithumyanon P et al., Dengue infection complicated by severe hemorrhage and vertical transmission in a parturient woman. *Clinical Infectious Diseases*, 1994, 18(2):248-249.
12. Carroll ID, Toovey S, Van Gompel A. Dengue fever and pregnancy – a review and comment. *Travel Medicine and Infectious Disease*, 2007, 5:183-188.

Swine Flu in Pregnancy

– Dr. Santosh D. Jagtap

Senior Resident (DNB),

– Dr. Manjula Rao (H.O.D.)

Durgabai Deshmukh Hospital, Andhra Mahila Sabha, Hyderabad.

Introduction

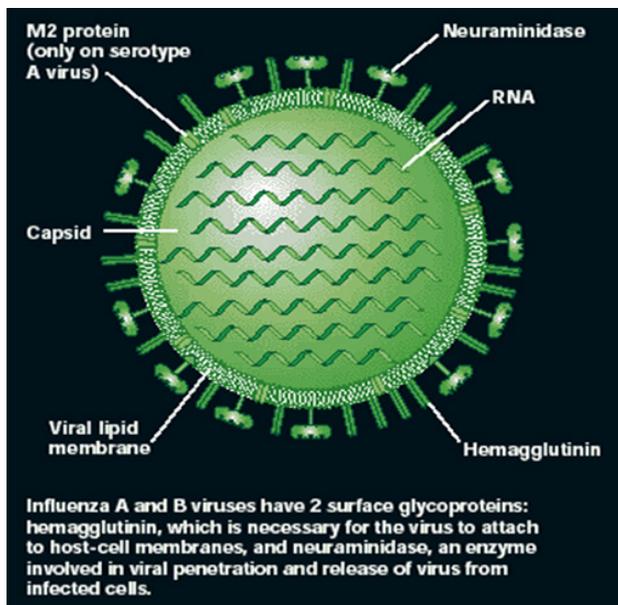
The H1N1 (H-Hemagglutinin, N-Neuraminidase) virus, which causes swine flu, first appeared in Mexico in April 2009 and then rapidly spread around the world. In India, the first case of influenza A H1N1 was reported on May 16, 2009 from Hyderabad. Globally influenza is responsible for 250,000 to 500,000 deaths annually.³

In India, The virus killed – 981 people in 2009, 1,763 in 2010, 75 in 2011, 405 in 2012 and 692 in 2013. (2014 Official statistics not available till date).

Epidemiology

The Agent

Genetic sequencing shows a new sub-type of influenza A (H1N1) virus with segments from four influenza viruses: North American Swine, North American Avian, Human Influenza and Eurasian Swine.



Family – Orthomyxoviridae

Genus and species - Influenza A (H1 to 18, N1 to 11).

Serotype – H1N1 (Negative sense, SS RNA, Segmented)

Genetic Plasticity

Influenza virus undergoes mutation that can take place within the genome (Antigenic drift) / or re-assortment among the genetic materials of subtypes (Antigenic Shift) resulting in a new virus.

Antigenic Drift is responsible for new seasonal strains that make necessary surveillance to detect these strains

and to prepare new seasonal influenza vaccine (yearly basis).

Antigenic Shift may result in a new virus easily transmissible from man to man for which the population has no immunity: Results in Pandemics.

Public Health Importance

Influenza virus causes Pandemics like

- Spanish Flu [A (H1N1)] 1918-19;
- Asian Flu [A (H2N2)] 1957-59;
- Hongkong Flu [A (H3N2)] 1968-68;
- "Swine Flu" [A (H1N1)] 2009-10

Causes Epidemics, seasonal Influenza outbreaks and sporadic cases too.

Host Factors

The majority of these cases have occurred in otherwise healthy young adults.

Maximum numbers of positive cases (35.1%) are from 20-39 year age group, comprising (22.5%) males and (14.8%) females.⁴

Environmental Factor

Predominance of pandemic 2009 influenza A H1N1 positive cases is seen during winter seasons and those of seasonal flu in the rainy season.⁴

Transmission

The transmission is by droplet infection and fomites.

Incubation Period : 1-7 days.

Communicability

From 1 day before to 7 days after the onset of symptoms. If illness persists for more than 7 days, chances of communicability may persist till resolution of illness. Children may spread the virus for a longer period.

Risk Factors for Severe Disease⁵

- Infants and young children, in particular <2 years.
- Pregnant women.
- Persons of any age with chronic pulmonary disease (e.g. asthma, COPD).
- Persons of any age with chronic cardiac disease (e.g. Congestive cardiac failure).
- Persons with metabolic disorders (e.g. diabetes).

- Persons with chronic renal disease, chronic hepatic disease, certain neurological conditions (including neuromuscular, neurocognitive, and seizure disorders), hemoglobinopathies, or immunosuppression, whether due to primary immunosuppressive conditions, such as HIV infection, or secondary conditions, such as immunosuppressive medication or malignancy.
- Children receiving chronic aspirin therapy.
- Persons aged 65 years and older.

A higher risk of severe complications from pandemic (H1N1) 2009 virus infection has also been reported in individuals who are obese (particularly in those who are morbidly obese) and among disadvantaged and indigenous populations.

Clinical Features

Majority of the patients have fever followed by exhibiting fever and cough. Some patients presented with fever, cough, nasal catarrh and a small proportion of patients present with fever and shortness of breath.⁴

Head ache, body ache, fatigue diarrhea and vomiting have also been observed.

