# Neo Clips

## NATIONAL NEONATOLOGY FORUM DELHI

MONTHLY E-BULLETIN

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# *From President Pen*


## DR. PRADEEP KUMAR DEBATA

Professor
Division of Neonatology
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President NNF Delhi

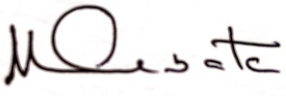

Dear NNF Delhi Members
Wish you all a happy and healthy new year on behalf of NNF Delhi. May this year bring new accolades to our most loved NNF Delhi branch.

It's a feeling of pride that we are coming with our new edition of NeoClips (Neonatal Clinical Practice) for the 2nd year. In this occasion, I am feeling privileged to congratulate our Editorial Board Members, chaired by Dr. T J Antony and our Editor in chief Dr. Navin Prakash Gupta, who are working tirelessly, to make each edition of NeoClips to see the light.

NeoClips is the platform for the Neonatologist to publish their studies, review articles and their clinical experience as case reports which help adding to the current knowledge. I am thankful to the authors who are contributing with their research works for publication and request all our Esteemed Members to contribute their research works and experience in a big way to enrich it further.

We all, as Neonatologists, are contributing towards the better survival of the neonates. Many national and international programs are floating to decrease the neonatal mortality and successfully we are approaching towards our set target by reducing the deaths among extreme preterm and asphyxiated neonates. Is this enough? Mere survival of neonates is not the solution. I feel we have to rethink about our current strategy not only to decrease the neonatal mortality but also to have intact survival of our children. In this year of 2023, let us all focus on the intact survival of neonates rather than just decreasing the Neonatal mortality.

With Regards



**Dr. Pradeep Kumar Debata**
President, NNF Delhi

# *From Secretary's Pen*

## DR KUMAR ANKUR

Secretary, NNF Delhi

**Dear friends**,

**Warm greetings from National Neonatology Forum, Delhi!**

It gives me immense happiness to see the success of NNF Delhi monthly E- Bulletin, launched in February 2022 with the name of '***NeoClips' (Neonatal Clinical Practice)***. Every month it's getting better & better. And credit goes to the Chief editor Dr Naveen Gupta & his exceptional team. OSCE as system wise which would be very helpful for Neonatal fellows/Residents/Postgraduates. We have covered review article on developmentally supportive care in NICU which is an important aspect of premmie care. Some interesting cases like gastrointestinal mucormycosis and arachnoid cyst has been covered in this edition.

We are requesting all the esteemed members to contribute to these E-bulletins. We shall be giving the due credits to all the contributors.

We eagerly look forward to your feedback and hope to give you an experience that you will cherish forever!

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**Dr. Kumar Ankur**
Secretary, NNF Delhi

# Editor's Desk


## DR NAVEEN PARKASH GUPTA

Chief Editor, Neo Clips


**Dear Friends,**

**Greetings from the NeoClips team.**

As Editor I would like to start by thanking the editorial team for the effort and the authors for their contribution to this and previous issues.

We have covered some interesting topics in the present issue.

An interesting case series of gastric perforation has been covered in case report section.

Developmentally supportive care in NICU is an important aspect of premature care. It has been covered nicely in review section by Dr Amitava Sen Gupta.

An interesting case of neonatal gastrointestinal mucormycosis has been covered in the picture of the month.

The image section describes a case of arachnoid cyst.

Few questions on renal disorders are covered in OSCE section.

We hope that you will enjoy reading this issue. Please share your feedback with us. It will help us improve the journal.

**Dr Naveen Parkash Gupta**

# An interesting case of pneumoperitoneum in a neonate

**Dr Swetha[1], Dr Naveen Parkash Gupta[2], Dr Shandip Kumar Sinha[3], Dr Anil Batra[2], Dr Praney Gupta[3]**

1. NNF Fellow, Department of Neonatology, Madhukar Rainbow Children's Hospital, Delhi
2. Senior Consultant, Department of Neonatology, Madhukar Rainbow Children's Hospital, Delhi
3. Senior Consultant, Department of Pediatric Surgery, Madhukar Rainbow Children's Hospital

Case – Late preterm (35 weeks) small for gestational age, second of the twins, born to a G3P2L2A0 mother through vaginal delivery with a birth weight of 1.5 kg vigorous at birth. The baby was shifted to NICU in view of very low birth weight. The baby was comfortable on room air and was hemodynamically stable. The baby was initially started on formula feeds which he tolerated well on day 1 of life. On day 2 of life, the baby started developing abdominal distention which was gradually progressing and hence baby was referred to a higher centre for further management. At that centre, the Sepsis screen was positive with raised CRP and platelet count of 1.6 lahks. X-ray abdomen was suggestive of gas under the diaphragm- football sign+ve (figure 1).

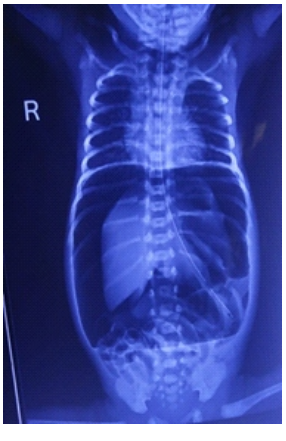


*Fig. 1 : Xray showing pneumoperitnoeum*

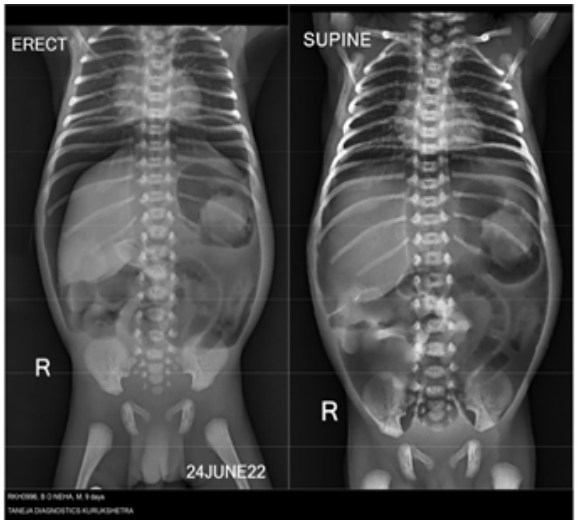


*Fig. 2 : Xray abdomen on post operative day 4 showing pneuoperitoneum (anastomotic leak)*

Ultrasound abdomen was suggestive of moderate ascites with diffuse circumferential edematous mural thickening of bowel loops suggestive of intestinal perforation. Baby underwent exploratory laparotomy which showed a burst-out stomach with a large tear along the greater curvature and small tear in the anterior and posterior wall of the stomach. Primary gastric perforation repair was done. Baby was kept NPO, started on parenteral nutrition and IV antibiotics were continued. Baby further developed abdominal distension on day 7 of life (Post op day 4). Anastomotic leak was suspected. Xray abdomen was repeated which was suggestive of gas under the diaphragm (figure 2). Reexploration was planned. The parents decided to shift the baby to our center.

At admission, baby was on room air, hemodynamically stable. On examination, the abdomen was distended and shiny with absent bowel sounds. The baby was kept NPO, and started on TPN/ IV antibiotics after sending sepsis workup. X-ray abdomen was suggestive of perforation. Exploratory laparotomy was done showed dehiscence of the previously

repaired stomach with generalized peritonitis (figure 3).

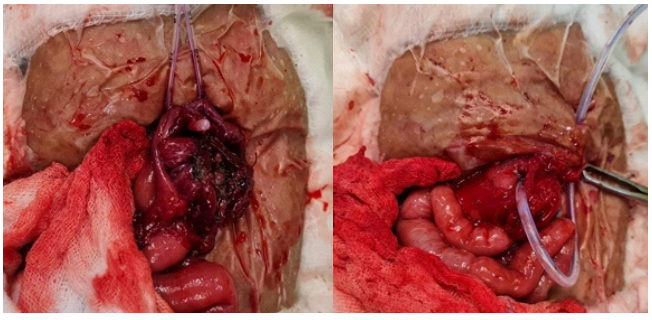

*Fig. 3 : a. Peroperative pictureshowing necrotic gastric wall*
*b. Post repair Gastrojueneostomy tube put for postoperative feeding purpose*

Gastric wall was friable and was not holding sutures. Necrosed, friable part of the stomach was resected, (sent for biopsy) and a partial gastrectomy was done with feeding jejunostomy tube on day 9 of life. Baby showed persistent thrombocytopenia with lowest platelet counts of 80,000 with culture positive sepsis suggestive of Vancomycin resistant enterococcus faecium. Peritoneal fluid culture was suggestive of candida. Baby received iv antibiotics Inj Linezolid and Amphotericin B for 14 and 21 days respectively. Biopsy was suggestive of congested acute necrotizing suppurative gastritis with large dilated vascular channels with fibrin thrombus and hemorrhage. Post operatively, continued on mechanical ventilation, developed hypovolemic shock with poor peripheral pulses and mean BP <30 mm hg for which baby received NS bolus and inj dopamine infusion which was gradually tapered over 48 hours. One PRBC transfusion was given. Chest Xray was done on post op day 1 which was suggestive of left lung collapse. Baby was continued on mechanical ventilation, IV antibiotics. Chest physiotherapy was initiated. Repeat chest Xray after 48 hours showed collapse of right upper lobe lung suggestive of VAP (figure 4).

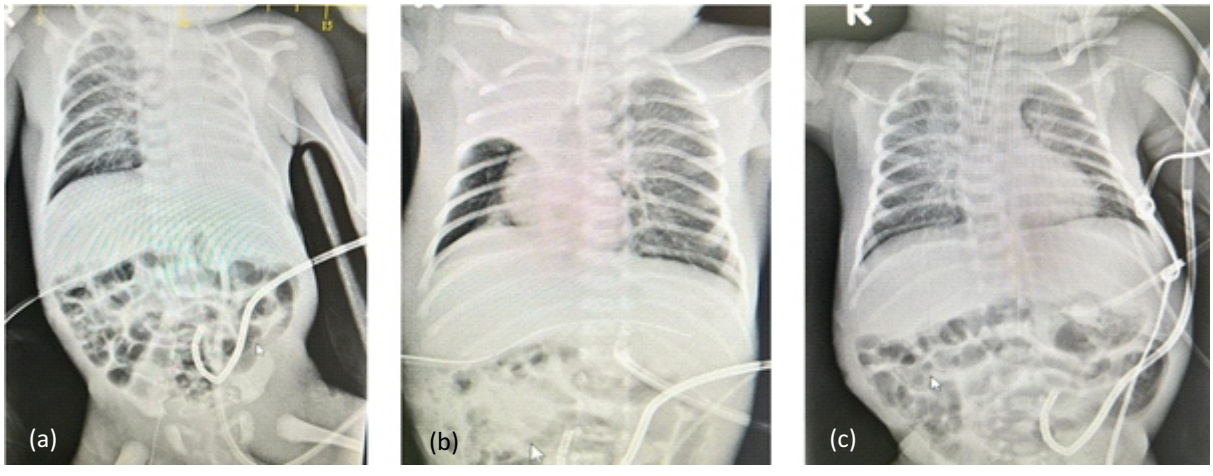


*Fig. 4 : Serial xrays of baby showing left lung collapse (a), Right upper zone collapse (b), no collapse in both lungs (c)*

Baby was extubated on day 18 of life to HHHFNC and subsequently to room air on Day 19 of life. Baby was restarted on minimal jejunostomy continuous feeds at 1ml/ hour from post op day 3. Baby had one episode of bilious vomiting for which feeds were withheld for 24 hours. Anti-reflux medication was started and feeds were restarted from post op day 5 of life. Feeds were gradually increased and baby reached full feeds by day 20 of life. In view of prematurity with prolonged ventilatory support, fungal positive sepsis (peritoneal fluid) ROP screen was on day 21 of life which showed bilateral eye zone 2 posterior, stage 2-3 plus disease. No fungal lesion was seen in retina or vitreous. Laser photocoagulation was done for the same. 2D Echo/ USG Cranium and KUB was done-normal. Paladay feeds (small amount) were initiated from day 28 of life gradually which the baby tolerated well. Baby was discharged on day 30 of life on jejunostomy feeds and paladay feeds 5ml every 2 hourly. 4 days after discharge, baby developed vomiting, was admitted at a local hospital, where sepsis screen sent was positive, given IV antibiotics. Feeding jejunostomy and gastrostomy tubes were removed, started on paladay feeds which baby tolerated well. Presently baby doing well, on direct breast feeds, present weight- 2.5 kg at 2.5 months age.

