May 2023

DPT

DEVELOPMENTAL PEDIATRICS TODAY

Monthly e-Newsletter of IAP Chapter of Neurodevelopmental Pediatrics

Inside

Editorial

Respected seniors and dear friends,

Greetings from the Neurodevelopmental chapter of IAP!

May 15th marks the International Kangaroo mother care(KMC) awareness day. Kangaroo mother care refers to the practice of providing continuous skin-to-skin contact between the mother, father or any other family member and the baby. KMC, especially with mothers promotes breastfeeding and early discharge from the hospital. Further benefits of kangaroo care are that mom has better lactation and a better chance of breastfeeding. Baby stays warmer, body temperature is better and has improved heart rate, respiratory rate and improved weight gain. Baby cries less, has lower stress levels and has improved sleep.

There are some interesting case reports in this issue and journal scan on KMC.

Our sincere request to all of you especially fellows to send us case studies and writeups related to neurodevelopment.

Happy reading.

Long live IAP and our Chapter!

Dr. Lata Bhat

Chief Editor

Chairperson's Message

Dear Readers,

Summer greetings to all of you !

As the warm sun is spreading its energy, flowers blooming in vibrant colours and butterflies hovering around they bring good news from everywhere. They also bring the good news of the holding of the 20th Conference of our Chapter in Guwahati on 6-8 October this year.

Two decades of relentless work of our dedicated members have paved the path towards new thoughts and better understanding in the field of neurodevelopmental pediatrics. This year's Conference focusses on the 5 Ps : Parent and Primary givers Proactive Partnership with Pediatricians for childcare. We look forward to your participation in the Conference in October so that we can empower people who matter the most, and each one of us become an agent of change for a better inclusive world.

This month also celebrates 'Kangaroo Mother Care'. It amplifies the warm relationship and scaffolding that is required lifelong. This issue has interesting dimensions on understanding neurodevelopmental problems.

Happy reading !

See you soon in Guwahati !

Dr. Shabina Ahmed MD, FIAP

National Chairperson

Neurodevelopmental Pediatrics Chapter of IAP

Snippets from the Secretary

Respected Seniors and dear friends,

Greetings from the Neurodevelopmental chapter.

Hope this issue of newsletter find you all and your families in good health.

The chapter is having its 20th national conference at Guwahati this year in the first week of October in association with IAP Assam branch and we look forward to having a great academic and cultural feast in natures lap at Guwahati. I urge the chapter members to register and participate in large numbers to make the conference to make it a big success.

Fellowship program of the chapter under the aegis of IAP has been flourishing well and we are entering into the ninth year this year. We are hoping to add few more centres for fellowship this year. With increasing recognition of neurodevelopmental disorders in the community, there is a strong need to increase the number of trained personnel working in the field and the chapter is striving hard in this direction.

The chapter's quarterly journal of developmental and behavioral pediatrics is now out with second issue and is being appreciated by all. Members can submit their scientific papers and case reports to the journal for publication.

May month has many important health days - Kangaroo care day, International Women Health Day to name a few. We have some interesting articles in the journal scan section.

Happy reading and stay healthy and stay safe. Jai Hind! Jai IAP !

Jai Hind! Jai IAP !

Wg Cdr (Dr) KS Multani

National Secretary

IAP Chapter of Neurodevelopmental Paediatrics

Case Report

Does Face Predict The Brain? – A Case Series on Subtypes of Holoprosencephaly with Varied Facial Features

ABSTRACT:

Holoprosencephaly (HPE) is a complex brain malformation characterized by a failure of the forebrain (prosencephalon) to separate completely into two distinct cerebral hemispheres, a process normally complete by the fifth week of gestation. HPE typically is divided into four subtypes based on the degree of nonseparation of the prosencephalon.

"The face predicts the brain approximately 80% of the time," refers to the observation that the degree of facial malformation frequently reflects the degree of brain malformation. In the present case series it was observed that the facial features were not consistent with the severity of the cerebral malformation noticed on the neuroimaging.

Keywords: holoprosencephaly, subtypes, neuro imaging, facial features

CASE SERIES:

ALOBAR HOLOPROSENCEPHALY:

There were two children who had alobar HPE on MRI with varying facial features.

