

Case Report

Chronic Upper Airway Obstruction as a Main Cause of Pulmonary Hypertension

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Abstract

Introduction

The obstruction of the superior airways in the childhood turns from several causes, among them the commonest is the adenotonsillar hypertrophy [1,2]. This chronic obstruction frequently is associated by syndrome of the apnea of the sleep and, in the most serious cases, they evolve with pulmonary hypertension [3]. The pulmonary hypertension is an indisputable indication for realization of adenotonsillectomy [4,5].

Objective

To present a case of pulmonary hypertension secondary of the adenotonsillar hypertrophy in Cardiology department of a university hospital.

Case Relate

3.5 years old girl admitted with respiratory difficulty, coughing, nocturnal snores and mouth breath. Cardiomegaly was found in chest XR. The Doppler echocardiogram study showed pulmonary arterial hypertension. The lateral X ray films showed important adenoid hypertrophy. Adenotonsillectomy was carried without problems. After six months, the patient did a new Doppler echocardiogram that showed normal, showing clear reversion of all alterations shown in the first echocardiogram.

Conclusion

Patients with adenotonsillar hypertrophy and chronic upper airway obstruction, may present with evaluated a pulmonary hypertension. The Doppler echocardiogram is safest exam and little invasive for determination of the diagnosis [5,6] and the adenotonsillectomy is the definitive treatment

Keywords: Adenoid Hypertrophy; Adenotonsillar Hypertrophy; Pulmonary Hypertension; Upper Airway Obstruction

Introduction

The obstruction of the superior airways in the childhood turns from several causes, among them the commonest is the adenotonsillar hypertrophy [1,2]. This chronic obstruction frequently is associated by syndrome of the apnea of the sleep and, in the most serious cases; they

evolve with pulmonary hypertension [3]. The pulmonary hypertension is an indisputable indication for realization of adenotonsillectomy [4,5]. Because that we describe a case of sever pulmonary hypertension secondary of the adenotonsillar hypertrophy in the cardiology department of a university hospital.

Case Report

3.5 years old girl admitted to the hospital with cough ,history of recurrent chest infections from 2 years (4 to 5 times a year) treated by oral antibiotics without hospitalization , breathing difficulty with any exercise

No history of cyanosis or syncope, Failure to thrive (WT=12kg <3%, HT=90cm <3%), History of snoring and mouth breathing

On physical examination: Loud S2, soft systolic murmur II/VI, chest: Harsh crackles bilateral

Hypertrophied tonsils, other systemic examination was normal

Labs: WBC: 6200(N 33%, L 66%), Hgb 11g/dl, CRP negative

Electrolytes, liver function, renal function and Immune globulins were normal.

Blood gases: During the day: PH=7.41, PCO2=34, PO2=72, Hco3=25, So2=96%

During sleep: PH = 7.35, PCO2=42, PO2 =60, Hco3=26, So2=94%

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Chest XR: Cardiomegaly, increased pulmonary vascularity bilateral (figure 1)

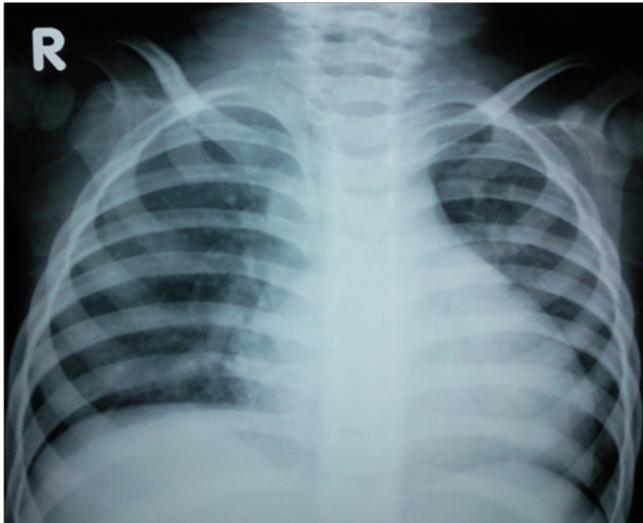


Figure 1: chest XR: Cardiomegaly , increased pulmonary vascularity bilateral

ECG: signs of dilated right atrium, dilated right ventricle (Figure 2)

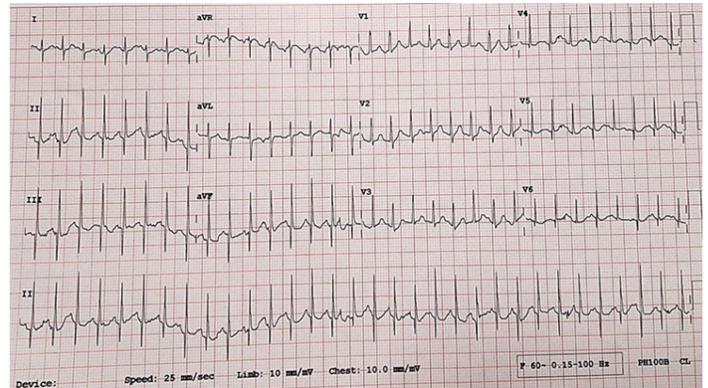


Figure 2: ECG: signs of dilated right heart.

