Neo Clips

NATIONAL NEONATOLOGY FORUM DELHI

MONTHLY E-BULLETIN

Vol.14 | March 2023

DR MAMTA JAJOO
President, NNF Delhi

DR NAVEEN PARKASH GUPTA
Secretary, NNF Delhi

DR NAVEEN PARKASH GUPTA
Chief Editor, Neo Clips

www.nnfdelhi.org

CONTENTS

NNF Delhi Office Bearers

Dr Mamta Jajoo
President

Dr Pradeep Debata
Past President

Dr Poonam Sidana
Vice President

Dr Naveen Parkash Gupta
Secretary

Dr Anup Thakur
Joint Secretary

Dr Dinesh Goel
Treasurer

Executive Members

Dr Vivek Choudhury

Dr Jai Kishore

Dr Vinay Kumar Rai

Dr Tapas Bandyopadhyay

Dr Shekhar Biswas

Jubilant James

Central NNF Office Bearers

Dr Sushma Nangia
President (2024)

Dr V C Manoj
President Elect (2024)

Dr Praveen Kumar
Immediate Past president

Dr Dinesh Chirla
Vice President (2024)

Dr Surender Singh Bisht
Secretary General 2022-24

Dr Amit Upadhyay
Treasurer (2022-24)

Dr Srinivas Murki
Joint Secretary (2022-24)

Dr Dinesh Tomar
Immediate Past Secretary
General-Ex-officio

NeoClips Committee Members

Dr T J Antony
Chairperson

Dr Naveen Parkash Gupta
Chief Editor

Dr Avneet Kaur
Co-Chairperson

Dr Swati Upadhyay
Member

Dr Sidharth Nayyar
Member

From President Pen

DR. MAMTA JAJOO

President NNF Delhi

Dear NNF Delhi Members

It's a feeling of pride that we are coming with the 14th issue of NeoClips. On this occasion, I am feeling privileged to congratulate our Editorial Board Members, chaired by Dr T J Antony and our Editor in chief Dr Naveen Parkash Gupta, who are working tirelessly, to make each edition of NeoClips 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 add 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 neonates. Many national and international programs are being launched to decrease neonatal mortality and we are approaching our set target by reducing the deaths among extremely preterm and asphyxiated neonates. Is this enough? The mere survival of neonates is not the solution. I feel we have to rethink our current strategy not only to decrease neonatal mortality but also to ensure the intact survival of our children.

With Regards

Dr. Mamta Jajoo
President, NNF Delhi

From Secretary and Editors desk

DR NAVEEN PARKASH GUPTA

Secretary, NNF Delhi

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 the current (14th issue) and previous issues.

We have covered some interesting topics in the present issue.

The case report covers an interesting case of neonatal thrombocytopenia and hyperbilirubinemia.

Congenital Diaphragmatic hernia is an important condition in neonates. Dr Shandip Kumar Sinha has covered it comprehensively in the review section

An interesting case of the Omphalocele has been covered in the picture of the month.

The image section describes an interesting X-ray of the Eventration of Diaphragm.

OSCE is covering a few interesting questions on mixed topics.

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

This is the last issue from the existing NeoClips team. I want to thank all the authors and NeoClips team members from the bottom of my heart for working hard in bringing out these 14 issues.

Next time, we have decided to publish NeoClips as a quarterly journal with the addition of a few areas. My best wishes to the new team for all the subsequent editions. Lets hope that NeoClips get indexed soon.

Thanks and Regards

Dr Naveen Parkash Gupta
Secretary, NNF Delhi

An Interesting Case of Neonatal Thrombocytopenia, Conjugated Hyperbilirubinemia and Skin Lesions

Mithun Mohan[1], Vidya Gupta[2], Saroja Balan[3]

1. NNF Fellow, Indraprastha Apollo Hospital, New Delhi
2. Senior Consultant, Neonatology, Indraprastha Apollo Hospital, New Delhi

INTRODUCTION:

Neonatal lupus is a rare and complex autoimmune condition that affects newborns, presenting unique challenges in both diagnosis and management. Neonatal lupus is a consequence of maternal autoimmune antibodies, primarily anti-SSA/Ro and anti-SSB/La, crossing the placenta during pregnancy, leading to a range of clinical manifestations in the newborn –(1). The most common presentation is a nonscarring, non-atrophic skin lesion which resemble subacute cutaneous lupus erythematosus. The infants may have no skin lesions at birth but develop them during the first weeks of life. Cardiac, haematological, hepatobiliary, central nervous, and pulmonary systems may also be involved. Despite its infrequent occurrence, the condition necessitates early recognition and multidisciplinary care to ensure optimal outcomes of affected infants. This case report highlights the intricacies of diagnosing neonatal lupus. Babies may present with unusual findings like calcifications which maybe present in multiple areas or very early onset jaundice with acute liver failure. This is in addition to the usual presentation of a rash with thrombocytopenia.

Case:

A Late preterm (35 weeks), appropriate for gestational age male baby was born by emergency LSCS in view of meconium stained liquor and fetal distress to a 28-year-old primigravida mother with antenatal history suggestive of oligohydramnios. IgG CMV and IgG rubella were positive in pregnancy. Baby cried immediately after birth and developed respiratory distress soon after birth requiring HFNC support for a suspected diagnosis of meconium aspiration syndrome/ transient tachypnoea of newborn. Baby had conjugated hyperbilirubinemia, firm hepatosplenomegaly, thrombocytopenia from birth. Ultrasound examination of the head showed intracranial calcifications at Bilateral basal ganglia region. Baby was given intravenous antibiotics and referred to a higher centre with a probable diagnosis of sepsis. Baby got admitted in our NICU on Day-3 of life. On admission general condition of the baby was sick (Figure 1). Baby was hemodynamically stable on high flow nasal canula. On examination baby was deeply icteric with pin point to macular rashes visible on chest, trunk, lower limbs and associated firm hepatosplenomegaly with hypotonia. First line investigations showed significant thrombocytopenia (8000/mm3), conjugated hyperbilirubinemia (TB-20mg/dl/ CB-14.4mg/dl), transaminitis (AST, ALT- 514 IU/L, 145 IU/L), coagulopathy (INR-2.1). On further evaluation, baby was found to have calcifications in bilateral periventricular, thalamic and basal ganglia region on ultrasound cranium (Figure 2), calcifications in the lateral wall of right ventricle on echocardiography (Figure 3) and calcifications around external iliac vessels. A multidisciplinary approach was taken in this case involving neonatology, paediatric gastroenterology, paediatric neurology, paediatric haematoncology. Diagnoses considered were congenital TORCH infections, Gestational alloimmune liver disorder and storage disorders. Multiple calcifications did not fit into any of the above diagnosis. TORCH workup was negative. Enzyme assay for storage disorders specifically Gaucher's disease and Niemann Pick Type-C were negative. Iron profile yielded a normal serum iron, TIBC, and transferrin saturation although ferritin levels were high (8000 ng/ml). In view of multiple calcifications, Serum calcium, PTH, Vitamin D3, Urine calcium/creatinine ratio were done. Reports were normal thereby excluding hyperparathyroidism. Baby was seen by the geneticists who suggested a possibility of Aicardi Goutières syndrome and Pseudo torch syndrome. This was ruled out with a whole exome sequencing which was otherwise normal but showed evidence of non G6PD related haemolytic anaemia and MTHFR

heterozygous mutations which were non-contributory for the diagnosis. Rash evolved during the period of hospital stay from a pin point rash to a macular and eventually a scaling rash (Figure 4,5,6,7). With persistent thrombocytopenia, haemolytic anaemia, low WBC counts and the evolving rash; a dermatologist opinion was sought. An ANA profile and skin biopsy was done. ANA profile showed a positive Anti Ro/ La antibody with skin biopsy suggestive of a lichenoid pattern of dermatitis consistent with cutaneous Lupus. Although there was no maternal history suggestive of SLE, she was screened and was also positive for Anti Ro/ La antibody. A diagnosis of neonatal Lupus was made. Baby had already received multiple platelet transfusions. IVIG at 2g/kg was given. Baby was finally discharged after 1.5 months of hospital stay at a platelet count of 24000/mm3. At this time baby was 2months old, on full demand feeds and gaining weight steadily. Baby was followed up on OPD basis at 3 months of age. There was steady weight gain, development was normal and the rash had disappeared (Figure 8,9). Liver and spleen had decreased in size and transaminitis had improved. Platelet count had increased (2.5 lakhs/mm3). A repeat echo showed a decrease in size of cardiac calcifications but the other areas of calcifications were unchanged.

INVESTIGATIONS:

	Day 1 of admission	Day 4 of admission	Day 15 of admission	Day 30 of admission	At discharge	Follow-up (3months of age)
Hb (g/dl)	12.4		11.1	10	9.7	10.10
TLC (/mm3)	13450		7610	5170	13440	8900
Platelet(/mm3)	8000	21000 (post transfusion)	44000	16000	20000	253000
AST/ALT	514/145	62/53	237/ 184	281/ 180	142/108	61/58
PT/INR	24.3/ 2.1		12.1/ 1.1			
TB/ CB (mg/dl)	20/ 14.4	20/14.4	8.7/ 6.6	7.3/ 5.7	6/ 4.8	0.6/ 0.52
CRP	145		10.4		3.6	

CASE PICTURES:

