Introduction

Asthma affects an estimated 300 million individuals worldwide. It is a serious global health problem affecting all age groups, with increasing prevalence in many developing countries, rising treatment costs, and a rising burden for patients and the community. Asthma still imposes an unacceptable burden on health care systems, and on society through loss of productivity in the workplace and, especially for pediatric asthma, disruption to the family. Health care providers managing asthma face different issues around the world, depending on the local context, the health system, and access to resources (GINA, 2014).

International guidelines for the treatment of asthma have identified that the primary clinical goal of asthma management is to optimise asthma control (minimisation of symptoms, activity limitation, bronchoconstriction and rescue $\beta 2$ -agonist use) and thus reduce the risk of lifethreatening exacerbations and long-term morbidity. The Asthma Control Questionnaire (ACQ) was developed to meet these criteria. It measures both the adequacy of asthma control and change in asthma control, which occurs either spontaneously or as a result of treatment (*Juniper et al.*, 2006).

Asthma guidelines indicate that the goal of treatment should be optimum asthma control. In a busy clinic practice with limited time and resources, there is need for a simple method for assessing asthma control with or without lung function testing (*Nathan et al.*, 2004).

The good news is that for most kids with asthma, it can be well controlled sometimes so well that flare-ups are rare. For many families, the learning process is the hardest part of controlling asthma. Between diagnosis and good control, there's much to learn and a lot to do. Don't be surprised or discouraged if your child has flare-ups while learning to control asthma. Asthma control can take a little time and energy to master, but is worth the effort. Identifying triggers and symptoms can take time and good detective work. But once patterns are discovered, some of the triggers can be avoided through (*Kidshealth.org*, 2015).

The reason why some patients develop asthma that is difficult to manage and relatively insensitive to the effects of current antiinflammatory asthma therapy especially inhaled corticosteroids is not well understood. Common associations in patients with difficulttomanage asthma include poor compliance with treatment and psychological and psychiatric disorders. However, genetic factors may also contribute in some. Many patients have difficult to treat asthma from the onset of the disease, rather than progressing from milder asthma. In these patients airway closure leads to air trapping and hyperinflation. Although the pathology



appears broadly similar to other forms of asthma, there is an increase in neutrophils, more small airway involvement, and more structural changes (*Manning*, 2008).

Aim of the Work

Study the demographic and clinical characteristics of bronchial asthma patients and their relation to level of asthma control in Al-Mahalla chest hospital.

Review of Literature BRONCHIAL ASTHMA

The word 'asthma' is derived from the Greek aazein, meaning "sharp breath." The word first-appears in Homer's Iliad; Hippocrates was the first to use it in reference to the medical condition, in 450 BC. Hippocrates thought that the spasms associated with asthma were more likely to occur in tailors, anglers, and metalworkers (*Marketos and Ballas*, 1982).

Bronchial asthma is a disease that is becoming a major health issue in many developing countries. Many factors may have contributed to the rise of the problem of bronchial asthma. Increasing air pollution, fast modernization, and widespread construction work are some of the reasons for asthma to thrive. The situation is complicated by poor access to medical services, high price of effective drugs, and poor health education among the affected population. Increased urbanization may have modified the traditionally low incidence of bronchial asthma in the Third World. Diets becoming more westernized, improvement in standard of living, decrease in exercise rates, more dust mites, and more pollution has been blamed (Ramos et al., 2006).

Definition:

Asthma (AZ-ma) is a chronic (long-term) lung disease that inflames and narrows the airways. Asthma causes recurring periods of wheezing (a whistling sound

when you breathe), chest tightness, shortness of breath, and <u>coughing</u>. The coughing often occurs at night or early in the morning. Asthma affects people of all ages, but it most often starts during childhood. In the United States, more than 25 million people are known to have asthma. About 7 million of these people are children (*National Heart, Lung and Blood Institue, 1996*).

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the 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 (GINA, 2014).

The burden of asthma:

Prevalence, Morbidity, and Mortality:

Asthma affects an estimated 300 million individuals worldwide. It is a serious global health problem affecting all age groups, with increasing prevalence in many developing countries, rising treatment costs, and a rising burden for patients and the community. Asthma still imposes an unacceptable burden on health care systems, and on society through loss of productivity in the workplace and, especially for pediatric asthma, disruption to the family. Health care providers managing asthma face different issues around the world, depending on the local context, the health system, and access to resources. *The Global Initiative for Asthma (GINA)* was established to

about increase asthma health awareness among professionals, public health authorities and the community, and to improve prevention and management through a coordinated worldwide effort. GINA prepares scientific asthma. encourages dissemination reports on implementation of the recommendations, and promotes international collaboration on asthma research (GINA, *2014*).

Working in continued collaboration with leaders in asthma care from many countries, GINA sponsors World Asthma Day (first Tuesday in May) which has been extremely successful. A vast number of people have made a commitment to bring awareness about the burden of asthma to their local health care officials, and to implement programs of effective asthma care (*GINA*, *2010*).

