The Child and newborn

West Bengal Academy of Pediatrics

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Special Correspondance
Dr Kaustav Nayek
Editor-in-Chief, The Child and Newborn
"Oriental Apartments" Flat H1
15C, Canal Street, Kolkata 700 014
Email : kaustav25@yahoo.co.in
wbapeditorinchief@gmail.com

Editorial

It is indeed a pleasure to present the last issue of Child and Newborn for 2024. As we step into another year of this journey, it is heartening to reflect on the successful two years of publishing this journal - a feat that would not have been possible without the unwavering support and contributions of our esteemed pediatric colleagues.

When we embarked on this endeavor two years ago, there was a fair share of skepticism. However, the overwhelming response and active participation from our fellow pediatricians have made this journal a platform for academic and clinical exchange. The credit for its smooth journey belongs entirely to you, our contributors, who have enriched each issue with your valuable articles and insights.

We acknowledge that there have been challenges, particularly in ensuring the timely delivery of the journal to all our members. We appreciate your patience and trust and assure you that we are working diligently to overcome these hurdles. Our commitment remains steadfast in making Child and Newborn a high-quality publication that serves as a vital resource for pediatricians.

Once again, we invite you to submit original articles that will further enhance the journal's academic value. Your contributions will play a crucial role in helping us achieve our ultimate goal of getting the journal indexed, thereby increasing its reach and impact. We urge you to continue sharing your research and experiences, as they form the backbone of this publication.

On another note, we are thrilled to announce that PEDICON 2026 will be held in Kolkata and will be organized by us, the West Bengal Academy of Pediatrics family. This presents an incredible opportunity for us to showcase our organizational prowess and create a conference that will be remembered by the entire pediatric fraternity. We encourage all members of WBAP to actively participate, bring forth innovative ideas, and contribute towards making this event unique and extraordinary.

Let us come together to ensure that PEDICON 2026 sets new benchmarks and stands out as an event that fosters knowledge, collaboration, and excellence in pediatric care. Looking forward to your continued support and enthusiasm in both the journal and the upcoming conference.

Warm regards,

Prof.(Dr.) Kaustav Nayek
Editor-in-Chief

President's Address

Message from the President, West Bengal Academy of Pediatrics

Dear Esteemed Colleagues and Readers of Child and Newborn,

It is both an honour and a privilege to address this distinguished platform as we turn our focus to a crucial yet often underappreciated aspect of pediatric healthcare—Cognitive Dysfunction in Children with Oropharyngeal Abnormality or Dysfunction.

Oropharyngeal abnormalities, encompassing a spectrum of congenital and acquired conditions such as cleft palate, Pierre Robin sequence, neuromuscular disorders, and swallowing dysfunction, extend beyond their immediate impact on feeding, respiration, and speech. Increasing research underscores the intricate interplay between these conditions and neurodevelopmental outcomes, with long-term consequences on cognition, learning, and social adaptation.

Children facing persistent oropharyngeal challenges often endure chronic malnutrition, recurrent infections, hypoxia, and prolonged medical interventions—factors that collectively impede optimal brain growth and cognitive function. Additionally, speech and language delays stemming from impaired oral-motor coordination may lead to deficits in communication, executive functioning, and academic performance.

This issue of Child and Newborn seeks to highlight critical insights into:

- The neurological implications of early-life oropharyngeal dysfunction
- The role of early intervention, feeding therapies, and neurodevelopmental suppor

As paediatricians and child health advocates, our mission must extend beyond survival to ensuring quality of life and developmental potential for every child. Early recognition, comprehensive management, and holistic rehabilitation strategies hold the key to mitigating cognitive deficits and fostering better outcomes for these vulnerable children.

I encourage researchers, clinicians, and policymakers to collaborate in shaping a future where early intervention is prioritized, multidisciplinary care is strengthened, and no child is left behind in their cognitive and developmental journey.

Let us continue our collective pursuit of excellence in pediatric care.

Warm Regards,

Dr.Asok Kumar Datta
President
West Bengal Academy of Pediatrics

Determinants of Quitting Tobacco in School-children

Dr Kamirul Islam
Pediatrician , Katwa Sub divisional Hospital

Introduction: Tobacco use is prevalent among Indian school-children. Data regarding predictors of successful quitting are scarce. Aim: To identify the determinants of quitting tobacco in school-children. Materials andMethods: Thisprospective cohort study was conducted between January 2018 and December 2019 among 2458 school-children selected by complete enumeration. Data were collected by direct interview using pre-designed, pre-tested, semi-structured schedule. Different study variables were compared among children who were able to quit tobacco and who were not able to quit. Results: Six hundred and fifty two (26.5%) school-children were able to quit tobacco successfully. Nicotine dependence (Relative risk=1.67) was the most significant determinant of quitting, followed by daily use of tobacco (Relative risk=1.65). Conclusions:Nicotine dependence of an individual is the most important determinant of quitting tobacco.

Keywords: Smoking, Cigarette, Dependence, Nicotine, Adolescent

Introduction:Excessive use of tobacco, the foremost cause of preventable morbidity andmortality, and is responsible for 12% of global deaths.[1] Majority of these deaths will occur in developing countries, like India.[1]Asian countries have an additional burden due to excessive popularity of smokeless tobacco.[2]Prevalence of tobacco use among students is highest in Bangladesh (43.3%), followed by India (34.6%).[3]Decreased awareness, risky mind set-up, poor literacy and repeated advertisements by tobacco companies-all are responsible for high prevalence of tobacco use in Indian school-children.[1, 2] Tobacco use is associated with diseases including coronary artery diseases, chronic obstructive airway disease, cancers of oral cavity, upper airway and lungs, fetal growth restrictions etc. Onset of this disease process occurs in late adolescence/ early adulthood. Cessation of smoking is associated with reversibility of disease process, increase in life expectancy and better quality of life.[2]But, successful quitting is difficult to achieve. Nearly 50% of tobacco users were able to quit tobacco permanently with help of modern pharmacotherapy.[4] Detailed information regarding predictors of successful quitting is scarce in Indian context, especially in adolescent population. Available few articles suffer from methodological issues (assessing quit attempts/ intention to quit instead of successful quitting, small sample size, risk ratio was not calculated).[5,6] Hence, we had undertaken this study to find the determinants of quitting tobacco in school-children.

Materials and Methods: This prospective cohort study was conducted between January 2018 and December 2019, after taking necessaryapproval from Institutional Ethics Committee. Informed written consents were taken from each participant/ their legal guardians. All the current tobacco-user school-children from 5th to 12th standard were included.Eleven schools (9- government, 2-private) with 18653 children were selected for the study (Source- Office of District Inspector of Schools). Who were severely ill, absent despite 3 visits, denied consent and lost in follow-up were excluded. A pre-designed, pre-tested, semi-structured schedule was used to collectdata. They were treated (counseling and/or pharmacotherapy) and followed-up at out-patient at quarterly interval/ earlier, if clinically indicated. Tobacco smoking was defined as inhaling

Correspondance :Dr Kamirul Islam, Medical Officer, *Burdwan Medical College and Hospital. Email kamirul.islam7@gmail.com*

smoke of burning tobacco into mouth and lung at least once in last week.[7]Smokeless tobacco use was defined as chewing, sniffing or keeping tobacco preparation in mouth at least once in last week.[2]Socioeconomic status was assessedby modified Kuppuswamy scale.[8]Problem families are those which lag behind the rest of community.[9] Nicotine dependence of an individual was measured by Fagerström Test for Nicotine Dependence (FTND). Low, medium and high level of nicotine dependence was defined as FTND score <4, 4-6 &>6, respectively.[10, 11]Two separate scoreswere calculated for the users who use both smoking and smokeless tobacco products and the higher score was taken for analysis.Mini-International Neuropsychiatric Interview for Children and Adolescents (MINI Kid) was used as a screening tool to identify associated psychiatric disorders.[12]Registered student of school at the time of study was considered as the school-children. Abstinence from tobacco for at least 12 months or morewas defined as quitting.[13]

SPSS version 19.0 (Statistical Package for the Social Sciences Inc, Chicago, IL, USA) was used for data analysis. Kolmogorov Smirnov test (as n>2000) was used to check normal distribution. Categorical and continuous variables were expressed in proportion and mean values, respectively. Significance of association between two attributes in contingency table was assessed by Pearson's chi-square (?2) test. Significance of difference between two means was checked by Student's unpaired t test.Risk ratio (RR) with 95% Confidence Interval (CI) was calculated taking quitting tobacco as positive outcome.[14] Binary logistic regression model (stepwise method) was generated to calculate Cox and Neglekarke's R2 to identify individual contribution of different factors in quitting tobacco.P=0.05 was considered as statistically significant.

Results:Two thousand six hundred and seventy nine (2679) tobacco-users were identified and221 were excluded(3 with serious illness, 26 were absent despite three visits, 16 refused consent and 176 did not complete follow-up). Hence, 2458 school-children (93.2% male) were included with a mean age of 14.8±2.8 years. Majority of them belonged to Hindu religion (1302, 53.0%), lower socioeconomic status (1261, 51.3%) and joint family (1538, 62.6%). Psychiatric co-morbidities and other substance abuse were observed in 218 (8.9%)and 185 (7.5%) children, respectively. Nine hundred and thirty four (934, 38.0%) children were exclusive smokers, 909 (37.0%) were using smokeless tobacco exclusively and 615 (25.0%) were using both. High nicotine dependence was seen among 869 (35.4%) children. After repeated counseling and pharmacotherapy, 652 children (26.5%) were able to quit tobacco. Different variables of two groups of children(who quitted tobacco and who were not able to quit tobacco) are illustrated in table 1.

Nicotine dependence (RR=1.67, 95% CI=1.59-1.73, P<0.0001) emerged as most significant determinant of quitting tobacco, explaining 52.3-52.5% variance of it; followed by daily use of tobacco (RR=1.65, 95% CI=1.57-1.74, P<0.0001) which can explain another 13% variance of quitting tobacco. Risk ratios of different determinants with 95% CI are represented in detail in table 2. Overall, our model could correctly predict75.6%-76.3% variance of quitting tobacco.

Discussion:Nicotine dependence emerged as the most significant determinant of quitting tobacco, followed by habit of tobacco use. We observed that 26.5% school-children were able to quit tobacco. Different studies from abroad reported a successful cessation rate of 7.7-27.8%.[15, 16]Multiple Indian studies estimated intention to quit among tobacco-users, which varies between 39%-63.3%.[5, 6, 17]Heterogeneity of population and differences in operational definitions might lead to this discrepancy. Level of nicotine dependence among school-children corroborates with findings of other researchers from India and abroad.[18, 19]Bernstein, et al and Islam, et al also noted that FTND score is the single most significant factor responsible for quitting.[5, 20] Nicotine alters the mesolimbic pathway and responsible for temporarily pleasing mood enhancement and makes quitting difficult.[21]Similar to the findings of current study, multiple authors reported that different socio-demographic variables (age, sex, type of family, socioeconomic status), type of school, variables related to tobacco use (age of initiation of tobacco use, duration of tobacco us, habit of tobacco use), presence of psychiatric

Table 1: Distribution of the study population according to quitting tobacco and different variables (n=2458).

Variables	Quitted Tobacco (n=652)	Not Quitted Tobacco (n=1806)	Significance
Age (y)*	14.6±2.3	14.9±3.1	t=2.2568, P=0.0241
Sex#			
Male	584 (25.5)	1708 (74.5)	χ^2=19.0415,
Female	68 (41.0)	98 (59.0)	P=0.0001
Religion#			
Hindu	348 (26.7)	954 (73.3)	χ^2=0.0582,
Others	304 (26.3)	852 (73.7)	P=0.8093
Type of Family#			
Nuclear	368 (40.0)	552 (60.0)	χ^2=136.971,
Joint	284 (18.5)	1254 (81.5)	P<0.0001
Socioeconomic Status#			
Upper	179 (39.2)	278 (60.8)	χ^2=47.9222P<0.0001
Middle	188 (25.4)	552 (74.6)	
Lower	285 (22.6)	976(77.4)	
Type of School#			
Government	364 (29.7)	862 (70.3)	χ^2=12.5675P=0.0004
Private	288 (23.4)	944 (76.6)	
Belonged to Problem Family#			
Yes	323 (24.5)	997 (75.5)	χ^2=7.8154
No	329 (28.9)	809 (71.1)	P=0.0052
Age of Onset of Tobacco Use (y)*	13.9±2.0	12.3±2.9	t=13.0140P<0.0001
Duration of Tobacco Use(y)*	4.3±0.9	6.1±1.2	t=34.9179P<0.0001
Variables	**Quitted Tobacco (n=652)**	**Not Quitted Tobacco (n=1806)**	**Significance**
Nicotine Dependence*			
Low	388 (49.2)	400 (50.8)	χ^2=513.0442,
Medium	256 (32.0)	545 (68.0)	P<0.0001
High	8 (0.9)	861 (99.1)	
FTND score#	3.1 ±1.2	5.9 ±1.5	t=42.9572, P<0.0001
Habit			
Daily	88 (7.5)	1089 (92.5)	χ^2=420.4862,
Occasional	564 (44.0)	717 (56.0)	P<0.0001
Psychiatric co-morbidities			
Present	41 (18.8)	177 (81.2)	χ^2=7.3119,
Absent	611 (27.2)	1629 (72.8)	P=0.0068
Other Substance Abuse			
Yes	22 (11.9)	163 (88.1)	χ^2=21.9818,
No	630 (27.7)	1643 (72.3)	P<0.0001
Knowledge about injurious effect of tobacco			
Absent	209 (18.2)	938 (81.8)	χ^2=21.9818,
Present	443 (33.8)	868 (66.2)	P<0.0001

**No (%), # Mean±SD, FTND-Fagerström Test for Nicotine Dependence*

Table 1: Distribution of the study population according to quitting tobacco and different variables (n=2458).

