10. Poisonous Plants

ALLERGENS

ALLERGY:

Allergic reactions occur to normally harmless environmental substances known as allergens; these reactions are acquired, predictable, and rapid. Allergy is a disorder of the immune system often also referred to as atopy.

The term Allergy was first defined in 1906 by Clemens von Pirquet as a changed or altered reaction in the body. “Allergy” is derived from the Ancient Greek words *allos* meaning “other” and *ergon* meaning “work”.

When a person develops an unusual response to a substance or condition that is harmless to others, the individual is said to be Allergic.

CLASSIFICATION:

Historically, all forms of hypersensitivity were classified as allergies, and all were thought to be caused by an improper activation of the immune system. Later, it became clear that several different disease mechanisms were mixed up, with the common link to a disordered activation of the immune system.

In 1963, a new classification scheme was designed by Philip Gell and Robin Coombs that described four types of hypersensitivity reactions, known as Type I to Type IV hypersensitivity. With this new classification, the word “allergy” was restricted to only type I hypersensitivities (also called immediate hypersensitivity), which are characterized as rapidly developing reactions.
HYPERSENSITIVITY TYPES

<table>
<thead>
<tr>
<th>Type</th>
<th>Alternative names</th>
<th>Often mentioned disorders</th>
<th>Mediators</th>
</tr>
</thead>
</table>
| I    | Allergy (immediate) | • Atopy  
• Anaphylaxis  
• Asthma | IgE |
| II   | Cytotoxic, antibody-dependent | • Autoimmune hemolytic anemia  
• Thrombocytopenia  
• Erythroblastosis fetalis  
• Goodpasture's syndrome | IgM or IgG |
| III  | Immune complex disease | • Serum sickness  
• Arthus reaction  
• Systemic lupus erythematosus (SLE) | IgG |
| IV   | Delayed-type hypersensitivity (DTH), cell-mediated immune memory response, antibody-independent | • Contact dermatitis  
• Mantoux test  
• Chronic transplant rejection  
• Multiple sclerosis | T-cells |
| V    | Autoimmune disease | • Grave's disease  
• Myasthenia Gravis  
• Hashimoto's thyroiditis  
• Systemic lupus erythematosus | IgM or IgG |

Type V is an additional type that is sometimes used as a distinction from Type II. Instead of binding to cell surface components, the antibodies recognize and bind to the cell surface receptors, which either prevent the intended ligand binding with the receptor or mimics the effects of the ligand, thus impairing cell signalling.

CHARACTERISTICS:

It is characterized by excessive activation of certain white blood cells called mast cells and basophils by a type of antibody known as IgE, resulting in an extreme inflammatory response.

Common allergic reactions include eczema, hay fever, asthma, food allergies, and reactions to the venom of stinging insects such as bees. In some people, severe allergies to environmental or dietary allergens or to medication may result in life-threatening anaphylactic reactions and potentially death.
The exact cause of allergy is still undetermined. The reason, that why some pollens are causing allergy to certain individuals and not to others, is still unknown.

**GENETIC BASIS:**

There is also a probability of inheriting a tendency towards allergy.

- Over 50% of allergic patients give a history of allergic diseases in other members of the family.

- Transmission of the allergic tendency seems to occur twice as frequently through the female as through the male.

- A child with both allergic parents may develop allergy at earlier stage than a child with only one allergic parent.

- A child does not always inherit the same type of allergy as the parent, although this is usually the case.

<table>
<thead>
<tr>
<th>Affected Organ</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nose</td>
<td>swelling of the nasal mucosa (allergic rhinitis)</td>
</tr>
<tr>
<td>Sinuses</td>
<td>allergic sinusitis</td>
</tr>
<tr>
<td>Eyes</td>
<td>redness and itching of the conjunctiva (allergic conjunctivitis)</td>
</tr>
<tr>
<td>Airways</td>
<td>Sneezing, coughing, broncho constriction, and dyspnea, sometimes outright attacks of asthma, in severe cases the airway constricts due to swelling known as angioedema</td>
</tr>
<tr>
<td>Ears</td>
<td>feeling of fullness, possibly pain, and impaired hearing.</td>
</tr>
<tr>
<td>--------------------------</td>
<td>---------------------------------------------------------</td>
</tr>
<tr>
<td>Skin</td>
<td>rashes, such as eczema and hives (urticaria)</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>abdominal pain, bloating, vomiting, diarrhoea</td>
</tr>
</tbody>
</table>

**ALLERGEN:**

Allergens are antigenic substances capable of sensitizing the body in such a way that unusual responses occur in hypersensitive individuals. Almost any substance, whether of biologic, chemical or synthetic origin may prove to be allergenic. In addition, numerous other factors are allergy producing:

- Emotional factors
- Atmospheric factors
- Psychosomatic factors
- Chronic types of infection

The allergen concerned with the patient’s symptoms must be antigenic; i.e., it must be capable of initiating an antibody response.

The antigenic fraction of ragweed pollen causes a susceptible person’s body to produce special protein molecules (antibodies), some of which circulate in the blood (circulating antibodies) and other which become attached to the cells of nasal membranes (fixed antibodies). These particular antibodies have a special
affinity for the chemical components of ragweed family pollens only and not to other pollens or allergenic substances. Thus antibodies are considered specific.

When the body is first subjected to the allergen, the condition is referred to as primary exposure. As no antibodies are formed previously therefore no symptoms of allergy are produced. However during the subsequent exposures the allergen contacts the fixed antibodies and an antigen antibody reaction occurs. The *antigen-antibody reaction* causes liberation of histamine & other mediators of allergic symptoms, including leukotriene and bradykinin from the cells of certain tissues and organs called shock tissues or shock organs.

If the state of shock is confined to the area of introduction of the allergen, the condition is localized reaction; if its effects extend beyond this area, it may be a generalized or constitutional reaction.

**MECHANISM OF ACTION:**

The constant region of IgE antibodies (shown in blue) has a binding site for a receptor present on the surface of basophils and their tissue-equivalent the mast cell. These cell-bound antibodies have no effect until and unless they encounter allergens (shown in red) with epitopes that can bind to their antigen-binding sites.

When this occurs, the mast cells to which they are attached, explosively discharge their granules by exocytosis.