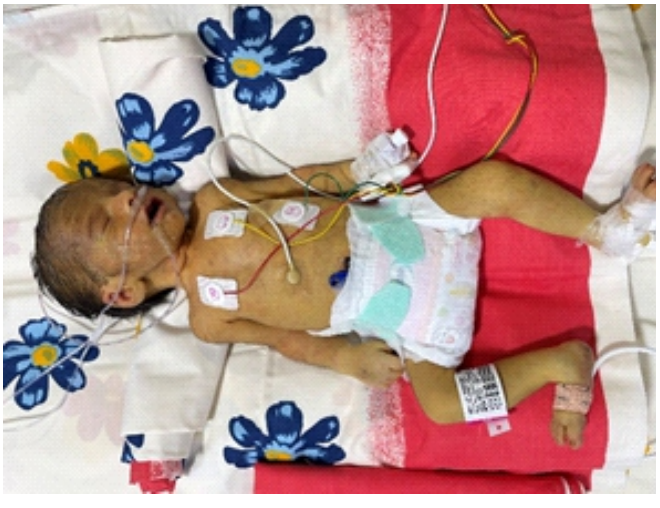


Fig.1

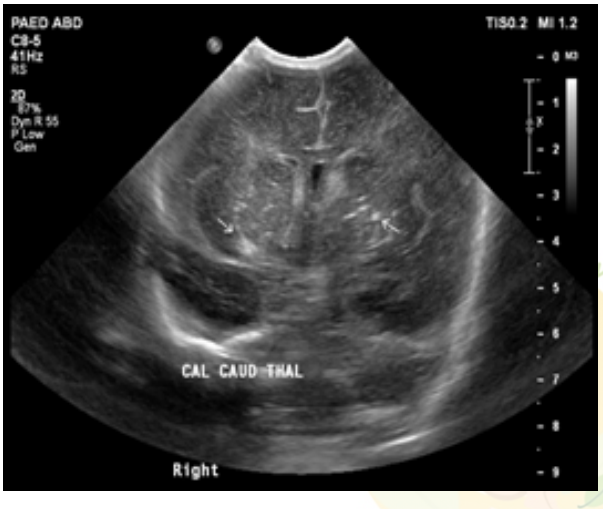


Fig.2

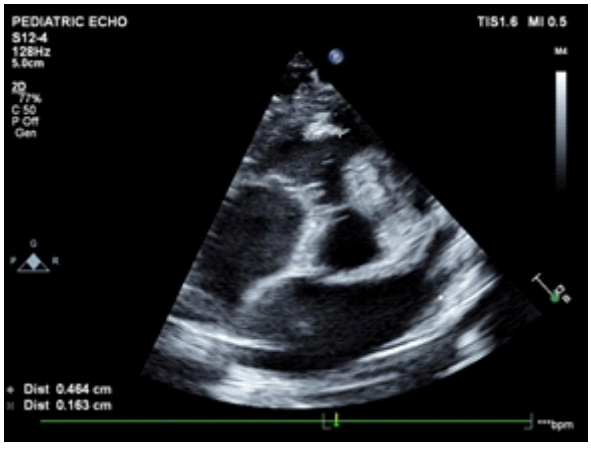


Fig.3

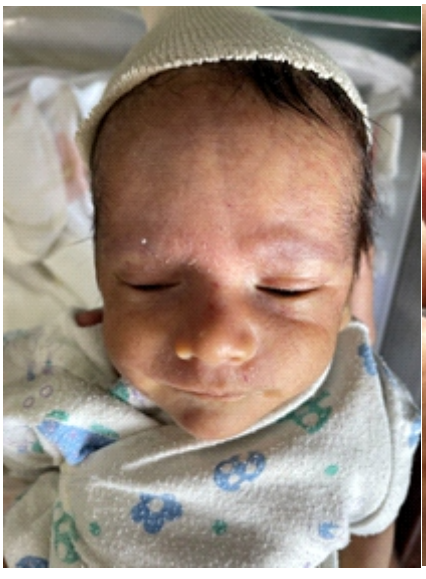


Fig.4

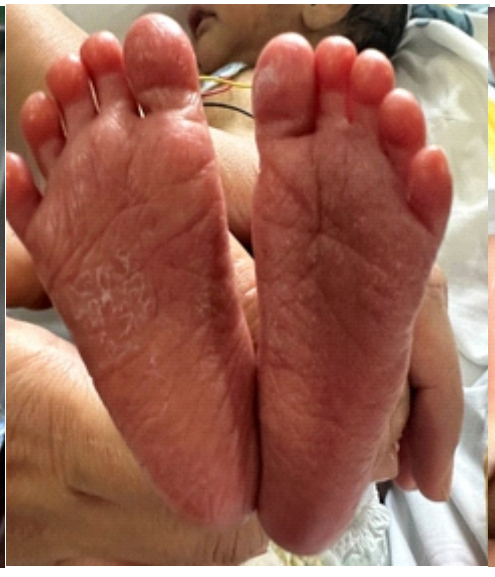


Fig.5

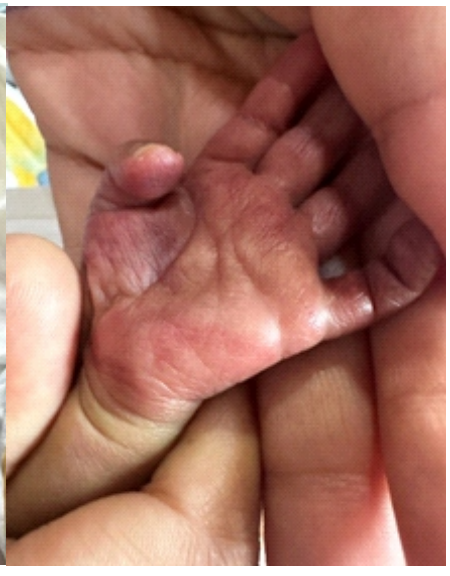


Fig.6

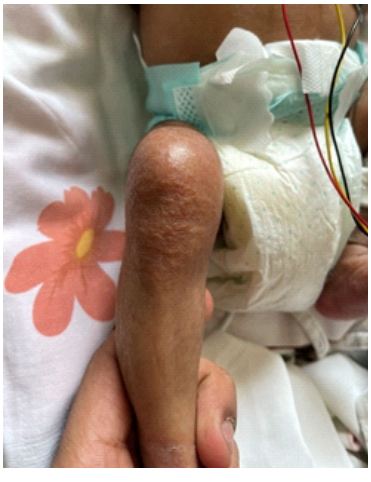


Fig.7

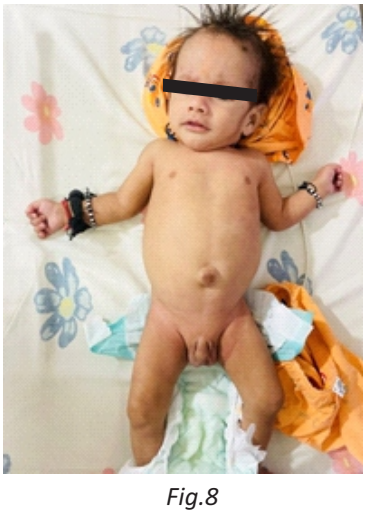


Fig.8

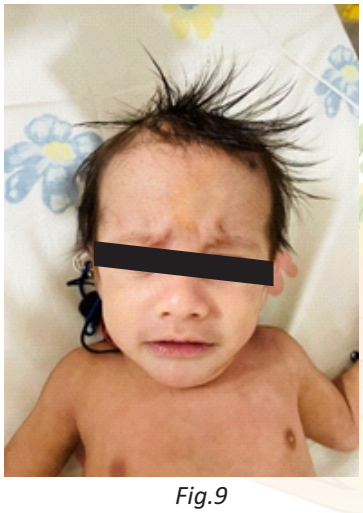


Fig.9

DISCUSSION:

Neonatal Lupus (NL) is a rare disease in neonates and infants caused by autoantibodies transmitted from the mother. Ninety-eight percent of the causative Antibodies include anti-SSA/Ro Ab and anti-SSB/La Ab, in addition to U1 ribonucleoprotein Ab (13). These antibodies migrate to the fetus via placenta, causing fetal tissue damage and neonatal lupus symptoms. NL is common in females rather than males, and typical symptoms include annular or spotty rashes on the face (especially periorbital), torso, and scalp (2). Symptoms other than skin include the hepatitis, neurological abnormality, defective spleen, congenital heart block, and hematocytopenia such as anemia, neutropenia, and thrombocytopenia (14). The rash is detected in 15%–25% of children with NL (4). However 10%–20% of patients manifest residual skin abnormalities such as telangiectasias, dyspigmentation, pitting, scarring, and skin atrophy (5). The overall risk of CHB in anti-SSA/Ro–positive women is estimated to be 2% to 5% (6). Congenital heart block ranges from the prolonged PR interval to the complete heart block. During development of the heart, Ro and La antigens can be exposed to the surface of the cardiac cells near the atrioventricular node, and these antigens react with the autoantibodies received from the mother. This antigen-Ab binding causes a localized immune response, that induces fibrosis in the conduction system and causes heart block ' (7,8). Confirmation is by fetal echocardiography at 18 to 24 weeks of gestation –'(5,9). Once diagnosed, fetal echocardiograms should be routinely monitored on a weekly or bi-weekly basis starting from 16–18 weeks of gestation. (10) While the non-cardiac symptoms are usually reversible, the congenital heart block is permanent and approximately 20% may be accompanied by cardiomyopathy and death from heart failure (2). Hepatobiliary disease can have three distinct presentations: Transient conjugated hyperbilirubinemia with mildly raised liver function tests (LFTs) in the first weeks of life; mild elevations of LFTs at 2 to 3 months of life; or severe liver failure during gestation presenting with conjugated hyperbilirubinemia, transaminitis and coagulopathy at birth (6).

Calcification in neonatal lupus is not a very well-known entity. Case reports with cranial calcifications have been reported only in infants and paediatric age groups. Neurological manifestations had been reported since early 1990's. There are two previous reports of echogenic lenticulostriate vessels in NLE. The first, a large sonographic study of 586 infants with various perinatal disorders, reported lenticulostriate vasculopathy without calcifications in one of three patients with NLE (11). Its postulated that following vasculopathy calcifications occurs later at the sites. Later, Cabanas et al (12) reported the same finding in three of four infants with complete congenital heart block and serologic evidence of NLE. One of these patients also had skin lesions and thrombocytopenia. Serial ultrasound examinations up to 18 months of age indicated the changes were persistent but not progressive. There are two previous reports of neurologic disease in children with a history of cutaneous NLE: an infant girl who had a residual myelopathy at 16 months of age (13) ,and congenital nonobstructive hydrocephalus was described in two sisters (14). There was a series of 11 infants of NLE studied, 9 of which had Computerized tomography (CT) of the brain done (15). There was diffuse, markedly reduced attenuation of the cerebral white matter in four infants studied in the first week of life, and also in an infant 5 weeks of age. Patchy reduced subcortical white matter attenuation was observed in another 5-week-old infant. Basal ganglia calcifications were present in two infants at 2 months of age, one of whom also had mild ventriculomegaly. As these studies indicate, calcifications in infantile and paediatric lupus are not unknown entities but calcifications in a neonate including cranial, right ventricle of heart, around external iliac vessels as in our case; isn't yet reported in literature. So, in this case we report a confirmed case of neonatal lupus with multiple calcifications.

Other rare manifestations of neonatal lupus include more than 15 cases of Chondrodysplasia punctata associated with maternal autoantibodies (16, 22), and it is therefore likely that this is a skeletal manifestation of NLE.

Conservative treatments are indicated depending on the symptoms. Most skin symptoms improve without scarring, without the need for special treatment. Topical steroid ointment, anti-malarials, and laser treatments are available. In addition, sunscreens and protective clothing are beneficial because exposure to sunlight can worsen the lesion (1).

Treatment of CHB in NLE remains controversial.

Prevention of progression to complete CHB may be achieved by treating the mother with fluorinated steroids (dexamethasone or betamethasone), which are not metabolized by the placenta and are available to the fetus in an active form. IV immunoglobulin had been used to prevent the development of CHB (6). The current recommendation is to screen anti-Ro/SSA antibody–positive mothers with serial echocardiograms and obstetric sonograms biweekly starting from week 16 of gestation. Early detection of cardiac manifestations of NLE including premature atrial contractions or moderate pericardial effusion preceding CHB may potentially be targeted with preventive therapy (6). First-degree heart blocks can be reversed by dexamethasone treatment given to the mother (6). Once third-degree block is unequivocally identified, reversal is unlikely to be achieved. The majority of children with CHB require pacemakers (6).

Medical history of the mother is crucial for the diagnosis, but caution is needed because approximately half of the mothers have no symptoms at the time of their diagnosis (2). In this case, retrospectively there was history of lower limb rash in the mother which was never evaluated or treated. She was found to have anti-SSA/Ro Ab and anti-SSB/La Ab positivity on evaluation during the management of this case. We should suspect NL when neonates or infants manifest typical skin rash, with or without a maternal history of connective tissue disorders.