**Review of Literature:**

Neonatal gastric perforation (NGP) is a very rare, serious, and life-threatening problem. The reported incidence is around 1:5000 live birth. It accounts for approximately 7% of all gastrointestinal perforations in neonates. The first case of gastric perforation was reported in 1825 by Siebold and first successful surgical treatment by Leger in 1950.

Although multiple theories have been advocated explaining the aetiology of NGP, pathogenesis and aetiology remain unknown. Previously, NGP was often described as spontaneous, a perforation in the stomach of a newborn of no demonstrable cause. In one of largest study of 168 neonates with gastric perforation, majority (79 neonates) had idiopathic perforation. In the rest of the babies prematurity, necrotizing enterocolitis (NEC), asphyxia, postnatal corticosteroid administration, nasal ventilation, and gastric trauma were implicated as contributing factors.

**Pathogenesis -** Anatomic defects of the gastric muscular wall have been suggested to potentiate perforation of the stomach among neonates, especially in prematurity.

The circular muscle layer of the newborn stomach normally contains several gaps, most prominently in the fundus, near the greater curvature. These gaps are more common in premature infants. Under normal circumstances, such gaps may have little clinical

significance, but they are potential weak points in the stomach wall that might be susceptible to rupture if intragastric pressure increases. Additionally recent findings suggest that lack of the interstitial cells of Cajal (gastrointestinal pacemaker cells), may predispose some neonates to NGP. Incoordination and immaturity of esophageal motility causing increased intragastric pressure and reduced blood flow during hypoxic events or NSAID treatments are other important factors implicated in pathogenesis of disease.

Peak incidence of presentation occurs at 2-7 days of life with common presenting features abdominal distention, feeding intolerance, respiratory distress, poor activity, gastrointestinal bleeding, abdominal erythema, hemodynamic changes as shock.

Greater curvature of the stomach is the most common affected site (73.8%) followed by lesser curvature( 13.1%), anterior wall (9.0 %) and posterior wall (4.1%).

Treatment of choice is Surgical repair with necrotic tissue excision and gastrorrhaphy.

Rarely partial gastrectomy is done in case of massive gastric disruption. Sometimes gastrostomy is needed. Feeding jejunostomy helps in the early initiation of feeds in post-op period. Gastric perforation carries a high mortality ranging from 27-83%.

Poor prognostic factors include male sex, hyponatremia and metabolic acidosis

In a largest case series report, Sepsis was the only variable associated with high mortality.

**Key messages**:

- Neonatal gastric perforation is an uncommon but life-threatening condition.
- Abdominal distension and pneumoperitoneum on x-ray should raise suspicion.
- Hemodynamic status of the child, lab parameters and bowel pattern on x-ray may differentiate it from NEC when pneumoperitoneum is present.
- Primary surgical repair is the treatment.
- Carries poor outcome.

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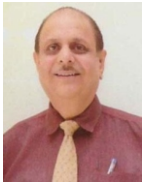

# Overview of Developmentally Supportive Care (DSC) in Preemies & Neuroprotection in the NICU

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**Fellowship Neonatology (Neth)**
**FNNF, MICP, DCH**
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Chairperson & Executive Director DSC Foundation for newborn & children (India)

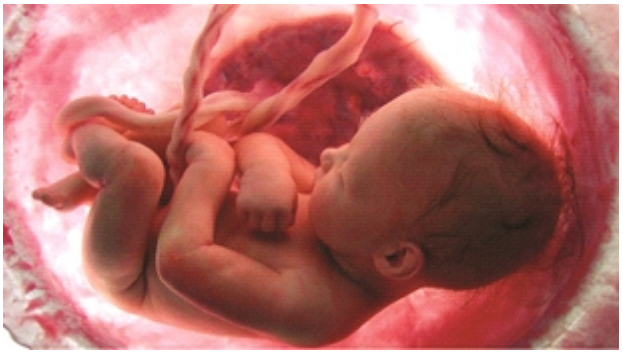
## Background

Significant technologic advances with enhancement of knowledge over the past three to four decades in antenatal, perinatal and neonatal medicine have resulted in increased survival of Extremely Low Birth Weight (ELBW) in tertiary level NICUs across the globe. Infants born as early as 24 weeks gestation now have a fair chance of survival.

However, this progress comes with great physical, emotional, and financial costs because premature infants spend many weeks and months in the neonatal intensive care unit (NICU). Because development continues outside the protective environment of the womb for the prematurely born foetus, many have impaired short- and long-term outcomes.

Preterm birth interrupts the precise process of foetal maturation in utero, hence forcing critical neurologic growth to continue within the Neonatal Intensive Care Unit (NICU). There is increasing concern for the impact of the NICU experience on the developing brain. The typical NICU provides a very different environment for the continued maturation of the preterm infant's CNS.

**INTRAUTERINE ENVIRONMENT (MATERNAL WOMB)** is favourable for **POSITIVE SENSORY INPUTS** which are crucial for normal brain development in a growing foetus. It protects the developing foetus against harsh outside stimulation and provides a variety of sensory stimuli in an integrated Multimodal fashion. These include: (1) Tactile and Vestibular, (2) Chemical and Hormonal (3) Auditory and Visual systems.



The Intrauterine milieu is characterized by:

1. Generalized flexion and gentle, secure containment
2. Limited light and noise exposure
3. Normal sleep cycle preservation and development.
4. Unrestricted access to mother via somatosensory, chemosensory, and auditory pathways.

## NICU Environment

In contrast, this exposes the micro preemie to an array of painful procedures with separation from mother and disturbance in sleep cycles.

FLUCTUATIONS IN STIMULI include:

- Temperature and Tactile (Touch)
- Vestibular
- Olfaction (Smell) and Gustatory (Taste)
- Auditory (Sound) and Visual (Light)

There are also FLUCTUATIONS in Oxygenation and Nutrients and the infant experiences scenarios which are very different from those experienced in Utero.

**Negative Sensory inputs replace the Positive Sensory inputs into the developing brain, which can permanently alter normal brain development.**

## Preterm Birth

In the present times, the incidence of prematurity is high in both western and eastern worlds. An estimated 15 million are born preterm every year and

India leads with 3,519,100 preterm births (almost 24 %) of the total number.

Infants born prematurely miss an important intra-uterine motor milestone which is **development of flexor tone. This critical component of muscle development occurs throughout the third trimester of pregnancy.**

Preemies born at 26 to 28 weeks Gestational Age are in a posture of Extension with Hypotonia.

| 26 to 28 weeks Gestational Age - Extension | |
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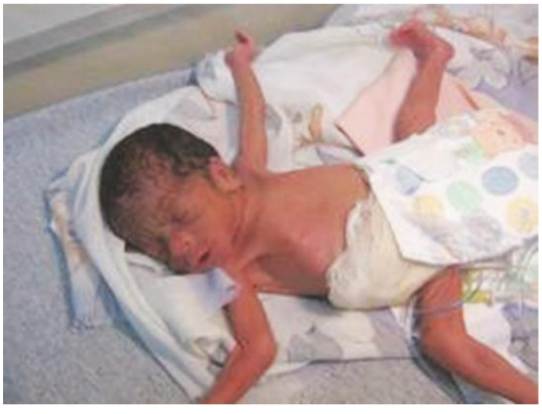
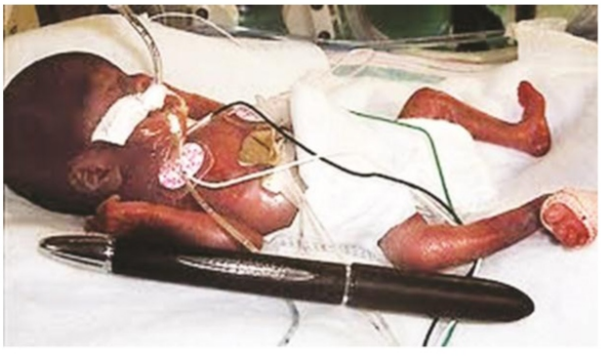
|  |  |
|  | **Gravitational spull-on body further enhances the posture of Extension** |

| 34 to 36 Weeks Gestational Age - Flexion | | |
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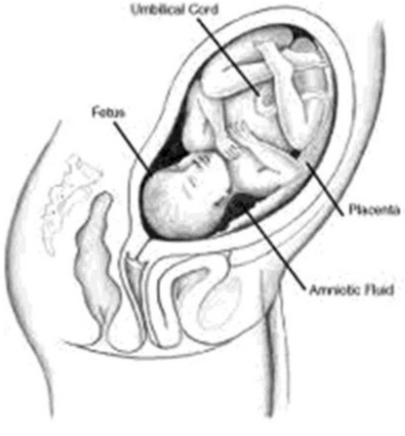
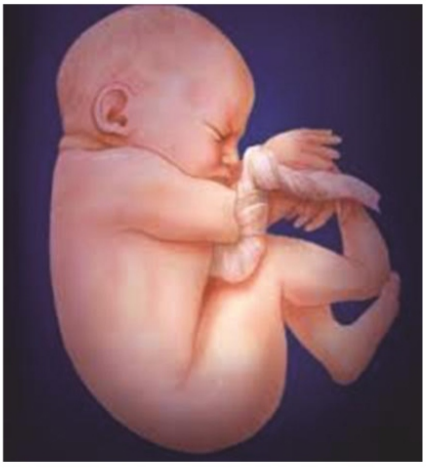
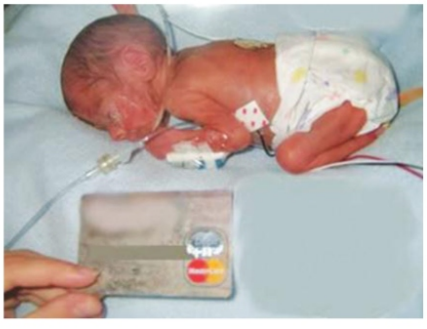
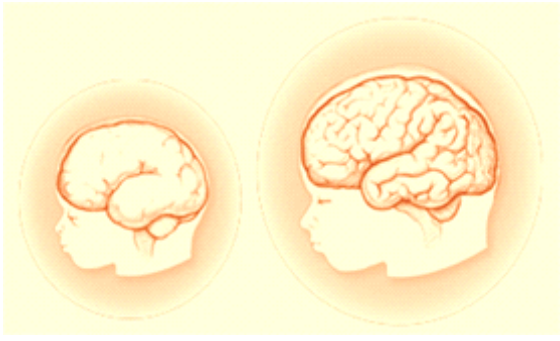
|  |  |  |

### Preterm Birth & Neuroplasticity



"In 3rd trimester of foetal development and even in early infancy the brain is drastically changing with new brain cell production and migration, synaptic pruning and brain organization".

Third trimester of pregnancy is a most crucial period, during which the basic Foundation for the Neuromotor and Neurobehavioral systems is being laid in the growing foetus.

**"(Vanderberg, 2007; Volpe, 1995 as cited by Legendre, Burtner, Martinez, & Crowe, 2011)**

However, in spite of the rapid progress in neonatal medicine, the morbidity rate of Neurodevelopmental Impairment has not decreased for this population. Neurodevelopmental outcome still remains a major issue of concern for these infants.

**Our Goal Should be to Improve Functional Outcome, have Positive Neuro Developmental Outcome and Achieve the Gold Standard of "INTACT SURVIVAL" of the Preterm, Fragile and/or Critically Ill Infants.**

**Developmentally Supportive Care for High-Risk Infants**

For preemie infants born early, we should aim to mimic the "In-Utero" environment as far as achievable in the infant care area and NICU setting.

### What is Developmentally Supportive Care (DSC)?

Developmentally Supportive Care (DSC) practices are evidence-based interventions that promote newborn brain and neurobehavior development. They support autonomic stability, normal motor, sensory neurological development and promote behaviour state organization.

