



UNIVERSITE CADI AYYAD
FACULTE DE MEDECINE ET DE PHARMACIE
MARRAKECH

Année 2015

Thèse N° 73

Management of childhood asthma in the emergency department

THESE

PRESENTEE ET SOUTENUE PUBLIQUEMENT LE 26/05/2015

PAR

Mme. **Chaimaa KOUDRI**

Née Le 15 Juillet 1987 à Casablanca

POUR L'OBTENTION DU DOCTORAT EN MEDECINE

MOTS-CLES:

Asthma – Children – Emergency – Management

JURY

MR. S. YOUNOUS Professeur d'Anesthésie-réanimation	PRESIDENT
MR. M. BOURROUS Professeur Agrégé de Pédiatrie	RAPPORTEUR
Mme. G. DRAISS Professeur Agrégée de Pédiatrie	} JUGES
Mme. L. AMRO Professeur Agrégée de Pneumo-phtisiologie	

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

"ربِّهِ أَوْزَعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ

الَّتِي أَنْعَمْتَ عَلَيَّ وَعَلَى وَالِدِي

وَأَنْ أَعْمَلَ حَالًا تَرْضَاهُ وَأُطِيعَ

لِي فِي خُرَيْتِي إِنْ بَدَأَ بِكَ إِلَيْكَ

وَأِنْ بَدَأَ مِنَ الْمُسْلِمِينَ"

صَدَقَ اللَّهُ الْعَظِيمَ.



Serment d'hypocrate

Au moment d'être admis à devenir membre de la profession médicale, je m'engage solennellement à consacrer ma vie au service de l'humanité.

Je traiterai mes maîtres avec le respect et la reconnaissance qui leur sont dus.

Je pratiquerai ma profession avec conscience et dignité. La santé de mes malades sera mon premier but.

Je ne trahirai pas les secrets qui me seront confiés.

Je maintiendrai par tous les moyens en mon pouvoir l'honneur et les nobles traditions de la profession médicale.

Les médecins seront mes frères.

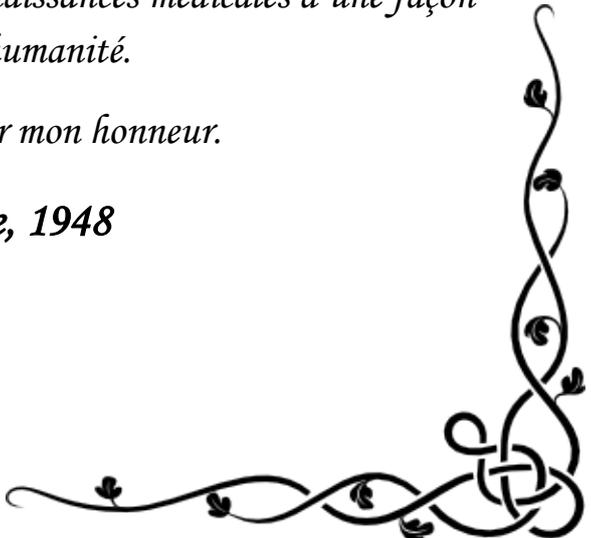
Aucune considération de religion, de nationalité, de race, aucune considération politique et sociale, ne s'interposera entre mon devoir et mon patient.

Je maintiendrai strictement le respect de la vie humaine dès sa conception.

Même sous la menace, je n'userai pas mes connaissances médicales d'une façon contraire aux lois de l'humanité.

Je m'y engage librement et sur mon honneur.

Déclaration Genève, 1948





*LIST OF
PROFESSORS*

UNIVERSITE CADI AYYAD

FACULTE DE MEDECINE ET DE PHARMACIE MARRAKECH

Doyen Honoraire: Pr Badie Azzaman MEHADJI

ADMINISTRATION

Doyen : Pr Mohammed BOUSKRAOUI

Vice doyen à la recherche et la coopération : Pr.Ag. Mohamed AMINE

Secrétaire Générale : Mr Azzeddine EL HOUDAIGUI

Professeurs de l'enseignement supérieur

Nom et Prénom	Spécialité	Nom et Prénom	Spécialité
ABOULFALAH Abderrahim	Gynécologie- obstétrique	FINECH Benasser	Chirurgie – générale
AIT BENALI Said	Neurochirurgie	GHANNANE Houssine	Neurochirurgie
AIT-SAB Imane	Pédiatrie	KISSANI Najib	Neurologie
AKHDARI Nadia	Dermatologie	KRATI Khadija	Gastro- entérologie
AMAL Said	Dermatologie	LMEJJATI Mohamed	Neurochirurgie
ASMOUKI Hamid	Gynécologie- obstétrique B	LOUZI Abdelouahed	Chirurgie – générale
ASRI Fatima	Psychiatrie	MAHMAL Lahoucine	Hématologie - clinique
BENELKHAÏAT BENOMAR Ridouan	Chirurgie - générale	MANSOURI Nadia	Stomatologie et chiru maxillo faciale
BOUMZEBRA Drissi	Chirurgie Cardio- Vasculaire	MOUDOUNI Said Mohammed	Urologie
BOUSKRAOUI Mohammed	Pédiatrie A	MOUTAOUAKIL Abdeljalil	Ophtalmologie
CHABAA Laila	Biochimie	NAJEB Youssef	Traumato- orthopédie
CHELLAK Saliha (Militaire)	Biochimie- chimie	OULAD SAIAD Mohamed	Chirurgie pédiatrique
CHOULLI Mohamed Khaled	Neuro pharmacologie	RAJI Abdelaziz	Oto-rhino-laryngologie

DAHAMI Zakaria	Urologie	SAIDI Halim	Traumato- orthopédie
EL FEZZAZI Redouane	Chirurgie pédiatrique	SAMKAOUI Mohamed Abdenasser	Anesthésie- réanimation
EL HATTAOUI Mustapha	Cardiologie	SARF Ismail	Urologie
ELFIKRI Abdelghani (Militaire)	Radiologie	SBIHI Mohamed	Pédiatrie B
ESSAADOUNI Lamiaa	Médecine interne	SOUMMANI Abderraouf	Gynécologie- obstétrique A/B
ETTALBI Saloua	Chirurgie réparatrice et plastique	YOUNOUS Said	Anesthésie- réanimation
FIKRY Tarik	Traumato- orthopédie A		

Professeurs Agrégés

Nom et Prénom	Spécialité	Nom et Prénom	Spécialité
ABKARI Imad	Traumato- orthopédie B	EL OMRANI Abdelhamid	Radiothérapie
ABOU EL HASSAN Taoufik	Anesthésie- réanimation	FADILI Wafaa	Néphrologie
ABOUCHADI Abdeljalil (Militaire)	Stomatologie et chir maxillo faciale	FAKHIR Bouchra	Gynécologie- obstétrique A
ABOUSSAIR Nisrine	Génétique	FOURAIJI Karima	Chirurgie pédiatrique B
ADALI Imane	Psychiatrie	HACHIMI Abdelhamid	Réanimation médicale
ADERDOUR Lahcen	Oto- rhino- laryngologie	HAJJI Ibtissam	Ophtalmologie
ADMOU Brahim	Immunologie	HAOUACH Khalil	Hématologie biologique
AGHOUTANE El Mouhtadi	Chirurgie pédiatrique A	HAROU Karam	Gynécologie- obstétrique B
AIT AMEUR Mustapha (Militaire)	Hématologie Biologique	HOCAR Ouafa	Dermatologie
AIT BENKADDOUR Yassir	Gynécologie- obstétrique A	JALAL Hicham	Radiologie
AIT ESSI Fouad	Traumato- orthopédie B	KAMILI El Ouafi El Aouni	Chirurgie pédiatrique B
ALAOUI Mustapha (Militaire)	Chirurgie- vasculaire périphérique	KHALLOUKI Mohammed	Anesthésie- réanimation
AMINE Mohamed	Epidémiologie- clinique	KHOUCHANI Mouna	Radiothérapie
AMRO Lamyae	Pneumo- phtisiologie	KOULALI IDRISSEI Khalid (Militaire)	Traumato- orthopédie
ANIBA Khalid	Neurochirurgie	KRIET Mohamed (Militaire)	Ophtalmologie
ARSALANE Lamiae (Militaire)	Microbiologie - Virologie	LAGHMARI Mehdi	Neurochirurgie

BAHA ALI Tarik	Ophthalmologie	LAKMICH I Mohamed Amine	Urologie
BASRAOUI Dounia	Radiologie	LAOUAD Inass	Néphrologie
BASSIR Ahlam	Gynécologie-obstétrique A	LOUHAB Nisrine	Neurologie
BELKHOU Ahlam	Rhumatologie	MADHAR Si Mohamed	Traumato- orthopédie A
BEN DRISS Laila (Militaire)	Cardiologie	MANOUDI Fatiha	Psychiatrie
BENCHAMKHA Yassine	Chirurgie réparatrice et plastique	MAOULAININE Fadl mrah rahou	Pédiatrie
BENHIMA Mohamed Amine	Traumatologie - orthopédie B	MATRANE Aboubakr	Médecine nucléaire
BENJILALI Laila	Médecine interne	MEJDANE Abdelhadi (Militaire)	Chirurgie Générale
BENZAROUEL Dounia	Cardiologie	MOUAFFAK Youssef	Anesthésie - réanimation
BOUCHENTOUF Rachid (Militaire)	Pneumo- phtisiologie	MOUFID Kamal(Militaire)	Urologie
BOUKHANNI Lahcen	Gynécologie-obstétrique B	MSOUGGAR Yassine	Chirurgie thoracique
BOUKHIRA Abderrahman	Toxicologie	NARJISS Youssef	Chirurgie générale
BOURRAHOUE Aicha	Pédiatrie B	NEJMI Hicham	Anesthésie- réanimation
BOURROUS Monir	Pédiatrie A	NOURI Hassan	Oto rhino laryngologie
BSISS Mohamed Aziz	Biophysique	OUALI IDRISSE Mariem	Radiologie
CHAFIK Rachid	Traumato- orthopédie A	QACIF Hassan (Militaire)	Médecine interne
CHAFIK Aziz (Militaire)	Chirurgie thoracique	QAMOUSS Youssef (Militaire)	Anesthésie- réanimation
CHERIF IDRISSE EL GANOUNI Najat	Radiologie	RABBANI Khalid	Chirurgie générale
DRAISS Ghizlane	Pédiatrie	RADA Nouredine	Pédiatrie A
EL BOUCHTI Imane	Rhumatologie	RAIS Hanane	Anatomie pathologique
EL HAOURY Hanane	Traumato- orthopédie A	ROCHDI Youssef	Oto-rhino- laryngologie
EL MGHARI TABIB Ghizlane	Endocrinologie et maladies métaboliques	SAMLANI Zouhour	Gastro- entérologie
EL ADIB Ahmed Rhassane	Anesthésie- réanimation	SORAA Nabila	Microbiologie - virologie
EL ANSARI Nawal	Endocrinologie et maladies métaboliques	TASSI Noura	Maladies infectieuses
EL BARNI Rachid (Militaire)	Chirurgie- générale	TAZI Mohamed Illias	Hématologie- clinique

EL BOUIHI Mohamed	Stomatologie et chir maxillo faciale	ZAHLANE Kawtar	Microbiologie - virologie
EL HOUDZI Jamila	Pédiatrie B	ZAHLANE Mouna	Médecine interne
EL IDRISSE SLITINE Nadia	Pédiatrie	ZAOUI Sanaa	Pharmacologie
EL KARIMI Saloua	Cardiologie	ZIADI Amra	Anesthésie - réanimation
EL KHAYARI Mina	Réanimation médicale		

Professeurs Assistants

Nom et Prénom	Spécialité	Nom et Prénom	Spécialité
ABIR Badreddine (Militaire)	Stomatologie et Chirurgie maxillo faciale	FAKHRI Anass	Histologie- embryologie cytogénétique
ADALI Nawal	Neurologie	FADIL Naima	Chimie de Coordination Bioorganique
ADARMOUCH Latifa	Médecine Communautaire (médecine préventive, santé publique et hygiène)	GHAZI Mirieme (Militaire)	Rhumatologie
AISSAOUI Younes (Militaire)	Anesthésie - réanimation	HAZMIRI Fatima Ezzahra	Histologie – Embryologie - Cytogénétique
AIT BATAHAR Salma	Pneumo- phtisiologie	IHBIBANE fatima	Maladies Infectieuses
ALJ Soumaya	Radiologie	KADDOURI Said (Militaire)	Médecine interne
ARABI Hafid (Militaire)	Médecine physique et réadaptation fonctionnelle	LAFFINTI Mahmoud Amine (Militaire)	Psychiatrie
ATMANE El Mehdi (Militaire)	Radiologie	LAHKIM Mohammed (Militaire)	Chirurgie générale
BAIZRI Hicham (Militaire)	Endocrinologie et maladies métaboliques	LAKOUICHMI Mohammed (Militaire)	Stomatologie et Chirurgie maxillo faciale
BELBACHIR Anass	Anatomie- pathologique	LOQMAN Souad	Microbiologie et toxicologie environnementale
BELBARAKA Rhizlane	Oncologie médicale	MARGAD Omar (Militaire)	Traumatologie - orthopédie
BELHADJ Ayoub (Militaire)	Anesthésie - Réanimation	MLIHA TOUATI Mohammed (Militaire)	Oto-Rhino - Laryngologie
BENHADDOU Rajaa	Ophtalmologie	MOUHSINE Abdelilah (Militaire)	Radiologie
BENLAI Abdeslam (Militaire)	Psychiatrie	NADOUR Karim(Militaire)	Oto-Rhino - Laryngologie
CHRAA Mohamed	Physiologie	OUBAHA Sofia	Physiologie
DAROUASSI Youssef (Militaire)	Oto-Rhino - Laryngologie	OUERIAGLI NABIH Fadoua (Militaire)	Psychiatrie
DIFFAA Azeddine	Gastro- entérologie	SAJIAI Hafsa	Pneumo- phtisiologie

EL AMRANI Moulay Driss	Anatomie	SALAMA Tarik	Chirurgie pédiatrique
EL HAOUATI Rachid	Chiru Cardio vasculaire	SERGHINI Issam (Militaire)	Anesthésie - Réanimation
EL HARRECH Youness (Militaire)	Urologie	SERHANE Hind	Pneumo- phtisiologie
EL KAMOUNI Youssef (Militaire)	Microbiologie Virologie	TOURABI Khalid (Militaire)	Chirurgie réparatrice et plastique
EL KHADER Ahmed (Militaire)	Chirurgie générale	ZARROUKI Youssef	Anesthésie - Réanimation
EL MEZOUARI EI Moustafa (Militaire)	Parasitologie Mycologie	ZIDANE Moulay Abdelfettah (Militaire)	Chirurgie Thoracique



DEDICATIONS

*All letters cannot find the right words...
All words cannot express the appreciation,
Love, Respect, recognition...
Also, it is simply that*



🌸 I dedicate this thesis ... ✍️

To my dear mother Lhajja Amina

Affable, honorable, amiable you represent for me the symbol of ultimate goodness, tenderness source and example of dedication that has not stopped encouraging me and praying for me. No dedication is enough to express what you deserve for all the sacrifices you have stopped giving me since my birth, during childhood and even in adulthood. I dedicate this work to you as a token of my deep love. May Allah, the Almighty, you preserve and grant you health, long life and happiness.

To my dear father Lhaj Mustapha

This modest work, which is primarily yours, is that the consecration of your great efforts and your immense sacrifices. Without you I cannot get where I am. I hope to stay still worthy of your esteem. Your kindness and generosity are endless. Your prayers have been for me a great moral support all through my studies. May Allah Almighty protect you from harm, fill you with health, happiness and grant you a long and happy life so I can make you a minimum of what I owe you.

To my dear husband Doctor Abdelilah

When I met you, I found the man of my life, my soul mate and the light of my way. My life by your side is full of surprises. Your sacrifices, your support, your unequalled kindness, your deep commitment have allowed me to succeed my studies. Without your help, your advice and your encouragement this work could not exist. May God meet our paths for a serene common long and that this work is testimony of my gratitude and my sincere and faithful love.

To my dear little girl Chaïd

My little darling, the icing on the cake that has given meaning to my life and illuminates by all its splendor my life, I thank you for making our sweet lives, our best days and our lives as filled. No cannot express my pride and my love for you. May God Almighty protect you and gives you a life full of happiness and success.

To my dear brothers, Anas, Hamza and Saad

In recognition of the commitment, love and affection I have for you. You have supported me and filled all through my career. Hoping this work to be testimony of my deepest feelings and gratitude. Thank you again for your encouragement that have never been lacking. May God provide you happiness, health and prosperity.

To my dear stepfather Lhaj Abdellatif and my stepmother Lhajja Keltoum

*You have welcomed me with open arms in your family.
In recognition of the commitment, love and affection I have for you.
I dedicate this work with you my best wishes for happiness, success, health and long life.*

IN MEMORY OF MY GRANDPARENTS

I wish you to be present that day to share with me the best moments of my life, but alas ... God wanted otherwise.

*Hoping this work is a prayer for the repose of your souls.
May Allah the almighty, the merciful, reward yourself and your souls rest in peace.*

To all my uncles and aunts

Please accept the expression of my deep gratitude for your support, encouragement, and affection.

I hope you find in the dedication of this work, the evidence of my sincere feelings and my wishes for health and happiness.

To all my family members, big and small

Please find in this modest work the expression of my affection.

To all my friends and colleagues,

*To all the times we had together, all our memories!
I wish you long life full of happiness and prosperity.
I dedicate this work to you as a token of my gratitude and my respect.*

To all my teachers of primary, secondary, and faculty of medicine of Marrakech.

