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Original Research Article

An Observational Study on the Etiology and Associated Clinical Profile of Congenital, Neonatal and Pediatric Stridor

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ABSTRACT

Background
This study was conducted to analyse the aetiology and associated clinical profile of congenital, neonatal and paediatric stridor.

Methods
This was a hospital-based retrospective, observational, analytical, single-group study conducted among 50 children presenting with stridor, fulfilling the inclusion criteria(s) in the Department of Otolaryngology, Manipal hospital, HAL airport road, Bangalore from October 2017 to May 2018. After a detailed case history of all selected patients were taken along with ear, nose and throat examination and detailed systemic examination, Flexible fiberoptic laryngoscopic examination was conducted to visualize the larynx.

Results
Significant change in spO2 (%)was noted in subjects with activity;spO2 at feeding was found to be significantly lower compared to spO2 while sleeping. Also, spO2 at crying was significantly higher compared to that while feeding. In association of etiology with respiratory distress-Laryngomalacia was significantly associated with respiratory distress.A significantly higher frequency of failure to thrive was seen in subjects with respiratory distress. In comparison of spO2 at various occasions between subjects with and without respiratory distress, spO2 was found to be significantly decreased while sleeping, feeding, and crying in subjects with respiratory distress compared to those without respiratory distress.

Conclusion
The most common cause of stridor in neonatal age group is laryngomalacia;in the paediatric age group it is subglottic stenosis;both more common in male children.
The most common associated clinical presentation of congenital and paediatric stridor was respiratory distress.Chronic stridor was associated with failure to thrive.

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Laryngomalacia was significantly associated with children with syndromes. Whereas there may not be a direct association with any maternal factors or term delivery with respect to congenital/paediatric stridor.
Babies born preterm are prone to present with stridor.
Keywords: Etiology, Associated Clinical Profile, Congenital, Neonatal, Pediatric Stridor.

INTRODUCTION

Stridor is a high-pitched, monophonic sound caused by turbulent airflow through a narrow airway. The term originates from the Latin *stridere*, which means “creaking" or “grating noise." It may present as an inspiratory or expiratory type, often limited to inspiration. Readaptation of respiratory mechanisms from an intrauterine to an extrauterine environment occurs in neonatal life. Stridor in this period may be caused by anatomical or developmental defects in the airway. It may also be caused by any obstruction narrowing the airway, including edema/inflammation, masses, aspirated foreign bodies, and decreased tone of airway walls. When stridor presents with significant suprasternal tug and intercostal recession, stridor indicates an extremely complicated airway (<1 mm away from complete obstruction). The production of stridor is related to the Venturi effect: as air passes through a constricted lumen, the air speed increases while the pressure decreases.

This causes a vacuum, soft tissue flapping in the airways, and finally the audible sound known as stridor. A stidor might be biphasic, expiratory, or inspiratory. Clinically, there is a correlation between supraglottic and glottic lesions and inspiratory stridor. Anomalies of the trachea, bronchi, and subglottis are linked to the expiratory stridor. Pathologies of the glottis and subglottis can produce biphasic stridor.[1] It is unusual for stridor to be present at birth, that is, with the child's first breath. This typically indicates a permanent congenital narrowing, such as tracheal narrowing, subglottic stenosis, or laryngeal web.[2] In the first few weeks of birth, dynamic disorders like laryngomalacia manifest. Congenital vocal cord palsy frequently manifests as stridor right after delivery, which raises the possibility that the condition is more of an incoordination than a true paralysis. Gradually worsening stridor or compromised airway suggests the development of a blockage, which could be external (as in a mediastinal mass) or luminal (as in a subglottic haemangioma).[3]

Pattern of Stridor

Stridor is not always reliable. Any variation in the stridor with regard to the child's activity, the time of day, or any other related factors may be helpful in determining the reason. For instance, laryngomalacia improves while the infant is sleeping or at rest, but it deteriorates when the child is crying, eating, or in a stressful state. A pedunculated laryngeal mass may cause airway blockage in a supine infant, but more frequently, a degree of supralaryngeal obstruction, such as micrognathia and the ensuing tongue base occlusion, is to be blamed. When there is a significant nasal blockage, such as bilateral choanal atresia, the airway improves with crying.