DSC practices are designed to minimize the stress of the NICU environment and include elements such as control of external stimuli (auditory, visual, tactile, vestibular), clustering of nursing care activities to avoid disrupting sleep, positioning or swaddling of the preterm infant and calming techniques'

The aim is to provide a structured care environment which supports, encourages, and guides the developmental organization of the premature and/or critically ill infant

### Benefits of DSC

When in the NICU neonates are under severe and often life-threatening stress. They have immature and or fragile autonomic and nervous systems. DSC can give them more reserve to heal, minimize effects of trauma, and promote normal development of nervous system.

It also decreases length of hospital stay, improves weight gain and shortens the time to full enteral feeding. The neuro-developmental scores at 9-12 months age were seen to be improved.

### Who provides DSC? NICU Team


**Providers of developmentally supportive care (DSC) in the neonatal intensive care unit (NICU). Image Courtesy: With permission from DSC Foundation for Newborn and Children, India**

### Neuroprotective Care

Neuroprotection encompasses all strategies that support the developing brain, facilitating normal development and reducing disability. In the event of neuronal injury, these interventions are intended to help the brain limit neuronal cell death and permit healing by creating functional synaptic connections and pathways **(Bader, 2014; McGrath et al., 2011).**

It is mandatory that every effort be made to minimize negative experiences for the preterm infant. Parents and professional caregivers can work together to minimize the adverse impact of the NICU experience, hopefully reducing subsequent impairment and disability **(Altimier & Phillips, 2013).**

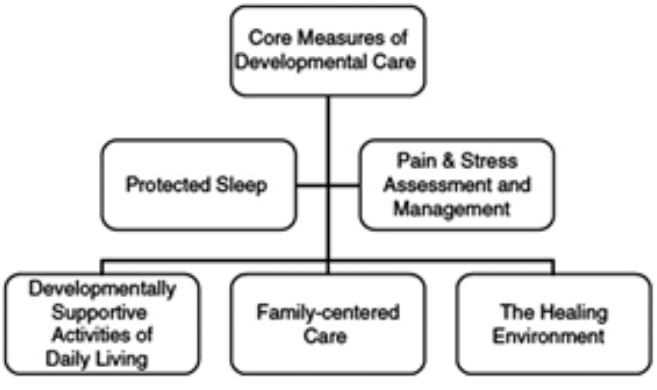
### Evolution of DSC Models

**1. COUGHLIN ET AL, 2009 - PROPOSED FIVE DEVELOPMENTAL CARE CORE MEASURES.**

**Focus on Care Actions**

Core measures are focused on neonatal caregiver actions which are disease independent but nonetheless essential to promote healthy growth and development of the infant and family. The proposed five core measures represent the first step in operationalizing evidence-based developmental care.

1. Protected sleep
2. Pain and stress assessment and management
3. Activities of daily living (positioning, feeding and skin care),
4. Family-centred care
5. The healing environment.

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**2. THE NEONATAL INTEGRATIVE DEVELOPMENTAL CARE MODEL**

**The Neonatal Integrative Developmental Care Model** utilizes neuroprotective interventions as strategies to support optimal synaptic neural connections, promote normal development and prevent disabilities.

(Leslie Altimier Raylene M. Phillips, 2013)

This provides more practical guidance for NICU staff in delivering developmental care to preterm infants in the NICU. The five neonatal core measures first introduced by Coughlin et al in year 2009 have been **recategorized and expanded into Seven distinct core measures of Neuroprotective neonatal care.**

**(Leslie Altimier Raylene M. Phillips, 2013, Newborn & Infant Nursing Reviews 13 (2013) 9–22)**

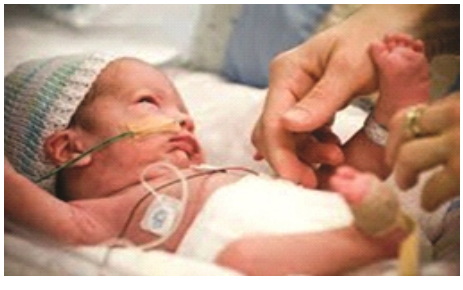
1. The Healing Environment
2. Partnering with Families
3. Positioning & Handling
4. Safeguarding Sleep
5. Minimizing Stress & Pain
6. Protecting Skin
7. Optimizing Nutrition and Feeding.

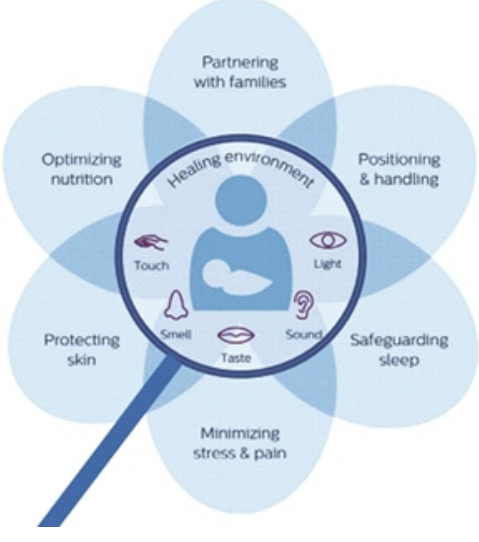
**Neuroprotection in the NICU**


**Seven neuroprotective core measures of DSC depicted on petals of Lotus. (DSC: Developmentally Supportive Care; NICU: neonatal intensive care unit)**

**Photo courtesy: With permission from DSC Foundation for Newborn and Children, India**

**Source: Adapted from the Integrative Developmental Care (IDC) Model; Philips Healthcare Andover MA, USA**



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**The Neonatal Integrative Developmental Care Model (IDC) (Philips Healthcare Andover, MA. USA)**

# Therapeutic Positioning in the NICU

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## Background

**"Therapeutic Positioning" is a very cost-effective intervention to promote Neuromotor and Neurobehavioral development in the "Preterm/fragile and Critically ill new-borns"**

**In the third trimester the foetus is establishing the important milestone of physiologic flexion which will impact motor development and the ability of the infant to self-calm and gain neurobehavioral control.**

A new-born preterm infant is deprived of these basic developmental needs upon transition from the womb to the hostile environment of the newborn intensive care unit (NICU).

It is now increasingly clear that new-born babies respond to and are affected by their posture, position and environment around them.

## Aims of Positioning the Preterm or Sick Neonate in NICU

The preterm or sick infant requires support to adapt and maintain postures that enhance motor control, physiological functioning and reduction of stress.

The developmental goals of positioning are:

- Provide flexion in the limbs and develop midline orientation
- Assist the infant in self-regulation and maximize stability.
- Energy and Heat conservation
- Promote Growth and CNS organization

## Practical considerations for positioning

The position should optimise the infant's ability to breathe independently and we should ensure that upper airway is not compromised. Infants with increased respiratory demands may be more stable in prone. Evidence suggests prone position provides improved respiration and greater chest wall synchrony and improved gas exchange.

Preterm infants are hypotonic (floppy) at less than 28 weeks gestational age (GA) and start to develop increased tone (resistance) at around 30 weeks GA. Flexor strength develops first in the lower limbs and gradually progresses to the upper limbs. Extensor muscles are stronger than flexor muscles until they balance out around term age (corrected). Muscle imbalance and hypotonia hamper the preterm infant from achieving the midline position and adequate self-regulation.

The loss of a fluid environment and the impact of gravity further limit movements of the preterm infant. The loss of boundaries (uterine wall) to resist and strengthen against also confine movements of the infant.

The use of high boundaries achieved by nesting provides the baby containment and facilitates midline movements (eg. hands to face). Boundaries of nest need to be high enough to contain the legs and close enough to enable the baby to brace his feet against it.

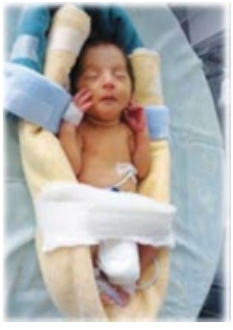
As preterm infants get closer to term their movements become smoother and they become capable of bringing limbs into flexion. All positions should reduce the impact of gravity and support the infant with hands to midline and legs flexed. Side lying with nesting is the position that facilitates this best.

We are now going to share with you "**Potentially better DSC practices**" /formats, in **Therapeutic Positioning** which are being implemented in our neonatal unit to **support neurodevelopment of infants.**

## Practical Aspects in Therapeutic Positioning and Nesting

In the preterm infant, it is noteworthy to understand the **importance of good infant positioning in**

**achieving reduction of stress, good self-regulation (calming), protected sleep and well-defined Arousal states.**



**Courtesy:**
**With permission Dr Amitava Sengupta.**
**DSC Foundation for Newborn and Children, India**

**POSITON CHECK LIST**

| **Details** | **Status** |
|---|---|
| Change Position with each Care | ☐ |
| Head: midline | ☐ |
| Neck: neutral | ☐ |
| Shoulder: rounded forward | ☐ |
| Trunk: support for flexion | ☐ |
| Limbs: support for flexion | ☐ |
| Hips: knees midway | ☐ |
| Feet: neutral | ☐ |
| IVs can flow? | ☐ |
| Breathing comfortably? | ☐ |
| Baby calm? | ☐ |

## Modified Infant Position Assessment Tool (IPAT) 2020

Patient's Name: : DOB :
Gestational Age: Birth Weight:
Clinician performing assessment:
Date/Time of Assessment:
Infant position (circle); Supine Side -lying Prone

| | | | | Date | | | Date | | | Date | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Indicator | 0 | 1 | 2 | M | E | N | M | E | N | M | E | N |
| Head | Head rotated laterally (L/R) greater than 45° from midline | Head rotated laterally (L / R) 45° from midline | Head positioned to less than 45° from midline (L or R) | | | | | | | | | |
| Neck | Neck hyper extended | Neck neutral but poorly aligned with spine | Neck in neutral position and slightly flexed to align with spine | | | | | | | | | |
| Shoulders | Shoulder retracted | Shoulders flat/in neutral | Shoulders softly rounded forward | | | | | | | | | |
| Hands Arms | Hands away from the body Arms Extended | Hands touching torso Arms Extended | Hands touching face Hands on chest in midline Arms Flexed | | | | | | | | | |
| Hip Pelvic position | Hips abducted/externally rotated and/or in extension | Hips in alignment but extended | Hips aligned and softly flexed | | | | | | | | | |
| Knees, ankles, feet | Knees extended, ankles and feet externally rotated | Knees, ankles and feet extended | Knees, ankles and feet are aligned in midline orientation and softly flexed | | | | | | | | | |
| | | | Total Score | | | | | | | | | |
| | | | Heart Rate/Min | | | | | | | | | |
| | | | Respiratory Rate/Min | | | | | | | | | |
| | | | Oxygen Saturation (SPO) | | | | | | | | | |

Modified and prepared by Dr Amitava Sengupta, DSC Foundation for Newborn and Children (India) 2020. Adapted from Infant Position Assessment Tool (IPAT), Courtesy with permission: Philips Children's Medical Ventures

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**Courtesy: With permission from Philips Children's Medical Ventures.**
**Sources: Modified and prepared by Dr Amitava Sengupta, DSC Foundation for Newborn and Children, India, 2020; Adapted from Infant Position Assessment Tool (IPAT)**

## Modified Infant Position Assessment Tool (IPAT) 2020

### SCORING PATTERN

- Six items are scored on an ordinal scale of 0, 1, and 2.
- Minimum score: 0, Maximum score 12.
- Optimal IPAT core 10-12; ideal acceptable score ≥9.
- If score ≤8: infant needs correction in positioning.

Interval between two recordings of position scoring should preferably be three to four hours.

