REVIEW PAPER

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Stridor in pediatrics is not only laryngomalacia

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ABSTRACT

Introduction. Stridor in newborns and infants is a common manifestation of airway obstruction that can be caused by both benign and severe pathologies.
Aim. This literature review examines the diseases that can present with stridor in children after birth, with the aim of better understanding their etiology, clinical course, and treatment.
Material and methods. The literature reviewed included published studies, reports, guidelines, and consensus statements on pediatric laryngeal pathologies associated with stridor.
Results. Laryngomalacia is the leading cause of stridor (up to 75% of cases) with a mostly mild course that requires only observation. However, stridor may also indicate other, often more serious laryngeal pathologies, such as vocal cord paralysis, subglottic stenosis, laryngeal cleft, and subglottic haemangioma, usually requiring surgical intervention.
Conclusions. Stridor is not a specific symptom of only laryngomalacia. Laryngeal pathologies manifested as stridor can have different etiology and, as a result, require a fundamentally different approach to treatment.

Introduction

Stridor is included as an entity in the International Classification of Diseases (R06.1 in ICD 10 and MD11.B in ICD 11), but it is only a symptom, not a diagnosis. It is described as noisy breathing that occurs due to obstruction at different segments of the airway. Due to the areas of obstruction in the airways of the child, a turbulent airflow is formed, which causes a characteristic whistling noise while breathing. Depending on the level of airway obstruction, the stridor can appear both in the inspiratory and the expiratory stages, and it can be biphasic in case of pathology at the level of the vocal fold, subglottis, or upper trachea areas. To make a correct diagnosis, it is important

to know the anatomy and understanding of the nature of the stridor, a thorough anamnesis, and of course, modern airway examination capabilities and methods. Voice condition also provides additional information to understand the level of obstruction and possible diagnosis. Hoarseness of the voice usually indicates an obstruction at the level of the vocal folds. A muffled voice is usually a symptom of processes at the level of the epiglottis. Stridor with congenital diseases can develop from the first hours of a child's life, as well as during the first days, weeks, or even months after birth. Patients of different age groups often have different congenital and acquired conditions that can manifest as stridor, which are important to recognize. A thorough examination of the patient, collecting anamnesis, and a deep understanding of the nature of the stridor will help to establish a preliminary diagnosis of the patient and provide an algorithm of actions for further invasive diagnostics and evaluation of the possibilities of conservative and surgical treatment of such patients. The authors aim to review the most common causes of stridor in children as well as modern approaches to the diagnosis and treatment of these conditions in pediatric patients.

Laryngomalacia

Laryngomalacia is the most common congenital laryngeal disease in children and reaches up to 75% of all stridor cases according to the literature [1,2]. Laryngomalacia is characterized by the collapse of the supraglottic structures during inspiration, which leads to a spurting airflow and high inspiratory stridor typical for the disease. In children with congenital laryngomalacia, stridor does not manifest immediately after birth, but rather a more typical progression of symptoms and the appearance of an obvious stridor at the age of several weeks. Symptoms may become more severe during the first 4–8 months of life. The stridor worsens with the baby's supine position, when the baby is nervous, during the crying and feeding process, and with upper respiratory tract infections. Approximately half of children with mild to moderate laryngomalacia will have feeding problems and problems in addition to respiratory symptoms. However, almost all children with mild laryngomalacia have feeding difficulties, which may be associated with episodes of cyanosis, cough, and choking. In children with severe disease, there is a lag in the rate of weight gain due to feeding disorders, which may be accompanied by aspiration pneumonia. Such patients have an unpredictable amount of calories consumed due to more frequent feeding and increased metabolic demand due to elevated respiratory work [3]. In the literature, there are usually three different types of laryngomalacia characterized by prolapse of excess mucosa into the lumen of the larynx, a combination of shortened scooped epiglottis and twisted omega epiglottis, a mobile epiglottis that collapses into the larynx during breathing [4].