Associated Features

Along with stridor, other symptoms caused by airway obstruction include coughing, hoarseness, cyanosis, dyspnoea, tachypnea, chest recession, and apneas. Parents may not notice recession, even if it is fairly severe, although it is an obvious indication of inspiratory blockage. Apneas accompanied by cyanosis are sometimes referred to as "dying spells" and are indicative of severe tracheobronchomalacia. There are other causes of tachypnea and dyspnea besides upper airway blockage. However, a detailed account of exertional dyspnoea in an older child offers a helpful functional evaluation of its severity. Cough is rarely the result of "infant asthma" and is typical of

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tracheomalacia and tracheooesophageal fistula.[4] Although vocal cord palsy can sometimes cause hoarseness, it is obviously indicative of a laryngeal lesion such as laryngeal papillomatosis.

Feeding History

Breathing and feeding are intimately related, especially for infants because of the sucking swallow feeding reflex. It is necessary to have a precise image of the feeding pattern. When an airway obstruction occurs, breastfed newborns will typically "come up for air"; bottle-fed babies might need to have their meals increased or use a "slow teat" (one with tiny openings). Aspiration may indicate a tracheo-oesophageal fistula, vocal cord palsy, or infrequently, a cleft larynx. Recurrent chest infections may be linked to significant repeated aspiration. In newborns, regurgitation, often known as "posseting," is common but may not always indicate severe gastro-oesophageal reflux. Poor feeding practices can lead to failure to thrive with observably poor weight gain, or they can only cause slow feeding that bothers the mother more than the infant.

General Medical Conditions

A history of general medical health must be taken. The presence of any medical problems may explain a lot of associated medical conditions, along with stridor. For instance, a vocal cord palsy brought on by a neurological condition, a heart surgery, or vascular compression linked to a congenital cardiac condition could be indicated. History and examination of the presence of any birthmarks must be emphasized, as they may be associated with a subglottic haemangioma.

Approach to a Child with Stridor

Prior to a formal examination, the child should be observed while at rest in order to earn the kid's trust and make an initial assessment of the severity of respiratory distress and any stridor characteristics. It is necessary to monitor the traits of the stridor in addition to the consequences of airway obstruction, such as recession. Coughing, wheezing, or an abnormal voice are helpful localizing indicators. Despite its importance, the pre-endoscopy evaluation can only serve as a guide for the kind and extent of pathology found during the endoscopy. In certain cases (such as moderate laryngomalacia), a complete history, physical examination, and restricted investigation can be enough to provide enough diagnostic confidence to forego a first endoscopy. A certain pathology may be characterized by the type of stridor.[5] Up to 20% of cases may have a second pathology, although few will need treatment.[4] An endoscopy is required for definite diagnosis confirmation. This does not imply that endoscopy is necessary for every child with stridor. Endoscopy is usually the gold standard for most disorders and will be necessary for the majority of children examined in a secondary or tertiary referral centre. Dynamic diseases, including tracheobronchomalacia and vocal cord palsy are frequently challenging to establish or rule out with a normal endoscopy.

AIMS AND OBJECTIVES

- To analyse the etiology and associated clinical profile of congenital, neonatal and pediatric stridor
- What is the most common cause of stridor in neonatal and pediatric age groups?
- What is the most common associated clinical presentation of congenital neonatal and pediatric stridor?
- What is the most common associated maternal factor(s) and birth history in congenital, neonatal and pediatric stridor?

MATERIALS & METHODS

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This was a hospital-based retrospective, observational, analytical, single-group study conducted among 50 children presenting with stridor, fulfilling the inclusion criteria(s) in the Department of Otolaryngology, Manipal hospital, HAL airport road, Bangalore from October 2017 to May 2018. After a detailed case history of all selected patients were taken along with ear, nose and throat examination and detailed systemic examination, flexible fiberoptic laryngoscopic examination was conducted to visualize the larynx.

Inclusion Criteria

- Neonates, infants, or children with breathing difficulties with respect to airway pathologies
- Neonates, infants or children presenting with stridor

Exclusion Criteria

- Children with breathing difficulties with systemic pathologies (pulmonary, cardiac, neurological and hepatobiliary).
- Children with breathing difficulties due to metabolic derangements.