In one of the follow-up studies, none of more than 100 children with NLE developed a connective tissue disease; however, most of these children have been followed for less than 20 years. A report from Japan suggested a less optimistic outcome; 8% of children had persistent or recurrently positive autoantibodies, and one met the American College of Rheumatology classification criteria for SLE (23). In a second study, 12% of children with NLE developed an autoimmune disease (i.e., juvenile rheumatoid arthritis, Hashimoto's thyroiditis, psoriasis, diabetes mellitus, and nephrotic syndrome), although only a minority had autoantibodies, and none had anti-Ro antibodies or SLE (23). The increased risk of autoimmune disease in these offspring may reflect the genetic predisposition of a child born to a mother with an autoimmune disease, as opposed to a direct delayed consequence of having had NLE.

Key Points:

- Multiple calcifications especially cranial calcifications are known entities.
- Cutaneous manifestations are the most common findings reported in NL.
- Suspect NL when neonates or infants manifest typical skin rash, with or without a maternal history of connective tissue disorders.
- All the clinical manifestations apart from the cardiac ones appears during the first 3 months after birth, lasts for a mean of 4 months, and disappear spontaneously by 6–8 months of age as the maternal Ab in baby's circulation disappear.
- Treatment of CHB including antenatal fluorinated steroids remains controversial.
- Neonatal lupus has got excellent long-term outcome when only skin lesions are present and if there are no cardiac manifestations.
- There is no apparent increased risk of systemic lupus erythematosus in the children. They may have a genetic predisposition to develop some form of autoimmune disease usually seen after adolescence and young adulthood.

References:

1. Lun Hon K, Leung AKC. Neonatal Lupus Erythematosus. Autoimmune Dis. 2012;2012:301274.
2. Perez MF, Torres ME de, Buján MM, Lanoël A, Cervini AB, Pierini AM. Neonatal lupus erythematosus: a report of four cases. An Bras Dermatol. 2011 Apr;86:34751.
3. Ramphul K, Mejias SG, Ramphul-Sicharam Y. Cutaneous Neonatal Lupus Erythematosus: A Case Report. Cureus. 10(2):e2212.
4. Silverman E, Jaeggi E. Non-Cardiac Manifestations of Neonatal Lupus Erythematosus. Scand J Immunol. 2010;72(3):2235.
5. University of Mansoura, Nasef N. Neonatal Lupus Erythematosus. Neonatol Clin Pediatr. 2014 Oct 20;1(1):110.
6. Firestein GS, Kelley WN. Kelleys textbook of rheumatology [Internet]. 9th ed. Philadelphia, PA: Elsevier/Saunders; 2013 [cited 2023 Sep 10]. 1 p. Available from:

http://cuml1.md.chula.ac.th/login

7. Izmirly PM, Buyon JP, Saxena A. Neonatal Lupus: Advances in Understanding Pathogenesis and Identifying Treatments of Cardiac Disease. Curr Opin Rheumatol. 2012 Sep;24(5):46672.
8. Capone C, Buyon JP, Friedman DM, Frishman WH. Cardiac Manifestations of Neonatal Lupus: A Review of Autoantibody Associated Congenital Heart Block and its Impact in an Adult Population. Cardiol Rev. 2012 Mar;20(2):726.
9. Yildirim A, Sedef Tunaodlu F, Karaadaç AT. Neonatal congenital heart block. Indian Pediatr. 2013 May 1;50(5):4838.
10. Diagnosis and Treatment of Fetal Cardiac Disease | Circulation [Internet]. [cited 2023 Sep 10]. Available from: https://www.ahajournals.org/doi/10.1161/01.cir.0000437597.44550.5d
11. Wang HS, Kuo MF, Chang TC. Sonographic lenticulostriate vasculopathy in infants: some associations and a hypothesis. AJNR Am J Neuroradiol. 1995 Jan;16(1):97102.
12. Cabañas F, Pellicer A, Valverde E, Morales C, Quero J. Central nervous system vasculopathy in neonatal lupus erythematosus. Pediatr Neurol. 1996 Sep 1;15(2):1246.
13. Kaye EM, Butler IJ, Conley S. Myelopathy in neonatal and infantile lupus erythematosus. J Neurol Neurosurg Psychiatry. 1987 Jul;50(7):9236.
14. Nakayama-Furukawa F. Hydrocephalus in Two Female Siblings With Neonatal Lupus Erythematosus. Arch Dermatol. 1994 Sep 1;130(9):1210.
15. Prendiville JS, Cabral DA, Poskitt KJ, Au S, Sargent MA. Central nervous system involvement in neonatal lupus erythematosus. Pediatr Dermatol. 2003;20(1):607.
16. Chitayat D, Keating S, Zand DJ, Costa T, Zackai EH, Silverman E, et al. Chondrodysplasia punctata associated with maternal autoimmune diseases: Expanding the spectrum from systemic lupus erythematosus (SLE) to mixed connective tissue disease (MCTD) and scleroderma report of eight cases. Am J Med Genet A. 2008 Dec 1;146A(23):303853.
17. Elcioglu N, Hall CM. Maternal systemic lupus erythematosus and chondrodysplasia punctata in two sibs: phenocopy or coincidence? J Med Genet. 1998 Aug 1;35(8):6904.
18. Austin-Ward E, Castillo S, Cuchacovich M, Espinoza A, Cofré-Beca J, González S, et al. Neonatal lupus syndrome: a case with chondrodysplasia punctata and other unusual manifestations. J Med Genet. 1998 Aug;35(8):6957.
19. Honda R, Ichiyama T, Maeba S, Sunagawa S, Furukawa S. Male siblings with tibia-metacarpal type of chondrodysplasia punctata without maternal factors. Brain Dev. 2008 Apr;30(4):3014.
20. Kelly TE, Alford BA, Greer KM. Chondrodysplasia punctata stemming from maternal lupus erythematosus. Am J Med Genet. 1999 Apr 23;83(5):397401.
21. Shanske AL, Bernstein L, Herzog R. Chondrodysplasia Punctata and Maternal Autoimmune Disease: A New Case and Review of the Literature. Pediatrics. 2007 Aug 1;120(2):e43641.
22. Kozlowski K, Basel D, Beighton P. Chondrodysplasia punctata in siblings and maternal lupus erythematosus: CDP and MLE. Clin Genet. 2004 Oct 20;66(6):5459.
23. James T. Cassidy REP. Textbook of Pediatric Rheumatology (Fifth Edition) [Internet]. Usa: Elsevier; 2005 [cited 2023 Sep 10]. Available from: https://www.biblio.com/book/textbook-pediatric-rheumatology-fifth-edition-james/d/1471304569

Congenital Diaphragmatic Hernia

Shandip Kumar Sinha
Director (Pediatric Surgery and Pediatric Urology)
Medanta - The Medicity, Gurugram, India.

Congenital diaphragmatic hernia (CDH) is a condition in which because of a defect in the diaphragm, abdominal viscera to herniate into the thorax. The newborn may be minimally symptomatic at birth, but often the symptoms are severe and may result in neonatal mortality. The major factor for mortality is irreversible pulmonary hypoplasia or severe persistent pulmonary hypertension. The diagnosis of congenital diaphragmatic hernia can be made in utero with a great deal of accuracy, which allows time for parents to receive genetic and pediatric surgical consultation. Outcome for neonates born with congenital diaphragmatic hernia has improved because of combination of early diagnosis and advances in prenatal and postnatal therapies.

Incidence and Associated Malformations

The incidence of CDH is estimated to be 1 per 2,000 to 4,000 births.Excluding conditions that are considered to be part of the "CDH syndrome" (pulmonary hypoplasia, patent ductus arteriosus, patent foramen ovale, and malrotation), approximately 40% of liveborn patients who have CDH have one or more associated anomalies. Approximately 60% of these anomalies are cardiac, 23% are genitourinary, 17% are gastrointestinal, 14% involve the central nervous system (CNS), and 10% are chromosomal.

Pathophysiology

- The diaphragm develops largely from three structures:
  - o The pleuroperitoneal membrane
  - o The septum transversum
  - o Marginal ingrowths from the muscles of the body wall.
- Congenital diaphragmatic hernia results from failure of formation or fusion of the components of the diaphragm, such that abdominal contents can move through a defect into the chest.
- Sometimes, failure of muscularisation may produce a thin, weak diaphragm, referred to as an eventration of the diaphragm.
- Postero-lateral diaphragmatic defect (usually on the left side) with communicating thoracic and abdominal spaces
- In left-sided hernias the stomach, the small bowel, and part of the colon are usually located in the thorax. The left lobe of the liver and the spleen may also be involved. In cases of dorsal aplasia of the diaphragm, the adrenal gland and even the kidney may be displaced into the thorax
- In right-sided hernias the liver and the small and large bowel may be located in the thorax

The pathophysiology of CDH involves pulmonary hypoplasia, pulmonary hypertension, pulmonary immaturity, and potential deficiencies in the surfactant and antioxidant enzyme system.

Prenatal Diagnosis

Polyhydramnios is reported to complicate up to 80% of pregnancies in which CDH occurs. The diagnosis is suggested strongly by the presence of a fluid-filled stomach or intestine at the level of the four-chamber view of the heart. The absence of a fluid-filled stomach in the abdomen or the presence of liver (confirmed by Doppler vascular flow) in the thorax also increases the likelihood of the diagnosis. The overall prenatal detection rate is 54%.

The association of polyhydramnios with CDH is believed to result from a kinking of the distal esophagus caused by translocation of the stomach into the chest. This effectively obstructs fetal swallowing, resulting in polyhydramnios, and in some instances, a very small fetal stomach that may be difficult to see in either the chest or abdomen.

Prenatal Care

Other congenital anomalies, particularly those affecting the cardiovascular and central nervous systems should be searched for when a prenatal diagnosis of CDH has been made. Fetal karyotype should be determined by amniocentesis or chorionic villus sampling. If the diagnosis of CDH is made before 20 weeks' gestation, identification of a second major, life-threatening malformation or chromosomal aberration may lead the parents to consider elective termination of the pregnancy. Ultrasonographic lung biometry including Lung area-to-head circumference ratio (LHR) can be used to predict prognosis.

- LHR = lung diameter (width) (mm) × lung diameter (length) (mm)/head circumference (mm)
- 95% survival if LHR >1.4
- 75% mortality if LHR <1

The Prognostic factors can be classified as

- Genetic Factors
  - o Karyotype Abnormalities
  - o Syndromic
- Hernial Factors
  - o Sac
  - o Placement of liver in thorax
- Cardiac Factors
  - o Congenital Heart Disease
  - o LV/RV Ratio (Cardiovascular Index)
  - o Modified McGoon Index - pulmonary artery diameter at the point before its branching, divided by the diameter of the descending aorta at the level of the diaphragm.
- Lung Factors
  - o Lung to Head Ratio (LHR) - Validated predictor of CDH Survival
  - o Total Lung Volume (TLV)
  - o Percent Predicted Lung Volume (PPLV)

A composite Prognostic index is proposed(table 1)

Obstetrics Care- Delivery

For a fetus that has CDH, a plan for maternal delivery at a tertiary perinatal center offering all advanced strategies for respiratory failure usually is most appropriate. A spontaneous vaginal delivery should be anticipated unless obstetric issues dictate otherwise. The best timing and mode of delivery of infants with CDH is not known with certainty. There is no advantage to scheduled cesarean delivery though, based on retrospective reviews, some believe that there might be an advantage to early term delivery (37–38 weeks). Nevertheless, most recommend scheduled induction of labor at 38–39 weeks, preferably at a center where proper monitoring and all aspects of advanced care are available. For children with low LHR ratio, centre with neonatal ECMO should be preferred, although it may be difficult in most Indian cities. Delivery by cesarean section is performed when indicated by standard obstetric criteria.