Risk Factors	Relative Risk	95% Confidence Interval	P Value
Age >15 y	1.18	1.04-1.35	0.0124
Male Sex	1.26	1.11-1.44	0.0004
Hindu Religion	0.99	0.95-1.04	0.8092
Joint Family	1.36	1.28-1.44	<0.0001
Lower Socioeconomic Status	1.12	1.06-1.17	<0.0001
Reading in Private School	1.09	1.04-1.14	0.0004
Belonged to Problem Family	1.06	1.00-1.11	0.0136
Age of onset of tobacco use ≤12 y	1.34	1.17-1.52	<0.0001
Duration of Tobacco Use ≥5 y	1.44	1.26-1.64	<0.0001
High Dependence to Nicotine	1.67	1.59-1.73	<0.0001
Daily Use of Tobacco	1.65	1.57-1.74	<0.0001
Presence of Psychiatric co-morbidities	1.12	1.04-1.20	0.0017
Other Substance Abuse	1.22	1.15-1.29	<0.0001
No knowledge about injurious effect of tobacco	1.24	1.17-1.30	<0.0001

FTND-Fagerström Test for Nicotine Dependence

comorbidities and concurrent other substance abuse also influence quitting among tobacco users.[5, 6, 15] They are also important determinants of nicotine dependence.[19, 22] Hence their effect on quitting might be due to interplay of these variables.

This study may be associated with recall bias. Self-reporting of the school-children was main mode of data collection. Thus, concealed information may lead to inaccuracy. Only urban schools were included with a small fraction of girls (approximately 7%). Nearly one-fourth factors responsible for successful quitting remained unknown.

To conclude, the present study identified the predictors of successful quitting of tobacco among school-children. Tobacco use had its origin in early childhood and adolescence;[5] hence, school-children should be targeted and made aware of injurious effect of tobacco.[21] Considering high prevalence of tobacco use in school-children, proper cessation program is the need of hour to decrease future burden of tobacco related morbidity and mortality.[21] Appropriate categorization of school-children depending on their dependence level maybe helpful.[21, 23]

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Exploring the Connection between Childhood Cognition and their Oral Health: A Comprehensive Review

Prof. Dr Arun K Singh, Dr Kanupriya Rathore
Paediatric Dental Consultant, Model Early Intervention Centre,
All India Institute of Medical Science, Jodhpur

Abstract : The interrelationship between cognitive development and oral health in children is complex and multifaceted. This review examines how cognitive factors, such as memory, problem solving ability, and psychological well-being, influence oral health outcomes and vice versa. The article highlights how oral health problems, such as early childhood caries (ECC), can affect cognitive functioning and how cognitive development impacts oral health practices.

Introduction

Oral health is crucial for children's overall development, influencing physical, emotional, and cognitive growth. Early Childhood Caries (ECC), a severe form of tooth decay affecting children aged 71?months (5?years) or younger, has been shown to negatively impact cognitive abilities, speech development, and psychomotor function, with long-term consequences. According to the National Oral Health Survey & Fluoride Mapping (2019-20) by the, Government of India (MoFHW)in India, the prevalence of ECC is alarmingly high, affecting around 50-60% of preschool children, primarily due to poor oral hygiene, dietary habits, and limited access to dental care. The prevalence up to 85% has been found in underprivileged group of children. Understanding how ECC affects cognitive development highlights the need for early intervention, preventive care, and improved oral health strategies to promote overall child well-being. This article aims to review the literature on ECC and its effects on cognitive development, providing insights into how oral health influences cognitive outcomes.

Cognition and Child Development:

Child development is generally categorized into four key domains: physical, cognitive, linguistic, and socio-emotional development. Physical development involves growth, motor skills, and self-care tasks, while cognitive development refers to processes such as problem-solving, memory, and mathematical abilities. Linguistic development spans the progression from babbling to communicative speech, and socio-emotional development focuses on emotional growth through caregiver relationships.

Research has demonstrated that cognitive development is interconnected with oral health, influencing behaviors such as regular brushing and dental visits. Cognitive development also impacts decision-making, problem-solving, and emotional regulation, all of which affect health practices (Jackson et al., 2022; Mouradian et al., 2015). Hence for better cognitive development, the general health of child cannot be neglected.

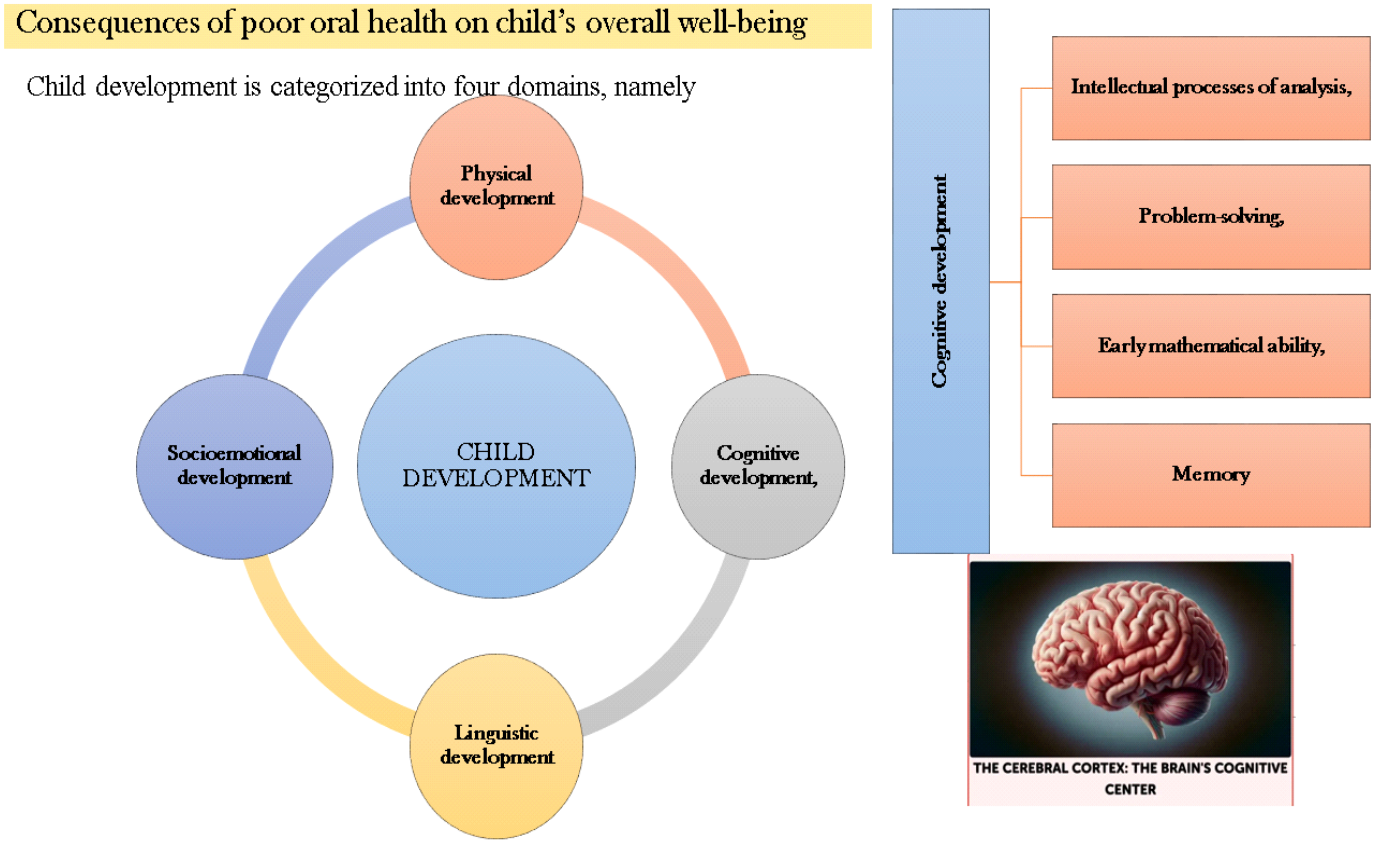
Fetal Brain Development and its Significance:

Fetal brain development occurs in several key stages, each critical to the growth of cognitive, motor, and emotional functions:

1. Early Prenatal (Weeks 3–8): Neural tube formation and basic brain structures develop, with disruptions leading to neural tube defects (Kaufman, 2004).
2. Mid Prenatal (Weeks 9–24): Rapid neuronal proliferation and migration occur, forming the cerebral cortex and brainstem, essential for sensory, motor, and cognitive functions (Rice & Barone, 2000).The most active period of baby brain development takes place during middle of

Correspondance : Prof. Dr Arun K Singh, Paediatric Dental Consultant, All India Institute of Medical Science, Jodhpur. Email : drarunsingh61@yahoo.co.in

Fig1: Domains of child development



the second trimester, when 250,000 neurons are created every minute.

3. Late Prenatal (Weeks 25–40): Brain maturation continues with synaptogenesis and myelination, which improve neuronal communication efficiency (Berlouin, 2018).
4. Early Postnatal (0–2 Years): Brain plasticity peaks, supporting motor skills, sensory processing, and early cognitive functions (Huttenlocher & Dabholkar, 1997).
5. Preschool Years (2–6 Years): Synaptic pruning strengthens neural connections, enhancing cognitive abilities such as attention and problem-solving (Casey et al., 2005).

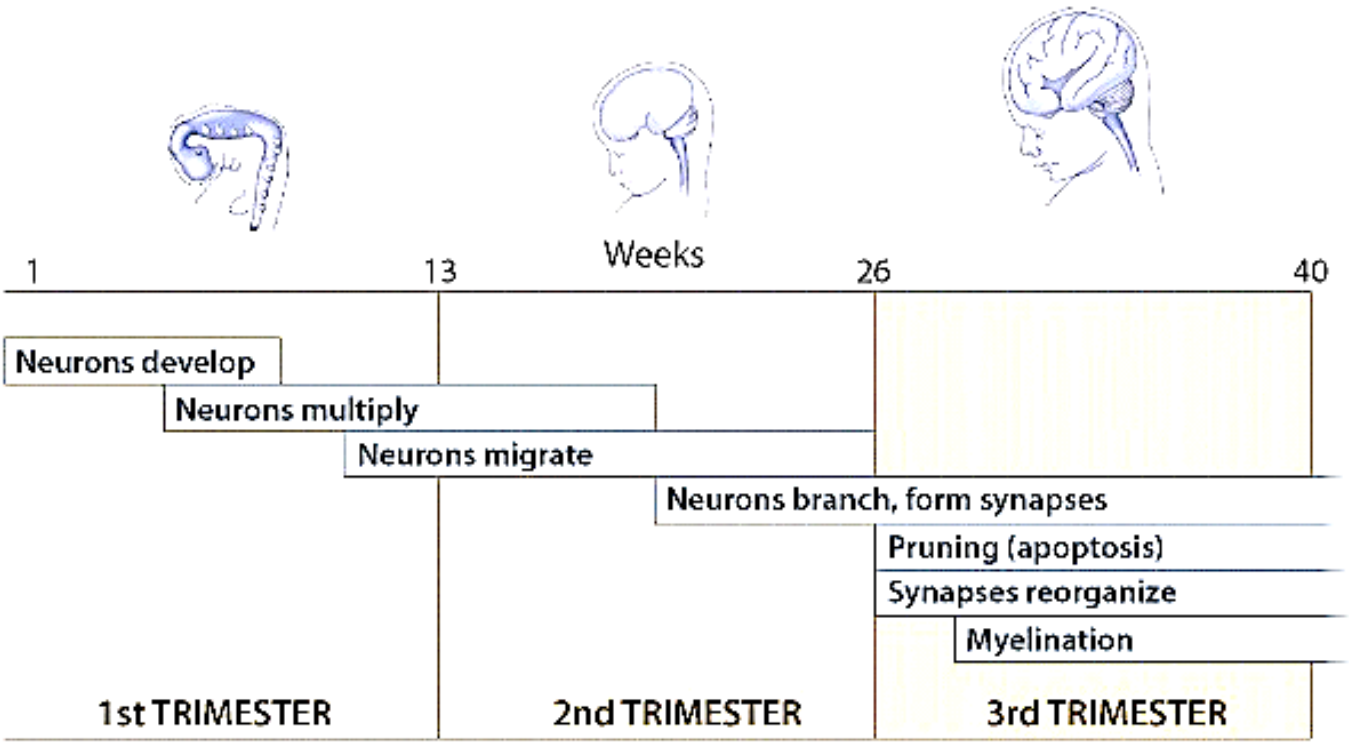
Each developmental stage is essential for optimal brain maturation, with environmental factors such as maternal health and nutrition playing a pivotal role in shaping cognitive outcomes (Kaufman, 2004; Rice & Barone, 2000).Up to 700 neural connections are formed every second through stimulation.At birth 20-25% of Adult brain volume, by one year 72% of the Adult brain volume and by two years 83- 85% of the Adult brain volume is formed.

