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Pediatric Education and Research

July – September 2020
Volume 8 Number 3

Contents

Original Research Article

Pediatric Education and Research
Volume 8 Number 3, July–September 2020
DOI: https://dx.doi.org/10.21088/per.2321.1644.8320.5

To Study the Awareness and Significance of Blood Pressure Measurement of Children Among Parents

Pranita Tambe[1], Sunil Natha Mhaske[2], Veenita Pande[3]

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How to cite this article:
Pranita Tambe, Sunil Natha Mhaske, Veenita Pande. To Study the Awareness and Significance of Blood Pressure Measurement of Children Among Parents. Pediatr Edu Res. 2020;8(3):97–101

Abstract

Hypertension is a common disease associated with high mortality and morbidity. With globalization bringing more lifestyle modifications, adolescents are exposed to multiple risk factors like obesity, diet, academic stress, inactive lifestyle combined with hereditary risk factors. Early diagnosis of hypertension is an important strategy in its control, effective treatment and prevention of complications.

Keywords: Hypertension; Prehypertension; Parents; Blood Pressure.

Introduction

Hypertension and Prehypertension are one of the commonest diseases with worldwide prevalence of 1 billion. 3rd National Health and Nutritional Assessment Survey revealed that in United States America, one-third of people were unknown of hypertension.[1] Epidemiological transition with increasing burden of cardiovascular risk factors is evident in adults and children both.[2] The data on the prevalence of prehypertension and hypertension in children show large regional differences in India.

Early diagnosis of hypertension and Pre-hypertension is an important strategy in its control. Previous studies have documented that hypertension may begin in adolescence, perhaps even in childhood. Elevated blood pressure, systolic or diastolic at any age, in either sex is a contributor for all forms of cardiovascular disease.[4] Identifying and modifying risk factors reduces the incidence and complications in young and adult. Prevalence of hypertension varies across countries and states. Hypertension - multifactorial disease, is influenced by genetics, race, geography, cultural and dietary patterns.

Paediatrics hypertension is seen in 2% to 5% of all Paediatrics patients. It is one of the top five chronic diseases in children and adolescents. Pediatric hypertension affects approximately 65 children per million.[5]

Hypertension is a common disease associated with high mortality and morbidity. With globalization bringing more lifestyle modifications, adolescents are exposed to multiple risk factors[6] like obesity, diet, academic stress, inactive lifestyle combined with hereditary risk factors. Early diagnosis of hypertension is an important strategy

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Table 1: Criteria for diagnosis of Childhood hypertension[7].

Age	Normal	Prehypertension	Stage I Hypertension	Stage II Hypertension
3-11 years	<90th percentile	90th–<95th Percentile	95th–99th percentile + 5mm Hg	>99th percentile + 5 mm Hg
12-17 years	<90th percentile	90th–<95th percentile or > 120/80 mm Hg	95th-99th percentile + 5 mm Hg.	>99th percentile + 5 mm Hg.

in its control, effective treatment and prevention of complications.

For many children, hypertension is only diagnosed when it is severe, or once they reach adulthood. However, the importance of early and accurate diagnosis cannot be overstated, given the long-term health consequences of untreated hypertension and the fact that pediatric hypertension is a diagnostic indicator for some serious underlying medical conditions.

For the Children Aged 3-11 and 12-17 Yrs of Age (Table 1.1).

1. 90th percentile indicates a healthy child.
2. 90th - 95th percentile indicates a prehypertension stage.
3. 95th -99th percentile +5mm hg indicates stage 1 hypertension.
4. > 99th percentile + 5mm hg indicates stage 2 hypertension.

The prevalence of childhood obesity, the risk of developing left ventricular hypertrophy, and evidence of the early development of atherosclerosis in children would make the detection of childhood prehypertension and hypertension important to reduce long-term health risks.[8] Guidelines for the screening for and diagnosis, evaluation, and management of hypertension in children have been available for 40 years.[9] Unfortunately, clinicians consistently fail to recognize the problem, and the majority of hypertensive children remain undiagnosed. Several reasons for this have been documented including lack of knowledge of the problem and the complexity of blood pressure standards among children, Parents and Pediatricians.

Aim and Objectives

- To study the awareness of blood pressure measurements of children amongst parents.
- To create importance of Hypertension in children and Parents.
- To make aware of Prehypertension is an emerging disease in adolescents and Parents.

Material and Methods

A Google doc questionnaire was created and was circulated to all Parents on WhatsApp. The questionnaire included simple questions like education of parents, awareness of Hypertension and Prehypertension, importance of measurements of their Childs blood pressure etc.

The results were interpreted according to their responses.

Observations

Total 280 parents responded in these questionnaires. The following observations were made from their responses.

Table 2: In These Questionnaires Out of 240 Participants 85.7% Were Father of Children.

Respondents	Number	Percentage
Father	240	85.7
Mother	20	7.15
Any other	20	7.15
Total	280	

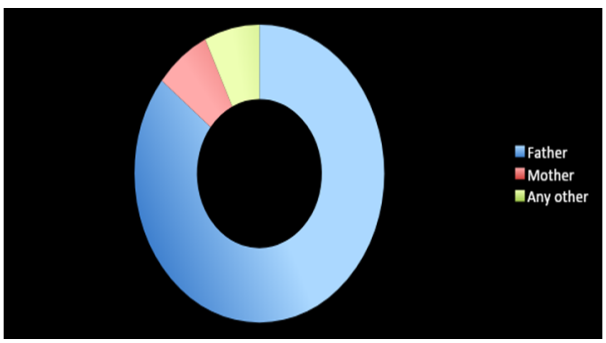

Fig. 1: Out of the 280 Members that Answered, Fathers of Children Were 240 in Number and 20 Were Mothers of Children, 20 Were Others.

Table 3: Qualification of Respondents.

Qualification	Number	Percentage
Matriculations	40	14.3
Graduates	80	28.6
Postgraduates	140	50
Any other	20	7.15
Total	280	

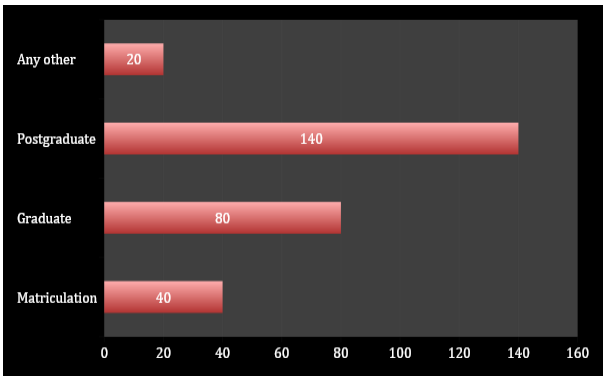

Fig. 2: Educational Qualification of the 280 members was as follows.

Educational Qualification of the 280 members was as follows (Table3, Fig. 2).