Postnatal Diagnosis

Babies born with CDH have a scaphoid abdomen and develop progressive respiratory distress Poor perfusion and hypotension may develop. The presentation of CDH after birth is determined primarily by the severity of the pulmonary hypoplasia and pulmonary hypertension. The most severely affected babies will be symptomatic with their first breath, and most infants will develop symptoms within the first 24 hours of life. The diagnosis is confirmed by a chest radiograph that demonstrates bowel loops within the chest. A nasogastric tube should be placed immediately to locate the gastric bubble and decompress the intestines. Once the diagnosis of CDH has been confirmed, a careful search for associated anomalies should be performed with renal and cranial ultrasonography, echocardiography, and karyotyping.

Although most patients who have CDH present within the first day of life, 10% to 20% present later with recurrent respiratory distress, chronic pulmonary infection, or acute gastrointestinal symptoms caused by gastric volvulus or intestinal obstruction.

Preoperative Care

The management of CDH is based on the understanding that pulmonary hypoplasia and pulmonary hypertension caused by CDH represent a physiologic emergency, not a surgical one. The principles of initial medical management of the infant

with CDH include:

- Using gentle ventilation techniques (permissive hypercapnia) to minimize iatrogenic lung injury.
- Minimizing the onset and impact of pulmonary hypertension
- Performing diagnostic studies to rule out associated anomalies and to prepare the infant for the possibility of ECMO
- Delaying surgical repair until hemodynamic stability has been maintained for at least 24 h.

Resuscitation and Monitoring

All severely symptomatic patients presenting after birth or who have CDH diagnosed prenatally should have endotracheal intubation. A nasogastric tube should be placed to minimize intestinal distension within the thoracic cavity. Arterial and venous access should be achieved through the umbilicus, and fluids (crystalloid, colloid, and blood) and inotropes (dopamine or dobutamine) administered to support blood pressure and perfusion. In addition to the umbilical arterial line, which is postductal, it is useful to have a preductal monitor (either a right radial arterial line or right upper extremity pulse oximeter probe) as well. The oxygen saturation gradient allows trending of the right-to-left shunt fraction and offers an estimate of the severity of pulmonary hypertension. It is very important to avoid systemic hypotension, which may lead to right-to-left shunting in the presence of pulmonary hypertension. Acidemia is predominantly metabolic (related to hypoperfusion) and should be corrected with administration of fluids and bicarbonate. Stress is considered a stimulus of pulmonary vasoconstriction, so the infant should be sedated with combinations of narcotic (morphine or fentanyl) and hypnotic (midazolam) agents.

Preoperative stabilization

- Chest X-ray- In left-sided defects, loops of bowel can be seen in the left chest. The heart is deviated to the right. Little room is left for the lungs, particularly the left lung which is markedly compressed. Sometimes, the appearance may be difficult to distinguish from basal lung cysts, in which case a repeat chest x-ray is performed after a nasogastric tube has been inserted, the tip of which can be seen in the chest. Alternatively, a barium study will show bowel within the thoracic cavity when there is a diaphragmatic hernia
- ECHO and USG KUB for associated anomalies should be done
- Care must be taken to avoid hyper-inflation and barotrauma of the small hypoplastic lungs.
- Ventilation with a face mask ('bagging') should be avoided as this may force air into the stomach, increasing its volume at the expense of the already compromised lungs.
- Vigorous endotracheal ventilation should also be avoided because of the risk of causing barotrauma and a tension pneumothorax, which can lead to the rapid demise of the infant
- High Frequency Oscillatory ventilation if required
- Arterial and venous pressure monitoring
- Pulse oximetry
- Transcutaneous preductal and postductal PaO2 monitoring
- Correction of acidosis
- ECMO, if required
- Sudden deterioration of the infant's condition during initial resuscitation or during transport suggests the development of a tension pneumothorax, and this may necessitate prompt drainage by needle aspiration or insertion of an intercostal drain.

Ventilator Strategies

The goals of ventilation for the infant who has CDH are to

- Achieve acceptable postductal oxygen saturation to meet the tissue's metabolic needs,
- Avoid severe hypercarbia and respiratory acidosis
- Avoid iatrogenic lung injury.

Surfactant

The therapeutic role of exogenous surfactant in the postnatal management of respiratory failure associated with CDH remains uncertain. Human studies of surfactant phospholipids in amniotic fluid yield conflicting data regarding fetal lung maturity. A recent study of surfactant composition of bronchoalveolar lavage fluid failed to demonstrate a difference between infants who had CDH and were ventilated, those who received ECMO, and age-matched controls. Reports of empiric exogenous

surfactant therapy for CDH are anecdotal and without demonstrated benefit.

Nitric Oxide

Nitric oxide mediates smooth muscle relaxation by liberating cyclic guanosine monophosphate (cGMP) from vascular endothelium. The responsiveness of infants who have CDH to iNO has been generally disappointing, with only anecdotal success reported. Recent Cochrane review reports that the outcome of infants with diaphragmatic hernia was not improved; indeed there is a suggestion that outcome was slightly worsened.

Extracorporeal Membrane Oxygenation (ECMO)

ECMO is useful for infants who have CDH and respiratory failure refractory to conventional and high-frequency modes of ventilatory support. ECMO provides a means of maintaining oxygen delivery only temporarily. Its success depends on the reversibility of the pathologic factors that led to respiratory failure within the time frame that ECMO can be used. If the pulmonary hypoplasia associated with CDH recovers in this time of ECMO support, the results will be good. Unfortunately, often it exceeds that provided by ECMO bypass. This accounts for the significant differences in survival observed between patients who have CDH and those who have more rapidly reversible causes of respiratory failure, including PPHN, meconium aspiration syndrome, and sepsis.

Timing of Repair

CDH is a physiologic emergency rather than a surgical one. Historically, infants who had CDH were rushed to the operating room under the (false) belief that decompression of the lungs by reduction of the abdominal viscera offered the greatest chance for survival. Usually, after a brief “honeymoon” period, progressive hypoxemia would develop as increased pulmonary vascular resistance led to right-to-left vascular shunting and accelerated respiratory deterioration. The realization that early surgical repair was associated with unfavorable changes in lung compliance and gas exchange resulted in the rationale for a period of preoperative stabilization and delayed surgical repair. The optimal timing of surgery for delayed repair is unknown. A recent report from the CDH Study Group, which included data from 62 centers in North America, Europe, and Australia, indicated that the mean age at surgery in patients not treated with ECMO was 73 hours. This practice of delaying repair beyond just a few hours may facilitate the pulmonary vascular remodeling purported to occur much more slowly in CDH lungs than normal lungs and may decrease the sensitivity of these vessels to vasoconstrictive stimuli, including surgical stress.

However a recent Cochrane review reports that there is no clear evidence which favors delayed (when stabilized) as compared with immediate (within 24 hours of birth) timing of surgical repair of congenital diaphragmatic hernia, but a substantial advantage to either one cannot be ruled out. A large, multicenter randomized trial would be needed to answer this question.

As early repair was definitely not associated with improved survival, it is reasonable to continue the practice of delayed repair until evidence based data are generated.

Surgical Technique

A subcostal incision is usually used. The herniated viscera are reduced gently from the chest and eviscerated from the abdomen, and the defect is visualized. In patients with (20%) with a hernia sac,it should be excised. Usually the anterior leaf of diaphragm is fully developed whereas posterior leaf is partially developed. The posterior leaf typically is rolled up into a muscular ridge in the retroperitoneum, which requires that the overlying peritoneum be incised and the muscle “unrolled” to assess its adequacy fully. If there is enough muscle, the defect can be closed primarily with interrupted, nonabsorbable suture. If there is inadequate diaphragm to accomplish a primary repair, a number of reconstructive techniques make use of nearby musculature, such as the latissimus dorsi or the internal oblique and transversus abdominus muscles. Abdominal wall closure after diaphragmatic hernia repair may result in unacceptably high intra-abdominal pressures, even after extensive stretching of the abdominal wall. In these instances, ventral hernia (simple skin closure without closure of abdominal wall musculature) may be created. Alternatively, a prosthetic silo may be created. Chest tubes are surgeons prerogative, and many surgeons will not use it.

Postoperative Management

Postoperatively, meticulous attention must be given to ventilator management, fluids, and cardiovascular performance. Ventilator support should ensure

adequate tissue oxygenation and the avoidance of hypercarbia and acidosis. Postoperative fluid requirements may be significant, and hypotension must be avoided at all costs. The infant must receive adequate narcotic analgesia and sedation and should begin nutrition by the parenteral route shortly after surgery because the time to resolution of intestinal ileus permitting enteral nutrition often is prolonged.

Systemic antibiotics are administered perioperatively, with the duration of the postoperative course (usually 1 to 5 d) determined by whether a prosthetic patch was used. Weaning from the ventilator is gradual and determined by the degree of pulmonary hypoplasia present. Regular chest radiographs are helpful in monitoring the size and growth of the lungs and the return to midline of the mediastinum.

Surgery and ECMO

The best strategy for timing of repair in infants who have CDH and require ECMO remains unclear. Currently, the options range from "early" repair (usually within 2 days of institution of bypass) to repair after decannulation. No comparative study has been performed, but most centers have adopted a policy of delayed repair on ECMO after successful weaning, but prior to decannulation.

Outcome

- Mortality remains between 20% and 50%
- The prognosis is better when the signs appear 6 h or more after birth
- ECMO may modify the prognosis in worst cases

The newborns not requiring or given ECMO has a survival rate of 77%. Wide variations in reported survival rates occur throughout the literature. In a recent population based study ,only 60% of CDH were live born , with rest undergone elective termination or were still born. The population of infants reaching the tertiary surgical center represented only 40% of the total cases of CDH. Ninety-two percent of postoperative infants survived beyond 1 year of age, as did 80% of infants who reached the surgical referral center. However, only 52% of live-born infants, 32% of all cases, survived. Therefore, the overall mortality rate for this condition remains high, despite increased prenatal detection, transfer to tertiary institutions for delivery, and advances in neonatal care, and is influenced significantly by the rate of prenatal termination.

Long Term Follow Up

Gastroesophageal reflux is a common clinical problem for patients who have undergone repair of a CDH. Failure to thrive or complications such as aspiration pneumonia, reactive airways disease, or life-threatening events related to aspiration are indications for antireflux surgery. Children with concomitant feeding difficulty might also benefit from placement of a gastrostomy button.

Fetal Therapy for CDH

As mortality in newborns with CDH is mainly due to pulmonary hypoplasia and severe associated pulmonary hypertension, fetal therapy in order to improve fetal lung development were developed. The first phase of fetal surgery consisted in open repair with one-stage surgical correction of the anatomic defect. It was followed by tracheal occlusion technique based on the decrease egress of lung fluid in order to improve lung growth. Initial approach of tracheal occlusion used clips on the trachea. It is now performed with intra-tracheal inflatable balloon.