Oral Health during Pregnancy and Foetal Cognitive Growth:

Oral health during pregnancy significantly affects both maternal and foetal health, including cognitive development. Conditions such as periodontal disease, dental caries, and oral infections can trigger systemic inflammation, potentially harming foetal brain development. Maternal infections can lead to complications like preterm birth or low birth weight, which may contribute to later cognitive impairments (Offenbacher et al., 2006).

Using fMRI scans, Thomason (2017) provided the first direct evidence that premature infants may have different brain structure before birth. These scans showed that premature infants, compared with full-term foetuses, had weaker connections in the left hemisphere, an area that later develops into language processing regions. Premature infants experience reduced brain volume in several areas later in life,

Fig 2: Key stages of foetal brain development



including the basal ganglia, cerebellum, and cortical gray and white matter, although the left hemisphere is more commonly affected. Furthermore, premature boys are more susceptible to white matter abnormalities than girls, and the frontotemporal and hippocampal regions appear to be particularly susceptible to volumetric changes.

How Do Oral Health Conditions Affect Babies and Do cavities during pregnancy increase the likelihood of babies developing cavities?

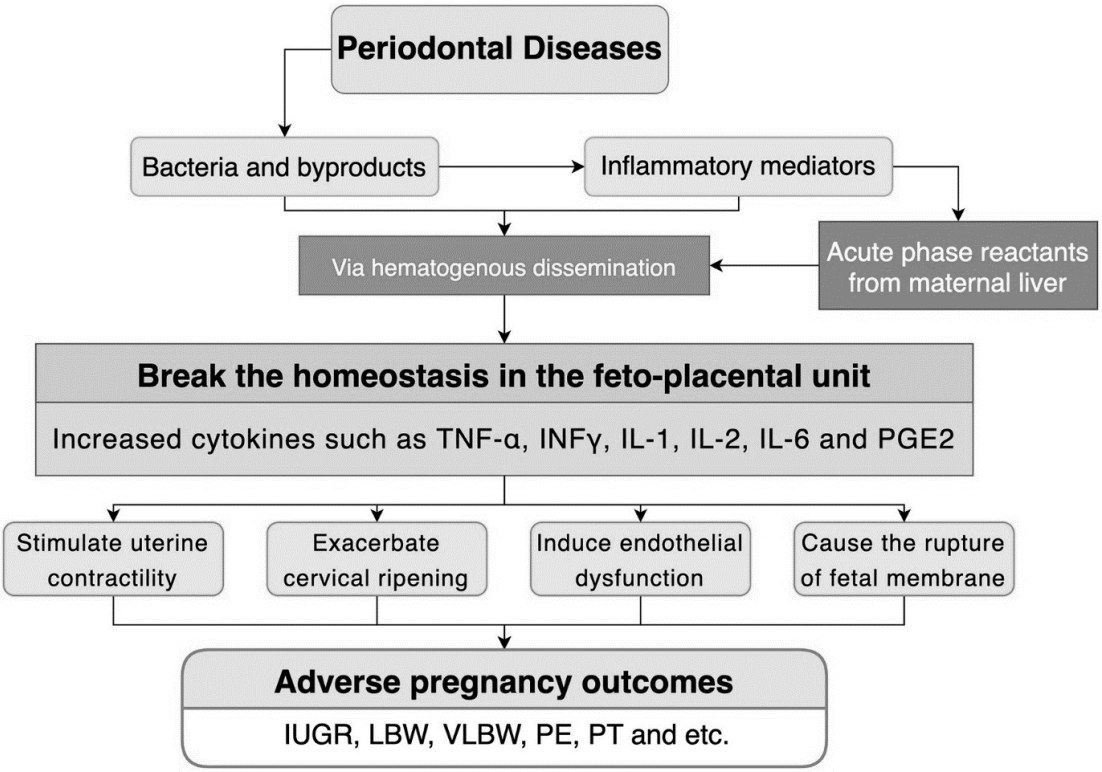
- Maternal oral flora is transmitted to the newborn infant, and increased cariogenic flora in the mother predisposes the infant to development of caries
- Children are about 75% more likely to develop ECC if the mom or caregiver has active cavity.
- Children can also acquire cavity-causing bacteria (particularly strep mutans) at a young age through transmission.
- Poor oral health can cause adverse pregnancy outcomes
- The inflammatory effects of gum/periodontal disease have been linked to low birth weight, preterm birth, and preeclampsia.
- Pre-term infants with low birth weight are more likely to have harmful cavity-causing bacteria colonizing in their mouths as early as one week after birth.

Impact of Poor Oral Health on Child Well-being:

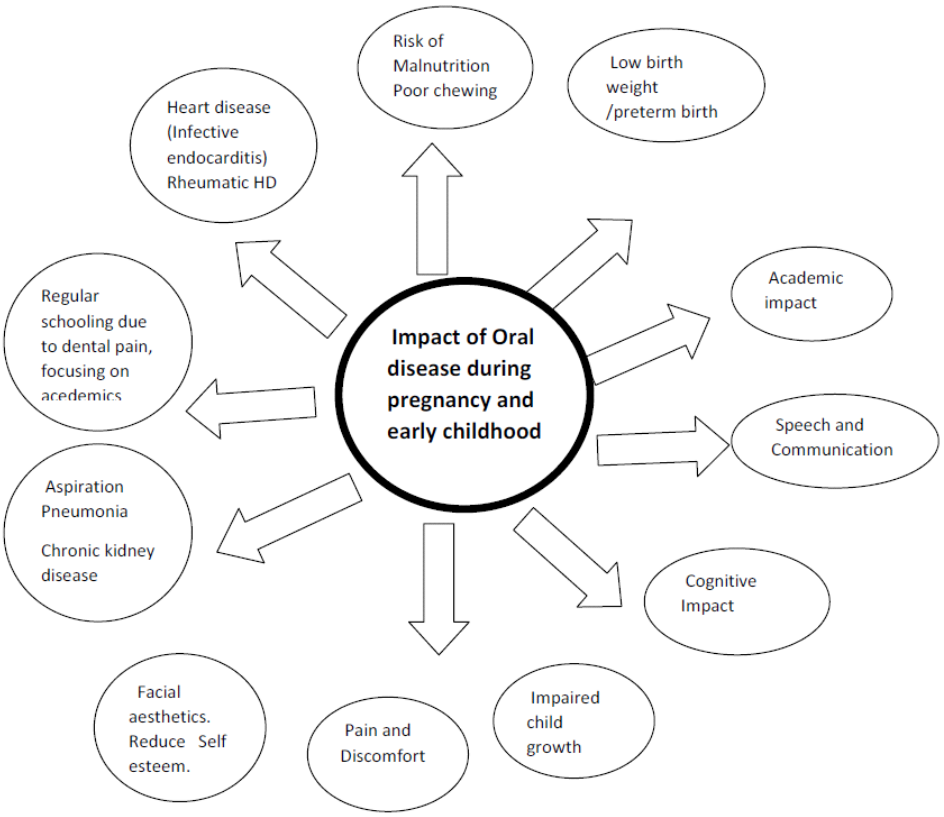
Children with poor oral health are at risk of various physical, emotional, cognitive, and social issues. Dental problems such as cavities can lead to pain, infection, and difficulty eating, hindering growth and development. Emotional consequences, such as low self-esteem, anxiety, and social withdrawal, can also emerge due to visible oral issues, further affecting a child's development (Warren et al., 2018; Kramer et al., 2021).

Common oral health problems in Preterm babies are notching of the alveolar ridge, palatal grooving, high arched palate, dental cross bite, dental crowding, deep bite, and palatal asymmetry. There is higher probability of developing malocclusion - enamel defects such as quantitative loss of enamel (hypoplasia) and qualitative change in the translucence (opacity) of the enamel in these babies.

The Link between Dental Caries and Cognitive Development in Children:



Inflammatory pathway and its role in adverse pregnancy outcomes (Wen, Xingyue et al).



Dental caries is a widespread issue among children, with significant effects on cognitive development. Pain and discomfort from untreated caries can hinder concentration, leading to lower academic performance and increased absenteeism (Lima et al., 2016). Additionally, caries can lead to nutritional deficiencies, impairing brain function and cognitive growth (Agha-Hosseini et al., 2021). Long-term, untreated dental issues may result in speech delays, behavioral changes, and overall cognitive delays (Tuna et al., 2020).

Relation between stress (infection and pain) and cognitive functions

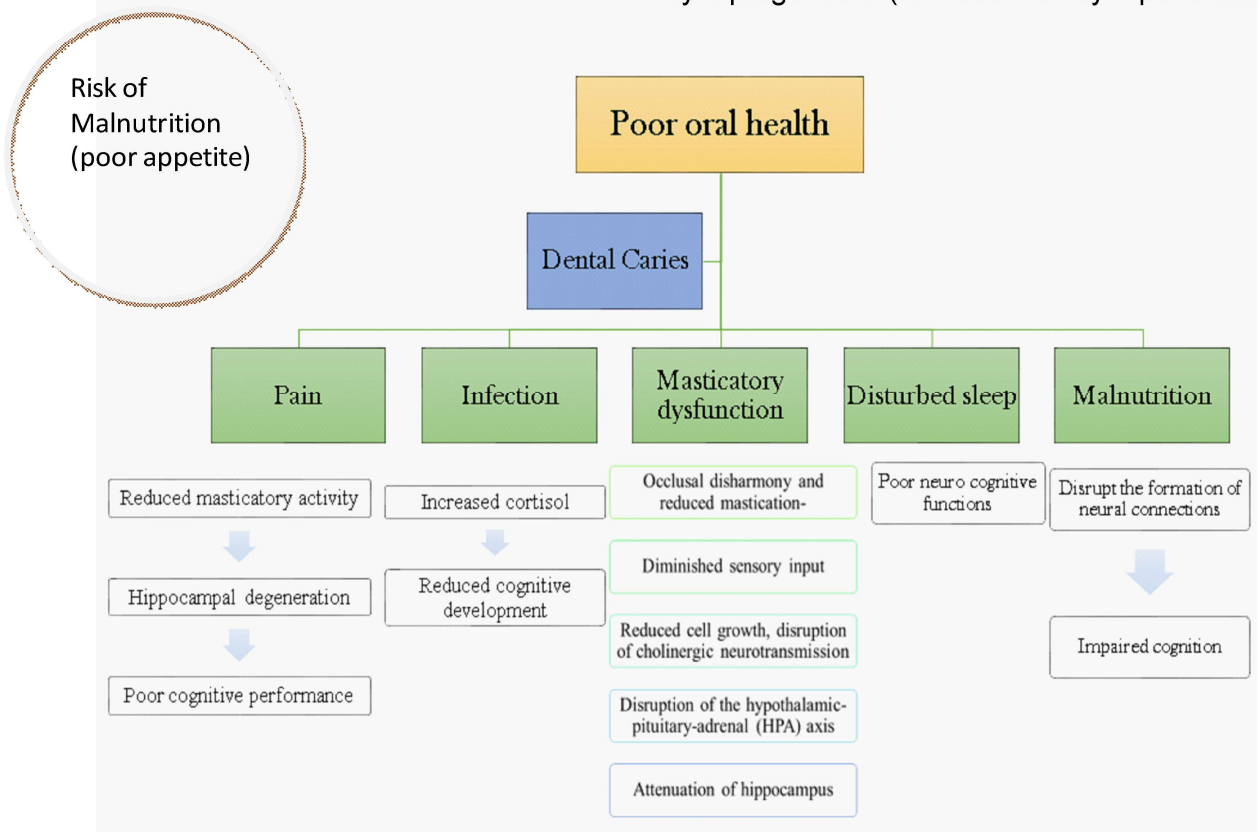
Acute and chronic stress exposure impairs hippocampal neurogenesis by reducing progenitor cell proliferation, suppressing neuronal differentiation, and decreasing cell survival in the dentate gyrus (McEwen, 2007; Kim & Diamond, 2002). Prenatal stress, along with excessive glucocorticoid (GC) exposure in utero, disrupts brain development and maturation in offspring, potentially leading to long-term consequences such as impaired neurogenesis and cognitive deficits (Meaney, 2001; Lupien et al., 2009). These effects suggest that stress not only alters adult hippocampal function but also has transgenerational impacts via epigenetic changes, affecting the offspring's cognitive health throughout life (Franklin & Mansuy, 2010).

