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REVIEW ARTICLE

PARTHENIUM ALLERGY IN INDIAN POPULATION: CLINICAL SIGNIFICANCE

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ABSTRACT: Parthenium hysterophorus is a deep rooted three to four feet tall tree at fully flowered stage. The origin of the weed is Caribbean Islands and it can withstand any adverse climate. It is found all over India. It bears white flowers which in turn bears numerous seeds which disperse due to wind or water. July and august is the peak season for these weeds. The parthenium weed produces as much as 3,000 million pollen grains per square meter during the flowering season. They may cause allergic type reactions like asthma, skin rashes, puffy eyes, peeling skin, eczema, running nose, swelling and itching of mouth and nose etc. The main cause of parthenium allergy is direct or indirect contact with the parthenium pollen. Here we have reviewed the harmful effects of Parthenium on human beings.

KEYWORDS: Parthenium hysterophorus, allergy, Bronchial asthma, contact dermatitis.

INTRODUCTION: The term allergy, coined by Von Pirquet, is used to define the series of events which occurs when an antigen which is not harmful in itself, causes an immune response, leading to symptoms and diseases in genetically predisposed individuals. An antigen that induces allergic response is called an allergen. The development of allergies involving IgE antibody formation, also known as atopy, involves both complex and environmental influences that are only now being elucidated. Among the 10 most common allergens in the Indian environment, Parthenium Hysterophorus is one among them.

The word Parthenium is derived from the Latin word 'parthenice', suggesting medicinal uses. Parthenium hysterophorus is a species of flowering plant in the aster family, Asteraceae, that is native to the American tropics. Common names include carrot gass, congress grass, gajar gass, Santa Maria Feverfew and Whitetop Weed. Other common names include featherfew, febrifuge plant, featherfoil, mid-summer daisy, and wild chamomile. It is a common invasive species in India, Australia, and parts of Africa. P. hysterophorus invades all disturbed land, including farms, pastures, and roadsides. In some areas, outbreaks have been of almost epidemic proportions, affecting crop production, livestock and human health. In India, it was first present as a contaminant in imported wheat in 1956.(1) It can trigger allergies and is a common cause of pollen allergy.

Parthenium is a species of flowering plant in aster family, Asteraceae, native of the American tropics. It is an obnoxious weed, colonizes grassy land, and grows rapidly in bare areas, along road side and especially near water points. It belongs to the family Asteraceae/Compositae (Daisy family), which is one of the largest and most important families in the plant kingdom (Fig. 1). The family includes troublesome weeds, ornamental annuals, herbaceous perennials, medicinal and food plants. The plants of Compositae family have many tiny flowers (florets)

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clustered to form a flower head (capitulum). This flower head is surrounded by bracts (modified leaves) that form an involucre beneath or around a flower cluster. P. hysterophorus has two life cycles: Juvenile or rosette stage, and Mature or adult stage. At juvenile stage the plant has a rosette with large, dark green, simple, radicle, pinnetisect small leaves and flowering is absent. During adult stage, the plant branches profusely, leafy herb resembles a bush or shrub because of its height (1-2.5m). The stem becomes tough and woody as the plant matures into a hardy bush. The plant contains enormous number of pollen grains (624 millions per plant) are produced by anemophilous (by wind) pollination. It is an extremely prolific seed producer with upto 25,000 seeds (achenes) per plant.[2] One of the world's seven most devastating and hazardous weeds, parthenium invaded 14.25 million hectares of farm land during 2001-07, compared to 2 million hectares in 1991-2000. Parthenium has invaded 35 million hectares across the country including crop land, wasteland and forest areas, according to the DWSR, which is preparing a report on this. Initially, the deadly weed occupied largely non-crop areas like wasteland, open forests and roadsides. Now it has now spread to cropping land at an alarming rate.