The granules contain a variety of active agents including histamine; Synthesize and secrete other mediators

including leukotrienes and prostaglandins.
Release of these substances into the surrounding tissue causes local anaphylaxis: swelling, redness, and itching. In effect, each IgE-sensitized mast cell is a tiny bomb that can be exploded by a particular antigen. The most common types of local anaphylaxis are:

- Allergic rhinitis (hay fever) in which airborne allergens react with IgE-sensitized mast cells in the nasal mucosa and the tissues around the eyes;
- Bronchial asthma in which the allergen reaches the lungs either by inhalation or in the blood;
- Hives (urticaria) where the allergen usually enters the body through food.

Leukotrienes are far more potent than histamine in mediating these reactions. Leukotrienes and prostaglandins are derivatives of arachidonic acid (AA) an unsaturated fatty acid produced from membrane phospholipids. The principal pathways of arachidonic acid metabolism are:

- The 5-lipoxygenase pathway, which produces a collection of leukotrienes (LT) and
- The cyclooxygenase pathway, which yields a number of prostaglandins (PG) and thromboxanes (Tx).

All three are synthesized by monocytes and macrophages. Mast cells and basophils generate a mixture of leukotrienes. The products of both pathways act in concert to cause inflammation with prostaglandins producing fever and pain.
Aspirin, ibuprofen, and certain other NSAIDs achieve their effects (fever and pain reduction) by blocking the activity of the enzyme cyclooxygenase.

Systemic Anaphylaxis:
Some allergens can precipitate such a massive IgE-mediated response that a life-threatening collapse of the circulatory and respiratory systems may occur.

Frequent causes:
- Insect stings (e.g., bees, wasps)
- Many drugs (e.g., penicillin)
- A wide variety of foods e.g. egg white, cow’s milk, and nuts are common offenders in children; fish and shellfish are frequent causes of anaphylaxis in adults.

Treatment of systemic anaphylaxis centres on the quick administration of adrenaline, antihistamines, and IV fluid replacement, if shock has occurred.
CASE HISTORY:

To determine the circumstances surrounding the patient's allergy, the allergist must record all details regarding the allergic attacks, including data on the type of occupation and the family background. Information concerning the place, time and mode of onset of past symptoms, as well as those causing the most recent attack, is recorded in the case history or allergic history of the individual.

As stated on a typical case history report, the entries include:

Name and sex

Marital status

Chief complaint

Present illness

   Age of onset

   Date

   Place, time, and mode of onset

Seasonal variation

Duration

What relieves attacks

Present attack

   Date of onset

   Place of onset

   Mode of onset

   Sneezing
Nasal discharge
Wheeze
Cough
Headache

Symptoms affected by

Meals
Drugs
Exertion
Excitement
Weather changes
Season Changes
Wind
Smoke or fumes
Time of the day
Mowing lawn
Rain
Working in garden
Automobile rides
Playing golf
Riding horse
Cleaning house

Change of environment

Change of occupation

Other points of information include the types of medication the patient may be taking and the conditions of the home environment (heating system, type of floor covering, presence of household pets, kinds of cosmetics used, nature of bed covers and pillows, and numerous other details. Past medical history may be inquired. Allergic symptoms of the paternal and maternal relatives can be a good clue.

A complete case history includes both a physical and a laboratory examination, the latter includes reports on urine, blood, sputum, and nasal smears. In addition, results of a radiograph and ECG are customary. Skin tests and blood tests are also concurrent with all this practice.

**SKIN TEST:**

For assessing the presence of allergen-specific IgE antibodies, allergy skin testing is preferred over blood allergy tests because it is more sensitive and specific, simpler to use, and less expensive. Skin testing is also known as “puncture testing” and “prick testing” due to the series of tiny puncture or pricks made into the patient’s skin.

Small amounts of suspected allergens and/or their extracts (pollen, grass, mite proteins, peanut extract, etc.) are introduced to sites on the skin marked with pen or dye (the ink/dye should be carefully selected, lest it cause an allergic response itself).

A small plastic or metal device is used to puncture or prick the skin. Sometimes, the allergens are injected "intradermally" into the patient's skin, with a needle and
syringe. Common areas for testing include the inside forearm and the back. If the patient is allergic to the substance, then a visible inflammatory reaction will usually occur within 30 minutes. This response will range from slight reddening of the skin to a full-blown hive (called "wheal and flare") in more sensitive patients.

Interpretation of the results of the skin prick test is normally done by allergists on a scale of severity, with +/- meaning borderline reactivity, and 4+ being a large reaction.

Increasingly, allergists are measuring and recording the diameter of the wheal and flare reaction. Interpretation by well-trained allergists is often guided by relevant literature. Some patients may believe they have determined their own allergic sensitivity from observation, but a skin test has been shown to be much better than patient observation to detect allergy.

**Clinical Designations of Skin reactions**

<table>
<thead>
<tr>
<th>Designation</th>
<th>Symbol</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>-</td>
<td>No reaction or no different than control.</td>
</tr>
<tr>
<td>Doubtful</td>
<td>±</td>
<td>No appreciable difference from control other than slight erythema</td>
</tr>
<tr>
<td>One Plus</td>
<td>+</td>
<td>Erythema smaller than 20 mm in diameter.</td>
</tr>
<tr>
<td>Two Plus</td>
<td>++</td>
<td>Erythema larger than 20 mm in diameter but no wheal.</td>
</tr>
<tr>
<td>Three Plus</td>
<td>+++</td>
<td>Definite wheal with surrounding erythema.</td>
</tr>
<tr>
<td>Four Plus</td>
<td>++++</td>
<td>Wheal with definite pseudopods and erythema.</td>
</tr>
</tbody>
</table>
If a serious life threatening anaphylactic reaction has brought a patient in for evaluation, some allergists will prefer an initial blood test prior to performing the skin prick test. Skin tests may not be an option if the patient has widespread skin disease or has taken antihistamines sometime the last several days.

**BLOOD TEST:**

Various blood allergy testing methods are also available for detecting allergy to specific substances. This kind of testing measures a “total IgE level” - an estimate of IgE contained within the patient's serum. This can be determined through the use of radiometric and colormetric immunoassays. Radiometric assays include the radioallergosorbent test (RAST) method, which uses IgE-binding (anti-IgE)
antibodies labeled with radioactive isotopes for quantifying the levels of IgE antibody in the blood. Other newer methods use colorimetric or fluorometric technology in the place of radioactive isotopes. Some “screening” test methods are intended to provide qualitative test results, giving a “yes” or “no” answer in patients with suspected allergic sensitization.

