2019-2020

NEWSLETTER

PAKISTAN SOCIETY OF ANAESTHESIOLOGISTS
KARACHI - CHAPTER

Volume: 20
Issue 01, July 2019

Inside this Issue:

EDITOR'S NOTE

I feel great pleasure for revival of PSA newsletter after a long period. It is a difficult uphill task but with the grace of Almighty Allah and help of our all colleagues we be able to publish first issue. The purpose of PSA newsletter is primarily, to disseminate the knowledge to peripheral/non-teaching hospitals/anaesthesiologists, to make them abreast with latest ongoing research/ guidelines in the field of anaesthesiology, critical care and pain management; secondarily; to make the fraternity aware of PSA activities.

It was decided in executive committee meeting to publish PSA newsletter thrice a year or more. Each issue will be dedicated to a special topic. So the first issue is regarding Obstetric anaesthesia. As you know the obstetric morbidity and mortality is very high especially in peripheral area of Sindh where medical facilities and trained staff particularly anaesthesia provider are lacking behind. We hope that our first issue will refresh some knowledge about obstetric anaesthesia and may improve healthcare at peripheral areas.

As this is a combined effort, so suggestions are always welcome.

Prof. Zahid Akhtar Rao
Editor, Newsletter PSA Karachi
Email: newsletter@psacentre.org

RECENT UPDATE ON POST-DURAL PUNCTURE HEADACHE (PDPH)

The International Headache Society has recently defined PDPH as "Headache occurring within five days of a lumbar puncture, caused by cerebrospinal fluid (CSF) leakage through the dural puncture. It is usually accompanied by neck stiffness and/or subjective hearing symptoms. It remits spontaneously within two weeks or after sealing of the leak with autologous epidural lumbar patch"[1]

The exact etiology of PDPH is due to the CSF leakage from the subarachnoid space through the dural perforation and the CSF leak via the dural breach being more rapid than its production.Subsequent headache occurs as CSF leaking leads to intracranial structure's tractionand stretching of intracranial and sensory nerves in the erect position. Secondly, as per Monro-Kellie doctrine, compensatory venous expansion occurs due to loss of CSFto preserveconstant volume of intracranial contentsleading to headache. Thirdly, altered distribution of cranio-spinal elasticity as postulated by Levine et al. could also be a possible mechanism for causing positional headache.

Treatment alternatives alleviate the symptoms by attempting to replace CSF which is lost due to leaking, decreasing cerebral vasodilatation and sealing the site of dural puncture.Abdominal binder, hydrationand bed rest are conservative therapies. Pharmacological therapies include simple analgesics which may provide relief of symptoms. Methylxanthine derivatives like caffeine and theophylline cause cerebral vasoconstriction by blocking adenosine receptors.Triptans used for migraine headaches, ACTH and synthetic analogues which increase CSF secretion and pain threshold, gabapentin, pregabalin, steroids, desmopressin, ondansetron and mannitol all have been used for symptomatic relief of PDPH but the evidence is limited. Kracoff et al. have recommended that hyperbaric oxygen therapy improves fibroblast proliferation at dural puncture site and facilitates closure and may have a role in PDPH management.[2]Recent study showed that intravenous neostigmine plus atropine improved PDPH symptoms without recurrence of headache.[3]

Epidural blood patch is still the treatment of choice for moderate to severe PDPH. Leaving an intra-thecal catheter insitu for 24 hours after accidental dural puncture stimulates a fibrotic response causing reduced dural defect and may be beneficial in reducing PDPH. Newer treatment include Spheno-palatine ganglion block (SPGB) which block parasympathetic activity leading to inhibition of cerebral vasodilation and cause symptomatic relief.[4]Greater occipital nerve block (GONB) is another modality which is simple and safe. It gives rapid symptomatic relief and effects may last for several weeks. Fibrin glue, an invasive modality, seals dural defect and prevents further CSF leak. It can be considered if there has been repeated failure of blood patch.

References:

1 J. Olesen. International classification of headache disorders. Lancet Neurol., 17 (5) 2018, 396-397
2. S.L. Kracoff, V. Kotlovker. Post dural puncture Headache. Review and suggested new treatment Open J.Anesthesiol., 09,2016,
3. Mahmoud et al. Addition of Neostigmine and Atropine to Conventional Management of Postdural Puncture Headache: A Randomized Controlled Trial. AnesthAnalg. 2018 (6):1434-1439
4. S. Cohen et al. Sphenopalatine Ganglion Block Compared with Epidural Blood Patch for Postdural Puncture Headache Management in Postpartum Patients: a Retrospective Review. RegAnesth Pain, Med 2018.