***To all who are dear to me and I inadvertently failed to mention.
To all who have contributed in any way to the develop.***



ACKNOWLEDGMENTS

***To our Master and thesis president, Professor Said YOUNOUS,
Professor of Anesthesia and reanimation.***

*Thank you for the honor you have done us by accepting to chair this jury.
Your seriousness, your competence and sense of duty have greatly impressed us.
Please find here the expression of our respectful consideration and deep admiration for all of
your scientific and human qualities.
This work is an opportunity for us to express our deep gratitude.*

***To our Master and thesis judge, Professor Ghizlane DRAISS,
Professor of Pediatrics.***

*Thank you for your valuable participation in the development of this work,
Allow us to express our admiration for your human and professional qualities.
Please find here the expression of our esteem and consideration.*

***To our Master and thesis judge, Professor Lamya AMRO,
Professor of Pneumophysiology***

*You have honored us with great sympathy to accept to sit among our thesis jury.
Please find here the expression of our respect and our acknowledgments.*

***To our Master and thesis supervisor, Professor Mounir
BOURROUS, Professor of Pediatrics.***

*You have entrusted us this rich work of interest and guided us every step of its implementation.
You always reserved for us the best reception, despite your professional obligations.
Your tireless encouragement, your kindness, your kindness deserve all of admiration.
We take this opportunity to express our deep gratitude while witnessing you our respect.*



ABBREVIATIONS

List of Abbreviations

AD	: Atopic Dermatitis
BHR	: bronchial hyperreactivity
BMI	: body mass index
CBC	: complete blood count
CRP	: C-reactive protein
ED	: emergency department
EIA	: exercise induced asthma
FEV	: Forced Expiratory Volume
GERD	: Gastro-oesophageal reflux
GINA	: the Global Initiative for Asthma
GOAL	: Gaining Optimal Asthma Control
GST	: Glutathione S-transferase
HDM	: house dust mite
HRV	: human rhinovirus
ICU	: intensive care unit
ICS	: inhaled corticosteroids
IM	: intramuscular
ISAAC	: International study of asthma and allergy in childhood
IV	: intravenous
LABA	: long-acting beta-2 agonist
MDI	: metered-dose inhaler
NAC	: National Asthma Council
NAEPP	: National Asthma and Education and Prevention Program
NHLBI	: National Heart, Lung, and Blood Institute
OCS	: oral corticosteroids
PEFR	: The peak expiratory flow rate
PSA	: Pediatric status asthmaticus
RCT	: randomized controlled trials
RSV	: respiratory syncytial virus
SABA	: short-acting beta-2 agonists
SHS	: second hand tobacco smoke
Th	: T-helper



PLAN

INTRODUCTION	1
PATIENTS AND METHODS	3
I. Patients:	4
1. Inclusion criteria:	4
2. Exclusion criteria:	4
II. Methods:	4
1. Type of work:	4
2. Collecting data:	5
3. Statistical analysis:	5
RESULTS	6
I. Anamnestic Profile:	7
1. Age :	7
2. Sex	7
3. Socioeconomic level:	7
4. Type of residence	8
5. Personal history:	9
6. Family history of asthma and / or atopy:	10
II. Precipitating factors:	10
III. Natural history of disease	11
1. Reason for consultation	11
2. functional signs:	11
3. Duration of the crisis	12
4. Seasonal predominance	13
IV. Clinical examination:	13
V. Paraclinical investigation	14
VI. Therapeutic profile	15
VII. Classification	15
VIII. Follow up and Evolution	16
DISCUSSION	18
I. Definition of asthma	19
II. Pathophysiology of asthma:	19
1. Inflammation and Airway Remodeling	19
2. Bronchoconstriction and airway hyperresponsiveness.	21
III. EPIDEMIOLOGY:	21
1. Prevalence	21
2. Mortality	22
3. Morbidity	22
IV. Risk factors:	23
1. Age of onset:	25
2. Atopy	25
3. Genetic predisposition:	26

4. Infection–Related Asthma.....	26
5. Allergens:.....	27
6. Gastro–esophageal reflux:.....	28
V. Environmental factors:.....	29
1. Passive smoking:.....	29
2. Socio–economic factors:.....	29
VI. Associated Allergic reactions:.....	30
VII. Clinical diagnosis:.....	31
1. Paroxysmal asthma attack.....	31
2. Status asthmaticus or acute severe asthma.....	32
3. Exercise induced asthma:.....	33
VIII. Classification of asthma.....	33
IX. Complications:.....	34
1. Infection:.....	34
2. Mechanical complications: Pneumo–mediastinum, pneumothorax and subcutaneous emphysema:.....	35
X. Differential diagnoses:.....	36
XI. Para clinical investigations:.....	37
1. Chest X–ray :.....	37
2. Skin tests:.....	38
3. Respiratory function tests:.....	38
4. Peak Expiratory Flow Rate:.....	39
5. Blood gases:.....	40
XII. Therapeutic management:.....	40
1. Treatment of the usual acute asthma attack:.....	40
2. Treatment of status asthmaticus:.....	46
3. Proposed treatment to return home:.....	48
4. Maintenance treatment of asthma:.....	49
XIII. Control and follow of asthma:.....	50
XIV. Education:.....	53
1. Self–monitoring and Periodic Assessment:.....	53
2. Environmental Control and Avoidance:.....	54
XV. Evolution:.....	55
XVI. Prevention.....	55
XVII. Summary:.....	57
CONCLUSION.....	58
APPENDICE.....	60
RESUMES.....	63
BIBLIOGRAPHY.....	67



INTRODUCTION

Asthma is the most common chronic lower respiratory disease in childhood throughout the world which affects more than 6.6 million children in the United States. It is estimated that in economically developed countries, approximately 10% of the pediatric population is affected by this disease.

Asthma most often starts early in life and has variable courses and unstable phenotypes which may progress or remit over time (1).

It is among the leading causes of school absentees, hospitalizations and frequent visits to hospital emergency, but the most worrying still increasing constant of the prevalence in the last decades. The Pediatric asthma exacerbations account for more than 1.8 million emergency department (ED) visits annually (2).

The management of an asthma exacerbation is complex, involving a temporal and multi-disciplinary evaluation and reevaluation to adjust asthma medications and make a disposition decision. It is challenging to provide standardized care in a fast-paced and overcrowded environment like the ED (3).

The evolution of asthma is relatively unknown, 40%–50% of children followed for asthma will not be bothered by this disease as adults and nearly 50% of children follow for asthma will not disappear this disease after puberty. Morbidity and asthma mortality in children have become a problem in recent years, the most often due to inadequate therapeutic management (2,4).

The assessment of asthma control has become pivotal in the management of asthma. Uncontrolled asthma can lead to exacerbations requiring the patient to seek immediate care, frequently in an ED setting.

Our study will focus on asthma in children between 2–15 years, based on the analysis of anamnesis, clinical and para-clinical of children's cases presented to the pediatric emergency department (ED) of University Hospital Mohamed VI Marrakech.

The goal of this study is to assess the management of asthma and make an asthma care protocol in the pediatric ED to help standardize care and reduce time to disposition decision.



*PATIENTS
AND
METHODS*

I. Patients:

1. Inclusion criteria:

The criteria for inclusion of subjects in our study were:

- ✓ An age range from 2 to 15 years
- ✓ The admission of patients to pediatric emergency department of university hospital Med VI.
- ✓ A diagnosis of asthma retained on anamnestic, clinical, and para-clinical data.
- ✓ The occurring of an asthma attack, whatever its gravity.

2. Exclusion criteria:

- ✓ Age less than 2 years and more than 15 years.
- ✓ Other respiratory diseases than asthma.
- ✓ Improperly filled records.

II. Methods:

1. Type of work:

Our work is a prospective study of 216 of asthmatic children, which were presented to the pediatric emergency department of university hospital Med VI Marrakech for 1 year from 1st January 2014 to 31 December 2014.

2. collecting data:

This study included data collected through the exploitation records (see appendice) and filled out by duty doctors with the following information:

- Epidemiological data.
- Precipitating factors.
- Natural history of disease.
- Clinical data.
- Paraclinical data.
- Therapeutic data.
- Evolutive data.

3. Statistical analysis:

We had simple and univariate data, processed by Microsoft Excel in duration of two weeks after consulting the Epidemiological Laboratory of the Faculty of Medicine and Pharmacy of Marrakech.



RESULTS

I. Anamnesic Profile:

1. Age :

In our population studied, the age of our patients was between 2 and 15 years. Average age was 6.32 years.

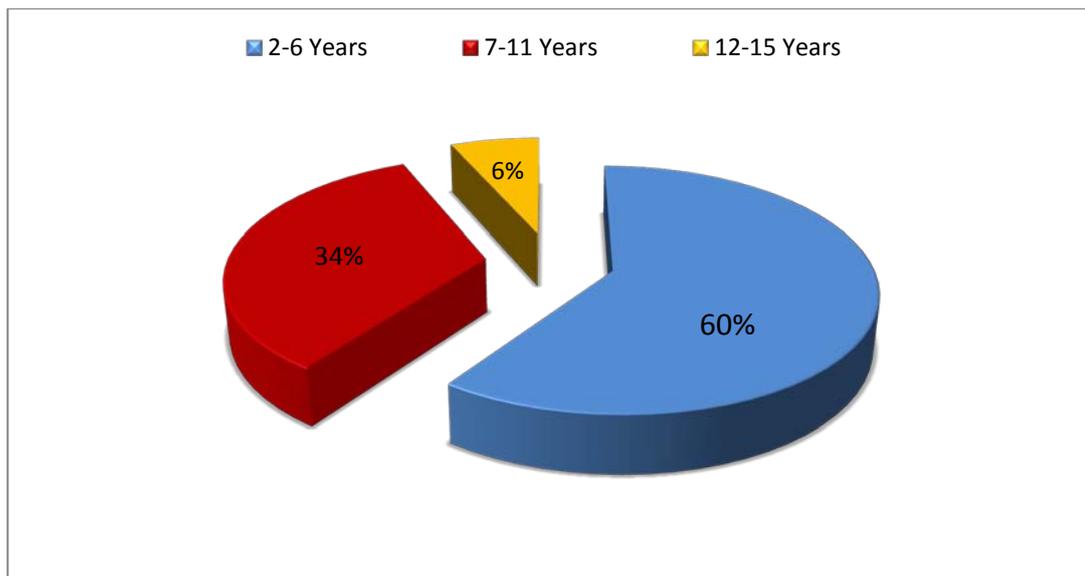


Figure 1: Distribution of patients according to age ranges

2. Sex

The sex ratio was 1.5 with male predominance (60%).

3. Socioeconomic level:

The majority of patients (55%) in our study were of medium socioeconomic level.

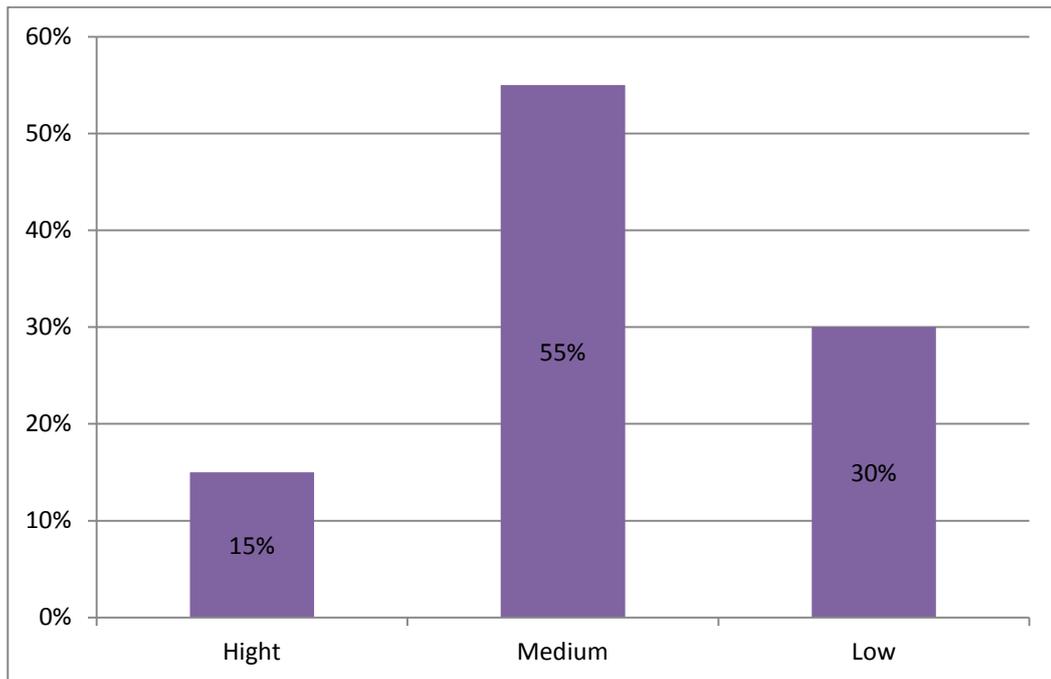


Figure 2 : Distribution of patients according to socioeconomic level

4. Type of residence

The residents were unhealthy in 23.1% (50) of cases.

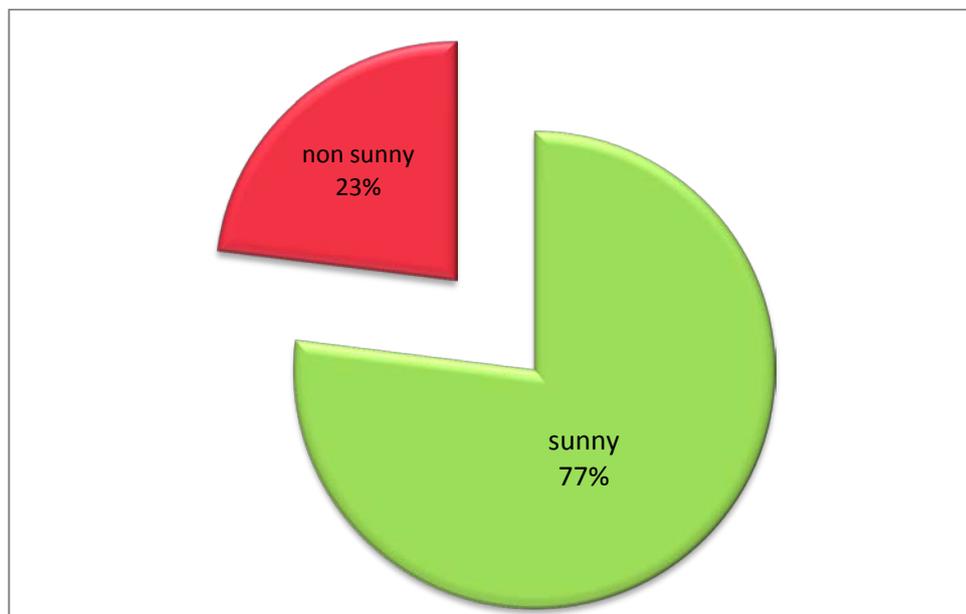


Figure 3: Distribution of patients according to their residence

5. Personal history:

5.1. History of Asthma

The majority of children (83.8%) included in our study had already developed an asthma attack and only 63.8% of them had received previous treatment.

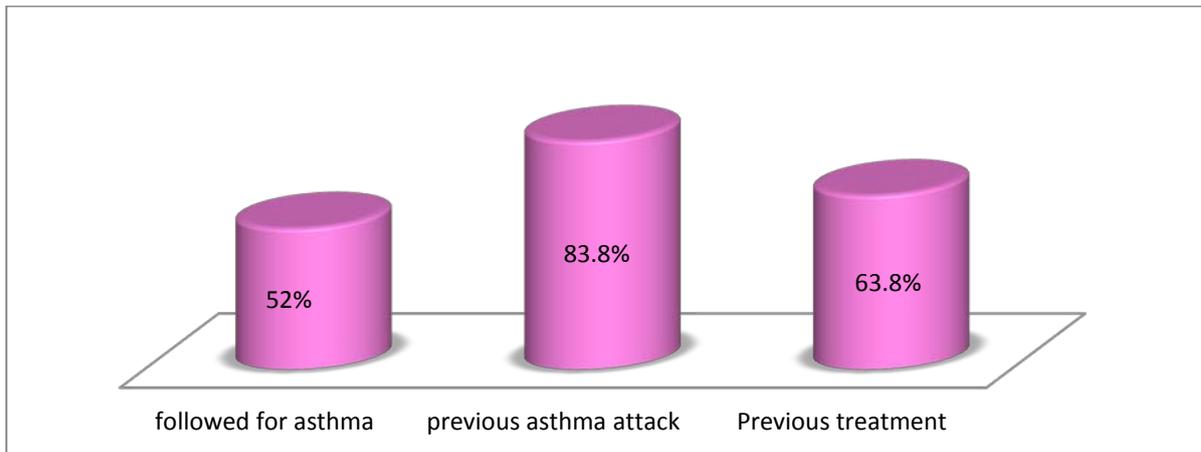


Figure 4: Distribution of patients according to their history of asthma

5.2. Personal atopy:

The personal atopy was associated with asthma in 95 children (44%), these types were represented in the following figure:

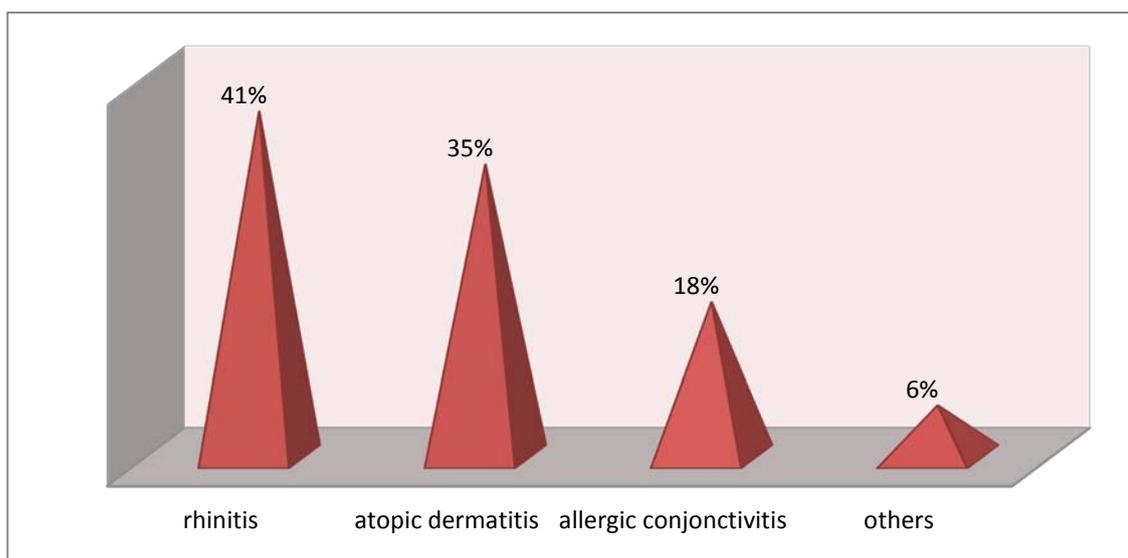


Figure 5: Distribution of patients according to their personal atopy.

In addition to the asthma and atopic history, children also presented others. The main one was respiratory infection in 141 children (65.27%) and the chronic vomiting in only 19 (8.7%).

6. Family history of asthma and / or atopy:

More than half of the patients (57%) had a family history of asthma and /or atopy, most frequently in mothers (29%).

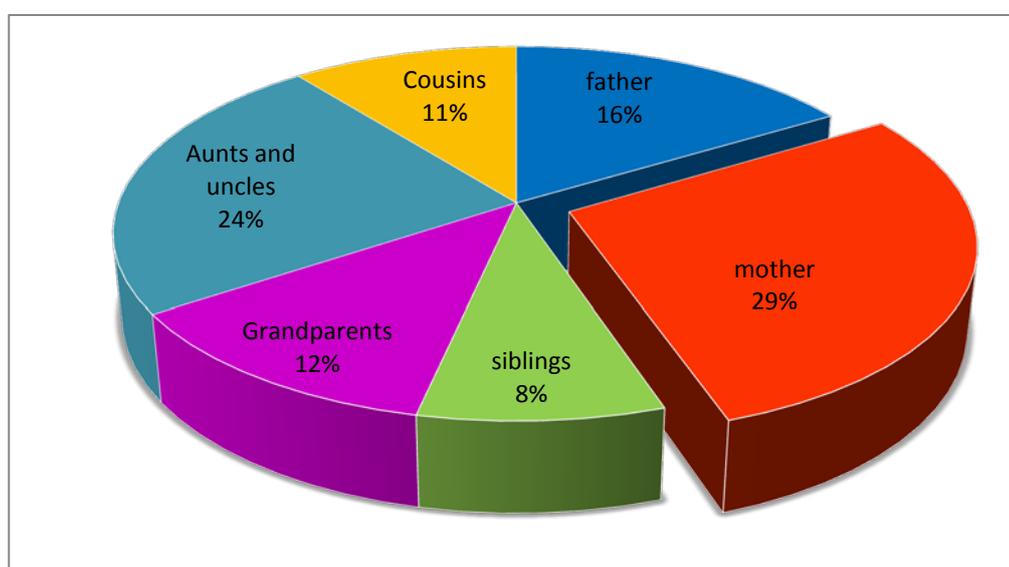


Figure 6: Distribution of patients according to their family history

II. Precipitating factors:

Table I: Distribution of patients according to precipitating factors.

Precipitating factors	Number of cases	Percentage
passive smoking	112	51.85%
Allergens	203	94%
physical exercise	177	81.9%
Respiratory infection	123	57%
Chimney smoke	3	1.38%
air pollutant	37	17.1%

In our study, we found that the allergens were the main precipitating factors (94%). This percentage include:

- Dust mites (64%)
- Mildew (24.4%)
- Indoor plant (47.8%)
- Domestic animals (5.3%)

III. Natural history of disease

1. Reason for consultation

In our study, the reason for consultation was: cough (104 children), dyspnea (84 children) or both (28 patients).