Post graduates –50%

Graduates –28.6%

Matriculation –14.3 %

And remaining participants that did not complete matriculations were 7.5%

Table 4: Number of Children.

Number	Number	Percentage
One	40	14.3
Two	200	71.4
Three	40	14.3
More than three	00	00
Total-	280	

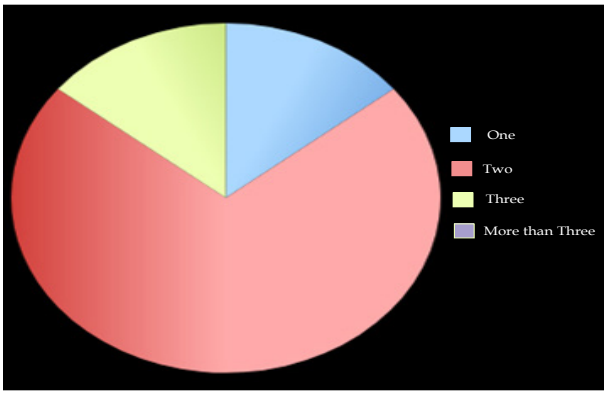

Fig. 3: Above graph shows 14.3% Parent had a Single Child , 14.3% had 3 Children and 71.4% had 2 Children.

Table 5: Age of Children.

	First child	Second child	Percentage
Below 10	14.3 % (40)	21.6% (60)	14.3
11-19	71.4 % (200)	71.4% (200)	71.4
More than 20	14.3% (40)	7.3%.(20)	14.3
Total-		280	

Table 6: Are You Aware of Hypertension (Raised Blood Pressure) in Children?

	Response	Percentage
Yes	140	50
No	140	50
Total	280	

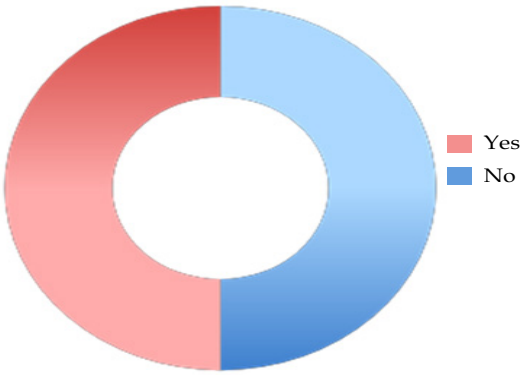

Fig. 4: Of the 280 People that Answered 50% of Them Knew About the Prevalence of Hypertension in Children.

Table 7: Are You Aware of Prehypertension (Phase Of Higher Blood Pressure Than Normal) in Children?

	Response	Percentage
Yes	120	42.9
No	160	57.1
Total	280	

Fig. 5: Out of the 280 People that Answered, 42.9% Knew About Prevalence of Pre Hypertension in Children and 57.1% Had No Knowledge of it.

Table 8: Have you Checked your Child's Blood Pressure any Time?

	Response	Percentage
Yes	160	61.5
No	120	38.5
Total	280	

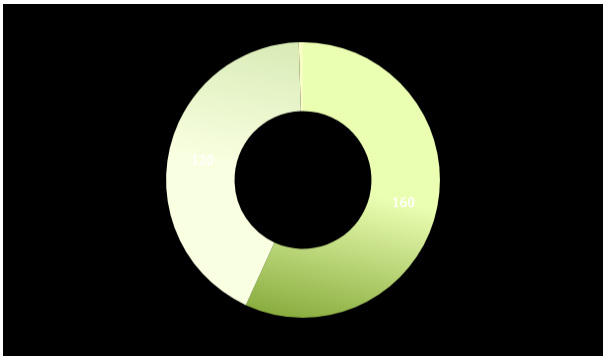

Fig. 6: Out of total 280 parents reviewed, 160 actually have checked BP of their child & 120 did not check it.

Table 9: Are you aware of effect of Junk food, overweight, school stress on blood pressure of child?

	Response	Percentage
Yes	140	50
No	20	7.1
May be	120	42.9
Total-	280	

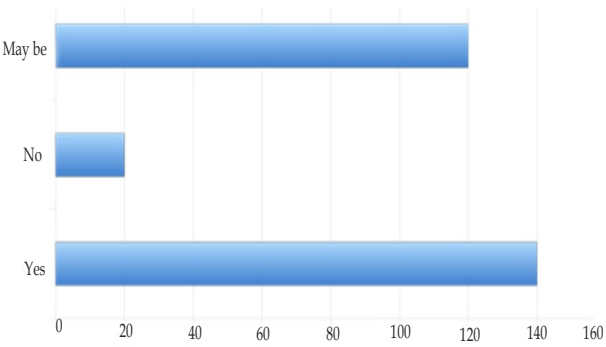

Fig. 7: Depicts the Awareness Among Parents About the Epidemiology of Pre Hyprtension and Hypertension, Findings Revealed that 50% of Them Were Aware of the Effect of Junk Food, Overweight, School Stress on Blood Pressure of Child.

7.1% were not aware and 42.9 % gave the answer as maybe.

Discussion

- Collect an accurate family history to identify primary and secondary forms of hypertension.
- Use standardized methods and suitable instruments for a correct measurement of blood pressure in the child and interpret the values according to the most extensive and updated tables.
- Monitor blood pressure during annual control visits from the age of three.
- Repeat the blood pressure measurement on at least three different occasions when values are observed that could indicate hypertension or high normal blood pressure.
- Learn to make a first differential diagnosis between primary and secondary forms of hypertension on the basis of clinical history, physical examination, targeted examinations.
- Send patients with suspect secondary hypertension to referral centers.
- Apply the principles of the dietary and behavioral interventions in the treatment of the primary forms.
- Send patients with suspect secondary hypertension and cases of primary hypertension who do not respond to dietary and behavioral therapy to specialist centers.
- Cooperate with the specialist centers in the follow-up of the hypertensive child.

Conclusion

Hypertension among the adolescent age group was alarmingly high; there was no difference in prevalence among government and private schools and among various types of curriculum. Awareness of hypertension was very low. There was no association with socio economic status. Periodic surveys should be done in schools to identify the "at risk" groups.

Conflicts of interests: No

Funding: No

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Original Research Article

Pediatric Education and Research
Volume 8 Number 3, July–September 2020
DOI: https://dx.doi.org/10.21088/per.2321.1644.8320.6

Study of Body Growth in Boys with Steroid Sensitive Nephrotic Syndrome

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How to cite this article:

Nagaraju A S, A K Bhalla, Deepti Suri et. al., Study of Body Growth in Boys with Steroid Sensitive Nephrotic Syndrome. Pediatr Edu Res. 2020;8(3):103-108.

Abstract

Background: Steroids are mainstay of the treatment of Nephrotic syndrome and their effect on growth of children studied by earlier workers for lack of consensus, presented conflicting views. Therefore, in this study, we attempted to study pattern of growth of Indian boys with steroid sensitive nephrotic syndrome in terms of some selected anthropometric parameters.