Nutrition's Role in Cognitive Development and Oral Health:

Proper nutrition is crucial during brain development, especially in prenatal and early childhood stages. Key nutrients, such as omega-3 fatty acids, proteins, and vitamins, are vital for neuronal formation and brain maturation (Huskisson et al., 2007). A deficiency in these nutrients can impair cognitive and neuronal development, emphasizing the importance of a balanced diet for optimal brain growth.

Role of Nutrition in the Formation of Neurons

Nutrition plays a fundamental role in the formation and development of neurons, which are the building blocks of the brain. Proper nutrition is crucial during critical periods of brain development, particularly during prenatal development and early childhood, when the brain undergoes rapid growth and synaptogenesis (formation of synapses between



The Link between Dental Caries and Cognitive Development in Children

neurons). Nutrients like omega-3 fatty acids, proteins, and vitamins are essential for the structural and functional maturation of neurons.Both DHA and AA are 'neural lipids' which make up 60% of human brain. DHA enhances phospholipid synthesis and increased phospholipids promotes neurite (axon or a dendrite) outgrowth

1. Omega-3 Fatty Acids:

Omega-3 fatty acids, especially docosahexaenoic acid (DHA), are vital for the formation of neuronal membranes and synapses. DHA contributes to the fluidity and function of the cell membrane, essential for proper neuronal communication and cognitive function (Huskisson et al., 2007). A deficiency in omega-3 fatty acids during critical developmental periods can result in impaired neuronal growth and function.

2. Proteins and Amino Acids:

Proteins are essential for the synthesis of neurotransmitters, which are crucial for communication between neurons. Amino acids, the building blocks of proteins, are also important in the formation and maintenance of neuronal structures (Hasselmo, 2006).

3. Vitamins and Minerals:

Vitamins like B-complex vitamins (especially B12 and folate) are essential for proper neuronal development, as they support DNA synthesis and neuronal maturation. Minerals such as iron are necessary for oxygen delivery to the brain and for maintaining optimal cognitive function (Ramakrishnan et al., 2012).

The Link between Malnutrition and Cognitive Development in Children

Due to poor oral health, dental pain and difficulty in chewing, child is malnourished. Malnutrition may affect neurocognitive development in children by directly affecting structural brain development (reduced synapses and synaptic neurotransmitters, delayed myelination, abnormal timing of brain maturation and thus disruption of neural circuit formation) or indirectly affecting cognitive abilities. Children are more affected by nutritional deficiencies. Malnutrition is associated with both structural and functional brain diseases. Various cognitive disorders have been reported in malnourished children. It has been reported that long-term changes in brain function may be related to the long-term cognitive impairment associated with malnutrition.

Poor oral health, especially dental pain and reduced chewing ability, can lead to malnutrition in children. The Child may avoid certain foods due to the pain, resulting in a diet that lacks important nutrients required for optimal growth and cognitive development. This can lead to deficiencies in vitamins and minerals essential for brain function (Agha-Hosseini et al., 2021). Furthermore, dental problems, if left untreated, can affect a child's ability to chew properly, leading to poor nutritional intake and associated growth and development delays (Lima et al., 2016).

The Bidirectional Relationship between Cognition and Oral Health:

The connection between cognition and oral health is bidirectional. Cognitive development directly impacts oral health behaviours. Children with better cognitive abilities are more likely to understand the importance of oral hygiene and dental visits (Jackson et al., 2022). Children with oral health issues may experience psychological distress, which impairs their cognitive development and social engagement (Kramer et al., 2021; Gates et al., 2019).

Conclusion:

The relationship between cognition and child oral health is complex, with oral health affecting cognitive function and cognitive development influencing oral health . A trans-disciplinary approach that integrates cognitive development with oral health interventions could lead to better outcomes for children's overall well-being. Future research should explore comprehensive strategies that address both cognitive and oral health needs to ensure the optimal development of children.

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Management of PDA in Newborn(Term and Preterm)

Dr Dipsikha Jana*, Dr. Debasree Gangopadhaya**
**Resident, HOD, Senior consultant, Department of Pediatric Cardiology.*
Narayana Health- Rabindranath Tagore International Institute of Cardiological Sciences.

Abstract: Preterm infants are at increased risk of PDA. There may be several grave clinical consequences of long term ductal patency in preterm neonates. Although the molecular mechanisms underlying regulation of postnatal ductusclosure are not fully understood, multiple clinical experience and research trials have suggested recent changes in its management strategies and refocused treatment strategies. This article aims to provide thecurrent diagnostic and management approaches to PDA in term and preterm neonates.

Objectives: After completing this article, readers would be able to understand:

- What is hemodynamically significant ductus,
- Timing of pharmacotherapy and drugs,
- Role of prophylactic pharmacotherapy,
- Timing of invasive treatment,
- Intervention or surgery how to make the decision

he interrelationship between cognitive development and oral health in children is complex and multifaceted. This review examines how cognitive factors, such as memory, problem solving ability, and psychological well-being, influence oral health outcomes and vice versa. The article highlights how oral health problems, such as early childhood caries (ECC), can affect cognitive functioning and how cognitive development impacts oral health practices.

Introduction

The ductus arteriosus (DA), a physiological necessity in the intrauterine life, is a fetal vascular connection between the main pulmonary artery and the aorta that diverts blood away from the pulmonary bed. After delivery, the DA undergoes active constriction and eventual obliteration.PDA occurs when the DA fails to close postnatally.It is the most common cardiovascular condition in preterm infants,due to the anatomical characteristics of the DA tissue, results in a number of hemodynamic alterations and an increase in preterm morbidity. Despite of recent advances in the field of neonatology, PDA still is a challengeto neonatologists and pediatric cardiologists.

Correspondance : Dr. Debasree Gangopadhaya, HOD, Senior consultant, Narayana Health- Rabindranath Tagore International Institute of Cardiological Sciences. Email : debasreeganguly23@gmail.com

Epidemiology:

It is the most common cardiovascular condition of preterm infants, and the incidence of PDA is inversely related to gestational age at birth.About 15-40% of very low birth weight infants (<1500 g) have a PDA. In extremely preterm infants (<28 weeks), the incidence is as high as 70-80%. Data among term infants suggest that PDA are observed in ~1 in 2000 birth,accounting for 5% to 1% of all CHD. Longitudinal cohort studies suggest that the incidence of Silent PDA (these are discovered by cardiac imaging without any clinical manifestation) is 1 in 20 births. [1].

Rate of spontaneous closure:

In healthy full-term infants, the DA closes within 48 to 72 hours. In premature infants born weighing > 1kg, the ductus closes spontaneously in 67% by day 7 and in 94% by discharge. Overall, only 3% of infants weighing >1kg mayrequire intervention for a PDA.

However, in extremely premature infants weighing <1kg at birth (ELBW), 57% to 69% will still have a PDA at 7 to 10 days of age. Of those that close, up to 30% will reopen and, either may reclose with median time of 56 days, or become hemodynamically significant which require pharmacologic or surgical closure. [1]

Because hemodynamically significant PDA has been associated with IVH,pulmonary hemorrhage, NEC, CLD, and death, more substantial studies are required before nonintervention approaches can be widely adopted.

What is hemodynamically significant ductus?[2]

A consensus definition for hemodynamically significant PDA is lacking. The diagnosis is often suspected clinically, when an infant demonstrates signs of excessive shunting from the arterial to pulmonary circulation:

- Continuous or systolic murmur; note, a "silent" PDA may also occur when the ductus shunt is large enough that nonturbulent flow fails to generate a detectible murmur.
- A low diastolic blood pressure (due to runoff into the ductus during diastole, more frequent in the most premature infants)
- A wide pulse pressure (due to ductus runoff or steal)
- Hypotension (especially in the most premature infants)
- Bounding pulses
- Increased serum creatinine concentration or oliguria
- Hepatomegaly

Signs of pulmonary edema are often seen, including tachypnea, decreased oxygen saturation, and increasing respiratory support. Chest radiography can show signs of pulmonary edema. The neonatal cardiac output can increase as much as 25% in an effort to overcome ductus steal.

The echocardiographic parameters aimed at identifying term and preterm infants in whom ductal shunt volumes are estimated to be primary pathological contributors to physiologic instability:

Table A. : Comprehensive Grading Schema for HSPDA among Preterm Infants[1]

Clinical	Echocardiography	
Asymptomatic	No PDA	No evidence of ductal flow on 2D or Doppler interrogation
Mild symptoms • MAP <8 mmHg (on respiratory support of NCPAP or mechanical ventilation) • Feeding intolerance	Small, non-HSPDA	• Transductal diameter <1.5mm. • Restrictive continuous transductal flow (DA Vmax >2.0m/s). • No signs of left heart volume loading (eg, mitral regurgitant jet <2.0m/s or LA:Ao <1.5:1) • No signs of left heart pressure loading (eg, E/A ratio <1.0 or IVRT <50)
Moderate symptoms • MAP 9–12 mmHg (ventilation requirement) • Evidence of abdominal distention and/or persistent emesis	Moderate, HSPDA	Transductal diameter 1.5–3.0 mm • Unrestrictive pulsatile transductal flow (DA Vmax <2.0 m/s) • Mild-moderate left heart volume loading (eg, LA:Ao 1.5–2.1) • Mild-moderate left heart pressure loading (eg, E/A ratio ≥1.0 or IVRT 50–60) • Decreased or absent diastolic flow in the superior mesenteric, middle cerebral, and/or renal arteries
Severe symptoms • MAP >12 mmHg (high ventilation requirements or HFOV) • Marked abdominal distention and/or erythema	Large HSPDA	• Transductal diameter >3.0mm. • Unrestrictive pulsatile transductal flow. • Severe left heart volume loading (eg, LA:Ao >2.1, mitral regurgitant jet >2.0m/s). • Reversal of mitral E/A ratio. • Severe left heart pressure loading (eg, E/A ratio >1.5 or IVRT >60). • Reversal of end-diastolic flow in superior mesenteric, middle cerebral, and/or renal arteries. • LPA diastolic velocity—mean velocity >0.42 m/s, enddiastolic velocity >0.2 m/s.

Predictive scoring system for HS-PDA :

A. Clinical Criteria:

The scoring systems were developed with the target of early prediction of HSPDA .These depend on patient characteristics, prenatal and perinatal history, physical examination, and other clinical and vital signs. However,they could not be used instead of echocardiographic evaluation, but they help to predict who need early echocardiographic assessment, and to provide objective follow-up for HSPDA.E.g.: Scoring system mentioned in Gonen et al (2021)- Simple,with 90-100% sensitivity and 89-94% specificity –Includes maternal chorioamnionitis, antenatal steroid exposure ,birth weight ,cord blood base deficit, hypotension ,tachycardia ,invasive MV ,maximum PIP, FiO2, surfactant use.[3]

B. Laboratory Criteria:

1. Platelet count and indices

   Platelet count and other platelet indices such aslow platelet distributionwidth (PDW), low mean platelet volume (MPV), low platelet counts in first 3 postnatal days, correlate with HSPDA.

   Evidence suggests that, low value of these indices causes potential failure of pharmacologic treatment of PDA.

2. Cardiac peptides

   Pressure and volume loading in the cardiac ventricles cause therelease of pro-brain natriuretic peptide (proBNP), which activates into BNP. Studies, suggested that, BNP with =250 pg/mL in preterm infants (< 30 weeks) with PDA indicates HSPDA.