Fetoscopic Approach (FETENDO)

- Temporary fetal tracheal occlusion reverses the pulmonary hypoplasia seen in congenital diaphragmatic hernia (CDH) and provides an alternative treatment strategy for some fetuses with CDH by distending the hypoplastic lungs. The safety and efficacy of the following methods are being tested in ongoing trials. Only those fetuses most severely affected, with liver herniated into the chest, a diagnosis before 24 weeks, and a ratio of lung to head of less than 1.4, are eligible for this treatment. Less severely affected fetuses are best managed after birth
- PLUG (Plug the Lung Until it Grows)
- This fetoscopic approach uses small scopes and video equipment to place a detachable balloon by fetal bronchoscopy, without opening the uterus
- The balloon occludes the trachea and enables the lungs to grow.
- Although the lungs grow in size the lung function is not necessarily increased
- EXIT (Ex utero Intrapartum Treatment)-This procedure is used to reverse tracheal occlusion devices in fetuses with severe CDH who have been treated with the PLUG procedure. Carried out before the umbilical cord is severed, this procedure offers the advantage of ensuring uteroplacental gas exchange while the fetus is still receiving placental support
- For this procedure a well-trained interdisciplinary

Neonate With Giant Omphalocele

Pinaki Dutta[1], Naveen Parkash Gupta[2], Shandip Kumar Sinha[3], Chanchal Singh[4], Anil Batra[2], Jayasree Sundar[5]

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. Director, Department of Pediatric Surgery, Medanta Hospital, Gurgaon
4. Lead Consultant, Department of Fetal Medicine, Madhukar Rainbow Children's Hospital, Delhi.
5. Director, Department of Obstetrics and Gynecology, Madhukar Rainbow Children's Hospital, Delhi.

Case - Term(37 weeks), appropriate for gestational age male baby with birth weight of 2314 gm born via Caesarian section whose antenatal scan showed ventral abdominal wall defect containing bowel, part of liver and gall bladder (30 mm * 28 mm), the base of defect measuring 21 mm suggestive of omphalocele with single umbilical artery (Figure 1). The baby cried immediately after birth. A large swelling was observed at birth with a sac over it (Figure 2). Baby was shifted to NICU and started on intravenous fluids.

Diagnosis – Giant omphalocele

Further course – Baby was operated on day 3 of life. The baby was extubated on day 5 of life, feeds were started which the baby tolerated well. Echocardiography and ultrasound abdomen was normal. The baby was discharged from hospital on day 12 of life. In follow up baby is now 3 months old, thriving well. He is having an incisional hernia for which surgery is planned.

Condition – An omphalocele is a type of abdominal wall defect. An omphalocele occurs when there is a failure of the migration of lateral folds to form the umbilical ring

and failure of the herniated midgut to return to the abdominal cavity early in gestation. Incidence is 1 in 4000 live births. Varying amounts of bowel may be contained within the omphalocele sac along with other intra-abdominal viscera including liver, bladder, stomach, ovary, and testis.

Diagnosis - The prenatal diagnosis is based on ultrasound examination and made in a fetus with a midline abdominal wall defect of variable size in the area of the umbilicus, covered by a membranous sac, and containing herniated abdominal contents (typically bowel, but often liver, and occasionally stomach or bladder). The cord inserts into the apex of the sac. Ascites may be seen in the sac or the abdomen.

A "giant" omphalocele can be defined as an omphalocele containing >75 per cent of the liver or a defect greater than 5 cm.

It's important to rule out associated anomalies on prenatal ultrasound. Omphalocele is accompanied by an 18-24% incidence of cardiac anomalies(septal defects like ASD/VSD, tetralogy of Fallot, tricuspid atresia, ectopic cordis). Associated syndromes such as Beckwith-Weidman syndrome, cloacal exstrophy, Donnai - Barrow syndrome, pentalogy of Cantrell, and OEIS syndrome (omphalocele, exstrophy of bladder, spinal defect, imperforate anus) are seen. Chromosomal abnormalities most commonly associated are trisomies 13, 18 and 21.

Postdiagnostic evaluation –

Fetal chromosomal microarray to rule out aneuploidy. An isolated omphalocele-containing liver is usually associated with fetal euploidy, whereas associated anomalies and an intracorporeal liver are commonly associated with aneuploidy.

Fetal Echocardiogram

Testing for Beckwith-Wiedemann syndrome in euploid fetuses

Differential diagnosis – Gastroschisis is the major disorder to consider in differential diagnosis.

Differentiating features between omphalocele and gastroschisis

	Omphalocele	Gastroschisis
Sac	Present	Absent
Associated anomalies	Common	Uncommon
Location of defect	Umbilicus	Right of umbilicus
Maternal age	Average	Younger
Surgical management	Not urgent	Urgent
Prognostic factors	Associated anomalies	Condition of bowel

Management

Timing of birth – Preterm birth offers no advantage. Wait for spontaneous labour until at least 39 weeks of gestation in the absence of standard indications of early delivery.

Route of birth – A trial of labour should be given. Only in cases of giant omphalocele, caesarean section is preferred.

Place of delivery – Preferably a tertiary care centre having an appropriate level of neonatal medical and surgical care.

Delivery room care

a. Cover the defect with gauze dressings soaked in thermally neutral sterile saline and cover the dressing with clear plastic wrap
b. Insert an orogastric tube to decompress the stomach
c. Stabilize the airway to ensure adequate ventilation
d. Establish peripheral intravenous access

Surgical management - Treatment options depend on size of the defect/gestational age/the presence of associated anomalies.

Small omphalocele- In infants with small defects, primary closure consists of excision or inversion of the sac with closure of fascia and skin.

Large omphalocele- Primary closure is sometimes not possible due to large defect as there are chances of compartment syndrome. Multiple methods like flaps and non absorbable patches are available in case surgeon opts for primary closure. Sometimes staged closure is done using silicone plastic 'silo' to provide staged reduction. Another option for medium-sized omphalocele with thick sac is to do sequential ligation of the sac itself for gradual reduction of viscera. Escharotic therapy which results in gradual epithelialization of the omphalocele sac is another form of staged closure for those neonates who cannot tolerate operation due to prematurity, pulmonary hypoplasia,congenital heart disease or other anomalies.

Postoperative course- Majority of patients post surgery will require mechanical ventilation for few days during which abdominal wall and bowel wall edema will decrease leading to decrease in intrabdominal pressure. Tube feeds can be started once bilious aspirates are no more there.

Outcome – Overall survival of live-born infants is approximately 90 per cent. This high rate reflects the ability to diagnose abdominal wall defects prenatally and the decision of many families to proceed with termination of pregnancy when the defect is severe or multiple associated anomalies are present.

With large omphaloceles issues like Gastroesophageal reflux disease, pulmonary insuffiency, recurrent lung infections or asthma and feeding difficulty with failure to thrive are present. They may occasionally require gastrostomy tube feeding to ensure adequate growth.

References:

1. Christison-Lagay ER, Kelleher CM, Langer JC. Neonatal abdominal wall defects. Semin Fetal Neonatal Med. 2011 Jun; 16(3):164-72. doi:10.1016/j.siny.2011.02.003. Epub 2011 Apr 6.

   PMID: 21474399.

2. Henrich K, Huemmer HP, Reingruber B, Weber PG. Gastroschisis and omphalocele: treatments and long-term outcomes. Pediatr Surg Int. 2008 Feb;24(2):167-73. doi: 10.1007/s00383-007-2055-y. Epub 2007 Nov 6. PMID: 17985136.
3. Van Eijck FC, Hoogeveen YL, van Weel C, Rieu PN,

Wijnen RM. Minor and giant omphalocele: long-term outcomes and quality of life. J Pediatr Surg. 2009 Jul;44(7):1355-9. doi: 10.1016/j.jpedsurg.2008.11.034. PMID: 19573661.

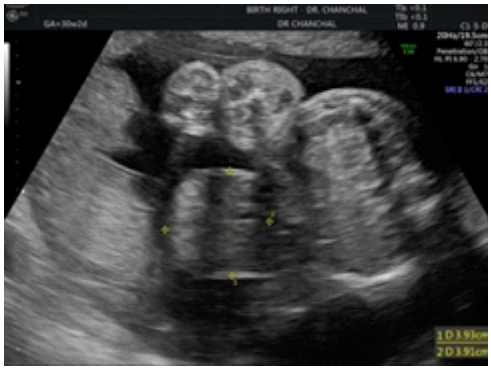


Fig. 1 : Exomphalos in antenatal scan

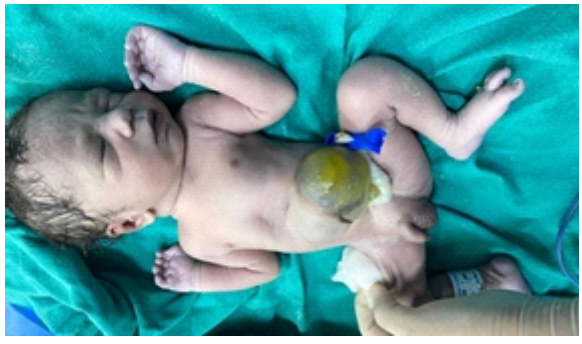


Fig. 2 : Neonate with abdominal mass after birth. Note the mass is covered with sac and position of umbilical cord

Respiratory distress in a term newborn

Deepika Rustogi[1], Neelam[2]

1. Senior Consultant & Head NICU, Yashoda Superspeciality Hospital, Kaushambi, Ghaziabad, UP.
2. Junior Consultant, Yashoda Superspeciality Hospital, Kaushambi, Ghaziabad, UP.

Clinical Presentation: Early term male baby (37 weeks) was delivered through emergency caesarean section (indication: fetal distress) to a primigravida mother with gestational diabetes mellitus well controlled on insulin, with otherwise uneventful antenatal period. The anatomy scan was reported normal. His birth weight was 2.660 kg (26th centile). Baby cried immediately after birth with the Apgar score of 9 at 1 and 9 at 5 minutes of life. He developed respiratory distress soon after birth requiring CPAP support in the delivery area and was shifted to NICU after initial stabilisation. He required a maximum PEEP support of 7cm of water and oxygen up to 40%. Chest X-ray done showed homogenous opacity in the right lower zone of the lung field with no air bronchogram (Figure 1A). The upper margin of the opacity was sharp but smooth, seen up to the 4th intercostal space and had a contour of the diaphragm. No obvious intra-abdominal contents were seen in the chest cavity and the distal end of the feeding tube was seen on the left side at the level of fundus of the stomach. On auscultation, air entry was slightly reduced on the right side, especially in the infra-axillary and infrascapular areas. Urgent bedside ultrasonography of the chest was performed which revealed the liver just below the level of the nipple on the right side with elevated right hemidiaphragm, though the diaphragmatic movement was not well visualized. The possibility of Eventration of diaphragm was kept. No other obvious anomaly was noted, there was no facial dysmorphism or asymmetry. There was no preductal to postductal saturation split. A 2D echo done by a pediatric cardiologist was reassuring and ruled out concealed cardiac anomaly. MRI chest was done on day 5 of life as respiratory distress settled after being transitioned to room air which confirmed the congenital diaphragmatic eventration (Figure 1B).

Final Diagnosis: Right sided Eventration of Diaphragm

Further course: Work of breathing got better and child was off CPAP on 4th day of life. He was started on gavage feeds initially which were transitioned to oral feeds as the clinical condition improved. The paediatric surgical consult was taken and was advised watchful waiting with a plan to intervene surgically on an elective basis between 1 to 3 months of age. He was discharged home on room air on exclusive breast feeds on day 6. In Follow-up visits, child is gaining weight well and there is no breathing difficulty. Presently he is 2 months old and weighs over 3.8 kg. Plan is to do open thoracotomy with plication of the diaphragm around 3 months of age.