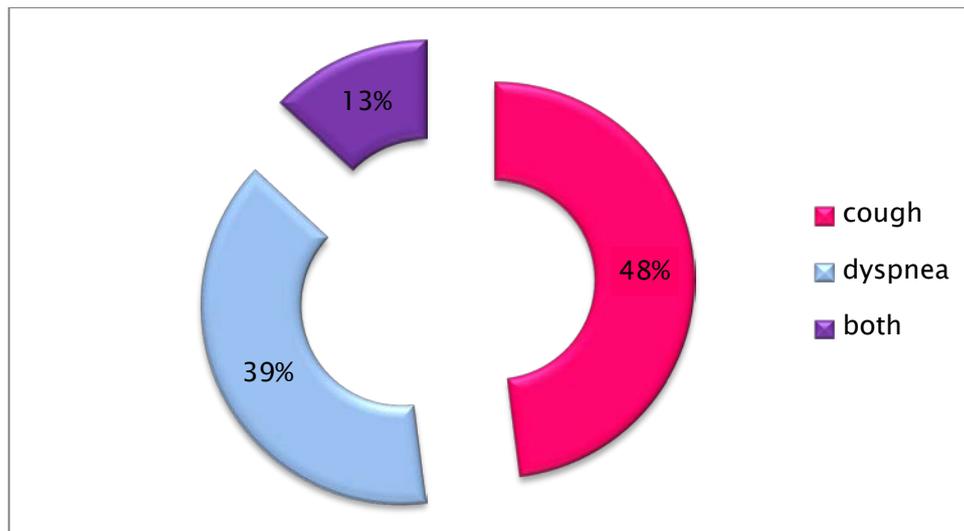


Figure 7: Distribution of patients according to their reason for consultation

2. Functional signs:

Dyspnea and cough were found, respectively, in 211 and 203 patients. However, other signs like wheezing (65 patients) and rhinorrhea (127 patients) were also presents.

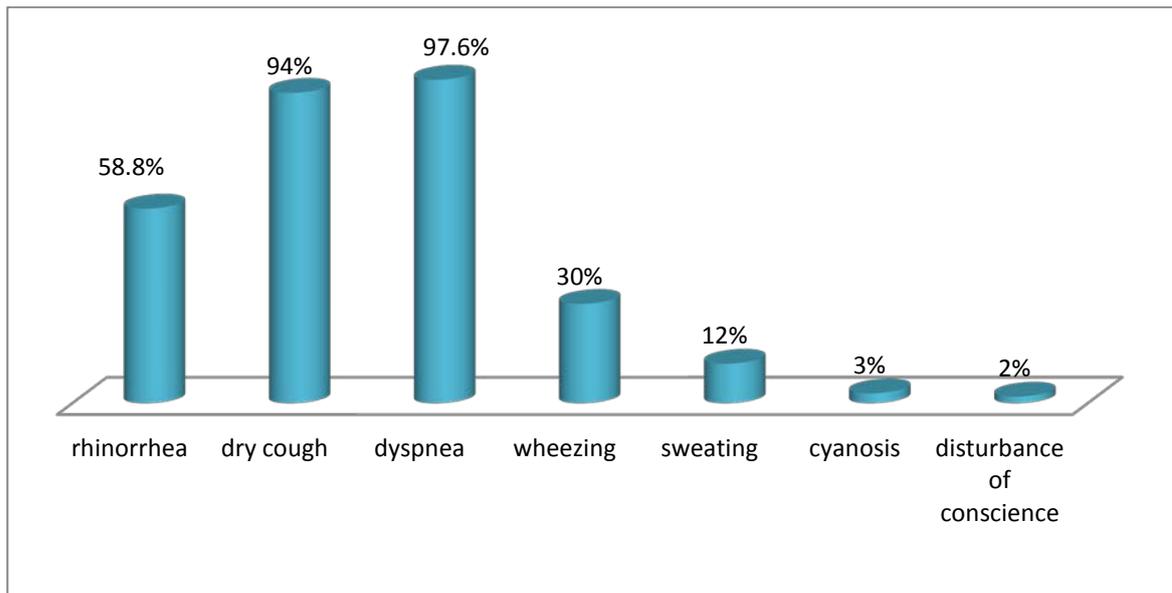


Figure 8: Distribution of patients according to their functional signs

3. Duration of the crisis

The duration of the crisis was varied between 2 and 14 hours, the average was 5.39 hours.

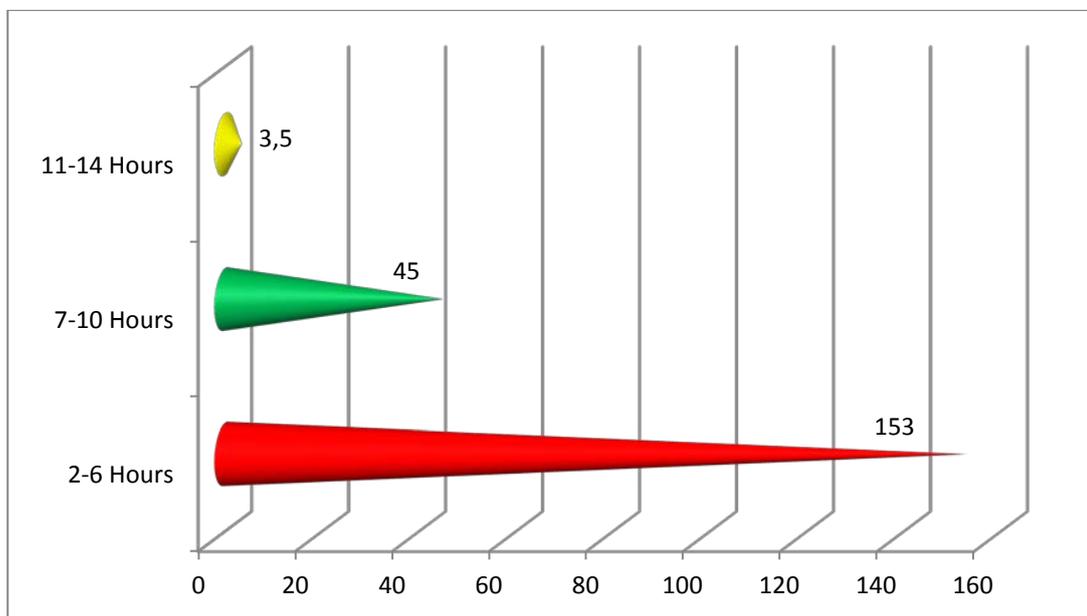


Figure 9: Distribution of patients according to the duration of their crisis

4. Seasonal predominance

Children included in our study were admitted frequently in autumn and winter period with a pick in November when 35 children (16.2%) were admitted.

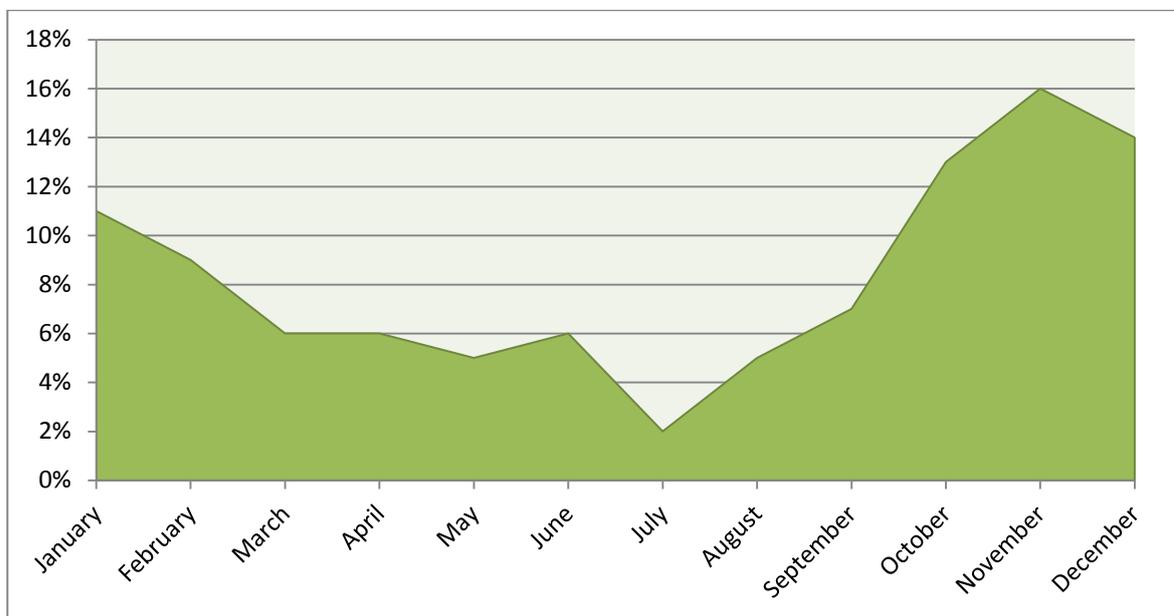


Figure 10: Distribution of patients according to their seasonal predominance

IV. Clinical examination:

All patients had wheezes (100%). Indeed, clinical signs are shown in the table below:

Table II: Distribution of patients according to clinical signs

CLINICAL SIGNS	Number of cases	Percentage (%)
polypnea	137	63.4%
Fever	71	32.9%
distended chest	82	38%
wheezes	216	100%
tachycardia	31	14.3%
subcutaneous emphysema	3	1.4%
decreased breath sounds	13	6%

V. Paraclinical investigation

C-reactive protein (CRP) and complete blood count (CBC) were requested respectively in 52 (24%) and 65 (30.1%).

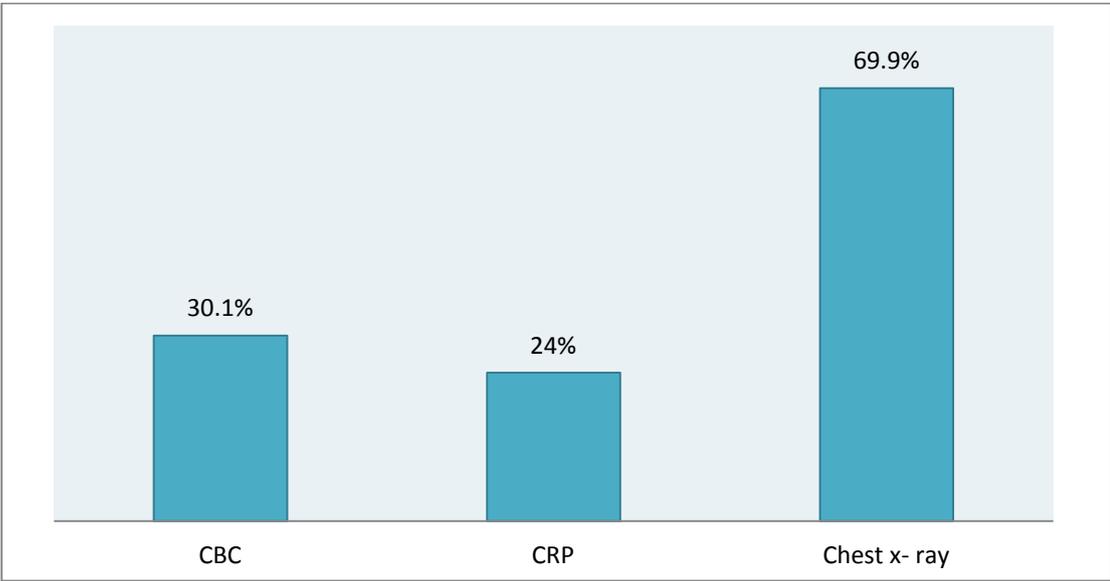


Figure 11: Paraclinical investigation requested

The chest X-ray was performed in 151 cases (69.9%) with results below:

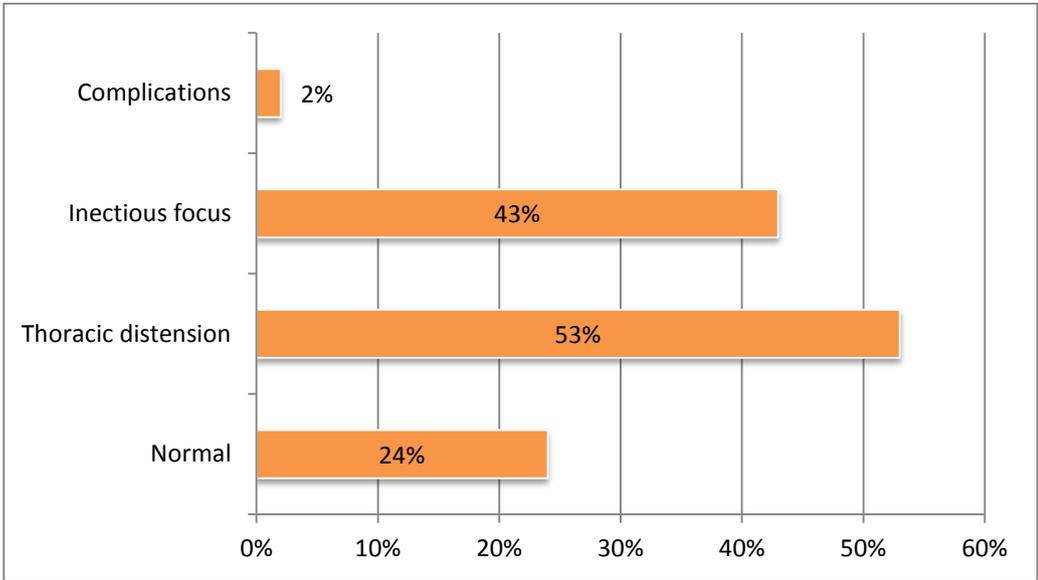


Figure 12: The result of the chest x-ray

VI. Therapeutic profile

We found in our study that the treatment of choice was nebulization of salbutamol used for 208 (96.3%) children.

Table III: Distribution of patients according to their Therapeutic profile

Treatment		Number of cases	Percentage
Oxygen Therapy		119	55.1%
Salbutamol	nebulization	208	96.3%
	injection	8	3.7%
Corticotherapy	oral	109	50.5%
	injection	121	56%
Hydration		72	33.33%
Antibiotic therapy		127	58.8%
Other treatment		5	2.3%

Antibiotics were prescribed for 127 children (58.8%), the most used drug was Amoxicillin-clavulanic acid (57: 26.4%), followed by Amoxicillin (39: 18%), and macrolides (31: 14.4%).

VII. classification

The crisis severity was classified according only to clinical signs. Indeed, the crisis was mild in the majority of cases (80%).

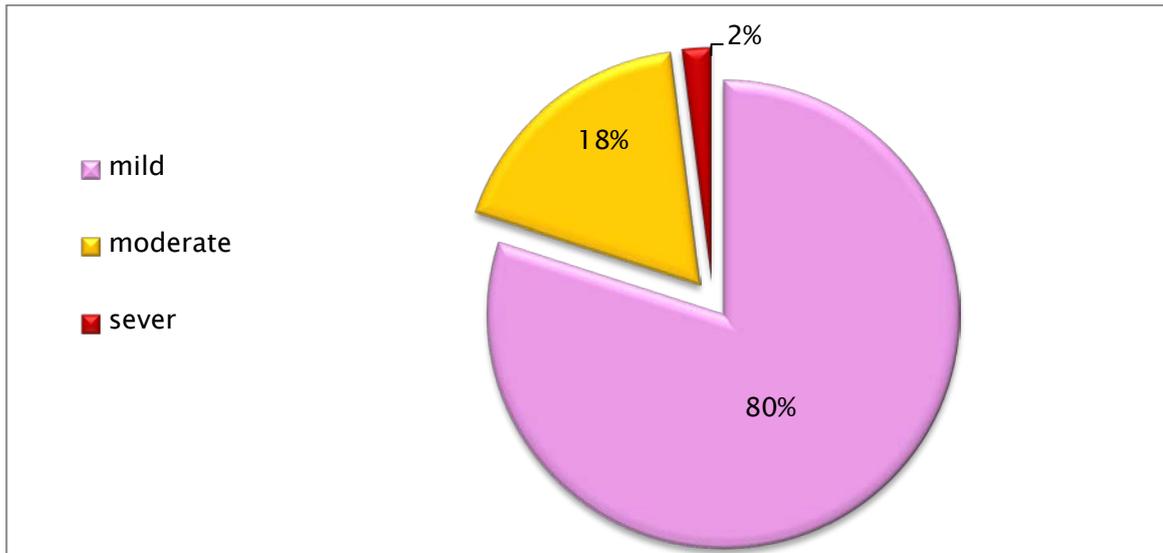


Figure13: Distribution of patients according to their crisis's severity

VIII. Follow up and Evolution

- We found that 62 children had regular follow of their asthma (28.7%). The rest of the patients were distributed among poor compliance (58: 26.8%), not followed (37: 17%) and disregarded (59: 27.5%).

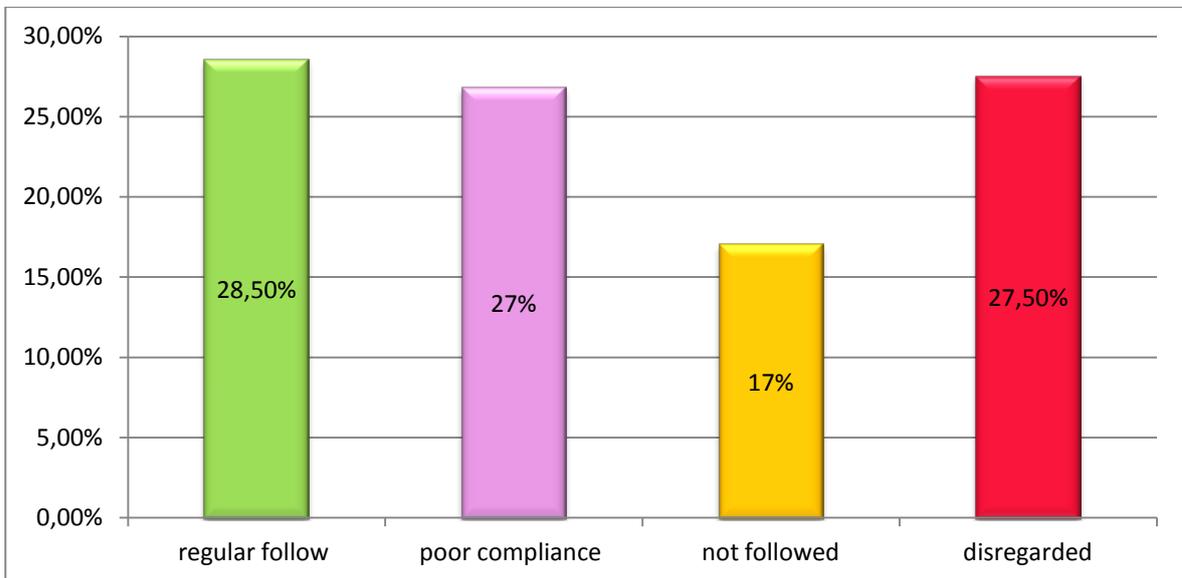


Figure14: Distribution of patients according to their following

- We found that 185 children (85.6%) presented an improvement and received ambulatory treatment, 27 (12.5%) had been hospitalized on pediatric service A and only 4 (1.9%) was admitted in Intensive Care Unit (ICU).