A low total IgE level is not adequate to rule out sensitization to commonly inhaled allergens. Statistical methods, such as ROC curves, predictive value calculations, and likelihood ratios have been used to examine the relationship of various testing methods to each other. These methods have shown that patients with a high total IgE have a high probability of allergic sensitization, but further investigation with specific allergy tests for carefully chosen allergens is often warranted.

**TYPES OF ALLERGY:**

The type of symptoms depends on the shock organ affected by the particular allergen and its path of entry into the body.

1) Inhalant allergens are distributed in the atmosphere and contact the nasal or buccal mucosa during respiration.

2) Ingestant allergens occur in food stuffs and are swallowed.

3) Injectant allergens may be present in solutions intended for parenteral administration.

4) Contactant allergens come into direct contact with epithelium.

5) Infectant allergens are metabolic wastes and growth products of pathogenic microorganisms.

6) Infestant allergens are parasitic microorganisms on or in the body.
Moreover, allergy may be caused by heat or cold (physical allergy), changes in climate (environmental allergy), anger or frustration (psychosomatic allergy) and others.

1) INHALANT ALLERGENS:

An inhalant allergen is usually indicated, if the symptoms are restricted to the nasal mucosa and are manifested by sneezing, lacrimation, itching and swelling of nose and eyes. (However certain food allergens may also cause such symptoms e.g. chillies). The condition is known as sinusitis or hay fever.

Occurrence of the symptoms during certain months of the year indicates seasonal hay fever. Because this condition is usually associated with the release of pollen grains from certain plants, the term pollinosis is often used. Non seasonal hay fever or perennial rhinitis may be caused by inhalants other than pollens e.g. mold spores, dust, animal epidermis or dander, feathers, cotton linters, volatile oils and numerous other factors.

Atmospheric pollens are liberated chiefly by wind pollinated plants and are usually small (15–45 µ in dia), light, non adhesive and relatively smooth e.g. oak/walnut trees, Bermuda/timothy grasses and ragweed are examples of plants having such flowers. The Paper Mulberry (Broussonetia papyrifera, syn. Morus papyrifera) is an example of tree spreading such pollens causing allergy.

In contrast pollens of insect pollinated plants are usually larger (up to 200 µ in dia), heavier, adhesive and somewhat spiny. Rose, clover and honeysuckle are examples of such plants. Wind pollinated flowers are rarely colored and generally not fragrant as they don’t need to attract insects for pollination process.

Non seasonal hay fever can occur at any time of the year without any regularity. Such inhalant allergens may occur at home, work place or any place visited frequently by patient. In the home cotton pillows, sheets, blankets etc usually shed
“linters” or fragments of cotton fibers, that are light enough to float in the air. Feather pillows are also a source of allergens, specially if these are old enough and feathers are disintegrating. So these allergies can only be prevented by avoiding the known allergens.

Volatile oils from plants being used in perfumes and toiletries may cause allergies e.g. sandalwood oil has been reported in the medical literature to be allergenic.

Animal epidermis or animal dander (epithelial scales) is a frequent source of allergenic matter. For example cats, dogs, guinea pigs and others are occasionally responsible for the cough, wheeze or asthmatic attack. Much of the non seasonal hay fever is thought to be caused by different types of fungal spores e.g. Aspergillus etc.

Persons allergic to mold spores are usually allergic to dust as well. Dust is almost indefinable, not as it differs from one place to another place but it is composed of mold spores, cotton linters, animal danders, sizing from rugs and carpets and numerous others.

2) INGESTANT ALLERGENS:

Food allergen usually cause gastrointestinal symptoms, & they may also cause skin rash, puffed lips and tongue, migraine, rhinitis or some other serious effects such as bronchial asthma. In food allergy, the activity of the allergen is not localized in one organ or area of the body, but is transferred to other parts of the body through blood. Thus an atopic dermatitis, such as tomato rash, strawberry rash or that caused by eating oranges, chocolate or shell fish, is developed by the patient.

Some persons may not know that they have allergy but they do know about some particular food stuffs which lead to dire consequences, if eaten.
Some of the most common allergens ingested by the children are foods considered essential to proper diet and growth such as cow’s milk, orange juice, cod liver oil or other vitamin containing fish liver oils. The major symptom for these may be colic.

Different extracts are available for the diagnosis of these allergies but they have little or no value in treatment. The most satisfactory method for combat is elimination of offending substance from diet.

Milk allergy is a specific immunologic, antigen antibody response owing partially to a lactalbumin. Because heating or boiling alters this protein, evaporated milk may be used as an effective substitute for cow’s milk. Milk allergy may result in severe dermatitis, bronchitis and asthma.

3) **INJECTANT ALLERGENS:**

Allergic reactions to penicillin injections are well known to most of the people. Allergic reaction in response to penicillin leads to anaphylactic shock. These reactions occur with a frequency of 1 – 5 per 10,000 patient courses of penicillin. Skin testing for penicillin allergy is of definite value, but tests must be conducted in controlled condition.

6-Amino penicillinic acid (6APA) and 7-Aminocephalosporinic acid as well as semi synthetic penicillins and cephalosporins, cause +ve intracutaneous reaction in most susceptible patients. Therefore such antibiotics should be given with care and always after applying the test dose.

Other injectable products causing allergies are liver extracts, antitoxins and the glandular products. The symptoms in each case are similar to those of antibiotic; itching of the palms of hands and soles of the feet, erythema, and peeling of the skin are characteristics.
Stinging insects e.g. bees etc are also considered as source of injectant allergens. Stings of such insects can induce severe local and constitutional reactions, sometimes even leading to death. Such patients can be immunized by using injections of antigens because one injection is common to all bees; however, each species has its own additional specific antigen(s).

4) CONTACTANT ALLERGENS:

A lot of substances have been identified as contactant allergens. One of them is Poison ivy (toxicodendron radicans). Other species are western poison oak and eastern poison oak and poison dogwood. All of them contain a non-volatile Phenolic Principle known as urushiol and all produce allergenic symptoms in hypersensitive individuals. Indication of this allergy are watery blisters and pruritis. The blisters break open and the exuding fluid forms new blisters rapidly.

Other examples of plants causing this type of allergy are buttercups, chrysanthemums, daffodils, English ivy, lobelia and dozens of others.
Sometimes contact dermatitis has also been caused by aeroallergens, such as pollen grains having oils, hairs from different kind of leaves & flowers and even small fragments of plant tissues carried by smoke emanating from brush fires, grass fires and burning leaves.

A term hypoallergenic cosmetic is used for the products from which manufacturers have removed certain known allergenic substances.