Child AL:

Child AL(fig.1) was a 1.5 years old female child presented with global developmental delay. She had macrocephaly, hydrocephalus and subtle facial dysmorphisms like depressed nasal bridge, hypotelorism. Neurologically she had bipyramidal signs, brisk reflexes. She had seizures and her hearing and vision were preserved. Her genetic evaluation including Chromosomal microarray and Whole exome sequencing were normal.

Child OB:

Child OB (fig.1) is an infant with global delay and spastic quadriparesis. His facial features included synophrys, depressed nasal bridge, hypotelorism, midline cleft lip and palate. He didn't

have seizures, hearing or visual impairments. His karyotyping was normal. Unfortunately, he succumbed to probable aspiration. The neuroimaging was similar to the first child.

Figure 1

Child AL Child OB

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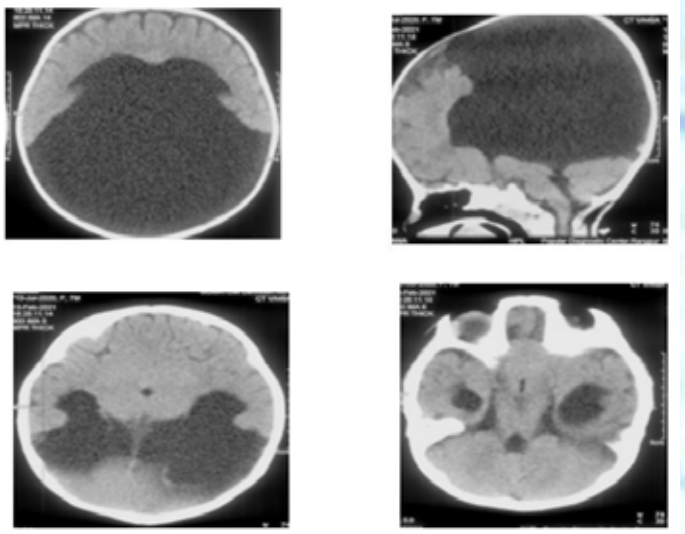
Fig 2: MRI features of alobar HPE



MRI features of alobar HPE (fig.2) shows absence of interhemispheric fissure, single ventricle and hydrocephalus.

SEMILOBAR HOLOPROSENCEPHALY:

There were two children semilobar HPE with similar radiological features but had different facial and neurological features.

Child SM:

Child SM (fig. 3) was a 3rd born child to nonconsanguineous couple with uneventful antenatal and perinatal periods. He presented with global developmental delay, microcephaly. His facial features included triangular face, flat nasal bridge, prominent skin tag over nasal bridge and high arched palate. Neurologically he had spastic diplegia, oromotor insufficiency. His vision and hearing were preserved and there were no seizures. His chromosomal microarray showed trisomy 5q32. Whole exome sequencing is awaited.

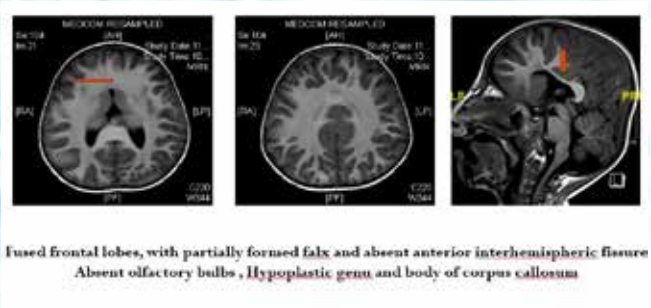
Child EI:

Child EI (fig. 3) was a 6 years old girl who had predominant motor delay, normal facial features, microcephaly and spastic diplegia. There were no seizures or oromotor insufficiency. The MRI of both the children was similar.

Figure.3

Radiological features of semilobar HPE have been shown in Fig 4.

Fig 4: MRI of semilobar HPE



LOBAR HOLOPROSENCEPHALY:

There were 3 children with lobar HPE with varying facial, clinical and developmental profiles yet had similar radiological features.