Parthenium plant is thermo- and photo-insensitive; hence, it grows round the year except in severe winters; in other words, it survives environmental extremes. It is a rapid colonizer and competes out other vegetation in its vicinity within two growing seasons. It grows in almost all types of soil except near the seashore as the saline soil is not conducive to parthenium flowering. Parthenium is a curse for the biodiversity. Scientists describe it as a "noxious, poisonous, allergic and aggressive weed posing a serious threat to human beings and livestock." It squeezes grasslands and pastures, reducing the fodder supply. It is also responsible for bitter milk disease in livestock fed on grass mixed with parthenium.[3] The reasons for its fast spread are: high germination ability throughout the year, large seed production ability, high survival rate, extreme adaptability in a wide range of habitats and easy dispersal of seeds.

Allergenicity of Parthenium: The allergens in Asteraceae are allergenic sesquiterpene lactones (SQLs) and thus the patients with contact dermatitis to Compositae can react to many other non-Compositae SQL containing plants. The SQLs are found in the leaves, stems, flowers, and some pollens. The highest concentrations are found in trichomes which are present on stems, the underside of leaves and in the flowering heads.[4] The major component of these toxins being Parthenin and other Phenolic acids such as caffeic acid, vanillic acid, anisic acid, chlorogenic acid, parahydroxy benzoic acid, Panisic acid are lethal to human and animals. Cross-reactivity between SQLs does not follow any rules. Over 200 skeletal types and 1350 individual types of SQLs have been described, and each of these may have multiple functional groups attached to them. SQLs are characterized by the presence of a γ-butyrolactone ring bearing an exocylic γ-methylene group. Parthenin has enhanced toxicity due to the presence of a cyclopentene group that can cause chromosomal damage in animal cells, uncouple phosphorylation and inhibit the key cellular enzymes.

Risk for Human Health: Around three decades ago, serious human health risks from P. hysterophorus were reported from Pune.[5] Several thousands of cases of allergic contact dermatitis with some fatalities have been reported. An outbreak of epidemic proportion followed a

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dam burst. After 1-10 years of exposure to the weed, 10-20% of the population will develop severe allergenic reactions. There may be hay fever, allergic rhinitis, asthma or dermatitis and can be caused by dust and debris from the plant as well as pollen. Aeropollen sampling in Bangalore (Southern India) over a 6-year period revealed that 40-60% of the total pollen count was from P. hysterophorus. Allergenicity to P. hysterophorus pollen extracts was recorded in 34% allergic rhinitis and 12% bronchial asthma patients from Bangalore.[6] Parthenium pollen is now a major cause of allergic rhinitis in Bangalore with 7% of the population affected and 40% sensitive to the pollen. Such a high incidence of allergic rhinitis to specific pollen has not been reported from any other place in the world. Subsequent studies in Northern India (Punjab) showed that a significant proportion of bronchial asthma patients is sensitized to P. hysterophorus.[7] In New Delhi, out of 63 patients with airborne contact dermatitis, 62 showed a positive reaction to the parthenium weed.[8] The severity of dermatitis in India is greater in comparison to America because the plant grows more vigorously in India and contains large amounts of the sesquiterpene lactone, parthenin, which is absent in the plants in South America. It clinically involves the adult males in both USA and India.[9] Studies have estimated a ratio of 20:1 between men and women. This cannot be explained in terms of degree of exposure since Indian women and children also work in fields. Possibly women and children are less frequently sensitized. However, the studies on plant dermatitis from India have shown a male-to-female ratio of 1:1 and 5:5:1. Initially, the exposed sites of the face, neck and flexures are affected with erythema, blistering and intense pruritus resulting later in skin thickening, hyperpigmentation and development of a leonine facies. Unexposed sites may get involved late in the course of the disease. A seasonal variation is initially observed with the dermatitis flaring in the summers corresponding to the growing season and disappearing in winters. After several years, persistent pruritic lichenified dermatitis develops without seasonal variation. Winter exacerbation is seen in the months of September, October and November and may be due to the increased growth of parthenium following the North-East monsoon showers.[10]