Methods: 121 boys between 9 to 16 years of age diagnosed as cases of steroid sensitive Nephrotic syndrome were measured for Weight, Height, Chest Circumference, Biacromial diameter, Bicristal diameter, Triceps skinfold thickness and Subscapular skinfold thickness at half yearly age intervals following a mixed longitudinal growth research design. Tanner's method was used to compute mean (±SD) distance and velocity growth values for different body parameters of boys.

Results: Growth of height, weight, chest circumference, biacromial diameter and bicristal diameter in boys with steroid sensitive Nephrotic syndrome in general, was found to be compromised as compared to their normal counterparts but was severely affected for height between 14 to 16 years of age and they became short statured individuals. Exceptions to this were triceps and subscapular skinfold thicknesses which in general, measured fatter than normal boys.

Barring triceps skinfold thickness, peak growth velocity for all other body parameters measured amongst boys with SSNS was attained at the same age of 14.5 years. Attainment of peak height velocity (PHV) and peak weight velocity (PWV) as compared to their normal Indian and western counterparts was found to be substantially delayed and also measured lesser in magnitude.

Conclusions: The relatively impaired auxological status recorded amongst boys with SSNS appears to be due to influence of chronic nature of the disease itself as well as effect of steroid therapy which is known to impair growth of children. On the contrary, growth of subcutaneous fat measured in terms of triceps and subscapular skinfold thicknesses exhibited relatively fatter attainments amongst boys with SSNS than their normal counterparts.

Keywords: Nephrotic Syndrome; Growth; Steroids.

Introduction

Nephrotic syndrome is a manifestation of glomerular disease, typically characterized by heavy proteinuria (>40mg /m2 /hr), hypoalbuminemia (<2.5 g/dl), edema and hyperlipidemia (serum cholesterol >200 mg/dl).[1,2] The worldwide prevalence is approximately 12-16 cases per 100,000 children with an annual incidence of 2-7 per 100,000 children.[5] It occurs, most commonly in

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age of 2-8 years which corresponds to a period of relatively steady growth. Daily exposure to supraphysiologic concentration of glucocorticoids over prolonged period is known to affect growth of these children.[3] The loss of insulin-like growth factor-I (IGF-1) and IGF binding protein-3 (IGFBP-3) found in Nephrotic children may prompt growth retardation. In addition, glucocorticoid therapy is believed to be associated with elevation of serum IGF-I levels suggesting potential development of IGF resistance.[4] considered as one of main factors responsible for growth retardation.

Available information relating to effect of steroid therapy on growth of patients with steroid sensitive Nephrotic syndrome remains inconclusive as it lacks clear consensus. Studies conducted by earlier researchers[3,6-12,22-25] show that growth of steroid-sensitive nephrotic syndrome patients gets affected, while those carried out by others[17,18,19,20] reveal no effect on growth of these patients. However, studies by some workers[13-16] revealed effect of steroid therapy on growth of children with nephrotic syndrome during initial years of disease, as these patients ultimately became adults with normal height. It has also been further noticed that most of auxological information on these patients has emanated from developed western world, and their growth in majority of instances, was studied in terms of height, and hardly any attention was paid to study growth of other body parameters. As compared to western world, longitudinal studies conducted on the growth of Indian children with SSNS are scarce. Therefore, we attempted to explore and understand growth dynamics of some selected body parameters of adolescent boys with steroid-sensitive Nephrotic syndrome using mixed longitudinal growth research design.

Patients and Methods

121 boys between 9 to 16 years of age diagnosed as cases of steroid sensitive Nephrotic syndrome (as per criteria given by Indian Academy of Pediatrics)[1] who had successfully completed their treatment comprised sample for this study. These children were born to parents representing mixed socio-economic strata, and inhabited north western parts of India. These subjects were enrolled from the Nephrotic Clinic of Advanced Pediatrics Center, PGIMER, Chandigarh. Children with other chronic diseases, severe malnutrition and secondary Nephrotic syndrome were excluded from the study. Children who had received cyclophosphamide/ cyclosporine/levamisole and those who developed steroid resistance during course of the study were also not included.

Every child was measured for Weight, Height, Chest Circumference, Biacromial diameter, Bicristal diameter, Triceps skinfold thickness and Subscapular skinfold thickness at 6 monthly intervals with time tolerance of ±1 month in Growth Laboratory/Clinic of the department following mixed-longitudinal growth research design. Body weight was measured with the help of Electronic Weighing Scale up to the accuracy of ±50g. Height was measured using Stadiometer (Make:Holtain Ltd) upto accuracy of ±1 mm. Fiber glass tape was used to measure chest circumference up to ±1mm of accuracy. Harpenden Skinfold Caliper with a least count of 0.2 mm was used to measure skinfold thicknesses. A Spreading caliper was used to measure the two body diameters (i.e.biacromial and bicristal) up to accuracy of ±1 mm. Prior to actual data collection a thorough training with respect to all anthropometric measurements was provided to the investigator until magnitude of intra/inter-observer error became ±50g for body weight, ±2mm for height, ±0.2 mm for skinfold thicknesses and up to ±1 mm for chest circumference as well as two body diameters. Tanner's 1951 method[26] was employed to compute mean ±SD of distance (gross size) as well as velocity (rate of growth) related statistics from mixed- longitudinally obtained data for different growth parameters.

Results

Mean and standard deviation (SD) computed for different body parameters measured among boys with steroid-sensitive Nephrotic syndrome (SSNS) are shown Table 1. Height and Weight of children with SSNS showed a regular increase between 9 to 16 years of age (Fig. 1 and 2). This increase however, remained relatively sharper upto 13.5years, thereafter it became comparatively slower. As compared to their normal American (CDC 2000)[28] and Indian affluent counterparts (Agarwal et. al. 1992)[27], height of boys with steroid sensitive Nephrotic syndrome measured shorter throughout. (Fig. 1)

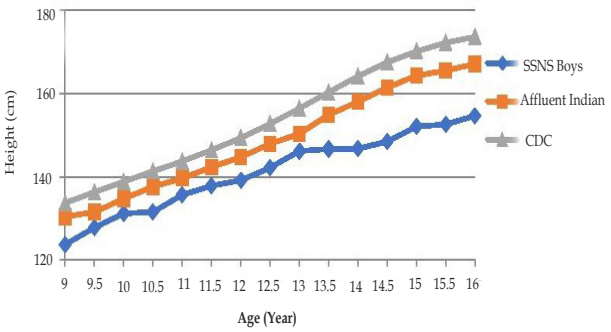

Fig. 1: Comparison of Mean Height (cm) of Normal Boys and Steroid-Senstive Nephrotic Syndrom Boys.