The following tests were done to know the severity (to decide the need for possible intervention) or exclude children from the study group:

- Complete blood count
- Serum electrolytes
- Arterial blood gas analysis
- 2D echo
- Sleep study

Statistical Methods

The data were presented as a mean ± standard deviation and as a percentage. To ensure that the data were linear, Kolmogorove-Smirnove analysis was done. The significance of the difference between two parameters in parametric data was assessed using the Student's t test (paired and unpaired), while the significance of the difference between two parameters in non-parametric data was assessed using the Mann Whitney U test. The Friedman test, followed by the post hoc Dunn test, was used to test the significance of the difference between more than two parameters in parametric data. Fisher's exact test or Chi square test was used to analyze the significance of the difference between the frequency distribution of the data. P-value <0.05 was considered statistically significant.

Study Procedure

After prior approval from the ethical and scientific committee, FIFTY patients who clinically presented with stridor, fulfilling the inclusion criteria(s), were recruited in this study, and if they agreed to participate in the study, written informed consent was obtained from them. For each individual consenting to participate in the study, a case record form was filled out. A detailed case history of all selected patients was taken along with detailed ear, nose, and throat examinations along with detailed systemic examinations. All patients underwent FFOL (Flexible Fiberoptic Laryngoscopy) through the oral route. The laryngoscope was passed till the larynx was visualized and beyond vocal cords till subglottis. Findings were noted. A real time video recording of the same was obtained along with photographs (done with prior consent). In cases of impending severe airway obstruction, a similar procedure was done in an operation theatre set up with monitored anesthesia care.

Patients were assessed, and results were charted as:

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- Age of presentation
- Gender of child
- Presence/absence of respiratory distress (characterized by xiphisternal retraction, suprasternal retractions, tachypnea, and nasal flaring)
- Oxygen saturation during sleeping, crying, and feeding and its association with children presenting with respiratory distress.
- Presence/absence of failure to thrive.
- Most common etiology based on FFOL findings.
- Association of genetic syndromes with stridor.
- Association of full-term/preterm delivery.
- Associated maternal illness during pregnancy.

RESULTS

Age (months)	Frequency	Percent
</=1 month	4	8.0
1-12 months	25	50.0
>12 months	21	42.0
Total	50	100.0
Age Distribution		
Gender	**Frequency**	**Percent**
F	21	42.0
M	29	58.0
Total	50	100.0
Sex Distribution		

Table 1: Demographic Distribution

Age distribution in study subjects was studied. Maximum subjects (50%) were found to be in the age group 1-12 months. While least frequency (8%) was found in the subjects </= 1 month. The gender distribution of study subjects was studied. Out of 50 subjects, 29 (58%) were males and 21 (42%) were females.

SpO2 (%)	Mean	S.D.	Min	Max	P-Value
Sleeping	89.70	4.89	78.00	96.00	0.018
Feeding	88.68[a]	5.35	78.00	96.00	
Crying	90.04[b]	4.18	82.00	96.00	

Table 2: Change in Oxygen Saturation (SpO2-%) during Various Activities

p<0.05 Vs Sleeping, p<0.05 Vs crying

Change in spO2 (%) with activity was assessed using the Friedman test. Significant change in spO2 was noted with activity. Further on post hoc analysis, spO2 at feeding was found to be significantly lower compared to spO2 while sleeping. Also, spO2 at crying was significantly higher compared to that while feeding.

Etiology	Respiratory Distress		P-Value
	No	Yes	
Glottic Web	0 (0%)	3 (6.8%)	069

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Laryngeal Cleft	0 (0%)	1 (2.3%)	0.87
Laryngeal Papillomatosis	0 (0%)	1 (2.3%)	0.87
Laryngeal Web	0 (0%)	1 (2.3%)	0.87
Laryngomalacia	5 (83.3%)	14 (31.8%)	0.02
Laryngomalacia with Tracheomalacia	0 (0%)	1 (2.3%)	0.87
Subglottic Stenosis	1 (16.7%)	16 (36.4%)	0.32
Tracheal Stenosis	0 (0%)	2 (4.5%)	0.77
Tracheomalacia	0 (0%)	1 (2.3%)	0.87
Vallecular Cyst	0 (0%)	1 (2.3%)	0.87
Vocal Cord Palsy	0 (0%)	2 (4.5%)	0.77
Vocal Cord Palsy with Glottic Stenosis	0 (0%)	1 (2.3%)	0.87

Table 3: Association of Etiology with Respiratory Distress

Association etiology with respiratory distress was performed using Fisher's exact test. No significant association was noted with any etiology except for laryngomalacia being significantly associated with respiratory distress.

	Failure to Thrive	**Respiratory Distress**		**Total**	**Chi Square**	**P-Value**
		No	**Yes**			
	No	6	20	26	6.294	0014
		100.0%	45.5%	52.0%		
	Yes	0	24	24		
		.0%	54.5%	48.0%		
Total		6	44	50		
		100.0%	100.0%	100.0%		

Table 4: Association of Failure to Thrive with Respiratory Distress in Study Subjects

The association of failure to thrive with respiratory distress in study subjects was assessed using the Chi square test. A significant association was found to exist between two parameters, indicating a significantly higher frequency of failure to thrive in subjects with respiratory distress.

Spo2	**Respiratory Distress**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**T**	**P-Value**
Sleeping	Yes	89.1591	4.93183	.74350	-2.199	.033
	No	93.6667	1.96638	.80277		
Feeding	Yes	87.9545	5.23385	.78903	-2.767	0.008
	No	94.0000	2.52982	1.03280		
Crying	Yes	89.6136	4.26023	.64225	-4.226	<.0001
	No	93.1667	1.32916	.54263		

Table 5: Comparison of SpO2 at Various Occasion between Subjects with and without Respiratory Distress

Comparison of spO2 at various occasions between subjects with and without respiratory distress was performed using students unpaired t-tests. SpO2 was found to be significantly decreased while sleeping, feeding, and crying in subjects with respiratory distress compared to those without respiratory distress.

DISCUSSION

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A physical indication of upper airway blockage is stridor. The most prevalent congenital abnormality, laryngomalacia, is responsible for 60%–70% of stridor occurrences in newborns and infants.[6] The airway obstruction (in laryngomalacia) is caused by collapse of supraglottic tissue, and abnormal and/or reduced laryngeal tone. The term Laryngomalacia (soft larynx-latin) was coined by Jackson and Jackson[7] in 1942 and replaced the term congenital laryngeal stridor. The phrase described the larynx's flaccidity and distinguished it from other causes of stridor.[8]

The Infant Larynx: Anatomical Considerations

It is imperative to comprehend the paediatric larynx in order to navigate through lesions that result in stridor. The supraglottis, glottis, and subglottis are the three regions that make up the larynx. The larynxes of newborns and infants differ from those of older kids and adults.

The first three years of life are a time of rapid larynx growth. Postnatally, the hyoid bone and larynx develop. Neonates are forced to breathe via their noses because of the elevated location of the larynx after birth, which makes it easier for them to switch to spontaneous breathing and prevent aspiration. The first year of life is when the larynx undergoes significant postnatal changes.[9]

Clinical Presentation

When the baby is crying, nursing, or lying supine, stridor frequently gets worse. Stridor is typically accompanied by feeding issues. There is a disruption in the suck-swallow sequence and how it works with respiration. Stridor can happen during inspiration, expiration, or both (biphasic) phases of breathing.

The main symptom of laryngomalacia is inspiratory stridor.

Partial obstruction at the supraglottis level is the source of inspiratory stridor. The most common cause of stridor on expiration is lower respiratory tract blockage.

In our study, the most common etiology was found to be laryngomalacia (38% patients) and the next most common etiology was subglottic stenosis (34%). Holinger and Brown reported that laryngomalacia is the commonest congenital laryngeal anomaly.[10] This was also consistent with the study conducted by Rupa V and Raman R.[11] Endoscopic appearances included one or more of the following: elongated overhanging epiglottis, short aryepiglottic fold and bulky arytenoids which collapse with inspiration.

Comparison of Studies in Incidence of Laryngomalacia in Children with Stridor

The results obtained are consistent with Rupa V.[11] Higher rates of incidence may be seen in other groups due to a larger sample size, a longer duration of study period, and differences in the study groups.

In the same study by Rupa V, laryngotracheobronchitis was found to be the most common infectious cause producing stridor, also supported by Lum SG, etc., where it is stated that the incidence of infective etiology is now significantly lower, probably attributed to the advent of antibiotics and the effectiveness of current childhood immunization programmes.[12]

In our study, children presenting with an infective etiology were excluded. The second most common cause was found to be subglottic stenosis in our study (34%).

Other causes of stridor were glottic web (6%), vocal cord palsy (4%), and tracheal stenosis (4%). A small number of cases were diagnosed to have laryngeal cleft, laryngeal papillomatosis, and laryngeal web.

Some of the cases presented with synchronous laryngeal lesion-laryngomalacia with tracheomalacia (2%). Vocal cord palsy presented with glottic stenosis (2%). Studies have revealed that 21%–47.3% of patients had at least a synchronous airway lesion contributing to airway compromise.[13-17] Lum SG et al. found it to be 9.9% in their study.[12]

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In neonates and infants, the most common congenital cause of stridor was found to be laryngomalacia, whereas in the pediatric age group for more than 1 year, it was found to be subglottic stenosis. It occurred as a primary lesion and also secondary to intubation/instrumentation in some cases. In our study, maximum subjects were found to be in the age group of 1–12 months. (50%). In a study conducted by Sundari K M and Kumar P M, they found the majority of the cases falling in the age group of less than 2 years (62%). [18] In another study conducted by Lum SG, Liza Noor, 84 out of 121 children (69%) were between the ages of 1 and 12 months.[12]

The incidence of male child involvement was found to be more than 58% (29 out of 50). The incidence of male child involvement was also seen to be higher in the studies conducted by Lum SG, Liza Noor (73 out of 121),[12] Sundari K M, Praveen KM (30 out of 50)[18] and Olney DR et al. (64% males).[17]

In our study, stridor was seen to have syndromic association in 12 % of the patients. In the study conducted by Lum SG, Liza Noor congenital pathologies were seen in 15.9% of children with stridor, especially Down syndrome.[12] In our study, syndromes noted were Down's syndrome, Pierre Robin syndrome, and Guillian Barre syndrome. Sanchez et al. reported that 23% of patients with tracheobronchial anomalies had associated genetic disorders.[19] Olney DR et al. also found a syndromic association in children diagnosed with laryngomalacia.[17]

Some of the other incidental findings during the course of the study were:

Respiratory distress was found to be in 88% of children. They presented with stridor along with xiphisternal retractions, suprasternal retraction during inspiration, tachypnea, and nasal flaring, but absent cyanosis.

54.5% of children presented with failure to thrive. Failure to thrive, as noted by Jeffery P. Simons MD et al. in their study, was 9.6%, whereas symptoms of dysphagia were present in 50.3% of patients.[20] As it is known that swallowing interrupts breathing, infants with airway compromise or respiratory distress may not be able to safely coordinate sucking, swallowing, and breathing, leading to dysphagia and aspiration.[21-24] Hence resulting in failure to thrive eventually. Patients with laryngomalacia can have coughing and choking during feeding, feeding difficulty, dysphagia, aspiration, failure to thrive, or worsening of stridor during feeding.[22-24]

7.52% of babies were born at full term by delivery, and 48% were either born preterm or by LSCS, not indicting any significant association with term delivery of the child. Maternal illness was not found to be an important factor in children presenting with stridor. In contrast, Olney DR et al.[17] observed that only 12% were preterm infants and the rest were full term, again signifying no strong association with mode of delivery or term delivery. Although he observed nearly 41% of pregnancies were complicated due to maternal illness.

CONCLUSION

The purpose of the study was to analyze the etiology and associated clinical profile of congenital, neonatal, and pediatric stridor. From our study, we found that the most common cause of stridor in the neonatal age group is laryngomalacia; in the pediatric age group, it is subglottic stenosis, both of which are more common in male children. The most common associated clinical presentation of congenital and pediatric stridor is respiratory distress (characterized by xiphisternal retraction, suprastrnal retractions, achypnea and nasal flaring). Laryngomalacia is significantly associated with children with syndromes. Whereas there may not be a direct association with any maternal factors or mode of delivery with respect to congenital/pediatric stridor. Having noted that, a significant number of babies with stridor have been observed to be born preterm.

REFERENCES

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[1] Mitchell RB, Periera KD. Paediatric otolaryngology for the clinician, XI, New York City, Humana Press 2009.
[2] Pedraza-Pena LR, Rodriguez-Santana JR, Sifontes IE. Neonatal stridor: a life-threatening condition. Puerto Rico Health Sciences Journal 1994;13(1):33-6.
[3] David albert scott brown 7th edition .pg 1114-1125.
[4] Mancuso RF, Choi SS, lalzal GH, Grundfast KM. Laryngomalacia. The search for the second lesion. Arch Otolaryngol Head Neck Surg 1996;122(3):302-6.
[5] Papsin BC, Abel SM, Leighton SE. Diagnostic value of infantile stridor: a perceptual test. Int J Pediatr Otorhinolaryngol 1999;51(1):33-9.
[6] Daniel SJ. The upper airway: congenital malformations. Paediatr Respir Rev 2006;7(Suppl 1):S260-3.
[7] Jackson C, Jackson CL. Diseases and injury of the larynx. New York: The Macmillan Company 1942.
[8] Richter GT, Thompson DM. The surgical management of laryngomalacia. Otolaryngol Clin North Am 2008;41(5):837-64.
[9] Dobbie AM, White DR. Laryngomalacia. Paediatr Clin Am 2013;60(4):893-902.
[10] Holiger PH, Brown WT. Congenital webs, cysts, laryngoceles and other anomalies of the larynx. Ann Otol Rhinol Laryngol 1967;76(4):744-52.
[11] Rupa V, Raman R. Aetiology of paediatric laryngeal stridor in an Indian hospital. Ann Trop Paediatr 1991;11(2):137-41.
[12] Lum SG, Noor Liza I, Priatharisiny V, Saraiza AB, Goh BS. Aetiological profile of paediatric stridor in a Malasian tertiary hospital. Malays Fam Physicia 2016;11(1)2-6.
[13] Zoulmalan R, Maddalozzo J, Holinger LD. Etiology of stridor in infants. Ann Otol Rhinol Laryngol 2007;116(5):329-34.
[14] Richter GT, Thompson DM. The surgical management of laryngomalacia. Otolaryngol Clin N Am 2008;41(5):837-64.
[15] Sichel JY, Dangoor E, Eliashar R, Halperin D. Management of congenital laryngeal malformations. Am J Otolaryngol 2002;21(1):22-30.
[16] Sakakura K, Chikamatsu K, Toyoda M, Kaai M, Yasuoka Y, Furuya N. Congenital laryngeal anomalies presenting as chronic stridor: a retrospective study of 55 patients. Auris Nasus Larynx 2008;35(45):527-33.
[17] Olney DR, Greinwald JH, Smith RJ, Bauman NM. Laryngomalacia and its treatment. The Laryngoscope 1999;109(11):1770-5.
[18] Sundari KM, Kumar PM. A Study of clinical profile of paediatric stridor in tertiary Medical College. JMSCR 2017;5(7):24521-4.
[19] Sanchez I, Navarro H, Mendex M, Holmgren N, Caussade S. Clinical characteristics of children with tracheobronchial anomalies. Paediatr Pulmonol 2003;35(4):288-91.
[20] Simons JP, Greenberg LL, Mehta DK, Fabio A, Maguire RC, Mandell DL. Laryngomalacia and Swallowing Function in children. Laryngoscope 2016;126(2):478-84.
[21] Thompson DM. Abnormal sensorimotor integrative function of the larynx in congenital laryngomalacia: a new theory of etiology. Laryngoscope 2007;117(Suppl.114):1-33.
[22] Richter GT, Wootten CT, Rutter MJ, Thompson DM. Impact of supraglottoplasty on aspiration in severe laryngomalacia. Ann Otol Rhinol Laryngol 2009;118(4):259-266.
[23] Rastatter JC, Schroeder Jr JW, Hoff SR, Holinger LD. Aspiration before and after supraglottoplasty regardless of technique. Int J Otolaryngol 2010;2010(1):912814.
[24] Schroeder JW, Thakkar KH, Poznanovic SA, Holinger LD. Aspiration following CO2 laser-assisted supraglottoplasty. International Journal of Pediatric Otorhinolaryngology. 2008;72(7):985-90.