Historically, laryngomalacia was considered to be a congenital anatomical pathology, but modern theories relate the disease more to a neuromuscular etiology, which is based on the failure of the peripheral nerves to maintain the shape of the airway, which ensures its patency and coordinates the act of swallowing in children. Some studies have investigated the threshold of stimulus to induce a normal motor response of the laryngeal structures in children with LM, the results of which were correlated with the severity of the disease [1]. As well as histological studies that revealed differences in the size of the branches of the upper laryngeal nerve in children with LM and healthy children of the same age. The neuromuscular etiology and the possibility of further development of the nervous system of newborns may explain the spontaneous improvement and recovery that is usually observed in children with LM [5].

A preliminary diagnosis of laryngomalacia can be made based on a characteristic anamnesis, symptoms, and a thorough physical examination of the child. The most characteristic of congenital laryngomalacia is the manifestation of symptoms from 2–4 weeks of life, unlike other pathologies that can manifest themselves with stridor from the first hours of life. Inspiratory stridor is characterized by worsening in the supine position, when the child is worried and during crying, in severe cases of the disease, weight lag is characteristic, so it is important to measure the child's height and weight. Feeding disorders may be accompanied by frequent aspiration pneumonia. Respiratory disorders can be accompanied by suffocation and cyanosis, it is important

to examine the child's chest for chest retraction or contraction during the act of inhalation. In most cases, a definitive diagnosis can be made by fibrolaryngoscopy without anesthesia during spontaneous breathing. Although fibrolaryngoscopy is the gold standard for the diagnosis of LM, it still has its limitations, such as the inability to assess structures below the vocal folds. It is also important to remember that the course of stridor and the severity of symptoms do not always have a direct correlation with the identified collapse of laryngeal structures and the severity of this collapse. If concomitant airway pathologies are suspected or if the clinical picture is inconsistent with the results of fibrolaryngoscopy, a deeper laryngotracheoscopy using rigid optics under general anesthesia should be performed. Such a technique makes it possible to visualize the larynx more accurately, unlike fiberoptic examination, which has its limitations in image quality due to the optical fibers. As well as performing endoscopy with rigid optics, it is even possible to examine the airways more completely to exclude comorbidities. There are also recommendations for performing polysomnography in children with severe LM and in children in whom surgical treatment has not given a significant improvement [6].

In children with mild laryngomalacia manifested by only a minor inspiratory stridor, a control examination should be performed once a month, in case of symptom stability or improvement, the interval between control examinations can be increased to 3–6 months. In the case of severe laryngomalacia that is additionally manifested by cough, choking, regurgitation, feeding difficulties, apnea, cyanosis, failure to thrive, pulmonary hypertension, and cor-pulmonale, it is necessary to start treatment with acid suppression therapy and consider feeding therapy and swallowing evaluation as well as laryngotracheoscopy and supraglottoplasty (which is the gold standard of surgical treatment). In the case of multiple severe comorbidities or multilevel airway obstruction not amenable to surgical management, it is necessary to consider tracheostomy [6].

Vocal cord paralysis

Vocal Cord Paresis or Paralysis (VCP) is another common cause of stridor in infants and older children, constituting up to 60% of stridor cases [7]. It is defined as an absence of vocal fold movement and can be bilateral (BVCP) or unilateral (UVCP). VCP is the effect of damaged nerve supply to the muscles of the larynx [8]. In approximately 40% of patients with VCP, another airway disease coexists, and laryngomalacia is the most common [3]. UVCP is more frequent, with a left fold predominance. Around 70% of UVCP cases have an iatrogenic origin, being mainly a result of cardiovascular or thoracic procedures. Prolonged intubation along with neurologic diseases and birth trauma compose approximately 50% of cases of BVCP and 21% are considered idiopathic [9]. In patients with BVCP who did not undergo any surgical procedures, neurologic diseases such as Arnold Chiari malformation or congenital myasthenic syndromes (CMS) need to be suspected, and so, further imaging and a careful neurologic examination is recommended [10]. In patients with Arnold Chiari malformation the protrusion of the brainstem through the foramen magnum is connected with compressing the vagus nerve which in effect contributes to VCP development [11].

Symptoms differ between patients with UVCP and BVCP and are more severe in BVCP. Neonates with BVCP present with a weak cry, dyspnoea, inspiratory stridor, or even cyanosis, whereas in patients with UVCP, a typical sign is dysphonia. In both cases, symptoms intensify upon agitation, although some UVCP might pass unrecognized [12]. Another great concern amongst patients with VCP is the risk of silent aspiration, reaching over 50% according to recent data, making the evaluation of swallowing function necessary [13]. To set the diagnosis in a stable patient a flexible fiberoscopy seems to be a procedure of choice [7].

The main goal of surgery in VCP is to achieve safe airway and feeding at the same time, as many neonates with VCP have concomitant problems with nursing and might require a gastrostomy or nasogastric tube feeding until a "safe swallow" is obtained [9]. The surgical approach depends on the patient's general condition, his comorbidities, especially the risk of aspiration, and spreads from endoscopic procedures such as adenoidectomy, vocal fold cordectomy, fold lateralization to open laryngotracheoplasty with a posterior cartilage graft or in some cases – a tracheostomy [14]. In cases of BVCP where surgical management is

focused on widening the posterior glottic space, the risk of future aspiration and voice disorders increases [15].

When establishing the best moment for surgical intervention, the surgeon should keep in mind that up to 70% of VCP cases recover spontaneously. The rehabilitation of damaged laryngeal nerve is frequently unpredictable and might last up to 12 months, with iatrogenic VFP unfortunately having the lowest percentage of healing. According to that, a patient should be observed for at least a year and, in cases of open surgeries, even up to two years before a surgical procedure is undertaken [7,16].

Congenital subglottic stenosis

Congenital subglottic stenosis (SGS) is usually identified by the authors as the third most common congenital laryngeal malformation after laryngomalacia and vocal fold paralysis [17]. About 30% of all congenital anomalies of the larynx that provoke stridor may be congenital subglottic stenosis [18]. Congenital SGS is defined as a constriction of less than 4 mm for full-term infants and less than 3 mm for preterm infants [19] at the level of the cricoid cartilage with the maximum level of constriction 2–3 mm below the vocal folds in children who had no previous episodes of intubation or endoscopic examinations. Congenital SGS requires evaluation of the larynx with fiberoptic endoscopy for dynamic assessment of the vocal folds and a further endoscopy using rigid optics as the best possible method of visualization and evaluation of the larynx.

Congenital SGC can be divided into two types: a membranous and a cartilaginous type, both of which occur as a result of incomplete recanalization of the laryngeal lumen during embryogenesis. The membranous type is much more common and occurs due to hypertrophy of the submucosa with an excess of fibrous connective tissue and is a milder type of congenital SGS. The cartilaginous type occurs due to an abnormal, usually elliptical, shape of the cricoid cartilage and is a more severe form of congenital SGS that can be classified as grade III-IV stenosis according to the Cotton-Myer grading system [20].

In children with mild subglottic stenosis, manifestations may appear only during recurrent respiratory infections, during which mild edema of the laryngeal mucosa can provoke airway obstruction (recurrent croup). In most cases, such mild forms of subglottic stenosis resolve spontaneously with the patient's growth, in other cases, endoscopic surgery with radial incisions of the membrane and following laryngeal dilation can be performed [21]. More severe cases can manifest as biphasic stridor and even as acute airway compromise during delivery, and such patients may require endotracheal intubation or even an emergency tracheostomy. In the case of endotracheal intubation, the risk of more severe acquired subglottic stenosis increases significantly, which may require open laryngotracheal reconstruction using costal cartilage as a laryngeal graft or cricotracheal resection [22].

Laryngeal webs

Congenital laryngeal webs (LW) constitute 5% of laryngeal congenital defects causing airway obstruction and arise between the 6th and 10th week of embryogenesis [23]. LW can present a wide range of symptoms – from voice disorders – such as hoarseness to severe dyspnea with stridor depending on the grade of larynx lumen involved in the process [24]. Since 1985, LW grading has been based on the Cohen scale, where type 1 are described as thin, membranous webs, occupying less than 35% of the glottis. Type 2 is defined as a web covering up to 50% of glottic space. Type 3 involves not only 50–75% of the glottis, but might spread to the anterior cricoid in the subglottic space. In type 4 – the most severe type – up to 99% of glottis is involved [25]. In cases of complete congenital laryngeal stenosis, a rare and fetal congenital high airway obstruction syndrome (CHAOS) might be suspected, as laryngeal atresia is its most frequent cause [26].

The way of treatment depends on the type of web and symptoms, where type 1 and 2 might not require any surgical intervention if asymptomatic, and remain only under observation. If symptomatic, they are almost always managed endoscopically. Web can be cut with the use of a cold steel laryngeal knife or a CO2 laser. In some cases, the laryngeal keel needs to be fixed in the anterior part of the glottis to inhibit scar-

ring. Types 3 and 4 usually require an emergency tracheostomy in the first days or months of life, before the target surgical approach is taken, which often consists of open laryngoplasty with rib graft placement [24,27].

Cases of type 2 webs give most concerns regarding surgical approach as they present a wide range of both morphological features and in consequence symptoms. They can be thin and membranous or thick with subglottic involvement which would demand different surgical treatments, and the approach must be tailored to a certain case not only to a web type [28].

In patients with laryngeal webs, there is a higher prevalence of other laryngeal abnormalities such as laryngeal subglottic stenosis or trachea-esophageal fistulas [24]. According to recent data, 30% up to 65% of patients with the laryngeal web have 22q11.2 deletion syndrome, commonly called DiGeorge syndrome. It is the most common chromosomal microdeletion and its characteristic features as well as comorbidities should be kept in mind when diagnosing a patient with LW [29].

Subglottic hemangioma

Hemangiomas are benign tumors, appearing in childhood, caused by abnormalities in angiogenesis. They are built up of endothelium cells and some parts of stroma such as fibroblasts, macrophages, and pericytes[30]. They are quite common as they occur in 4–10%of infants and they have a phase of rapid proliferation and involution [31]. Hemangiomas are a challenge in case they are part of some syndrome or are located in the airway tract, narrowing air flow, causing respiratory stridor and other severe breathing disorders.

Hemangiomas have two times higher prevalence among females [32]. They are more common among caucasian infants [33]. Around 60% of infantile hemangiomas are located around the head and neck but in contrast, subglottic hemangiomas (SGH) are infrequent benign tumors of airways and are responsible for 1,5% of congenital laryngeal anomalies [30]. Syndromes connected with hemangioma are hemangiomatosis, PHACE(s), sacral, and PELVIS syndrome.

The typical clinical course in infants with subglottic hemangioma (SGH) is asymptomatic in the early weeks of life. The first symptoms are biphasic stridor, recurrent or prolonged croup, and barking cough occur between 2–4 months. [32]. The basic diagnostic method consists of medical reconnaissance and rigid endoscopy. The most frequent SGH occurs in the left posterior portion of the subglottis. Pink or bluish, submucosal, smooth, round, compressible subglottic mass which leads to asymmetry and stenosis of the airway is a typical view in endoscopy. Diagnosis is based on endoscopic findings. Performing a biopsy is not recommended in this case [30].

Infantile hemangiomas are an interdisciplinary issue. There are a lot of advantages to using propranolol, a nonselective beta blocker. Treatment is initiated usually in the hospital to observe patients for unwanted reactions, at 1 mg/kg of propranolol 3 times a day, then increase to 2 mg/kg and is continued for 6–12 months [34]. Propranolol is considered the best treatment option for SGH nowadays but it is still under evaluation [32]. Response to treatment is confirmed by serial endoscopy. Other alternative treatment methods include intralesional and systemic steroids, CO2 laser resection, microdebrider submucosal resection, or open surgery. All these options have side effects and are considered only in certain cases [31].

Laryngeal cleft

Laryngotracheoesophageal clefts are a group of rare congenital anomalies characterized by the presence of pathological communication between the esophagus and the airway tract and are estimated to occur from 1 in 10,000 newborns to 1 in 20,000 newborns [35].

Classification of laryngeal clefts proposed by Benjamin and Inglis in 1989 remains the most popular and functional and identifies 4 main types [36]: Type I is a supraglottic intraaratenoid defect above the level of the true vocal folds; Type II includes a partial defect of the posterior cricoid cartilage below the level of the true vocal folds; Type III extends completely through the entire cricoid cartilage; Type IV extends to the intrathoracic trachea. Laryngeal cleft occurs due to abnormal embryogenesis, due to the absence of the interaretinoid muscle, the first type develops, the second type occurs due to incomplete develop-

ment of the posterior cricoid cartilage, the third and fourth types due to incomplete formation of the tracheoesophageal septum. Depending on the degree of cleft, the symptoms and the time of their manifestation may be different. In infants born with a low degree of cleft, the course may be asymptomatic at first. The main symptoms are respiratory distress, frequent coughing, recurrent croup or stridor, and recurrent respiratory tract infections. Deeper clefts of the larynx manifest more severely and quickly after birth and may present with severe respiratory distress immediately after birth. The most serious consequences of an undiagnosed laryngeal cleft are chronic aspiration and recurrent aspiration infections that can lead to lung damage.

Medical treatment is possible for type 1 laryngeal clefts, which includes primarily thickening of the fluid and food to prevent aspiration, as well as treatment of associated diseases that may lead to swallowing dysfunction or additional swelling (e.g. GERD or food allergies). Types 2, 3, and 4 clefts should be managed through surgical treatment. For type 1 and type 2 clefts, and with the rare exception of type 3, endoscopic cleft repair is the method of choice. Surgical treatment of clefts of types III and IV is mainly performed via the open approach [37].

Laryngeal cysts and Laryngocele

Congenital laryngeal cysts are a rare congenital anomaly occurring in 1.82 [38] to 3.49 [39] per 100,000 newborns or 2% of all patients with stridor [39] (congenital laryngeal saccular cysts represent 1.5% of all patients with stridor) [40]. There are several classifications of congenital laryngeal cysts, among which the classification of DeSanto [41], who identified saccular and ductal types of cysts, should be highlighted. Forte's classification, which was based on the differentiation into types as a basis for treatment approaches, defined two types depending on the size and embryologic tissue origin. Type I cysts consist only of endodermal elements are located entirely in the larynx, and can be removed completely with endoscopic surgery. Cysts of the second type have extralaryngeal extension, open surgical access is used for their removal, and depending on the presence of mesodermal tissues in addition to endodermal tissues, the author divided the type into additional subtypes 2a and 2b [42]. The most common symptoms in children with laryngeal cysts are stridor, feeding difficulties and failure to thrive, episodes of cyanosis. It should also be noted that in recent studies [43] it was reported that 2/3 of patients with vallecular cysts had coexisting laryngomalacia, and in a study by Yang Xiao et al. 35.7% of patients with congenital laryngeal saccular cysts were misdiagnosed as laryngomalacia [44]. Flexible fiberoptic laryngoscopy is used for initial diagnosis, endoscopic laryngotracheoscopy under general anesthesia is the gold standard, and CT scanning can also be useful for the evaluation of cyst extralaryngeal extension [44]. Treatment of congenital laryngeal cysts consists of surgical removal or marsupialization. In the case of ductal cysts and saccular cysts without extralaryngeal extension, it is recommended to perform surgery at the time of diagnostic laryngotracheoscopy [45].

Laryngocele is a very rare congenital anomaly of the larynx, which is an air-filled cystic dilated laryngeal saccule [46]. It is manifested by a stridor, in severe cases it can cause respiratory distress, and significant airway obstruction in the infant, and can be life-threatening [47]. A tracheostomy should be performed as an emergency treatment for a newborn with severe dyspnoea and laryngocele. Treatment of laryngocele is only surgical, marsupialisation of the cyst walls under endoscopic control is the optimal minimally invasive treatment.

Conclusions

Although stridor is usually a symptom of laryngomalacia, which in most cases has a benign course, this symptom is not specific only to laryngomalacia and can be a sign indicating the presence of another serious disease. Children with stridor require significant attention and appropriate diagnostics because stridor can be a manifestation of many different laryngeal pathologies in terms of symptoms, etiology, course, and consequences, which may require completely different approaches to the treatment of such patients. Stridor in children after birth should not be underestimated or ignored.

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