   El-Khuffash et al. found that the levels of cardiac troponin T werecorrelated with the echocardiographic findings of PDA.[3]

3. Near infrared spectroscopy, perfusion index

   It continuously measures regional tissue oxygenation non-invasively. It could help identify impaired cerebral, renal, and mesenteric circulation for early diagnosis of HSPDA. It could help to predict a significant PDA.[3]

Clinical Consequences Of Hemodynamically Significant PDA :

A left-to-right ductal shunt causes increased pulmonary blood flow and ductal steal from the systemic circulation and thus can have adverse effects on premature infants, although a causal relationship is not well defined.

A. Pulmonary Sequelae and CLD:

There is increased need for respiratory support and mechanical ventilation, contributing to lung injury. Pulmonary overcirculation can lead to hemorrhagic pulmonary edema(incidence 10 % in neonate <1000g), Pulmonary hypertension (post capillary). A largePDA also cause left atrium (LA) and leftventricle (LV) overload leading to LAand LVdilation , later LV dysfunction.

Sustained PDA exposure may contribute to BPD. However, an association between PDA and BPD has not been found in other studies (PDATOLERATE trial).[4]

B. Neurologic Morbidities :

PDA decreases regional cerebral oxygenation saturation and increases fractional tissue oxygen extraction in preterm infants. On the other hand, Intraventricular hemorrhage,periventricular leukomalacia, and school-aged performance are important outcomes associated with prematurity. Hence,the role of PDA in these outcomes versus the effect of PDA treatments is hard to explain. The Trial of Indomethacin Prophylaxis in Preterm (TIPP) revealed decreased IVH rates after prophylactic indomethacin but without long-term benefit. (The data for prophylactic ibuprofen or paracetamol are less compelling).[4]

C. NEC or Focal Intestinal Perforation :

HSPDA can cause diminished intestinal blood flow and may predispose preterm infants to NEC or focal intestinal perforation (FIP).An RCT from 1989 revealeddecreased NEC risk in infantsundergoing prophylactic surgicalligation (8% vs. 30%). On the contrary, there is no difference on prophylactic pharmacotherapy. Althoughthere are few data suggested that early feeding during indomethacintreatment may improve the time to reach full feedings, may preservepostprandial mesenteric perfusion and decrease therisk of NEC or FIP.[4]

TREATMENT OF PDA IN THE PRETERMINFANT

Indications:

The therapeutic indications for treatment of a symptomatic PDA include respiratory compromise (e.g. requiring persistent mechanical support), heart failure, or large left-to-right ductus shunt with evidence of hemodynamic compromise, such as reversal of flow in the descending aorta during diastole, oliguria or rising serum creatinine concentration, hypotension, or wide pulse pressure.

Some clinicians choose to use indomethacin prophylacticallyin ELBW with a goal of preventing IVH, PDA, and the adverse consequences of HSPDA. On the contrary, with prophylactic treatment, although there is successful reduction in short-term outcomes (IVH, pulmonary hemorrhage, hypotension, symptomatic PDA, need for ligation), the long-term benefits are uncertain. The echocardiographic criteria can be used to guide selective approach for prophylactic treatment, which may improve outcomes or less drug exposure. Well-designed studies are needed to clarify the risks/ benefits of this approach.

Conservative management: [1]

In patients of more than 1,000 g birth weight with few risk factors, a PDA can generally be successfully managed conservatively. In patients at higher risk of PDA, or who weigh less than 1,000 g at birth, conservative treatment is recommended before starting pharmacologic treatment.

A. Modest fluid restriction and use of positive end expiratory pressure to treat pulmonary edema.
B. Certain diuretics, such as furosemide, can prevent a ductus from closing and are not recommended in the first 1 to 3 weeks, when the greatest decrease in ductus diameter occurs spontaneously.
C. Avoidance of other drugs that promote ductus arteriosus relaxation.
D. Proactive use of agents like caffeine that are associated with lower rates of PDA.

Timing of Treatment:[2]

It is generally not necessary in the first few days after birth when the pulmonary vascular resistance is still elevated. However,during the second week, treatment should be considered if conservative measures have failed to control pulmonary edema, or if there is cardiac or renal failure. After 3rdweek, pharmacologic measures are less likely to be successful.

Pharmacological Closure of PDA:

The most widely studied and used drugs in the closure of PDA are the non-steroidal anti-inflammatory agents,(indomethacin and ibuprofen) and Acetaminophen (Paracetamol). All are equally efficacious, with a closure rate of 70-80% but differ in complications. Indomethacin and Ibuprofen are nonselective CoX inhibitors. Whereas Paracetamol inhibits prostaglandin production by inhibiting the peroxidase enzyme.

Effect on Lung by these agents:

1. Early pharmacologic closure improves incidence of CLD.
2. Decreased pulmonary edema and improved alveolarization (in premature baboons). This may be due at least in part to a direct effect on the lung.
3. Use of ibuprofen or indomethacin is associated with increased amiloride-sensitive alveolar epithelial sodium channels, increased lung water clearance, and improved lung compliance.
4. Prophylaxis with indomethacin reduced the risk for serious pulmonary hemorrhage by 35% over the first week of life; 80% of this beneficial effect was explained by reduced risk for PDA. Similarly, infants at ,29 weeks' gestation who were selectively treated prophylactically by 6 hours of age had less pulmonary hemorrhage than controls (2% vs 21%).

Role of prophylactic pharmacotherapy in HSPDA:[3]

A review of prophylactic indomethacin analyzed 19 trials with 2872 infants and demonstrated that, there were immediate short-term benefits in symptomatic PDA with exception of reduced renal output during treatment. However, there was no improvement in mortality or pulmonary outcomes.There is therefore no proven role for prophylactic indomethacin in terms of long-term outcomes.

Table B : Pharmacological management of hemodynamic PDA[1]

Preparation	Indomethacin	Ibuprofen	Paracetamol
Dosage (same for intravenous and oral formulation)	Intravenous preparation requires reconstitution. Oral suspension is not available but can be prepared by dissolving the powder content of a 25 mg indomethacin capsule in 25 ml of distilled water.	IV Ibuprofen is not available in India. The oral formulation is the most common and readily available form in India.	Available both as an injection (10 mg/ml) and oral suspension
Drug administration	Loading dose: 0.2 mg/kg/dose. Subsequent dose (adjusted as per postnatal age). • <2 days: 0.1 mg/kg/dose 12 hourly x 2 doses. • 2-7 days: 0.2 mg/kg/dose 12 hourly x 2 doses. • >7 days: 0.25 mg/kg/dose 12 hourly x 2 doses.	Loading dose: 10 mg/kg/dose. Subsequent dose: 5 mg/kg/ dose 24 hourly x 2 doses. (High dose Ibuprofen 20mg/kg/dose followed by 10 mg/kg/dose 24 hourly for two)	15 mg/kg every 6 hours for 3 days.
Drug administration	Administered as an infusion over 30 minutes or as a continuous infusion over 36 hours	Administered as an infusion over 30 minutes	Administered as an infusion over 15-30 min
Contraindications	Thrombocytopenia(< 60000/mm³) , the clinical bleeding tendency (bloody gastric aspirates, blood stools, pulmonary hemorrhage,oozing from puncture sites), raised serum creatinine, severe IVH, and NEC.	The clinical Same as Indomethacin	Elevated liver enzymes or hepatic failure.
Side effects	Transient or permanent renal impairment, NEC, gastrointestinal hemorrhage or perforation, alteration of platelet function, and impairment of cerebral blood flow or cerebral blood flow Velocity.	Lesser risk of NEC and transient renal insufficiency	No known renal adverse effects, Lower antiplatelet activity.
Persistence of a HSPDA after first course	A repeat course (3 doses) of indomethacin can be considered (maximum of 2 courses). The first course can be prolonged by giving two more doses of 0.1 mg/kg Q 24 hourly	A repeat course (3 doses) for a maximum of 2 courses can be considered. Extension of the initial course by giving 2 more doses with 5mg/kg, 24 hrly can be considered. However if the ductus fails to respond, it is unlikely to close with further courses.	A longer course of 15 mg/kg 6 hourly for 6-7 days can be considered based on echocardiography to guide the treatment.

The prophylactic treatment, once popular, has now become controversial as it unnecessarily exposes a large proportion of preterm babies to the side effects of treatment who would have closed the PDA spontaneously.

Catheter-Based Interventional Closure of the DA in Preterm Infants:

Devices:Various types of devices are used to occlude PDA. Ex : Sideris Buttoned Device, Gianturco-Grifka Vascular Occlusion Device , Duct-occlud and Nit-occlud Device,Amplatzer Devices (Amplatzer duct occluderI and II,Angled Amplatzer duct occlude, Amplatzer plug device , Amplatzer duct occlude additional sizes.The Amplatzer Duct Occluder II Additional Sizes (Piccolo Occluder device), recently received US Food and Drug Administration approval for PDA closure in premature infants weighing ?700 g with success rate of 95.5%

(191 of 200) in a multicenter trial of 200 patients, and 99% in those weighing 700 to 2000g.Selection of Device depends upon the size and type of PDA.

Surgical Ligation of the DA:Prophylacticand early ligation approaches are no longer indicated, but there remainsuncertainty about when and forwhom ligation has clinical benefit.

In a meta-analysis and RCT revealed that, ligation comparedto medical treatment was found tobe associated with increased odds of neuro-developmental impairment,CLD, and ROP.Incidence of complications up to 44%. The major complicationsare post ligation cardiac syndrome, acute kidney injury, vocal cord paralysis,prolonged mechanical ventilation, BPD.[2]

Prophylactic surgical ligation provides no benefit in terms of IVH, and surgical ligation itself may be an independent risk factor for poor neurodevelopmental outcome.

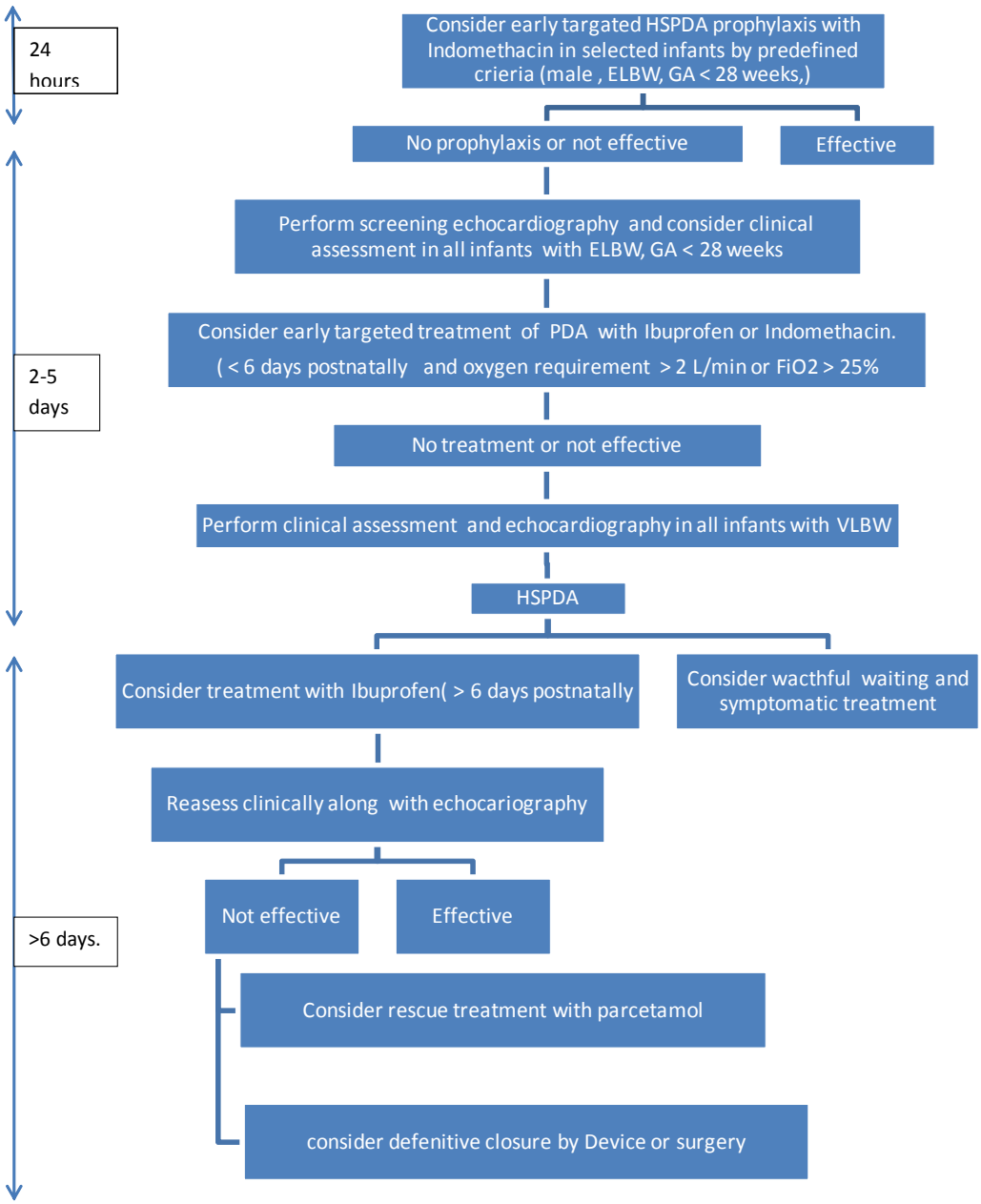

FIGURE 1: Treatment algorithm of PDA in the preterm infant. The proposed treatment algorithm incorporates the following 4 main treatment categories into a stepwise approach: (1) early targeted prophylaxis, (2) targeted therapy of asymptomatic infants (>6 days after birth), (3) symptomatic treatment of HSPDA (?6 days after birth), and (4) late symptomatic treatment after watchful waiting or rescue treatment.[4]

