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ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS

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INTRODUCTION

Allergic Bronchopulmonary Aspergillosis (ABPA) is an inflammatory disorder of lung caused due to immune hypersensitivity inflammatory reaction to Aspergillus fumigatus. It occurs mainly in patients of Asthma or Cystic fibrosis (CF). Sometimes an ABPA like disease is caused by fungi other than A. fumigatus, and that condition is termed as allergic bronchopulmonary mycosis (ABPM). ABPA was first described by Hinson et al. in 1952 from UK. Almost two-decade later it was again reported by Shah et al. from India.

The prevalence rate of ABPA in Asthmatics are very high and in India it is believed to have almost double that reported from other countries. Ritesh Agarwal et al. has quoted the number of ABPA cases in India is roughly about 1.4 million. The community prevalence in India is above 5% in Asthma cases[1].

ETIOLOGY & PATHOLOGY

Aspergillus fumigatus, an ubiquitous mould is the causative organism for ABPA. The conidia of this fungus very small about 2-3 micrometres in diameter are inhaled through breath and colonise in airways & alveoli. In healthy non susceptible individuals, it is being eliminated by body defence mechanism. But in susceptible asthmatics individuals having genetic predilection due to presence of HLA- DR2, DR5, and possibly DR4 or DR27[2] and mucus in the airway, the fungus grows there and behaves as an antigen, resulting intense immune hypersensitivity reaction leading to formation of IgE & IgG antibodies. It further stimulates a chronic allergic inflammatory response causing tissue injury which ultimately lead to clinical feature of ABPA.

CLINICAL PRESENTATION

Most of the patients of ABPA present with features of severe asthma. The other common features may be fever, haemoptysis, fleeting lung opacities in chest radiography & bronchiectasis.

The symptoms are very non-specific to diagnose ABPA, hence proper screening of asthmatics patients for ABPA is needed.

DIAGNOSIS OF ABPA

The investigations commonly done to diagnose ABPA include

1. Skin Test (Prick or intradermal). to check for Aspergillus sensitization.
2. Serum A. fumigatus specific IgE - > 035 kUA/L
3. Serum total IgE - > 500 IU/mL
4. Serum precipitin or IgG against A. fumigatus- >27 mgA/L
5. Peripheral Blood Eosinophil Count >500 cell/ micro L
6 Chest X-Ray
7. High Resolution CT scan of Chest.

The other tests that may be done as adjuvant investigation are Sputum culture & spirometry.

Chest x-ray has limited sensitively of around 50% for diagnosis of ABPA. It may show parenchymal infiltrates & bronchiectatic change, mainly in upper lobes, however other lobes may also be involved.

HRCT is the investigation of choice. It easily detects bronchiectasis and mucus filled bronchi and also delineate the distribution.

The diagnosis of ABPA is ultimately made on composite basis of clinical, radiological & immunological findings in blood. The Criteria for the diagnosis of ABPA was first proposed by Rosenberg & Patterson in 1977[3]. The Criteria were further reviewed by ISHAM. ISHAM-ABPA Working group modified criteria was put forward in 2021[4].

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The criteria are

(a). All should be present-

- A. fumigatus specific IgE > 0.35 kUA/L
- Serum total IgE > 500 IU/ml

(b). And 2 or more of the followings

- A. fumigatus specific IgG > 27 mg A/L
- Bronchiectasis on CT thorax
- Peripheral blood eosinophil count > 500 cells/micro L

TREATMENT

ABPA if left untreated, may lead to irreversible lung damage due to bronchiectasis, hence early diagnosis and treatment is warranted. The goals of treatment are

(a) Control of symptoms

(b) Preserving the normal lung structure & functions

(c) Prevention of exacerbation.

The current treatment options are directed towards reducing the inciting fungus load by antifungal drugs and suppressing the immune response to fungus by corticosteroids.

ORAL GLUCOCORTICOSTEROID :

It is the first line treatment of choice[5]. The usual dose is Prednisolone 0.5mg/kg/day for 4 weeks, 0.25 mg/kg/day - 4 weeks, 0.125 mg/kg/day- 4 weeks, then further tapered over one month. The total duration is 4 months.

ORAL ANTIFUNGAL DRUGS:

These drugs basically help in reducing the fungal load, thereby reducing the inflammatory activity thus acting as steroid-sparing agents. It may also help in decreasing the episodes of exacerbations.

Itraconazole 200mg twice a day for 16-24 weeks is given. Newer antifungal drug voriconazole 200mg twice for 16-24 may be given, but cost is the major deterrent.

All patients & ABPA, beside above drugs should also be optimally treated for their underlying asthma or Cystic fibrosis.

MONITORING OF TREATMENT

The response to treatment is monitored by assessment of clinical symptoms as well as re-evaluation of immunological and imaging features.

Decline in total IgE to the tune of at least 25% usually after eight weeks treatments along with clinical & radiological improvement is marker of response to treatment. With successful treatment, the serum total IgE falls progressively. ABPA remission is marked by clinical, radiological & immunological stability i.e. < 50% increase in total IgE for at least 6 months after stopping treatment of ABPA.

CONCLUSION

ABPA is prevalent in patients of Asthma & Cystic fibrosis. So, all patients of Asthma & cystic fibrosis having frequent episodes of exacerbation must be investigated for ABPA.

Early diagnosis & treatment is needed to preserve lung function & avoid permanent damage.

Initial treatment is with Glucocorticosteroid. Antifungal drug may be added to reduce fungal load and as cortisone sparing agent.

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