Child LA:

Child LA (Fig.5) was a 2 year old female child with global developmental delay. She had microcephaly, facial features included deep set eyes, low set ears, depressed nasal bridge and hypotelorism. She had a Solitary median maxillary central incisor (SMMCI). There were no seizures. Neurologically she had hypotonia with brisk reflexes. Ultra sound abdomen revealed absent left kidney. Screening

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Echocardiogram was normal. Her Karyotype showed 45, XO, which was suggestive of Turner syndrome. Since her phenotypic features were not correlating, Whole Exome sequencing is planned.

Child BO:

Child BO (Fig.5) was a 7 months old infant, whose mother also had midfacial hypoplasia and was operated for cleft palate and lip. He was born at term, by C- section, with birth weight of 2.2 kg. He had global developmental delay, microcephaly and normal tone. His facial features

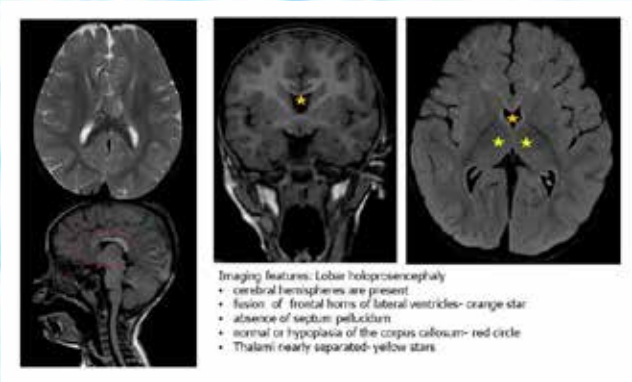
showed nasal bone hypoplasia, midline cleft lip and palate with feeding challenges. He had moderately severe hearing loss. There were no seizures. Genetic evaluation was suggested.

Child HP:

Child HP(Fig.5) was a 6yr old boy, who had a stormy perinatal period. His mother had preeclampsia, antepartum hemorrhage and he was delivered at term, by C section, with birth weight of 2.5 Kg. He had poor feeding, respiratory distress requiring Oxygen due to meconium aspiration. He had global developmental delay, microcephaly and Seizures. His facial features include synophrys, wide philtrum and depressed nasal bridge and hypotelorism. Neurologically he had spasticity and brisk reflexes. He had associated comorbidities like Gastro Esophageal Reflux Disease, persistent hypernatremia and chronic renal disease. His Ultrasound abdomen revealed smaller left kidney. There was Grade III Vesico ureteric Reflux on Lt side on MCU. His Karyotype was normal. Genetic evaluation including WES is planned.

Figure 6

Figure 6: MRI features of lobar HPE



MIDDLE INTERHEMISPHERIC VARIANT / SYNTELENCEPHALY

Child SN was a 2.5 yr old girl who was brought with delay in the motor domain. Her language and socio adaptive skills were reasonably fair. She had microcephaly. There was no facial dysmorphism. Neurologically her tone was increased with exaggerated deep tendon reflexes. She had drooling, but there was no history of feeding difficulties. Hearing and vision were preserved. EEG was normal and MRI of the brain(fig.7) showed fusion of the hemispheres in the posterior frontal and parietal regions with formation of the interhemispheric fissure in the anterior frontal and occipital lobes. A normal appearing genu and splenium with absence of the body of the corpus callosum was noted.

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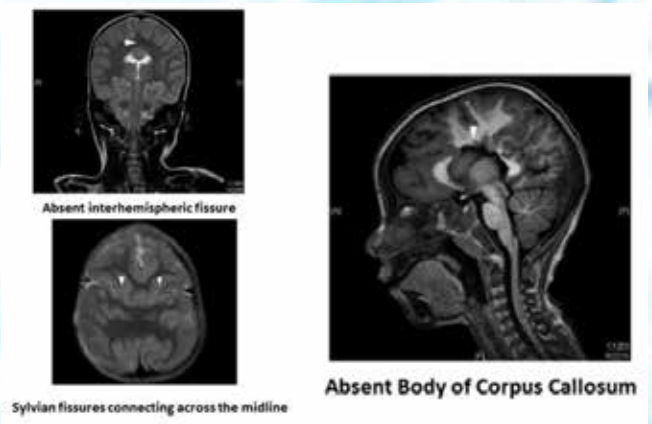
Figure 7: MRI features of Middle Interhemispheric Variant



DISCUSSION :

Holoprosencephaly (HPE) is the most common structural anomaly of the developing forebrain, resulting from incomplete midline cleavage of the prosencephalon and associated with neurologic impairment and dysmorphism of the brain and face. Studies suggest that the defects associated with HPE occur at approximately two to four weeks post-conception1, indicating that HPE is a disorder of gastrulation.