Role of Parthenium in Allergic Rhinitis and Bronchial Asthma: The pollen of the parthenium is a potential source of allergic airway disease –allergic rhinitis and bronchial asthma. Factors responsible for its extensive spread are its high seed production (almost throughout the year) and extreme light weight. One study done in Bangalore reported allergenicity for parthenium hysterophorus pollen extracts to be 34% for allergic rhinitis and 12% bronchial asthma.[11] Another study from Delhi also reported parthenium sensitivity in 45.1% of the patients having allergic rhinitis and 46.94% patients having nasobronchial allergy, which were proved either by skin test, nasal eosinophilia or blood eosinophilia.[12] Exposure to parthenium pollen is mainly anemophilous (i.e. wind mediated). Pollens contain submicronic allergens like starch granules and polysaccharide-containing wall precursor bodies which get released from pollen grains landing on the stigma of receiving plant and if genetically compatible activation occurs. For e.g. the pollen grains on angiosperms hydrate on contact and pollen components either diffuses or is expelled from the grains after which pollen tube development occurs through the style, leading to fertilization. It is this process that is thought to account for allergen exposure to humans when grains land on mucosal surface. Sometimes angiosperm pollen grains may also

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encounter environmental water and it has been shown that when this occurs, cytoplasmic granules are expelled from the grains, thus releasing submicronic allergen particles into the atmosphere. This process is thought to contribute to thunderstorm-related outbreaks of asthma in the community. Immunoblot assays of the parthenium pollen have identified a no. of allergens in a molecular range of 14- 70 kda. PAS staining and lectin overlay experiments have revealed that the allergens are mainly glycoproteins. Immunoscreening of crude extract of parthenium pollen with sera from parthenium sensitive individuals revealed that most significant IgE binding activity, in more than 90% of sera tested was associated with a 31 kda glycoprotein which is designated as Par h 1, function being β- extension.[13]

Inhalation of pollens can cause allergic rhinitis that can develop into bronchitis or asthma if the pollens enter the respiratory tract during breathing. We cannot predict which individuals will develop allergies and which will not based on simple Mendelian inheritance patterns. However, there does appear to be a higher incidence allergies among offspring's of allergic parents. The two step method by which one initially becomes "allergic" to a substance begins with sensitization. Similar changes happen during exposure to the parthenium allergens in the nearby vicinity. During the initial stage of sensitization, the individual develops significant amount of IgE antibodies against an inhaled allergen. Memory B cells develops at this stage. Second stage involves adherence of newly formed IgE antibody to circulating blood basophils, or to mast cells located in mucosal surfaces of skin or respiratory system. An individual is considered to be "sensitized" only after IgE antibodies against a certain substance have been produced and are bound to the surface of mast cells and basophils. The process of sensitization does not produce any of the symptoms of allergic manifestations, until the re-exposure of the allergens.[14] The second step in the process of becoming allergic involves the re-exposure of sensitized person to the allergen, with the production of symptoms ranging from negligible rhinorrhea to sudden death. Most cases lie somewhere in between. One should keep in mind that although the cellular and molecular events for all immediately hypersensitivity reactions are similar, differences in target organ responses, ultimately dictate the clinical patterns of disease activity once a reaction has been induced.

Role of Parthenium Hysterophorus in Contact Dermatitis: Parthenium hysterophorus and Tanacetum parthenium, members of the Compositae family, are important causes of allergic contact dermatitis due to plants. Parthenium dermatitis is a major problem in India and Australia. Parthenium hysterophorus causes a spectrum of clinical patterns. Parthenium dermatitis, in its classical form known as airborne contact dermatitis, primarily affects the exposed areas and the flexures. Other clinical patterns are photodermatitis (essentially a pseudo-photodermatitis) involving the eyelids, nasolabial folds, areas under the chin and behind the ears, atopic widespread dermatitis, actinic reticuloid dermatitis, exfoliative dermatitis and also photosensitive lichenoid dermatitis.[15] The trend of the clinical pattern is changing. The classic airborne contact dermatitis may change to photodermatitis resembling chronic actinic dermatitis or mixed pattern dermatitis. Hand dermatitis is observed in gardeners after contact with the weed. Vitiliginous skin appears to be spared perhaps due to the vacuolization of Langerhans cells in these areas. The allergens responsible for contact dermatitis are sesquiterpene lactones and are present in the