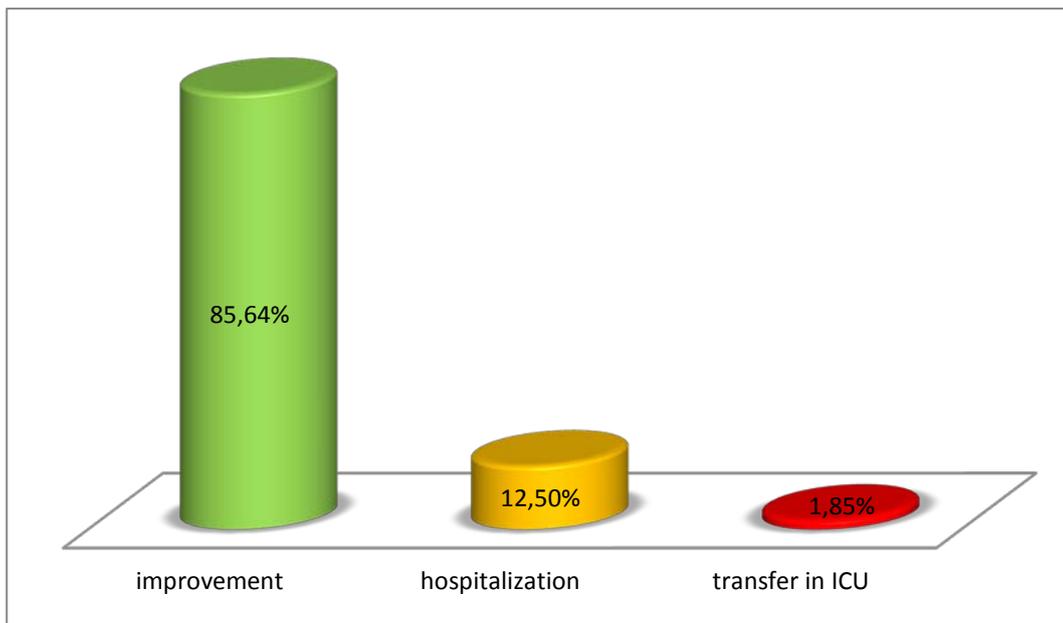


Figure 15: Distribution of patients according to their evolution.



DISCUSSION

I. Definition of asthma: (5)

The Global Initiative for Asthma (GINA) definition is:

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.

II. Pathophysiology of asthma:

1. Inflammation and Airway Remodeling

The inflammation in asthma is mediated by multiple cell types including mast cells, eosinophils, T lymphocytes, macrophages, neutrophils, and epithelial cells (6). Asthma has allergic and non allergic presentations, based on the presence or absence of IgE antibodies to common environmental allergens. Both variants are characterized by airway infiltration by T-helper (Th) cells, which secrete a predominantly Th2 milieu (cytokines IL-4, IL-5, and IL-13) (6), (7). These cytokines stimulate mast cells, cause eosinophilia, promote leukocytosis, and enhance B-cell IgE production.

Although mild asthma symptoms are episodic and reversible, with progression, severity long-term and permanent airway changes can be present. Long term changes can include airway smooth muscle hypertrophy and hyperplasia; increased mucus production (and associated risk of mucus plugs); and edema (6). In the subepithelial layer, thickening can range from 7 to 23 mm, versus 4 to 5 mm in normal subjects, and more commonly affects the smaller airways (2-6 mm) (7-9). Permanent changes can include thickening of subbasement membrane, subepithelial edema and fibrosis, airway smooth muscle hypertrophy and hyperplasia, blood vessel proliferation and dilation, and mucus gland hyperplasia and hypersecretion (6, 9, 10).

There is likely an occult process of bronchial inflammation that precedes clinical symptoms of asthma. Bronchial biopsies of children with early respiratory symptoms who progressed to asthma had higher concentrations of eosinophils in the bronchial mucosa and thicker subepithelial lamina reticularis than those who did not, and these findings were present before the clinical presentation of disease (11). This suggests that an inflammatory milieu may precede clinical symptoms in people with asthma. Furthermore, in patients who had clinical symptoms of asthma that then seemed to go into remission, evidence of inflammation and remodeling persist on follow-up biopsies (12, 13).

Clinical symptoms are a late manifestation of lower airway inflammation. There are many treatments for asthma symptoms, but asthma is not a curable disease, and there is evidence that inflammation is life-long and occurs even when no symptoms are present.

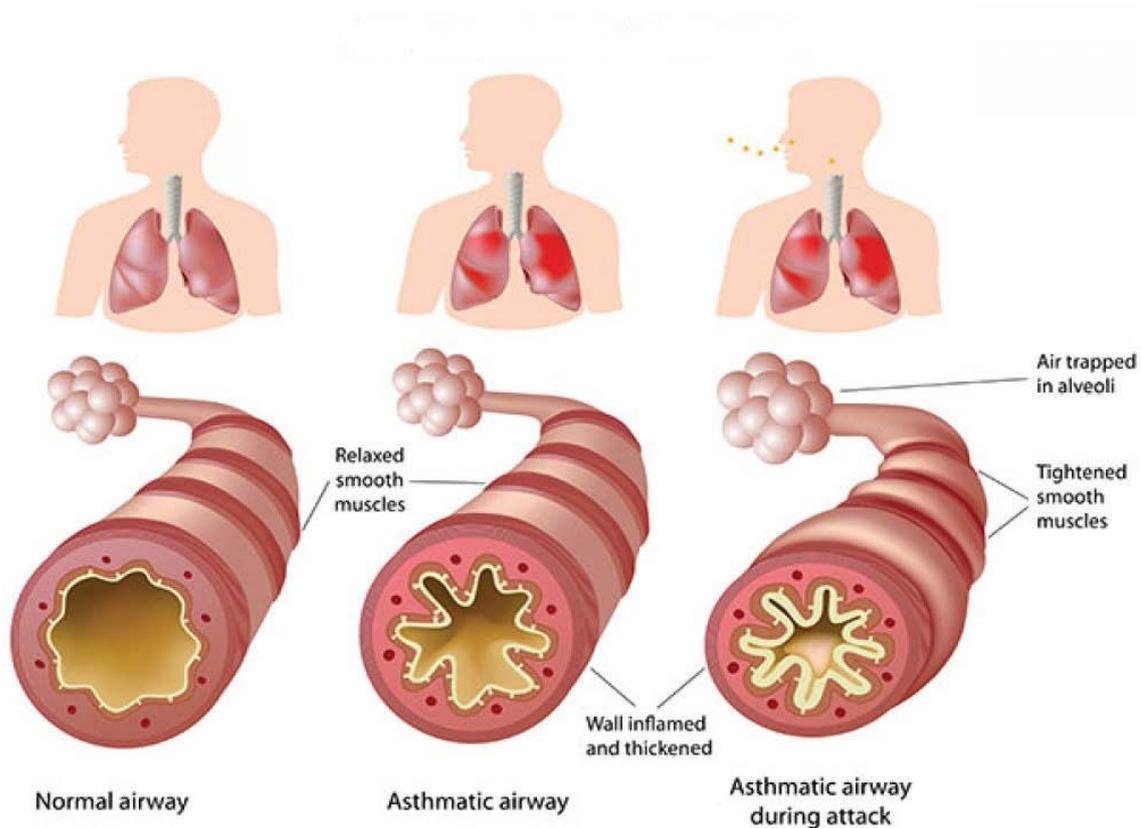


Figure 16: Illustrative representation of the pathophysiological changes in asthma

2. Bronchoconstriction and airway hyperresponsiveness.

The bronchoconstriction that occurs in asthma exacerbations is the main cause of obstructive symptoms. Airway hyperresponsiveness, or twitchy airways, occurs secondary to inflammation and airway remodeling. There is a distinct correlation between airway hyperresponsiveness and the degree of inflammation present. Bronchoconstriction can be induced by several pathways. Allergen-induced bronchoconstriction is caused by IgE-dependent mast cell degranulation, with resultant release of histamine, tryptase, leukotrienes, and prostaglandins (14–16). In addition to these mechanisms, bronchoconstriction by mast cell degranulation can also occur secondary to osmotic stimuli, which is likely the cause of exercise induced bronchoconstriction (6).

III. EPIDEMIOLOGY:

Known since antiquity, asthma was long considered a disease relatively benign. It became a concern after the Second World War, particularly in the early 1960s and was recorded deaths epidemic in Anglo-Saxon countries (17).

1. Prevalence

According to the global burden of asthma in 2003 the average prevalence of symptoms in Middle East children was 10.7%. The reported range for the prevalence of symptoms starts from 7.5% in Morocco to 17% in Kuwait (18). Based on the mentioned report; prevalence of asthma in Iran was about 5.5% in total population and 10% in childhood (18).

The International Study of Asthma and Allergy in Childhood (ISAAC) examined the epidemiology of asthma in Morocco. It included three Moroccan centers (Casablanca, Rabat, and Marrakech). The first surveys in Morocco in 1996 estimated the prevalence of asthma between 2 and 5.5%. Ten years later, as part of the same study, the prevalence of asthma was 6.6% among

children of Rabat and 12.1% in the same population in Casablanca. in contrast in Marrakech, less polluted city, the study found a prevalence of 17.9% (19). The prevalence of asthma in children in the Safi region was 3.4% in 2010 (20).

2. Mortality

Mortality from asthma has become a concern in recent years, due to the increase in the prevalence of asthma, increased severity of crisis, low patient compliance as well as poor management. Mortality among asthmatic children varies according to the authors from 0.4% to 0.7% (21). Asthma was recently reported to remain the sixth leading cause of death of children between 5 and 14 years of age in the United States. In Sweden, a study by Bergströma et al during 10 years period from 1994 to 2003, found 75 deaths suspected to be due to asthma (22).

In our study, we had no case of death.

3. Morbidity

Asthma is considered as an affection of the lifetime which 50% begins before the age of 2 years. Webb et al noted the persistence of crisis in 59% of children aged 3 years and a half after bronchiolitis (22). Similarly, Buffum still find 60% at 5 years with asthma among children who had asthma before the age of 2 years (23). In contrast, Park et al found that 20% of asthmatics at age 10 years among those who had presented dyspnea events were infants (24).

The frequency of hospitalization for asthma has also increased. This data is found in different countries: she has in fifteen years multiply by 3 in the USA and by 4 in Canada (25). In Morocco, the number of hospitalizations of children with asthma varies according to the months of the year, peaked in November and decreased in June and July (20).

In France, half of hospital admissions for children relate to asthma and hospitalization rates remained stable among children. A study of more than 1 year was conducted in 14 pediatric units in France, including children aged 3 years and older hospitalized for asthma

exacerbation. Data from 727 hospitalizations were collected. In 48% of hospitalizations, the children were 3–5 years. Among children with asthma, 57% had been admitted to the hospital for asthma exacerbation, 37% were admitted to the hospital or in the emergency department, and control of the asthma in the previous month was unacceptable in 46% of cases (26).

IV. Risk factors:

Numerous potential risk factors have been studied in relation to the development of asthma. Atopy is frequently identified as a strong risk factor for the development of asthma, yet there is no direct correlation between the two (27, 28). Some studies have demonstrated that early dust mite sensitization and maternal asthma are very significant predictors of asthma (29,30). Parental smoking is a significant risk factor for acute lower respiratory tract infections in infants, and the development of wheezing and asthma in children. Air pollution and viral infections are well-established triggers for asthma exacerbations, but there is conflicting data whether these factors contribute to developing asthma (33–36). Microbial exposure is inversely correlated with the development of asthma and atopy, and may account for the disparate prevalence of asthma in urban versus rural (specifically farming) environments (37–39). Ultimately, it is likely that asthma develops in genetically susceptible individuals through a combination of complex environmental exposures.

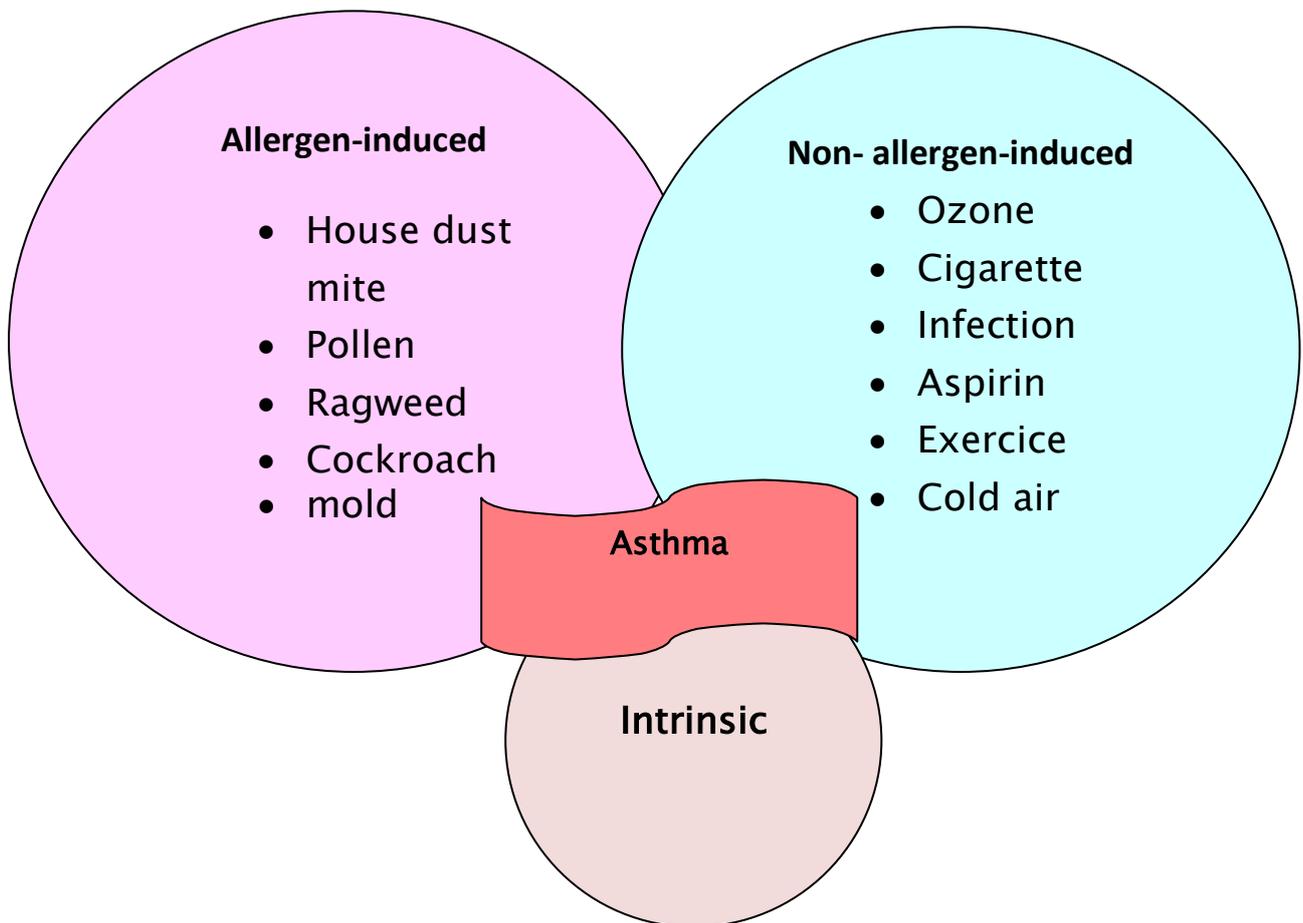


Figure 17: Effect of the interaction of various types of exposures on the asthma

1. Age of onset:

Most epidemiological studies report that the vast majority of children with asthma have their asthma during childhood.

Some authors have studied the influence of this factor on the asthma prognosis. The Barr et al studies found no dependence between the age of early onset and evolution of asthma. Gerritsen's study confirms this same observation (40).

Finally, a study by Sears in 2003 showed that the age of early onset is a risk factor for persistence and severity of asthma (41).

In our study, the age of the majority of our patients (60%) was between 2 and 6 years.

2. Atopy

The main personal risk factor for bronchial asthma is atopy in the individual or his/her family. The most important risk factor is atopy in the family. Some genes related to concurrent atopy, Ig E response and asthma have been found. These genes are located on chromosomes 5, 11 and 14. Children whose parents do not have extrinsic asthma have an asthma prevalence of 8 %, which increases to 15 % with asthma in one parent and 28.6 % with asthma in both parents. The incidence of asthma in first-degree relatives is 3–6 times of normal (42). The study by Martin et al found that eczema has a bad influence on the prognosis of asthma. Against, Wittis study showed that there is no relationship between eczema and the prognosis of asthma (42).

The study of Kocevar published in 2002, which estimated the number of hospital days and readmission in asthmatic Norwegian children with and without allergic rhinitis, has demonstrated that the allergic rhinitis is associated with an increase of the duration of hospitalization in these children (43).

Our study objectified that 44% had a personal atopy and/or family atopy, allergic rhinitis occupied the predominant place in 41% of children.

3. Genetic predisposition:

Asthma is a complex disease that results from the interaction between genetic predisposition and environmental factors

It seems that the inheritance of asthma and atopy does not follow the classical Mendelian patterns (43). In 1909, Drinkwar by studying an example of three generations suggested that asthma was transmitted in a Mendelian dominant mode (44).

Martinez et al studies in USA found the influence of several genes including one major gene transmitted respectively by an autosomal co-dominant and autosomal recessive. The role of HLA class 2 in the specific immune response to allergens has historically been initiated in the early years by the discovery of an association between IgE Specific Ra of 5 allergens and HLA-DW2 located almost exclusively in asthmatic (45).

A study in Italy in 2011 of 127 asthmatic children showed that imbalance between the oxidation forces and antioxidant defense systems has been implicated in the pathogenesis of asthma. Glutathione S-transferase (GST) plays an important role in cell protection against inflammation. The results suggest that the GSTA1 and GSTO2 are asthma genes involved in the increased risk of developing asthma in the Italian population (45).

In our study, the family history of asthma and/or atopy was recorded in more than half of cases (57%), most frequently in mothers (29%).

4. Infection-Related Asthma

4.1. Viruses:

The role of infection in asthma is varied in that it may exacerbate established asthma or be a contributing factor to the initial development of the clinical onset of asthma. Mounting evidence implicates both roles, with particular viral pathogens, namely human rhinovirus (HRV) and respiratory syncytial virus (RSV), among the most likely culprits in asthma inception (46,47).

In addition, outpatient wheezing illnesses caused by RSV and HRV early in life may increase the risk for subsequent wheezing episodes and the development of asthma (46). Recent studies have found that over 80% of wheezing episodes were associated with viral respiratory infections (47). In more than 60% of these children, the syncytial respiratory virus (RSV) was detected. The close link between bronchiolitis induced by viruses and the development of asthma has been demonstrated in several studies (48).

In our study, more than half of the children followed for asthma (57%) had a history of recurrent respiratory viral infections.

4.2. Bacterial pathogens:

Bacteria such as *Lactobacillus* and *Helicobacter pylori* are reported to be protective against asthma; whereas other bacteria are associated with an increased risk of asthma (49). One study demonstrates associations between neonatal hypopharyngeal colonization of *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*, with the subsequent increased risk of developing recurrent wheezing and childhood asthma (50).

It is unclear from these findings whether early colonization with these organisms in some way influences the development of asthma or if the presence of these organisms is a reflection of an altered immune system that predisposes to altered host airway responses to respiratory pathogens. Acute wheezing episodes of preschool children are associated with these bacterial pathogens, with a frequency similar to that seen with viruses (51).

5. Allergens:

High allergen exposure in the home and allergic sensitisation is a cause of acute exacerbations of asthma in children (52). Allergen exposure in schools might also be important. Low-dose exposure to cat allergen on the clothes of classmates at school is sufficient to cause deterioration of asthma (53).

The aeroallergens likely susceptible to intervention are pets, cockroaches, moulds, and house dust mites. House dust mites are a controversial area, although there is little evidence for routine use of avoidance measures for most children sensitised to these aeroallergens.