Children with SSNS in general, weighed lighter than their normal CDC (2000)[28] counterparts, but when compared with affluent Indian boys (Agarwal et. al.)[27] they depicted an inconsistent trend. (Fig. 2)

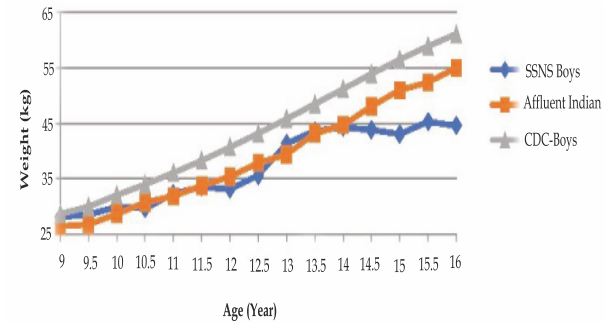

Fig. 2: Comparison of Mean Weight (kg) of Normal Boys and Steroid-Senstive Nephrotic Syndrom Boys.

Mean chest circumference (cm) amongst boys with SSNS grew regularly with advancement of age, yet magnitude of its increase was inconsistent. Growth of shoulder and hip measured in terms of biacromial diameter and bicristal diameter respectively, amongst boys with SSNS showed an uninterrupted increase in mean values between 9 to 16 years of age. As compared to other body parameters the pattern of growth of triceps and subscapular skinfold thicknesses exhibited highly fluctuating trend. In general, boys with steroid sensitive Nephrotic syndrome remained fatter than their normal affluent Indian counterparts depicting a tendency to become obese with advancement of age. (Table 1)

Yearly growth velocities computed for each of the body parameters measured in boys with steroid sensitive Nephrotic syndrome are shown in Table 2. Height growth velocity in boys with steroid sensitive Nephrotic syndrome in general increased regularly, to attain peak height velocity (PHV) measuring 2.25 cm/year between 14 to 15 years of age in magnitude. Weight velocity (kg/year) amongst boys with steroid sensitive Nephrotic syndrome experienced a regular increase to attain peak weight velocity (PWV) measuring 2.35 Kg/ year at 14.5 years. Thereafter, it showed sudden deceleration. The magnitude of yearly height and weight growth velocities measured lesser in boys with steroid sensitive Nephrotic syndrome when

Table 1: Mean and Standard Deviation of Height, Weight, Chest Circumference, Biacromial Diameter, Bicristal Diameter, Triceps Skinfold Thickness, Subscapular Skinfold Thickness in Steroid-Sensitive Nephrotic Syndrome Boys.

Age Interval (±year)	Height (cm) Mean ± SD	Weight (kg) Mean ± SD	Chest circumference (cm) Mean ± SD	Biacromial diameter (cm) Mean ± SD	Bicristal diameter (cm) Mean ± SD	Triceps skinfold thickness (mm) Mean ±SD	Subscapular skinfold thickness (mm) Mean±SD
9.0	123.7 ±8.13	28.1 ±9.22	62.3 ±6.90	25.8 ±2.75	18.6 ±2.19	13.1 ±6.23	11.8 ±8.58
9.5	127.7 ±6.61	28.6 ±6.34	62.5 ±5.94	26.4 ±2.20	18.7 ±2.28	11.2 ±5.22	8.9 ±6.19
10.0	131.1 ±5.13	29.9 ±3.90	62.9 ±3.83	27.2 ±1.72	19.3 ±1.35	11.1 ±4.07	8.8 ±4.55
10.5	131.5 ±5.85	29.6 ±4.54	62.9 ±4.39	27.2 ±2.01	19.3 ±1.68	10.7 ±4.21	8.7 ±4.08
11.0	135.6 ±6.93	32.3 ±6.31	63.1 ±4.46	28.0 ±2.09	19.7 ±1.40	11.1 ±3.89	8.2 ±2.80
11.5	137.8 ±7.94	33.5 ±7.17	64.3 ±5.12	28.3 ±2.35	19.9 ±1.77	12.5 ±5.04	9.1 ±3.34
12.0	139.1 ±7.34	33.1 ±7.07	63.9 ±5.81	28.6 ±2.38	19.7 ±1.52	11.7 ±7.04	8.7 ±4.63
12.5	142.1 ±6.66	35.6 ±7.04	65.6 ±6.40	29.2 ±2.00	20.3 ±1.94	12.8 ±5.79	8.9 ±3.89
13.0	146.1 ±9.66	41.3 ±10.93	69.7 ±7.41	29.9 ±2.19	21.1 ±1.77	16.1 ±6.14	12.4 ±6.65
13.5	146.6 ±8.87	43.6 ±10.44	71.2 ±8.52	30.2 ±2.20	21.6 ±2.54	17.2 ±7.63	15.6 ±9.09
14.0	146.7 ±5.43	44.2 ±10.31	72.0 ±10.30	30.3 ±1.79	21.6 ±3.33	19.9 ±9.57	17.3 ±11.07
14.5	148.4 ±7.14	43.8 ±7.97	71.7 ±7.73	30.5 ±2.21	21.7 ±2.89	17.0 ±8.32	14.2 ±11.14
15.0	152.0 ±8.32	43.0 ±9.33	70.1 ±5.57	31.7 ±2.98	22.1 ±1.34	12.1 ±5.65	9.2 ±3.98
15.5	152.5 ±9.60	45.3 ±8.92	72.5 ±6.59	32.3 ±2.55	22.7 ±2.35	14.6 ±6.05	11.5 ±5.91
16.0	154.6 ±5.11	44.5 ±7.73	72.7 ±8.28	32.9 ±1.28	22.6 ±1.59	13.9 ±7.05	11.1 ±6.02

Table 2: Mean and standard deviation of Yearly Height velocity (cm/year), weight velocity (kg/year), chest circumference velocity (cm/year), biacromial diameter velocity (cm/year), bicristal diameter velocity (cm/year), triceps skinfold thickness velocity (mm/year), subscapular skinfold thickness velocity (mm/year), in adolescent boys with Steroid-Sensitive Nephrotic Syndrome.