Frequently individuals can’t tolerate wool in clothing, blankets or even in the form of wool fat (lanolin) in cosmetics. Soaps and soap powders, plain detergents and enzyme detergents, nail polishes and nail polish removers, hair dyes and hair sprays are listed among the major causes of contact dermatitis.

5) **INFECTANT ALLERGENS:**

Numerous living organisms may cause allergy through the products they release during their metabolism in the human body.
Some individuals harbor certain types of bacteria, protozoans, molds, helminths, and other parasitic forms which are responsible for chronic illness by their continual presence in the body. The patient may or may not be aware of this infection because it may or may not show recognizable symptoms. Metabolic products of growth of these organisms may be of such nature that the individual becomes sensitized.

For example in the chronic bacterial infection of the bronchioles, known as bronchiectasis, the constant presence of bacterial wastes may sensitize the allergic individual. Thus, the person may exhibit allergic symptoms but does not respond positively to skin tests for inhalant allergens. In this case, the bacterial metabolic wastes are considered as infectant allergens.

6) INFESTANT ALLERGENS:

In a manner somewhat similar to the infectants, parasitic organisms may sensitize the human body. Invasions of hookworms, tapeworms, pinworms, threadworms, dermatophytes, and other forms have caused allergic response in susceptible individuals. Growth products and metabolic wastes of these parasites are constantly present in the body and are referred to as infestant allergens.

TREATMENT OF ALLERGY:

There have been enormous improvements in the medical treatments used to treat allergic conditions. There are different ways to treat allergy.

Some of them are:

- Pharmacotherapy
- Immunotherapy
- Allergy Shot treatment
Traditionally treatment and management of allergies involved simply avoiding the allergen in question or otherwise reducing exposure.

For instance, people with cat allergies were encouraged to avoid them. While avoidance may help to reduce symptoms and avoid life-threatening anaphylaxis, it is difficult to achieve for those with pollen or similar air-borne allergies.

Strict avoidance still has a role in management, and is often used in managing food allergies.

**PHARMACOTHERAPY:**

Several antagonistic drugs are used to block the action of allergic mediators, or to prevent activation of cells and degranulation processes. These include antihistamines, cortisone, dexamethasone, hydrocortisone, epinephrine (adrenaline), theophylline and cromolyn sodium.

Anti-leukotrienes, such as Montelukast or Zafirlukast, are FDA approved for treatment of allergic diseases. Anti-cholinergics, decongestants and mast cell stabilizers are also commonly used. These drugs help to lessen the symptoms of allergy, and are essential in the recovery of acute anaphylaxis. They play little role in chronic treatment of allergic disorders.
**Antihistamines**

<table>
<thead>
<tr>
<th>Non sedating</th>
<th>Sedating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cetirizine hydrochloride</td>
<td>Alimemazine tartrate</td>
</tr>
<tr>
<td>Desloratadine</td>
<td>Chlorphenamine maleate</td>
</tr>
<tr>
<td>Fexofenadine hydrochloride</td>
<td>Clemastine</td>
</tr>
<tr>
<td>Levocetirizine hydrochloride</td>
<td>Cyproheptadine hydrochloride</td>
</tr>
<tr>
<td>Loratadine</td>
<td>Hydroxyzine hydrochloride</td>
</tr>
<tr>
<td>Mizolastine</td>
<td>Ketotifen</td>
</tr>
<tr>
<td></td>
<td>Promethazine hydrochloride</td>
</tr>
</tbody>
</table>

**Anti leukotrienes**

- Montelukast
- Zafirlukast

**Decongestant**

- Pseudoephedrine Hydrochloride

**IMMUNOTHERAPY:**

Desensitization or hyposensitization is a treatment in which the patient is gradually vaccinated with progressively larger doses of the allergen in question. This can either reduce the severity or eliminate hypersensitivity altogether. It relies on the progressive skewing of IgG antibody production, to block excessive IgE production seen in allergies. In a sense, the person builds up immunity to increasing amounts of the allergen in question.

Studies have demonstrated the long-term efficacy and the preventive effect of immunotherapy in reducing the development of new allergy.
If the treatment is conducted prior to the time of pollination of the plants, it is called pre-seasonal; if it is maintained throughout the year, it is termed as perennial and if the treatment is started during the symptomatic period, it is known as co-seasonal.

A second form of immunotherapy involves the intravenous injection of monoclonal anti-IgE antibodies. These bind to free and B-cell associated IgE; signalling their destruction. They do not bind to IgE already bound to the Fc receptor on basophils and mast cells, as this would stimulate the allergic inflammatory response. The first agent of this class is Omalizumab. While this form of immunotherapy is very effective in treating several types of allergies, it should not be used in treating the majority of people with food allergies.

**SUBLINGUAL IMMUNOTHERAPY:**

A third type, Sublingual immunotherapy, is an orally-administered therapy which takes advantage of oral immune tolerance to non-pathogenic antigens such as foods and resident bacteria. This therapy currently accounts for 40 percent of allergy treatment in Europe. In the United States, sublingual immunotherapy is gaining support among traditional allergists and is endorsed by doctors who treat allergy. In a preliminary study, Italian researchers found that putting honeybee venom under the tongue was safe and significantly reduced reactions in people allergic to bee stings. It involves putting extracts of allergens under the tongue. Like the shots, sublingual immunotherapy reduces allergic sensitivity in many patients over time.

**ALLERGY SHOT TREATMENT:**

Allergy shot treatment is the closest thing to a ‘cure’ for allergic symptoms. This therapy requires a long-term commitment. [Allergy shots, also called "immunotherapy," are given to increase the tolerance to the substances (allergens) that provoke allergy symptoms. They usually are recommended for people who
suffer from severe allergies or for those who have allergy symptoms for more than 3 months each year. They do not cure allergies, but reduce your sensitivity to certain substances.

Allergy shots are given regularly (in the upper arm), with gradually increasing doses. When starting immunotherapy, you will need to go to your healthcare provider once or twice a week for several months. The dose is increased each time until the maintenance dose is reached. If the shots are effective, you will go to your healthcare provider every 2 to 4 weeks for 2 to 5 or more years. You may become less sensitive to allergens during this time, and your allergy symptoms will become milder and may even go away completely.

**TYPES OF ALLERGY:**

The type of symptoms depends on the shock organ affected by the particular allergen and its path of entry into the body.

1) **Inhalant allergens** are distributed in the atmosphere and contact the nasal or buccal mucosa during respiration.

2) **Ingestant allergens** occur in food stuffs and are swallowed.