Complications

There is insufficient information to date about clinical complications of the current pandemic influenza A (H1N1) virus infection.

Clinicians should expect complications to be similar to seasonal influenza: sinusitis, otitis media, croup, pneumonia, bronchiolitis, status Asthmaticus, myocarditis, pericarditis, myositis, rhabdomyolysis, encephalitis, seizures, toxic shock syndrome and secondary bacterial pneumonia with or without sepsis. Individuals at extremes of age and with preexisting medical conditions are at higher risk of complications and exacerbation of the underlying conditions

Swine Flu in Pregnancy

Antenatal check-ups during outbreak

Low risk pregnant women need to postpone antenatal visits during outbreaks. Separate care area for asymptomatic and ill pregnant women should be provided during epidemics. Apply triage criteria for separation. Display posters to self-separate upon arrival.

Effect of Pregnancy on Swine Flu

Pregnant women are vulnerable to complications of flu because of (more in 3rd trimester)

- Decrease in immune function
- Increase in body water
- Splinting of diaphragm, reduced lung volume
- Respiratory tract is more congested

Effect of Swine Flu on Pregnancy

- Viremia common
- Possibly reaches placenta / fetus (data insufficient)
- Fever (>38° C) in early pregnancy can cause
- Increased risk of fetal anomalies
- Spontaneous abortions
- Preterm labour
- Fetal distress in late pregnancy with adverse perinatal outcomes.
- Severe pneumonia
- Maternal death

Investigations / Diagnosis⁶

Confirmation of Pandemic influenza A (H1N1) infection is through:

- Real time RT PCR (Reverse Transcriptase Polymerase Chain Reaction), or
- Isolation of the virus in culture, or
- Four-fold rise in virus specific neutralizing antibodies.

For confirmation of diagnosis, clinical specimens such as

- Nasopharyngeal swab,
- Throat swab, nasal swab, wash or aspirate,
- Tracheal aspirate (for intubated patients) are to be obtained.

The sample should be collected by a trained physician / microbiologist preferably before administration of the anti-viral drug. Keep specimens at 4°C in viral transport media until transported for testing. The samples should be transported to designated laboratories within 24 hours. If they cannot be transported then it needs to be stored at -70°C. Paired blood samples at an interval of 14 days for serological testing should also be collected. Treatment.⁷

Case Definition of H1N1 – Acute Febrile Respiratory Illness

Suspected case:

1. Within 7 days of contact with positive case

2. Within 7 days of travel to area with positive case
3. Resides in a community where there are cases

Probable Case

1. Positive for Influenza A but not subtypable (H1 or H3)
2. Rapid Influenza test positive + suspected case
3. Died of acute respiratory illness in same community

Confirmed Case

1. WHO recognised RT-PCR positive
2. Viral culture positive
3. Four Fold increase H1N1 neutralizing antibodies

Guidelines on categorization of Seasonal Influenza-A H1N1 cases during screening for home isolation, testing, treatment and hospitalization (Revised on 11.02.2015). In order to prevent and contain outbreak of Influenza-A H1N1 virus for screening, testing and isolation following guidelines are to be followed:

Category-A

Patients with mild fever plus cough / sore throat with or without body ache, headache, diarrhoea and vomiting will be categorized as Category-A. They do not require Oseltamivir and should be treated for the symptoms mentioned above. The patients should be monitored for their progress and reassessed at 24 to 48 hours by the doctor. No testing of the patient for H1N1 is required. Patients should confine themselves at home and avoid mixing up with public and high risk members in the family.

Category-B

- (a) In addition to all the signs and symptoms mentioned under Category-A, if the patient has high grade fever and severe sore throat, may require home isolation and Oseltamivir; and
- (b) In addition to all the signs and symptoms mentioned under Category-A, individuals having one or more of the following high risk conditions shall be treated with Oseltamivir:
 - Children with mild illness but with predisposing risk factors.
 - Pregnant women;
 - Persons aged 65 years or older;
 - Patients with lung diseases, heart disease, liver disease kidney disease, blood disorders,

diabetes, neurological disorders, cancer and HIV/AIDS;

- Patients on long term cortisone therapy.

No tests for H1 N1 are required for Category-B (i) and (ii). All patients of Category-B (i) and (ii) should confine themselves at home and avoid mixing with public and high risk members in the family. Broad Spectrum antibiotics as per the Guideline for Community-acquired pneumonia (CAP) may be prescribed.

Category-C

In addition to the above signs and symptoms of Category-A and B, if the patient has one or more of the following:

- Breathlessness, chest pain, drowsiness, fall in blood pressure, sputum mixed with blood, bluish discoloration of nails.
- Children with influenza like illness who had a severe disease as manifested by the red flag signs (Somnolence, high and persistent fever, inability to feed well, convulsions, shortness of breath, difficulty in breathing, etc).
- Worsening of underlying chronic conditions.

All these patients mentioned above in Category-C require testing, immediate hospitalization and treatment.