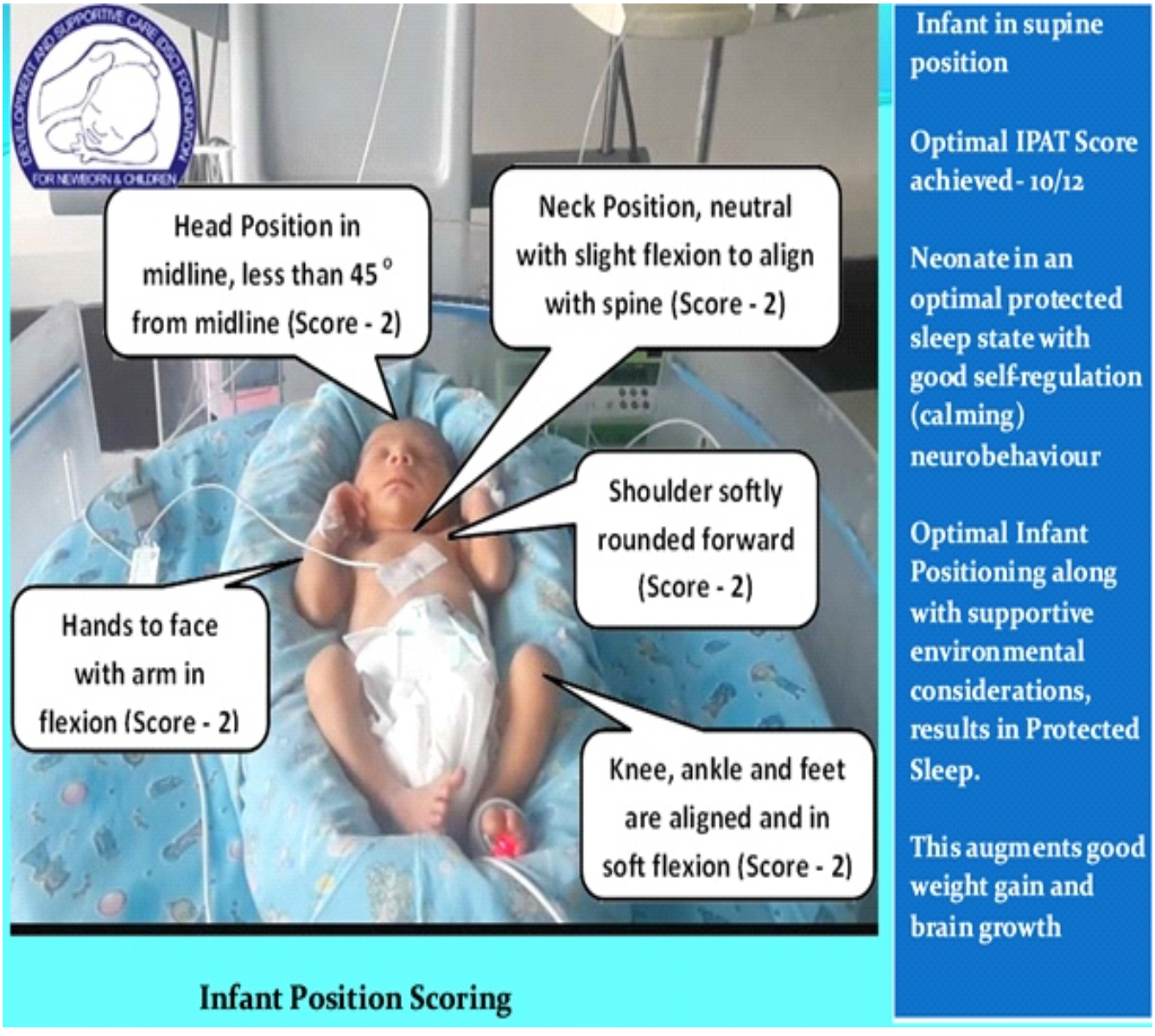

**Flexion position with Mid line orientation**

1) Provide nesting, to maintain Flexion position with Mid line orientation

2) Adopting Flexion posture is of prime importance in achieving good Self-Regulation (calming) and optimal Protected sleep

3) Optimal positioning & posture promote Deep sleep and thus protect the baby's developing nervous system/brain and development.

**Photo & captions courtesy:**
**With permission Dr Amitava Sengupta. DSC Foundation for Newborn and Children, India**




**Infant Position Scoring**

**Photo & captions courtesy:**
**With permission** Dr Amitava Sengupta. **DSC Foundation for Newborn and Children, India**

**INFANT POSITIONS**


**Photo courtesy:**
**With permission Dr Amitava Sengupta. DSC Foundation for Newborn and Children, India**


**Photo courtesy:**
**With permission Dr Amitava Sengupta. DSC Foundation for Newborn and Children, India**

The flexion posture is important for motor development, and also augments self-regulatory (calming), behaviours that support the baby's neuro-behavioural development during this critical time.

It is also recommended that these infants have experience in varied positions (prone, supine, side lying) with proper supports.

There are several circumstances that may impede your ability to provide the degree of optimal positioning (e.g., infants with venous/arterial access needs, drains, and ventilation procedures). Hence, it is very important to document the variance in your ability to provide optimal positioning for each infant.

**Salient considerations**

- Good positioning and support promote autonomic stability, give a sense of security, help maintain skin integrity and muscle tone. **Infants had the most stress when in side-lying unsupported**
- Nest size, shape and height can be according to the baby's size and need. No one size or shape fits all. Focus on what is needed for the desired position. For example, we provide a very shallow small nest for very young and tiny micro-preemies.
- Protocol has to be individualized as per the staff availability expertise of staff and workload of the unit


**Courtesy:**
**With permission** Dr Amitava Sengupta. **DSC Foundation for Newborn and Children, India**

**IMAGE-3 Infant on Mechanical Ventilation**

**Photo courtesy:**
**With permission Dr Amitava Sengupta**
**DSC Foundation for Newborn and Children, India**

**Position according to Medical Status**

1. Allow for alteration due to positional constraints for medical equipment
2. Caudal Nesting (½ nesting) Full nest not feasible as infant is on ventilation with accessory lines in-situ
3. Flexion Posture with Mid line orientation
4. Hands to face (Left) and bilateral Foot Bracing

**Suggested Reading**

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# Neonate with gastrointestinal mucormycosis

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4. Head of Department, Senior histopathologist/ Cytologist, PSRI Hospital, Delhi

**Clinical presentation**

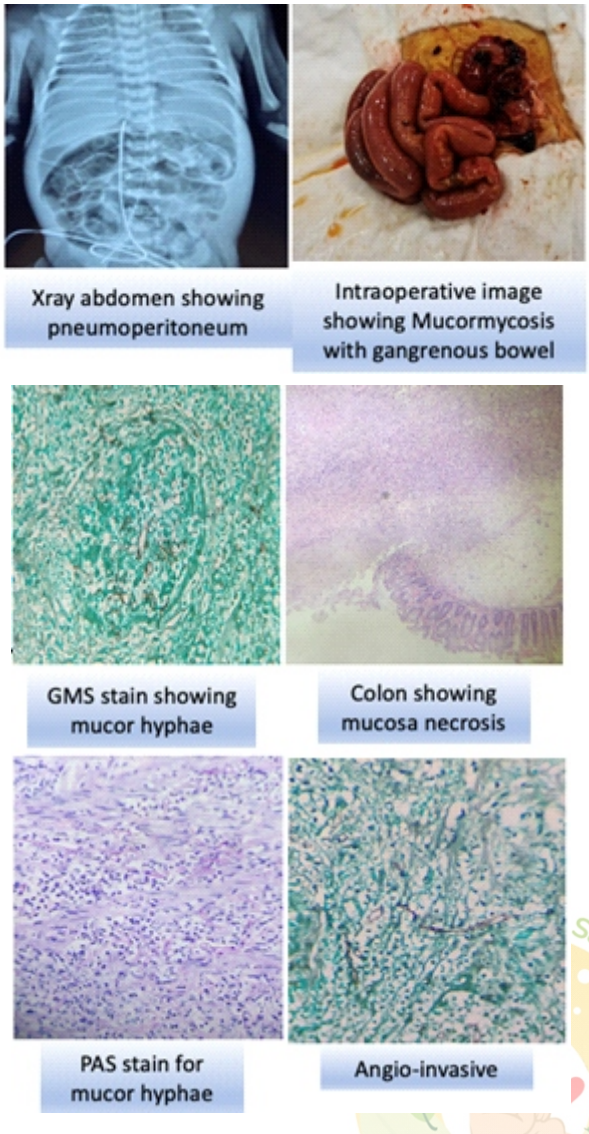
Late preterm (34 weeks) appropriate for gestation age male baby with birth weight 2.7 kg was admitted in our unit on day 3 of life in view of worsening respiratory distress. Baby was ventilated for the same, surfactant was given. Inotropes (Adrenaline, Milrinone) were started which were gradually tapered off in next 48 hours. Orogastric feeds were started on day 6 of life. Baby developed abdominal distension on day 8 of life.

Xray abdomen was suggestive of pneumoperitoneum. (Figure 1).

**Peroperative findings**

Exploratory laparotomy was done which was suggestive of multiple perforations in the left colon (Figure 2) for which the baby underwent left hemicolectomy with a colostomy. A biopsy sample was sent which was suggestive of mucormycosis (Figure 3) .

**Course** – Baby received intravenous amphotericin B for a total of 21 days. The baby was taken off ventilatory support by day 12 of life. Feeds were restarted by day 13 of life which were gradually increased and were tolerated well. Baby was discharged on day 26 of life with plan to complete 21 days of amphotericin B. On follow up, baby is gaining weight and colostomy is functioning fine.




**Condition:**

Mucormycosis is a fungal disease which rarely invades the gastrointestinal tract of new born, resulting in high

morbidity and mortality. Clinically, it is not distinguishable from the neonatal necrotizing enterocolitis and the diagnosis is usually made on autopsy or histopathology of the excised surgical specimen.

The first review of neonatal gastrointestinal Mucormycosis (GIM) was published in 1994. Of the 22 cases of neonatal GIM reported in English literature to date, 15 have been reported from India. GIM is the rarest form accounting for 7% of all cases. In neonatal GIM, the colon is the predominantly involved organ. Low birth weight (LBW), especially premature, neonates having immature immune system and fragile skin barriers, are at risk of Mucormycosis. Interventions such as oro-gastric tube placement, endotracheal intubation, and indomethacin therapy have been associated in increasing the risk of contracting GIM by causing mucosal injury. GIM should be considered in LBW babies with clinical NEC, who have prolonged neutropenia, negligible enteral feedings and treatment with multiple antibiotics.

**Management:**

1. Aggressive early surgery followed by intravenous amphotericin B after histological diagnosis is the mainstay of treatment.
2. Adequate surgical resection reduces the fungal load and the chances of perforation and long-term sequelae like stricture. IV amphotericin B has been the drug of choice for the treatment of GIM.

**Key points:**

1. GIM should be considered in neonates with a clinical picture of NEC, who have prolonged neutropenia, negligible enteral feedings and treatment with multiple antibiotics.
2. Aggressive early surgery followed by intravenous amphotericin B after histological diagnosis is the mainstay of treatment

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# Primary Arachnoid Cyst

**Pinaki Dutta[1], Naveen Parkash Gupta[2], Chanchal Singh[3], T J Antony[4], Rana Patir[5], Anil Batra[2]**

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2. Senior Consultant Neonatology, Madhukar Rainbow Children's Hospital, Delhi
3. Lead Consultant, Department of Fetal Medicine, Madhukar Rainbow Children's Hospital, Delhi
4. Director and Head, Department of Neonatology, Fortis Memorial Research Institute, Gurgaon
5. Director and Head, Department of Neurosurgery, Fortis Memorial Research Institute, Gurgaon.

**Clinical presentation** – Term appropriate for gestational age baby born to 30 years old primigravida mother. On Antenatal scan at 32 weeks a intracranial cyst was detected (Figure 1).

Baby was born at 37 weeks through LSCS with birth weight 3.157 kg, perinatal course was uneventful. Baby was kept with mother, started on feeds. USG brain done on day 1 of life(Figure 2) showed large avascular intracranial cyst(4.5*4.2*4 cm) with dilatation of both lateral ventricles( L>R). His neurological examination was normal.

Postnatally MRI brain was done on Day 2 of life which was suggestive of of uniloculated interhemispheric cyst projecting to left of midline (4.7*4.1*4.5 cm) with hypogenesis of corpus callosum with dilatation of lateral ventricles present( left>right) associated with areas of encephalomalacia and gliosis in parietal, temporal and occipital regions on left side and temporal and occipital regions on right side. Baby was discharged on day 3 of life.