Dr. Malika Hameed
Fellow Obstetric Anesthesia
Department of Anaesthesiology,Aga Khan University, Karachi, Pakistan

Dr.Ali Sarfraz Siddiqui
Assistant Professor,
Department of Anaesthesiology,Aga Khan University, Karachi, Pakistan

2019-2020

NEWSLETTER
PAKISTAN SOCIETY OF ANAESTHESIOLOGISTS
KARACHI - CHAPTER

Volume: 20
Issue 01, July 2019

UPCOMING CONFERENCES / MEETINGS / SYMPOSIA

Colombian Congress of Anesthesiology 2019
August 21 - 24, 2019
Bogotá, Colombia

2nd International Congress on Pain and Palliative Care
August 23 - 24, 2019
Chiapas

35th Congress for the Scandinavian Society of Anaesthesiology and Intensive Care Medicine (SSAI)
August 28 - 30, 2019
Copenhagen, Denmark

21st ASEAN Congress of Anesthesiologists 2019
September 6 - 7, 2019
Brunei Darussalam

SAARC-AA 2019 Congress
October 10 - 13, 2019
Lahore, Pakistan

Critical Care Congress
December 14 15, 2019
Karachi, Pakistan

PSA Karachi Office:
Room I, Annex Building II,
Aga Khan III Road,
PMA House Karachi.
Tel: +92-21-32294795
Email: psakarachi@psacentre.org
Website: www.psacentre.org

OBSTETRIC HAEMORRHAGE MANAGEMENT

Massive obstetric haemorrhage
Massive obstetric haemorrhage is a major cause of maternal death and morbidity. It is variably defined as: blood loss >1500 ml; a decrease in haemoglobin >4 g/dl; or acute transfusion requirements >4 units.1
OR
vital signs >15%change
OR
>110 HR
BP<85/45
O_2 sat< 95%

- **Obstetric haemorrhage in Early Pregnancy**
  - Incomplete Abortion
  - Septic Abortion
  - Ruptured Ectopic
- **Antepartum haemorrhage (APH)**
  This is bleeding after 24 weeks gestation and before delivery 2
  - Placenta Previa
  - Placental abruption
  - Trauma
  - Uterine rupture
- **Secondary postpartum haemorrhage**

This is blood loss greater than 24 hours after delivery.
- Retained products of conception
- Puerperal sepsis

Emergency treatment of massive obstetric haemorrhage

- **Call for help (Most senior obstetric anaesthetist and obstetrician)**

Early management should be always as follows
- Airway
- Breathing (Oxygen)
- Circulation

Choice of Anaesthesia General Anaesthesia
Rapid Sequence Induction, Intubation and controlled ventilation or
Conversion of Regional Anaesthesia into General Anaesthesia as patients bleed heavily.
Treatment must be aimed at shock and preventing Disseminated Intravascular Coagulopathy
- Maintain the following:
  - Systolic pressure >90mm Hg
  - Urine output >0.5 ml/kg/hr
  - Normal mental status
- Eliminate the source of haemorrhage
- Avoid overzealous volume replacement that may contribute to pulmonary edema

Concurrently:
Warm all resuscitation fluids
Crystalloid, 2 litres maximum
Colloid, 1.5 litre maximum
Use group specific or O - Rh negative blood.
Whilst waiting ask somebody to set up a level 1 warmer and Rapid infusion (or similar) device
Monitor haematocrit and haemoglobin
Restore normovolaemia

- **If massive bleeding continues:**
  - Give 4 units FFP, cryoprecipitate and platelets as per requirement3
  - Use coagulation studies to guide the use of further blood products
  - Peri-operative monitoring as per the AAGBI guidelines
  - Consider invasive monitoring

Physical Treatment
If the uterus is atonic, stimulation of uterine activity by rubbing up a contraction using vigorous bimanual fundal massage for at least 15seconds

Pharmacological
- Oxytocin
- Ergometrine
- Carboprost (Synthetic 15 Methyl -Pgf)
- Cytotec (Misoprostol)
- Prostin

Surgical
- Manual removal of placenta
- Uterine packing
- Uterine and hypogastric artery ligation
- B Lynch suture to uterus
- Hysterectomy

Radiological
- Selective embolization of the pelvic vessels using interventional radiological techniques

References:
1. ACOG Practice Bulletin, Clinical Management Guidelines for Obstetrician Gynaecologists Oct 2006
2. Sara Catrin Cook et al, Key Clinical Topics in critical care 2014, Obstetric emergencies- haemorrhage, 232-234
3. J N Cashman et al, Lee's Synopsis of Anaesthesia 2017,Obstetrics, Haemorrhage 655-56

Dr Muhammad Amim Anwar
Consultant Anaesthetist,
Liaquat National Hospital Medical College

2019-2020

NEWSLETTER
PAKISTAN SOCIETY OF ANAESTHESIOLOGISTS
KARACHI - CHAPTER

Volume: 20
Issue 01, July 2019

PAINLESS LABOUR

Worldwide, about 140 million women give birth every year. Labour pain is one of the most severe pains which has ever evaluated but history is full of myths and controversies regarding the relief of pain in labour.

