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Prevalence and associated factors of pulmonary hypertension in Kenyan children with adenoid or adenotonsillar hypertrophy

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ABSTRACT

Objectives: Adenotonsillar hypertrophy is a common condition in childhood, whose serious complications of pulmonary hypertension and cor pulmonale are devastating but local prevalence is unknown. This study determined the prevalence and associated factors of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at Kenyatta National Hospital, Kenya.

Methods: This was a cross sectional hospital based survey conducted among children below 12 years of age with clinical and radiological adenoid hypertrophy attending the ear, nose and throat (ENT) outpatient clinic and general pediatric wards. Doppler echocardiography was used to determine pulmonary hypertension defined as a mean pulmonary arterial pressure (mPAP) of ≥25 mm Hg using the Chemla equation. Children with mPAP of ≥25 mm Hg were compared to those with lower pressures and clinical and radiological factors associated with pulmonary hypertension determined using multivariate logistic regression analysis.

Results: Of the 123 eligible children in the study, 27 had pulmonary hypertension giving a prevalence of 21.9% (95% CI 14.64%–29.27%). Independent factors associated with pulmonary hypertension included nasal obstruction (OR = 3.0 [95% CI 1.08–8.44] $p = 0.035$) and hyperactivity on history (OR = 0.2 [95% CI 0.07–0.59] $p = 0.003$) and adenoid-nasopharyngeal ratio (ANR) >0.825 on lateral neck radiography (OR = 5.0 [95% CI 1.01–24.37] $p = 0.048$).

Conclusion: One in five children with adenoid or adenotonsillar hypertrophy had pulmonary hypertension with a 3-fold and 5-fold increased odds in those with nasal obstruction on history and ANR >0.825 on lateral neck radiography respectively and an 80% reduced odds in reportedly hyperactive children.

Introduction

Adenotonsillar hypertrophy is a common condition in childhood and is the most common cause of upper airway obstruction and sleep disordered breathing in children [1]. Pulmonary hypertension and cor pulmonale are serious potentially fatal complications of upper airway obstruction [2]. Upper airway obstruction causes hypoxemia, hypercarbia and respiratory acidosis, all of which are potent vasoconstrictors of the pulmonary vasculature [3]. In the acute phase, pulmonary vasoconstriction is reversible if airway obstruction is alleviated. However, chronic vasoconstriction results in structural remodeling of the pulmonary vascular bed and subsequently irreversible pulmonary hypertension and cor pulmonale ensue [3,4]. Globally, the prevalence of pulmonary hypertension among children with adenoid or adenotonsillar hypertrophy varies widely between 7.3% and 51.9% based on echocardiography studies [5–8].

Annually there are over 6000 clinical contacts made with children with adenoid or tonsillar hypertrophy at the ear, nose and throat (ENT) outpatient clinic at the Kenyatta National Hospital (KNH), Nairobi's leading referral health facility [9]. The objective of this study was to determine the prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH, Kenya and secondarily determine clinical-radiological factors associated with pulmonary hypertension. Estimating the burden of pulmonary hypertension via echocardiography and identifying associated factors in these children in our setting allows for early clinical recognition of children at risk of pulmonary

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hypertension and their prioritization for appropriate and timely interventions such as adenoidectomy or adenotonsillectomy to prevent fatal sequelae.

Materials and methods

A hospital based cross sectional descriptive study was carried out within the ENT department and pediatric wards of KNH over a period of 4 months among children aged 0–12 years with clinician diagnosed adenoid hypertrophy with or without tonsillar hypertrophy as the only cause of upper airway obstruction, confirmed on lateral neck radiography and had written informed consent from their guardians.

Children were excluded from the study if they had neurologic abnormalities such as cerebral palsy, genetic syndromes with craniofacial abnormalities such as Down syndrome, or other causes of upper airway obstruction such as deviated septum, nasal polyposis, gross turbinate hypertrophy or a body mass index (BMI) >95th percentile for the age. Gross inferior turbinate hypertrophy in this study was defined as complete occlusion of the nasal cavity (Grade III according to Friedman et al.) on either the left or right side [10]. Children were also excluded if they had other co-morbidities known to be associated with pulmonary hypertension such as a known diagnosis human immunodeficiency virus (HIV), sickle cell anemia, chronic lung disease, cardiac disease or cardiac disease discovered on echocardiography that could otherwise explain the pulmonary hypertension. In addition, children whose caregivers refused to grant written informed consent or for whom echocardiography was not performed were excluded.

Upon enrollment, history was obtained from the caregivers and physical examination performed on all children comprising a general, ENT and cardiovascular evaluation. Grading of tonsil size on physical examination employed the Brodsky classification [11]. Lateral neck radiography was performed in the KNH radiology department using a standardized technique and image quality was assessed. Good quality image was defined as one without rotation or magnification and was taken with the mouth closed.

A diagnosis of adenoid hypertrophy was made by any hospital clinician if a child had suggestive symptoms or signs of upper airway obstruction confirmed as adenoid hypertrophy on lateral radiography by any hospital radiologist and had tonsil grade 0–2 on physical examination assessed by the study clinician using the Brodsky classification. A diagnosis of adenotonsillar hypertrophy was made by any hospital clinician if a child had suggestive symptoms or signs of upper airway obstruction confirmed as adenoid hypertrophy on lateral radiography by any hospital radiologist and tonsil grade 3 or 4 on physical examination assessed by the study clinician using the Brodsky classification [11]. The anterior nasal examination was conducted by the study clinician by visual inspection using a head lamp as a light source. Children without Grade III inferior turbinate hypertrophy according to the Friedman classification (i.e., those with Grade I – mild nasal obstruction; and Grade II – between Grade I and Grade III) were included and the analysis was conducted on these children as a combined group [10].

The radiographs were reviewed by a pediatric radiologist who was blinded to the severity of the patients' disease. Adenoid size, nasopharyngeal size, tonsil size and pharyngeal size were measured in centimeters to the nearest decimal point with a ruler and the adenoid nasopharyngeal ratios (ANR) and tonsil pharyngeal ratios (TPR) were determined using a standardized technique proposed by Shintani [12]. In the literature an ANR cut-off of >0.63 has been used as a criteria for enlarged adenoids on lateral neck radiography although this was not used as a criteria for inclusion in our study [13]. An ANR cut-off of 0.825 was an arbitrary cut-off taken in this study that is between 0.63 and 1, as all children in our study had an ANR >0.63. Granzotto et al. previously reported a TPR cut-off of >0.66 on lateral neck radiography to be a predictor of pulmonary hypertension in children with adenoid hypertrophy [7]. These various cut-offs were used to report measurements taken on lateral neck radiography for our study patients.

Echocardiography was performed by the pediatric cardiologist who was also blinded to the severity of the patients' disease. Transthoracic M-mode, 2D echo and Doppler echocardiography were carried out using a portable VIVID I Echo Color Ultrasound System echocardiography machine. Cardiac measurements were performed according to the guidelines of the American Society of Echocardiography [14]. Systolic pulmonary artery pressure (sPAP) was measured and the modified Bernoulli equation applied using the tricuspid regurgitation jet [15]. Mean pulmonary artery pressure (mPAP) was then derived using the Chemla equation = $(0.61 \times \text{sPAP}) + 2$ mm Hg [16]. Estimated mPAP using the Chemla equation has been shown to have an acceptable accuracy of 77–98% within 10 mm Hg of right heart catheterization measured mPAP and thus suitable for clinical use [17]. In addition, an estimated mPAP $\geq$25.5 mm Hg using the Chemla equation is useful in the diagnosis of pulmonary hypertension with an excellent sensitivity (98%), specificity (100%), positive (98%) and negative (88%) predictive value [18].

Pulmonary hypertension was defined as an estimated mPAP of $\geq$25 mm Hg [15]. Digital images and videos of the echocardiographs were stored for future validation. During the procedure young infants were pacified with breastfeeding or distracted with toys. Light sedation with oral chloral hydrate at a dose of 25 mg/kg/dose was administered when needed. Ethical approval was sought from Kenyatta National Hospital Scientific and Ethics Committee and the study was performed in accordance with the guidelines of the Declaration of Helsinki.

Statistical analysis

Data were entered in a preformed Microsoft Access database and analysis performed using STATA version 12.0. Chi square test of association or Fisher's exact test where appropriate were used to compare categorical variables and an odds ratio was reported to give an estimate of risk. Student's *t*-test was employed when means were compared and Mann–Whitney *U* test was used to compare medians. A multivariate logistic regression analysis was conducted with the main outcome as presence or absence of pulmonary hypertension.

Results

From September to November 2012, a total of 127 children meeting study eligibility criteria were enrolled of whom 123 completed the required echocardiography evaluation and qualified for further analysis.

As shown in Table 1, the median age of the study population was 2.5 years, with a male to female ratio was 1.5:1. Overall, 94.4% of the study participants were well nourished with weight for height Z-scores >−2 with only six (4.8%) moderately wasted and one (0.8%) severely malnourished. The median duration of symptoms suggestive of upper airway obstruction was 14 months, ranging from 1 to 60 months and 93 children out of 123 (75.6%) were on intranasal steroids. Majority of the patients 115 out of 123 (93.5%) were recruited from the ENT clinic. Only 39 out of 123 (31.7%) were scheduled for corrective surgery.

The commonest symptoms reported by guardians of the 123 study participants were frequent upper respiratory infection (URTI) by 119 (97%), snoring 118 (96%), restless sleep 111 (90%) and frequent awakening 99 (80%). Moderately frequent reported