Volume 4 | Issue 18

SMART PEDI UPDATE

Pedismart Newsletter
Vol 4 | Issue 18 | 2024

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Dear Doctor,
Greetings from Micro Labs Limited. We are happy to bring you **"PEDI SMART NEWSLETTER**" which contain latest and interesting news in the field of Paediatrics.

This issue contains:

1. **Clinical characteristics and associated serological profiles of Lyme disease in children**
2. **Association between obesity and chronic rhinosinusitis, and asthma in children**
3. **Trends in childhood mental, behavioral, and developmental disorders**
4. **Case Report on Misdiagnosis of tracheal stenosis as asthma**

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team, Micro Labs Limited

SMART PEDI UPDATE
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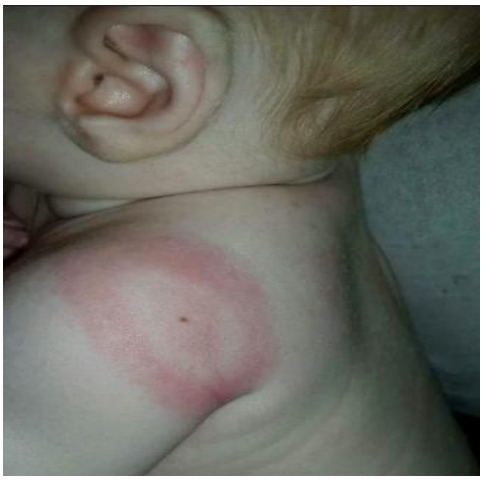
Clinical characteristics and associated serological profiles of Lyme disease in children

Introduction



Lyme disease (LD) is the most prevalent tickborne disease, caused by different genospecies of the bacteria Borrelia burgdorferi.[1] An average of 15% of Ixodes ricinus ticks in Europe are infected with B. burgdorferi infection, and 3% of people are affected with LD after an I. ricinus tick bite. The peak tick feeding seasons, May to October, are when most LD cases occur, with incidence varying across regions and countries. Children with LD can present with a variety of clinical symptoms. It typically affects the joints, skin, nervous system, and less frequently, the heart. Skin manifestations in children include erythema migrans, borrelial lymphocytoma, and acrodermatitis chronica atrophicans, which are rare.[2,3] Lyme neuroborreliosis (LNB) often presents with cranial nerve impairment and lymphocytic meningitis in children, while meningoradiculitis is often seen in adults, and is uncommon in children.

The diagnosis of LD is based on symptoms and the presence of antibodies specific to B. burgdorferi in serum and, in the case of LNB, cerebrospinal fluid (CSF). Intrathecal antibody production against B. burgdorferi in CSF remains the gold standard for diagnosing LNB. [3] Moreover, serum antibodies against B. burgdorferi persist for months to years following the initial infection, which is a significant drawback of serological testing. Furthermore, the diagnostic standards that are now in use for adults have not yet been approved for use with children. This is caused, at least in part, by a dearth of precise information regarding the immunological and clinical characteristics of LD in children. So, this study investigated the serological profiles linked to various manifestations of LD in children and provided a detailed description of the epidemiological, clinical, and laboratory features.

Methods

This retrospective cohort study included children and adolescents diagnosed with LD who were ≤18 years of age. Children whose etiology was unclear or who had other diagnoses and/or aetiologies were excluded. Patients who met diagnostic criteria based on current guidelines were finally included in the study. All patients with a positive serological test result, which consists of a complete IgM and/or IgG Western blot for all eight B. burgdorferi antigens were included in the subcohort. Clinical, laboratory, demographic, and epidemiological data were extracted from medical records. White blood cell count (WBC), C-reactive protein (CRP), serum glucose, CSF cell count, and CSF glucose, protein, and lactate were all considered. The study identified B. burgdorferi-specific IgM and IgG antibodies in serum and CSF using a two-

tier serology. An enzyme-linked immunosorbent assay (ELISA) was used for the first-tier screening of all serological samples. If screening was positive, a second-tier immunoblot was performed to detect specific antibodies. Fisher's exact tests for categorical variables and Kruskal-Wallis rank sum tests for continuous variables were used to evaluate differences between the three groups, and unadjusted P-values were reported.