3) **Injectant allergens** may be present in solutions intended for parenteral administration.

4) **Contactant allergens** come into direct contact with epithelium.

5) **Infectant allergens** are metabolic wastes and growth products of pathogenic microorganisms.

6) **Infestant allergens** are parasitic microorganisms on or in the body.

Moreover, allergy may be caused by heat or cold (physical allergy), changes in climate (environmental allergy), anger or frustration (psychosomatic allergy) and others.
POISONOUS PLANTS:

1. **Oleander** (Kaneer, Sim al khamaar)
2. **Thevetia peruviana** (Yellow oleander, Peela kaneer)
3. **Castor oil seed** (Arand, baid e injeer)
4. **Croton tiglium** (Jamal ghota)
5. **Abras precatorius** (Chashm e kharoos, gangachi, ratti)
6. **Calotropis** (Ashkhar, aak)
7. **Datura** (tatura, tatura sufai)
8. **Atropa belladonna** (Shah beezak, yabraj, angoor e shifa)
9. **Colchicinie** (Suranjan e shireen, gul e hasrat, Darkom)
10. **Nux – vomica** (Dog button, Kuchla)

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1. **OLEANDER:**

*Scientific Name:* (i) Nerium odorum (ii) Nerium oleander

*Common Name:* White oleander, Kanair (Urdu), Ganderey (Push). Khar zehr, sim al khamaar (N. oleander)

*Family:* Apocynaceae

*Toxic Parts:* All parts are toxic. Even smoke from burning plant is toxic.

*Geographical Source:* Found in all parts of Pakistan, India and Afghanistan. Also cultivated in gardens for ornamental purposes

*Plant Description:* The plant is evergreen shrub; leaves are dark green in color with prominent midrib and usually rise in groups of 3 on a stem. Flowers are pink, or white which are having 5 petals. The fruit is a capsule with fluffy seeds. The plant exudes a thick white sap when branches or stem is broken out.
CONSTITUENTS:

- The plant contains crystalline glycoside – Nerin which is 6 – 7 times more poisonous than strychnine. Nerin consists of:
  - Nerodine: its effects are similar to digitalis.
  - Neiodorein: Its effects are ‘picrotoxin’ like, i.e. It causes muscular twitching and spasm more powerful than strychnine
  - Karabin: Its effects on heart are similar to digitalis and on spinal cord it acts like strychnine.

According to new research oleander also contains:

- Oleandrin
- Nerioside
- Oleandrigenine
- Diginoside
- Rosagenin

POISONING:

FATAL DOSE:

- Even a single leaf chewed is lethal for children.
- Between 7 to 20 or a handful of leaves have been ingested by adult patients but recovered completely.
• Animals poisoned by eating the plant often develop bloody diarrhea, due to a direct effect on the gastrointestinal tract.

• In birds, as little as 0.12 to 0.7 g of the plant extract has caused death.

• As little as 100 g of dried leaves can be fatal to a horse.

FATAL PERIOD:

☐ If not treated, Death occurs within 24 hours.

CAUSES OF POISONING:

• When children playing in gardens nearby the plant taste the plant mistakenly.

• When meat is cooked directly on smoky fire of plant or stirred with the stems.

• Honey made by bees visiting the flowers has produced toxic effects.

• Inhalation of smoke emanating from burning oleander.

• Due to over dosage, when used by women as abortifacient.

• When used for suicidal purposes.

SIGNS & SYMPTOMS:

GIT: Nausea, vomiting, bloody diarrhoea, abdominal pain, difficulty in swallowing, increased salivation and locked jaws.

Body temperature: Hypothermia

Respiration: Breathing and respiration is hurried & rapid but finally respiratory failure occurs.

CVS: Slow and weak pulse, hypotension, cardiac arrhythmias, bradycardia and finally cardiac failure.

CNS: Restlessness, anxiety, drowsiness, and finally coma occurs secondary to cardiac failure.

Eyes: Mydriasis.
**Muscles:** Muscular twitching of extremities deepens into tetanic spasms which frequently affect one side more than the other.

**TREATMENT:**

1) **Supportive treatment:**
   - Stomach wash with KMnO₄ solution.
   - Gut decontamination by means of emesis or lavage.
   - Administration of activated charcoal.
   - Saline and glucose solution for correction of electrolyte imbalance.

2) During severe respiratory paralysis artificial respiration is essential.
3) Correction of electrolyte imbalance.
4) Correction of severe bradycardia with atropine or electrical pacing.
5) Corrections of ventricular arrhythmias could be treated with phenytoin or lignocaine.
6) Treatment of hyperkalaemia should aim at lowering the serum potassium level with insulin, glucose, sodium bicarbonate and ion-exchange resins.
7) Calcium chloride is contraindicated.

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**2. YELLOW OLEANDER:**

- **Scientific Name:** Thevetia peruviana
- **Common Name:** Yellow oleander, Peela kanair, Zard kanair.
- **Family:** Apocynaceae
- **Toxic Parts:** All parts are toxic specially the kernels of the fruit. The absorption of the equivalent of 2 *thevetia* leaves may be sufficient to kill a 12½ Kg child.
- **Geographical Source:** The plant is native to central & South America but is now frequently grown in parks and gardens in tropical and subtropical areas.
**Plant Description:** It is a small ornamental tree which grows to about 10 to 15 feet high.

- Leaves are spirally arranged, linear and about 13 – 15 cm in length.
- Flowers are bright yellow and funnel shaped with 5 petals spirally twisted.
- The fruits are somewhat globular, slightly fleshy and have a diameter of 4 – 5 cm. The fruits which are green in colour, become black on ripening. Each fruit contains a nut which is longitudinally and transversally divided.
- All parts of the plant contain milky juice.

**CONSTITUENTS:**

**Cardiac glycosides:**

- Thevetine A
- Thevetine B
- Thevetoxin
- Peruvoside
- Ruvoside &
- Neriifolin

**POISONING:**

*Causes of Poisoning:*
Accidental ingestion of the seeds by the children may cause poisoning.

Suicidal ingestion is also common in some areas.

**Mode of Action:**

- Cardiac glycosides exert a digoxin like effect by inhibiting the Na–K ATP enzyme system. The increased intracellular Na concentration and the increased serum K⁺ concentration produce negative chronotropic and positive ionotropic effect e.g. ↓ H.R. & ↑ C.O.

- The resulting syndrome resembles digitalis poisoning with marked hyperkalemia.