ECHO cardiogram: dilated right atrium, dilated right ventricle, moderate to severe tricuspid regurgitation, RVSP=80mmHg, no structure abnormality was seen .

Conclusion

Pulmonary hypertension, normal cardiac anatomy, (figure3)

So we started to search for extra cardiac reason for the pulmonary hypertension, we prescribed Sildenafil but the parents didn't start it.

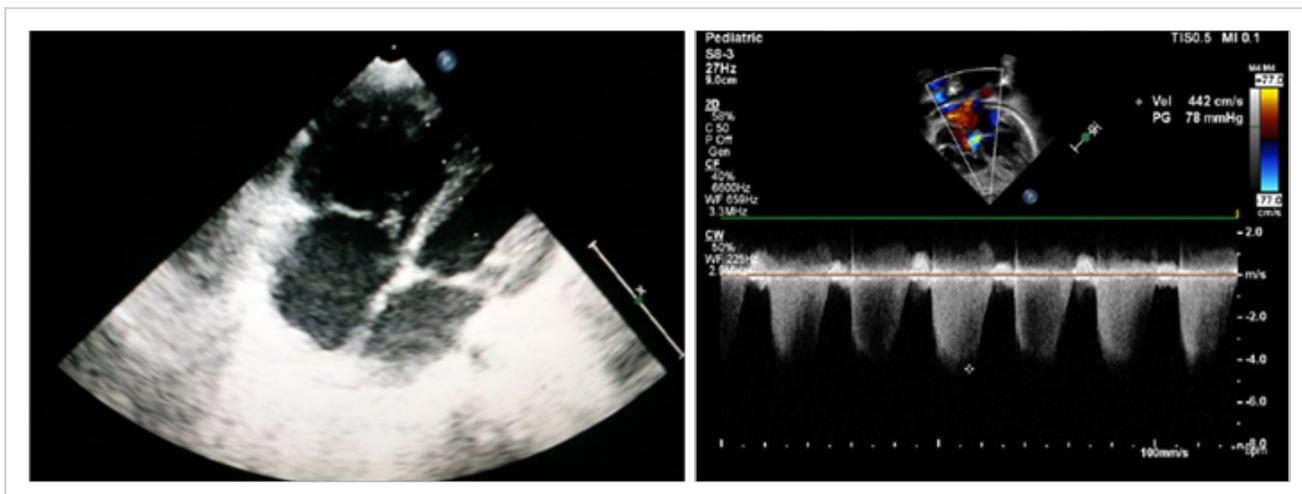


Figure 3: Echo: pulmonary hypertension.

Chest CT scan: cardiomegaly , dilated main pulmonary artery and its branches , lungs about normal (figure4)

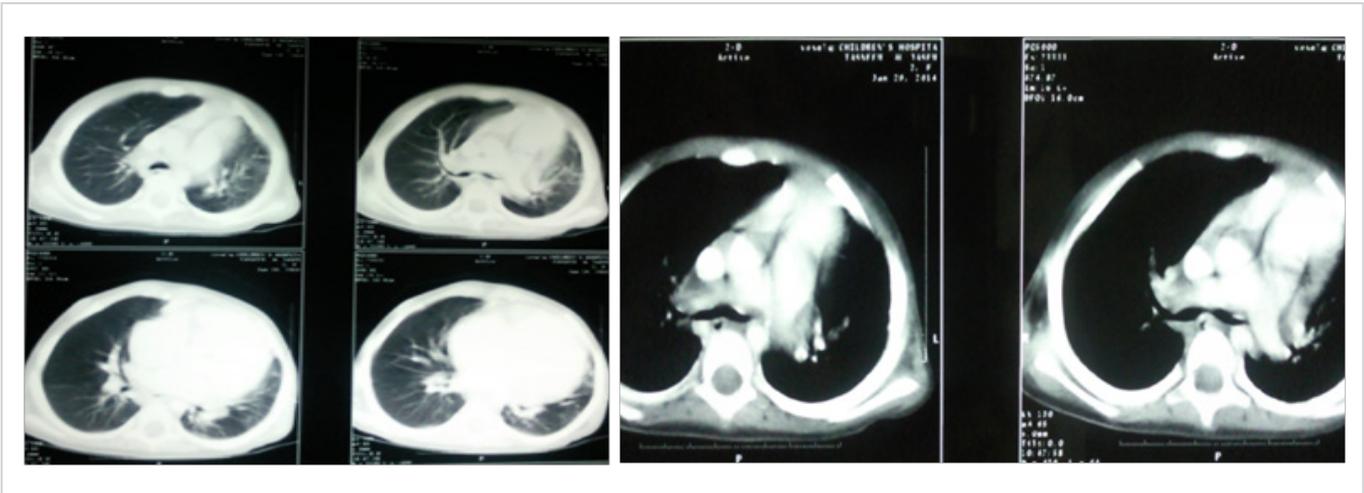


Figure 4: CT scan : cardiomegaly with dilated pulmonary artery

Lateral skull XR showed Large adenoids in the nasal pharynx and narrow air way (figure 5)