**Diagnosis** – Primary Arachnoid Cyst

**Course** - Baby was readmitted on day 13 of life in view of persistent vomiting. Baby underwent interhemispheric cyst excision with cystoventriculostomy and left ventricular-peritoneal shunt insertion. Post operative course was uneventful, baby was discharged on 6th postoperative day. CT brain done on 2nd postoperative day (Figure 4) which showed reduction in cyst size with VP shunt in situ.

Baby is now 3 months old and doing well on follow up.

**Discussion** -

Arachnoid cysts are a rare central nervous system malformation, representing only 1% of all intracranial masses in newborns.(1) Primary (congenital) arachnoid cysts are benign accumulation of clear fluid between the dura and the brain substance and do not communicate with the subarachnoid space.(2) Secondary (acquired) arachnoid cysts result from hemorrhage, trauma, and infection and usually communicate with the subarachnoid space . It's pathogenesis is based on a discontinuity in the neural mesencephalic crest that occurred during formation of the meninges, with consequent discontinuity of the primordial membrane filled with the CSF. The common locations of arachnoid cysts are the surface of the brain at the level of main brain fissures, such as sylvian, rolandic and interhemispheric fissures, sella turcica, the anterior cranial fossa, and the middle cranial fossa.(3)

Arachnoid cysts may be associated with intracranial-associated anomalies like corpus callosum agenesis, absence of cavum septi pellucidum, cerebellar anomalies, Arnold–Chiari I malformation, cerebral sulcus disorders, and arteriovenous malformations.(4) The extracranial-associated anomalies are sacrococcygeal tumors, tetralogy of Fallot, and type 1 neurofibromatosis. The risk of chromosomal abnormalities is very low.

If the cyst obstructs the foramina of Monro or displaces the aqueduct, the mass effect of the cyst can alter the CSF dynamics, leading to ventriculomegaly.

**Management** – Depends upon the size of cyst and compression on adjacent structures. Surgical drainage includes shunting, open or endoscopic fenestrations,

and stereotaxic aspiration. Rate of surgery in sysmptomatic infants with isolated arachnoid cyst is around 35%.(5) Currently, endoscopic cystoventriculostomy and cystocisternostomy have become less invasive surgical alternatives.(6)

**Prognosis** – Prognosis depends on the presence or absence of other congenital malformations, parenchymal hemorrhages, the rate of the growth of the cyst, and progression of ventriculomegaly.

Fetal arachnoid cysts without associated structural anomalies or chromosomal abnormalities can have a favorable outcome. The prognosis depends more on the brain integrity rather than the cyst volume or location. (7)

**Key messages**

1) Arachnoid cysts are a rare central nervous system malformation, representing only 1% of all intracranial masses in newborns.
2) Fetal arachnoid cysts can be evaluated prenatally by ultrasound and/or MRI.
3) Early surgical or endoscopic intervention should be done in symptomatic cases.
4) The prognosis depends on the brain integrity rather than the cyst volume or location.
5) Currently, endoscopic cystoventriculostomy and cystocisternostomy have become treatment of choice.

**References**


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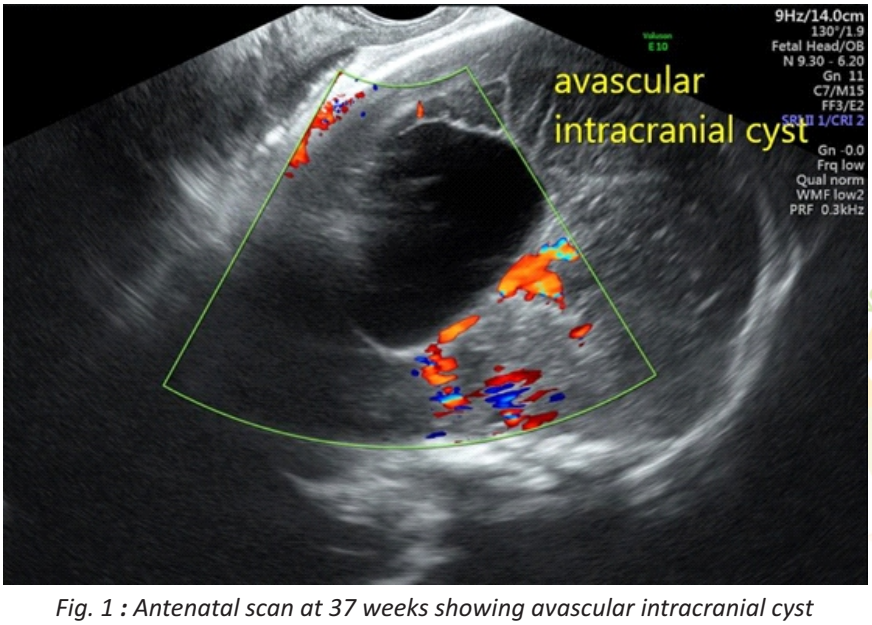


*Fig. 1 : Antenatal scan at 37 weeks showing avascular intracranial cyst*

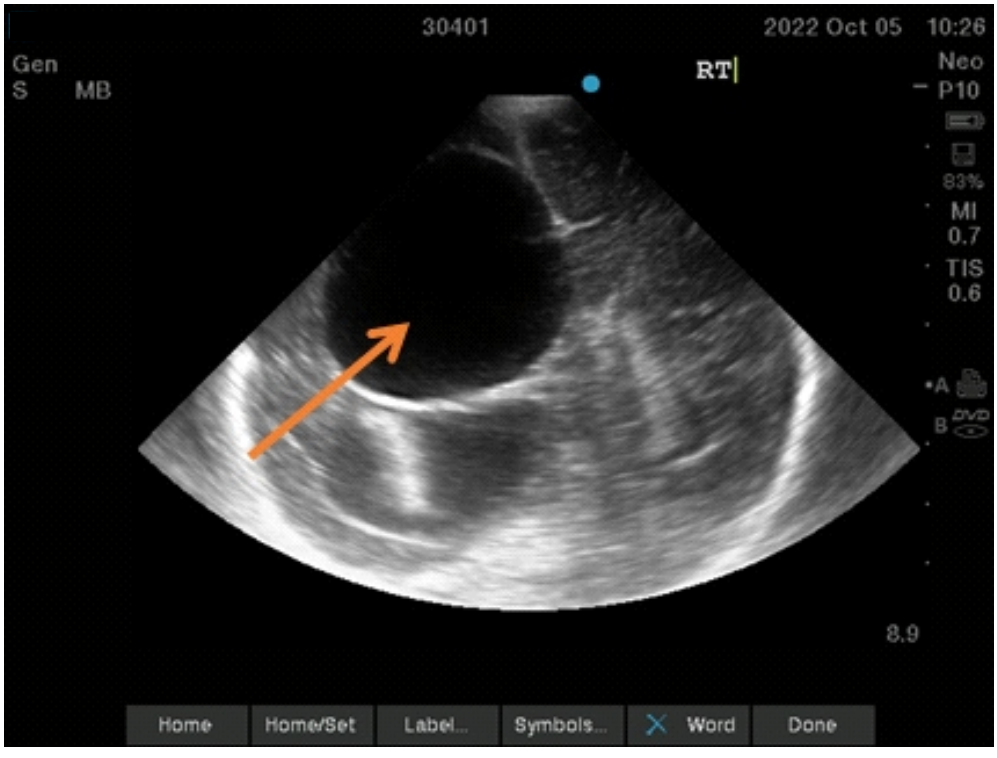


*Fig. 2 : Postnatal USG cranium showing large intracranial cyst with dilatation*

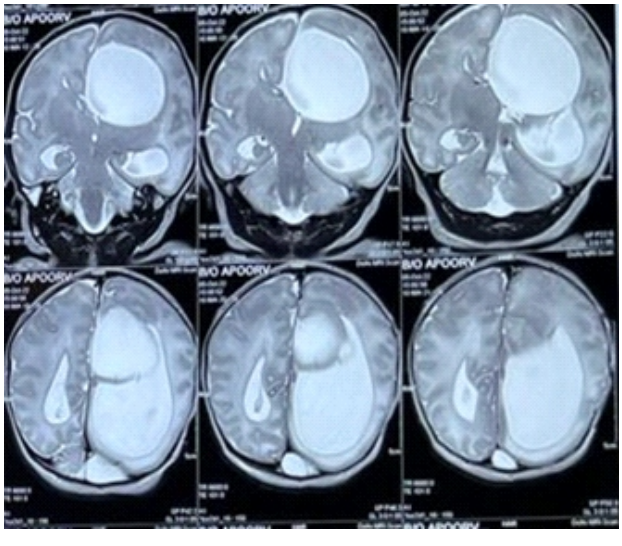

*Fig. 3 : MRI Brain showing uniloculated interhemispheric cyst projecting to left of midline with dilatation of lateral ventricles*

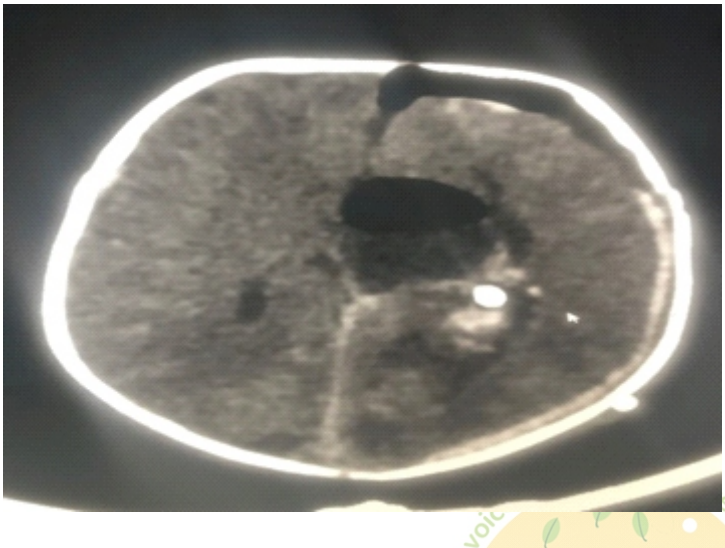

*Fig. 4 : Postoperative CT brain showing left ventriculoperitoneal shunt with decreased cyst size.*

# Journal Scan

Reviewed by
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OBSTETRICS

## Umbilical cord milking in nonvigorous infants: a cluster-randomized crossover trial

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**Am J Obstet Gynecol 2022 Aug 13;S0002-9378(22)00649-4.**

**Research question:**

Does Umbilical cord milking (UCM) reduce admission to NICU in non-vigorous neonates born between 35 and 42 weeks compared to Early cord clamping (ECC)?

**Hypothesis:**

Population : Non-vigorous neonates born between 35 and 42 weeks

Intervention : Umbilical cord milking

Control : Early cord clamping

Outcome : UCM will reduce NICU admission with in 24 hours of non-vigorous neonates born between 35 and 42 weeks in NICU from 25% to 16% compared to ECC.

**Methods**

- Design: Multicentre Randomized crossover trial at 10 sites ( 7 in US, 2 in Canada and 1 in Poland)
- Allocation /Randomization: Hospitals were randomized through computer-generated allocation sequence to UCM or ECC in period 1(5 hospitals per treatment group from Jan 19 to Jan 20) until 600 consented subjects and then after washout period of 1 to 2 months crossed over to other intervention during period 2 (Feb 2020 to May 21). The waiver of consent was taken to minimise selection bias.
- Blinding: Only outcome assessors could be blinded due to the nature of the intervention.
- Settings; Community and University hospitals with few centres having > 5000 deliveries per year.
- Patients: 1730 infants with primary outcome data and 1207 with consent.

**Inclusion criteria:**

Neonates delivered at 35 to 42 weeks gestation and non-vigorous at birth.