The asthma mortality comparison between countries has been made using the asthma mortality rates in the 5- to 34-year age group because the diagnosis of asthma mortality is firmly established in this group. It has been shown that in this age group false-positive reporting (i.e., deaths from other causes being falsely attributed to asthma) and false-negative reporting (i.e., asthma deaths being falsely assigned to other categories) are extremely low. However, the accuracy of this approach declines with increasing age, with false positive reporting rates of >30% in those aged 65 years or more (*Masoli et al., 2004*).

In considering the impact of a disease in terms of mortality, it is informative to extend the concept of life expectancy to that of health expectancy. In this way an attempt is made to generalize the concept of years of life lost to that of years of healthy life lost, representing a health gap measure which incorporates both loss of life and the loss of quality of life. This allows a composite measure of the burden of both fatal and non-fatal disease. As a result, the years lost to disability (YLD) is added to the years of life lost to premature mortality (YLL) to yield an integrated unit of health - the "disability adjusted lifeyear" (DALY), with one DALY representing the loss of one year of healthy life. The DALYs lost due to asthma worldwide in 2001 are presented, together with the 30 leading causes of DALYs. These data were obtained from the recently published (WHO World Health Report 2002) (Masoli et al., 2004).

The World Health Organization has estimated that 15 million disability-adjusted life years (DALYs) are lost annually due to asthma, representing 1% of the total global disease burden. Annual worldwide deaths from asthma have been estimated at 250,000 and mortality does not appear to correlate well with prevalence. There are insufficient data to determine the likely causes of the described variations in prevalence within and between populations (*Masoli et al.*, 2004).

Social and economic burden:

Social and economic factors are integral to understanding asthma and its care, whether viewed from the perspective of the individual sufferer, the health care professional, or entities that pay for health care. Absence from school and days lost from work are reported as substantial social and economic consequences of asthma in studies from the Asia-Pacific region, India, Latin America, the United Kingdom, and the United States (*Mahapatra*, 1993 and Neffen et al., 2005).

The monetary costs of asthma, as estimated in a variety of health care systems including those of the United States (*Weiss et al., 1992 and Weiss and Sullivan, 1993*) and the United Kingdom are substantial. In analyses of economic burden of asthma, attention needs to be paid to both direct medical costs (hospital admissions and cost of medications) and indirect, non-medical costs (time lost from work, premature death) (*Marion et al., 1985*).

For example, asthma is a major cause of absence from work in many countries (*Asher et al.*, 2006), including Australia, Sweden, the United Kingdom, and the United States (*Karr et al.*, 1978). Comparisons of the cost of asthma in different regions lead to a clear set of conclusions:

 The costs of asthma depend on the individual patient's level of control and the extent to which exacerbations are avoided.

- Emergency treatment is more expensive than planned treatment.
- Non-medical economic costs of asthma are substantial.
- Guideline-determined asthma care can be cost effective.
- Families can suffer from the financial burden of treating asthma.

Although from the perspective of both the patient and society the cost to control asthma seems high, the cost of not treating asthma correctly is even higher (*Accordini* et al., 2006).

Factors influencing the development and expression of asthma:

Factors that influence the risk of asthma can be divided into those that cause the development of asthma and those that trigger asthma symptoms; some do both. The former include host factors (which are primarily genetic) and the latter are usually environmental factors. However, the mechanisms whereby they influence the development and expression of asthma are complex and interactive. For example, genes likely interact both with other genes and with environmental factors to determine asthma susceptibility (*Ober, 2005 and Holgate, 1999*).

Asthma is generally believed to be a heterogeneous disease whose inception and persistence is driven by gene–environment interactions. The most important of these interactions may occur in early life and even in-utero.

There is consensus that a 'window of opportunity' exists during pregnancy and early in life when environmental factors may influence asthma development. Multiple environmental factors, both biological and sociological, may be important in the development of asthma. Data supporting the role of environmental risk factors for the development of asthma include a focus on: nutrition, allergens (both inhaled and ingested), pollutants (particularly environmental tobacco smoke), microbes, and psychosocial factors (GINA, 2014).

Host Factors:

Genetic

According to genetic association theory, various genes such as IL10, SPINK-5 and STAT-6, play a major role in the transfer of asthma from one generation to another. These genes are mostly associated with the modulating inflammation near the bronchial walls. However, these genes have not been able to produce consistent results over a large set of individuals, thus leading to the conclusion that these genes do not lead to the development of bronchial asthma under all set of conditions and only influence in the development of certain subsets (*Sandford et al.*, 1996).