TREATMENT FOR TERM INFANTS.[1]

In case of HSPDA, the term infants are initially managed with pharmacotherapy. Those who not responded , are managed symptomatically with diuretics to treat pulmonary overcirculation followed by ultimate definite decision for PDA closure (either by surgery or transcatheter). Here consideration to be taken on the direction of ductal shunting and pulmonary artery systemic pressure and/or pulmonary vascular resistance indexed to systemic pressure. The frequency and timing of outpatient follow-up are determined on the basis of PDA classification. According to 2011 guidelines from the American Heart Association on cardiac catheterization in pediatric heart disease, transcatheter PDA occlusion is indicated in Term infants and older children patients for the treatment of an HSPDA with "left-to-right shunt that results in any of the following: congestive heart failure, failure to thrive, pulmonary overcirculation, or an enlarged left atrium or left ventricle, provided the anatomy and patient size are suitable"(class I recommendation, level of evidence B).

In case of PDA With Associated PAH in Term Infants and Older children : In this context, health care providers may consider transcatheter closure following short-term pulmonary vasodilator therapy.

Conclusions:

With increase chance of survival in ELBW newborn, PDA remains an important cardiovascular condition among them. Recent advances in early detection of HSPDA; pharmacological treatment (with paracetamol) along with transcatheteric management in newborn age improves morbidity and mortality associated with HSPDA.

ABBREVIATIONS

PDA – Patent ductus arteriosus

BNP – B-type natriuretic peptide

CLD – chronic lung disease

BPD – Bronchopulmonary Dysplasia

IVH – intraventricular hemorrhage

LA/Ao –left atrium to aortic ratio

FIO2 – fraction of inspired oxygen

MAP – Mean airway pressure

NCPAP – nasal continuous positive airway pressure

Vmax – maximum velocity

NEC – necrotizing enterocolitis

HFOV – high-frequency oscillatory ventilation.

ROP – retinopathy of prematurity

ELBW – Extremely Low Birth Weight Baby

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SRY Gene Negative 46 XX Testicular Disorder Of Sexual Differentiation: A Rare Case ofSex Reversal Syndrome

Dr Kakali Roy*, Priyanka Gupta,Soumik Goswami*****

**Assistant Professor, Department of Pediatrics, Nilratan Sircar Medical College and Hospital, Kolkata*

***Consultantin Department of Pediatrics, Krishna Institute of medical sciences, Kondapur, Hyderabad.*

****ClinicalTutor in Department of Endocrinology, Nilratan Sircar Medical College and Hospital, Kolkata*

Abstract

46XX testicular disorder of sexual differentiation (DSD) is a rare form of gender dysplasia comes under sex reversal syndrome, characterized by inconsistency between chromosomal and gonadal sex. A child of 46XX testicular DSD tested negative for SRY gene and had heterozygous mutation in RSPO1 gene whose homozygous loss-of-function mutation is reported to cause similar phenotype or sex reversal. This case aims of adding a new phenotype-genotype to the previously reported cases.

Keywords: SRY negative XX testicular DSD, ambiguous genitalia, sex reversal syndrome, RSPO1 gene

Sex reversal syndrome (SRS) is a rare disorder of sex differentiation (DSD) which is characterized by discrepancy between the gonadal and chromosomal sex. It includes 46, XY females and 46, XX males [1]. 46, XX male was first reported by Albert de la Chapelle in 1964, hence is also known as de la Chapelle syndrome. It has an incidence of 1:20000–1:100000 of live male births [2]. The sex determining region Y (SRY) gene was once considered essential for the development of internal and external phenotype of male. Recent reports suggest that male phenotype can develop in the absence of SRY gene. Approximately 80-90% of individuals with 46XX testicular DSD are SRY positive [3] however the exact etiopathogenesisof SRY negative cases is not known. The proposed mechanisms include over expression of pro-testis genes (SOX-3, SOX-9 or SOX 10) or decreased expression of anti-testis genes (DAX-1, WNT-4 and RSPO1 gene) [4].

Here an extremely rare case of SRY negative 46, XX testicular DSD case is being reported, who was heterozygous for RSPO1 gene mutation, with the aim of adding a new phenotype-genotype to the previously reported cases of SRY negative 46XX testicular DSD.

Case Report

A 8-month-old child born out of non-consanguineous marriage was brought with atypical genitalia and was being reared as a male baby. Birth and developmental history were normal. On genital examination, child had penoscrotal hypospadias, microphallus [Stretched penile length (SPL): 2.2 cm], bifid scrotum with normal rugosity and bilaterally palpable gonads within scrotum (Fig1). External masculinisationscore (EMS) was 6. There were no features of dysmorphism or palmoplantar hyperkeratosis. Anthropometry, other systemic examination, hearing assessment and ophthalmological examination were normal. There was a family history of atypical genitalia in the child's cousins (maternal aunt and uncle's children) but they had not been medically evaluated so far.

Ultrasonography revealed presence of Wolffian duct derivatives (epididymis, spermatic cord and seminal vesicles);gonads within scrotum had homogeneous echotexture with adjacent epididymis and no identifiable cystic area suggesting the gonads as testes(right testis: 0.98*0.64*1.29 cms; left testis: 1.24*0.57*0.89 cms). Ovary and Mullerian structures were absent on imaging. On biochemical investigations, electrolytes were normal (Na/K: 142/4.9 mEq/L), anti-Mullerian hormone was in male range (>46ng/mL) and other hormonal analysis was normal for age(LH/FSH:0.13/0.89mIU/mL, testosterone: <0.15nmol/L, Dihydrotestosterone: 11.70pg/mL, oestradiol: <10pg/mL). Meanwhile karyotype revealed 46XX chromosomal patterns and FISH analysis was negative for SRY gene.Clinical exome using next generation sequencing (NGS)

Correspondance : Kakali Ray Assistant Professor, NRS Medical College and Hospital Email :kakaliroy.nrs2007@gmail.com

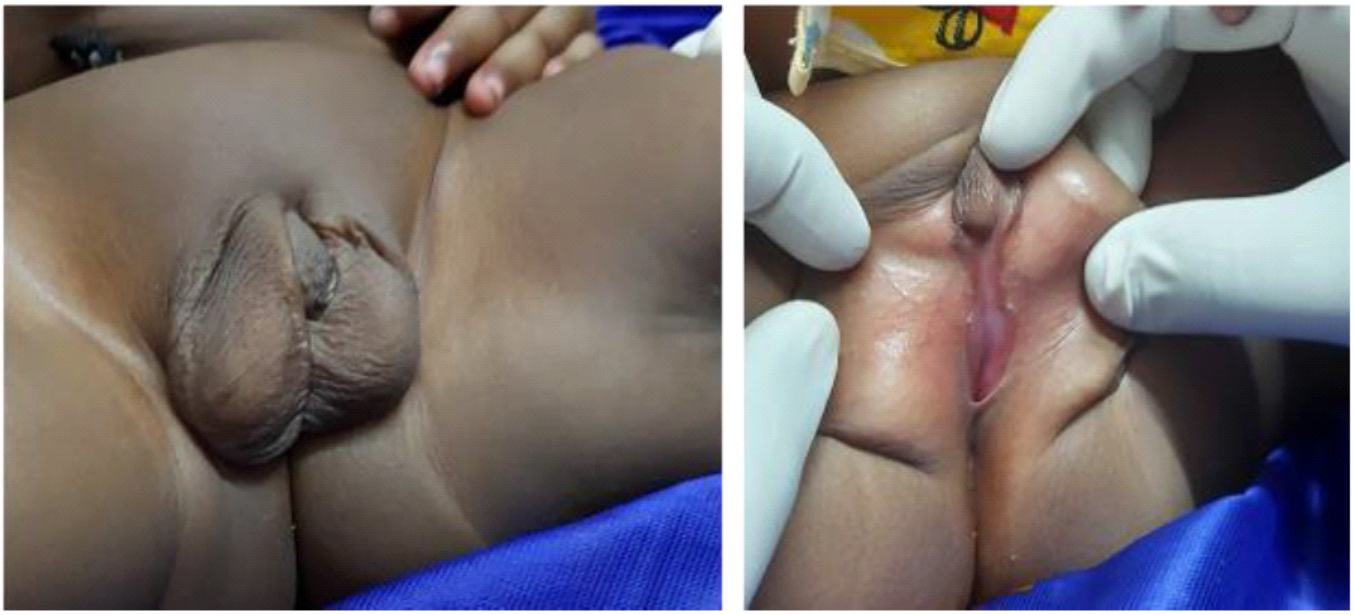

Fig1: 8-month male child with atypical genetalia: Bifid scrotumwith normal rugosity, microphallus, penoscrotal hypospadias and bilateral testes in scrotum.

method revealed heterozygous mutation in RSPO1 gene [Exon6, c.512G>C (p.Gly171Ala)] of uncertain significance (homozygous loss of function mutation in RSPO1 gene is reported to cause 46 XX testicular DSD) and was negative for mutation in more commonly involved Pro-testes genes like SOX9, SOX3, SOX8, SOX10, DAX1 andWNT4. Due to financial constraints and unwillingness of the family to undergo testing despite multiple counseling, further work up with MLPA analysis for detection of any missed deletion of RSPO1 gene and/or duplication of genes including SOX9, SOX3 etc andgonadal biopsy for microscopic evidence of ovarian tissue and/or dysgenetic testes could not be performed. With a provisional diagnosis of 46, XX SRY negative testicular DSD, parents were counselled regarding the management and prognosis, and the patient was referred for male genitoplasty including hypospadias repair.

Discussion

Sex determination and differentiation of gonads is a complex process and requires balance in the timing and level of expression of various genes. Ovarian development was once thought to be a passive process and SRY gene as essential for testicular development. RecentlySOX9, SOX3, SOX10, DMRT1 and FGF9have been identified which function downstream of SRY gene and play a critical role in testicular differentiation. Up regulation of these genes in the form of duplication or triplication can result in testicular or ovo-testicular differentiation in XX individualsin the absence of SRY gene [4].

Decreased expression of anti-testis genes including RSPO1, DAX-1 andWNT-4, leading to SRY negative XX male is extremely rare. RSPO1 gene is located on chromosome 1p34.3 and encodes for R-spondin protein which synergizes with WNT4 to stabilize ß-catenin in XX gonads leading to normal ovarian development. Biallelic loss-of-function mutation of RSPO1 geneis reported to cause46XXSRS, palmoplantar hyperkeratosis and predisposition to squamous cell carcinomaof the skin. Homozygous RSPO1mutation leading to testicular or ovo-testicular differentiation and ambiguous genitalia in 46 XX male was first described by Vernole et al. in 2000. Since then, total of 9 cases from 5 families have been reported with this rare syndrome [5]. Heterozygous mutation in exon5 of RSPO1 gene has also been Children with 46, XX SRY negative testicular DSD may present with ambiguous genitalia, failed puberty or infertility. Infertility is due to absence of 'azoospermia factor' in the Yq locus leading to absence of spermatogenic cells and testicular atrophy. Such patients should be offered adoption or sperm donation to father a child. Hormone replacement therapyat the pubertal age is essential

to maintain the physical and sexual well-being of the individual. They should also be counselled for self-examination and regular evaluation, keeping in mind the risk of gonadoblastoma, although reported rate of testicular tumor in such cases is low even with dysgenetic testes probably because of absent Y chromosome [7,8].