Age interval (±year)	Height velocity (cm/year) Mean ± SD	Weight velocity (kg/year) Mean ± SD	Chest circumference velocity (cm/year) Mean ±SD	Biacromial diameter velocity(cm/year) Mean± SD	Bicristal diameter velocity (cm/year) Mean±SD	Triceps SFT velocity (mm/year) Mean ± SD	Subscapular SFT velocity (mm/year) Mean± SD
9.0-10.0	1.5 ±0.82	1.2 ±1.26	1.2 ±1.13	0.7 ±0.93	1.1±1.55	0.08±1.57	0.3 ±1.63
10.0-11.0	1.7 ±1.15	1.2 ±1.24	1.2 ±1.05	0.5 ±0.61	1.1± 0.90	0.4 ±1.27	0.2 ±0.79
11.0-12.0	1.7 ±1.09	1.3 ±1.15	1.3 ±1.33	0.7 ±0.80	0.7±0.97	0.09±2.31	0.2 ±2.07
12.0-13.0	1.7 ±1.07	1.4 ±1.49	1.5 ±1.33	0.8 ±0.68	1.0±0.93	0.3 ±2.74	0.1 ±1.67
13.0-14.0	2.0 ±2.01	1.3 ±1.75	1.6 ±1.64	0.4 ±0.88	0.7±0.66	0.3 ±3.40	0.03±2.60
14.0-15.0	2.2 ±2.61	2.3 ±3.47	2.1 ±2.42	1.2 ±1.18	1.5±1.47	0.5 ±3.10	0.7 ±2.77
15.0-16.0	1.5 ±1.34	1.5 ±0.22	0.7 ±0.31	0.3 ±0.59	0.5±0.35	0.8 ±2.32	0.2 ±1.49

compared with their normal well-off Chandigarh[30] and Leeds counterparts.[31] Further, attainment of peak height (PHV) and weight (PWV) velocities in boys with steroid sensitive nephrotic syndrome was delayed as compared to normal Chandigarh and Leeds children.

Mean chest circumference growth velocity measuring 1.23±1.13 cm/year between 9 to 10 years of age showed regular increase to attain a peak value measuring 2.11cm/year at 14.5 years of age. Thereafter, like height and weight it experienced a rapid deceleratory trend to measure 0.78cm/year between 15 to 16 years of age. In general, growth velocities of biacromial diameter, bicristal diameter, triceps skin fold thickness and subscapular skinfold thickness depicted inconsistent trend with a high degree of variability around the mean values (Table. 2)

Discussion

The distance growth curves plotted for height and body weight (Fig 1 and 2) of children with steroid sensitive nephrotic syndrome (SSNS) demonstrated a regular increase in mean values of these two auxological parameters, throughout the period of this study. However, rapidity of this increase was relatively sharper upto around 13.5 years, whereafter it became slower. Boys with SSNS measured shorter than their normal American (CDC)[28] and affluent Indian[27] counterparts as their height growth curve ran below those plotted for children of American and Indian origin. The relative magnitude of this statural growth retardation was found to be greater when contrasted with their normal American[28] than Indian[27] counterparts and it increased in magnitude beyond 13.5 years with advancement of age. This may be due to effect of chronic nature of the disease itself and influence of steroid therapy with which boys with SSNS were treated. Shorter height attainments noticed amongst our study subjects i.e. boys with steroid sensitive Nephrotic syndrome resemble with the findings of Donatti et. al.[6], Emma et. al.[7], Kitamura[8], Tsau et. al.[9], Hung et. al.[10], Osamu et. al.[11], Salim et. al.[12] Mohan et. al.[3], Rees et. al.[21], Motoyama et. al.[22], Ayoub et. al.[23], Alan M[24] and Allen DB[25] who also observed shorter height in SSNS children. Significantly, more severe height growth retardation yielding short stature amongst boys with SSNS during peripubertal age recorded by Kitamura et. al.[8], Emma et. al.[7] and Salim et. al.[12] are similar to our findings. However, these observations are at variance with those of Saha et. al.[17], Adhikari et. al.[18], Ruth et. al.[19] and Abbas et. al.[20] who did not notice any difference in mean height attainments of children with steroid-sensitive nephrotic syndrome as compared to their normal peers. Height growth curve plotted for children with SSNS not only ran below those of the American and Indian children but exhibited parallelism until 13.5 years, afterwards it suddenly diverged to impair height more severely, to make them short individuals.

It has further been observed that like height and weight, growth of chest circumference, biacromial diameter and bicristal diameter in boys with steroid sensitive Nephrotic syndrome was also compromised as compared to their normal counterparts.[27,28,29] Exception to this were triceps and subscapular skinfold thicknesses which in general had more of fat than normal boys and became fatter between 12 to 14 years due to excessive deposition of appendicular fat at triceps, as well as truncal fat in the subscapular region.

Boys with steroid sensitive nephrotic syndrome not only experienced delay in attainment of peak height and weight velocities, but the magnitude of peak height velocity (PHV) and peak weight (PWV) velocity was found to be substantially lesser than their normal Chandigarh[30] and Leeds counterparts[31], which may be the reason for relatively shorter height (Fig 1) and lighter weight (Fig. 2) attainments noticed amongst boys with SSNS as compared to their normal peers. However, inter-population auxological comparison for other body parameters could not be attempted because of the non-availability of suitable sets of comparative data on normal subjects belonging to different population stocks.

Conclusions

The relatively impaired auxological status recorded amongst boys with SSNS appears to be due to influence of chronic nature of the disease itself as well as effect of steroid therapy which is known to impair growth in children. On the contrary, growth of subcutaneous fat measured in terms of triceps and subscapular skinfold thicknesses exhibited relatively fatter attainments amongst boys with SSNS than their normal counterparts.

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Original Research Article

Pediatric Education and Research
Volume 8 Number 3, July–September 2020
DOI: https://dx.doi.org/10.21088/per.2321.1644.8320.7

To Study Incidence of Neurosonographic Abnormalities in Newborns with Birth Asphyxia

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How to cite this article:
Abhijit Shinde, Sunil Natha Mhaske, Shreya Bhate. To Study Incidence of Neurosonographic Abnormalities in Newborns with Birth Asphyxia. Pediatr Edu Res. 2020;8(3):109-112.

Abstract

Neonatal sonography of the brain is now an essential part of newborn care, particularly in high risk and unstable premature infants. Cranial ultrasound is the most available and easily repeatable imaging technique for the neonatal brain showing brain development and the most frequently occurring forms of cerebral injury in the preterm and terms. This study aims to assess the importance of cranial ultrasound as an investigatory modality for high-risk neonates and to find out the morphology of various cerebral lesions and correlate clinically.

Materials and Methods: This was a Descriptive Longitudinal Prospective study conducted in Neonatal Intensive Care Unit at Paediatric Department of *Dvvpf Medical College and Hospital*, which is a tertiary care hospital for surrounding districts, during the period of two years. In our period 155 neonates having perinatal asphyxia was studied to evaluate the usefulness of Neurosonogram in diagnosis of various lesions in symptomatic neonates with history of birth asphyxia.

Results: In our study 70 (45.1%) neonates had abnormal NSG findings of total of 155 neonates. In our study abnormal NSG among preterm neonates was maximum with weight in 1-1.5kg (88.5%). In our study abnormal NSG among term neonates was maximum in neonates with weight in 2-3kg (72%) range [2- 2.5kg (25%) and 2.5-3kg (47%) range]. Among 70 abnormal NSG, 25 had mydriasis, 38 had normal anterior fontanelle,58 had negative transillumination test. Abnormal NSG finding were found when done after 72 hours.

Conclusion: This study shows that Neurosonography is a sensitive, easy, simple, non-invasive, value-effective, initial choice of investigation for detection of abnormal changes in brain among neonates. High efficacy of NSG in detecting presence of brain damage and evolution of brain lesions on regular follow up guides clinical decisions and prognosis of the neonate.