Results

The study included 469 children with LD (median age, 7.9 years), with 190 (40.5%) having Lyme neuroborreliosis (LNB), 171 (36.5%) having skin manifestations (erythema migrans, n = 121; multiple erythema migrans, n = 11; borrelial lymphocytoma, n = 37; and acrodermatitis chronica atrophicans, n = 2), and 108 (23%) having Lyme arthritis. LD was most diagnosed between May and October, with peaks in June and July for cutaneous symptoms and July and August for LNB. Clinical symptoms varied significantly by age, with Lyme arthritis patients being older than those with cutaneous signs and LNB. Patients under 5 years (18.6%) primarily had EM and lymphocytoma (60.9%). In total, 52 patients (11.1%) had underlying illnesses. 203 (43.3%) LD patients reported a tick bite, while 164 (35.0%) had a history of a rash like EM. In addition, patients with Lyme arthritis showed significantly higher WBC counts, including neutrophils and monocytes, and CRP levels compared to patients with skin manifestations and LNB. Significant differences were observed in serological profiles across the various clinical groups. IgG antibodies in skin manifestations and Lyme arthritis targeted B. burgdorferi antigens p100, VlsE, p58, p41, p39, and p18, while LNB patients showed IgG reactivity to VlsE, p41, and OspC (Figure 1). Skin manifestation and Lyme arthritis showed stronger IgG responses to p100, VIsE, p58, p39, and p18; minimal reactivity was found for OspA (IgG) and OspA, p58 and p18 (IgM).

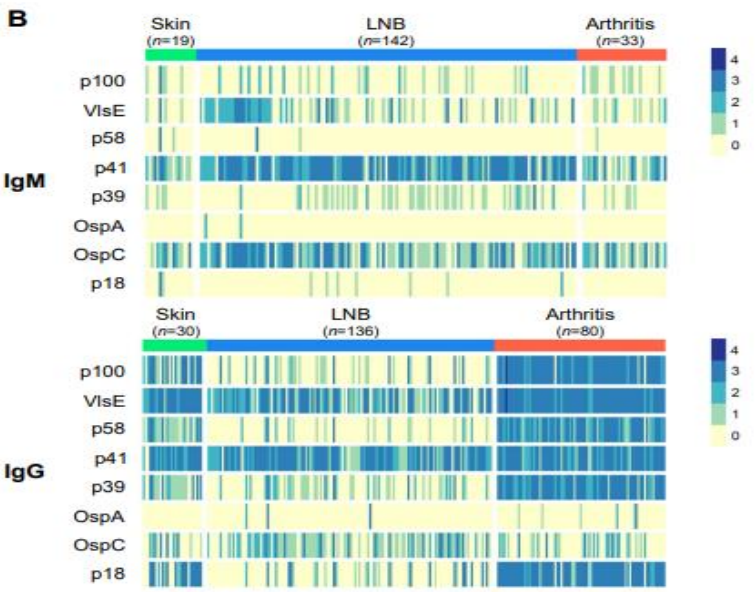


Figure 1. Serum antibody responses to distinct Borrelia burgdorferi antigens in various LD symptoms.

Discussion

This study found that IgG isotype responses against B. burgdorferi antigens were associated with Lyme arthritis, while IgM isotype responses against B. burgdorferi antigens may indicate LNB. A prior study supports these findings, demonstrating that a panel comprising these three antigens could enhance early disseminated LD diagnosis and also notice distinct serologic correlates of LD stage, indicating a part played by the host immune response in clinical manifestation.[4] Better diagnostic techniques that can accurately identify B. burgdorferi infection at all stages of the disease are required in light of the rising prevalence of LD and the difficulties in diagnosing it.[5]

Conclusion

Clinical characteristics and specific serum antibody response patterns varied amongst LD manifestations. Distinct serum antibody responses of the IgG isotype against six B. burgdorferi antigens (p100, VlsE, p58, p41, p39, and p18) were associated with Lyme arthritis, and the IgM isotype against three B. burgdorferi antigens (VlsE, p41, and OspC) may aid in the prediction of LNB.

Early recognition of symptoms and targeted serological testing are critical for effective management of lyme disease.