Non vigorous: presence of any of following in first 15 seconds as determined by obstetrician

a. Poor tone

b. Lack of breathing despite initial resuscitation efforts (stimulation, warmth /suctioning )

c. Pallor

**Exclusion criteria:**

a. Major congenital anomalies

b. Cardiac defects other than VSD

c. Placental abruption, monochorionic twins

d. Non reducible nuchal cord

**Primary outcome:**

Admission to the NICU in the first 24 hours of life for predefined criteria as follows: respiratory distress (tachypnea, grunting, or retractions), bradycardia or tachycardia, hypotonia, lethargy or difficulty arousing, hypertonia or irritability, poor feeding or emesis,

hypoglycemia, oxygen desaturations or cyanosis, need for oxygen, apnea, seizures or seizure-like activity, hyperbilirubinemia, and/or temperature instability.

**Sample size calculation:** A sample size of 1200 was estimated as necessary to test the efficacy of UCM vs ECC for the primary outcome on basis of following :

1. A clinically meaningful 35 % relative reduction in NICU admissions (16% for UCM vs 25% for ECC)

2. A 2 sided type 1 error alpha=0.05, 85% power, 0,02 rho,0.02 eta and a correction factor (4 x cluster size) for the small number of clusters.

**Intervention:** UCM during caesarean was provided by placing the newborn below the level of incision (at edge of table or on sterile blanket on mother's legs) with second member milking cord 4 times. 20 cm of cord was milked over 2 seconds. For vaginal delivery infant was placed on mother's abdomen. For ECC clamping was done within 60 seconds.

**Results :** The frequency of NICU admission between the UCM(23%) and ECC(28%) groups was different, however difference did not remain statistically significant after adjusting for study design by site. The most common cause for NICU admission was respiratory distress. UCM was associated with significantly lower odds of abnormal 1 minute APGAR score, receipt of delivery room cardiorespiratory support (61 % vs 71%) and therapeutic hypothermia (3% vs 4%). There was no difference in any grade HIE but moderate to severe HIE was less in UCM group (1% vs 3%). There were no cases of polycythemia, hyperbilirubinemia requiring exchange transfusion or pulmonary haemorrhage in any group. The UCM group had significantly higher haemoglobin (mean difference 0.68g/dl) and peak serum bilirubin (mean difference 1.4 mg/dl).

Significant interaction was observed for site with 22% of the difference in primary outcomes being due to the site of birth. Post hoc analyses also revealed a significant interaction between treatment group and delivery volume with large primary treatment effect in NICU admission by treatment arm . Hospitals with higher volume load ( > 5000 births per year) had lower NICU admission in UCM group (22.4% vs 36.7%, OR 0.42 (0.26-0.70) but NICU admissions were similar in both groups in hospitals with < 5000 deliveries per year.

**Reviewers comments:**

Nearly 6 million infants need resuscitation at birth annually. The currently recommended cord management strategy for such infants is early cord clamping. Umbilical cord milking is an attractive alternative to such infants as it can be performed quickly and may provide benefits of placenta transfusion to such infants. The current multicentre pragmatic cluster cross over trial compared the two strategies of DCC and UCM and did not find any difference in NICU admissions. UCM was associated with reduction in need of cardiorespiratory support, fewer moderate to severe HIE, reduction in use of Therapeutic hypothermia and higher hemoglobin levels.

**Strengths:**

This is the largest trial comparing two different cord management strategies in non vigorous neonates. The issue of selection bias in the resuscitation trial was mitigated by use of cluster randomised trial design and of antenatal consent. All obstetrical care providers were trained in both performing the intervention and identification of non vigorous infants. The primary outcome had specified criteria and all admissions were also reviewed to confirm it. The protocol was adhered in most neonates with nearly 95% of neonates in both arms treated as per protocol.

**Limitations:**

The intervention could not be blinded due to the nature of intervention per se. The policy of providing therapeutic hypothermia to mild encephalopathy was not uniform among the centres which might have contributed to significant difference in use of therapeutic hypothermia between the two groups, though the incidence of moderate to severe HIE which is a definite indication for provision of hypothermia was also different. There was significant difference in NICU admission across various sites with nearly ¼ of difference because of the hospital of birth . **SIte delivery load** was a significant effect modifier with lower delivery centres not observing any difference in

NICU admission but high delivery centres having significant difference. The cause of this variation cannot be determined from the above study and needs to be studied further in future. Pallor as a criteria for identification of non vigorous neonates could have had high interobserver varaiability though most of neonates also had other parameters to be labelled as non vigorous neonate.

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**Question1.**

A 30-year-old primigravida mother is admitted in LR with labour pains. On reviewing the antenatal records, you find the second trimester scan showed fetal renal pelvis dilatation of 6mm on right side and 4mm on left side. On repeat scanning in third trimester, anteroposterior diameter (APD) of renal pelvis was 9mm on right and 8mm on left side, with normal fetal bladder and amniotic fluid level. Baby is delivered at 38weeks gestation with birth weight of 2.8kg..

(i) How do you define and classify fetal hydronephrosis? What is the most common etiology for antenatal hydronephrosis (ANH)?

(ii) In the above case what is the grade of hydronehprosis by APD (Antero-Posterior Diameter of renal pelvis) classification system?

(iii) How will you evaluate this baby prenatally? What are the treatment options in antenatal period?

(iv) What is SFU classification system? How do you classify postnatal hydronephrosis?

(v) What will be the indications for micturating cystourethrogram (MCUG) and diuretic renography (DR) scan?

(vi) On post-natal evaluation, there was persistent right sided hydronephrosis with APD 12 mm at 7 days of life and right sided APD 15 mm at 4 weeks. MCU at 4 weeks did not show evidence of lower urinary tract obstruction or VUR. DTPA scan was done at 8 weeks and image was as shown in Fig. 1. What is the interpretation and what is the likely diagnosis?

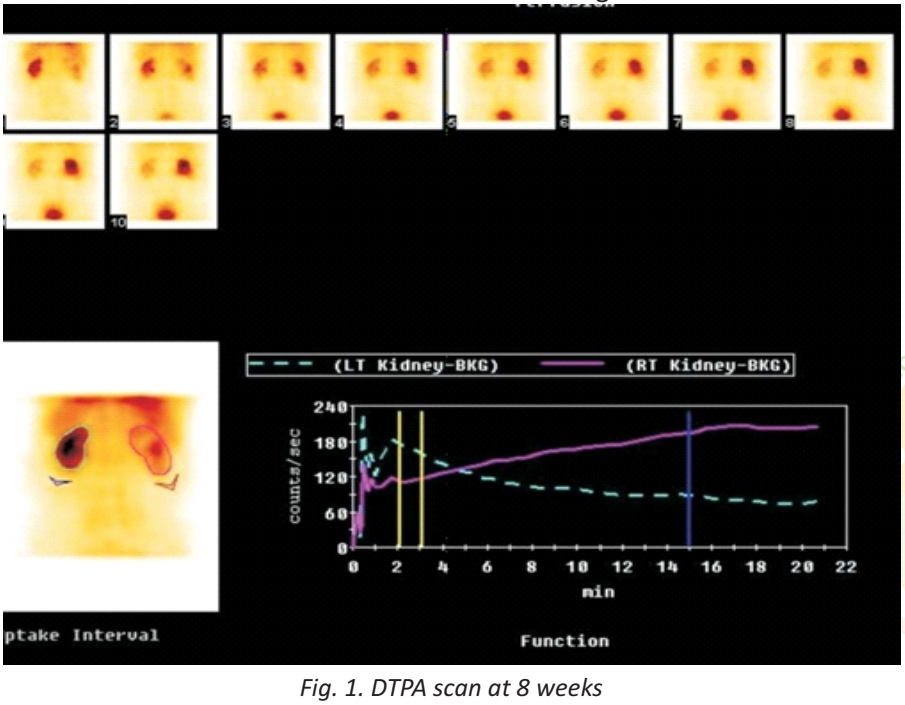


*Fig. 1. DTPA scan at 8 weeks*

(vii) What are the indications for starting antibiotic prophylaxis in hydronephrosis?

**Question 2.**

A 1-day old male neonate is referred to your hospital with complaints of dribbling micturition. Prenatal ultrasound had showed oligohydramnios with distended bladder. On examination, you find palpable bladder and palpable mass in bilateral flank.

(i) What is the immediate medical intervention you will do at admission?

(ii) Post-natal ultrasound showed gross bilateral hydroureteronephrosis with thickened bladder wall. What next investigation/ imaging would you consider for evaluation and confirmation of the diagnosis? Imaging is done as shown in Fig 2. What are the findings and what is the diagnosis?

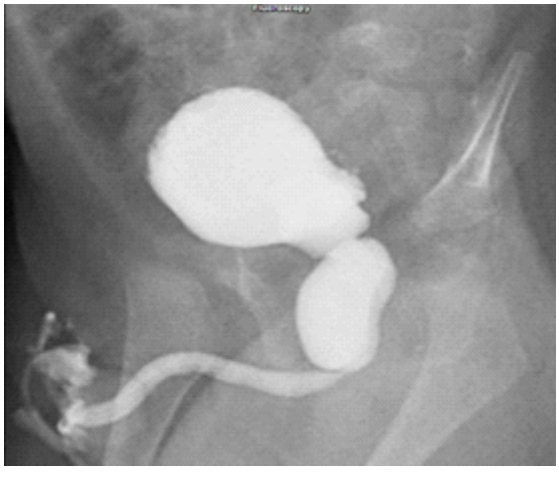

*Fig. 2.*

(iii) What is the most common clinical presentation in the above condition?

(iv) What is the definite treatment for the above condition?

(v) What is the most common residual renal complication post treatment?

**Question 3.**

A 20week scan in a primigravida mother shows multiple large bright cysts in left fetal kidney with loss of normal renal architecture.

(i) What is the likely diagnosis?

(ii) What are the common associated anomalies in the contralateral kidney?

(iii) What are the outcomes in neonates with bilateral form of the above disease?

(iv) What serial monitoring is required on follow up in this case?

**Question 4.**

A 6weeks old infant, former 28weeker preterm is admitted in your NICU with BPD on supplemental oxygen. You have noticed that in the last one week the blood pressure readings have been mostly above 100/65 mm Hg with none less than 85/65mmHg.

(i) How do you define neonatal hypertension? What charts will you use to define hypertension in this infant?

(ii) What are the common causes of neonatal hypertension?

(iii) What is the gold standard technique of blood pressure monitoring in neonates? Which investigations and interventions will you plan in the above case and why?

(iv) When do you consider anti-hypertensive medications in a case of neonatal hypertension?

(v) What is the prognosis of the above case?

**Question 5.**

2 weeks old late preterm, formula fed neonate is admitted in your NICU with complaints of poor feeding and anuria for last 2 days. Baby is lethargic, hypothermic and has generalized petechiae on examination. On evaluation, baby had Na 134 meq/l, K 5.8 meq/l, Creatinine 3.8 mg/dl, Urea 80 mg/dl, along with positive infection work up.

(i) What is the KDIGO classification of neonatal Acute kidney injury (AKI)?

(ii) What is the likely cause of AKI in the above case?

(iii) Name 2 newer biomarkers of kidney injury.

(iv) When will you consider fluid challenge in a case of neonatal AKI?

(v) Which drug has been shown to prevent neonatal AKI?

**Question 6.**

37 week late preterm neonate is admitted in your hospital at 3weeks of life with encephalopathy, anuria for 2days (stage 3 AKI) and hemodynamic instability. On examination baby has signs of fluid overload.

(i) What are the indications of Renal Replacement Therapy (RRT) in neonatal AKI?

(ii) What are the modalities available for RRT? Which is the preferred mode in neonates?

(iii) At what blood ammonia levels is initiation of dialysis recommended in neonatal hyperammonemia?

(iv) What is the preferred modality of RRT in neonatal hyperammonemia?

**Question 7.**

A 4-day old neonate weighing 2.5kg, born at 36weeks with birth weight 2.1kg is now admitted to your hospital with complaints of generalised edema and oliguria. Antenatal ultrasound showed bilateral enlarged hyperechoic fetal kidneys with fetal ascites and placentomegaly.

(i) How would you like to evaluate this baby?

(ii) On evaluation, baby has hypoalbuminemia and urinalysis showed significantly elevated protein (4+) and hyperlipidemia. What will you suspect?