EPIDEMIOLOGY:

HPE is the most common developmental defect of the forebrain and midface in humans and occurs in 1 in 250 conceptuses2, however only 3% of the fetuses with HPE survive to delivery, so the incidence in live births is only approximately 1 in 8,0003.

ETIOLOGY:

The etiology of holoprosencephaly is extremely heterogeneous and is still being elucidated. Prenatal exposure to various environmental factors and teratogens have been implicated, including maternal diabetes (infants born to diabetic mothers have a 200-fold risk of holoprosencephaly), ethanol, cytomegalovirus infection, salicylates, antiepileptic medications, retinoic acid, and maternal hypocholesterolemia4.

GENETIC CAUSES:

Between 18%–25% of live births affected by holoprosencephaly have a recognizable monogenic syndrome, including Smith-Lemli-Opitz syndrome, Pallister Hall syndrome and Rubinstein-Taybi syndrome5.Chromosomal anomalies have been implicated in 24–45% of live births affected by holoprosencephaly, most frequently in chromosomes 13, 18, and 216.

HPE is inherited as an autosomal-dominant disease. Mutations in the following nine genes have been identified in about 28% of HPE cases patients with HPE – sonic hedgehog (SHH), patched homolog 1 (PTCH1), glioma-associated oncogene family zinc finger 2 (GLI2), teratocarcinoma-derived growth factor 1 (TDGF1, also known as CRIPTO), TGF-β–induced factor homeobox (TGIF), forkhead box H1 (FOXH1), zinc finger protein of the cerebellum 2 (ZIC2), SIX homeobox 3 (SIX3), and dispatched homolog 1 (DISP1)7

EMBRYOLOGY OF HPE:

Just before neural tube closure (neurulation) around 4wks, the anterior end of the tube begins to expand forming the three primary brain vesicles, or pouches. The most anterior of these embryonic brain vesicles is the “prosencephalon” which is the embryonic precursor of the forebrain. The middle vesicle is the “mesencephalon” which is the precursor of midbrain structures, and the most posterior is the “rhombencephalon” which will become the hindbrain. These three segments further subdivide and by the end of the embryonic period (7wks) the five secondary brain vesicles are present. The prosencephalon divides into the telencephalon and the diencephalon which further develop into brain structures. Failure of this cleavage and rotation of these primary brain vesicles leads to holoprosencephaly.

The sine qua non of HPE is incomplete cleavage of midline structures involving the telencephalon and diencephalon. DeMyer et al. (1964)8 (fig 8) classified

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the brain anomalies in HPE patients as alobar, semilobar and lobar types, depending on the degree of the midline cleavage of the cerebral hemispheres.

In the most severe type, alobar HPE, nearly complete lack of separation of the cerebral hemispheres is characteristic, with a single midline ventricle very often communicating with a dorsal cyst. The interhemispheric fissure and corpus callosum are completely absent.

In the intermediate form, semilobar HPE, the anterior hemispheres are not separated, but some degree of separation of the posterior hemispheres is seen. The genu and body of the corpus callosum are absent, but the splenium is present. The frontal horns of the lateral ventricles are not developed, but the posterior horns are present.

The mildest form, lobar HPE, is characterized by lack of separation of the most rostral and ventral aspects of the cerebral hemispheres. The splenium and body of the corpus callosum are present, but the genu is absent. Rudimentary frontal horns may be present.

The middle interhemispheric variant, the midportion of the cerebral hemispheres is continuous across the midline, with absence of the corpus callosum seen only in this region. There is separation of the anterior frontal lobes, basal forebrain, and occipital lobes.

"Face predicts the brain"

Face predicts the brain is the maxim coined by DeMyer (1964)8.