People with asthma of all levels of severity are commonly exposed to allergens in the home (54). Pet and cockroach sensitisation might be a marker for high morbidity, although whether cockroach sensitisation can be separated from the effects of low socioeconomic status is arguable (56). An interaction between passive smoking and pet sensitisation might exist. The use of synthetic bedding might be associated with severe wheeze (55).

The study of Platts-Mills et al (56) showed, in patients allergic to dust mites, improved clinical signs and decreased bronchial hyperresponsiveness (BHR), after a sojourn of two months in a hospital without mites. Recent studies have revealed a recurrence of symptoms and worsening non specific BHR when children were staying again at home, full of mites, reflecting the likely role of the latter in asthma symptoms (57, 58).

In our study, we found that the dust mite was the main precipitation factor, present in 203 patients (94%).

6. Gastro-esophageal reflux:

Gastro-esophageal reflux disease (GERD), which is the passive regurgitation of gastric contents retrograde into the esophagus may be associated with asthma (59). However, exact causal relationship has not been confirmed between asthma and GERD (60). The relationship between asthma and GERD has been detected in many studies by 24 h pH monitoring (61, 62).

The GERD was present only in 19 children (8.5%) from our patients studied.

V. Environmental factors:

1. Passive smoking:

Children are likely to be exposed to second hand tobacco smoke (SHS) at home (63). A study in Japan by Kaneita et al has shown that 64.8% of 6 month old children live with smoking parent(s), and of those, 57.9% of parents smoke indoors at home. Although, many previous studies have revealed the risk of SHS for childhood asthma (64).

There has been no prospective study of the risk of paternal smoking for asthma in children aged 2 years or less and only one study for children aged 3–4 years. Further, a wide range of estimated effect size of postnatal maternal smoking on incidence of childhood asthma was observed, indicating a need to confirm the results. A previous study by Kanoh et al. using data from the Longitudinal Survey of Newborns in the 21st Century, reported a positive hazard risk between parental smoking and childhood asthma incidence (65). Furthermore, although parents are encouraged to smoke outdoors or not to smoke (66), the difference in the contribution of these parental smoking behaviors to the risk reduction of asthma among their children has not been sufficiently evaluated (67).

In our population, 51.8% of asthmatic children were exposed to parental smoking.

2. Socio-economic factors:

Factors related to lifestyle and socioeconomic status appears to be involved in the expression of the manifestations of asthma (68).

The prevalence of asthma may vary according to life styles, it is higher in children living in rural areas, and especially if they live in the country during the first two years of life (69). The prevalence of asthma also varies according to the socio-economic level. Asthma is more common in people of higher social class as shown by the English and Swiss studies with prevalence of 13 and 9% in the highest class against 8 and 5% in the lowest (70). The social class

difference involves multiple environmental factors that may be more directly involved in the determinism of asthma.

In addition to the different lifestyles (heating, types of bedding, carpets ...), other factors may explain this predominance: better medical knowledge of diseases, excessive medical consumption, the use of higher hygiene products and finally the highest maternal age.

The majority of patients (85%) in our study had a low or medium socioeconomic level.

VI. Associated Allergic reactions:

Seasonal allergic rhinitis is one of the most common allergic diseases and its prevalence is steadily increasing. Asthma and allergic rhinitis are two manifestations of one common allergic respiratory syndrome. The relationship between asthma and allergic rhinitis is complex and upper and lower airways interact with each other. They often occur together and allergic rhinitis increases the risk of asthma development (71).

Although the relationship between allergic rhinitis and asthma has been well established, direct links between nasal and bronchial inflammation in seasonal allergic rhinitis without asthma remain to be exactly investigated. Several authors have focused on the effect of natural allergen exposure or nasal challenge on lower airway, but the dynamics of bronchial inflammation in patients with pollen allergic rhinitis during and out of the pollen season are not completely clear yet (71).

One study (72) was made in four cities of Mexico, about prevalence allergy (rhinitis) in children aged 6 to 7 years and adolescents aged 13 to 14 years of primary and secondary schools. In children aged 6 to 7 years, the results were in this order: current rhinitis (27.9%), rhinoconjunctivitis (24.2%), and associated allergies (9.2%). The corresponding frequencies in adolescents 13 to 14 years were respectively 33.3%, 34.1% and 18.4%. All children with rhinitis also had asthma symptoms, and symptoms of atopic dermatitis (AD).

AD is a common condition in infancy but disappears around age 3 years in a significant proportion of children. The prognosis is mostly determined by the severity and the presence of atopic sensitization. Early AD is associated with asthma at school age, but in many of these asthmatic children, wheezing manifests before or with the onset of AD. Children with AD and wheeze have a marked loss in lung function, suggesting a distinct phenotype rather than a progressive development from AD to asthma. (73).

In our study, the allergic rhinitis was associated with asthma in 41% of cases followed by atopic dermatitis in 35%.

VII. Clinical diagnosis:

1. Paroxysmal asthma attack: (5)

The classic triad of asthma attack includes cough, wheeze, and dyspnea. However, patients often present with only 1 of these symptoms, which can make diagnosis challenging. In studies of patients presenting solely with wheeze, cough, or dyspnea, only 24 to 35% were eventually diagnosed with asthma. Symptoms of asthma are typically worse at night or early in the morning. A personal and family history of asthma and atopy favor the diagnosis.

Clinical examination revealed chest distension, increased the sound and especially wheezing predominate in expiration

The exacerbation of asthma in children is defined by GINA 2014 as an acute or sub-acute deterioration in Symptom control that is sufficient to cause distress or risk to health, and necessitates a visit to a health care provider or requires treatment with systemic corticosteroids.

Early symptoms of an exacerbation may include any of the following:

- An acute or sub-acute increase in wheeze and shortness of breath.
- An increase in coughing, sneezing, nasal itching.
- Lethargy or reduced exercise tolerance.

- Impairment of daily activities, including feeding.
- A poor response to reliever medication.

In our study, 14.3% of children had tachycardia and only 3% had cyanosis.

2. Status asthmaticus or acute severe asthma: (74)

Pediatric status asthmaticus (PSA) is a medical emergency warranting prompt recognition and intervention. A status asthmaticus or severe asthma exacerbation is defined as an acute episode that does not respond to standard treatment with short acting b₂-agonists and corticosteroids. Although, a large variation exists in this definition between authors. In other definitions, need for hospitalization, emergency room visit or decline in peak expiratory flow (PEF) is also taken into account. PSA can result in respiratory insufficiency as well as circulatory failure and is potentially life-threatening.

Clinically, it is a dramatic asphyxia, acute respiratory failure made of:

- Respiratory syndrome with dyspnea, cyanosis, inability to cough and, Sweating, pallor, chest distended, tympanic, disappearance of wheezing
- Tachycardia with presence paradoxical pulse and cardiovascular collapse
- Neuropsychiatric syndrome with anxiety, disturbance of consciousness, confusion and stupor.
- The Radiography is essential to ensure the absence of complications (pneumothorax, pneumomediastinum and infection).
- The arterial blood gas shows severe hypoxia, but especially hypercapnia indicator of alveolar hypoventilation, importance of bronchial obstruction and depletion of the patient.

Finally, the onset of acidosis is a poor prognostic factor. In case of hypercapnia, the gasometric response to treatment should be evaluated within two hours after startup.

Data on the incidence or prevalence of PSA are scarce. In a USA cohort, admission for status asthmaticus between 1992 and 2006 approximately halved from 1.92 to 0.93 per 1000 children. In contrast, ICU care related to asthma increased from 0.09 to 0.31 per 1000 patients (75).

In our study, 4 patients presented a Status asthmaticus and were admitted to ICU.

3. Exercise induced asthma:

The clinical expression of Exercise induced asthma (EIA): is that of an asthma attack whose only characteristic is to follow an exercise. Typically, it occurs in the exercise, in a subject with normal respiratory function, and reaches its maximum intensity 5 to 10 minutes after cessation of exercise (76). In some cases, the EIA may be the only manifestation of asthmatic disease (77).

A retrospective study was conducted in four high schools in Rabat, with 1179 students and 16 teachers of physical education. The study found 70 asthmatic children (6%), 62.5% teachers felt that children with asthma cannot continue the effort and only 18% of students use asthma prophylaxis before the sports sessions (78).

The EIA is not a cons-indication to the sport in asthmatic children and does not justify any exemption in physical education. It remains accessible to administered therapeutic few minutes before exercise (79).

In our study, the physical exercise induced asthma attacks was found in 81.9% of children.

VIII. Classification of asthma: (80)

To address diversity and guide management, several factors have been used to classify pediatric asthma.

According to the National Asthma Education and Prevention Program's (NAEPP) Guidelines for the Diagnosis and Management of Asthma , asthma can be divided into four levels of asthma severity: mild intermittent, mild persistent, moderate persistent, and severe persistent.

In children older than 5 years, three major features are recommended in determining level of severity: frequency of asthma symptoms during the day, frequency of nighttime asthma symptoms, and measures of pulmonary function. These Guidelines divided the Forced Expiratory Volume (FEV) predicted into three levels: above 80% predicted typical of mild asthma (both intermittent and persistent), from 60 to 80% predicted typical of moderate asthma, and below 60% predicted typical of severe asthma.

Table IV: Classification of asthma severity (80)

	Symptoms	Nocturnal symptoms	FEV or PEF
Severe persistent	Continuous limited physical activity	frequent	< 60% predicted Variability >30%
Moderate	Daily attacks physical activity	> 1 Time a week	60 to 80 % predicted Variability >30%
Mild persistent	> 1 Time a week But < 1 Time a day	> 2 Time a month	> 80% predicted Variability 20 to 30%
Intermittent	< 1 Time a week Asymptomatic And Normal PEF between attacks	< 2 Time a month	> 80% predicted Variability <20%

FEV: Forced Expiratory Volume ; **PEF:** Peak Expiratory Flow

In our study, the crisis severity was classified according only to clinical signs. Indeed, the crisis was mild in the majority of cases (80%).

IX. Complications:

1. Infection:

It's a classic but rare complication, antibiotic therapy is justified if the patient is febrile, if there is purulent secretion and radiological home. The frequency of respiratory infections associated with bacteria during asthma attacks varies according to the authors from 10 to 17% (81).

More than half of our patients (58.8%) were treated for infection.

2. Mechanical complications : Pneumo–mediastinum, pneumothorax and subcutaneous emphysema:

The frequency of pneumomediastinum in children's asthma attacks is 0.3–5%. The diagnosis should be suspected in a sudden deterioration of respiratory status, retro–sternal pain radiating to the arms and neck, aggravated by breathing movements and sometimes swallowing. The essential clinical sign is the perception of a snowy crepitation of the upper cervical and thoracic region painful on palpation. The pneumomediastinum results on chest x ray deal by Hyper-V linear lights along the mediastinum, lifting the two pleural layers (82).

In our patients, the subcutaneous emphysema was found in 3 children (1.9%), with improvement under treatment.

The vast majority of cases, pneumomediastinum and subcutaneous emphysema are benign complication of asthma. Treatment coincides with that of the crisis itself, and their disappearance is done in a few days. The hospitalization of the child is indispensable because of the risk of pneumothorax. Pneumothorax, it is the consequence of a pneumo–mediastinum or pleural bubble rupture; it is a rare event (83).



Figure 18: Chest x–ray showing a subcutaneous emphysema

X. Differential diagnoses:

Table V: Important differential diagnoses in pediatric asthma (84).

Condition	Characteristics
Transient infant wheezing (eg, recurrent bronchiolitis)	Onset in infancy No associated atopy Associated with maternal smoking
Cystic fibrosis	Recurrent wheeze and failure to thrive
Primary ciliary dyskinesia	Associated recurrent otitis media and sinusitis Initial oxygen requirement postnatally Situs inversus in 50%
Primary ciliary dyskinesia	Persistent moist cough in combination with wheeze Purulent sputum suggests bacterial cause
Structural abnormality (eg, tracheomalacia, bronchomalacia)	Onset usually from or shortly after birth but occasionally later
Vocal cord dysfunction	High-pitched inspiratory stridor and dyspnea May be spontaneous or exercise induced Blunting of inspiratory volume loop on spirometry
Inhaled foreign body	Sudden onset Differential air entry or wheeze on examination
Cardiac failure	Associated with congenital or acquired heart disease (eg, dilated cardiomyopathy after viral infection)
Eosinophilic lung disorders including allergic bronchopulmonary aspergillosis	Skin prick test positivity to <i>Aspergillus fumigatus</i> Raised serum IgE Infiltrates on chest radiograph
Anxiety causing hyperventilation	No wheeze audible Spirometry at the time of symptoms may help distinguish
Exercise induced asthma	No respiratory symptoms other than with exercise Exercise testing may distinguish
Milk aspiration/cough during feeds	Symptomatic particularly with liquids Associated with developmental delay

XI. Para clinical investigations:

1. Chest X-ray :

Chest radiographs can be used to help exclude other diagnoses of wheezing or cough, especially in patients with first-time wheezing (85). Acute-onset, unilateral wheezing suggests foreign body aspiration, and patients may show hyperinflation on chest radiographs. Chest radiographs may show a structural abnormality in a patient with chronic wheezing that fails to respond to bronchodilator therapy (86–88).

Chest radiograph in patients with respiratory failure may help rule out complications from asthma such as pneumothorax or pneumomediastinum as well as other contributing causes for respiratory distress such as infection or cardiac disease. Although no clear guidelines exist, chest radiographs should be considered in the following instances: asymmetric wheezing, wheezing that fails to respond to bronchodilator therapy, or patients with respiratory failure (88).

Two patients in our study, which was initially treated as asthma had presented a foreign body in the chest X-ray.

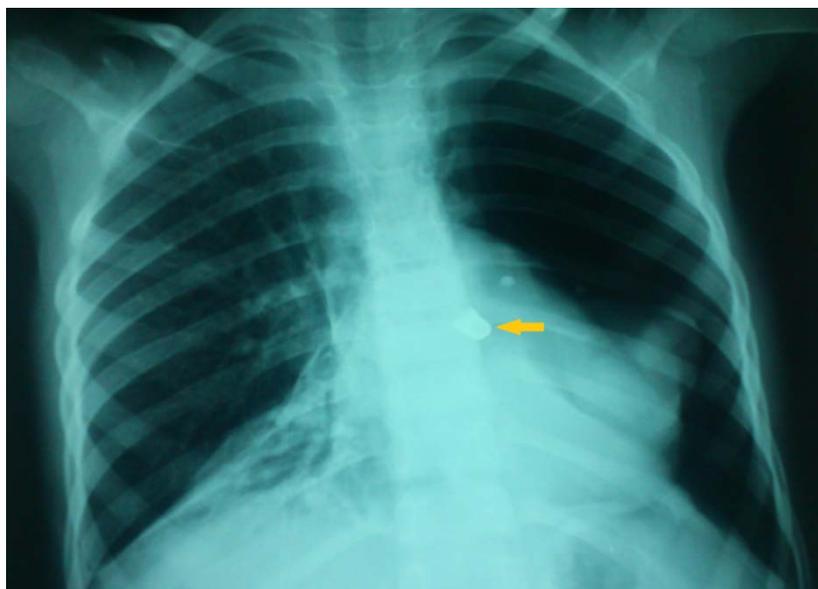


Figure 19: Chest X-ray objectified a foreign body in a 5 year old girl who was treated initially as asthma

2. Skin tests:

The skin prick test is a commonly used procedure in secondary care for assessing a specific sensitization in allergic respiratory diseases. Up to 85% of asthma patients show positive reactions to skin tests for common aeroallergens (89). Research has demonstrated the reliability and predictive validity of allergy skin testing.

Failure to use this combined approach could result in deficiencies in diagnosis and asthma management. Tschopp et al. recently concluded that the skin prick test should be used as a primary tool by clinicians to assess respiratory allergic diseases, as the test is comparably economical and provides immediate educational information for patient and physician (89).

3. Respiratory function tests:

Evaluation of lung function is important for both diagnosis and monitoring. Nevertheless, normal lung function tests do not exclude a diagnosis of asthma, especially for intermittent or mild cases (89). Therefore, these tests are considered supportive. Performing the tests when the child is symptomatic may increase sensitivity.

In children younger than 5 years, newer lung function tests that require less cooperation have been used (such as oscillometry or specific airway resistance). However, these are not generally available outside specialized centers (90).

Respiratory function tests are recommended for children old enough to perform it properly; the proposed range of minimum age is between 5 and 7 years.

Assess the disease by the only clinic, causes an under appreciation and under treatment which can be harmful to the respiratory future of the child. Only lung function test confirms a sense of well being breathing. It is used to classify the severity of the disease and adapt treatment; it must be repeated at regular intervals to adjust the therapeutic stage of the disease (91).

4. Peak Expiratory Flow Rate:

The peak expiratory flow rate (PEFR) is the measure of airflow during a brief, forceful exhalation. The measurement of peak flow rates can be taught to the patient and routinely used at home to monitor disease severity (92).

Reduced peak flow measurements do not differentiate between obstructive and restrictive diseases; spirometry and sometimes measurement of lung volumes are necessary to distinguish the two. Peak flow measurements are not sufficient to distinguish upper airway obstruction (eg, vocal cord dysfunction) from asthma. The validity of PEFR measurements depends on patient effort and technique. There is also no standardization among peak flow measurements. Despite the shortcomings, many patients use PEFR to successfully follow the progression of their asthmatic disease (93).

Fonceca et al followed 75 children aged 5–16 years with persistent asthma for a 12–week period. Their study showed little or no evidence of correlation between clinical severity scores and the values of FEV and PEF but showed a positive correlation between PEF and FEV obtained by spirometry and they establish PEF as a reasonable measure for use in the home when spirometry cannot be obtained. The researchers state that their findings reinforce the use of PEF for the management of children with asthma but state that PEF should not be the only objective parameter used in monitoring asthma control (94,95).

The PEFR is not available in our emergency department.



Figure 20: Peak Expiratory Flow (PEF)

5. Blood gases:

It is required in severe asthma attacks to assess respiratory failure. Respiratory alkalosis is common at a moderate asthma attack, marked by hypoxia with hypocapnia. The transition to normocapnia and especially to hypercapnia indicates decompensation and respiratory acidosis.

In our study, the blood gas was used only in ICU, and the oxygen saturation was less than 92% in 32.5% of our patients.

XII. Therapeutic management:

1. Treatment of the usual acute asthma attack:

Early recognition and treatment of the asthma exacerbation is essential for success in overall management. Patients presenting to the ED with acute asthma should be quickly evaluated

for the adequacy of airway, breathing, and circulation. This evaluation should include a complete set of vital signs, pulse, oximetry, respiratory rate, and an assessment of respiratory effort.

Treatment should be started immediately. Current recommendations are outlined in the 2007 National Asthma and Education and Prevention Program (NAEPP), Expert Panel Report (EPR-3) coordinated by the National Heart, Lung, and Blood Institute of the National Institutes of Health and the 2005 Canadian Asthma Consensus Guidelines (96). Goals of therapy include correction of hypoxemia, rapid reversal of airway obstruction, and treatment of inflammation.

1.1. B2- Agonists:

B2-Agonists are potent bronchodilators that act on b receptors to quickly and effectively relax bronchial smooth muscle. Short-acting b-agonists are the recommended first-line therapy for the acute asthma exacerbation (96). Albuterol is the most commonly used b2-agonist for acute asthma. B2-Agonists can be administered in multiple forms, including metered-dose inhaler (MDI), nebulizer, subcutaneous injection, and intravenous injection. Traditionally, aerosolized bronchodilators have been administered by continuous-flow nebulization. However, multiple studies have found little difference in efficacy between MDI and nebulizer therapy (96). Hospital admission rates are equivalent between the 2 modalities, MDI is more cost-effective and time-effective than nebulizer therapy (96). An additional concern with aerosolized or nebulized medication is its potential to spread infectious agents.