Category A	Category B	Category C
Mild fever +cough / sore throat ± body-ache, headache, diarrhea and vomiting	High grade fever + severe sore throat and risk groups having signs of category A	Signs and symptoms of category A & B + breathlessness, chest pain, drowsiness, convulsions, fall in BP, blood in sputum, blue nails (or) worsening of underlying chronic conditions
✓ No testing for H1N1 required ✓ No oseltamivir (anti viral tablet) required	✓ No testing for H1N1 required	
✓ Stay at home ✓ Do not mix with public and high risk members	✓ Home isolation ✓ Oseltamivir (anti viral tablet) as per doctor's prescription	✓ H1N1 testing ✓ Immediate hospitalization treatment

Recommendations in pregnancy and breastfeeding

Early (<48 hrs) initiation of antiviral treatment for pregnant women without waiting for results of diagnostic testing.

Pregnant women presenting with uncomplicated illness due to influenza, and who have no evidence of systemic disease, can be offered either zanamivir (Relenza) or oseltamivir (Tamiflu).

Continue treatment even if negative laboratory test but clinical suspicion of influenza.

Pregnant women developing severe systemic or complicated disease due to influenza will typically be treated as an inpatient and should be offered treatment with oseltamivir.

Consideration should be given to extending the duration of antiviral treatment in critically ill pregnant women.

Oseltamivir Medication⁸

Oseltamivir is the recommended drug both for prophylaxis and treatment.

Dose for treatment is as follows:

By Weight:

<15 kg = 30 mg bd for 5 days

15-23 kg = 45 mg bd for 5 days

24-40 kg = 60 mg bd for 5 days

>40 kg = 75 mg bd for 5 days

For infants:

< 3 months = 12 mg bd for 5 days

3-5 months = 20 mg bd for 5 days

6-11 months = 25 mg bd for 5 days

It is also available as syrup (12 mg per ml.) If needed dose and duration can be modified as per clinical condition.

There are insufficient safety data for doses higher than 75 mg twice daily in pregnancy.

Zanamavir Medication⁸

10 mg (2 inhalations) inhaled twice daily for 5 days.

Pandemic influenza (H1N1) virus is currently susceptible to the neuraminidase inhibitors (NAIs) oseltamivir and

zanamivir, but resistant to the M2 inhibitors amantadine or rimantadine.⁹

Supportive therapy

- I.V. Fluids.
- Parenteral nutrition.
- Oxygen therapy / ventilatory support.
- Antibiotics for secondary infection.
- Vasopressors for shock.
- Paracetamol is prescribed for fever, myalgia and headache.
- Patient is advised to drink plenty of fluids.
- Smokers should avoid smoking.
- For sore throat, short course of topical decongestants, saline nasal drops, throat lozenges and steam inhalation may be beneficial.
- Salicylate / aspirin are strictly contra-indicated in any influenza patient due to its potential to cause Reye's syndrome.
- The suspected cases would be constantly monitored for clinical / radiological evidence of lower respiratory tract infection and for hypoxia (respiratory rate, oxygen saturation, level of consciousness).
- Patients with signs of tachypnea, dyspnea, respiratory distress and oxygen saturation less than 90% should be supplemented with oxygen therapy.

Delivery

Decision for Delivery

Most mothers with symptoms of influenza in labour will be able to tolerate labour with adequate pain relief and hydration. When a symptomatic woman is admitted with complications of influenza, early involvement of the obstetric anaesthetists and respiratory physicians is important to set out a clear management plan.

In most cases, the decision to deliver will be made for an obstetric indication. In the event of a critically ill woman close to term, there would be a clinical indication to deliver her, usually by caesarean section, to help with her mechanical ventilation. This should be done once her clinical condition is stabilized and other potential complications such as coagulopathy have been excluded or corrected.

Most of the respiratory complications have been shown to occur in the 2nd and 3rd trimesters. There may be

situations where a preterm baby needs to be delivered in order to improve the outcome for ventilation of a very ill mother. The decision is a clinical one, in conjunction with the obstetric, critical care and neonatal teams. In order to improve the outcome for the premature infant, 2 doses of 12 mg betamethasone should ideally be administered at least 24 hours prior to delivery.

Lactation and Newborn

Keep ill babies with mother.

If mother is ill with influenza,

- Cover with mask while breastfeeding.
- Frequent hand hygiene.

Initiate breastfeeding within one hour of birth.

Breastfeed frequently and exclusively.

Continue breastfeeding even on antiviral medication. If severe illness prevents feeding, expressed breast milk can be given.

Mothers who are breast feeding may continue breastfeeding while ill and receiving oseltamivir or zanamivir.

For pregnant women or mothers who are breast feeding, ensure that antimicrobials for treating any secondary infection are safe for use during pregnancy and lactation, e.g. avoid tetracycline, chloramphenicol, and quinolones.

Prevention and Control¹⁰

Personal Protection Equipment (PPE)

PPE reduces the risk of infection if used correctly. It includes:

Gloves, mask / three layered surgical mask, long-sleeved cuffed gown, protective eyewear (goggles / visors / face shields), cap (may be used in high risk situations where there may be increased aerosols), plastic apron if splashing of blood, body fluids, excretions and secretions is anticipated.

Chemoprophylaxis:

Oseltamivir is the drug of choice

Chemoprophylaxis for health care workers at high risk

Chemoprophylaxis for contacts-with high risk (with underlying systemic diseases, extremes of age < 5 and > 65 years, pregnant women).

Mass Chemoprophylaxis

If the virus is lethal and causing severe morbidity and mortality - to every individual in prescribed geographic limit of 5 kms from the epicenter.