Keywords: Neurosonography; Asphyxia; Seizures; Anterior fontanelle; Newborn; Cranial sonography.

Introduction

The advent of cranial ultrasound or neurosonography (NSG) as a routine tool in neonatology has greatly improved our knowledge of the presence and incidence of brain lesions in the newborn infant. Cranial ultrasound has been used routinely for infants at risk of neurological impairment, such as those born prematurely[1-5] or who have suffered from birth asphyxia.[6,7] World Health Organization (WHO) states that about 9 million neonates develop birth asphyxia every year. Of them 1.2 million die

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and same number develop severe consequences such as cerebral palsy, epilepsy and developmental delay. Cranial ultrasound is the most available and easily repeatable imaging technique for the neonatal brain showing brain development and the most frequently occurring forms of cerebral injury in the preterm and terms.[8]

Ultra sonogram through the anterior fontanelle forms the best acoustic window and is as use full as CT with added advantages as it is easy, value effective, can be repeatable at bedside, free of radiation, minimum discomfort to the baby. And thereby enables visualization of ongoing brain maturation and the evolution of brain lesions. In addition, it can be used to assess the timing of brain damage.[9]

Hence this study is undertaken to evaluate the utility of Neurosonography (NSG) for diagnosis of various assorted brain lesions in symptomatic neonates having history of birth asphyxia.

Methodology

This was a Descriptive Longitudinal Prospective study conducted in Neonatal Intensive Care Unit at Paediatric Department of DVVPF MEDICAL COLLEGE and HOSPITAL, which is a tertiary care hospital for surrounding districts, during the period of two years. In our period 155 neonates having perinatal asphyxia was studied to evaluate the usefulness of Neurosonogram in diagnosis of various lesions in symptomatic neonates with history of birth asphyxia.

Inborn Term and Preterm neonates with perinatal asphyxia admitted to Neonatal Intensive Care Unit during the study period. The probe used for neurosonogram (Sonosite machine) was Linear high frequency probe and was done in NICU. Findings on neurosonogram were periventricular leukomalacia grading 1, 2 and 3, Haemorrhage, Infarct, Ventricular and white matter haemorrhage. No follow up is available.(Fig. 1&2)

All cases of Birth asphyxia fulfilling inclusion criteria were included in the study. No baby amongst these were on anticonvulsants having abnormal or absent pupillary reflexes.

Inclusion Criteria

A. All In-born term and preterm neonates with features indicative of perinatal asphyxia.

B. Criteria for asphyxia includes
   1. Apgar score of 3 at 1min.
   2. Requirement of Positive pressure ventilation for more than 1 min at resuscitation.
   3. Fetal heart rate abnormalities (Fetal bradycardia 160 beats/minute) and/or presence of meconium stained amniotic fluid.
   4. Abnormal neurological findings including altered muscle tone, altered sensorium and seizures.
   5. Need for chest compression during neonatal resuscitation.

Exclusion Criteria

1. Outborn neonates.
2. Neonates with major congenital malformations e.g.- anencephaly, open neural tube defects, diaphragmatic hernia etc.
3. Neonates with extremely low birth weight (< 1 kg)
4. Neonates of extreme prematurity (less than 28 weeks of gestation)
5. Neonates who did not respond to resuscitation

Informed consent was obtained from the parents/guardian regarding inclusion of the neonate in the study. All babies received standard care during and after resuscitation. The relevant maternal and neonatal data was recorded in the standard proforma.

Gestational age in completed weeks was obtained on basis of mother's last menstrual cycle and confirmed where neccessary by routine early antenatal USG examination. In some cases where LMP was not available and antenatal USG was not done, then gestational age was assessed by Modified New Ballard's scoring system

The images were obtained through the anterior fontanelle. Image quality was maximized by fine adjusting the preset already available for transcranial scans.

All the data was arranged in a tabulated form and was analysed using Epi info software version 7.1.2. Chi square test and student t test was used for comparison. Probability value of less than 0.05 was considered significant.

Results

Table 1A: Distribution of Various Clinical Findings V/S Neurosonography (According To Pupil Reflex)

Presentation	Pupil Reflex (N= 155)			
	Normal	Mydriasis	Miosis	Absent
Total (n=155)	80	30	15	30
NSG - Normal (n= 85)	75	07	03	00
NSG - Abnormal (n= 70)	05	25	15	25

X2= 103.85, p < 0.001, Significant

Significant association was seen between the NSG normal-abnormal and pupillary reflexes (p < 0.001). Out of 85 normal NSGs, 75 cases had normal pupillary reflex, while out of 70 abnormal NSGs, only 5 were normal.(Table 1A)

Table 1B: According to Anterior Fontanelle.

Presentation	Anterior Fontanelle (N= 155)		
	Bulged	Depressed	Normal
Total (N= 155)	30	14	111
NSG- Normal (n= 85)	00	10	75
NSG-Abnormal(n=70)	30	02	38

X2 = 46.43, p < 0.001, Significant

Significant association was seen between the NSG normal-abnormal and anterior fontanelle (p < 0.001). Out of 85 normal NSGs, 75 cases had normal anterior fontanelle, while out of 70 abnormal NSGs, only 38 were normal.(Table 1B)

Table 1C: According to Translumination.

Presentation	Translumination (n= 155)	
	Positive	Negative
Total (n= 155)	10	145
NSG- Normal (n= 85)	00	85
NSG-Abnormal(n=70)	12	58

X2 = 15.79, p < 0.001, Significant

Significant association was seen between the NSG normal-abnormal and anterior fontanelle translumination (p < 0.001). Out of 85 normal NSGs, 85 cases had negative translumination, while out of 70 abnormal NSGs, only 12 were positive. (Table 1C)

Table 2: Relation of Central Cyanosis and Neurosonography Findings.

NSG	Central Cyanosis	
	Present	Absent
Total (n= 155)	15	150
NSG- Normal (n= 85)	00	85
NSG-Abnormal(n=70)	17	53

X2 = 23.19, p < 0.001, Significant

Significant association was seen between the NSG normal-abnormal and central cyanosis (p < 0.001). Out of 85 normal NSGs, 0 cases had cyanosis, while out of 70 abnormal NSGs, only 17 had cyanosis. (Table 2)

Table 3: Distribution of Birth Asphyxia Neonates Based on Timing of Nsg

Timing of NSG	Normal	Abnormal	Total
< 24 Hrs	98	57	155(100%)
24-72 Hrs	90	65	155(100 %)
>72 Hrs	85	70	155(100%)

X2 = 2.29, p = 0.32, Not Significant

No significant association was seen between the NSG normal-abnormal and timing of NSG (p < 0.001).(Table 3)

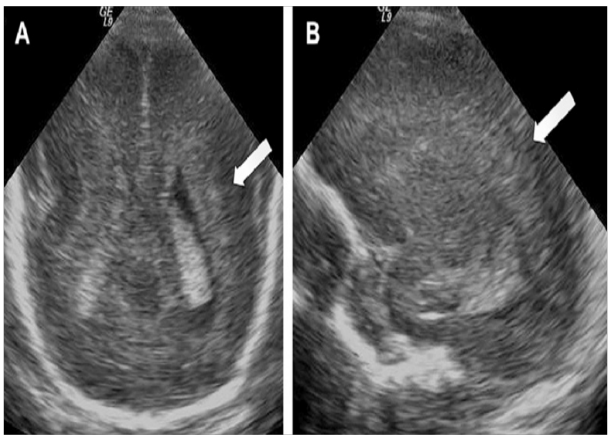

Fig. 1 A: Neurosonogram showing periventricular leukomalacia.
Fig. 1 B: Neurosonogram in which ventricles are visualized.