Figure 8: DeMyer's classification of HPE (1964)8

DEMYER'S CLASSIFICATION OF HPE

	ALOBAR	SEMILOBAR	LOBAR	MIHV*
Interhemispheric separation	**Complete non-separation** Absent falx cerebri	**No anterior separation,** some posterior separation	Nonseparation only **rostral/ventral frontal neocortex**, with hypoplastic falx cerebri	**Non-separation of posterior frontal and parietal lobes**
				
Corpus callosal characteristics	**Corpus callosum absent completely**		Absent in **affected region**	**Absent BODY** of the corpus callosum
Additional findings	Absent olfactory bulbs, **Fused deep gray nuclei (thalami and caudate)** Absent or hypoplastic olfactory bulbs, Varying degrees of **septum pellucidum** Hypoplastic olfactory bulbs, hypoplastic falx cerebri, and azygous anterior cerebral artery Heterotopias, Cortical dysplasias			

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The brain and face have a common origin. As the brain undergoes its own morphogenesis, the developing face is shaped in response. Both the pattern and the rate of growth of the brain provide unique influences on facial morphogenesis. The neural crest cells that give rise to the facial skeleton originate from the dorsal neural tube, and information about their spatial patterning is based on their anterior-posterior level of origin. Brain serves as a structural platform, and as such imparts physical forces on adjacent tissues that help shape the location and directional growth of facial primordia. The brain and facial tissues communicate via molecular interactions that control the cellular mechanisms responsible for facial morphogenesis9. Thus, the development of the face is highly contingent on normal brain development, and consequently, events and disruptions to the processes that regulate the brain often produce facial malformations

HPE is typically associated with midline facial anomalies which range from ocular hypotelorism, Proboscis, with single nostril in severe alobar HPE to ocular hypotelorism, midline cleft lip, flat nose in the milder forms. The severity of the craniofacial phenotype usually mirrors the severity of the brain malformations and correlates inversely with survival which may be true approximately 80% of the time10. But exceptions are present – For example mutations in ZIC2, severe HPE with extensive neurologic impairment and characteristic clinical sequalae, but have a much milder facial phenotype11.

Along with the facial features children with HPE are frequently found to have myriad of clinical features like tonal abnormalities like hypotonia, dystonia, and/or spasticity. They may have oromotor dysfunction, thirst and appetite disturbances resulting from hypothalamic dysfunction, exacerbate feeding challenges secondary to cleft lip and palate. Due to the nonseparation of thalamus and the presence of a dorsal cyst causes the blockage of CSF egress from the third ventricle and hydrocephalus, seizures. Other complications include recurrent respiratory infections. Due to the medial and rostral location of the hypothalamus, nonseparation of the hypothalamus occurs frequently, leading to a variety of issues involving homeostatic dysfunction

leading to temperature instability, and dysfunction of thirst, appetite, sleep-wake cycles and electrolyte imbalance, as well as hypothalamic-pituitary endocrine dysfunctions like central diabetes Insipidus, hypothyroidism, hypocortisolism, Growth Harmone deficiency, and multiple pituitary hormone deficiency12

CONCLUSION:

HPE can present with wide spectrum of phenotype and facial features always may not always correlate with brain malformation. Severe forms can present with mild facial phenotype while there may be severe facial defects in not so severe forms. Each child needs a detailed history and clinical examination, neuroimaging and genetic evaluation and relevant investigations to understand the entirety of the disorder

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Case Report

Noonan Syndrome

ABSTRACT:

Noonan syndrome (NS) is one of the most common RASopathies that result from mutations in genes in the RAS / mitogen-activated protein kinase (MAPK) pathway, with mostly an autosomal dominant inheritance. It is characterized by typical facial features, congenital heart defects, cryptorchidism and short stature. An increased risk of different types of malignancy, mainly hematologic, has been reported in patients with NS compared with the general population. Hence along with the neurodevelopmental, systemic, visual and audiologic assessments, careful screening including radiological and hematological evaluation is needed to rule out malignancies.