Prophylaxis should be provided till 10 days after last exposure (maximum period of 6 weeks)

By Weight:

- <15 kg = 30 mg OD
- 15 to 23 kg = 45 mg OD
- 24 to 40 kg = 60 mg OD
- > 40 kg = 75 mg OD

For infants:

- < 3 months not recommended unless situation judged critical due to limited data on use in this age group
- 3–5 months = 20 mg OD
- 6–11 months = 25 mg OD

Swine Flu Vaccine

Only inactivated influenza vaccine can be given in pregnancy and children less than 2 years of age. Live attenuated vaccine should not be used in this population. Vaccination is recommended for all health care workers.

Indications for Swine Flu Vaccine

Chronic health conditions including cardiac or pulmonary disorders (Broncho-pulmonary dysplasia, COPD, Cystic fibrosis and asthma).

- Anaemia and Haemoglobinopathies
- Diabetes Mellitus
- Cancer
- Renal disease
- Immunodeficiency / immunosuppression

VAXIGRIP® is 0.5 mL of liquid vaccine in a single dose syringe.

Active Ingredients

Vaccine has been prepared on eggs and is made from inactivated parts of the following Influenza virus strains:

- A/California/7/2009 NYMC X-179A (A/California/7/2009 [H1N1]pdm09 - like),
- A/Texas/50/2012 NYMC X-223A (A/Texas/50/2012 [H3N2] – like),
- B/Massachusetts/2/2012 NYMC BX-51B (B/Massachusetts/2/2012-like)

Other Ingredients

Buffered saline solution composed of:

- Sodium chloride
- Potassium chloride
- Sodium phosphate – dibasic dihydrate
- Potassium phosphate – monobasic
- Water for injection

Vaccine may also contain traces of egg proteins, formaldehyde, octoxinol-9 and Neomycin.

Vaccine Dosage

Adults and children over 35 months: A single injection (0.5 mL)

Children 6 months to 35 months: A single injection (0.25 mL)

Vaccine Response

After receiving vaccine, the body will start making antibodies after 2 to 3 weeks. These antibodies help to recognise the virus and prevent the infection. Protection lasts for 6 to 12 months. The vaccine will only protect you against three types of influenza virus contained in the vaccine. It will not protect you from influenza caused by other types of viruses.

Ministry of Health and Family Welfare, Directorate General of Health Services, (Emergency Medical Relief)

Seasonal Influenza A (H1N1): Guidelines for Vaccination of Health Care Workers (Updated on 14th February 2015)

1. World Health Organization recommends vaccination of high risk groups with Seasonal Influenza Vaccination.
2. In India, neither the actual disease burden of Influenza, nor differentials on the way influenza impacts high risk groups are known. Hence, evidence based decision is not possible for all high risk groups.
3. Health Care Workers working in close proximity to influenza patients are at higher risk of acquiring the disease. Hence, vaccination is recommended for them. Such category would include:
 - Health Care Workers working in casualty/emergency department of identified hospitals treating Influenza cases.
 - Health Care Workers working in ICU and Isolation Wards managing influenza patients.
 - Health Care Workers identified to work in screening centres that would be set up for categorization of patients during Seasonal Influenza outbreak.
 - Health Care workers treating/managing the High Risk Group.
 - Laboratory personnel working in virological laboratories testing Influenza samples.

- Rapid Response Team members identified to investigate outbreaks of Influenza.
 - Drivers and staff of vehicles/ambulances involved in transfer of Influenza patients.
4. The vaccine should be used every year.
 5. Influenza vaccination is most effective when circulating viruses are well-matched with vaccine viruses. Even with appropriate matching, efficacy of vaccine may be about 70% to 80%, especially in geriatric age group. In case the locally circulating virus is different from vaccine virus recommended by WHO, it may not be effective at all. Hence, vaccine should not give a false sense of security. Considering the risk perspective, the preventive modality of infection prevention and control practices like use of PPEs should be strictly adhered to. The available vaccine takes about 2-3 weeks for development of immunity. The use of chemoprophylaxis during this period may be considered.

Conclusion

- H1N1 is a novel virus with high communicability spreading worldwide with variable mortality rates.
- It causes pandemics, epidemics and sporadic cases too.
- Antigenic drift and shift phenomenon makes the virus more communicable and less susceptible to established treatment, due to formation of new strains to which humans may not have immunity and vaccines for previous strains may not work.
- Pregnancy and infancy are itself high risks for contracting swine flu and hence included in high risk category for management. Initiating treatment within 48 hours of symptoms has good clinical outcome.
- Vaccine and treatment with oseltamivir in pregnancy and lactation is safe. Vaccine to be taken every year.
- Data in Indian scenario is still insufficient / unpublished. Hence we need more numbers of large multicentric, double blind, randomized controlled trials to form guidelines.