Our child had 46, XX male testicular DSD. Presence of functional testis in our child was supported by absence of Mullerian structure, male range AMH, masculinization of internal and external genitalia. Histopathology of gonad is confirmatory which could not be performed in our case. Work up was negative for SRY gene, mutation in pro-testis and anti-testis gene. Whole exome sequencing revealed a novel heterozygous mutation in exon6 of RSPO1 gene, significance of which is uncertain at present. MLPA for copy number variations in SOX9, SOX3 or SOX10 could not be performed.

Care of children with DSD requires a multidisciplinary approach along with patient's representative on the same table to provide a practical solution to the family [9]. Gender assignment, psychosexual orientation, fertility, risk of gonadoblastoma and irreversible nature of genital surgeries are main medical concerns while parents are anxious about social stigma, disclosure to other relatives, financial burden, social and emotional functioning of the child.

To conclude, we report a rare case of 46XX SRY negative testicular DSD, an extremely rare form of SRS, who was heterozygous for RSPO1 gene mutation of unknown significance which has not been reported previously.

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A Curious Case of Freeloaders in The Liver And Lung

Dr Joydeep Das
Professor and head ,Dept of Pediatrics
Jagannath Gupta institute of medical sciences, Budge budge

Abstract :

Background: Hydatid cyst is an important health disease caused by parasitic infestation which is endemic in many sheep and cattle raising areas. It is relatively uncommon in children. Liver hydatid cyst affects approximately 95% of the world's echinococcosis cases.

Clinical Description: A 9-year-old male residing on Bihar presented to paediatric emergency of our medical college with history of paint in the left chest & abdominal pain for last 1year.Abdominal pain is colicky in nature associated with intermittent fever which is radiating to thehypochondric and epigastric region.

Management: CECT whole abdomen showed feature of multifocal cystic lesion on segment V & VII of liver with echinococcus antibody positivity. The cyst was excised and a course of anthelminthic was given.

Conclusion: In children the incidence of liver hydatid cyst increased by age. A conservative surgical technique (cystotomy and partial pericystectomy) is sufficient in most cases. Mostly the postoperative results are favorable.

Keywords: Liver Hydatid Cyst, Abdominal Mass, Diagnosis, Treatment, Children

Introduction :

Human echinococcosis is a zoonotic disease.Echinococcosis occurs in 4 forms:

- cystic echinococcosis, also known as hydatid disease or hydatidosis, caused by infection with a species complex centred on Echinococcus granulosus;
- alveolar echinococcosis, caused by infection with E. multilocularis;
- two forms of neotropical echinococcosis: polycystic caused by infection with E. vogeli; and
- unicystic caused by E. oligarthrus.[1]

A number of herbivorous and omnivorous creatures act as halfway of Echinococcus. They ended up tainted by ingesting the parasite eggs in sullied nourishment and water, and the parasite at that point creates into larval stages in the viscera.

Correspondance : Joydeep Das, Professor, JIMS, Budge Budge. Email : jddasjoydeep@gmail.com

Carnivores act as conclusive for the parasite, and harbours the develop tapeworm in their digestive tract. The conclusive are contaminated through the utilization of viscera of that contain the parasite larvae.[2]

Humans act as so-called intermittent accidental host in the sense that they obtain contamination in the same way as other host , but are not included in transmitting the contamination.[3]

The liver is the most commonly included organ with the right lobe being most regularly influenced. All sorts of Hydatid Cyst can be seen inside the liver. Type I Hydatid Cyst ended up particularly imperative when they apply a mass impact. The conclusion is moderately simple in endemic locales and in single Hydatid cyst. In any case, a basic liver cyst along with a singular Hydatid cyst as well as polycystic liver infection in the presence of multiple type 1 Hydatid cyst can cause symptomatic issues. Living in endemic locales is vital clue along with research facility discoveries.

Case scenario

A 9-year-old male residing on Bihar came to pediatric emergency of our college with history of pain in the left side of the chest which was radiating over the right hypochondric and epigastric region of abdomen. There was also history of colicky associated pain with non-bilious vomiting.History of intermittent fever was noted which was relieved after giving antipyretics. There was no history of mass effect, abdominal distension or palpable lump or jaundice. Also, there was no history of altered bowel habit or dribbling during micturition.

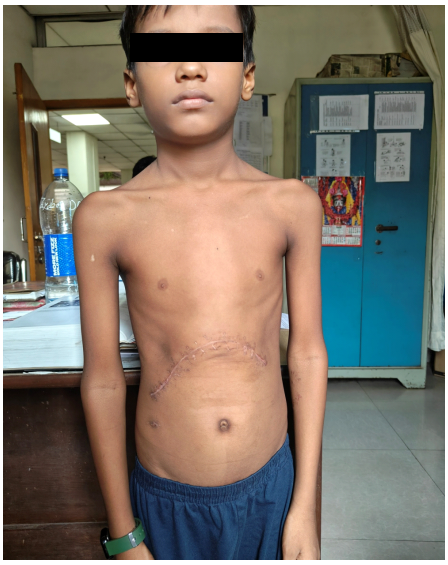

Fig 1: Index case showing scar mark over upper abdomen in post-operative follow up.

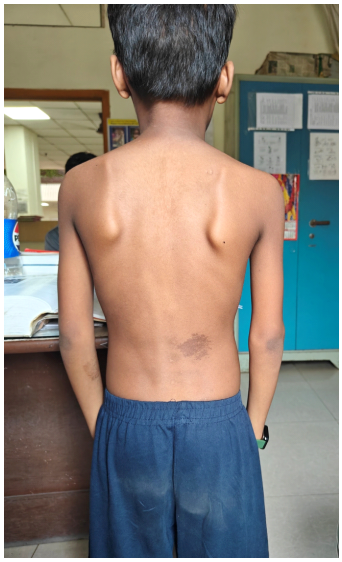

Fig 2: Index case showing drooping of scapulae on right side

Ultrasound whole abdomen shows features of hydatid cyst in liver and contrast enhanced computerised tomography shows multifocal non enhancing cystic lesion with enhancing septae and soft calcific foci on segment V &VII of liver (1.3x2.8x3.8 cm).

Chest Xray revealed haziness in left middle and lower zone of lung and Contrast enhanced computerised tomography also shows cystic lesion in the lung.

Echinococcus IgG antibodies was also positive.

Management

Patient was admitted and after initial routine examination, radiological examination was done. Albendazole was started and the patient underwent excision of hydatid cyst in the liver. Microscopic examination of the excised mass also revealed features of hydatid cyst.

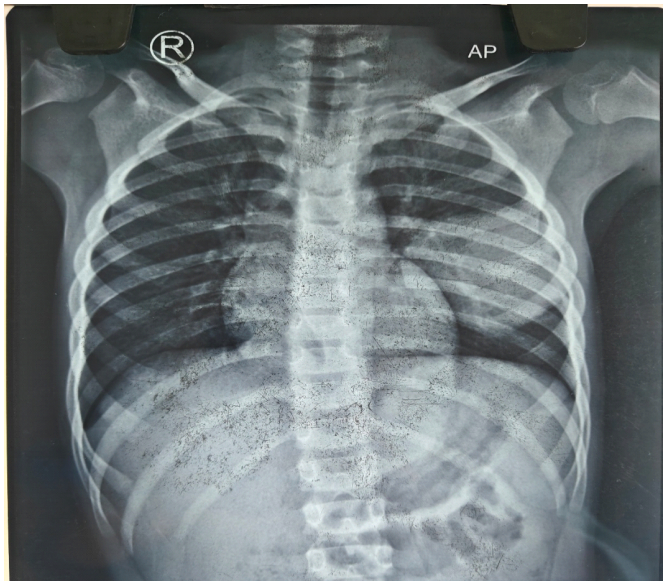

Fig 3: An oval-shaped, well-defined and rounded homogeneous density mass is observed in left lung.

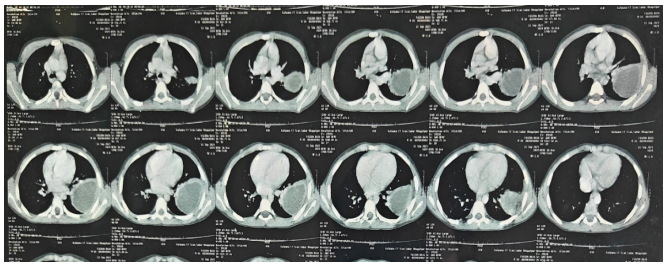

Fig 4: Images showing cystic lesion in anterior and lateral basal segments of the lower lobe of the left lung.

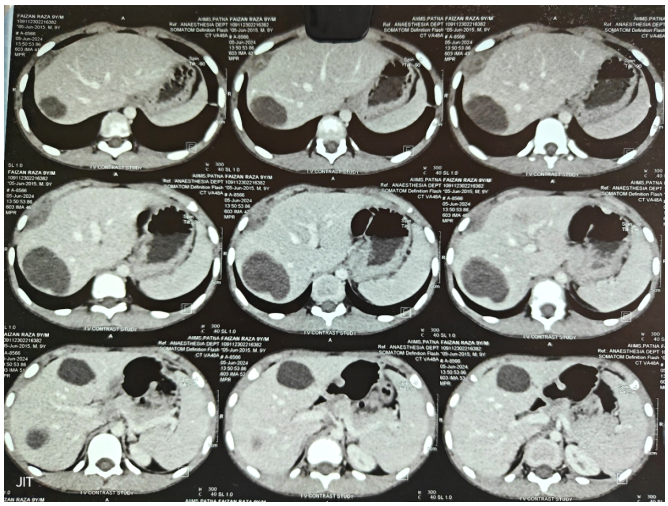

Fig 5: Well-circumscribed, round, unilocular hypodense cystic lesion centred in segment V and VII of the right hepatic lobes.

As for lung cyst, patient was given a course of albendazole and was advised for regular follow-up.

Discussion

Hydatid disease is still a national problem in highly endemic countries and needs epidemiologic prevention for its eradication[5].A risk of surgery is that hydatid cyst ruptures and spreads tapeworm heads throughout the patient's body. To reduce this risk, we prescribe high doses of the drug albendazole in conjunction with surgery [6].Hydatid cysts in children involves more commonly lungs than liver but Schitogullri[7] believes that liver cysts are more common in childhood and Talaizadeh[8] found nearly equal incidences (41% -43%) of hydatid cyst in lung and liver in pediatric patients. Ultrasound (US) reveals an anechoic, well-defined cystic lesion with small echogenic foci or “falling snowflakes” consistent with hydatid sands changing with patient position . Ultra Sonogram should be the first imaging choice in abdominal hydatid cysts with sensitivity rates between 93% and 97%.[9]

Therapy protocol for hydatid cyst

Stage	Size	First-option treatment	Alternative treatment
Refusal of intervention or contraindications for invasive treatment		**ABZ (6 months)**	
CE1, CE3a	Small	Only ABZ (6 months)	PAIR + ABZ (1 month)
	Medium	Surgical treatment + ABZ (1–6 months)	PAIR + ABZ (month)
	Large	Surgical treatment + ABZ (1–6 months)	MoCaT + ABZ (1 month)
CE2, CE3b	Small	Only ABZ (6 months)	MoCaT + ABZ (1 month)
	Medium	Surgical treatment + ABZ (1–6 months)	MoCaT + ABZ (1 month)
	Large	Surgical treatment + ABZ (1–6 months)	MoCaT + ABZ (1 month)
CE4, CE5	Any diameter	“Watch-and-Wait” attitude	“Watch-and-Wait” attitude
Complicated cysts, no matter what stage	Any diameter	Surgical treatment (+/- interventional endoscopy in case of rupture into the biliary tract)+ ABZ (6 months)	Surgical treatment in case of rupture; Percutaneous drainage in case of infection + ABZ (1 month)

Fig 6: Treatment of hydatid cyst 4(PAIR -puncture, aspiration, injection, and re-aspiration, MoCaT-modified catheterization technique, ABZ-albendazole).