Discussion: Eventration of diaphragm refers to an abnormal contour of the diaphragmatic dome with no disruption of its continuity. It was first described in 18th century by Jean Louis Petit during a postmortem finding (1). Eventration can be congenital or acquired. Congenital diaphragmatic eventration (CDE) results from inadequate development of the muscle or a thin, abnormal diaphragm or absence of the phrenic nerves at birth (2). CDE is a rare anomaly with a reported incidence of 1 per 10,000 live births and male sex preponderance (3). The incidence is likely higher than reported as most cases remain undiagnosed. The exact aetiology of congenital diaphragmatic eventration is unknown. Acquired eventration is caused by injury to the phrenic nerve, resulting from either a traumatic birth or thoracic surgery for congenital heart disease.

Most cases of diaphragmatic eventration are reported from adult population due to its asymptomatic presentation and incidental diagnosis (4). However, newborns can present with respiratory distress in the form of cyanosis, tachypnea, or work of breathing. CDE is one of the extrapulmonary causes of respiratory

distress. It can occasionally present with vomiting secondary to gastric volvulus. The closest differential for eventration at birth is congenital diaphragmatic hernia (CDH). Their differentiating features have been summarised in Table 1. There is no standard imaging of choice for diagnosis of Eventration. It can be diagnosed on chest radiographs. Ultrasound can be done to confirm the diagnosis by the presence of minimal or paradoxical movement of the diaphragm. Fluoroscopy can also be used to demonstrate real-time abnormal diaphragmatic movements. Dynamic contrast-enhanced MRI or CT scan can also be used to establish the diagnosis. Even in the best of hands and technology, possibility of CDH cannot be ruled out in 10% of cases as per available scientific evidence.

The treatment of CDE is surgical. The goal of operative management when detected early during infancy is to restore normal parenchymal volume. Plication is the surgical treatment of choice (5). It can be done through standard thoracotomy or a thoracoscopic approach. Thoracoscopy is associated with reduced pain scores, postoperative morbidity, and gastrointestinal dysfunction when compared with thoracotomy (6). Therefore elective diaphragmatic eventration repair by thoracoscopy is considered feasible, safe, and efficient in children (6). Surgical repair should be considered earlier if conservative treatment fails or in case of recurrent distress, pneumonia, and failure to gain weight, feeding difficulty or gastrointestinal complications. The long-term outcome of patients who undergo surgical repair is good (4).

Key messages

1. Diaphragmatic eventration can present in neonatal period with respiratory distress
2. Any neonate with persistent tachypnea should have a radiograph (X ray), to pick up concealed congenital anomalies in a timely manner.
3. Early diagnosis and timely repair of CDE can improve quality of life by preventing complications like recurrent respiratory infections, and gastrointestinal disorders.
4. Timing of intervention depends on the clinical profile rather than the diagnosis itself.

References

1. París F, Blasco E, Cantó A, Tarazona V, Casillas M. Diaphragmatic eventration in infants. *Thorax*. 1973;28(1):66-72. doi:10.1136/thx.28.1.66
2. Garcia-Prats, J.A. Eventration of the Diaphragm in Infants. 2011
3. Borruto FA, et al. The thoracoscopic treatment of congenital diaphragmatic eventration in children: lessons learned after 15 years of experience. *Eur J PediatrSurg*. 2014;24(4):328–31.
4. Wu S, Zang N, Zhu J, et al. Congenital diaphragmatic eventration in children: 12 years'experience with 177 cases in a single institution. *J Pediatr Surg.* 2015;50:1088–92.
5. Barakat, N.A., Maaty, S.H. and Al-Koly, A. Outcome of Congenital Diaphragmatic Defects: 3 Years Experience. *International Journal of Academic Research*. 2010; 2:183-187.
6. Becmeur, F., Talon, I., Schaarschmidt, K., Philippe, P., Moog, R., Kauffmann, I., Schultz, A., Grandadam, S. And Toledano, D. Thoracoscopic Diaphragmatic Eventration Repair in Children: About 10 Cases. *Journal of Pediatric Surgery*. 2005;40:1712-1715. http://dx.doi.org/10.1016/j.jpedsurg.2005.07.008

Table 1: Differentiating features between congenital diaphragmatic eventration and congenital diaphragmatic hernia

Differentiating feature	**CDE**	**CDH**
Continuity of the diaphragm	Continuous with abnormal contour	Disrupted continuity and attachment to costal margin
Predisposition for side	Complete eventration is common on left side	Most common is left sided postero-lateral defect (Bochdalek)
Age at diagnosis	Usually an incidental finding	Antenatally or soon after birth
Clinical Presentation	Most cases are asymptomatic	Life threatening respiratory distress requiring ventilatory support
Pulmonary Hypoplasia	No pulmonary hypoplasia	Associated with significant pulmonary hypoplasia
Chest X-ray	Dome appears uniform, smooth and intact	Sac filled with bowel loops seen in chest cavity with mediastinal shift
Associated Anomalies	Usually an isolated anomaly, rarely associated with other defects or congenital infections, may be associated with phrenic nerve palsy, ipsilateral horner's syndrome, ptosis	Associated with chromosomal, non- chromosomal syndromes, malformation sequence, non-syndromic. Multiple anomalies, most common are cardiovascular, urogenital, musculoskeletal and CNS malformation
Management	Plication of the diaphragm	Surgical correction of the defect itself
Timing of surgery	Can be elective and later during infancy	Early neonatal period after initial stabilisation
Prognosis and Outcome	Overall outcome is very good	Significant short-term and long-term morbidities and risk of mortality is high

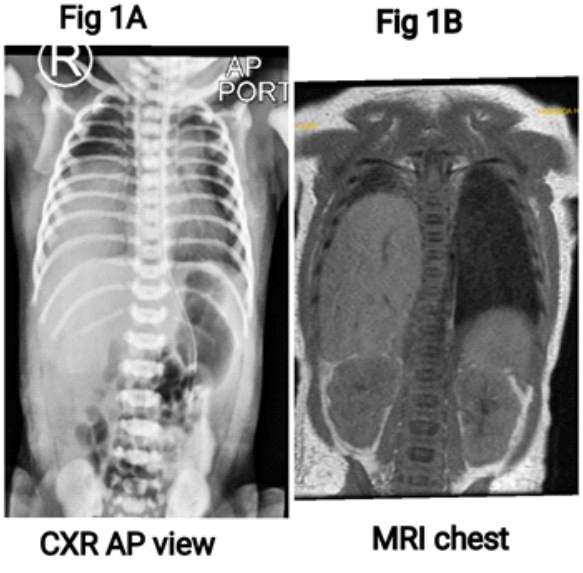


Fig. 1:
1A- Chest radiograph anteroposterior (AP) view showing elevated right dome of diaphragm reaching the level of the fourth rib posteriorly.
1B- Magnetic resonance imaging (MRI) chest revealed that right hemidiaphragm was raised as compared to the left side with a smooth contour and there was no herniation of abdominal contents, confirming the Right CDE

Journal Scan

Reviewed by
Dr Ashwin Vekhande
NNF Fellow, Department of Neonatology
Madhukar Rainbow Children's Hospital, Delhi.

JAMA Pediatrics | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Noninvasive High-Frequency Oscillatory Ventilation vs Nasal Continuous Positive Airway Pressure vs Nasal Intermittent Positive Pressure Ventilation as Postextubation Support for Preterm Neonates in China
A Randomized Clinical Trial

Xingwang Zhu, MD; HongBo Qi, MD; Zhichun Feng, MD; Yuan Shi, MD, PhD; Daniele De Luca, MD, PhD; for the Nasal Oscillation Post-Extubation (NASONE) Study Group

JAMA Pediatrics June 2022, Volume 176, Number 6

Research question

What is the best noninvasive ventilation mode to reduce post-extubation invasive respiratory support in preterm neonates?

Hypothesis

Population	Neonates between 25 to $32^{+}6$ weeks deemed suitable for extubation
Intervention	Nasal high frequency ventilation
Control	Nasal IPPV and Nasal CPAP
Outcome	Total duration of IMV during NICU stay, need for reintubation and ventilator free days

Methods:

Study site – 69 referral tertiary care NICUs in China

It's an assessor-blinded, multicentric, three arms parallel randomized control trial.

Inclusion Criteria:

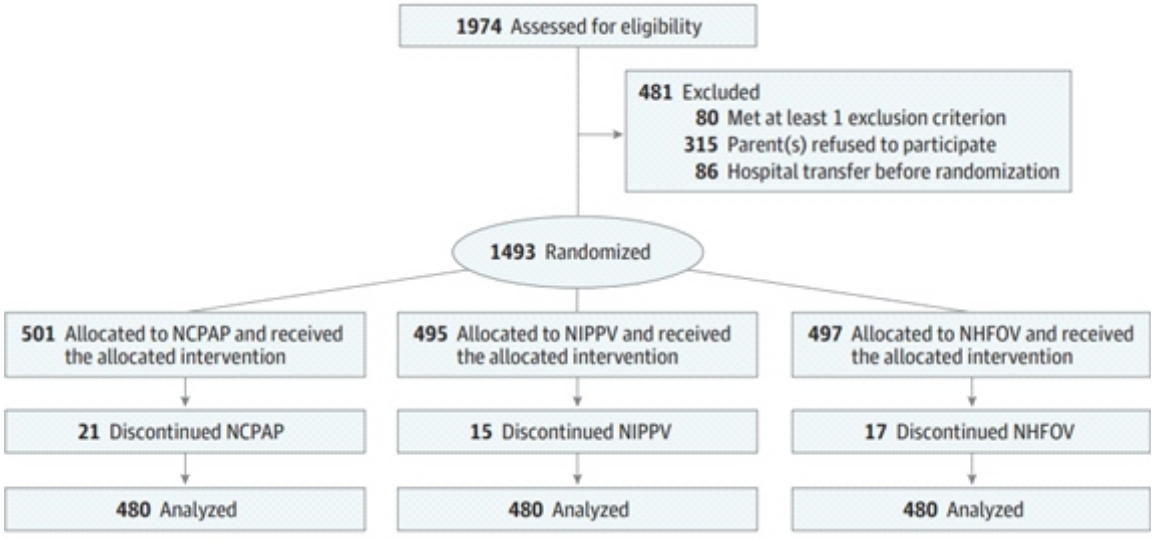
i) Gestational age between 25^{+0} and 32^{+6} weeks
ii) Assisted with any endotracheal intubation
iii) Post-conceptional age less than 36 weeks
iv) Ready to be extubated for first time

Exclusion criteria:

i) Major congenital anomalies or chromosomal abnormalities
ii) Neuromuscular disease
iii) Upper respiratory tract abnormalities
iv) Need for the surgery known before first extubation
v) Grade IV IVH has occurred before first extubation
vi) Birth weight less than <600 gm
vii) Suspected congenital lung disease or malformation.

Primary outcome – Total duration of IMV during NICU stay

Sample size calculation: Considering a 20% difference in duration of mechanical ventilation with an alpha error of 0.05(with Bonferroni correction at 0.017) and power of 95%, 480 neonates were needed in each arm (with a 1:1:1 design). A total of 1440 neonates were planned to be enrolled. Sample size estimation was performed in software GPower 3.1.9.3.