Discussion

In our study 70 (45.1%) neonates had abnormal NSG findings of total of 155 neonates.

In our study abnormal NSG among preterm neonates was maximum with weight in 1-1.5kg (88.5%) range which is consistent with Eastman NJ et. al. although we had higher percentage (88.5%) in that rage as compared to Eastman NJ et. al. (41%).

In our study abnormal NSG among term neonates was maximum in neonates with weight in 2-3kg (72%) range [2- 2.5kg (25%) and 2.5-3kg (47%) range] this is also consistent with Eastman NJ et. al. which had 51% preterm with abnormal NSG in this range.[10,11,12]

Primhak RA et. al. in their study found out that up to 50% Of neonates weighing less than 1500 g

exhibited some abnormality on the initial NSG.[13]

In our study we have found that out of 155 neonates, 80 had meconium of which 36 (45%) had abnormal scan. 70 mothers had anemia of all these deliveries 30 (42.8%) had abnormal NSG. PROM as risk factor was present in 36 pt. of these deliveries 22 (61.1%) had abnormal NSG. Out of 25 deliveries with PIH as risk factor, 8 (32%) neonates had abnormal scan. In only 5 deliveries cord around neck was present, 3 (60%) of these neonates had abnormal scan. Prolonged 2nd stage of labour was present in 22 deliveries, 15 (68%) of these asphyxiated neonates had abnormal neurosonography.

Reddy et. al. reported that PROM and preeclampsia influenced the presence of NSG abnormalities and risk of developing periventricular intraventricular hemorrhage(PVH).[14]

Conclusion

Neurosonography is a sensitive, easy, simple, non-invasive, value-effective, initial choice of investigation for detection of abnormal changes in brain among neonates. High efficacy of NSG in detecting presence of brain damage and evolution of brain lesions on regular follow up guides clinical decisions and prognosis of the neonate.

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Review Article

Pediatric Education and Research
Volume 8 Number 3, July–September 2020
DOI: https://dx.doi.org/10.21088/per.2321.1644.8320.8

Surfactant Replacement Therapy for Respiratory Distress Syndrome in the Newborn

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How to cite this article:
Rucha Nitin Tipare, Sunil Natha Mhaske. Surfactant Replacement Therapy for Respiratory Distress Syndrome in the Newborn. Pediatr Edu Res. 2020;8(3):113-116.

Abstract

Surfactant deficiency Causing respiratory failure is the major cause of morbidity and mortality in low birth weight premature infants. Surfactant therapy gradually reduces mortality and morbidity for this population. Exogenous surfactant therapy is well established in newbornbabies with respiratory distress. Many aspects of its use have been well assessed in high-quality trials and systematic reviews. In late-preterm and term neonates with meconium aspiration syndrome, pneumonia/sepsis, and maybe pulmonary haemorrhage, secondary surfactant deficiency also leads to acute respiratory morbidity; surfactant substitution can be helpful for these babies. This paper reviews the evidence and provides guidelines for the use of respiratory distress syndrome (RDS) surfactant therapy in newborns.

Keywords: Surfactant therapy Distress Syndrome in newborn

Introduction

As an effective preventive and treatment therapy for prematurity-related surfactant deficiency, exogenous surfactant substitution has been developed.More advanced children with primary pulmonary hypertension or meconium aspiration syndrome may also be recommended for surfactant therapy. The use of surfactant replacement in preventive or treatment modes has been shown to be safe and efficient in single and multicenter randomised controlled trials using synthetic, modified animal, purified animal, and human surfactants. Reduced death rates and increased short-term respiratory status have been reported for preterm babies with respiratory failure due to surfactant-deficiency. New experiments continue to tackle refinements in the use of surfactants that can maximise their efficacy. Among the challenges that can boost the effect of surfactants are new materials, spacing, dosage, ways of administration, and adjustment for various gestational age groups.[1,2] Surfactants are organic compounds that lower the surface tension of a liquid lining the alveoli.[2] Surfactants decrease the surface tension of the fluid by adsorbing the liquid-gas interface.[3] A surface-active lipoprotein complex (phospho lipoprotein) produced by type II alveolar cells is a pulmonary surfactant. The key lipid surfactant portion, dipalmitoyl phosphatidylcholine (DPPC), decreases surface tension by adsorbing alveoli to the air-water interface with the hydrophilic head groups in the water and the hydrophobic tails facing the air.[4]

Surfactant Functions

- To improve pulmonary compliance.
- Atelectasis (collapse of the lung) can be

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avoided at the end of expiration.

- In order to promote the recruiting of collapsed airways.

Benefits of Surfactant Replacement Therapy in RDS

The occurrence of acute respiratory failure with disrupted gas exchange in a preterm baby with a normal clinical course or x-ray (ground glass presentation, air bronchograms and reduced lung volume) is typically described by RDS.[5] The lungs of preterm infants with RDS are both anatomically and biochemically immature; they do not synthesise or secrete RDS. Surfactant usually lines the alveolar surfaces of the lung, thus decreasing surface stress and avoiding atelectasis.[5] Surfactant replacement therapy decreases death and morbidity of babies with RDS, either as a rescue procedure or as a prophylactic natural surfactant therapy.[6,7] These morbidities include oxygenation defects, the occurrence of leakage of pulmonary air (pneumothorax and interstitial pulmonary emphysema) and the length of ventilatory assistance. Replacement of surfactants improves the chance of survival without bronchopulmonary dysplasia (BPD, also referred to as chronic lung disease) mainly by improving survival rather than the occurrence of BPD. Compared to randomised placebo children without surfactants, babies administered with surfactants had shorter hospital stays and reduced costs of intensive care treatment.[8] The rise in longevity is done without a rise of adverse neurodevelopmental results.[9]

Risks of Exogenous Surfactant Therapy

Bradycardia and hypoxemia during instillation as well as endotracheal tube blockage are short-term risks of surfactant replacement therapy.[10] There can also be a rise in pulmonary haemorrhage after surfactant therapy; however, mortality associated with pulmonary haemorrhage is not increased and total mortality is lower after surfactant therapy.[11] In surfactant-treated children that are deficient in surfactants, there is also a very fast increase in gas exchange.[12] Natural surfactants include proteins (surfactant protein-A, surfactant protein-B) from bovine or porcine origins and concerns about the immunological consequences have been raised.[13] To date, there is no proof that there are immunological improvements of therapeutic concern.[14] Babies with RDS have observable circulating immune complexes targeted at proteins of surfactants, but these do not seem to be more common in surfactant-treated babies.[15] One study found a lower occurrence of protein-A and anti surfactant protein Banti surfactants in surfactant-treated babies compared to controls.[16,17]

Which is Better: Natural or Synthetic Surfactants?