CASE REPORT:

7.8 years old boy, Master A, was brought with concerns of delayed developmental milestones since infancy, short stature. He was detected to have heart disease at local center and brought here for further evaluation. He is the firstborn child to non-consanguineous parents. Antenatal scans reportedly showed polyhydramnios at 7months of gestation. He was delivered at term by C- Section (cord around neck) with normal birth weight. Immediately after birth, he had respiratory distress and was treated as sepsis and meconium aspiration syndrome and was admitted for 1month, and was diagnosed to have congenital heart disease. He was noticed to have atypical facial features since birth. He had delay in all the domains of development since early infancy, however he had caught up, now at present he can pedal a tricycle, can jump from height. He is able to care for self with minimal supervision, however he has few deficits in his communication and cognitive skills. He started going to school lately due to his cardiac problem and has achieved preschool skills.

On examination he was found to have microcephaly (HC<-2SD), weight and height below 5th centile. He had proportionate short stature (<3rd centile). His facial features (Fig 1) include bilateral ptosis, down slanting eyes, low set ears, wide philtrum, depressed nasal bridge, median epicanthal folds, tented upper lip, occipital plagiocephaly, low posterior hair line and webbed neck. His phenotypic features fulfill the scoring system for Noonan syndrome. He had pectus excavatum. His vision and hearing were normal. He had a systolic murmur on auscultation. There were no

neurological deficits and organomegaly per abdomen. He had bilateral cryptorchidism. Ultrasound abdomen showed Right undescended and left intraabdominal testis, with mild splenomegaly and few non necrotic lymph nodes. Routine blood investigations including complete blood picture, thyroid profile, blood sugars, glycosylated Hemoglobin were normal. His vision and hearing were clinically normal. Echocardiogram showed Large Ostium Secundum type of Atrial Septal Defect of 15mm with left to right shunt, with an additional defect near mitral rim, measuring 8mm, left to right shunt, dilated right sided chambers for which he underwent surgery. An elective orchiopexy is planned by the Pediatric surgeons. In view of the distinctive phenotypic features and associated cardiac disease, Noonan syndrome was suspected and genetic evaluation was done. RASopathy Panel showed mutations in PTPN11 gene which was confirmatory of the diagnosis of Noonan syndrome. Parental validation by Sanger sequencing confirmed the heterozygous PTPN11 variant in proband. Parents are wildtype for the variant.

Case Report

DISCUSSION:

Noonan syndrome (NS, OMIM 163950), first described by a Pediatric cardiologist Jacquelin Noonan in 19681, is one of the most common RASopathies that result from mutations in genes in the RAS / mitogen-activated protein kinase (MAPK) pathway, with mostly an autosomal dominant inheritance and an estimated incidence at birth between 1:1,000 and 1:2,5002,3. Different genes

have been implicated, of these, the most commonly mutated gene in NS is PTPN11, with mutation frequency of nearly 50%4. The presence of heterozygous missense point mutations of the PTPN11 gene (12q24) in NS patients has been demonstrated first in 20015. PTPN11 codes for a ubiquitous non-receptor tyrosine phosphatase, the Src homology region 2 (SH2) domain-containing tyrosine phosphatase 2 or SHP2 protein, a member of RAS /MAPK pathway, which is involved in cell proliferation, differentiation, metabolism, apoptosis and cell survival5.

It is characterized by typical facial dysmorphism, congenital heart defects (most commonly pulmonary valve stenosis and atrial septal defects and hypertrophic cardiomyopathy), postnatal growth retardation, thoracic deformity and webbed neck, while neurologic, genitourinary, lymphatic, ocular, and skin findings may also occasionally be present. The distinctive facial features of NS include a broad forehead, hypertelorism, down-slanting palpebral fissures, ptosis of the eyelids, epicanthal folds, and posteriorly rotated low-set ears. The diagnostic criteria6 for NS has been tabulated in table 1.