For acute asthma, 2 to 6 inhalations of albuterol are given with MDI and spacer device. Therapy is repeated every 20 minutes for up to 4 hours until there is maximal improvement in respiratory symptoms. Alternatively, if MDI is unavailable or if the patient is unable to use proper techniques, 2.5 to 5 mg of nebulized albuterol is given every 20 minutes to a total of 3 doses (maximum of 15 mg in one hour). Albuterol can also be given as a continuous nebulization at a rate of 10 to 15 mg over 1 hour (96). Therapy should be titrated to an objective measure of airflow obstruction (eg, FEV1 or PEF) and clinical response. B-Agonists should only be used for relieve of acute symptoms. Adverse effects associated with b-agonists include tremor,

tachycardia, palpitations, hyperglycemia, and hypokalemia. Current evidence does not support the use of intravenous β_2 -agonists for the treatments of acute severe asthma (96). A meta-analysis including 21 pediatric studies and more than 2000 children demonstrated that in acute asthma spacers were as effective as nebulizers in limiting hospitalization rates and reducing the time spent in emergency department (96).

In our study, the majority of children (96%) used nebulized salbutamol.



Figure 21: the metered-dose inhaler with spacer

1.2. Anticholinergics:

Anticholinergics block the action of acetylcholine on the parasympathetic autonomic system. They decrease vagally mediated smooth muscle contraction in the airways leading to bronchodilation. Anticholinergics are recommended for the treatment of severe asthma, in combination with short-acting β -agonists (97). The synergistic effects of these 2 agents decrease hospitalization rates and improve lung function. Ipratropium bromide is the most commonly used anticholinergic agent for the treatment of asthma.

For the acute exacerbation, 0.5 mg of ipratropium is given by nebulization every 20 minutes for 3 doses. Alternatively, 8 puffs with MDI and spacer can be given every 20 minutes for up to 3 hours. A recent study (97) showed the benefit of a multiple-dose protocol of ipratropium combined with albuterol in patients with severe asthma exacerbations ($FEV_1 < 50\%$).

Both medications were administered through MDI every 10 minutes for 3 hours. Patients had significant improvement in pulmonary function and decreased admission rates.

In a 2008 Cochrane Review, the addition of multiple doses of anticholinergics to b2-agonists decreased the risk of hospital admission by 25% in children with moderate and severe asthma (97).

The anticholinergic is not available in our emergency for that it was not administered in our patients.

1.3. Corticosteroids

Corticosteroids are recommended for asthma exacerbations that are incompletely responsive to inhaled beta-agonists. Doubling the dose of inhaled corticosteroids (ICS) at the onset of an exacerbation is ineffective in improving lung function and controlling symptoms (98). A review of published pediatric studies concluded that ICS given at high doses seem to have a modest benefit compared with placebo but are inferior to oral corticosteroids (OCS) in preventing hospitalization in more severe attacks (99).

The National Asthma Council (NAC) recommends that treatment with ICS should be considered for patients with any of the following:

- Exacerbations of asthma in the last two years.
- Use of reliever medication three times a week or more.
- Asthma symptoms three times a week or more.
- Waking at least one night per week due to asthma symptoms; and/or
- Impaired lung function.

The most commonly used OCS is prednisolone, often chosen because of its palatability rather than because of comparative OCS data. Pediatric randomized controlled trials (RCT) has examined differing doses of prednisolone, in a mild to moderate severity cohort, and found no difference between 0.5, 1, and 2 mg/kg. At present, the recommended dose of oral prednisolone is 1 mg/kg every 12 to 24 hours (maximum dose: 50 mg) (99). The optimal duration of

treatment also is unclear. A single dose of OCS on admission has failed to show consistent benefit. A recent pediatric RCT comparing 3 and 5-day courses demonstrated equivalent efficacy. Currently a 3-day course is recommended, lengthened to 5 days in more severe exacerbations (100). Early administration of OCS, within the first hour of arrival, has been shown to reduce admission rates in children (101).

IV corticosteroids should be used in patients who are unlikely to tolerate OCS (children with severe exacerbation). Benefit is seen by 4 to 6 hours. A single dose of oral or intramuscular dexamethasone (0.6 mg/kg to a maximum of 15–18 mg) was comparable to a 5-day course of oral prednisolone (2 mg/kg/d) in three pediatric RCTs (101). Systemic side effects may be more common with IM long-acting corticosteroids.

In our study, the use of oral corticosteroids was found in the half of children (50.5%). Indeed 75% of this corticosteroids was prednisolone.

1.4. Magnesium sulfate

Magnesium is a potential therapeutic agent in asthma because of its bronchodilating effect on smooth muscle cells and reduction of the neutrophilic burst associated with inflammation (102). In a meta-analysis of five pediatric RCTs, IV magnesium sulfate decreased hospitalizations and improved pulmonary function and symptom scores, despite variation in dosage (25–75 mg/kg) (103). Another predominantly adult meta-analysis that included two pediatric RCTs confirmed its safe and beneficial role in severe acute asthma (103). Meta-analysis of trials of inhaled magnesium sulfate, including two pediatric RCTs, demonstrated significant improvement in lung function only in severe acute asthma and no difference in hospitalization (103).

In our emergency department, the magnesium sulfate was not used in any of our patients.

1.5. Other Therapies:

a. Heliox:

Heliox is a blend of 70 to 80% helium and 20% to 30% oxygen, which has a lower gas density than air. Heliox can potentially decrease resistance to airflow and enhance delivery of

nebulized bronchodilators (104). The role of heliox in asthma management remains unclear. Currently, it is not recommended as an initial treatment of asthma (104). There have been few controlled studies and the optimal duration of heliox treatment is unknown. Current guidelines recommend that heliox-driven albuterol nebulization should be given for life-threatening exacerbations or if the exacerbation remains severe (PEF <40%) after 1 hour of conventional therapy (104).

A Cochrane Review concluded that heliox improved pulmonary function only in the subgroup of patients with the most severe obstruction. However, this conclusion was based on a small number of studies (104). A randomized controlled trial of 30 children with moderate to severe asthma showed a greater degree of clinical improvement in those treated with heliox-driven albuterol nebulization (105).

The heliox is not yet available in morocco.

b. Leukotriene modifiers:

Leukotrienes are potent inflammatory mediators. Leukotriene modifiers improve lung function and decrease asthma exacerbations (106). Three leukotriene modifiers are currently available for long-term therapy for asthma: montelukast, zafirlukast, and zileuton. However, many studies have found that overall efficacy of inhaled corticosteroids is superior to that of leukotriene modifiers for the long-term control of asthma (106).

Leukotriene modifiers are alternative chronic treatments of patients with mild persistent asthma, who are unable to use inhaled corticosteroid (101). Leukotriene modifiers have not been used traditionally in the emergent setting. However, promising new research shows that leukotriene modifiers may have a future role in the ED.

A randomized multicenter trial evaluated the effects of oral zafirlukast in 641 acute asthmatics. Those receiving zafirlukast in the ED had significant improvement in dyspnea and FEV₁, decreased risk of relapse, and decreased need for extended hospital care. Intravenous leukotriene modifiers have also shown promise in the setting of acute asthma (107).

A randomized controlled trial of 201 asthmatics showed significant improvement in FEV in the first 20 minutes after treatment with intravenous montelukast (106).

c. Antibiotics:

There is a paucity of evidence to support the use of antibiotics in the routine management of acute asthma, because most exacerbations are triggered by viral infections. The Everyday Clinical Practice indicates that antimicrobials are commonly prescribed in patients with asthma both in Europe and the United States. This attitude finds little support from current guidelines. The recent National Asthma Education and Prevention Program Expert Panel Report indicates that antibiotics are not currently recommended for the treatment of acute asthma exacerbations except when fever, purulent sputum or clear evidence of infection are present.

Both of these rather old studies found no association between antibiotic treatments and improvement in any asthma outcome (108).

We saw in our study a significant use of the antibiotic in the treatment of asthma in more than half (58.8%) of the cases.

2. Treatment of status asthmaticus:

2.1. First-line treatments:

Children with status asthmaticus have a greater frequency of hypoxemia than adults. These children are at a higher risk of ventilation–perfusion mismatch because of age related differences in pulmonary mechanics including lower functional residual capacity/total lung capacity ratio, increased chest wall compliance, and higher peripheral airways resistance (109). This mismatch may be exacerbated initially by bronchodilators and can cause desaturation during the early stages of therapy.

- Oxygen should be delivered to relieve dyspnea .Children, and especially infants, are at risk for respiratory failure and develop hypoxemia more rapidly than adults. Therefore, monitoring of oxygen saturation is necessary. National Heart, Lung, and

Blood Institute (NHLBI) guidelines recommend oxygen administration to maintain saturations greater than 90%. SaO₂ must be monitored until a clear response to bronchodilator therapy has occurred. High-flow oxygen despite good saturations can lead to poorer outcomes (109).

- B-Adrenergic agonists, such as albuterol, are the most effective and commonly used bronchodilators in the United States. Continuously administered albuterol has been shown to improve outcomes and to be more cost effective than continued intermittent therapy. High doses (as much as 20–30 mg/hour) are generally well tolerated and have been used for days in this population. Orally administered b-adrenergic agents are not indicated for status asthmaticus (109,110).
- Corticosteroids are used to treat airway inflammation and edema in children with acute asthma (110). There is little published evidence regarding the duration and dosage of corticosteroids for the treatment of status asthmaticus in children. Duration of therapy is typically driven by the severity of the exacerbation and the rapidity of response to therapy (110). If corticosteroid treatment is longer than 7 days, a slow dosage is recommended (111). Currently, National Heart, Lung and Blood Institute guidelines recommend administration of corticosteroids systemically rather than by an inhaled route. Orally administered medications may be used if a child can tolerate oral medication, but if not, intravenous (IV) medication is preferred. The National Heart, Lung and Blood Institute guidelines suggest that 2 mg/kg/d of systemic prednisone or methylprednisolone be used for acute asthma, but offer no recommendations for children with respiratory failure. In this population of children with more severe disease, some authors have suggested using dosages as high as 4 mg/kg/d (111).
- The need for IV fluid boluses should not be overlooked in children with status asthmaticus (109). Several pathophysiologic mechanisms contribute to the need for robust intravascular volume in these children. Specifically, an increased intrathoracic pressure from air-trapping can lead to decreased venous return, which coupled with

bronchodilator-induced tachycardia can reduce filling time and also potentially decrease cardiac output. Additionally, children with acute disease frequently present dehydration because of decreased oral intake and elevated respiratory rate. This dehydration can also be exacerbated by the nausea and vomiting associated with β -adrenergic agonist therapy. All of these factors contribute to the need to restore euvolemia in this population. However, previous authors have identified a syndrome of inappropriate antidiuretic hormone in children with status asthmaticus, so fluid balance should be monitored carefully (112).

In our study, we found that the first line of treatments in pediatric ICU was based on the antihistaminic, nebulized salbutamol, corticosteroid, Anticholinergics and antibiotics. The Magnesium sulfate was used in one patient only.

2.2. Second-line treatments:

Any child who does not sufficiently respond to first-line treatment of status asthmaticus should be strongly considered for admission to an ICU setting. These children require close monitoring of their clinical status including continuous cardiorespiratory monitoring. Second-line therapies should be considered, but these should be additive and not replace the first-line therapies.

There are several second-line treatments available; however, few comparative studies have been performed, and none have been shown to be superior to any of the others (113).

3. Proposed treatment to return home:(113)

This point is important when an asthma attack is associated with airway inflammation and where recent data show that at least 25% of children remain symptomatic within 15 days after the return home.

The notion of an unbalanced chronic asthma prior to admission to the emergency pediatric and / or the occurrence of a severe asthma attack should be prescribed a maintenance treatment for at least 3 months and are part of a specialist consultation as soon as possible.

The short acting β_2 mimetic administration should be continued at least 7 days with a proposed dose of 2–4 puffs or 200 mcg equivalents to 400 μ g salbutamol x 3 to 4 puffs.

All of our duty doctors prescribed the short acting β_2 mimetic, but nobody was there to educate the patient how to use it.

4. Maintenance treatment of asthma:

Maintenance treatment does not mean lifelong treatment. Only persistent asthma requires long-term treatment. The basic treatment aims to allow the subject's personal and social life as normal as possible, permanent disappearance of symptoms, nocturnal asthma, exercise-induced asthma and avoid the need for hospital.

This requires well-estimate the severity inter-critical phase and well master the environmental factors. The occurrence of severe exacerbation or loss of control requires consultation to reassess treatment.

Inhaled corticosteroids are indicated according to the latest international consensus in the pediatric in the treatment of moderate and persistent asthma in children. They considerably improved the basic treatment of asthma. It now represents the main weapon of the long-term treatment of moderate to severe asthma (114).

International GOAL (Gaining Optimal Asthma Control), included 3416 patients treated for one year by fluticasone fixed combination salmeterol + fluticasone. The proportion of controlled patients was higher in fluticasone + salmeterol combination in subjects treated with inhaled corticosteroids alone (115).

Initiation of treatment is variable, some start with a moderate dose gradually increase if asthma is not improved, and others begin immediately by a high dose they decrease after a few

weeks. Usually the dose reduction is only possible after a minimum period of 6 to 8 weeks and often more (116).

Gaga et al in their study showed that European children with asthma were generally maltreated (117). Only 26% of children were receiving inhaled corticosteroid therapy while 46% had persistent asthma. This under-use of inhaled corticosteroids, particularly in the more severe stages, contributed to poor control of asthma observed in the study.

XIII. Control and follow of asthma:

The assessment of asthma control has become pivotal in the management of asthma. However, several surveys in developed nations have shown that the majority of patients with asthma do not enjoy adequate asthma control (118).

Asthma control refers to control of the clinical manifestations of the disease, and it is the ultimate goal of asthma management (119). There is a clear relationship between asthma severity and asthma control. The underlying severity of asthma in a patient may be modified by changes in the environment and by the treatment received for asthma. Ultimately, the changes in these environmental and treatment factors will impact on the patient's symptoms and their ability to have a normal activity.

Asthma control reflects the combined effect of underlying disease severity, environmental exposures and the effectiveness of treatment. Laforest et al. identified several independent patient-related determinants of inadequate asthma control, including female gender, active smoking and overweight status (120). Control also varied according to the type of asthma supervision. Patients supervised exclusively by specialists rather than general physicians were more likely to have their asthma properly controlled.

Patients who were dispensed combined long-acting beta-2 agonist (LABA) and ICS therapy were also more likely to have their symptoms properly controlled, particularly at higher doses.

Current asthma guidelines suggest a series of criteria to assess if asthma is controlled, as displayed in table:

Table VI: Levels of asthma control (120).

Characteristic	Controlled (all of the following)	Party (any measure present in any week)	Uncontrolled
Daytime symptoms	None (twice or less/week)	More than twice/week	Three or more features of partly controlled asthma present in any week
Limitations of activities	None	Any	
Nocturnal symptoms/awakening	None	Any	
Need for reliever/ rescue treatment	None (twice or less/week)	More than twice/week	
Lung function (PEF or FEV1)	Normal	<80% predicted or personal best	
Exacerbations	None	One or more/year	One in any week

Asthma control tools are a useful aid in measuring a patient’s asthma status and are designed to support patient consultations. The Asthma Control Test is a patient-based tool developed and validated for identifying patients with poorly controlled asthma. The five-item test is designed to measure the patient’s level of asthma control during the preceding four weeks. Items relate to activity limitation, shortness of breath, nighttime symptoms, use of short-acting beta-2 agonists (SABAs) and a self-assessment of asthma control.

The overall score (the ‘Asthma Score’) out of 25 is given by the addition of the response to each item, with a score of 20–25 classed as ‘on target’ and a score of 19 or less as ‘off target’ (Figure 21). It has been demonstrated that the asthma score has a good predictive ability against outcomes related to asthma and also a good ability to detect risk factors. The responsiveness of the Asthma Control Test to changes in asthma control and lung function has also been reported (121).

The Asthma Score is a helpful screening tool to distinguish patients with good symptom control from patients with poor symptom control in an objective and feasible way (121).

In our study, we found that only 62 children (28.7%) had regular control of their asthma.

Asthma Control Test™ (ACT)

1. In the past 4 weeks, how much of the time did your asthma keep you from getting as much done at work, school or at home?

All of the time	1	Most of the time	2	Some of the time	3	A little of the time	4	None of the time	5
-----------------	----------	------------------	----------	------------------	----------	----------------------	----------	------------------	----------

Score

1. During the past 4 weeks, how often have you had shortness of breath?

More than once a day	1	Once a day	2	3 to 6 times a week	3	Once or twice a week	4	Not at all	5
----------------------	----------	------------	----------	---------------------	----------	----------------------	----------	------------	----------

1. During the past 4 weeks, how often did your asthma symptoms (wheezing, coughing, shortness of breath, chest tightness or pain) wake you up at night, or earlier than usual in the morning?

4 or more nights a week	1	2 or 3 nights a week	2	Once a week	3	Once or twice	4	Not at all	5
-------------------------	----------	----------------------	----------	-------------	----------	---------------	----------	------------	----------

1. During the past 4 weeks, how often have you used your rescue inhaler or nebulizer medication (such as salbutamol)?

3 or more times per day	1	1 or 2 times per day	2	2 or 3 times per week	3	Once a week or less	4	Not at all	5
-------------------------	----------	----------------------	----------	-----------------------	----------	---------------------	----------	------------	----------

1. How would you rate your asthma control during the past 4 weeks?

Not controlled at all	1	Poorly controlled	2	Somewhat controlled	3	Well controlled	4	Completely controlled	5
-----------------------	----------	-------------------	----------	---------------------	----------	-----------------	----------	-----------------------	----------

Patient Total Score

Figure 21: Asthma control test

XIV. Education:

Educational strategies for patients with asthma and their families are based on numerous factors, including patient age and the family situation and dynamics. Sufficient evidence exists to support the value of educating children and their families about self-management skills in improving asthma-related outcomes (122).

The guidelines describe the educational process as a partnership between the clinician, patient, and the caregivers. The clinician should make an assessment of the child's ability and receptivity to receive educational messages, information about the home environment and usual routine for care of the child, and the family's involvement in the patient's illness.

Education should be offered at multiple points of care. One of the important components of education relates to the use of inhalation devices. Inhalation therapy is a common method of treating asthma, and proper use of inhalation devices is essential to achieve the therapeutic benefit. Inhalation devices differ significantly in their directions for use, and patients require initial instruction and observation, as well as periodic follow-up, to ensure proper use (122).

1. Self-monitoring and Periodic Assessment: (123)

Periodic assessment by a health professional is essential for optimal asthma management. One important focus should be on the self-management skills used by the patients and their families to monitor asthma control and take actions when the asthma condition changes. All patients should receive a written asthma action plan that includes instructions about daily management and actions to take to manage worsening symptoms. Written action plans are especially important for patients with moderate or severe persistent asthma, patients whose asthma is poorly controlled, and those who have experienced a severe exacerbation. Written action plans can be based on peak flow results or recognition of symptoms because both approaches have been proved to be equally effective with no clear advantage of one over the other. Common strategies used for worsening symptoms include increased use of

rescue therapies, initiating a short course of oral corticosteroids, and early action to seek medical care.

Table VII: Key educational messages (123).