According to scientists, certain genetic variants lead to the development of bronchial asthma only when they are exposed to certain environmental conditions. Genes such as CD14, C159-T are prime examples of such variants. These

genes cause bronchial asthma in individuals only when they are exposed to endotoxin, a byproduct of bacteria (Sandford et al, 1996). Although environmental factors are clearly important determinants of asthma, numerous studies have revealed that asthma has a strong genetic component but does not follow monogenic patterns of inheritance (Aberg, 1993 and Litonjua et al., 1998). For a long time, asthma has been known to cluster in families, and family studies were the first to suggest that the disease was genetically inherited. More recent family studies found, for example, a 60 percent increased risk of atopy when both parents were affected (ABERG, 1993), and the odds of asthma in a child increased from 3 when one parent was affected to 6 when both were (Laitinen et al., 1998). Maternal asthma appears to be more influential than paternal asthma (Laitinen et al., 1998).

In addition to genes that predispose to asthma there are genes that are associated with the response to asthma treatments. For example, variations in the gene encoding the beta-adrenoreceptor have been linked to differences in subjects responses to β_2 -agonists (*Israel et al.*, 2004).

Obesity

Since the late 1990s, obesity has been reported to be associated with asthma, (*Rönmark et al.*, 2002) and an increase in the prevalence of obesity in Western society has been reported along with a parallel increase in asthma prevalence (*Jubber*, 2004). However, Chinn and Rona

(Somerville et al., 1984) have suggested that the increase in asthma prevalence cannot be explained by the increased prevalence of obesity in the population. Further, obesity may be a consequence of asthma. A convincing relationship between asthma and obesity has not been established. Three prospective studies have found obesity or an increased body mass index (BMI) to be associated with new onset of asthma among females (Gilliland et al., 2003). In another study, pre-teenage overweight preceded asthma and bronchial hyperreactivity in female children (Jubber, 2004). In contrast, a recent study found obesity to be significantly related to incident asthma only among male and nonallergic children (Gilliland et al., 2003).

Obesity has also been shown to be a risk factor for asthma. Certain mediators such as leptins may affect airway function and increase the likelihood of asthma development (*Shore et al.*, 2005 and Beuther et al., 2006).

Sex

Male sex is a risk factor for asthma in children. Prior to the age of 14, the prevalence of asthma is nearly twice as great in boys as in girls (*Horwood et al.*, 1985). As children get older the difference between the sexes narrows, and by adulthood the prevalence of asthma Is greater in women than in men. The reasons for this sex-related difference are not clear. However, lung size is smaller in males than in females at birth (*Martinez et al.*, 1995) but larger in adulthood.

Environmental Factors:

There is some overlap between environmental factors that influence the risk of developing asthma, and factors that cause asthma symptoms-for example, occupational sensitizers belong in both categories. However, there are some important causes of asthma symptoms-such as air pollution and some allergens-which have not been clearly linked to the development of asthma (*GINA*, *2010*).

Allergens

Although indoor and outdoor allergens are well known to cause asthma exacerbations, their specific role in the development of asthma is still not fully resolved. Birthcohort studies have shown that sensitization to house dust mite allergens, cat dander, dog dander (*Wahn et al.*, 1997 and Sporik et al., 1990), and Asperglllus mold (*Hogaboam et al.*, 2005) are independent risk factors for asthma like symptoms in children up to 3 years of age. However, the relationship between allergen exposure and sensitization in children is not straightforward. It depends on the allergen, the dose, the time of exposure, the child's age, and probably genetics as well.

For some allergens, such as those derived from house dust mites and cockroaches, the prevalence of sensitization appears to be directly correlated with exposure (*Horwood et al.*, 1985). However, although some data suggest that exposure to house dust mite allergens may be a causal factor in the development of asthma, other studies have

guestioned this interpretation (Sporik et al., 1995 and Charpin et al., 1991). Cockroach infestation has been shown to be an important cause of allergic sensitization, particularly in inner-city homes (Rosenstreich et al., 1997).

In the case of dogs and cats, some epidemiologic studies have found that early exposure to these animals may protect a child against allergic sensitization or the development of asthma (*Platts-Mills et al.*, 2001 and Gern et al., 2004), but others suggest that such exposure may increase the risk of allergic sensitization (*Ownby et al.*, 2002; Celedon et al., 2002 and Almqvist et al., 2003). This issue remains unresolved.

Infections

During infancy, a number of viruses have been associated with the inception of the asthmatic phenotype. Respiratory syncytial virus (RSV) and parainfluenza virus produce a pattern of symptoms including bronchiolitis that parallel many features of childhood asthma (Sigurs et al., 2000 & Gern and busse, 2002). A number of long-term prospective studies of children admitted to the hospital with documented RSV have shown that approximately 40% will continue to wheeze or have asthma into later childhood (Sigurs et al., 2000). On the other hand, evidence also indicates that certain respiratory infections early in life including measles and sometimes even RSV, may protect against the development of asthma (Stein et al., 1999). Parasite infections do not in general protect against asthma. but infection with hookworm may reduce the risk (Leonardi-Bee et al., 2006).