A total of 11 randomised trials were subject to systematic analysis comparing natural and synthetic surfactants for babies with RDS.[10] The study found that overall mortality is decreased relative and synthetic surfactants due to the use of natural surfactants. In babies treated with natural surfactants, pulmonary air leak syndrome is less frequent. In babies given natural or synthetic surfactants, the occurrence of BPD is not different, but since mortality is minimised in babies given natural surfactants, the cumulative result of death or BPD is decreased. Natural surfactants thus enhance longevity without BPD and with a lower rate of air leakage and should be favoured to synthetic surfactants.[18]

Surfactants Prescribed as Prophylaxis or Rescue Therapy for Preterm Babies with RDS-

Multiple trials have examined whether surfactants should be prescribed to all babies at serious risk of developing RDS or only after RDS development. Seven RCTs in prophylactic versus rescue treatment were analysed by Soll and Morley.[19] These were both experiments that used natural surfactants. Six of the RCTs enrolled children less than 30 weeks gestational and one enrolled children 29 to 32 weeks gestational. Prophylactic surfactant therapy reduced mortality both before 28 days and before hospital discharge. The occurrence of RDS, pneumothorax and pulmonary interstitial emphysema all decreased in babies treated prophylactically. There was no difference in the occurrence of BPD.[19] With the existing mortality rates at tertiary centres, prophylactically providing surfactant to all babies less than 26 weeks gestation and to all 26 to 27 weeks gestation who did not benefit from antenatal steroids would be a fair choice. hInfants at serious risk of RDS should undergo prophylactic natural surfactant therapy as soon as they are stable within a few minutes after intubation.[19]

Surfactant Replacement Therapy Procedure

Surfactant was instilled through the endotracheal tube in liquid form for all surfactant replacement therapy trials.[17] Some studies instilled all the surfactant at once, while othergave in small parts.[18] Just one very small trial compared a slow infusion with bolus surfactant administration. It concluded that slow infusion was at least as effective as bolus therapy.[20] There is no evidence to support the practise of putting the baby in several different positions during surfactant administration.[20]

Dosage of Surfactant

In the various clinical trials, dosages ranged from 25 mg to 200 mg phospholipids/kg body weight as single doses. At a dosage of 120 mg/kg, Surfactant-TA (a natural bovine surfactant) was more effective than 60 mg/kg.[20] At 200 mg/kg, Curosurf (a natural porcine surfactant) was more acutely effective than 100 mg/kg.[21] Lower doses may well be ideal for prophylaxis, although higher doses may be needed for the treatment of known RDS when antisurfactant proteins are present in the airspace. Therefore, improvements in results tend to be seen up to a dose of approximately 120 mg phospholipids/kg body wt for first dose, first larger doses do not cause improvement in outcome.

Requirements for Retreatment and Timing

Retreatment should be considered when there is a requirement of 30 percent or more for chronic or repeated oxygen and can be administered as early as 2 h after the initial dose or, more generally, 4 h to 6 h after the initial dose.[22]

Ventilatory Management After Surfactant Therapy

Due to the rapid changes in lung mechanics and the ventilation/perfusion matching that occurs after rescue surfactant therapy and the avoidance of serious lung disease by the prophylactic use of natural surfactants, many infants can be very easily weaned and extubated to nasal continuous positive airway pressure (CPAP) within 1 hour of intubation and surfactant therapy.[17] To do this, only a brief period of respiratory distress should be triggered by the premedication used for intubation and personnel must be educated and certified in rapid ventilator weaning. Such weaning is sometimes carried out with little to no blood gases, depending instead on the clinical state of the baby and spontaneous respiratory effort and taking into account the criteria for oxygen as determined from pulse oximetry and also using measurements of transcutaneous carbon dioxide. There is currently no evidence that, compared with the more conventional weaning approach, a fast weaning and extubation approach enhances long-term performance. Such an approach resulted in a decrease in the need for more than 1 h of mechanical ventilation in two small randomised trials.[21]

Surfactant therapy V/S antenatal steroids- A single course of steroids should be offered to expectant mothers with threatened preterm labour, according to current guidelines. Wide cohort studies suggest that surfactant and steroid combinations are more effective than surfactant alone, which is exogenous. A secondary analysis of evidence from surfactant research also suggests a decline in the incidence of disease in infants receiving antenatal steroids. Two additional RCTs have shown that prenatal steroids appear to minimise the risk of bad outcomes even in centres where surfactants are available; one has shown a decrease in RDS and an increase in survival without ventilatory assistance, and both have shown a substantial decrease in extreme intraventricular haemorrhage.[23]

Conclusion

The therapy of exogenous surfactants is safe and has important benefits in the treatment of many neonatal respiratory diseases. Excellent quality RCTs have been well studied and have clearly reported that their administration should be normal in the treatment of RDS and as prophylaxis in identified preterm baby classes. In other infant respiratory disorders, data continues to be gathered for its use. The following guidelines are provided by the Canadian Paediatric Society:-

- Antenatal steroids should be prescribed to mothers at risk of delivering babies less than 34 weeks gestation, in compliance with established guidelines, regardless of the availability of postnatal surfactant therapy.
- Exogenous surfactant therapy should be offered to intubated infants with RDS.
- As soon as they are healthy within a few minutes of intubation, infants who are at high risk for RDS should receive prophylactic natural surfactant care.
- Repeated doses of surfactants should be given to infants with RDS who have chronic or recurrent oxygen and ventilatory requirements during the first 72 h of life. It has not been shown that administering more than three doses has a benefit.
- Retreatment may be considered when an oxygen demand of 30 percent or more is persistent or chronic, and can be administered as early as 2 h after the initial dose or, more generally, 4 h to 6 h after the initial dose.
- Ventilatory management options to be considered following prophylactic surfactant therapy include very rapid weaning and extubation within 1 h of CPAP.
- If at all necessary, mothers with a threatened delivery before 32 weeks of gestation should be moved to a tertiary centre.
- Infants that have been gestated for less than 29 weeks outside the tertiary centre should

be recommended for immediate intubation followed by the administration of surfactants until stabilisation, provided that qualified personnel are available.

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