Basic Asthma Facts	Contrast airways in asthma from normal airways Signs and symptoms of acute worsening
Role of Medications	Differentiate long-term control and quick-relief medications
Patient Skills	Correct use of medications: adherence and optimal inhalation technique Practicing effective trigger avoidance Self-monitoring: peak flow or symptom based Utilizing a written asthma action plan Seeking medical care when appropriate

2. Environmental Control and Avoidance:

Control of environmental factors that may be asthma triggers as well as effective management of comorbid conditions are important strategies of management. Potential allergen exposure should be evaluated during the medical history and possibly confirmed by skin testing. It is clear that a multifaceted, comprehensive approach is required for effective allergen management (124).

Common strategies to control exposure to allergens and irritants the recent asthma guidelines also include a comprehensive list of co-morbid conditions that can disrupt asthma control. Recognition and effective management of these conditions can contribute to improved asthma control.

In addition, patients with asthma should receive the influenza vaccine annually (124).

In our study, only 28.7% of patients was educated about their disease, and had a regular follow up.

XV. Evolution:

The long-term prognosis of asthma remains relatively unknown as there are few prospective studies from childhood to adulthood.

The Blair Study of 200 asthmatic children showed that two-thirds of children with mild asthma had an excellent state twenty years later: life is normal, their attacks are absent or rare and very sensitive to bronchodilators (125). In contrast, two-thirds of severe asthma remain so 20 years later. However, this study is mainly clinical.

The study of Gerritsen of 101 children evaluated clinically and functionally shows that 43% of children remain symptomatic in adulthood and more than half of asthmatics clinically heal. This study confirms that it is the moderate asthmatics who are most likely to be dumb as adults and that is around the age of 15 years (126).

The Study of Melbourne group involved more than 300 asthmatic children followed clinically and functionally from infancy to the age of 21 and 28 years, showed that three quarters of mild, moderate asthma at the age of 14 years are in this category to 28 years and a quarter of them worsened (126). The asthmatic child deserves a prolonged illness surveillance and regular monitoring. This monitoring should be clinical and pulmonary function.

Finally, it is important to note that the restoration and maintenance of respiratory function of children protect the adult respiratory capital.

We found in our study, that 185 children (85.6%) presented an improvement and received ambulatory treatment, 27 (12.5%) had been hospitalized on pediatric service A and only 4 (1.9%) was admitted in ICU.

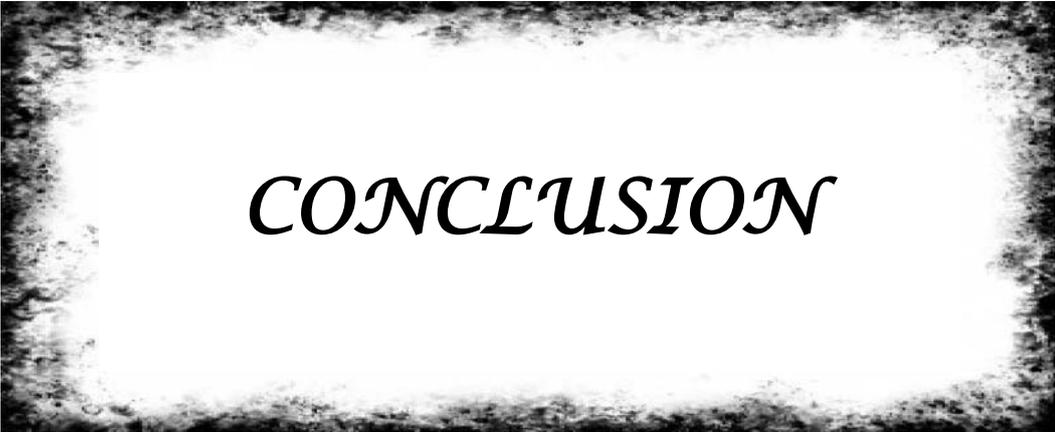
XVI. Prevention

Asthma prevention can be divided into primary (preventing onset of established risk factors), secondary (preventing development of asthma once established risk factors have developed) and tertiary prevention (care of established asthma and preventing exacerbations).

- Potential environmental factors in asthma are supported by marked geographic and temporal variation in asthma prevalence. Initial studies of environmental manipulation were promising, with the House Dust Mite (HDM) and food allergen avoidance measures in the first 12 months of life resulting in decreased sensitization and asthma diagnosis persisting until the age of 8 years in a high-risk birth cohort in the Isle of Wight (127). Subsequent larger, multifaceted studies in Canada and Australia have failed to reproduce these results. The Canadian Childhood Asthma Primary Prevention Study showed a benefit in asthma symptoms but not bronchial hyper-responsiveness at 7 years, whereas the Australian Childhood Asthma Prevention Study failed to show any clinical benefit at 5 years despite a 61% reduction in HDM and successful dietary manipulation (128).
- The Early Treatment of the Atopic Child study reported no overall benefit of prolonged cetirizine (H1 receptor antagonist) treatment in infants who had atopic dermatitis at 18-month post treatment follow-up. Benefit was seen only in sensitized subgroups, persisting to 36 months in grass pollen-sensitized infants but only transiently in those sensitized to HDM.

A subsequent study, the Early Prevention of Asthma in Atopic Children study specifically targeted these subgroups but failed to show any benefit (128).

- The rationale for tertiary prevention studies is the observation that a large number of persistent asthma cases start early in life, but the three trials conducted to date, at differing ages or stages of “asthma development,” have had disappointing results. Intermittent courses of ICS and maintenance ICS, for varying durations, in infants who had recurrent wheeze demonstrated no difference in asthma prevalence (129–131).
- Vaccination in asthma may prevent exacerbations and serious complications such as pneumonia.



CONCLUSION

Asthma is a chronic inflammatory disease that is commonly encountered in the emergency department. Early signs of worsening asthma should be recognized and immediate treatment given. B-Agonists, anticholinergics, and corticosteroids are mainstays of treatment of the asthma exacerbation.

In light of our results, it is clear that there was no specific protocol for the management of asthma attack in our pediatric emergency department.

Indeed the antibiotic was used in abused way in more than half (58.8%) of our patients, followed by the oral corticosteroids in 50.5% of cases.

We found also that the majority of our patients (71.5%) had no regular follow of their asthma because of a lack in education of children and their families about the disease.

We speculated that a local asthma educational programme intended to reduce the number of emergency department visits for asthma exacerbations should consider the inclusion of an explanation and discussion about the chronic nature of the disease and the importance of the long-term administration of “asthma controllers” medications, even in asymptomatic periods.

Further prospective studies are needed to corroborate this hypothesis.



APPENDICE

- Refus de biberon trouble de conscience Sueur
- Durée de la crise :
 - Notion de traitement antérieur : Oui NON
- Type : antihistaminique salbutamol corticoïde oral corticoïde inhalé

L'examen Clinique :

- FR : FC : T° : SaO₂ :
- Thorax distendu oui non
- Râles sibilants oui non
- Râles ronflants oui non
- emphysème sous cutané oui non
- Murmure vésiculaire :
Normale diminué abolie

Paraclinique :

- NFS : CRP :
- Rx thorax :
- Distension thoracique hyper clarté
- Foyer infectieux complications

Traitement :

- O₂ : oui non
- Bronchodilatateur :
 - ✓ B₂ mimétiques :
Salbutamol : -injectable oui non
-nébulisation oui non : nombre.....

 - Terbutaline : -injectable oui non
-nébulisation oui non : nombre.....
- Corticothérapie :
 - injectable oui non moléculeDose.....
 - per os oui non moléculeDose.....
- Hydratation IV: Oui Non
- ATB : Oui Non
Moléculedose indications
- Autres traitements :
.....

Evolution :

- Amélioration (traitement en ambulatoire) Oui Non
- Hospitalisation Oui Non
- Transfert en réanimation Oui Non

Observation :

.....



Abstract

Asthma is the most common chronic disease in childhood. The morbidity and asthma mortality have become a problem in recent years, the most often due to inadequate therapeutic management.

The work described in this study was directed at learning more about how asthma is managed in the community, and steps that can be taken to improve the management of these conditions. For this goal, we performed a prospective study with 216 patients; the population studied included children between the ages of 2 and 15 who presented to ED in The Mother-Child Hospital (UH Mohamed VI) Marrakech over one year from January to December 2014.

We found that The majority of the patient (83.8%) had already developed an asthma attack The personal and family history of asthma and /or atopy are present in more than half of the patients (57%). We also found that the allergens were the main precipitating factors (94%). The reason for consultation was cough in 94% of children.

In Clinical examination, all patients had wheezes (100%) followed by Polypnea (63.4%).

The treatment of choice was nebulization of salbutamol used for (96.3%) of children, followed by injection Corticotherapy (56%) and oral corticotherapy (50%). Antibiotics were prescribed for 127 children (58.8%). In the result of this management 85.6% of patients presented an improvement and received ambulatory treatment.

Childhood asthma remains under diagnosed and subcontracted it must be evoked as soon as the respiratory symptoms are repeated. In addition to the thoracic radiography which must be required systematically, the respiratory functional exploration not only contributes to the control of asthma but also for the evaluation of the effectiveness of the treatment prescribed, without forgets education during each consultation.

Adhering to asthma regimes implies not only taking medications to relieve asthma attacks, but also adjusting their life styles in order to prevent asthma attacks.

Resumé

L'asthme est la maladie inflammatoire chronique la plus fréquente dans la population pédiatrique. La morbidité et la mortalité de l'asthme chez l'enfant sont devenues préoccupantes ces dernières années, le plus souvent dues à une prise en charge thérapeutique inadéquate.

La présente étude a été effectuée pour évaluer la prise en charge de l'asthme dans la communauté et les mesures qui peuvent être prises pour améliorer cette prise en charge. Pour ce but, nous avons réalisé une étude prospective incluant 216 patients; la population étudiée comprenait des enfants entre les âges de 2 et 15 ans qui se sont présentés aux urgences pédiatriques de l'Hôpital Mère-Enfant du CHU Mohamed VI de Marrakech durant l'année 2014.

Nous avons constaté que la majorité des patients (83,8%) avaient déjà développé une crise d'asthme. L'histoire personnelle et familiale de l'asthme et / ou d'atopie était présente dans plus que la moitié des enfants (57%). Nous avons constaté également que les allergènes étaient les principaux facteurs déclenchants (94%). Le motif de consultation était la toux chez 104 enfants, la dyspnée présentée par 84 enfants ou les deux. La dyspnée et la toux ont été souvent associées avec le diagnostic de l'asthme. A l'examen clinique, tous les patients avaient des râles sibilants (100%) et de la polypnée (63,4%). Le traitement de choix était la nébulisation de salbutamol utilisé pour 96,3% des enfants, suivie par la corticothérapie injectable (56%) puis orale (50%). Les antibiotiques ont été prescrits chez 127 enfants (58,8%). Sur le plan évolutif, 85,6% des patients avaient présenté une amélioration et avaient reçu un traitement ambulatoire.

L'asthme de l'enfant reste sous-diagnostiquée et sous-traitée, le diagnostic de l'asthme de l'enfant est essentiellement clinique, il doit être évoqué dès que des signes respiratoires se répètent. Outre, la radiographie thoracique qui doit être demandée systématiquement, les explorations fonctionnelles respiratoires aident au contrôle de l'asthme, et l'évaluation de l'efficacité du traitement prescrit sans omettre l'éducation lors de chaque consultation.

Il faut non seulement prendre des médicaments pour soulager les crises d'asthme, mais également ajuster leur mode de vie afin de les prévenir.

ملخص

الربو هو المرض الالتهابي الاكثر شيوعا بين الاطفال, أصبحت معدلات الاعتلال والوفيات من الربو عند الأطفال مشكلة في السنوات الأخيرة، في معظم الأحيان بسبب عدم كفاية الإدارة العلاجية. وقد أجريت هذه الدراسة بهدف تقييم كيفية معالجة الربو في المجتمع، والتدابير التي يمكن اتخاذها لتحسين الرعاية. ولهذه الغاية، أجرينا دراسة استطلاعية تشمل 216 مريضاً من الأطفال الذين تتراوح أعمارهم ما بين 2 و 15 عاماً الذين تقدموا إلى قسم الطوارئ للأطفال بمستشفى الأم والطفل محمد السادس في مركش عام 2014.

لقد وجدنا ان 83,8% من لاطفال قد تعرضوا مسبقا الى نوبة الربو, كان التاريخ الشخصي والعائلي للربو و / أو الحساسية موجودة في أكثر من نصف الأطفال (57%). كما وجدنا أن المواد المسببة للحساسية كانت من المسببات الرئيسية (94%) لهذه النوبات. وكان سبب الزيارات الطبية اما السعال في 104 طفلا، او ضيق التنفس قدم من قبل 84 طفلا أو كليهما وغالبا ما يرتبط ضيق التنفس والسعال مع تشخيص الربو. اما بالنسبة للفحص السريري فقد كان جميع الاطفال يعانون من الصفير عند التنفس بنسبة (100%) و التنفس السريع لذا 63.4% تحسنت حالة 85.6% من المرضى، و تلقوا العلاج في العيادات الخارجية.

الربو عند الاطفال لا ازال تشخيصه و علاجه غير كافيين,نوبات ضيق التنفس والصفير تسهل التشخيص باستثناء الفحص بالاشعة السينية للصدرالتي تعد ضرورية هناك الفحص لوظائف الرئة الذي يفيد في السيطرة على الربو مع الاسرارعلى التوعية عند كل فحص. لا يجب علينا فقط تناول الدواء لتخفيف نوبات الربو لذا الاطفال، ولكن يجب أيضا ضبط نمط حياتهم لمنع تكرار هذه النوبات.



BIBLIOGRAPHY

1. **Kupczyk M, Haahtela T, Cruz AA, Kuna P.**
Reduction of asthma burden is possible.
National Asthma Plans. Allergy 2010; Apr;65(4):415–9.
2. **Aichane A**
Asthme infantile: l'importance d'un diagnostic précoce et précis.
Rev Docti News. fév 2011;16:50.
3. **Paul D. Robinson et al**
Asthma in Childhood
Ped Clin Nor Am, 2013; Feb 56(1): 191–226.
4. **Andrew Bush, MD and Sejal Saglani.**
Management of severe asthma in children.
Lancet. 2010 Sep 4; 376(9743): 814–825.
5. **Global Initiative for Asthma.**
Global strategy for asthma management and prevention.
GINA; 2014; May 6(1):16–25.
6. **Busse WW, et al.**
Expert Panel Report Three. Guidelines for the diagnosis and management of asthma.
National Institutes of Health. 2011;12:160.17.
7. **Cohn L, Elias JA, Chupp GL.**
Asthma: mechanisms of disease persistence and progression.
Annu Rev Immunol 2004;22:789–815.
8. **Roberts CR, Okazawa M, Wiggs B, et al.**
Airway wall thickening. In: Barnes PJ,
Grunstein MM, Leff AR, et al, editors. Asthma.
Philadelphia: Raven Publishers;1997. p. 925–35.
9. **Homer RJ, Elias JA.**
Consequences of long-term inflammation. Airway remodeling.
Clin Chest Med. 2000;21:331–43.
10. **Krouse JH, Brown RW, Fineman SM, et al.**
Asthma and the unified airway.
OtolaryngolHead Neck Surg. 2007;136:S75–106.

11. **Pohunek P, Warner JO, Turzi´kova´ J, et al.**
Markers of eosinophilic inflammation and tissue re-modelling in children before clinically diagnosed bronchial asthma.
Pediatr Allergy Immunol. 2005;16:43-5.
12. **van Den Toorn LM, Prins JB, Overbeek SE, et al.**
Adolescents in clinical remission of atopic asthma have elevated exhaled nitric oxide levels and bronchial hyperresponsiveness.
Am J Respir Crit Care Med. 2000;162:953-7.
13. **van den Toorn LM, Overbeek SE, Prins JB, et al.**
Asthma remission: does it exist?
Curr Opin Pulm Med 2003;9:15-20.
14. **Busse WW, Lemanske RF.**
Asthma.
N Engl J Med 2001;344:350-62.
15. **Stevenson DD, Szczeklik A.**
Clinical and pathologic perspectives on aspirin sensitivity and asthma.
J Allergy Clin Immunol 2006;118:773-86 [quiz: 787-8].
16. **Vally H, Taylor ML, Thompson PJ.**
The prevalence of aspirin intolerant asthma(AIA) in Australian asthmatic patients.
Thorax. 2002;57:569-74.
17. **Saye SV, Kong FJ et al.**
Relation of exposure to airway irritants infancy to prevalence of bronchial hyperresponsiveness school children.
Lancet 1995; 345: 217- 220.
18. **The International Study of Asthma and Allergies in Childhood (ISAAC).**
Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema Steering Committee.
Lancet 2003;351:1225-32.
19. **Tokkyyama K, Shigeta M, Maeda S**
Diurnal variations of Peak in asthmatic children after 10 years.
Pediatr Pulmal 1996; 2: 141-146.

20. **Aniba J.**
Prévalence de l'asthme chez l'enfant dans la région de Safi.
Thèse Doctorat Médecine, Marrakech ; 2011, n° 41, 53 pages.
21. **Albertini M, Bourrier T, Chiche V et al.**
Les décès par asthme chez l'enfant.
Arch Pediatr 1994 ; 1 :333-336.
22. **Sten-Erik Bergströma et al.**
Asthma mortality among Swedish children and young adults, a 10-year study
Respiratory Medicine 2008 ; 102 : 1335-134.
23. **Buffum WP.**
The prognosis of asthma in infancy.
Pediatrics 1999; 32: 453-457.
24. **Park ES, Golding J, Carswell F et al**
Preschool wheezing and prognosis at 10.
Arch Dis Child 2006; 51: 642- 646.
25. **Bousquet J, Charnez P**
Eosinophilic inflammation in asthma.
N Eng J Med 1990; 323:1033.
26. **Fuhrman C, Delacour C, Blic J, Dubus C, et al**
The hospitalizations for childhood asthma exacerbation.
j.arcped. 2009 ;11 :0-30.
27. **British Thoracic Society Scottish Intercollegiate Guidelines Network.**
British Guideline on the management of asthma.
2008 (www.brit-thoracic.org.uk).
28. **Priftanji A, Strachan D, Burr M, et al.**
Asthma and allergy in Albania and the UK.
Lancet 2001;358:1426-7.
29. **Lau S, Illi S, Sommerfeld C, et al.**
Early exposure to house-dust mite and cat allergens and development of childhood asthma: a cohort study. Multicentre Allergy Study Group.
Lancet 2000;356:1392-7.

30. **Sears MR, Herbison GP, Holdaway MD, et al.**
The relative risks of sensitivity to grass pollen, house dust mite and cat dander in the development of childhood asthma.
Clin Exp Allergy 1989;19:419-24.
31. **Strachan DP, Cook DG.**
Health effects of passive smoking. 6. Parental smoking and childhood asthma: longitudinal and case-control studies.
Thorax 1998;53: 204-12.
32. **Strachan DP, Butland BK, Anderson HR.**
Incidence and prognosis of asthma and wheezing illness from early childhood to age 33 in a national British cohort.
BMJ. 1996;312:1195-9.
33. **Patel MM, Miller RL.**
Air pollution and childhood asthma: recent advances and future directions.
Curr Opin Pediatr 2009;21:235-42.
34. **Corne JM, Marshall C, Smith S, et al.**
Frequency, severity, and duration of rhinovirus infections in asthmatic and non-asthmatic individuals: a longitudinal cohort study.
Lancet 2002;359:831-4.
35. **Von Mutius E, Martinez FD, Fritzsche C, et al.**
Prevalence of asthma and atopy in two areas of West and East Germany.
Am J Respir Crit Care Med 1994;149: 358-64.
36. **Von Mutius E.**
Infection: friend or foe in the development of atopy and asthma? The epidemiological evidence.
Eur Respir J 2001;18:872-81.
37. **Eder W, Ege MJ, von Mutius E.**
The asthma epidemic.
N Engl J Med 2006;355: 2226-35.
38. **Braun-Fahrländer C, Lauener R.**
Farming and protective agents against allergy and asthma.
Clin Exp Allergy 2003;33:409-1.

39. **Van Strien RT, Engel R, Holst O, et al.**
Microbial exposure of rural school children, as assessed by levels of N-acetyl-muramic acid in mattress dust, and its association with respiratory health.
J Allergy Clin Immunol 2004;113:860-7.
40. **Gerristen J, Koeter GH, Monchy JGR, et al.**
Change airway responsiveness to inhaled house dust from childhood to adulthood.
J Allergy Clin Immunol, 1990; 85: 1083-1089.
41. **Sears MR, Justina MB, Green M, Andrew R, et al.**
Population based cohort study of childhood asthma followed to adulthood.
N Eng Med 2003; 349: 1414-1422.
42. **Michael J. Parker, MD.**
Asthma.
Otolaryngol Clin N Am. 2011 ; 44 :667-684.
43. **Moffatt MF, Gut IG, Demenais F, Strachan DP, Bouzigon E, Heath S, et al.**
Large-scale, consortium-based genomewide association study of asthma.
N Engl J Med 2010;363:1211-21.
44. **The European community respiratory health survey group**
Genes for asthma. An analysis of the European community respiratory health survey.
Am J Resp City Care Med 1997: 1773-1780.
45. **Piacentini S, A Verrotti, Polimanti R, et al.**
Polymorphismes fonctionnels des gènes GSTA1 et GSTO2 associés à l'asthme chez les enfants asthmatiques en Italie.
Clin Exp Pharmacol Physiol. 2014 Mar;41(3):180-4.
46. **Thomas AO, Lemanske RF Jr, Jackson DJ.**
Infections and their role in childhood asthma inception.
Pediatr Allergy Immunol 2014;25:122-8.
47. **Mackenzie KJ, Anderton SM, Schwarze J.**
Viral respiratory tract infections and asthma in early life; cause and effect?
Clin Exp Allergy 2013;44(1):9-19.
48. **Murray M, Webb MSC, O'Callagan C et al.**
Respiratory status and allergy after bronchiolitis.
Arch Dis Child 1992; 67:482.

49. **Korppi M, Kotaniemi-Syrjanen A, Waris M, Vainionpaa R, Reijonen TM.**
Rhinovirus-associated wheezing in infancy: comparison with respiratory syncytial virus bronchiolitis.
Pediatr Infect Dis J 2004;23:995-9.
50. **Hansel TT, Johnston SL, Openshaw PJ.**
Microbes and mucosal immune responses in asthma.
Lancet 2013;381:861-73.
51. **De Schutter I, Dreesman A, Soetens O, De Waele M, Crokaert F, Verhaegen J, et al.**
In young children, persistent wheezing is associated with bronchial bacterial infection: a retrospective analysis.
BMC Pediatr 2012;12:83.
52. **Sulakvelidze I, Inman MD, Rerecich T, O'Byrne PM.**
Increases in airway eosinophils and interleukin-5 with minimal bronchoconstriction during repeated low-dose allergen challenge in atopic asthmatics.
Eur Respir J. 1998;11:821-27.
53. **Almqvist C, Wickman M, Perfetti L, et al.**
Worsening of asthma in children allergic to cats, after indirect exposure to cat at school.
Am J Respir Crit Care Med. 2001;163:694-98.
54. **Sheikh A, Hurwitz B, Shehata Y.**
House dust mite avoidance measures for perennial allergic rhinitis.
Cochrane Database Syst Rev. 2007;1:CD001563.
55. **Platts-Mills TA.**
Allergen avoidance in the treatment of asthma: problems with the meta-analyses.
J Allergy Clin Immunol. 2008;122:694-96.
56. **Platts mills TA, Tovey ER, Mitchell EB, et al.**
Reduction of bronchial hyperreactivity during prolonged allergen avoidance.
Lancet 1999; 2:675-8.
57. **Boner AL, Niero E, Antonili I, et al.**
Pulmonary function and bronchial hyperreactivity in asthmatic children with house dust mite.
allergy during prolonged stay in the Italian alps (Misurina, 1756 m).
Ann Allergy 1999; 54: 42-5.

58. **Gruchalla RS, Pongracic J, Plaut M, et al.**
Inner City Asthma Study: relationships among sensitivity, allergen exposure, and asthma morbidity.
J Allergy Clin Immunol.2005;115:478-85.
59. **Goldenhersh MJ, Ament M.**
Asthma and gastroesophageal reflux in infants and children.
Immunol Allergy Clin N Am 2001;21:439-48.
60. **Jung AD.**
Gastroesophageal reflux in infants and children.
Am Fam Phys 2001;64:1853-60.
61. **Harding S, Guzzol MR, Richter JE.**
24 h esophageal pH testing in asthmatics.
Chest 1999;115:654-9.
62. **Harding SM, Guzzo MR, Richter JE.**
The prevalence of gastroesophageal reflux in asthma patients without reflux symptoms.
Am J Resp Crit Care Med 2000;162:34-9.
63. **Human Services, Centers U.S.**
Department of Health and for Disease Control,
Office on Smoking and Health, 2006.
64. **Royal College of Physicians.**
Passive smoking and children. In: A Report of the Tobacco Advisory Group of the Royal College of Physicians.
Cambrian Printers Ltd, London, UK 2010.
65. **Kanoh, M., Kaneita, Y., Hara, M., Harada, S., Gon, Y., Kanamaru, H., Ohida,**
parental smoking habits and development of asthma in early childhood.
Prev. Med. 54, 94-96. 2012.
66. **Committee on Substance Abuse.**
American Academy of Pediatrics: tobacco's toll: implications for the pediatrician.
Pediatrics 107, 794-798. 2001.

67. **Leung, G.M., Ho, L.M., Lam, T.H.**
Second hand smoke exposure, smoking hygiene, and hospitalization in the first 18 months Of life.
Arch Pediatr. Adolesc Med. 2004. 158, 687-693.
68. **Li F1, Zhou YC, et al.**
Environmental risk factor assessment: a multilevel analysis of childhood asthma in China.
World J Pediatr. 2013 May;9(2):120-6.
69. **Nilson L, Castor O, Lofman O, et al.**
Allergic disease in teenagers in relation to urban and rural residence at various stages of childhood.
Allergy 1999; 54: 716-721.
70. **Wuthrich B.**
Epidemiology and natural history of asthma.
Allergy Clin Immunol Inter 1996; 8: 77-82.
71. **P. Panznera, I. Malkusová, M. Vachová, M. Liskaa.**
Bronchial inflammation in seasonal allergic rhinitis with or without asthma in relation to natural exposure to pollen allergens
Allergol Immunopathol. 2015 Jan-Feb;43(1):3-9.
72. **González-Díaz SN, Pietropaolo-Cienfuegos DR, et al**
Factors associated with allergic rhinitis in children and adolescents in northern Mexico
Allergy. 2008 Dec;63 Suppl 89:1-20.
73. **Abina Illi, Erika von Mutiuset al**
The natural course of atopic dermatitis from birth to age 7 years and the association with asthma.
Journal of Allergy and Clinical Immunology, 2004 ;113 (5) : 925-93.
74. **Christopher L. Carroll, MD, MSa,b, Kathleen A. Sala.**
Pediatric Status Asthmaticus.
Crit Care Clin. 2013 Apr;29(2):153-66.
75. **Muriel Koninckx et al.**
Management of status asthmaticus in children
Paediatric Respiratory Reviews, 2013 June;(14)2 :78-85.

76. **De Blic J.**
Asthma in children and infants.
Rev Mal Resp. 2005 Av;22(2):19-2.
77. **Anderson SD.**
Issues in exercise induced asthma.
J Allergy Clin Immunol 1999; 76: 763-772.
78. **Arsalane G, Mahraoui C.**
L'asthme et le sport en Milieu scolaire: Enquête auprès des enseignants et lycéens de Rabat.
Hopital d'enfants, Rabat, Maroc.
Arch Péd. June 2010 ; 176-186.
79. **Lobowitz MD, Barber R, Burrows F**
Family concordance of IgE, atopy and disease.
J Allergy Clin Immunol 2000; 73: 259-64.
80. **Papadopoulos et al.**
International consensus on (ICON) pediatric asthma pediatric asthma.
Allergy. 2012; 67: 976-997.
81. **Leroux P, Scheinmann P.**
Infections and asthma crisis.
Pediatr (Rio J). 2001 Mar-Apr;77(2):65-6.
82. **Lebourgeois M.**
Evolution de l'asthme.
Rev Mal Resp. 1992; p : 285-287.
83. **P. Charlier a,b, A.L. Naneix a, L. Brun c, J.C. Alvarez d, G.**
Asthma-related sudden death: Clinicopathological features in 14 cases.
La Revue de Médecine Légale, Sept 2012 ; (3) :115-119.
84. **M, Abu-Hasan M.**
Pseudo-asthma: when cough, wheezing, and dyspnea are not asthma.
Pediatrics 2007;120:855-7.
85. **C.E. Brightling, P. Bradding, F.A. Symon, S.T. Holgate, A.J. Wardlaw, I.D.**
Pavord, Mast-cell infiltration of airway smooth muscle in asthma.
N. Engl. J. Med. 346 (2002) 1699-1705.

- 86. NAEPP, National Asthma Education and Prevention Program.**
Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma, – National Institutes of Health (NIH) – National Heart, Lung, and Blood Institute. NIH, Publication. 2013–2014.
- 87. Al-Said A. Haffor a, Magdi Ismaeel.**
A simple, reliable quantitative score for grading chest X-ray in adult asthma. Egyptian Journal of Chest Diseases and Tuberculosis (2014) 63, 789–797.
- 88. Kaliner M, Lemanske R.**
Rhinitis and asthma.
J Am Med Assoc 1992;268:2807–29.
- 89. Bacharier LB, Strunk RC, Mauger D, White D, Lemanske RF Jr, Sorkness CA.**
Classifying asthma severity in children: mismatch between symptoms, medication use, and lung function.
Am J Respir Crit Care Med 2004;170:426–432.
- 90. Bisgaard H, Nielsen KG.**
Plethysmographic measurements of specific airway resistance in young children.
Chest 2005;128:355–362.
- .
- 91. Frey et al.**
Forced oscillation technique in infants and young children.
Paediatr Respir Rev 2005;6:246–254.
- .
- 92. Global strategy for asthma management and prevention.**
Who/Nhlp workshop report in: National institute of health national heart, lung and blood institute.
Publication Number 95–3659, 1995.
- 93. National Asthma Education and Prevention Program.**
Expert panel report III: guidelines for the diagnosis and management of asthma.
Bethesda (MD):National Heart, Lung, and Blood Institute; 2007 (NIH publication n^o 08–4051).
- 94. Newman KB, Milne S, Hamilton C, et al.**
A comparison of albuterol administered by metered-dose inhaler and spacer with albuterol by nebulizer presenting to an urban emergency department with acute asthma.
Chest 2002; 121(4):1036–41.

95. **Kimberly A. Callahan RN et al.**
Peak Flow Monitoring in Pediatric Asthma Management.
Journal of Pediatric Nursing. 2010;25:12-17.
96. **Davies A, Thomson G, Walker J, et al.**
A review of the risks and disease transmission associated with aerosol generating medical procedures.
J Infect Prev. 2009;10(4):122-6.
97. **Plotnick L, Ducharme F.**
Combined inhaled anticholinergics and beta2-agonists for initial treatment of acute asthma in children
Cochrane Database Syst Rev 2008.
98. **Hendeles L, Sherman J.**
Are inhaled corticosteroids effective for acute exacerbations of asthma in children?
J Pediatr. 2003;142:S26-32.
99. **Hendeles L.**
Selecting a systemic corticosteroid for acute asthma in young children.
J Pediatr 2003;142:S40-4.
100. **Van Asperen PP, Mellis CM, Sly PD.**
The role of corticosteroids in the management of childhood asthma.
Med J Aust 2002;176:168-73.
101. **Altamimi S, Robertson G, Jastaniah W, et al.**
Single-dose oral dexamethasone in the emergency management of children with exacerbations of mild to moderate asthma.
Pediatr Emerg Care 2006;22:786-93.
102. **Cheuk DK, Chau TC, Lee SL.**
A meta-analysis on intravenous magnesium sulphate for treating acute asthma.
Arch Dis Child 2005;90:74-7.
103. **Blitz M, Blitz S, Beasley R, et al.**
Inhaled magnesium sulfate in the treatment of acute asthma.
Cochrane Database Syst Rev. 2005;4:38-98.

- 104. Kress JP, Noth I, Gehlbach BK, et al.**
The utility of albuterol nebulized with heliox during acute asthma exacerbations.
Am J Respir Crit Care Med 2002;165(9):1317-21.
- 105. Kim IK, Phrampus E, Venkataraman et al.**
Helium/oxygen-driven albuterol nebulization in the treatment of children with moderate to severe asthma exacerbations:a randomized, controlled trial.
Pediatrics. 2005;116(5):1127-33.
- 106. Ostrom NK, Decotiis BA, Lincourt WR, et al.**
Comparative safety and efficacy of low-dose fluticasone propionate and montelukast in children with persistent asthma.
J Pediatr. 2005;147(2):213-20.
- 107. Silveira DR, Piva JP, Jose Cauduro MP, et al.**
Early administration of two intravenous bolus of aminophylline added to the standard treatment of children with acute asthma.
Respir Med 2008;102:156-6.
- 108. Francesco Blasi a, Sebastian L. Johnston b.**
The role of antibiotics in asthma.
International Journal of Antimicrobial Agents. 2007;29:485-493.
- 109. Bhogal SK, McGillivray D, Bourbeau J, et al.**
Early administration of systemic corticosteroids reduces hospital admission rates for children with moderate and severe asthma exacerbation.
Ann Emerg Med. 2012;60:84-91.
- 110. Warner JO, Naspitz CK.**
Third international pediatric consensus statement on the management of childhood asthma: international pediatric asthma consensus group.
Pediatr Pulmonol. 1998;25:1-7.
- 111. Baker JW, Yerger S, Segar WE.**
Elevated plasma antidiuretic hormone levels in status asthmaticus.
Mayo Clin Proc. 1976;51:31-4.

112. **Christopher L. Carroll, MD, MSa,b, Kathleen A. Sala, MPH.**
Pediatric Status Asthmaticus.
Crit Care Clin. 29 (2013) 153–166.
113. **Broek I, Harris N, Henkens M.**
Guide Clinique et thérapeutique.
Maladies Respiratoires: Asthme 2010; p : 74–77.
114. **Bateman ED, Boushey HA, Bousquet J, et al.**
Can guideline defined asthma control be archived? The gaining optimal asthma control study.
Am J Respir Crit Care med 2004; 170:836–44.
115. **Kuehni CE, Frey U.**
Age- related differences in perceived asthma control in childhood: Guidelines and reality.
Eur Resp J 2002; 20:880–9.
116. **Blanc FX, BLIC J, Scheinmann P.**
Etude AIRE : analyse des données recueillies chez 753 enfants asthmatiques en Europe.
Rev Mal Resp 2002 ; 19 :585–92.
117. **Gaga M, Papageorgiou N, Zervas E, et al.**
Control of asthma under specialistcare: is it achieved?
Chest. 2005; 128(1): 78–84.
118. **Bateman ED, Hurd SS, Barnes PJ, Bousquet J et al.**
Global strategy for asthma management and prevention: GINA executive summary.
Eur Respir J. 2008 Jan;31(1):143–78.
119. **Humbert M, Holgate S, Boulet LP, et al.**
Asthma control or severity: that is the question.
Allergy. 2007; 62(2): 95–101.
120. **Schatz M, Sorkness CA, Li JT, et al.**
Asthma Control Test: reliability, validity, and responsiveness in patients not previously followed by asthma specialists.
J Allergy Clin Immunol. 2006; 117(3): 549–556.
121. **Butz, A., Pham, L., Lewis, L., Lewis, C., Hill, K., & Walker, J., et al.**
Rural children with asthma: Impact of a parent and child asthma education program.
J Pediatr Health Care. 2008; 22(6): 343–350.

122. **Dennis M. Williams.**
Pharm D Management of Pediatric Asthma.
J Pediatr Health Care. (2009). 23, 357–368.
123. **Morgan, W. J., Crain, E. F., Gruchalla, R. S., O'Connor, et al.**
The Head-off Environmental Asthma in Louisiana.
N Engl J Med. 2004 ;351 :1068–1080.
124. **Blair H.**
Natural history of childhood asthma: 20 years follow up.
Arch Dis Child. 1977; 52:613–619.
125. **Roorda RJ, Gerritsen J, Vanaaldren, et al.**
Risk factors for persistence of respiratory symptoms in childhood asthma.
Am Rev Respir Dis 1993; 148:1490–1495.
126. **Arshad SH, Bateman B, Sadeghnejad A, et al.**
Prevention of allergic disease during childhood by allergen avoidance: the Isle of Wight prevention study.
J Allergy Clin Immunol 2007;119:307–13.
127. **Marks GB, Mahrshahi S, Kemp AS, et al.**
Prevention of asthma during the first 5 years of life: a randomized controlled trial.
J Allergy Clin Immunol 2006;118: 53–6.
128. **Yunginger JW, Reed CE, O'Connell EJ, et al.**
A community-based study of the epidemiology of asthma.
Am Rev Respir Dis. 1992;146:888–94.
129. **Bisgaard H, Hermansen MN, Loland L, et al.**
Intermittent inhaled corticosteroids in infants with episodic wheezing.
N Engl J Med. 2006;354:1998–2005.
130. **Murray CS, Woodcock A, Langley SJ, et al.**
Secondary prevention of asthma by the use of Inhaled Fluticasone propionate in Wheezy infants (IFWIN): double-blind, randomised, controlled study.
Lancet 2006;368:754–62.
131. **Long-term effects of budesonide or nedocromil in children with asthma.**
The Childhood Asthma Management Program Research Group.
N Engl J Med. 2000;343:1054–63.

قسم الطبيب

أقسم بالله العظيم

أن أراقب الله في مهنتي.

وأن أصون حياة الإنسان في كافة أطوارها في كل الظروف والأحوال باذلاً
وسعي في استنقاذها من الهلاك والمرض
والألم والقلق.

وأن أحفظ للناس كرامتهم، وأستر عورتهم، وأكتم سرهم.

وأن أكون على الدوام من وسائل رحمة الله، باذلاً رعايتي الطبية للقريب
والبعيد، للصالح والطالح، والصديق والعدو.

وأن أثابر على طلب العلم، أسخره لنفع الإنسان .. لا لأذاه.

وأن أوقر من علمني، وأعلم من يصغرني، وأكون أخاً لكل زميل في المهنة
الطبية

متعاونين على البر والتقوى.

وأن تكون حياتي مصداق إيماني في سرّي وعلانيّتي، نقيّة مما يشينها تجاه

الله ورسوله والمؤمنين.

والله على ما أقول شهيد



جامعة القاضي عياض
كلية الطب و الصيدلة
مراكش

سنة 2015

أطروحة رقم 73

تدبير الربو لدى الأطفال في قسم المستعجلات

الأطروحة

قدمت ونوقشت علانية يوم 2015/05/26

من طرف

السيدة شيماء كودري

المزداة في 15 يوليوز 1987 بالدار البيضاء

لنيل شهادة الدكتوراه في الطب

الكلمات الأساسية :

الربو - الطفل - مستعجلات - تدبير

اللجنة

الرئيس

السيد س. يونس

أستاذ في الانعاش والتخدير

المشرف

السيد م. بو الروس

أستاذ مبرز في طب الأطفال

السيدة غ. ضريس

أستاذة مبرزة في طب الأطفال

الحكام

السيدة ل. عمرو

أستاذة مبرزة في أمراض الرئة وطب السل