(iii) What is the classical postnatal presentation in the above condition?

(iv) What additional investigations would you consider to confirm diagnosis?

(v) How will you manage this condition? What is the definite treatment in congenital forms of the disease?

**Question 8.**

3.4kg male neonate, infant of diabetic mother is admitted in your NICU with respiratory distress since birth. On day 3, baby develops hematuria with a palpable flank mass

(i) What clinical diagnosis will you suspect in the above case?

(ii) What are the risk factors which predispose to it?

(iii) What is the investigation of choice to confirm diagnosis in a suspected case?

(iv) What are the treatment options available?

**Question 9.**

20 days old very preterm stable neonate on full orogastric tube feeds admitted in your NICU, develops new onset apnea with feed intolerance. Sepsis evaluation is done along with urinalysis. Urinalysis of catheterized urine sample shows numerous pus cells while culture report is awaited

(i) How do you define urinary tract infection (UTI)?

(ii) How do you define pyuria on urine routine test and what is the predictive ability of urinalysis to diagnose UTI?

(iii) When is renal ultrasound indicated in a case of UTI in neonate?

(iv) What are the indications for doing VCUG in a follow up case?

(v) Renal ultrasound in this baby was suggestive of bilateral hydronephrosis. MCU was done after few weeks and is as shown in Fig. 3. Interpret the image and what is the diagnosis? What are the indications for surgical intervention in VUR?

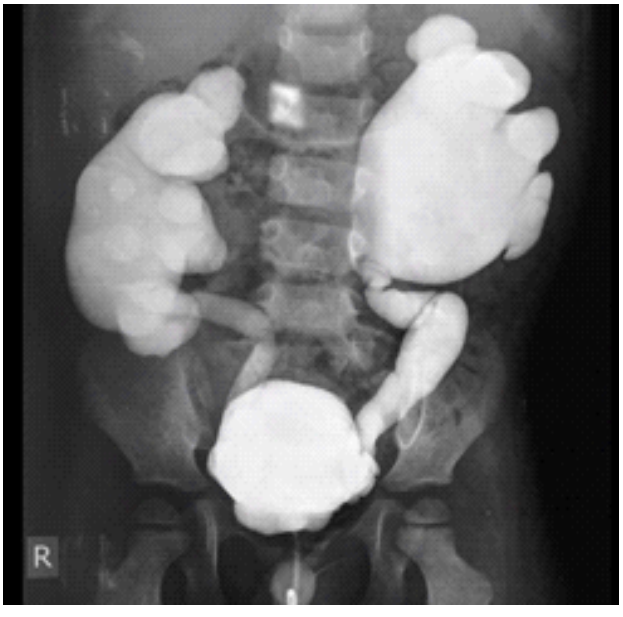


*Fig. 3 MCU*

**Question 10.**

Classify as true/false the following statements related to common renal physiology

(i) Filtration and first urine formation begins at 20weeks in fetal kidneys T/F

(ii) Fetal kidney receives only 2% of cardiac output as renal blood flow as opposed to adult values of 20-25% cardiac output T/F

(iii) GFR of a normal term neonate is same as that of an adult- T/F

(iv) VUR is a normal finding in a normal neonate at birth. T/F

**Question 11.**

Depicted below is thematic image of different

patterns of renogram curves. The curve shown in green is normal renogram curve.

(i) What will be the findings in non-obstructed drainage system? Which curve corresponds to non-obstructed drainage?

(ii) What will be the findings in obstructive hydronephrosis? Which curve corresponds to obstructed drainage?

(iii) Identify the curves shown in blue, orange and black color.

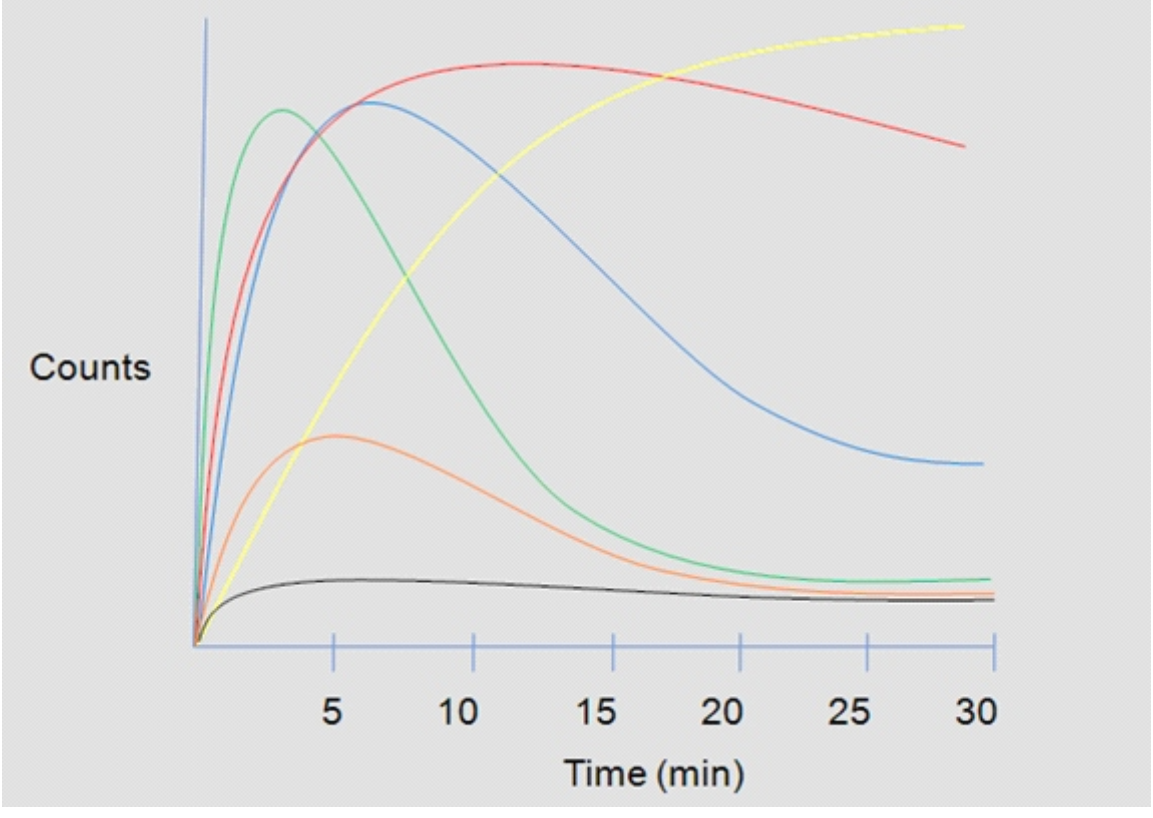


*Fig. 4. Renogram curves*


**Answer 1.**

(i) Fetal hydronephrosis is defined as dilatation of renal pelvis with or without dilatation of renal calyces with APD ≥ 4mm in 2nd trimester or ≥ 7mm in third trimester. It is further classified as:

| | 2nd Trimester APD | 3rd Trimester APD |
|---|---|---|
| Mild | ≥4mm | ≥7mm |
| Moderate | 7-10mm | 10-15mm |
| Severe | >10mm | >15mm |

The above case has bilateral moderate hydronephrosis with no signs of bladder outlet obstruction (normal bladder, normal amniotic fluid indices). The most common etiology is transient hydronephrosis, seen in 41-88% cases.

Other common etiologies include:

- Obstructive causes [Ureteropelvic junction obstruction (UPJ), vesicoureteric junction obstruction (VUJ), ureterocoele, posterior urethral valves (PUV)]
- Vesicouretric reflux (VUR)
- Congenital anomalies of kidney urinary tract-CAKUT (eg. Multicystic dysplastic kidney)

(ii) Moderate

(iii) Prenatal evaluation with follow up ultrasound scan is done in all cases of hydronephrosia (HDN) with the aim to look for disease progression/ resolution, involvement of renal parenchyma, bladder dilation or wall thickness, amniotic fluid volume and other systemic malformations.

Unilateral disease- atleast one follow up scan in third trimester

Bilateral disease- follow up scan every 4-6weeks

Vesicocentesis is indicated in selected cases with severe bilateral disease and signs of LUTO-lower urinary tract obstruction (oligohydramnios, thickened dilated bladder, keyhole urethra)

Karyotype/chromosomal studies are considered only if associated with multiple renal/systemic malformations.

Prenatal intervention: Vesicoamniotic shunt or in-utero endoscopic valve ablation

Indication: Bilateral severe disease with LUTO diagnosed early in 2nd trimester and favourable indices on vesicocentesis.

Evidence: PLUTO (Percutaneous shunting for Lower Urinary Tract Obstruction) trial, a multi-centre RCT (n=31) has shown improved perinatal survival with fetal intervention, however with no long-term benefit in renal outcomes.

(iv) Society of Fetal Urology classification system

| Grade | Central renal complex | Renal parenchyma |
|---|---|---|
| 0 | Intact | Normal |
| 1 | Slight splitting of renal pelvis | Normal |
| 2 | Evident splitting of pelvis and major calyces | Normal |
| 3 | Wide splitting of major and minor calyces | Normal |
| 4 | Further splitting of calyces | Reduced |

Neonatal hydronephrosis is defined as APD ≥ 7mm or SFU grade ≥ 1 on a postnatal ultrasound. It is further classified as mild (7-9mm), moderate (10-15mm) and severe (>15mm) based on APD of renal pelvis.

(v) Postnatal evaluation:

| Investigation | Indication | Optimal timing |
|---|---|---|
| Postnatal USG | In all cases of antenatally diagnosed hydronephrosis | If suspected LUTO/PUV, severe B/L hydronephrosis, severe U/L in solitary kidney: Early (<48hrs)<br>Others: between 3-7days of postnatal age |
| MCUG and VUR grading | -Suspected LUTO/PUV<br>-U/L or B/L moderate to severe hydronephrosis (APD>10mm or SFU grade 3-4)<br>-Mild hydronephrosis with ureteric/bladder/urethral dilation or abnormality | Suspected LUTO: 24-72hrs<br>Others: 4-6weeks |
| Diuretic renogram (DR) (Tc99 MAG3/EC/DTPA) to define differential renal function if obstructive kidney | -U/L or B/L moderate to severe hydronephrosis (APD>10mm or SFU grade 3-4) with no VUR (when MCUG normal) | Only when no VUR on MCUG: DR at scan 6-8week |

Postnatal management:

1. No postnatal hydronephrosis; follow up USG at 4-6weeks if normal no further evaluation
2. U/L or B/L hydronephrosis with LUTO unlikely- based on severity evaluate
   If APD<10/SFU grade 1-2: follow up USG at 4-6weeks, repeat every 6-12month until resolution
   If APD>10mm/SFU grade 3-4: do MCUG at 4-6weeks and if VUR present start antibiotic prophylaxis in all cases. If no VUR, then DR scanning at 6-8weeks.
3. If LUTO features present: Immediate catheterization.
   MCUG within 48hrs followed by early surgery in obstructive cases (eg: PUV- valve ablation)

(vi) Right kidney: Prominent concentration of radionuclide on right side, Hydronephrosis with mildly impaired function and features of obstructive pattern (significant retention of tracer and no response to diuretic).
Left kidney: Normal function with non-obstructive upper outflow tract.
Diagnosis: Right sided PUJ obstruction.

(vii) Indications for antibiotic prophylaxis: Infants with postnatally confirmed moderate or severe hydronephrosis (SFU 3-4; renal APD >10 mm) or dilated ureter, while awaiting evaluation; and all patients detected to have VUR to receive antibiotic prophylaxis through the first year of life.

**Answer 2.**

(i) Immediate intervention will include clinical stabilization, correction of dyselectrolytemia, starting antibiotic prophylaxis and temporary drainage of bladder with catheterization.

(ii) The above clinical scenario is suggestive of a case of lower urinary tract obstruction. The investigation to confirm clinical diagnosis is Voiding cystouretherogram (VCUG). Fig 2. Shows VCUG/ MCU reveals marked dilatation of the prostatic portion of the urethra consistent with posterior urethral valve.

(iii) Most common clinical presentation in a case of posterior urethral valve is urinary tract infection (UTI).