Results:1440 neonates were analyzed.

Total Duration of IMV was longer in NCPAP (mean difference 1.2,95%C.I.>0.001-2.3 days, P=0.04) and NIPPV (mean diff1.5 days;95% C.I.0.3 to 2.7 days) compared to NHFOV. No significant difference between the NIPPV and NCPAP groups.

The frequency rates of reintubation and reintubation within 48 hours were higher in the NCPAP ad compared to the NHFOV group (risk difference 12.5% 95% CI 7.5%- 17.4%, p<0.01) and NIPPV group (risk difference:8.1%, 95%CI 2.9%-13.3%; p=0.003). No difference between the NIPPV v/s NHFOV group.

Ventilator-free days in NCPAP group v/s NIPPV group significantly lower (median 25th – 75th percentile) difference -3(-6 to -1) days, p=0.01.

Secondary outcomes: Use of postnatal steroids is less in the NHFOV group compared to the CPAP group (risk difference 7.7; 95%C.I.,2.6%-12%; p=0.02).

Reviewers comment:

This is one of the largest clinical trial in preterm neonates comparing NCPAP, NIPPV and NHFOV post-extubation. Analysis based on intention to treat approach. There was 20 % reduction in IMV on NHFOV compared to NCPAP and 15% compared to NIPPV respectively. Reintubation with NHFOV is significantly less compared to NCPAP. Although results were not statistically significant reintubation and ventilator-free days are less in NHFOV compared to NIPPV.

Nasal HFOV may come out as a promising therapy post-extubation in preterm infants for preventing extubation failure.

Strengths:

Large sample size. Rigorous design. Well defined primary and secondary outcomes. Interim analysis was done after 50% enrollment.

Limitations: The blinding process was imperfect. Higher NCPAP levels were not achieved. Synchronized NIPPV was not been provided in every set-up. Very small neonates were excluded from the study as resuscitation depended on parent's wishes.

Gunjan Mishra
Senior Consultant Neonatology
Cloud Nine Hospital, Patparganj, Delhi.

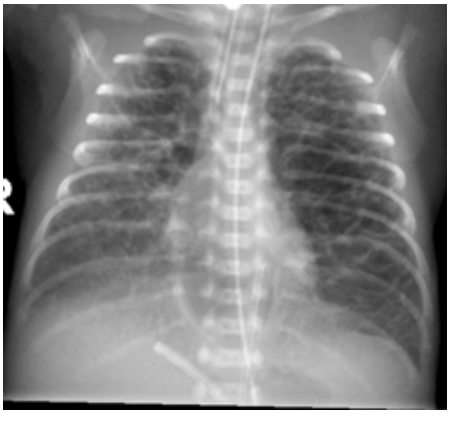
Question 1

A 34-week-old baby referred at 12 hours of life with increasing respiratory distress. X-ray chest showed bilateral pleural effusion. ICD was inserted which drained yellowish-orange fluid.

a. What is the most likely diagnosis?
b. Name some syndromes associated with this condition
c. What is the diagnostic criterion for pleural fluid analysis?
d. What is the initial line of management?
e. Name one pharmacological option available.
f. What are the complications associated with this condition?

Question 2

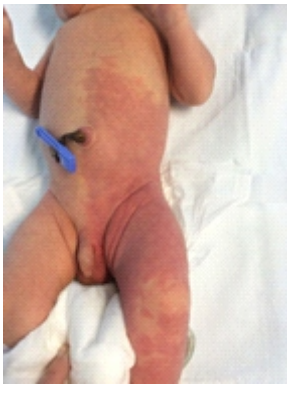
A 26-week neonate who received two doses of surfactant still needing mechanical ventilation on day 4 of life had worsening hypoxia and hypercarbia on ABG. An Xray was done which is as below



a. What is the finding in Xray?
b. What is the management?
c. What are anticipated complication

Question 3

A 38 week male newborn on first physical examination showed a port wine stain affecting abdomen, back and left limb and hypertrophy of affected limb. A biopsy was performed and histological findings revealed capillary malformations



a. What is the most likely diagnosis?
b. What is the triad of this syndrome?
c. what are the possible associated complications?
d. Name few syndrome associated with hypertrophy of limb

Question 4

Prevention of Mother-to-Child Transmission of HIV (PMTCT)

a. What is the risk of transmission of HIV from mother to child if baby is on ART and breastfeeding and when baby is not on ART and breastfeeding respectively?
b. What does EMTCT refer to in this context?
c. How do we do HIV risk assessment of infant born to HIV infected mother

d. How do we decide ARV prophylaxis in both the risk categories and how do we decide the duration

Question 5

Parent of a 2 week old neonate brought their baby to hospital with complaint of bilious vomiting and inconsolable crying. Xray was ordered which was non specific but the symptom persisted so a USG abdomen was ordered.

a. You as an attending neonatologist is suspecting malrotation. So, what all should be looked for while doing USG

b. What are the findings of malrotation in upper GI series

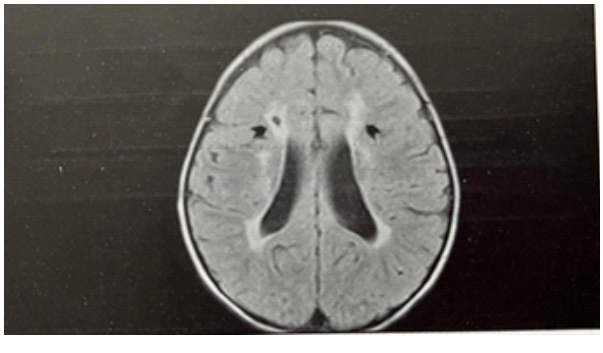
c. Why barium enema should not be used for diagnosis of malrotation?

d. Name few anomalies associated with intestinal malrotation

e. What is nonrotation?

Question 6

Below is the T1 weighted image of preterm baby born at 26 weeks gestational age. Present PMA at time of MRI is 36 weeks. Please answer following questions.



a. What are the findings of MRI?

b. What is the diagnosis?

c. Enumerate a few underlying causes that may lead to periventricular echogenecities in preterm baby

d. What is the likely prognosis /long-term outcome of the patient?

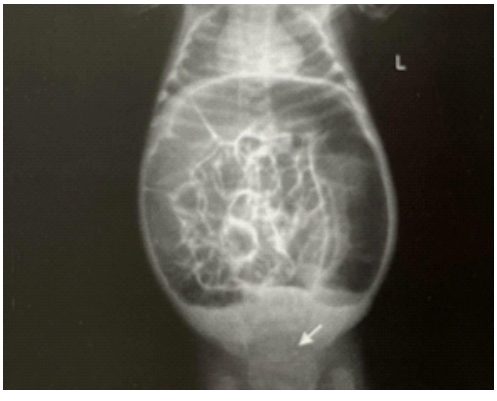
Question 7

a. A term born baby presented on day 14 of life with bilious vomiting, abdominal distension and non passage of stool for last 72 hours. Also baby had a history of delayed meconium passage noted at birth. Depicted below is xray abdomen erect AP view. What is the most likely diagnosis?

b. What are the associated anomalies with the condition?

c. What are the differential diagnosis ?

d. How would you confirm the diagnosis?



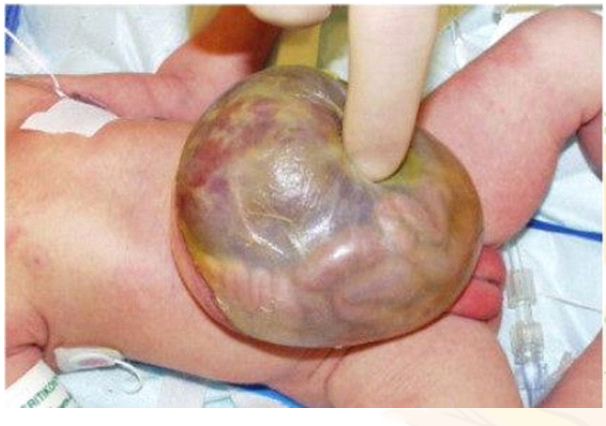
Question 8

a. A female neonate is born with this large abdominal mass in relation to umbilical cord (Below mentioned image). What is the likely diagnosis?

b. List 3 anomalies associated with this condition.

c. Prenatal diagnosis of omphalocele can be done reliably after 12 weeks of PMA. In which specific condition the diagnosis can be confirmed before this?

d. What is OEIS complex?



Question 9

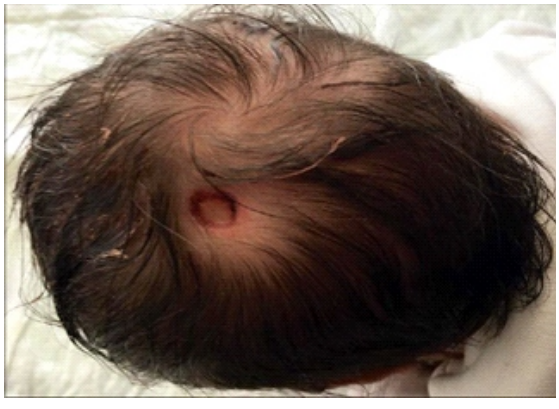
A tem baby is born to primigravida mother. On examination a defect is present in scalp (Below mentioned figure).

a. Identify the condition

b. In utero exposure to which drug can lead to this condition

c. List 3 syndromes which can be associated with this condition

d. What is the treatment of this condition?



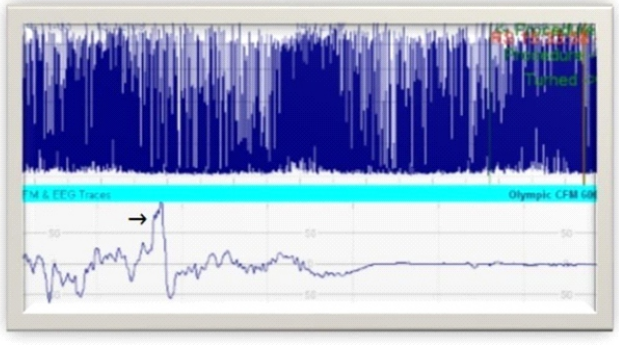
Question 10

Below mentioned is the amplitude integrated EEG trace in a term neonate at 6 hrs of life with history of delayed cry and extensive resuscitation at time of birth.

a. What is the abnormality seen in this aEEG ?

b. What are the effects of drugs (anticonvulsants/opiods/benzodiazepine) on aEEG ?

c. What are the features showing good and bad prognosis on aEEG ?