Summary of Clinical Management of Patients with Pandemic (H1N1) 2009 Virus Infection

Modalities	Strategies
Diagnosis	RT-PCR provides the most timely and sensitive detection of the infection. Performance of rapid influenza diagnostic tests (RIDT) is variable; a negative result cannot exclude infection with influenza. Consequently, clinical diagnosis in the context of local influenza activity should inform treatment initiation.
Antibiotics	In case of pneumonia, empiric treatment for community acquired pneumonia (CAP) as per published guidelines pending microbiologic results (e.g. 2-3 days); tailored therapy thereafter, if any pathogen(s) are present.
Antiviral therapy	If treatment is indicated, early initiation of treatment with oseltamivir or zanamivir, is recommended. Extended oseltamivir treatment (at least 10 days) and higher doses (up to 150 mg twice daily in adults) should be considered in severe cases. Sporadic oseltamivir resistance observed; treat unresponsive cases with suspicion.
Corticosteroids	Moderate to high dose systemic corticosteroids are NOT recommended as adjunctive H1N1 treatment. They are of unproven benefit and potentially harmful.
Infection control	Standard plus Droplet Precautions. For aerosol-generating procedures, use a particulate respirator (N95, FFP2 or equivalent), eye protection, gowns, gloves, and an adequately ventilated room, which can be naturally or mechanically ventilated, as per WHO guidance
NSAIDs, antipyretics	Paracetamol (acetaminophen) given orally or by suppository. Avoid administration of salicylates (aspirin and aspirin-containing products) in children and young adults (< 18 years old) due to the risk of Reye's syndrome.
Oxygen therapy	Monitor oxygen saturation and maintain SaO ₂ over 90% (92-95% for pregnant women) with nasal cannulae or face mask. High flow oxygen may be required in severe cases.
Pregnancy	Initiate oseltamivir treatment early. Do NOT treat with ribivirin. Safety data for elevated antiviral doses is not available. Ensure antimicrobials for secondary infections are safe for this patient group. NSAIDs should be avoided. Maintain SaO ₂ at 92-95%. Mothers can continue breastfeeding when ill and on antivirals.
Infants	Symptoms may be non-specific, so clinicians should act with a high index of suspicion. No aspirin should be given to children. Antiviral treatment should be initiated early.

References

1. Update: Novel influenza A (H1N1) virus infection. Mexico. March-May, 2009. CDC Morbidity and Mortality Weekly Report June 5, 2009/58; 585-9.
2. Ministry of Health and Family Welfare, India. Information on Swine Flu. <http://www.mohfw.nic.in/swineflu.htm>, accessed on November 18, 2011.
3. World Health Organization, April 2009. <http://www.who.int/mediacentre/factsheets/fs211/en/>, accessed on April 16, 2012
4. Indian J Med Res 135, April 2012, pp 534-537
5. WHO pandemic (H1N1) 2009 case summary form for clinical data collection: http://www.who.int/csr/disease/swineflu/guidance/surveillance/WHO_case_definition_swine_flu_2009_04_29.pdf (Annex 3)
6. <http://www.who.int/csr/disease/swineflu/guidance/laboratory/en/index.html>
7. http://www.who.int/csr/resources/publications/swineflu/h1n1_use_antivirals_20090820/en/index.html
8. http://www.emea.europa.eu/humandocs/PDFs/EPAR/tamiflu/Tamiflu_PI_clean_en.pdf (accessed 25 October 2009)
9. <http://www.cdc.gov/mmwr/PDF/wk/mm5817.pdf>
10. http://www.who.int/csr/resources/publications/infection_control/en/index.html
11. Kaji M, Watanabe A, Aizawa H. Differences in clinical features between Influenza A H1N1, A H3N2, and B in adult patients. *Respirology* 2003; 8 : 231-3.
12. Broor S, Gupta S, Mohapatra S, Kaushik S, Mir MA, Jain P, et al. Emergence of 2009A/H1N1 cases in a tertiary care hospital in New Delhi, India. *Influenza Other Respi Viruses* 2011; 5 : e552-7.

Malaria in Pregnancy

– Dr. S. R. Vamshi Krishna

– Dr. Abhishek Mufkalwar

– Dr. G. Sravan Kumar

Internal Medicine Postgraduates, Durgabai Deshmukh Hospital,
Andhra Mahila Sabha, Hyderabad.

Introduction

Malaria, a parasitic infection transmitted by mosquitoes, is one of the most devastating infectious diseases killing more than one million people annually. WHO describes malaria as a disease of poverty caused by poverty. Each year, 50 million women living in malaria endemic area become pregnant.¹ It is estimated that 10,000 women and 200,000 infants die as a result of malarial infection during pregnancy.

Etiology

Human malaria is caused by five species of Plasmodia

P. falciparum, *P. vivax*, *P. ovale*, *P. malariae* and *P. knowlesi*. Most infections are due to either *P. falciparum* or *P. vivax*, but mixed infections with more than one malarial species also occur. The majority of malaria-related deaths are due to *P. falciparum*.

Prevalence, Microbiology and Epidemiology

It is estimated that at least 25% of pregnant women living in areas endemic for malaria are infected with malaria, with the highest risk for infection and morbidity in primigravidae, adolescents, and those co-infected with HIV.²

P. falciparum is the predominant species, giving rise to heightened morbidity and mortality in pregnancy.

P. vivax infection can give rise to some of the same complications as *P. falciparum*; however, the complications are less frequent and less severe.