FINDINGS	A MAJOR	B MINOR
Facial	1A. Typical face	1B. Suggestive face
Cardiac	2A. Pulmonary valve stenosis and/or typical ECG	2B. Other defect
Height	3A. <3rd centile	3B. <10th centile
Chest wall	4A. Pectus carinatum/excavatum	4B. Broad thorax
Family History	5A. 1st degree relative definite NS	5B. 1st degree relative suggestive NS
Others	6A. All three (males): mental retardation, cryptorchidism and lymphatic dysplasia	6B. One of the three

Table 1: Noonan syndrome (NS) major and minor criteria - scoring system (Van der Burgt et al.[6])

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FINDINGS

A MAJOR

B MINOR Facial 1A. Typical face 1B. Suggestive face

Cardiac

2A. Pulmonary valve stenosis and/or typical ECG

2B. Other defect Height 3A. <3rd centile 3B. <10th centile

Chest wall

4A. Pectus carinatum/excavatum

4B. Broad thorax Family History 5A. 1st degree relative definite NS 5B. 1st degree relative suggestive NS

Others

6A. All three (males): mental retardation, cryptorchidism and lymphatic dysplasia

6B. One of the three

Table 1: Noonan syndrome (NS) major and minor criteria - scoring system (Van der Burgt et al.6)

Definite NS: 1A plus one of 2A–6A or two of 2B–6B; 1B plus two of 2A–6A or three of 2B–6B

An increased risk of different types of malignancy, mainly hematologic, has been reported in patients with NS compared with the general population7,8 while solid tumors are less frequent9,10. Among the NS with PTPN 11 gene mutations - soild tumours like Rhabdomyosarcoma, Neuroblastoma, central nervous system tumours were reported including Oligodendroglioma, Dysembryoplastic neuroepithelial tumour of the temporal lobe, Pilocytic astrocytomas, and low-grade astrocytoma and Acoustic neuroma11–13. Two solid tumours of the testis are reported in boys with clinically diagnosed NS, of those, testicular seminoma was diagnosed 14 years after orchiopexy for undescended testis14. Hepatoblastoma was reported in one case of an infant with the germline PTPN11 p.N308D mutation15.

CONCLUSION:

Noonan syndrome is the one of the most common RASopathies, frequently associated with cardiac defects, undescended testes and high incidence of malignant and solid tumors, careful monitoring and screening is mandatory. Since Master A has bilateral cryptorchidism, he needs an orchiopexy and close monitoring for testicular tumors along with his neurodevelopmental and anthropometric assessments. Since the child underwent surgery this visit, the family was suggested to bring him for periodic assessment and monitoring for his neurocognitive skills.

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Journal Scan

Immediate "Kangaroo Mother Care" and Survival of Infants with Low Birth Weight

WHO Immediate KMC Study Group; may 27, 202, N Engl J Med 2021; 384: 2028-2038; DOI: 10.1056/NEJMoa2026486

Abstract

BACKGROUND

"Kangaroo mother care," a type of newborn care involving skin-to-skin contact with the mother or other caregiver, reduces mortality in infants with low birth weight (<2.0 kg) when initiated after stabilization, but the majority of deaths occur before stabilization. The safety and efficacy of kangaroo mother care initiated soon after birth among infants with low birth weight are uncertain.

METHODS

We conducted a randomized, controlled trial in five hospitals in Ghana, India, Malawi, Nigeria, and Tanzania involving infants with a birth weight between 1.0 and 1.799 kg who were assigned to receive immediate kangaroo mother care (intervention) or conventional care in an incubator or a radiant warmer until their condition stabilized and kangaroo mother care thereafter (control). The primary outcomes were death in the neonatal period (the first 28 days of life) and in the first 72 hours of life.