**Fatal Dose:**

- For adult human – the *kernels of about 10 fruits* and for children – the *kernel of 1 fruit* may be fatal.

- Cattle grazing on grass under *Thevetia peruviana* trees are known to have died and nuts are lethal to chickens.

**ACUTE POISONING:**

**Ingestion:**

- *Thevetia peruviana* poisoning closely resembles digitalis poisoning with gastrointestinal and cardiac symptoms.

- Local irritation of mucus membranes and mouth is followed by nausea, vomiting and giddiness within hours.

- Other clinical features are severe diarrhoea, abdominal pain, dilated pupil and occasionally convulsions.

**Skin exposure:**

- Burning sensation of the skin due to sap.

**Eye contact:**

- Severe eye irritation is caused by the sap.

**SYSTEMIC EFFECTS:**
Cardiovascular effects:
In small doses it stimulates heart while at large doses it depresses and stops ventricular contractions. ECG changes include:

- Sinus bradycardia, P – R prolongation, Ventricular tachycardia leading to ventricular fibrillations which is the usual cause of death.

Respiratory system:
- It has little effect on respiration.

Neurological Effects:
- CNS: Drowsiness, coma, convulsions.
- PNS: Paraesthesia, weakness.

Skeletal & smooth muscles:
- It has a direct stimulant effect on the smooth muscles of the intestine, bladder, uterus and blood vessel walls.

Renal effects:
- Acute renal failure may occur secondary to cardiogenic shock.

Reproductive System:
- Abortifacient

ENT:
- Local effects, burning sensation and dryness of throat could occur.

Fluid and Electrolyte disturbances:
- GIT fluid loss often leads to dehydration and hypovolemic shock.

MANAGEMENT/TREATMENT:
General principles:
- Admit the patient to a hospital immediately.
ii. In severe poisoning admit to an intensive care unit for immediate cardiac monitoring.

iii. Treatment usually depends on the severity of poisoning and includes immediate gastric decontamination, correction of arrhythmias & electrolyte disturbance.

iv. Frequent ECG or continuous cardiac monitoring are necessary.

v. Check electrolytes regularly; particularly serum K⁺ ion levels.

vi. If consciousness is not impaired, induce emesis or perform gastric lavage.

vii. Activated charcoal should also be given.

**Biomedical analysis:**

- Bradycardia may require atropine.
- Ventricular arrhythmia may be controlled with lidocaine 1mg/kg (IV) followed by continuous infusion of 1 – 4 mg/ min.
- Hyperkalemia should be treated with insulin, glucose and NaHCO₃.

### 3. CASTOR OIL SEED:

**Scientific Name:** Ricinus communis

**Common Name:** Arand, baid e injeer, castor bean, African coffee tree.

**Family:** Euphorbiaceae

**Toxic Parts:** Broken seeds of castor.

**Geographical Source:** It is indigenous to India. It is also found in Pakistan. It is common in South America. Commercially supplied from India, South America, Brazil & Italy. It is also found in countries like Ethiopia, U.S.A., S. Africa and Tanzania.

**Plant Description:** It may be annual herb, shrub or tree depending upon the climate. In India it attains the height of 40 feet and becomes a perennial tree but in cooler regions it is either shrub or annual herb.
Leaves are large, alternate, simple, long stalked, palmately lobed having 5 – 12 lobes and dentate or serrate.

Fruit is a 3 – celled capsule having spines over it.

CONSTITUENTS:

- 46 – 53% fixed oil i.e. Ricinolic acid.
- Iso ricinolic acid.
- Stearic acid
- Dihydroxystearic acid.
- Ricin
- Ricinin
- Lipase and other enzymes.
- 26% protein as globulin, albumin, glycoproteins & nucleoalbumin.

POISONING:

*Causes of Poisoning:*

- Poisoning occurs by the ingestion of seeds.
If seeds are swallowed without chewing, then there will be no poisoning as the seed is covered with very tough testa and stomach is unable to digest it.

When testa is broken after chewing, Ricin is released & absorbed by the intestinal mucosa and causes poisoning.

Allergic reactions have been observed among workers exposed to Ricinus communis in commercial cultivation & gardening.

Also people living in vicinity of castor oil factories have developed allergies.

Mode of Action:

Ricin is the main toxic substance, similar in its action to that of bacterial toxin.

It has also got the antigenic properties, hence activates the body immune system to produce antibodies. This process leads to agglutination and lyses of erythrocytes.

Ricin is also converted into ricinolic acid & glycerine by the action of lipase enzyme. Ricinolic acid is neutralized in body leading to formation of Na/K ricinolates which produce gastric irritation.

Fatal Dose:

7 mg of Ricin or 8 seeds cause death from 2 – 6 days.

SIGNS AND SYMPTOMS:

BY INGESTION (AFTER CHEWING):

- Burning pain in throat
- Nausea and painful copious vomiting
- Diarrhoea which sometimes becomes bloody
- Excessive salivation
- Excessive thirst
- Abdominal pain and cramps
In the next few days following symptoms will be observed:

- Severe dehydration.
- ↓ urinary output.
- ↓ Blood volume
- Vertigo
- Rapid and feeble pulse
- Cold clammy skin
- Anaphylactic shock

AFTER INHALATION:

- Asthma
- Rhinitis
- Coma
- Convulsions

BY SKIN:

- Eczema
- Conjunctivitis
- If death does not occur within 3 – 5 days, the patient recovers.

TREATMENT:

GENERAL PRINCIPLES:

i. Induction of emesis.

ii. Gastric lavage.

iii. Administration of stimulants like tea or coffee.

iv. I/V Saline glucose in order to resolve dehydration.

v. Oral NaHCO₃ is given to alkalinize the urine

vi. Antispasmodics are given for colic.
vii. In case of excessive haemolysis blood is also transfused.

viii. Anti allergic drugs should be administered in case of exposure to powder.

ix. Corticosteroids should be given in bronchial asthma.

POST – MORTEM APPEARANCE:

i. Fragments of seeds can be found in stomach

ii. Inflamed bowel, occasional erosion and sub mucous haemorrhages.

iii. Haemorrhages of internal organs.

iv. Powder causes local irritation of mucus membranes.

USES:

i. It stimulates catharsis which increases peristalsis.

ii. As a constituent of flexible collodion (syrupy solution of pyroxylin in ether and alcohol, often used as a surgical dressing or to hold dressings in place).

iii. In soap manufacturing.

iv. In varnishes & paint manufacturing.

v. As a lubricant.