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Association between obesity and chronic rhinosinusitis, and asthma in children

Introduction

Childhood obesity affects 20% of children in United States and has become an increasing healthcare concern.[1] Obesity has been associated with several conditions in children such as reactive airway disease, hypertension, diabetes mellitus type II, obstructive sleep apnea (OSA), and sleep disordered breathing.[2,3] It is recognized as both risk factor and disease modifier in children with asthma. Chronic rhinosinusitis (CRS) and asthma often coexist, supported by unified airway theory. Although obesity has been linked to CRS and asthma in adults, study into the relationship in children are scarce.[3] This study aimed to assess the relationship between childhood obesity and the unified airway, specifically CRS and asthma, while accounting for comorbidities.

Methods

This cross-sectional study included paediatric patients diagnosed with CRS of age 18 years and younger. The endoscopic evidence of CRS was graded using the modified Lund Kennedy scoring system. The Lund-Mackay scoring system was used to assign scores to CT scans. After being chosen for the study, the patients were categorised according to their body mass index (BMI). According to Centres for Disease Control BMI for-age growth chart, patients who had a BMI at or above the 95th percentile were categorised as obese. Data were collected on demographic and comorbid conditions. OSA was identified with polysomnography, AR with positive skin or blood testing, and asthma with published recommendations from the Global Initiative for Asthma. Obesity and non-obesity groups were compared using univariate and multivariate binary logistic regression models.

Results

The study included 406 pediatric patients of with 130 children (32%) were classified as obese. The mean CT Lund-Mackay score for children with CRS (N=131) was 7.2 (SD of 6.3, N=63), and the mean endoscopy modified Lund-Kennedy score was 2.7 (SD of 2.9, N=81). Obese children had higher odds of having OSA (26.2% vs. 13%, P=0.001) and asthma (28.5% vs. 15.2%, P=0.002), and they were older (11.3 years vs. 10.2 years, P=0.039). Obesity is associated with asthma (OR=1.84, P=0.029) but not with CRS (OR=1.08, P=0.856) or AR (OR=1.05, P=0.856), according to multivariate logistic regression analysis (Figure 1).

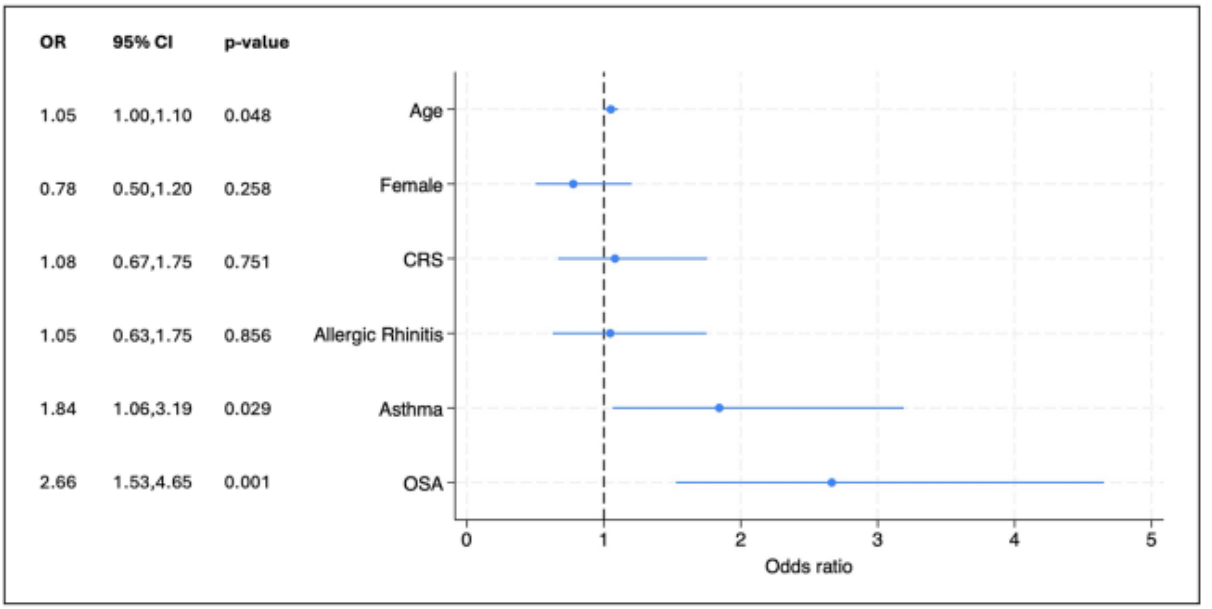

Figure 1. Association of obesity with chronic rhinosinusitis (CRS), asthma, and allergic rhinitis by multivariate logistic regression analysis

Discussion

Obesity has become a growing public health concern; therefore, it is crucial to understand the comorbidities linked to the disease. Although several studies have linked obesity to CRS in adults, studies on this subject in the pediatric population are limited. Sidell et al.'s analysis of school-aged children found no correlation between childhood obesity and CRS, a finding that was consistent with this study.[4] Obesity was strongly associated with asthma, consistent with earlier studies, suggesting that inflammation in asthma is amplified in obese patients.[5] The difference in the association between obesity and CRS findings between adults and children is intriguing. Although there is consistent evidence linking obesity and CRS in adults, the reason why this is different in children is unknown. Differences in the endotypes and inflammatory markers, Leptin's role in increasing the production of proinflammatory cytokines, may play a role in obesity-associated respiratory disease. Additional investigation is required to assess these differences.[3]

Conclusion

Obesity is associated with asthma in children but not with CRS. Further study is required to understand the selective impact of obesity on the lower airway and its potential impact on CRS outcomes.

Obesity is a significant risk factor for asthma in children, but no association was found with chronic rhinosinusitis.

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Trends in childhood mental, behavioral, and developmental disorders

Introduction

Childhood mental, behavioral, and developmental disorders (MBDD) are common conditions associated with chronic diseases, poor physical health and reduces educational and economic outcomes.[1] Early risk and health promotion factors that may impact population health can be highlighted by monitoring the prevalence of MBDDs in children and the related factors that may impact health outcomes. MBDDs include attention deficit/hyperactivity disorder [ADHD], behavioral/conduct issues, anxiety issues, depression, and Tourette syndrome, as well as developmental, learning, and language disorders, such as autism spectrum disorder [ASD], learning disorders, intellectual disability, developmental delay, and speech and other language disorders. MBDDs frequently co-occur and share protective and risk factors.[2,3] The COVID-19 pandemic worsened numerous stressors linked to MBDDs in children and had an impact on social determinants of health. According to studies, there were more risk factors for MBDDs during pandemic such as increasing financial stress, unmet healthcare needs, and barriers to early identification of delays.[3] Information is limited about trends in the prevalence of MBDDs and related health promotion and risk indicators over the period before and during the pandemic. This study examines trends in childhood MBDDs and related health care family, and community indicators to determine potential areas to consider for targeted efforts by healthcare and public health professionals to support children and families and improve access to resources.

Methods

This cross-sectional study analysed trends in childhood MBDDs and related risk factors and health promotion from 2016 to 2021 using information from the National Survey of Children's Health (NSCH). In non-institutional settings, parents of children ages 0 to 17 answered an address-based survey via mail or online. For this study, only children between the ages of three and seventeen were included in the analytical sample. Children (0.07% of our study sample) with missing data on more than three MBDD items were excluded in the analyses. MBDDs were categorized into two subgroups 1) mental, emotional, and behavioral disorders (MEB) and 2) developmental, learning, and language disorders (DLLD). Weighted prevalence estimated overall and stratified by MBDD diagnosis, MBDD subgroup (MEB, DLLD), sex, age, race or ethnicity, household income, education level, primary household language, and residence. MBDDs were compared on health care, family, and community indicators both overall and by MBDD subgroup.

Results

The lifetime prevalence of MBDDs in children between the ages of 3 and 17 increased from 25.3% to 27.7% (average annual percentage change, AAPC = 1.8; 95% CI, 1.1–2.5) from 2016 to 2021. Among MBDD subgroups, there were an overall increase in MEBs (AAPC = 2.3; 95% CI, 1.5–3.2) and DLLDs (AAPC = 1.8; 95% CI, 1.0–2.6). Increases were specific to depression (AAPC = 5.3; 95% CI, 1.9–8.9), learning disabilities (AAPC = 2.9; 95% CI, 0.1–5.9), developmental delays (AAPC = 2.5; 95% CI, 0.4–4.7), anxiety disorders (AAPC = 6.0; 95% CI, 2.8–9.2), and speech or other language disorders (AAPC = 3.0; 95% CI, 1.5–4.7) (Figure 1). During the study period, children's overall unmet health care needs in the previous 12 months increased by an average of 5% per year. In the previous 12 months, about 60% of children with MBDDs received mental and/or developmental services annually during the study period (MEB, 62.0%; DLLD, 69.1%). Every year, a greater percentage of parents of children with MBDDs than parents of children without MBDDs reported experiencing economic stress (17.6% MBDD, 10.2% no MBDD) and poor mental health (16.8% MBDD, 6.7% no MBDD).

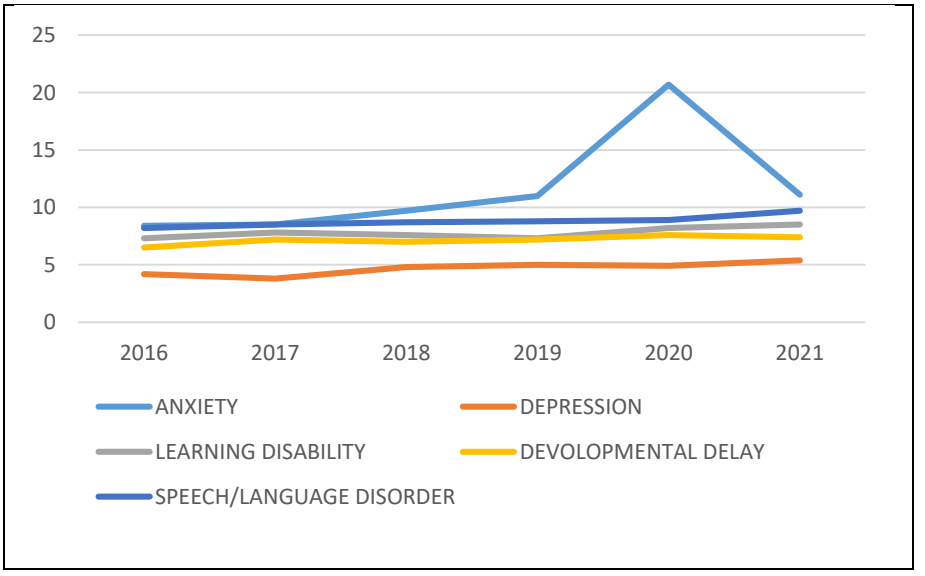

Figure 1. Prevalence of mental, behavioral and developmental disorders from 2016 - 2021

Discussion

This study observed an increase in the prevalence of MBDDs such as anxiety, depression, developmental delays, learning disabilities, and speech or language disorders, as well as associated risk indicators like unmet healthcare needs and poor parental mental health. This was consistent with previous studies showing the rising prevalence among some MBDDs before and during the pandemic.[4] Poor parental mental health has increased, particularly among parents of children with DLLDs. Approximately 2.5 times more children with DLLDs than those without MBDDs had a parent with poor mental health. Monitoring changes in the prevalence of these contexts and factors can help identify areas where intervention and prevention efforts are required.[5]

Conclusion

There was an increase in the prevalence of specific MBDDs and MBDD-related indicators before and during the COVID-19 pandemic, emphasizing the need for prevention and intervention initiatives, better pediatric mental health training for healthcare professionals, and policies that address financial stability and fair access to mental health services.

Enhanced pediatric mental health training is essential to address the increasing prevalence of childhood mental and developmental disorders

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Case Report on Misdiagnosis of tracheal stenosis as asthma

Introduction

Tracheal stenosis can result from a variety of causes such as trauma due to surgery, intubation, accidents, inhalation injuries, and inflammatory conditions.[1] Most cases are caused by tracheostomy or prolonged intubation. Up to 11% of patients who undergo tracheostomy or intubations may develop airway stenosis, even just 24 hours after intubation.[2] Tracheal stenosis is often challenging to diagnose and manage in children. The common presentation of tracheal stenosis includes general respiratory symptoms of wheezing, dyspnea, exercise intolerance, stridor, or cough. Even a mild respiratory infection can worsen breathing difficulties. It is important to consider tracheal stenosis in patients with a history of tracheostomy or intubation who present with dyspnea. These symptoms may not initially indicate airway stenosis but may be misdiagnosed as asthma, a common condition in pediatrics. By the time symptoms manifest, tracheal stenosis can affect 30 to 50% of the lumen diameter.[3]