Answer 1

a. Neonatal chylothorax. Chylothorax in neonates results from the accumulation of chyle in the pleural space. Congenital chylothorax is most likely due to abnormal development or obstruction of the lymphatic system and is often associated with hydrops fetalis. It can be idiopathic or may be associated with various chromosomal anomalies.

b. Trisomy 21, Turner syndrome, Noonan syndrome

c. Elevated triglyceride levels in the pleural fluid above the established cut-off limit of 110 mg/dL and elevated lymphocytes (>1000 cells/µL) on the fluid microscopy.

d. The use of formula with a high concentration of MCT and a low concentration of long-chain fatty acids has been shown to be effective in decreasing chyle flow with resolution of chylous effusion. Fat-free human milk produced by centrifugation and supplemented with MCT can also be used. In case of persistent chylous leak TPN can be started.

e. Octreotide. It is a synthetic analogue of somatostatin, a regulatory hormone that reduces intestinal blood flow and can decrease the production rate of chyle. Usage is reserved in refractory cases of chronic neonatal chylothorax after a thorough discussion with the parent/caregiver of the infant regarding the uncertainty of risk and benefits.

f. Because chyle contains significant amounts of protein, ongoing drainage can lead to **significant loss of albumin, coagulation factors and immunoglobulin**. Also, **profound cellular deficiency** occurs with prolonged chylous drainage resulting in lymphocyte depletion and thus significant immunodeficiency

Answer 2

a. Pulmonary interstitial emphysema.

Bilateral (whole left lung, right upper lobe) increased lucency with streaky opacification. Appearances suggestive of pulmonary interstitial emphysema. Pulmonary interstitial emphysema (PIE) consists of air trapped in the perivascular tissues of the lung. This results in decreased compliance and overdistention of the lung. The interstitial air also compresses airways, resulting in increased airway resistance.

b. There is no definitive treatment for PIE. Management is supportive and directed at providing adequate gas exchange and minimizing the risk of further air leak. This is best accomplished by decreasing the mean airway pressure as much as possible, which is achieved by reducing the peak inspiratory pressure and inspiratory duration. The Fio2 should be increased to compensate for the decreased mean airway pressure. High frequency ventilation is often used in infants with PIE to avoid large cyclic swings in tidal volume, although trials of this intervention are not available. Supportive management of unilateral PIE includes positioning the infant with the affected side down promoting aeration of the unaffected lung and reduces aeration of the lung with PIE, resulting in resolution of the PIE

c. Overdistention of the lung may cause vascular compression, resulting in decreased venous return and impaired cardiac output. PIE may precede the development of pneumothorax, Pneumopericardium or pneumomediastinum

Answer 3

a. Klippel-Trenaunay syndrome

b. Klippel-Trenaunay syndrome (KTS) is a complex congenital disorder defined as the triad of capillary malformation, venous malformation, and limb overgrowth, with or without lymphatic malformation.

c. Possible associated complications include risk of bleeding, thromboembolism, limb length discrepancy, chronic lymphedema and cellulitis

d. CLOVES (congenital lipomatous overgrowth, vascular anomalies, epidermal nevi, and scoliosis/spinal deformities) syndrome, Proteus syndrome, Park weber syndrome.

Answer 4

a. 15 -25% and 30-45%

ARV intervention	
No ARV and breastfeeding	30 to 45%
No ARV, no breastfeeding	20 to 25%
Short course with one ARV , breastfeeding	15 to 25%
Short course with one ARV , no breastfeeding	5 to 15%
Short course with two ARV , breastfeeding	5%
3 ARVs with breastfeeding	2%
3 ARVs with no breastfeeding	1%

b. Elimination of Mother to Child Transmission (EMTCT)

Targets for process indicators for validation of EMTCT (to be maintained for at least 2 years) are as follows:

i. >95% of all estimated pregnant women are registered for antenatal care and receive at least 1 antenatal care check-up

ii. >95% of all estimated pregnant women are tested for HIV

iii. >95% of all HIV positive pregnant women are on ART

c. HIV risk status

Low risk: Infants born to mothers with suppressed viral load (<1000 copies/ml) done any time after 32 weeks of pregnancy up to delivery

High risk: Infants born to HIV-positive mother not on ART

Maternal viral load not done after 32 weeks of pregnancy till delivery

Maternal viral load not suppressed between 32 weeks of pregnancy till delivery

Mother newly identified HIV positive within 6 weeks of delivery

d.

Low risk infant	High risk infant
Syrup Nevirapine (NVP) or Syrup Zidovudine# (in situations where NVP will not be effective):	Dual prophylaxis: Syrup NVP + Syrup Zidovudine
a) Infant born to a mother with confirmed HIV-2 or HIV-1 and HIV-2 combined infections	Duration of Dual ARV Prophylaxis: a) In case of Exclusive Replacement Feeding (ERF): From birth till 6 weeks of age
b) Infant born to a mother who had received single dose of NVP during earlier pregnancy or delivery	b) In case of Exclusive Breastfeeding (EBF):
c) Infant born to a mother who is on PI-based ART regimen due to treatment failure	
Duration of ARV prophylaxis: From birth till 6 weeks of age	From birth till 12 weeks of age

Answer 5

a.

- Third part of the duodenum is not in the normal retromesenteric position (ie, located between the mesenteric artery and the aorta in the retroperitoneal space)
- Abnormal position of the superior mesenteric vein (either anterior or to the left of the superior mesenteric artery [SMA]) [the superior mesenteric vein is normally located to the right of the SMA]
- The "whirlpool" sign of volvulus (caused by the vessels twisting around the base of the mesenteric pedicle)
- Dilated duodenum (indicating duodenal obstruction by Ladd bands)

b.

- A clearly misplaced duodenum with the ligament of Treitz on the right side of the abdomen
- A duodenum with a "corkscrew" appearance
- Duodenal obstruction, which may appear similar to that seen with duodenal atresia (dilated stomach and proximal duodenum or may present with a "beak" appearance if a volvulus is present.

c. Barium enema may be misleading and should not be used for the diagnosis of malrotation. Because the final fixation of the colon does not occur until near term, many newborns have a high or poorly-fixed cecum, which can mimic malrotation and result in a false-positive study Conversely, in approximately 20 percent of cases of malrotation, the cecum is normally located in the right lower quadrant (false-negative)

d. Congenital diaphragmatic hernia, Heterotaxia syndrome, Omphalocele and intestinal atresias can be associated with concurrent malrotation

e. During the fourth to eighth week of embryonic development, the embryonic coelom, or cavity, cannot accommodate the rapidly expanding gastrointestinal (GI) tract. As a result, the primary intestinal loop buckles into the area of the yolk stalk, which will be the future umbilicus. The axis of this loop is the developing superior mesenteric artery (SMA). As the primary intestinal loop buckles out of the abdomen, it begins the normal rotation of the bowel by twisting 90 degrees counterclockwise.The primary loop continues to grow, and then returns to the abdomen during the 8th to 10th week of gestation. With the return to the abdomen, there is an additional 180 degrees counterclockwise rotation. The overall effect is that the bowel rotates 270 degrees counterclockwise from the original primary loop.

   If both limbs of the primary loop return to the abdomen with no further rotation, nonrotation occurs. In this condition, the small bowel is located on the right of the abdomen and the colon on the left. Nonrotation is not as dangerous for the patient as malrotation because, in general, the base of the mesentery is wider than in malrotation, and the risk of volvulus is less.

Answer 6

a. Mild prominence of both the ventricles with periventricular FLAIR hyperintensity. Periventricular cystic change is also seen

b. Periventricular leucomalacia

c. Congenital malformation of the brain / metabolic insult of the brain / congenital infections like CMV, toxoplasma / periventricular hemorrhagic infarction/ choroid plexus cyst/ porencephaly

d. Major long term morbidity in infants with PVL is spastic diplegia. More severe lesions with extension to corona radiata are associated with visual and cognitive deficits

Answer 7

a. Large bowel obstruction mostly hirschprung disease or colonic aganglionosis

b. Congenital anomalies of the kidney and urinary tract (CAKUT), including hydronephrosis and renal hypoplasia, Ophthalmologic abnormalities and hearing impairment are fairly associated with the condition, so need to be ruled out

c. Intestinal atresias, malrotation, meconium plug syndrome and lazy colon syndrome

d. The diagnosis of HD is established if ganglion cells are absent in the rectal biopsy, provided that the tissue sample is adequate. Supportive findings include the presence of hypertrophic nerve fibers, increased acetylcholinesterase activity or staining in the muscularis mucosae, and decreased or absent calretinin-immunoreactive fibers in the lamina propria . Excessively thickened nerve fibers may not appear until after eight weeks of age.

   A suction rectal biopsy can be done at the bedside or in an ambulatory setting without the need for general anesthesia. A biopsy should be taken 2 cm above the level of the dentate line to avoid the physiologic 1- to 2-cm hypoganglionic or aganglionic zone that is normally present. A second biopsy should be taken proximal to the first one.

Answer 8

a. Omphalocele – herniation or protrusion of the abdominal contents into base of umbilical cord.

The sac is covered with peritoneum. Herniation of intestine into cord occurs in 1/5,000 births and herniation of liver and intestines occurs in 1/10,000 birth

b. Many infants with omphalocele (50–70%) have associated malformations and about 30% have chromosomal abnormalities.

Beckwith Weidmann syndrome (Omphalocele, Macrosomia and hypoglycemia)) Cardiac anomalies (TOF, ASD) Chromosomal abnormalities (various trisomies), genitourinary anomalies, diaphragmatic hernia

c. The sonographic diagnosis of a liver-containing omphalocele can be made before the 12th postmenstrual week because herniated liver is never a normal developmental finding.

d. OEIS complex comprises a combination of defects including omphalocele, exstrophy of the cloaca, imperforate anus, and spinal defects

Answer 9

a. Aplasia cutis congenita

b. In utero exposure to antithyroid drugs (methimazole/ carbimazole) can lead to aplasia cutis congenita

c. Syndromes associated with aplasia cutis are congenita – Adams – Oliver syndrome, Oculocerebrocutaneous syndrome, Johanson – Blizzard syndrome, 4p (-) microdeletion syndrome, Trisomy 13-15.

d. For small lesions, less than 4 cm without additional findings, daily cleansing of the lesion with topical antibacterial ointment is recommended until healing is complete. Lesions typically heal within a few weeks to a few months with an atrophic, hairless scar. Larger lesions greater than 4 cm are more commonly associated with underlying defects and are at increased risk of complications, including hemorrhage, venous thrombosis, and infection. Early surgical repair is recommended to avoid these complications.

Answer 10

a. Burst-suppression: It is seen in moderate or severe injuries. The longer it takes, worse the prognosis. Short-duration high-voltage bursts occur on a low-voltage trace that produce wide bands where the lower limit can be < 5 µV and the upper limit can exceed 10 µV

b. Several drugs affect the background of aEEG amplitude. Sedatives like chloral hydrate, opiates and anticonvulsant drugs like phenobarbital and benzodiazepines temporarily suppress the EEG. Fentanyl and high dose midazolam treatments can cause deep depression in aEEG. Therefore, aEEG assessment made within 30-60 minutes after drug exposure may result in misinterpretation of the encephalopathy

c. It has been reported that aEEG is a good tool as other clinical, radiological and neurophysiological evaluations in terms of predicting neurodevelopmental outcomes in newborns with HIE.

Features showing good prognosis:

1. Presence of normal background activity pattern
2. Even if the aEEG trace initially is abnormal, return to normal within 48 hours during cooling and within 24 hours during normothermia
3. The presence of sleep wake cycles (SWCs) or the beginning of SWCs within the first 36 hours after birth.

Features showing poor prognosis:

1. Abnormal background pattern
2. Interrupted pattern and low voltage
3. An amplitude of < 5 µV within the first postnatal 3 days
4. Absence of SWC
5. Status epilepticus
6. Prolonged and marked moderate to severe voltage disturbances.

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:

info@nnfdelhi.org
drgupta.naveen@gmail.com

Please call or Whats App at 9811758133