P. knowlesi is widely distributed, but relatively rare in pregnancy.³

Pregnant vs Non-Pregnant

The prevalence of peripheral parasitaemia is higher in pregnant than in age-matched nonpregnant women living in the same geographic area both in holo-endemic and mesoendemic areas.⁴⁻⁸ In the southeast Asia, malaria is a serious burden in pregnancy with a spectrum of ill effects as shown by slide positivity rate (1.1–58%, *n* = 45–365), parasitaemia (1–70%, *n* = 55–365), cerebral malaria (7–76%, *n* = 45–365), anaemia (8.6–90%, *n* = 45–365), maternal mortality (7–66.6%, *n* = 45–365), placental malaria (18–29%, *n* = 256–365), abortions (2–11%, *n* = 45–365) and intrauterine fetal development impairment (2–31%, *n* = 45–322), stillbirth (2–13%, *n* = 45–365), pre-term (4.2–60%, *n* = 45–322) and low birth weight (5.4–89%, *n* = 55–365) (Singh et al 2005).

In pregnant women, the increase in parasitaemia may be driven in part, by an increased susceptibility to mosquito bites. Exhaled carbon dioxide and body heat are both increased during pregnancy and may attract mosquitoes.⁹

It is hypothesized that the majority of sequelae in pregnancy result from two main factors: the immunocompromised state of pregnancy and placental sequestration of infected erythrocytes.

Pathogenesis

Pregnancy-associated *P. falciparum* malaria is characterized by sequestration and multiplication of a distinct population of malarial parasites in the placenta. These parasites express a specific class of variant surface antigens (VSAs) that mediate adhesion of parasite-infected erythrocytes to chondroitin sulfate A (CSA) on the syncytiotrophoblast lining the intervillous space.¹⁰⁻¹¹ This process appears to involve up-regulated transcription of *var2csa*¹² which is expressed on the surface of CSA-adherent infected erythrocytes. This suggests that the *var2csa* gene encodes a parasite adhesion molecule that initiates the pathology associated with Pregnancy Associated Malaria (PAM).

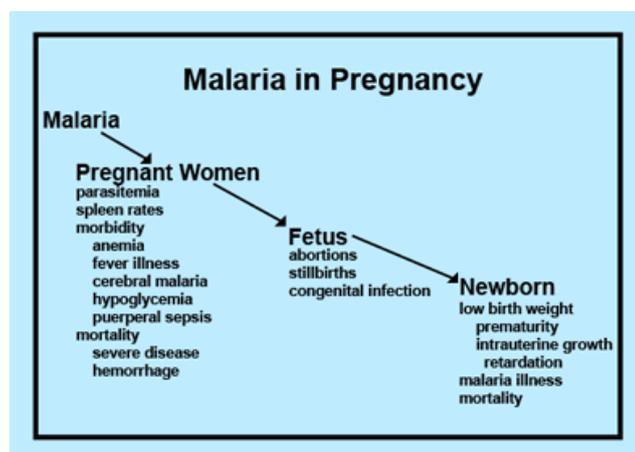
The unique VSA types expressed on pregnancy-associated malaria (PAM) parasites can be serologically classified as sex specific (i.e. men from malaria-endemic areas do not develop VSA antibodies) and gravidity dependent (i.e. women acquire anti-VSA immunoglobulin G as a function of gravidity). Thus, the observed protection afforded by multigravidae may result from maternal antibodies (e.g. anti-VSA) that prevent cyto-adhesion of the parasite to the placenta.¹³⁻¹⁴

Pregnancy-induced immuno-suppression may also account for the more severe disease experienced by primigravid nonimmune women (primigravidae express higher cortisol levels compared with multigravidae, which leads to greater depression of cell-mediated immunity).¹⁵

Clinical Manifestations

The clinical presentation varies according to the underlying endemicity of the region. In areas of stable malarial transmission (holo-endemic regions) where partial immunity is common, most malarial infections in pregnant women are asymptomatic; but the mother remains at risk for anemia and the fetus is at risk for

low birth weight.¹⁶⁻¹⁷ However, for women residing in mesoendemic areas, or for women returning to a holoendemic area after a prolonged absence, malaria is more likely to result in febrile illness, severe symptomatic disease, preterm birth, and death of mother or fetus.¹⁸



Diagnosis of Malaria

The diagnosis of malaria should be considered in any febrile woman who has resided in a malarious region, or traveled to a malarious region even if briefly or only in transit. Standard methods that detect peripheral parasitaemia can be used in pregnant women, and include Giemsa-stained thick and/or thin peripheral blood smears or rapid diagnostics. It is important to point out that women may have placental parasites that are not circulating in the peripheral blood, and hence, the blood film would be negative. No reliable peripheral biomarker for the presence of placental malaria has been identified; the diagnosis is made by histological examination of placenta after delivery.

Thick Smear

More sensitive in detecting malarial parasites because the blood is more concentrated allowing for greater volume of blood to be examined.

Thin Smear

Aids in parasite species identification and quantification. More recently, a number of rapid diagnostic tests have been developed that detect parasite proteins in peripheral blood. Some of these tests detect malaria histidine rich protein II (HRPII), such as the ParaSight F-test (Becton Dickinson), the ICT Malaria P.f.test/MalaQuick (ICT Diagnostic, Sydney, Australia), the PATH P. falciparum malaria IC strip (Program for Appropriate Technology in Health, Seattle, Washington), and the Determine Malaria

P.f. test (Abbot Laboratories, Japan).¹⁹⁻²¹ Early versions of these assays detected only HRPII of *P. falciparum*; newer-generation assays, however, detect antigens of both *P. falciparum* and *P. vivax* (such as the ICT Malaria P.f. /P.v. test).²² In addition, assays that detect all four malaria species are under development.²³

Another group of assays have been developed that detect plasmodial lactate dehydrogenase (pLDH) via immunochromatographic detection (OptiMAL kit; Flow, Inc., Portland, Oregon), or via enzymatic reaction (ICpLDH; Flow, Inc.).²⁴ The pLDH assays are able to detect antigens of both *P. falciparum* and *P. vivax*.²⁵

Prevention

Chemoprophylaxis

1. Travelers – are advised to defer travel to areas where risk of acquiring malaria is high, until delivery, if feasible. Non-immune pregnant women who cannot defer travel should take chemoprophylaxis.