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oleoresin fraction of the leaf, the stem, and the flower and also in pollen. The highest concentration of SQL is found in trichomes which are present on stems, the underside of leaves and in the flowering heads. The allergens can be extracted in various solvents (Such as acetone, alcohol, ether, and water) and then used for patch testing.[16] Acetone extract of Parthenium is better than aqueous extract in eliciting contact sensitivity. Initially the exposed sites of the face neck and flexures are affected with erythema, intense pruritus resulting later in skin thickening, hyperpigmentation and development of leonine faces. Unexposed sites may get affected late in the disease. Both type I and type IV hypersensitivity are responsible for parthenium dermatitis. Type I hypersensitivity mediated by IgE, particularly in sensitized atopic individual with parthenium dermatitis could be initiating and perpetuating the dermatitis.[17] The severity of dermatitis in a parthenium sensitive patient depends on the degree of contact hypersensitivity in the patient at that time and the quantity of antigen in contact with the patient. The degree of contact hypersensitivity to an agent can be determined by the titer of contact hypersensitivity.[18] Studies on cross-reactivity between ragweed (Ambrosia) and parthenium pollen suggest that individuals sensitized to parthenium may develop type-I hypersensitivity reactions to ragweed and vice versa when they travel to regions infested with the weed, to which they have not been previously exposed. Parthenium weed may have a more sinister effect on human health since it has been hypothesized that parthenium-contaminated animal feed leads to tainted milk and that the hepatotoxic parthenin reacts synergistically with copper in causing Indian childhood cirrhosis (ICC).[19]

Diagnosis: A detailed history has to be elicited for diagnosis of allergic manifestation due to exposure to the parthenium species. Atopy history in the family member is to be enquired. Thorough history regarding growth of parthenium in the surrounding places of residence or contact of the weed at work place has to be enquired. The following investigations can be carried out for the confirmation of the diagnosis:

1. Patch test – using plant material as it is.
2. Prick test - plant material is crushed and diluted in saline in order to obtain a solution that can easily be pricked. Both immediate reactions at 15 min and after 24-48 hrs are recorded.
3. RAST -for parthenium specific IgE antibodies.
4. Serum IgE measurement during the active phase of the disease.
5. Clinical Severity Scoring (CSS) system for contact dermatitis as devised by Verma et al.[16]

Treatment: The treatment of allergic rhinitis (AR) relies on an integrated approach to patient management. The physician has several strategies to use in treating the patient with AR, and the judicious use of these strategies will result in optimal therapeutic outcomes and improved symptoms and quality of life. Pharmacotherapy is only one of three major treatment arms in approaching the management of patients with allergic rhinitis. For pharmacotherapy to have maximal benefit, it should be used in conjunction with an appropriate program of environmental management.[17] Avoidance of offending agents, where possible, lessens antigen exposure, decreases inflammation, ameliorates patient symptoms, and lowers medication requirements. In addition, in difficult patients, patients who are not responding appropriately to medications and

patients who are seeking an alternative for their care, immunotherapy can be a useful treatment strategy.

Effective management of bronchial asthma requires establishing a long term working relationship with the patient. This enables the physician to understand the extent of disease, the role that the disease plays in patient's life, as well as encouraging the patient to take an active role in his/her own health care. Treatment measures in these patients include pharmacological therapy involving inhales corticosteroids along with long acting $beta_2$ agonists, for the control of inflammation and short acting $beta_2$ abonists as prn. These measures should be accompanied by environmental control and also immunotherapy when indicated.[18]

Treatment of Parthenium dermatitis is mostly symptomatic. Topical steroids, antihistamines, and avoidance of Parthenium are the mainstay of treatment for localized dermatitis. Systemic corticosteroids and azathioprine are frequently needed for severe or persistent dermatitis.

Allergen immunotherapy, also called Specific Immunotherapy, is the only recognized treatment of allergic disease that can ameliorate symptoms and alter the natural course of the disease, and it can lead to a long-term improvement which is sustained years after the discontinuation of this treatment.

Role of Immunotherapy: Immunotherapy is able to change the course of allergic disease in cases of contact dermatitis. It is also the only causal treatment of allergic disorders and therefore a cornerstone in the management of respiratory allergy. Immunotherapy is also effective in patients with allergic rhinitis and mild-to-moderate allergic asthma. Successful treatment is associated with decrease in allergic symptoms of the upper and lower airways.[19]

Immunological mechanisms underlying the immunotherapy treatment are: (i) Allergen-specific immunotherapy leads to a shift of Th2-dominated allergen-specific immune responses towards a Th1 response, (ii) Immunotherapy induces allergen-specific T cell tolerance. (iii) Immunotherapy has vaccination character and that injection of adjuvant-bound allergens induces a new type of allergen-specific immunity, (iv) Induction of IgG blocking antibodies, which antagonize the action of allergen IgE antibody. e.g. IgG4 antibodies.