Case Presentation

A 10-year-old male child with a history of adrenal insufficiency but no prior asthma history was admitted to the emergency department with coughing, wheezing, and increased work of breathing for 1 day. He had been admitted ten months prior with shock and an adrenal crisis, necessitating three days of intubation. After a successful extubation without complication, he was sent home. However, he made several visits to the emergency department following intubation coughing, wheezing, and increased respiratory effort. He was treated as having acute severe asthma due to his bilateral expiratory wheezing and respiratory distress. Similar symptoms, such as increased work of breathing, were present during this visit, necessitating admission to the pediatric intensive care unit (PICU) because his condition did not improve with initial treatment. Upon examination, he was tachypneic with bilateral wheezing on auscultation and hard vesicular breathing. Leukocytosis was found in laboratory tests, along with a left shift and a white blood cell count of 14400 cells/cu.mm. The differential count revealed 6.3% lymphocytes and 89% neutrophils. The arterial blood gas analysis revealed a pH of 7.42, a partial pressure of carbon dioxide of 32.9 mmHg, a partial pressure of oxygen of 75 mmHg, bicarbonate of 20.90 mEq/L, and a base excess of -2.8 mEq/L. Hemoglobin was 11.3 g/dL, and platelets were 3.27 lakhs/cu.mm. Despite the absence of usual evidence of hyperinflation on the patient's chest X-ray, asthma was assumed based on the patient's history of repeated episodes that responded to bronchodilator medication, bilateral wheezing, and increased work of breathing. Beta-agonists, anticholinergic medications, systemic steroids, magnesium sulfate, terbutaline injection, and non-invasive face mask-based bilevel positive pressure breathing were all used during treatment. His symptoms persisted, however, and he eventually developed hypoxic respiratory failure, necessitating intubation. A video

laryngoscope was utilized to perform rapid sequence intubation; however, the endotracheal tube was unable to pass through the vocal cords. A size 2.5 laryngeal mask airway was inserted, mask and bag ventilation were given, and an emergency tracheostomy was carried out and through this child's airway was able to be effectively controlled. A follow-up bedside bronchoscopy revealed grade III tracheal stenosis, but the tracheostomy was maintained intact. The patient was effectively weaned off mechanical ventilation, eliminating the need for additional asthma therapy. Three to four weeks after the patient's release, a follow-up plan for open resection and anastomosis or laser treatment was given. After three weeks, a carbon dioxide laser excision of subglottic stenosis was carried out, and the patient's tracheostomy tube decannulation succeeded. After an uneventful postprocedure course, he was sent home on oral medicine.

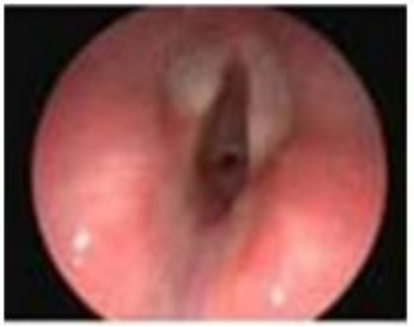

Figure 1. Preintubation tracheal stenosis

Discussion

Tracheal stenosis is often misdiagnosed as asthma even in the absence of typical signs such as hyperinflation on chest X-ray, particularly in pediatric patients with a history of intubation. In this case, the patient experienced long-term intubation for three days, which can lead to subglottic stenosis due to mechanical trauma and other factors like endotracheal intubation, including the endotracheal tube's size in relation to the child's larynx, its motion, and the length of the intubation. This is consistent with the findings of Schweiger et al. who demonstrated a higher risk of subglottic stenosis in children who were intubated and not properly sedated.[4] The most effective technique for identifying upper airway blockage is through flow-volume loops (FVL). A flow-volume loop could have been useful in distinguishing between extra-thoracic obstruction like subglottic stenosis and intra-thoracic obstruction like asthma.

Conclusion

Tracheal stenosis is frequently mistaken for asthma in patients with a history of intubation. When a patient shows signs and symptoms of respiratory distress, tracheal stenosis should be included in the differential diagnosis and the airway should be promptly secured in a controlled environment by an anaesthesiologist or otolaryngologist.

Misdiagnosis of asthma in patients with tracheal stenosis can delay critical interventions and delay outcomes.

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