Mechanism of Labour Pain

The pain of labour has two components: visceral and somatic, and its anatomy is well documented. The first stage is mediated by T10 to L1 spinal segments, where as the second stage is carried by T12 to L1, and S2 to S4 spinal segments.
By understanding the mechanism, the 1st stage of labour pain can be blocked by para-cervical plexus blockade whereas the 2nd stage pain can be abolished by blocking pudendal nerve. However, the labour pain is influenced by many factors which can decrease or increase its severity related to general physical and mental condition of the mother.
Labour pain is not just unpleasant for mother, it has also negative effects on fetus by reducing oxygen transfer to the fetus and fetal metaboloic acidosis.

Methods of Pain Relief

The methods of pain relief can be dived into two broad categories:
A. Non-pharmacological
B. Pharmacological

Non-pharmacological: There are number of non-pharmacological methods for the relief of labour pain which includes Emotional Support, Childbirth Education, Massage, Aroma Therapy, Hydrotherapy, Intradermal water injection, Biofeedback, TENS (transcutaneous electrical nerve stimulation), Acupuncture, Acupressure and Hypnosis. However, the number of women studied has been small and there have been no proven scientific data analysis of the quality of pain relief offered by these techniques.

Pharmacological Methods

Inhalational Analgesia

Entonox: which is an equal-proportional mixture of (50:50) oxygen and nitrous oxide. It is not a potent analgesic but some beneficial effects are definitely delivered if it is properly inhaled. It is particularly useful if neuraxial analgesia is contraindicated.

Sevoflurane: is an anaesthetic which has got a very rapid onset and termination of action. This property makes it somewhat an ideal inhalational anaesthetic agent for labour analgesia.
In practice, inhalational gases are not commonly utilized in labour due to technical difficulties in their safe administration, scavenging, requirement of specific vaporizers and concerns about atmospheric pollution.

Systemic Narcotics/Analgesics:

Pethidine (meperidine): It is one of the most commonly used opioid derivatives which is commonly administered intramuscularly (IM) in a dose of 1mg /kg. However, its effect on the general progress of labour is quite controversial and various studies have concluded that pethidine should not be administered in parturients with cervical dystocia. Its active metabolite (nor-meperidine) has convulsant properties. Neonatal sedation and respiratory depression is common.
Morphine: Its metabolites do not have convulsant effects. However there is possible respiratory depression in the newborn, addiction, nausea and vomiting. The dose used for maternal analgesia is 0.1 0.15mg/kg.
Fentanyl: It is a highly lipid soluble synthetic opioid with 100 times higher potency than that of morphine. It can be administered in boluses of 25-50mcg every hour or as a continuous infusion of 0.25mcg/kg/hr. Advantages include absence of active metabolites and rapid onset of action making it useful for patient-controlled analgesia. However repeated dosing may result in drug accumulation in both the fetus and the mother.
Remifentanil: It is an ultra-short acting synthetic opioid with a rapid onset of action and a half life of 6 minutes. It can readily cross placenta but is extensively metabolized by the fetus. The recommended infusion dose is 0.025 mcg/kg/min which can be increased in incremental manner upto a maximum dose of 0.15 mcg/kg/min
Nalbuphine: It is a synthetic mixed mu-agonist/antagonist and a kappa agonist. It is administered in doses of 10-20 mg intramuscularly for the relief of labour pain. Sedation and dysphoria are the main disadvantages of this drug.
Butorphanol: It is a synthetic narcotic which is five times as potent as morphine and 40 times as potent as pethidine. The dose of butorphanol is 1-2 mg intramuscularly. Neonatal respiratory depressionis reported to be less than with pethidine. It is not frequently used for labour analgesia as it produces greater sedation.
Tramadol: For labour pain it has been prescribed in doses of 50-100 mg 4 hourly. It has no clinically significant respiratory depression but incidence of nausea is more common with tramadol than with pethidine or morphine

Whenever one intends to use opioid for labour pain, they should be ready with injection naloxone which is the opioid antagonist. The dose of naloxone for reversing neonatal respiratory depression is 0.1 ml/kg intramuscularly while for maternal respiratory depression, the dose is 0.4 mg intravenously.

Regional Analgesia

Epidural: Epidural analgesia is thought to be the most effective method of providing pain relief in labour. Low dose techniques offer the best chance of spontaneous vaginal delivery. The Comparative Obstetric Mobile Epidural Trial (COMET) published in 2001 demonstrated a 25% higher incidence of operative vaginal delivery in women who received traditional bupivacaine 0.25% epidural top-ups compared with women receiving low dose local anaesthetic and opioid top ups or infusions. There was no increase in the rate of caesarean section.
Combined Spinal Epidural Analgesia: CSE provides the advantages of a spinal (speed of onset) with the ability to prolong labour analgesia with an epidural catheter. Combination of fentanyl 10-25 mcg or sufentanil 2.5-10 mcg +/- bupivacaine 2.5mg can be used. As per the guidelines of Obstetrical Anaesthesia Association of United Kingdom (2005) CSEA is indicated only in specific circumstances.
Patient Controlled Epidural Analgesia: It requires infusion device. Typical low dose infusion of bupivacaine 0.0625% - 0.1% + 2 ìg/ml fentanyl run at 8-12ml/h titrated to block height provides adequate analgesia and haemodynamic stability. It allows patient to match dose of analgesia to amount of pain as labour progresses.
Non-Narcotic Adjuvant for Labour Analgesia: Clonidine and dexmedetomidine are alpha-2 agonists that can be used as adjuvant to local anaesthetics and they act at the dorsal horn of the spinal cord to produce analgesia.

It is the right of every woman to have a painless labour. Labour analgesia has to be popularized and should be delivered to every demanding and desirous mother. We should ensure to offer her a comfortable and painless process of labour irrespective of the method thus employed.

References

1. New WHO guideline on intrapartum care, WHO News release - February 2018
2. Sukhwinder Kaur Bajwa et al Painless labour: how far have we travelled?, Sri Lanka Journal of Obstetrics and Gynaecology 2010; 32: 93-98
3. Simona Labor et al, the Pain of Labour, Reviews in Pain 2008 Dec; 2(2): 1519.

Dr. M. Kashif Iqbal
Anaesthesia Specialist,
The Indus Hospital Karachi

2019-2020

NEWSLETTER
PAKISTAN SOCIETY OF ANAESTHESIOLOGISTS
KARACHI - CHAPTER

Volume: 20
Issue 01, July 2019

PRE-ECLAMPSIA AND ITS MANAGEMENT

Pre-eclampsia can be defined as 'a blood pressure =140/90 mmHg after 20 weeks of gestation and involvement of one or more organ systems with previously normal blood pressure'.[1]

The ACOG defines preeclampsia as 'the development of hypertension with proteinuria, edema or both induced by pregnancy after the 20th week of gestation.[2]

The (ISSH) in its definition of preeclampsia does not include edema because it may be detected in 80% of normotensive pregnant women most of whom are healthy.[3]

Preeclampsia is a disease of abnormal placentation;pathogenesis is complex and incompletely understood but, there is consensus that the primary disease is an abnormality of placentation.

In normal placentation, maternal uterine spiral arteries run through the myometrium and into the endometrium,which in pregnancy is replaced by the decidua. Trophoblastsof foetal origin invade these spiral arteries resulting in a loss of elasticity and vascular smooth muscle tone.

Arteries are remodelled into low resistance capacitance vessels, which provide sufficient placental perfusion to sustain the growing foetus,but, in preeclampsia, trophoblastic invasion is much shallower, affecting only the spiral arteries in the decidua. The myometrial sections remain small and constricted, resulting in a defective uteroplacental circulation with a higher resistance. This gives rise to subsequent placental ischaemia. Cause is unknown, but genetic and immunological mechanisms are thought to be important contributors.

Preeclampsia is a two-stage disorder. The asymptomatic first stage occurs early in pregnancy and corresponds to the period of abnormal placentation. Symptomatic second stage of preeclampsia develop the maternal syndrome, characterised by

hypertension, proteinuria and multiorgan involvement.[4]

The underlying cause of the maternal syndrome is thought to be systemic endothelial dysfunction.

Management:

Preeclampsia can be further classified into mild, moderate or severe, and typically, worsening severity is associated with more abnormalities in other organ systems ofthe body.Management should be undertaken with a multidisciplinary team approach.Delivery of the placenta is the only cure.[5]

Worldwide, there is no consensus yet about what the target blood pressure should be and which drugs to use to achieveit. In the USA, targeted values are less than 150/100mmHg; in Canada and Australia, the target is a DBPof 90 to 109mmHg, and 90 to 105mmHg in Germany. All are higher than the UK recommendation, which is to aim for a DBP of 80 to 100mmHg[5]

Greatcare must be taken to avoid precipitous decreases in blood pressure, as this can adversely affect uteroplacentalperfusion.

Drugs that are in common use are Hydralazine, Labetalol, Nifedipine, Urapidil and Methyldopa.[5]

Pulmonary oedema is a recognized cause of death in women with Preeclampsia.

Despite this, preeclampsia is regarded as a pathologicalstate of intravascular volume depletion, and acute renal failure secondary to acute tubular necrosis is another well recognized, albeit rare, complicationConsequently, most units now have a fluid management protocol to guide

intravenous fluid replacement so that the risks of pulmonary oedema and renal failure are minimised, and this usually takes the form of a fluid restriction protocol[5]

Obstetricians decide on the timing of delivery having weighed up the benefits of continuing a pregnancy for the foetus against the risk of maternal morbidity and mortality rising with increasing gestation.[5]

Anesthetic Management

Anesthesia management of these patients is very chalanging.

The expertise of an experienced anesthesiologist is required with particular attention on

fluid management, stabilization of blood pressure and seizure prevention.

Epidural or combined spinal-epidural techniques are recommended for preeclamptic women in labour.

For Caesarean delivery, neuraxial techniques are also preferred to general anaesthesia. However, the traditionalview that epidural anaesthesia should be the neuraxialmethod of choice has now been rejected. It has been shown that women with severe preeclampsia experience less hypotension under spinal anaesthesia than normotensive patients.[6]

One well recognisedcomplication of preeclampsia is thrombocytopaenia, and a low platelet count is a relative contraindication to regional blockade because of concerns regarding epidural haematoma formation.

When considering this risk for Caesarean delivery, it must be balanced against the risks of general anaesthesia. There is no threshold for platelet count that divides high risk and low risk for epidural haematoma, and it is necessary to rely on expert and consensus opinion. A common view is that a stable platelet count of more than 75 000 in theabsence of other coagulation abnormalities should be well tolerated and should provide no greater risk than performing a general anaesthetic for a preeclampticwoman in labour with a full stomach.[5]

Occasionally, it is necessary to provide general anaesthesiafor Caesarean delivery. This may be appropriate for women with contraindications to regional anaesthesiasuch as coagulopathy, or for those who have developed severe complications such as pulmonary oedema or depressed consciousness following eclamptic seizuresIt is also extremely important to reduce the hypertensive response to laryngoscopy, which can be severe and has been identified as a cause of maternal mortality. Consideration should also be paid to the interaction between magnesium sulphateand nondepolarising muscle relaxants, whose action can be prolonged in the presence of a magnesium infusion.[5]

Reference:

1. Cantwell R, Clutton-Brock T, Cooper G, et al. Saving Mothers'Lives: reviewing maternal deaths to make motherhood safer:20062008. The Eighth Report of the Confidential Enquiries intoMaternal Deaths in the United Kingdom. British Journal ofObstetrics and Gynaecology 2011; 118: 1203.
2. American College of Obstetrcians and Gynecologists practicebulletin.Diagnosis and management of preeclampsia andeclampsia. Number 33, January 2002. International Journal of Gynaecology and Obstetrics 2002; 77: 6775.
3. TranquilliAL,DekkerG,Magee L et al.**The classification, diagnosis and management of the hypertensive disorders of pregnancy: A revised statement from the ISSHP.** *Pregnancy Hypertens.* 2014; **4**: 97-104
4. Uzan J, Carbonnel M, Piconne O, et al. Preeclampsia: pathophysiology,diagnosis, and management. Vasc Health Risk Manag 2011; 7:467474.
5. Stocks, G. **Preeclampsia: pathophysiology, old and new strategies for management.** *Eur J Anaesthesiol.* 2014;31:183189
6. Aya AGM, Mangin R, Vialles N, et al. Patients with severe preeclampsiaexperience less hypotension during spinal anesthesia for elective cesareandelivery than healthy parturients: a prospective cohort comparison. AnesthAnalg 2003; 97:867872.

Dr. Mujeeb Ahmed Khan
Consultant Anesthetist,
Department of Anaesthesiology, Kidney Center, Karachi, Pakistan