Agents of Choice : Chloroquine (chloroquine sensitive areas) Mefloquine (Chloroquine resistant areas)

2. Women living in endemic areas – intermittent preventive treatment during pregnancy (IpTp)
Sulfadoxine – pyrimethamine (SP) at each scheduled visit in second and third trimester.
3. Women infected with HIV – if on co-trimoxazole to prevent HIV related opportunistic infections, also provides protection against malaria, hence no need of SP-IpTp during pregnancy.

Mosquito Avoidance

- Covering exposed skin with Clothing and applying insect repellent
- Use of insecticide treated bed nets (INTs)

Treatment of Malaria

(National Vector Borne Disease Control Programme Guidelines)

Plasmodium Vivax – Chloroquine 25 mg/kg Body weight divided over 3 days i.e., 10 mg/kg on day 1, 10 mg/kg on day 2 and 5 mg/kg on day 3.

Primaquine is contraindicated in pregnancy and lactation. Hence radical treatment of vivax malaria to prevent relapse is not possible in pregnancy and lactation.

Plasmodium falciparum

Uncomplicated

First trimester – Quinine salt 10 mg/kg three times daily for 7 days.

Quinine may induce hypoglycemia; hence, pregnant women should not start taking quinine on empty stomach and should eat regularly while on quinine treatment.

Second and Third Trimesters – Area specific ACT (Artemisinin-derivatives based Combined Therapy).

- ACT-AL in northeastern states
- ACT-SP in other states.
- ACT-AL Artemether (20mg) - Lumefantrine (120mg) 80 mg twice daily for three days i.e., four tablets twice daily for three days.
- ACT-SP – Artesunate 4 mg/kg body weight daily for 3 days + sulfadoxin 25 mg/kg body weight – pyremethamine 1.25 mg/kg body weight on first day.

Chemotherapy of Severe and Complicated Malaria

Initial parenteral treatment for at least 48 hours followed by oral medication when patient can take orally.

CHOOSE ONE of Following Two Options

1. Quinine: 20mg quinine salt/kg body weight on admission (IV infusion or divided IM injection) followed by maintenance dose of 10 mg/kg 8 hourly; infusion rate should not exceed 5 mg/kg per hour. Loading dose of 20mg/kg should not be given, if the patient has already received quinine.

Quinine 10 mg/kg three times a day with Clindamycin- to complete 7 days of treatment. (Doxycycline contraindicated in pregnancy and lactation.)

Or

2. Full Oral Course of Area-Specific ACT :
In Northeastern states : Age specific ACT-AL for 3 days

In other states : Treat with ACT-SP for 3 days

Artemisinin derivatives used in the combination can be:

Artesunate : 2.4 mg/kg i.v. or i.m. given on admission

(time=0), then at 12 h and 24 h, then once a day.

Or

Artemether : 3.2 mg/kg i.m. given on admission then 1.6 mg/kg per day.

Or

Artemether : 150 mg daily i.m for 3 days.

Treatment of mixed infections (P.vivax + P.falciparum cases) : All mixed infections should be treated with full course of ACT.

In North-Eastern States : Treat with Age-specific ACT-AL for 3 days.

In Other States : ACT-SP for 3 days.

References :

1. <http://mosquito.who.int/>. (Accessed on May 08, 2008)
2. Desai M, ter Kuile FO, Nosten F, et al. Epidemiology and burden of malaria in pregnancy. *Lancet*
3. Barber BE, Bird E, Wilkes CS, et al. Plasmodium knowlesi Malaria During Pregnancy. *J Infect Dis* 2015; 211:1104.
4. Singh N, Saxena A, Chand SK, et al. Studies on malaria during pregnancy in a tribal area of central India (Madhya Pradesh). *Southeast Asian J Trop Med Public Health* 1998; 29:10.
5. Brabin BJ. An analysis of malaria in pregnancy in Africa. *Bull World Health Organ* 1983; 61:1005.
6. Diagne N, Rogier C, Cisse B, Trape JF. Incidence of clinical malaria in pregnant women exposed to intense perennial transmission. *Trans R Soc Trop Med Hyg* 1997; 91:166.
7. Bergström S, Fernandes A, Schwalbach J, et al. Materno-fetal transmission of pregnancy malaria: an immunoparasitological study on 202 parturients in Maputo. *Gynecol Obstet Invest* 1993; 35:103.
8. Sholapurkar SL, Gupta AN, Mahajan RC. Clinical course of malaria in pregnancy—a prospective controlled study from India. *Trans R Soc Trop Med Hyg* 1988; 82:376.
9. Lindsay S, Ansell J, Selman C, et al. Effect of pregnancy on exposure to malaria mosquitoes. *Lancet* 2000; 355:1972.
10. Salanti A, Staalsoe T, Lavstsen T, et al. Selective upregulation of a single distinctly structured

