Case Report

Acute asphyxic asthma: beware of a potential killer

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Keywords

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Abstract

Acute asphyxic asthma is a rare but severe presentation of an acute asthma exacerbation, not well known in children. We report the case of an eleven-year old girl, who presented with acute dyspnea at night, quickly evolving to cardiorespiratory arrest, for which cardiopulmonary resuscitation was required. An acute asphyxic asthma attack was diagnosed as the cause of the cardiopulmonary arrest. Acute asphyxic asthma has been previously described in adults, but studies in children are scarce. Further studies are needed to identify children at risk for acute asphyxic asthma and guide preventive measures. Awareness of this entity is crucial. If promptly recognized and treated, a near-fatal attack usually resolves rapidly and completely.

Introduction

The prevalence of asthma is high, with five to ten percent of children estimated to be affected. Only few children present with life-threatening episodes, but these are associated with potential mortality, morbidity and a high cost of treatment (1). Severe attacks of asthma may have a sudden or slow onset. Acute asphyxic asthma (AAA) is characterized by a sudden onset that may rapidly progress to a near-arrest state. This is a rare entity previously described in adult patients (2-3). There is evidence that AAA also occurs in children (1).

With this case report, we want to raise awareness of this rare but severe presentation of an acute asthma exacerbation in children. In addition, we want to describe the specific subgroup of patients who present with this type of exacerbation and highlight the differences in therapy and outcome in comparison to a classic asthma exacerbation.

Case Report

We report the case of an 11-year old girl, with previously known allergic rhinitis and asthma (total IgE 5814 kU/l, with sensitization for dog, cat, dust mite, tree pollen, grass pollen and weed), partly controlled with medium dose inhaled corticosteroids (ICS) and long acting beta2 agonist (LABA), but with moderate therapy compliance. She was exposed to second-hand tobacco smoke daily. No other environmental risk factors were identified. She had regular complaints of cough and was treated for asthma since the age of five, but without any need for oral corticosteroids or hospital admissions in the past. On January 4, 2019, 1 month before admission, therapy was increased to high dose ICS plus LABA and a leukotriene receptor antagonist was started because of daytime symptoms. Spirometry at that time showed a FEV1 of 83%, with an obstructive curve and significant reversibility after administration of SABA (short acting beta2 agonist) (figure 1).

On February 9, 2019, she was admitted to the pediatric intensive care unit (PICU) after an out of hospital cardiorespiratory arrest. She had developed acute dyspnea at night, without previous complaints during the day or evening. Her mother tried to administer salbutamol via inhaler, which was unsuccessful due to severe dyspnea. An ambulance was summoned. She subsequently rapidly lost consciousness and developed central cyanosis. Upon arrival of the ambulance, she was unresponsive, with apnea and extreme bradycardia (heart rate 20 bpm). CPR was started and she was intubated and managed according to standard pediatric advanced life support guidelines, with return of spontaneous circulation after administration of intravenous adrenaline. Intravenous corticosteroids were administered. She was transported to the PICU in stable condition where intensive treatment with inhaled bronchodilators (salbutamol, ipratropium bromide) was started. Investigations showed severe combined respiratory and lactic acidosis (pH 7.068, pCO2 72 mmHg, lactate 51 mg/dl) and a normal chest x-ray (figure 2). Her respiratory condition improved rapidly, with extubation after merely 9 hours of invasive mechanical ventilation (IMV). There were no signs of respiratory distress after extubation. Initial cardiological screening showed a prolonged QT(c) interval, which normalized spontaneously. Investigations for anaphylaxis showed a peanut sensitization but there was no history of recent ingestion of peanuts, and the attack happened at night. Unfortunately, the level of tryptase at admission was not performed. An AAA attack was diagnosed as the cause of the cardiopulmonary arrest. Thirty six hours after admission, spirometry was within normal range (figure 3) and she was discharged. Further investigations showed positive specific IgE for peanut h3, h8 and ara h2, but provocation test one month later was negative.

Discussion

When studying reports of life threatening asthma or near-fatal asthma, there is evidence that severe asthma can present with acute onset and rapidly progressive respiratory failure (1,2). This phenomenon is not rare, as 15 to 26 percent of asthma deaths in adults are attributed to sudden-onset asthma attacks (3). In near-fatal asthma in children, 17 percent of cases presented with sudden collapse without previous deterioration, while in an Australian asthma mortality study 79% of children presented with sudden collapse (2,4).