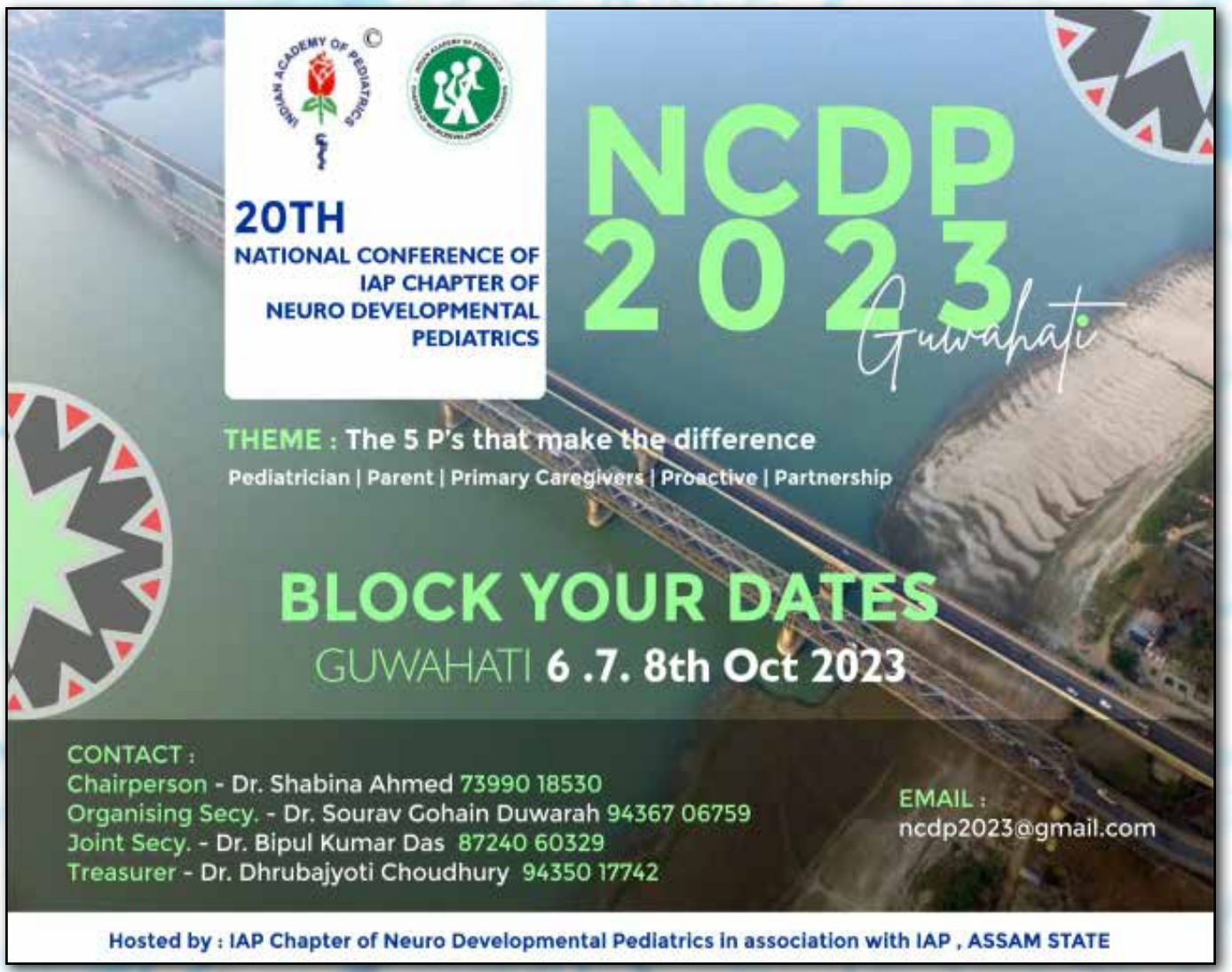
RESULTS

A total of 3211 infants and their mothers were randomly assigned to the intervention group (1609 infants with their mothers) or the control group (1602 infants with their mothers). The median daily duration of skin-to-skin contact in the neonatal intensive care unit was 16.9 hours (interquartile range, 13.0 to 19.7) in the intervention group and 1.5 hours (interquartile range, 0.3 to 3.3) in the control group. Neonatal death occurred in the first 28 days in 191 infants in the intervention group (12.0%) and in 249 infants in the control group (15.7%) (relative risk of death, 0.75; 95% confidence interval [CI], 0.64 to 0.89; P=0.001); neonatal death in the first 72 hours of life occurred in 74 infants in the intervention group (4.6%) and in 92 infants in the control group (5.8%) (relative risk of death, 0.77; 95% CI, 0.58 to 1.04; P=0.09). The trial was stopped early on the recommendation of the data and safety monitoring board owing to the finding of reduced mortality among infants receiving immediate kangaroo mother care.

CONCLUSIONS

Among infants with a birth weight between 1.0 and 1.799 kg, those who received immediate kangaroo mother care had lower mortality at 28 days than those who received conventional care with kangaroo mother care initiated after stabilization; the between-group difference favoring immediate kangaroo mother care at 72 hours was not significant.

Month in pics



Month in pics

ECD workshop at Srinagar on 13 May 2023

Month in pics