This immune response is dominated by high levels of allergen specific IgG1 and IgG4 antibodies which antagonize the effect of IgE. Therapy-induced IgG antibodies are able to block IgE binding and are thus able to inhibit allergen induced mast cell and basophil degranulation, thereby down regulating immediate-type of allergic reactions. Furthermore, the newly induced IgG antibodies may also inhibit IgE-facilitated allergen-presentation to T cells and thus suppress T cell activation and the release of proinflammatory cytokines. Immunotherapy involves the subcutaneous injection of allergen extracts in increasing concentrations and decreasing frequency, starting about twice a week to once a week and later once in 2 weeks. The aim of immunotherapy is to induce a state of "hypo-sensitization "with diminished clinical response on natural re-exposure of allergen.[20]

Prevention: One of the problems while coping with parthenium pollen allergy is how to escape from the stuff because it is impossible to create a pollen-free zone. Pollens of wind-pollinated

plants are small and light, easily get carried far and wide; and easily get inhaled right into the lungs. However, some preventive measures include:[21]

- Allergy symptoms should be under control before the start of the pollen season (February onwards).
- One should avoid the airborne pollen by staying indoors and using air purifiers.
- One should be aware of the times of day that are worse for your allergy during the pollen season.
- Eliminate weeds and grasses in your house garden.

The most effective treatment (if possible) is prevention by avoiding the weed. However, the attempts at eradication of the weed have been unsuccessful. Prevention also includes both biological and chemical control of the weed.

Physical Control: Manual uprooting of parthenium before flowering and seed setting is the most effective method. Uprooting the weed after seed setting will increase the area of infestation. Some landholders have achieved success in ploughing the parthenium weed in the rosette stage before it seeds, but this must be followed up by sowing a crop or direct seeding the perennial pasture. Physical control involves hand weeding, a time consuming and unpleasant job, made worse by the health hazards involved with handling parthenium weed.[22] Burning, another strategy employed to manage weed, is not a useful control strategy for parthenium. However, research suggests that burning for other purposes (e.g., woody weed control) will not result in an increased infestation of parthenium as long as the pasture is allowed to recover before stock is introduced. This too has proved to be inadequate due to two reasons; it requires large quantity of fuel and burning destroys all other economically important plants growing in its vicinity.[23]

Biological: Biotic factors suppress the plant within its native range compared to its increased fitness or vigor in their absence, as in Australia and India, and therefore, the biological control may offer the best long-term solution for the management of this weed.[24] Some of the biocontrol agents used are: Arthropods – Z. bicolorata is the arthropod commonly tried in Bangalore. Pathogens – mainly fungal herbicides called mycoherbicides are used for the control. Antagonistic plants – Cassina uniflora a plant belonging to leguminosae family and also Casia serraceae and Tagetes erecta are known to compete with parthenium and overgrow and thus can be used for its control.

Chemical: Well-known herbicides such paraquat, trifluralin, diphenamid, napropamide and propachlor fail to control parthenium weed. Timing of chemical control is critical. They should be treated when plants are small and before production of seeds and when they are growing to recolonize the infested area.[25] Use of selective chemical herbicide is important and few of them commonly used are: Ametryene, Atrazine, Fomesafan, Metribuzin, Linuron.

In conclusion, the noxious P. hysterophorus grows in a wide variety of habitats and causes changes in above ground vegetation as well as in below ground soil nutrients. It is capable of out-competing native and nonnative palatable plants that are important to livestock. Furthermore, the changes in vegetation and soil nutrients could lead to ultimate changes in other

trophic levels and alter the function of the ecosystem. Appropriate methods for the management of P. hysterophorus are necessary to avoid potential threats to biodiversity and economic losses.

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Fig. 1: Plant of Parthenium Hysterophorus

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