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Bronchial asthma is an episodic reversible inflammatory disorder of the obstructive airways disease, that is characterized by increased responsiveness of the tracheobronchial tree to a multiplicity of stimuli. Asthma is manifested physiologically by a wide spread narrowing of the air passages (Bronchospasm) increased bronchial mucus secretions and bronchial oedema. The patient presents with paroxysms of dyspnoea, chest tightness, cough and wheezing. In severe attacks there is tachycardia, pulsus paradoxus and central cyanosis. That is often reversible either spontaneously or with treatment. The air flow obstruction causes mismatching of alveolar ventilation and perfusion and increases the work of breathing. It is thought that varied etiology factors viz. biochemical, immunological, infective, endocrinial and psychological play and important role in causing an attack of asthma. These factors may or may not interact to produce classical symptomatology of the disease process.

The prevalence and incidence of asthma is observed that about 5-10 percent of children suffer from frequent wheezy episodes at some times in there childhood, the incidence increases to 20 percent if children having less
than 6 attacks of wheezing are included. Also, it has been observed that about half of the children presenting with asthma are atopic and show minor immuno-deficiencies (Sims et al 1981). Asthma could start at any age, although 80 to 90 percent of asthmatic patients have their first attack before the age of five years. Prior to puberty the sex incidence male : female is 2 : 1, thereafter, the sex incidence is equal (Ellis 1983).

Asthma may be extrinsic or atopic and intrinsic or nonatopic in nature. In extrinsic asthma, there is a strong family history of allergic diathesis in the family. High eosinophil count, abnormal CAMP metabolism and selective synthesis of IgE. Intrinsic asthma is often precipitated by infection specially by respiratory syncytial virus (RSV) or parainfluenza virus in infants and rhinovirus in patients who are more than two years of age. Recent studies show high IgE, atopic manifestation and eosinophilia in 10-30% cases of lower respiratory viral infections, indicating possible atopic component in these cases who subsequently develop asthma (Khatua et al 1987).
Asthma in children is usually, but not always of allergic type which occurs before 2 years of age and is precipitated by pollens, foods, house dust, mites, fungi and animal danger. It may be preceded by eczema. It is due mainly to type-I hypersensitivity reaction. Type-I hypersensitivity reaction is antigen antibody dependent. The reaction is initiated by allergen exposure, B cell stimulation and production of IgE. Then IgE binds with specific receptors, which are present on mast cell and basophil surfaces. On repeated challenge by specific antigen the second stage of reaction comes into play with binding of antigen to the surface bound IgE.

The interaction between IgE and antigen provokes the release of mediators from granules inside the mast cells. This process known as degranulation, releases histamine, leukotrienes, eosinophil chemotactic factor of anaphylaxis (ECF-A) and prostaglandins that cause the symptoms of type-I hypersensitivity (Behrman and Vaughan 1990).

In some cases of asthmatic patients, there is another mechanism which play a role to produce type-I hypersensitivity like reaction. This involves IgG4 (Sub-class of IgG).
Antibody mediating a complement form of type I reaction. According to Anderson (1984) non-immunologic mechanism also played a role in the pathogenesis of atopic disease named as anaphylactoid reaction (Non IgE mediated anaphylaxis like reaction).


The term complement is applied to a system of factors occurring in normal serum that are activated characteristically by antigen antibody interaction and subsequently mediate a number of biologically significant consequences. Complement as it participates in immune-haemolysis comprises of components symbol C is used to denote complement components. In the order of their entry into reaction process, the complement components are designated i.e. C1, C2, C3, C4, C5, C6.. C9. In bronchial asthma some worker had shown decreased level of C3 and C4 (Halprin 1973; Kay 1974, Godfrey 1975, Delaney 1976, Arroya 1976, Hutcheroft 1978, Srivastava 1982). Other have shown its levels increased (Baur 1980, Srivastava 1982).
Among all the pulmonary function tests, spirometry, which records the volume of air into and out of lungs, is simple to measure. Measurement of the forced expiratory volume in 1 second (FEV₁), forced vital capacity (FVC) and peak expiratory flow rate (PEFR) provide a fairly reliable indicator of the degree of the airflow obstruction in case of bronchial asthma. Asthma severity can be judged by symptoms, medication requirements and objective measures of lung function.


Keeping in view the above facts, a clinical and immunological study of bronchial asthma in children was undertaken to assess serum IgE, complement C3, C4, Absolute eosinophil count and spirometry.