AAA or sudden-onset asthma attacks have been defined as respiratory arrest or failure within three hours after the onset of the attack (3). This type of asthma exacerbation is more frequently observed in young male adults and is characterized by a brief duration of symptoms (<3h), few identifiable triggers and a rapid progression to respiratory failure. Sudden onset asthma attacks are characterized by severe mixed respiratory-metabolic acidosis, reflecting circulatory compromise besides acute hypercapnic respiratory failure. There is a higher incidence of respiratory arrest, and silent chest upon admission (1,3,5).

Studies in a pediatric population are scarce, but there is evidence suggesting that AAA in children shares characteristics with adults presenting with this type of asthma attack (1). It is unclear from the current literature, whether usual risk factors for asthma exacerbation like poor adherence and tobacco smoke exposure, as were present in the reported case, increase the risk for an AAA attack in children.

The exact pathophysiological differences between a sudden and a slow-onset asthma attack are not well-described. However, immunohistological differences have been described. Post mortem airway mucosa specimens from fatal asthma patients showed eosinophilic predominance in slow-onset asthma, and predominantly neutrophils in sudden-onset asthma (3). Other studies suggest bronchospasm as a predominant factor of deterioration (5,6). It is notable that in contrast with slow-onset attack, an upper respiratory tract infection or viral trigger is rarely found in AAA (6). It has been suggested that inhalation of large quantities of allergens by patients with high levels of specific IgE to this allergen may cause a sudden-onset asthma attack (3). However, anaphylaxis and AAA are distinct entities with differing treatments so a correct diagnosis is essential (7).

Despite the severity of the presentation, sudden-onset asthma attacks usually resolve rapidly with adequate treatment, both in adults and in children. The principles of the treatment do not differ between AAA and other asthma exacerbations, with bronchodilatory agents and corticosteroids being the first line of therapy. However, due to the rapid progression to respiratory failure, there is a higher need for IMV. Duration of IMV is shorter than in slow-onset attacks, with more rapid improvement of gas exchange (1,3,5). However, patients with a history of AAA are more at risk for a fatal asthma-related outcome (3). Therefore, we advise a strict follow-up for these patients, and would advise a careful approach when considering a step-down in therapy. To further guide preventive measures and treatment choices for pediatric AAA, further studies are warranted.

Conclusion

In conclusion, AAA is a distinct form of life-threatening asthma, that can occur in the pediatric population. In adults, it is characterized by a sudden onset of symptoms, few identifiable triggers and rapid progression to end-stage respiratory failure (1,3,5). Pediatric AAA shares certain characteristics with adult AAA but in this case poor adherence and smoke exposure were possible triggers. If promptly diagnosed and treated, a near-fatal attack usually resolves rapidly and completely, with most often early hospital discharge. Further studies are required to better define asthma patients at risk for AAA to guide both preventive measures and treatment choices.

Conflict of interest statement

The authors of this case report declare that they have no conflict of interest. They do not have any affiliations with or involvement in any organization or entity with any financial or non-financial interest in the subject matter of materials discussed in this case report.

Figure 2 : *Chest X-ray after intubation in the field. In the upper right lobe, we see an atelectasis, due to deep tube positioning (clinically evident, already corrected at the moment of the X-ray)*