- var gene in chondroitin sulphate A-adhering Plasmodium falciparum involved in pregnancy-associated malaria. *Mol Microbiol* 2003; 49:179.
11. Fried M, Duffy PE. Adherence of Plasmodium falciparum to chondroitin sulfate A in the human placenta. *Science* 1996; 272:1502.
 12. Salanti A, Dahlbäck M, Turner L, et al. Evidence for the involvement of VAR2CSA in pregnancy-associated malaria. *J Exp Med* 2004; 200:1197.
 13. Beeson JG, Rogerson SJ, Elliott SR, Duffy MF. Targets of protective antibodies to malaria during pregnancy. *J Infect Dis* 2005; 192:1647.
 14. Staalsoe T, Shulman CE, Bulmer JN, et al. Variant surface antigen-specific IgG and protection against clinical consequences of pregnancy-associated Plasmodium falciparum malaria. *Lancet* 2004; 363:283.
 15. Vleugels MP, Eling WM, Rolland R, de Graaf R. Cortisol and loss of malaria immunity in human pregnancy. *Br J Obstet Gynaecol* 1987; 94:758.
 16. McGregor IA, Wilson ME, Billewicz WZ. Malaria infection of the placenta in The Gambia, West Africa; its incidence and relationship to stillbirth, birthweight and placental weight. *Trans R Soc Trop Med Hyg* 1983; 77:232.
 17. Diagne N, Rogier C, Sokhna CS, et al. Increased susceptibility to malaria during the early postpartum period. *N Engl J Med* 2000; 343:598.
 18. Desai M, ter Kuile FO, Nosten F, et al. Epidemiology and burden of malaria in pregnancy. *Lancet Infect Dis* 2007; 7:93.
 19. Bustos DG, Olveda RM, Negishi M, Kurimura T. Evaluation of a new rapid diagnostic test "Determine Malaria PF" against standard blood film, ICT Malaria P.F and ParaSight F. *Jpn J Trop Med Hyg* 1999;27:417-425.
 20. Funk M, Schlagenhauf P, Tschopp A, Steffen R. MalaQuick versus ParaSight F as a diagnostic aid in travellers' malaria. *Trans R Soc Trop Med Hyg* 1999;93:268-272.
 21. Gaye O, Diouf M, Diallo S. A comparison of thick smears, QBC malaria, PCR and PATH falciparum malaria test trip in Plasmodium falciparum diagnosis. *Parasite* 1999;6:273-275.
 22. Tjitra E, Suprianto S, Dyer M, et al. Field evaluation of the ICT malaria Pf/P.v immunochromatographic test for detection of Plasmodium falciparum and Plasmodium vivax in patients with a presumptive clinical diagnosis of malaria in eastern Indonesia. *J Clin Microbiol* 1999; 37:2412-2417.
 23. Hanscheid T. Diagnosis of malaria: a review of alternatives to conventional microscopy. *Clin Lab Haematol* 1999;21:235-245.
 24. Piper R, Lebras J, Wentworth L, et al. Immunocapture diagnostic assays for malaria using Plasmodium lactate dehydrogenase (pLDH). *Am J Trop Med Hyg* 1999;60:109-118.
 25. Palmer CJ, Lindo JF, Klaskala WI, et al. Evaluation of the OptiMAL test for rapid diagnosis of Plasmodium vivax and Plasmodium falciparum malaria. *J Clin Microbiol* 1998;36:203-206

SOCIETY OF OBSTETRIC MEDICINE, INDIA (SOMI)

Membership Form

Surname : _____ First Name : _____

Qualification : _____ Date of Birth : _____

Designation : _____

Work Address : _____

Hospital _____

PHOTO

Type of Hospital: Women's / General / District / Nursing Home Total No. of Beds : _____

Year of Passing : MBBS _____ Diploma _____ MD _____ DNB _____ Fellowship / Other : _____

Medical Council Registration No. : MBBS : _____ PG : _____

Residence Address : _____

Email ID : _____

Tel No. : (Res) _____ (Off) : _____ (Mobile) : _____

Annual Membership Fees : Rs. 500/- (Rupees Five Hundred only)

Life Membership Fees : Rs. 5,000/- (Rupees Five Thousand only)

DD to be drawn in favour of "**Society of Obstetric Medicine, India**" payable at Hyderabad. (Send DD with two passport size photos to the address below).

Signature _____

For Office use only

Registration No. _____ Receipt No. _____

Received: Cash / DD / Cheque _____ Remarks: _____



Obstetric Medicine

NEWSLETTER

SOCIETY OF OBSTETRIC MEDICINE INDIA

Aims and Objectives

- To establish a forum for exchange and interchange of views and for enhancing fellowship among its members and to come out with evidence-based guidelines for management of medical disorders complicating pregnancy.
- To assist in the establishment of Obstetric Medicine as an important sub-specialty to advance clinical and scientific knowledge of medical illness complicating pregnancy.
- To promote research and training in Obstetric Medicine.
- To foster collaboration with other regional and international societies interested in Obstetric Medicine.
- To carry out all such activities as would contribute to the promotion of Obstetric Medicine.
- To promote formation of patient forums.

Inviting Articles for Future Issues

Please send in your articles (interesting case reports, review articles, original research articles, comments on previous articles) in Word document format to obsmedindia@gmail.com with a copy to the editor, drharikishan@gmail.com