Computerized Tomography should be performed in cases of uncommon locations of the disease. Computed tomography (CT) detects a water-attenuating lesion with well-defined borders.Once the ingested embryos enter the portal circulation, they primarily affect the liver, but can be spread hematogenous to all organs and tissues except hair. Other organ involvement is generally secondary to liver involvement, especially for lungs, spleen and kidneys. Another mode of spread other organs is cyst rupture into neighbouring organs or peritoneum. The external rupture of the cyst is generally iatrogenic during cyst surgery or trauma.Surgical treatment remains the "gold standard" in therapy, but minimally invasive methods with high applicability, less frequent complications and lower hospital requirements are starting to gain ground.[10]

A mortality rate between 0.29% and 0.6% has been reported. In symptomatic cases, the clinical manifestations are highly variable and depend on the following: (a) the organ involved; (b) size and site of the cyst; (c) interactions between expanding cysts and adjacent organs; and (d) complications caused by rupture of the cyst[11,12]. Pre-operative diagnosis of cystic echinococcosis is mandatory to prevent anaphylaxis or local recurrence.[13]

Conclusion

Echinococcus is a zoonotic parasitic disease with global existence. Though it can involve any organ, liver and lungs are the most commonly involved organ. Patients remain asymptomatic for a longer period as the cyst grows slowly. In children the incidence of liver hydatid cyst increased by age. The most involved organ was lung and then liver. Diagnosis is usually based on radiology supported by serological testing. Surgery is the treatment of choice of hydatid cyst in the liver.

Lesson Learnt

- A high index of suspicion and detailed diagnostic work up is required for early diagnosis of Hydatid cyst.
- Multidisciplinary holistic approach including a paediatrician, radiologist, surgeon is needed for early diagnosis .Prognosis of the disease is excellent after cystectomy.

Funding: No funding sources.

Conflict of interest: No conflict of interest.

Ethical approval: The study was approved by the Institutional Ethics Committee.

Confirmation of diagnosis

Proper clinical examination, history,microbiological findings and radiological imaging techniques is done for the confirmation of diagnosis of Hydatid cyst

Declaration of patient consent

I certify that I have obtained all appropriate consent from the patient. The patient understand that her name and initials will not be published and due efforts will be made to conceal the identity but anonymity cannot be guaranteed.

Limitations

The lung cyst was not resected due to smaller sizeand as the lung expansion is within the normal limit so a trial of pharmacotherapy by albendazole was given.[14,15]

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Apnoea – Easy To Treat, Easier To Overlook

Dr. Sumedha Bhattacharyya*, Prof Taraknath Ghosh**
**Pgt, Sail – Isp Hospital, Burnpur, **Professor, Burdwan Medical College*

Introduction

30-45% of preterm neonates exhibit periodic pattern of breathing, characterized by >= 3 respiratory pauses each lasting >= 3 seconds each. It is a normal entity in preterm infants, reflective of immaturity of respiratory control system and does not merit treatment.

In contrast, apnoea is pathological cessation of breathing lasting longer than 20 seconds or for shorter duration in presence of bradycardia (<80bpm) and significant desaturation (<80-85%).

Etiology:

1) apnoea of prematurity

Related to immaturity of brainstem and peripheral chemoreceptors resulting in abnormal ventilator response to hypercarbia and hypoxia along with immature reflex responses.

Apnoea occuring in first 24 hours of life and beyond 7 days of life is more likely to have a secondary cause than being apnoea of prematurity.

Secondary causes of apnoea include:

2) temperature instability –

Hypothermia and hyperthermia

3) metabolic

Acidosis, hypoglycemia, hypocalcemia, hyponatremia, hypernatremia

4) hematological –

Anemia, polycythemia

5) neurological –

Intracranial infections, intracranial hemorrhage, seizures, perinatal asphyxia and parenteral transfer of drugs like magnesium sulfate and narcotics

6) pulmonary –

Rds, pneumonia, pulmonary hemorrhage, obstructive airway lesions, hypoxemia, hypercarbia, airway obstruction due to neck flexion

7) cardiac –

Chd, hypotension, hypertension, pda, heart failure

Monitoring – all neonates <35 weeks of gestation should be monitored for apnoea in the first week of life.

TYPES

Central (40%)	Obstructive (10%)	Mixed (50%)
• Cessation of inspiratory efforts due to reduced central drive	• Infant unable to breathe due to obstructed airway	• Both components are present

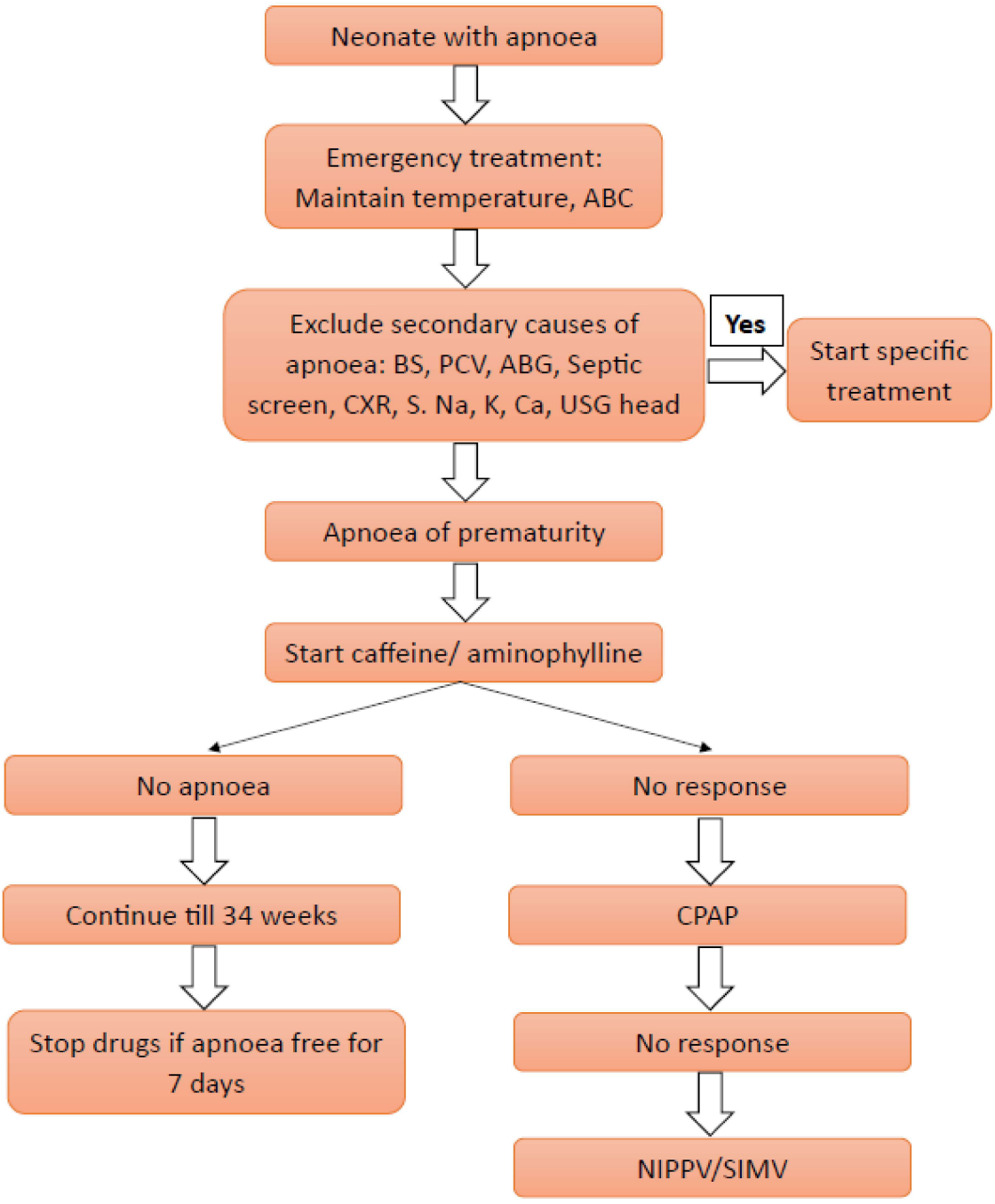
Prevention

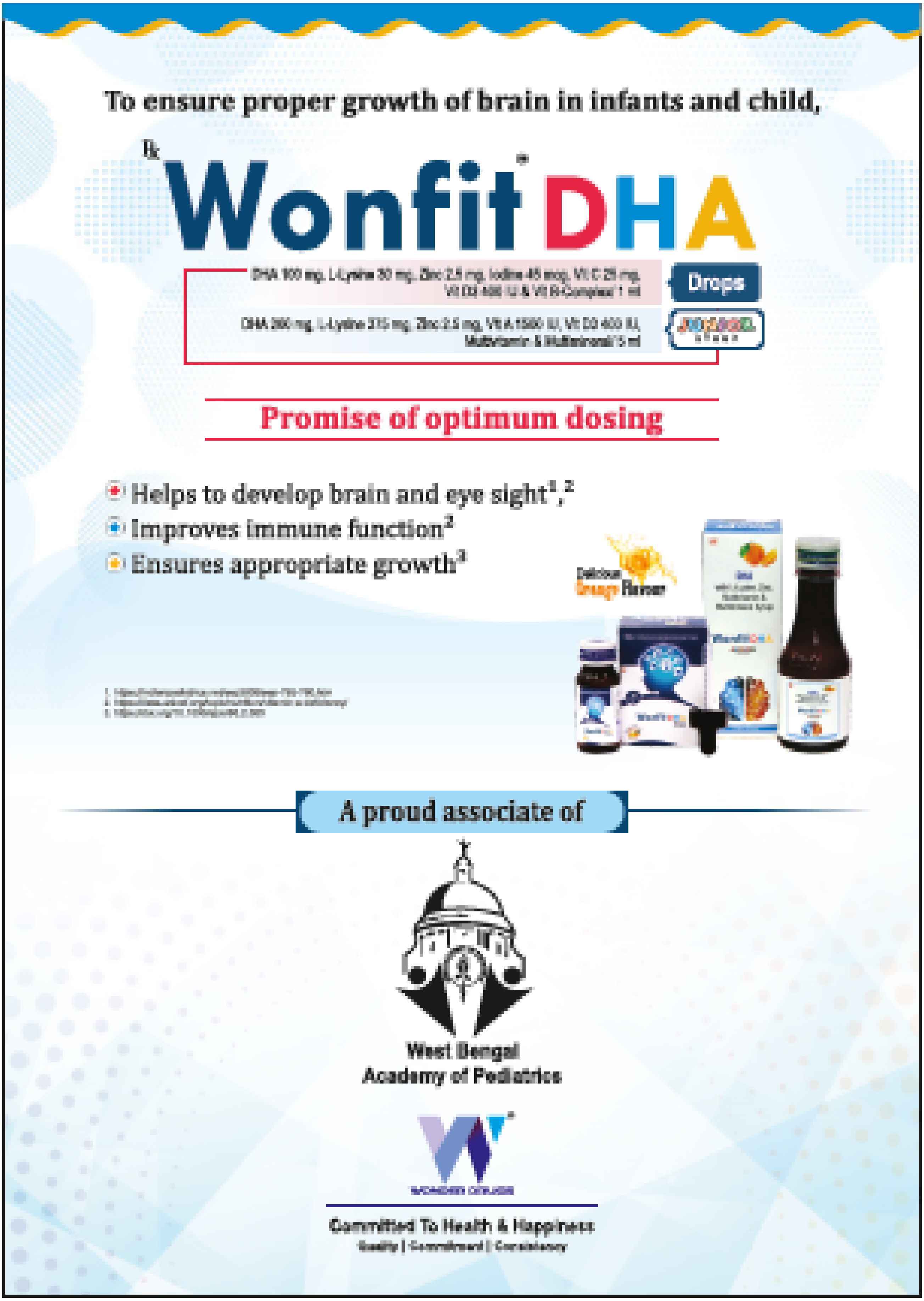
1) infant to be nursed in servo – controlled radiant Warmer or incubator to avoid fluctuations in body Temperature
2) head and neck to be maintained in neutral / slightly Extended position
3) upper airway patency to be maintained
4) oxygen saturation to be maintained between 90-95% By rational use of supplemental oxygen

Neurodevelopmental outcome – the precise of Effects of fluctuations is breathing, heart rate and Saturations during the episodes of apnoea is not Clearly defined.retrospective studies have suggested. That recurrent episode of apnoea are associated With worst neurodevelopmental outcomes at 1-2 Years of age.

Correspondance : Dr Taraknath Ghosh, Professor, Burdwan Medical College *Email tnghosh39@gmail.com*

MANAGEMENT


Neonate with apnoea
Emergency treatment: Maintain temperature, ABC
Exclude secondary causes of apnoea: BS, PCV, ABG, Septic screen, CXR, S. Na, K, Ca, USG head
Yes
Start specific treatment
Apnoea of prematurity
Start caffeine/ aminophylline
No apnoea
Continue till 34 weeks
Stop drugs if apnoea free for 7 days
No response
CPAP
No response
NIPPV/SIMV



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