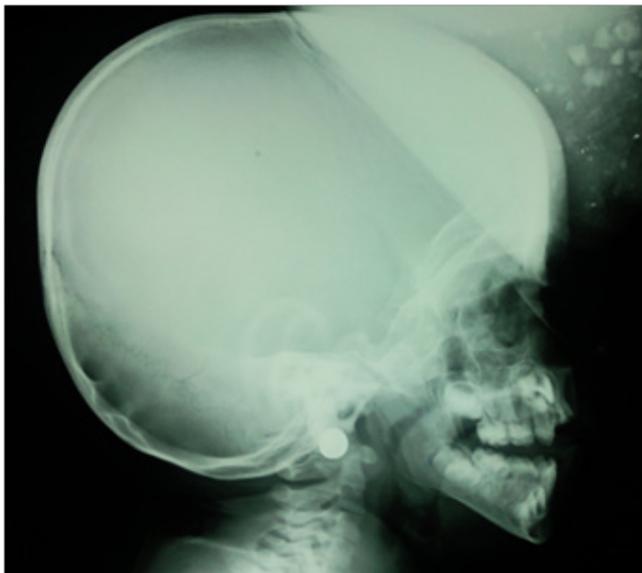


Figure 5 : Lateral skull XR

Sleep study done there were frequent episodes of apnea during sleep.

ENT consultation: kissing tonsils, hypertrophied adenoids, causes narrow air way, snoring and mouth breathing, needs surgical adenoidectomy and tonsillectomy

Then adenoidectomy and tonsillectomy surgery was done.

After surgery the girl became better, no more snoring or breathing difficulty and began to gain weight

Skull XR after surgery (wide upper air way)

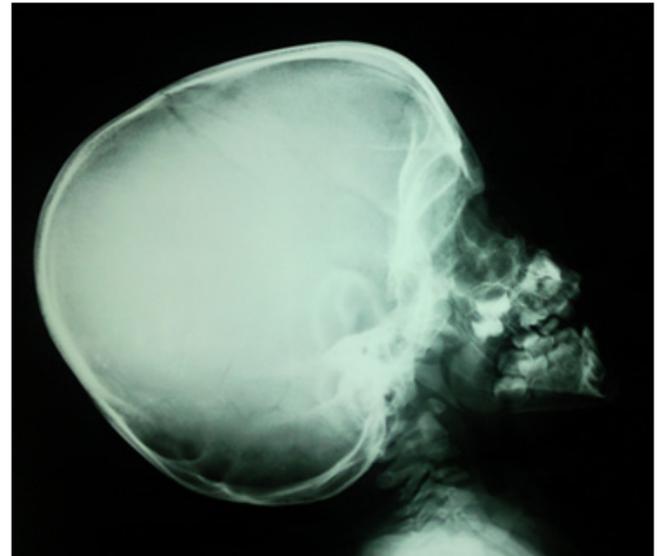


Figure 6: lateral skull XR : wide upper air way.

Chest XR after 3 month of surgery : mild cardiomegaly (figure 7)

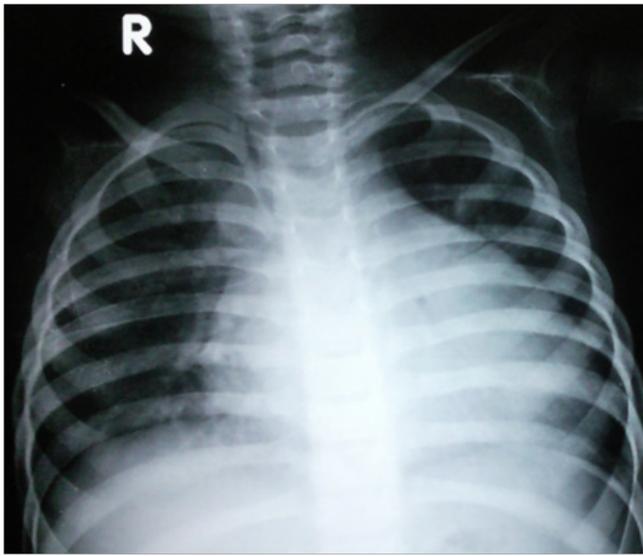


Figure 7 : chest XR : cardiomegaly.

She followed the cardiology clinic after 3 months

Echo done showed mild dilatation in the right heart , mild tricuspid regurgitation

(Right Ventricle Systolic Pressure = 38 mmHg) (figure 8)

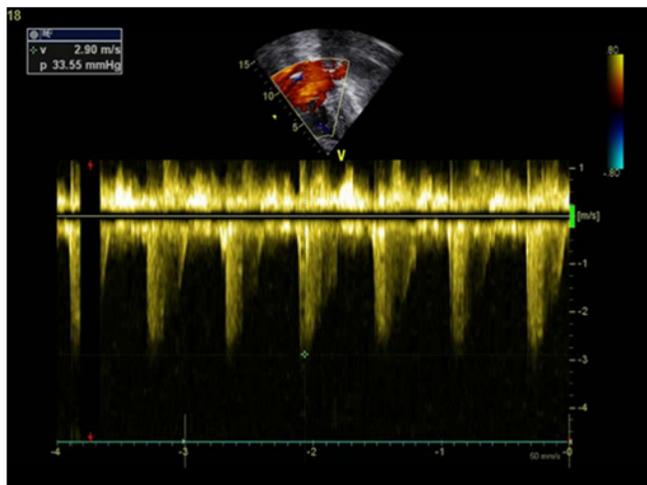


Figure 8 : Echo showed mild pulmonary hypertension .

After 6 months

No any complain or symptoms, gaining weight

The Echo showed trivial tricuspid regurgitation (RVSP =24mmHg), so the heart was about normal

Discussion

The symptoms caused by adenotonsillar hypertrophy can be nasal obstruction; mouth breathing, snoring, sleep apnea, irritability, and low development at school [5,6]. Complications of such obstruction cover from alterations on craniofacial development to pulmonary hypertension and Cor Pulmonale [1,3,4]. The current case already presented pulmonary involvement seen in the clinical history, assured by initial complaints.

Although adenotonsillar hypertrophy is very common, secondary pulmonary hypertension to it has been hardly diagnosed due to great amount of adenotonsillectomy approach performed in the past [4]. Doppler echocardiogram is highly useful when diagnosing pulmonary hypertension in infants with adenotonsillar hypertrophy, as it is considered a very safe, practical and non-invasive exam [5,6]. That exam was very important when diagnosing right heart involvement of the current patient. Chronic obstruction of the upper airways followed by pulmonary hypertension and Cor Pulmonale is an unquestionable sign of adenotonsillectomy, once it is already known that clinical and echocardiographic findings are fully reversible by surgery [3,6]. The current child was clinically benefited by adenotonsillectomy and few months later there was Doppler echocardiography in normal condition.

Final Comments

Adenotonsillar hypertrophy is a significant cause of pulmonary hypertension in children's.

Echocardiogram is the best exam to diagnose pulmonary hypertension for being safe and non-invasive. We suggest this exam becomes part of the investigations on children with adenotonsillar hypertrophy associated to sleep apnea [5,6].

Children with symptomatic adenotonsillar hypertrophy adenotonsillectomy required, to prevent pulmonary hypertension as complication of chronic obstruction by an enlargement of adenotonsil tissue [7,8].

References

1. Sie KC, Perkins JA, Clarke WR (1997) Acute right heart failure due to adenotonsillar hypertrophy. *Int J Pediatr Otorhinolaryngol* 41(1): 53-58.
2. Yates DW (1988) Adenotonsillar hypertrophy and cor pulmonale. *Br J Anaesth* 61(3): 355-359.
3. Blum RH, McGowan FX Jr (2004) Chronic upper airway obstruction and cardiac dysfunction: anatomy, pathophysiology and anesthetic implications. *Paediatric Anaesth* 14(1): 75-83.
4. sebusiani BB, Pignatari S, Armínio G, Mekhtarian Neto I, Stamm AEC (2003) Hipertensão pulmonar em pacientes com hipertrofia adenoamigdaliana. *Rev Bras Otorrinolaringol* 69: 819-823.

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5. Miman MC, Kirazli T, Ozyurek R (2000) Doppler echocardiography in adenotonsillar hypertrophy. *Int J Pediatr Otorhinolaryngol* 54(1): 21-26.
 6. Jacobs IN, Teague WG, Bland JW (1997) Pulmonary vascular complications of chronic airway obstruction in children. *Arch Otolaryngol Head and Neck Surg* 123(7): 700-704.
 7. Takashi N, Shirahata M, Yonezawa T, Honda Y (1985) Comparison of changes in the hypoglossal and phrenic nerve activity in response to increasing depth of anesthesia in cats. *Anesthesiology* 63: 404-409.
 8. Marcus CL, Loughlin GM (1996) Obstructive sleep apnea in children. *Semin Pediatr Neurol* 3: 23-28.