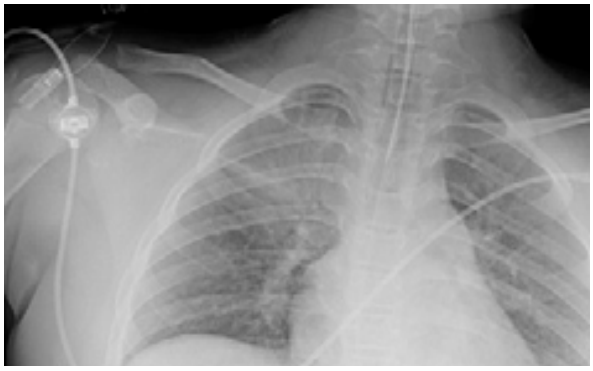

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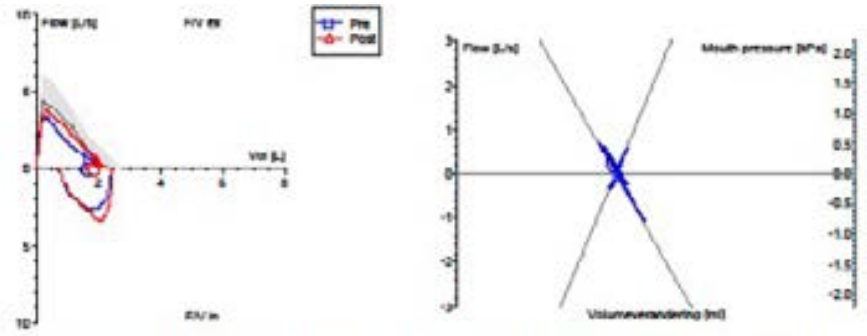
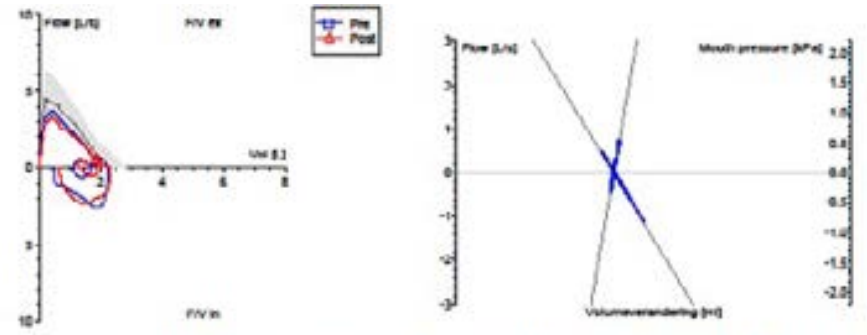
Figure 1 : *Spirometry one month before admission. Note the obstructive curve before administration of SABA and the 14.5% reversibility in FEV1 after administration of SABA.*



		Voorsp	Pre	%	Post	%	% Chg	z-score
R tot	kPa/(L/s)	0.35	0.71	202.2				3.97
SR tot	kPa²s		1.89					
ITGV	L	1.40	2.40	164.3				4.57
RV Pleth	L	0.77	1.86	241.8				6.19
TLC Pleth	L	2.99	4.22	141.9				4.63
ERV	L	0.72	0.54	74.8				-1.74
RV%TLC	%	25.4	44.0	173.5				4.77
ITGV % TLC	%	47.20	56.78	120.3				2.38
VC	L	2.21	2.36	107.1				0.67
VC IN	L	2.21	1.80	81.7	1.86	84.3	3.2	-2.10
FVC	L	2.23	2.40	107.6	2.42	108.8	1.1	0.64
FEV 1	L	1.86	1.60	83.1	1.85	95.1	14.5	-1.43
FEV 1 % VC MAX	%	89.37	68.69	76.9	77.76	87.0	13.2	-2.64
PEF	L/s	4.36	3.60	82.4	3.79	86.8	5.3	-1.25
MEF 25	L/s	1.23	0.47	37.9	0.69	56.0	47.5	-2.53
MEF 50	L/s	2.77	1.32	47.7	2.05	74.1	55.1	-3.84
MEF 75	L/s	3.92	2.60	66.4	3.21	81.9	23.5	-2.43
MFEF 75/25	L/s	2.66	1.11	41.7	1.70	64.0	53.4	-2.90
PIF	L/s		2.66		3.40		27.8	
FIV 1	L		1.79		1.73		-3.3	

Figure 3 : *Spirometry 36 hours after admission. Note the absence of obstruction and reversibility.*



		Voorsp	Pre	%	Post	%	% Chg	z-score
R tot	kPa/(L/s)	0.34	0.34	101.5				0.00
SR tot	kPa²s		0.80					
ITGV	L	1.52	2.19	144.6				3.17
RV Pleth	L	0.79	1.53	192.1				4.02
TLC Pleth	L	3.09	3.83	124.1				2.67
ERV	L	0.75	0.67	88.9				-0.77
RV%TLC	%	25.2	30.8	157.8				3.73
ITGV % TLC	%	47.32	57.18	120.8				2.45
VC	L	2.29	2.31	100.8				0.05
VC IN	L	2.29	1.79	78.0	1.69	73.8	-5.3	-2.53
FVC	L	2.31	2.24	96.9	2.30	99.5	2.6	-0.26
FEV 1	L	2.05	1.84	89.8	1.81	88.3	-1.7	-0.87
FEV 1 % VC MAX	%	89.29	82.37	92.3	78.01	88.4	4.2	-1.13
PEF	L/s	4.51	3.69	81.7	3.21	71.2	-12.9	-1.30
MEF 25	L/s	1.27	0.81	63.4	0.71	56.0	-11.6	-1.30
MEF 50	L/s	2.85	2.24	78.3	2.02	70.9	-9.5	-1.50
MEF 75	L/s	4.04	3.42	84.5	2.92	72.2	-14.6	-1.12
MFEF 75/25	L/s	2.73	1.90	69.9	1.70	62.3	-10.7	-1.44
PIF	L/s		2.62		2.33		-11.2	
FIV 1	L		1.75		1.67		-4.5	