4. CROTON TIGLIUM:

*Scientific Name*: Croton tiglium

*Common Name*: Jamal ghota, Croton oil seed.

*Family*: Euphorbiaceae
**Toxic Parts:** Ripened seeds or oil obtained from the seeds.

**Geographical Source:** It is indigenous to India and also found in some areas of Pakistan.

**Plant Description:** It is a small tree or shrub. It attains height of 15 – 20 feet. It has a few spreading branches which bear alternate, petiolate leaves.

- The leaves are ovate, smooth, dark green on upper surface and furnished by 2 glands at the base.
- Petals of the flowers are straw coloured. Female flowers are on lower side of the plant whereas male flowers are on the upper side.
- Fruit is a 3 – celled capsule having 3 seeds and lacks spines.
- Seeds resemble castor seeds in colour and readily lose their caruncles. Testa of seed is hard, brittle and dull coloured. Seed does not have marked odour and taste is oily following unpleasant acidity.
- Croton oil is obtained by expression of seeds after depriving from shells and has a disagreeable odour, acrid taste and pale- yellow color.

**CONSTITUENTS:**

i. Contains 50% fixed oil including:

- Croton resin
- Stearic acid glycerides.
• Palmitic acid glycerides.
• Myristic acid glycerides.
• Oleic acid glycerides.

ii. 2 – 18% protein as crotin which is a mix of croton globulin & croton albumin. This substance resembles with ricin.

iii. Diesters of tricyclic diterpenes phorbol (tumor – promoting phorbol formate, phorbol butyrate, and phorbol crotonate.)

iv. Crotonoside.

v. Crotonine.

POISONING:

Causes of Poisoning:

i. Poisoning occurs by eating the seeds or inhaling their dust.

ii. Accidental poisoning occurs from the use of croton oil as purgative or abortifacient.

iii. When oil is ingested by mistake.

Fatal Dose:

For a man, about four seeds & for a horse, about 15 seeds represent a lethal dose.

SIGNS AND SYMPTOMS:

➢ Ingestion:

  o Burning pain in mouth, stomach & throat
  o Nausea and vomiting.
  o Vertigo
  o Bloody stool
  o Excessive salivation.
  o Excessive thirst.
- Severe gripping pain.
- Tachycardia
- GIT irritation
- Powerful purgation (purging)
- Respiratory collapse.

**EXTERNAL APPLICATION OF OIL:**

- Blistering

**TREATMENT:**

**General principles:**

i. Induction of emesis.

ii. Gastric lavage.

iii. Demulcent drinks such as milk or egg white.

iv. Administration of stimulants like tea or coffee.

v. Morphine injection to suppress pain.

**Post-mortem appearance:**

i. Fragments of seeds can be found in stomach

ii. Inflamed bowel, occasional erosion and sub mucous haemorrhages.

iii. Haemorrhages of internal organs.

**USES:**

i. As counter irritant in gout, rheumatism, bronchitis and neuralgia.

ii. Abortifacient.

iii. Powerful cathartic used in sudden paralysis.

iv. Used in severe constipation.
5. ABRUS PRECATORIUS:

*Scientific Name:* Abrus precatorius

*Common Name:* Jequirity, Chashm e kharoos, Gangachi, Ratti, Prayerbeads, Rosary pea, Crab’s eyes, Buddhist rosary bead.

*Family:* Leguminosae

*Toxic Parts:* Broken seeds.

*Geographical Source:* It is a plant grown well in dry regions at low elevations. It grows best in tropical climates like India, Sri Lanka, Phillipine islands, Africa and West Indies.

*Plant Description:* It is a slender, perennial climbing woody shrub about 10 – 20 ft high. The stem is cylindrical, wrinkled with a smooth textured and brown bark.

- Leaves are glabrous, alternate, stipulate and compound paripinnate. Slightly hairy on lower side.
- Flowers are pale violet in colour with short stalk arranged in cluster.
- Fruit is pod
- Seeds are ovoid, globular, 5 – 9 mm in length, hard, smooth & shiny.

*Constituents:*

i. Abrin
ii. Lectin
iii. Hemagglutin
iv. Glycyrrhizin

*Poisoning:*

*Causes of Poisoning:*

They are frequently handled by the children because of their colour and attraction. Also they are made into rosary beads, necklaces and other jewellery. Therefore children are most likely to ingest them hence leading to poisoning.

*Mode of Action:*
- Toxins are released when seeds are chewed & swallowed. Poisoning is very serious & fatal if not accurately treated.

- The toxin abrin is a dimer consisting of two protein subunits, termed A and B. The B chain facilitates abrin's entry into a cell by bonding to certain transport proteins on cell membranes, which then transport the toxin into the cell. Once inside the cell, the A chain prevents protein synthesis by inactivating the 26S subunit of the ribosome. One molecule of abrin will inactivate up to 1,500 ribosomes per second.

**Fatal Dose:**
- Abrin can kill with a circulating amount of less than 3 μg.
- Fatal dose is 1 – 2 seeds and fatal period is 3 – 5 days. Shortest lethal period is 24 hours.
- *Boiling the seeds in milk and* drying will remove the toxic effect. The protein is denatured when subjected to high temperatures which remove its toxicity.

**SIGNS AND SYMPTOMS:**

*By Ingestion (Severe GIT irritation associated with):*
- Nausea
- Vomiting
- Diarrhoea
- Abdominal cramps
- Blood loss may occur due to haemorrhage.

**CVS effects:**
- No direct effect on heart
- Hypotension occurs due to ↓ blood volume, vomiting & diarrhoea.
- Hypotension is followed by reflex tachycardia and shock.

**Acid Base disturbances:**
• Prolong vomiting may cause alkalosis.
• Metabolic acidosis occurs from renal failure.

Effect on Fluid & Electrolytes:

✓ Loss of fluid and electrolytes occur due to vomiting and diarrhoea causing lethargy, fatigue, muscle weakness and muscle cramps.
✓ Dehydration also occurs.
✓ Degeneration of tubular cells occur leading to acute renal failure causing oligurea or anurea.

Effects on CNS:

☐ Drowsiness, convulsions, hallucinations and trembling of hands.

Effects on Body Temperature:

☐ Hyperthermia (104°F)

Effect on Liver:

➢ Necrotizing action of toxin causes liver damage.
➢ Serum level of enzymes i.e. Aspartate transaminase (AST), Alanine transferase (ALT) and lactic acid dehydrogenase are markedly ↑ed.
➢ Serum billirubin level is also ↑ed.

Effect on skin:

☐ Skin contact may cause irritation and deramtitis.

Effect on eye:

▪ Retinal haemorrhages may appear.
▪ There may be impaired vision, swelling and reddening of conjunctiva.

Haematological Effects:

☐ Abrin causes haemagglutination so it has a direct effect on red blood cells.
TREATMENT:

General principles:

i. No specific antidote is available but supportive therapy is done i.e.

ii. Induction of emesis

iii. Gastric lavage

iv. Fluid and electrolyte replenishes.

USES:

i. The seeds of Abrus precatorius are much valued in native jewelry for their bright coloration. One third of the bean with the hilum (attachment scar) is black, and the rest is bright red, suggesting a ladybug.

ii. Jewelry – making with jequirity seeds is dangerous, and there have been cases of death by a finger – prick while boring the seeds for beadwork.

iii. The plant is used in some traditional medicines to treat sores and wounds caused by dogs, cats & mice.

6. CALOTROPIS:

*Scientific Name:* Calotropis procera; Calotropis gigantea

*Common Name:* Akk (urdu), Spalmi (Push), Ashkhar, Mudar yercum.

*Family:* Asclepiadaceae

*Toxic Parts:* Milky juice from leaves, branches, bark.

*Geographical Source:* It is native to India & is naturalized in East & West Indies and Ceylon. In Pakistan it grows on dry, hot, & neglected areas.

*Plant Description:* The plant is typical xerophyte having branched stem.

- Plant is woody below and herbaceous above.
- The leaves are large, thick and dark green in colour.
The flowers are white or pale green in colour, have 5 petals and prominent symmetrical crown.

CONSTITUENTS:

i. Uscharin

ii. Calactin

iii. Calotoxin

iv. Madaralbum, a crystalline colourless substance;

v. Madarfluavil, an amber coloured viscid substance; and

vi. Caoutchouc

POISONING:

Causes of Poisoning:

i. Whenever fresh leaves and stalks are cut or crushed, a thick acrid & milky juice comes out which is acidic and bitter in taste.

ii. Upon standing or heating this juice forms a white clot and straw coloured serum is left behind. This serum contains an active principle called Giganitin. This is highly toxic and 20 times more potent than strychnine. The clot is also poisonous but to lesser extent.

Mode of Action:

Locally it acts as irritant poison and internally as GIT irritant and cerebrospinal poison.

Fatal Dose:

- Fatal dose is uncertain,
- Fatal period is 12 hours.

SIGNS AND SYMPTOMS:

BY INGESTION:
i. GIT & CSF poisoning

ii. Nausea

iii. Vomiting

iv. Diarrhoea

v. Burning pain in throat & stomach

vi. Mydriasis

vii. Tetanic convulsions

viii. Delirium

ix. Coma leading to death.

**EXTERNAL APPLICATION OF JUICE:**

- Redness of skin
- Inflammation
- Vesication

**Administration in the Eye:**

- Conjunctivitis

**TREATMENT:**

**GENERAL PRINCIPLES:**

i. Gastric lavage

ii. Administration of demulcent to skin for soothing effect on skin that has been irritated or inflamed.

iii. Stimulants for treating collapse.

iv. Morphine injection to suppress pain.

v. In over excitement, barbiturates are given.

**ANTIDOTE:**
Specific antidote is Atropine.

USES:

i. Flowers are used as digestive aid

ii. Leaves are used in external poultices (for sore skins)

iii. Powdered roots are used as emetic

iv. Juice is used as vesicant

v. Used in leather tanning to remove hair from Hyde and for deodorizing them

vi. Also for diarrhoea and dysentery

7. DATURA:

*Scientific Name:* Datura stramonium, D. metel, D. alba, D. fastuosa.

*Common Name:* Dhatura, tatura, tatura sufaid, Stramonium, Datura, Devil's Apple, Jamestown-weed, Jimson-weed, Stinkweed, Devil's Trumpet, Apple of Peru, Thorn apple.

*Family:* Solanaceae

*Toxic Parts:* All parts of plants are toxic but part used is seed.

*Geographical Source:* Native to tropical America. India, Pakistan. It is found in neglected areas e.g. Roadsides.

*Plant Description:* Plant is annual herb, about 3m tall woody below and herbaceous above. Stem is bifurcatedly branched.

- Leaves are simple, large and unequilateral at the base.
- Flowers are white in colour and infundibuliform (bell shaped).
- Fruits are white, egg shaped, capsular and spiny.

*CONSTITUENTS:*

i. Hyoscine (Scopolamine)

ii. Atropine (mixture of d- & l- hyoscyamine)
iii. Daturine

POISONING:

Causes of Poisoning:

i. The poisoning of datura is typically atropine poisoning which is the active principle of the plant.

ii. Most of its effects are produced due to its anti muscarinic effects.

iii. Accidental poisoning may occur but it is less common.

iv. Poisoning occurs in journey when unaware traveller is given some food or drink mixed with datura leaves, fruits or crushed seeds.

Typical signs & symptoms:

i. Dry as bone; Red as beet, Blind as bat, Hot as a hare, Mad as a wet hen.

ii. Delirium and coma appear within half an hour or earlier and immediately if decoction of seed is used.

iii. Earlier symptoms are bitter taste in mouth, dryness of mouth and throat due to inhibition of salivation (dry as bone).

Mode of Action:

- On ingestion of such material the travellers become unconscious and may lead to death if not treated in time.

Fatal Dose:

- About 100 – 125 seeds or 60 mg of the alkaloid for adult and 4 mg for children.

- Death usually occurs within 24 hours.

SIGNS AND SYMPTOMS:

First stage:
➢ Burning pain in stomach
➢ Bitter taste of mouth
➢ Nausea, vomiting & diarrhoea
➢ Thirst
➢ Headache
➢ Gastric irritation
➢ Dryness of mouth

Second stage:

- Muscle incoordination
- Flushing of face
- Hyperthermia
- Photophobia & conjunctivitis
- Pupilary dilation
- Urinary retention

Third stage:

✔ Restlessness
✔ Delirium
✔ Excitement
✔ Disturbed hearing
✔ Hallucination
✔ Visual disturbances
✔ Respiratory failure

TREATMENT:

GENERAL PRINCIPLES:
i. Gastric lavage with weak solution of KMnO₄ or 4 – 5% tannic acid.
ii. Symptomatic treatment
iii. Moistening of mouth and tongue
iv. Antidote physostigmine should be given
v. To make the patient comfortable pilocarpine should be given
vi. