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# *Congenital Stridor as seen in a Neonatal Intensive Care Unit in Saudi Arabia*

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**Summary**

**Fawehinmi Y, Fageeh N, Asindi A. Congenital Stridor as seen in a Neonatal Intensive Care Unit in Saudi Arabia.** *Nigerian Journal of Paediatrics* **2002; 29: 11.** A prospective study was carried out to determine the prevalent causes and management of congenital stridor in a referral neonatal intensive care unit of Assir Central Hospital, Abha, Saudi Arabia, between 1996 and 1999. Forty cases consisting of 25 males and 15 females were identified during the period. The predominant cause was laryngomalacia in 25 (62.5 per cent) of the cases. Other causes included subglottic stenosis 5 (12.5 per cent), tracheo-oesophageal fistula 3 (7.5 per cent), bilateral vocal cord palsy 2 (5 per cent), vascular compression 2 (5 per cent) and one each of laryngeal web, subglottic haemangioma and cystic hygroma. A majority of the laryngomalacia cases resolved spontaneously by the age of 2 years without surgical intervention.

Key words: Congenital stridor, neonates

## Introduction

STRIDOR is a sign of upper airway obstruction. It is a harsh, vibratory sound of variable pitch caused by partial obstruction of the respiratory passages that results in turbulent airflow through the airway[1] Stridor may be the result of a relatively benign process; it may also be the first sign of a serious and even life threatening disorder. The respiratory tract can be divided into three zones: (1) a supraglottic and supralaryngeal zone including the pharynx, (2) an extra thoracic tracheal zone including both glottis and subglottis and (3) intra-thoracic tracheal zone including the secondary bronchi.[2] Many non-airway lesions can also cause respiratory distress in neonates.[3] To our knowledge, there has been no documented survey of neonatal congenital stridor from the southwestern region of Saudi Arabia. The main focus of this survey was to determine the prevalent causes of congenital stridor in neonates admitted to Assir Central Hospital which serves as a tertiary and referral centre for all the 18 hospitals and 238 primary health centre in Assir province of Saudi Arabia.[4] It is the only hospital in the catchment area with a level three neonatal intensive care unit (NICU).[4] The NICU is a referral unit and therefore admits infants requiring specialized investigations and care from all over Assir region (population 2 million).[4] It has a capacity to accommodate 20 patients and the unit admits an average of 250 newborns a year. There are adequate facilities for incubator care, artificial ventilation, intravenous alimentation, stand-by portable radiography, ultrasonography, computed tomography (CT) scan and magnetic resonance imaging (MRI).

## Patients and Methods

This prospective study covers a period of four years (January 1996 to December 1999). All newborns admitted to the NICU with stridor were studied. The neonatology and otolaryngology teams jointly managed the patients. A detailed history was taken and a general physical examination was conducted on each infant on admission. The information obtained from the history included: age, sex, age of onset of respiratory symptoms and the referring hospital. For every infant, antero-posterior and lateral radiography was done to assess adenoidal, tonsillar, and epiglottic size and shape, retropharyngeal profile, subglottic and tracheal anatomy. The lateral radiography of the neck was taken with good extension of the neck and during inspiration so that the pharyngeal soft tissues were not mistaken for a retropharyngeal mass. Barium swallow was performed when vascular compression was suspected, while gastrograffin was used as contrast medium in the investigation for tracheo-oesophageal fistula. All the patients had fibreoptic-naso-pharyngoscopy examination as well as direct rigid bronchoscopy and oesophagoscopy under general anaesthesia. Rigid bronchoscopy performed under general anaesthesia gave a better view of the airway especially the part below the level of the vocal cords (subglottic area). It was also

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possible to take tissue biopsy with rigid bronchoscopy. Where endoscopy and contrast studies failed to demonstrate the lesion, computed tomographic (CT) scan and magnetic resonance imaging (MRI) were obtained to visualize the airway and the surrounding soft tissue structures including any evidence of vascular compression. To monitor the degree of respiratory distress, serial arterial blood gases were estimated to assess the degree of hypoxia and ventilatory status.

## Results

The total number of newborns with proven congenital stridor during the four-year period was 40 (25 males and 15 females). The overall admission in the corresponding period was 1050. Thus, the incidence of congenital stridor in the neonatal intensive care unit was four per 100 admissions. The stridor in these patients was inspiratory in 31 (77.5 per cent), expiratory in six (15 per cent) and biphasic in three (7.5 per cent ) of the patients. There were 29 (72 per cent) patients with lesions located in the extrathoracic tracheal zone, five (12.5 per cent), intrathoracic tracheal, and six (15 per cent), supraglottic and supralaryngeal zones. The causes of congenital stridor in the series are shown in Table I. Twenty-five (62.5 per cent) patients had laryngomalacia of whom only two (8 per cent) underwent supraglottoplasty; the remaining 23 (92 per cent) were managed conservatively with monitoring of their airways, weights, and heights in the clinic periodically, until the disease resolved spontaneously, usually before the age of two years. Three out of the five patients with subglottic stenosis were treated conservatively by close monitoring of their airways, weight and height until they were two years old. The remaining two who had marked subglottic stenosis underwent tracheostomy which was in place till the age of two years. This was necessary to maintain the infants at the normal weight and height for their age. In three infants, the stridor was associated with tracheo-oesophageal fistula and the defect was repaired by the paediatric surgeons. One female had subglottic haemangioma that was allowed to regress spontaneously. Two cases of bilateral vocal cord palsy were seen in the study and they were associated with hydrocephalus and cerebral palsy. The two cases of bilateral vocal cord palsy were managed by performing tracheostomies. The only case of laryngeal web was of thin membrane and this was treated surgically by passing bronchoscope and bougie. The cystic hygroma was surgically excised.

**Table I**

*Causes of Congenital Stridor in 40 Neonates*

| *Lesion* | *Male* | *Female* | *Total (%)* |
|---|---|---|---|
| Laryngomalacia | 15 | 10 | 25(62.5) |
| Subglottic stenosis | 3 | 2 | 5(12.5) |
| Tracheo-oesophageal fistula | 2 | 1 | 3(7.5) |
| Bilateral vocal cord palsy | 2 | - | 2(5.0) |
| Vascular compression | 2 | - | 2(5.0) |
| Laryngeal web | 1 | - | 1(2.5) |
| Subglottic haemangioma | - | 1 | 1(2.5) |
| Cystic hygroma | - | 1 | 1(2.5) |
| Total | 25 | 15 | 40(100) |

## Discussion

Congenital stridor is a common problem with various underlying causes. This study has identified laryngomalacia as the commonest cause of congenital stridor in the series. In a majority of cases, laryngomalacia is a benign condition requiring no surgical intervention. However in about 10 per cent of neonates, the condition can be life threatening.[5] In the present series, only eight per cent of the cases required supraglottoplasty. In the past, the standard treatment for these patients has been to perform tracheostomy but presently, better results are obtained with endoscopic surgery to the supraglottic structures in the form of epiglottoplasty or supraglottoplasty.[6,7] Studies have shown one type of laryngomalacia which is inherited in an autosomal dominant fashion.[8]

Anomalies of the aortic arch and pulmonary arteries may produce compression of the trachea with chronic stridor or wheezing aggravated during crying, feeding and flexion of the neck.[9] The trachea may also be compressed by mediastinal cysts, teratoma, lymphoma or lymphadenopathy resulting in tracheomalacia. We encountered only one case of vascular compression (vascular ring) caused by abnormal vessels passing between the trachea and oesophagus and this was referred to the cardio-thoracic surgeon. Tracheal compression due to solid mass or enlarged lymph nodes appear to be rare causes of congenital stridor.[10,11] Subglottic haemangioma occurs more commonly in girls, with a female ratio of 2:1.[9] The one case of subglottic haemangioma encountered in this study was in a female who was kept under observation till the mass spontaneously resolved.

This study has demonstrated that the commonest cause of congenital stridor in southwestern region of Saudi Arabia is congenital laryngomalacia. This is similar to global experience.[6,12] The treatment of stridor should be directed at the underlying cause. The airway should be established immediately in neonates with severe respiratory distress or actual airway obstruction. This can be done by endotracheal intubation. Investigations and examinations are not complete until the larynx and trachea are examined under general anaesthesia preferably with rigid bronchoscopes. It has been documented that acoustic analysis is a non-invasive procedure that can aid in the diagnosis of neonatal laryngotracheal pathology and to monitor the course of such disease.[13]

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