(iv) Definite treatment is surgery. Surgical options include- cystoscopic ablation or fulgration of

valves, vesicostomy or upper tract/ureteral diversions.

(v) Persistent bladder dysfunction is the common residual complication seen even after surgical valve ablation.

**Answer 3.**

(i) The most likely diagnosis is multicystic dysplastic kidney (MCDK). All other forms of renal cystic disease develop cysts much later in third trimester.

(ii) VUR (20%) is the most common finding in the contralateral kidney. 5% cases have associated UPJ or UVJ obstruction in contralateral kidney.

(iii) Bilateral forms of disease clinically present as potter sequence. Bilateral MCDK is fatal in immediate neonatal period.

(iv) U/L MCDK is associated with hypertrophy of contralateral kidney over time which leads to increased risk for hypertension, proteinuria and chronic kidney disease. U/L MCDK may sometimes involute over time. Serial monitoring with follow up scan to look for hypertrophy/involution is recommended. Serial renal function tests and BP monitoring should also be done on follow up in all cases which develop contralateral hypertrophy.

**Answer 4.**

(i) Neonatal hypertension is defined as sustained systolic and/ or diastolic blood pressure above 2 standard deviation or >95$^{th}$ percentile for post menstural age on three separate occasions, in infants of similar height, sex and gestational age.

The blood pressure charts commonly used in NICU are Zubrow's for first two weeks of life and Dionnes chart after 2 weeks of postnatal life.

(ii) Common causes of neonatal hypertension include

1. Renal causes:

   -Renovascular hypertension- renal artery thrombosis/stenosis, renal vein thrombosis

   -Obstructive renopathy- hydronephrosis, UPJ obstruction

   -Renal parenchymal causes- renal dysplasia, polycystic kidney disease, acute tubular necrosis
2. Cardiovascular causes- coarctation of aorta, patent ductus arteriosus
3. Endocrine and metabolic causes- Congenital adrenal hyperplasia, hypercalcemia, hyperthyroidism, hyperaldosteronism
4. Others: prematurity, BPD,
5. Itrogenic causes: ECMO, inadequately controlled pain and exposure of medications- corticosteroids, bronchodilators, caffeine/theophylline, vasopressors, indomethacin

(iii) Invasive arterial blood pressure monitoring is the gold standard technique of measuring BP in neonates.

Initial laboratory tests will include urinalysis, renal function tests, serum electrolytes and hormonal assays including Thyroid function test (TFT), aldosterone, cortisol, renin and angiotensin.

Imaging like ultrasound KUB with doppler and echocardiography.

Besides in the above case of a preterm with BPD, it is also important to consider any reversible or treatable cause like exposure to offending medications, pain or volume overload.

(iv) Antihypertensives are considered in cases with

1. Hypertensive emergency i.e. hypertension with cardiopulmonary failure or acute neurologic dysfunction or renal failure.
2. BP persistently >99 percentile +5mmHg despite all supportive measures

   Goal of antihypertensive therapy is to decrease BP and minimize injury to vital organs. Long standing hypertension can lead to left ventricular hypertrophy, retinopathy and encephalopathy.

(v) Hypertension secondary to BPD has a good prognosis and require only short-term treatment. In most cases BP normalizes by 12-18months of age.

**Answer 5.**

(i) KDIGO classification of Neonatal AKI

| Stage | Change in Serum (Sr.) Creatinine | Urine output |
|---|---|---|
| 0 | No change or <0.3mg/dl rise within 48hrs | ≥0.5ml/kg/hr |
| 1 | Rise of Sr Cr >0.3mg/dl in 48hrs or 150-190% of reference Cr within 7days | <0.5ml/kg/hr for 6-12hrs |
| 2 | Rise in Sr Cr 200-300% of reference Cr | <0.5ml/kg/hr for ≥12hrs |
| 3 | Rise in Sr Cr >300% od reference Cr or absolute value >2.5mg/dl or receipt of dialysis | <0.3ml/kg/hr for ≥ 24hrs or Anuria for ≥12hrs |

(ii) Most likely cause is late onset neonatal sepsis in the above case, which is also the most common cause of AKI in neonatal population.

(iii) NGAL (neutrophil gelatinase associated lipocalcin), Cystatin C, KIM-1 (Kidney Injury Molecule) and IL-18 are a few novel markers that can aid in early diagnosis of AKI

(iv) Fluid challenge in considered in all cases of prerenal AKI with signs of hypovolemia. Detailed history and thorough clinical examination should be done prior to fluid challenge to rule out CAKUT and systemic or pulmonary fluid overload (edema, weight gain, hypertension, tachypnea) and bladder outlet obstruction (palpable bladder, abnormal scans).

(v) Early caffeine therapy (started <7days of life in at risk preterm) has shown to decrease AKI incidence from 36% to 11% with NNT 4.3 (AWAKEN study). Caffeine has also shown to decrease progression to stage2/3 AKI.

**Answer 6.**

(i) The two broad categories for indication of RRT in neonates include:

-RRT in AKI: fluid overload >10%, persistent hyperkalemia, severe hyperphasphatemia or hypocalcemia, severe metabolic acidosis not responding to medical treatment, uremic encephalopathy

-Non renal indications: removal of toxins (ingested/IEM), prevention or treatment of tumour lysis syndrome

(ii) 3 modalities available for RRT in neonates includes

Peritoneal dialysis- most preferred mode

Hemodialysis- most efficient mode

Continuous renal replacement therapy (CRRT)- in severe hemodynamic instability and other modalities not feasible, in conjugation with ECMO

(iii) Neonatal hyperammonemia represents a medical emergency that requires prompt intervention to prevent brain damage. Though the mainstay of therapy involves decreasing ammonia production, but at blood ammonia >560µg/dl (>400µmol/L) immediate initiate of dialysis is recommended.

(iv) Hemodialysis (HD) and CRRT are both effective with their own advantages over one another. HD being relatively faster while CRRT allowing better hemodynamic stability and decreased risk of dyselectrolytemia. However, PD is clearly ineffective due to inability to clear ammonia rapidly.

**Answer 7.**

(i) Renal function test, Liver function test, Urinalysis, CBC, CRP, Blood Culture, Viral markers, TORCH, titer, Lipid profile, Ultrasound KUB

(ii) Congenital nephrotic syndrome (CNS), it is defined as nephrotic syndrome that presents at birth or within first 3 months of life.

(iii) Clinical features suggestive of CNS includes massive proteinuria, profound hypoalbuminemia, hyperlipidemia and generalized edema.

Primary or genetic forms of CNS may be

associated with other features like facial dysmorphism, ocular abnormalities, prematurity or small for gestational age. Secondary or acquired forms of the disease may additionally show features of TORCH stigmata.

(iv) Primary/genetic CNS- genetic mutational analysis

Secondary/acquired CNS- TORCH serology, HIV testing

Other supportive investigations- Renal function tests, urinalysis, immune profile, renal ultrasound and renal biopsy.

(v) Treatment includes initial stabilization with strict input output monitoring, fluid restriction, management of dyselectrolytemia, IV albumin infusions +/- glucocorticoids and immunosuppression.

Primary CNS shows poor response to treatment and may progress to end stage renal disease eventually requiring renal transplantation. Such cases are managed with renal dialysis until transplantation.

**Answer 8.**

(i) Renal vein thrombosis (RVT). The cardinal features include palpable flank mass, hematuria, thrombocytopenia with or without hypertension.

(ii) The common risk factors for RVT includes prematurity/SGA, Infant of diabetic mother (IDM), congenital heart disease, polycythemia, dehydration, asphyxia, sepsis, prolonged central venous catheter in situ and inherited thrombophilias

(iii) Colour doppler renal ultrasound is the investigation of choice. It shows absent or severely reduced intrarenal and renal venous flow during the initial stages of severe RVT. These findings become less characteristic as collaterals open in advanced stages of the disease

(iv) Management of case of RVT depends on presence of unilateral/bilateral disease, normal or deranged renal function tests and RVT with or without extension of thrombus to inferior vena cava.

Treatment options include- conservative management, systemic anticoagulation alone or anticoagulation with concomitant thrombolytic therapy.

**Answer 9.**

(i) Urine culture is gold standard technique to diagnose UTI.

- On a catheterised sample- UTI is generally defined as growth of a single uropathogenic pathogen with a colony count of ≥ 50,000 CFU/mL, or a colony count ≥ 10,000 CFU/mL with associated pyuria detected on urinalysis (or positive dipstick)
- On suprapubic aspiration- Any growth of a urinary pathogen is significant. The growth of one colony is equivalent to 1000 CFU/mL
- Bag sample is generally not recommended as the risk of contamination is very high

(ii) Pyuria is defined as presence of at least 5 WBCs per HPF in a centrifuged sample and >10 WBC/HPF in uncentrifuged sample. A WBC count of ≥ 10/mm$^3$ has a sensitivity of 91% and a specificity of 96% for predicting a positive culture of ≥50,000 CFU/mL

(iii) Renal ultrasound should is done in all cases of UTI in neonates at the time of diagnosis as CAKUT (congenital anomalies of kidney and urinary tract) is the most common risk factor for UTI in neonates.

(iv) VCUG- no routine use, however VCUG should be performed in high-risk neonates such as those with UTIs caused by bacteria other than E. coli, those with renal bladder ultrasound abnormalities, and uncircumcised males.

(v) Micturating cystourethrogram findings:

Bilateral ureters appear dilated and tortuous and bilateral pelvicalyceal systems appear grossly dilated with loss of papillary impressions, consistent with Grade V VUR.

Indications for surgery in VUR: Breakthrough UTI in Grade 3-5 VUR despite of antibiotic prophylaxis.

**Answer 10.**

(i) False, fetal kidneys start producing urine from 10-12weeks of intrauterine life.

(ii) True

(iii) False, GFR of term neonate is 21ml/min/1.73-meter square at birth which reaches adult values by 2years of age. It is further less in preterm neonates.

(iv) False, VUR is always pathological. It is secondary to aberrant insertion of ureter into bladder wall. 30% cases have first degree relative with VUR (autosomal dominant inheritance)

**Answer 11.**

(i) Non obstructive pattern: Rapid and almost complete washout of the radiotracer occurs before injection of diuretic. There may be slow emptying of the kidneys, if function is decreased. A T1/2 less than 10 min usually means the absence of obstruction. Green curve corresponds to non-obstructive pattern.

(ii) Obstructive drainage system: An obstructed system will not respond to the diuretic challenge. The curve rises continuously over 20 minutes or appears as a plateau, despite furosemide and post micturition. T1/2 greater than 20 minutes usually points towards obstruction. Red and yellow curves are suggestive of obstructive drainage.

(iii) Blue: Partial obstruction.

Orange: Renal failure with measurable renal function

Black: Renal failure with no significant renal function.

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# *Instructions for Authors*

| | |
|---|---|
| **Review Article** | **The article should be approximately 2-3 pages long with a word count of 2000-2500 words. Author should summarize key practice points at the end. Please include 5-6 references as suggested reading.** |
| **Case Report** | **This would be a summary of the case discussed in that months clinical meeting. Interesting cases even if not presented may also be submitted. It should include the clinical presentation and a brief discussion about the condition. Word count should be 1000-1500. Please include 2-3 references at the end.** |
| **Journal Scan** | **Some recent research paper of interest to pediatricians and neonatologists. The structure should include Introduction, Research question, Hypothesis, Methods, Results, Limitations and strengths of study, Reviewers comments. Word count should be approximately 1000 words. Please include 2-3 references if needed at the end.** |
| **Picture of Month** | **An interesting case related to neonatal practice. It should have a brief case history and a commentary, all fitting on one page along with the pictures.** |
| **Image section** | **Any interesting Xray, Ultrasound, CT or MRI of clinical interest. Brief clinical presentation and about the condition should be summarized on one page along with image.** |
| **OSCE** | **About 10-12 questions would be included in this section along with answers.** |

**Contact Us**

On behalf of committee, I request all members of NNF, Delhi to actively contribute to various sections of the newsletter.

You can contact us at following email addresses:

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**Please call or Whats App at 9811758133**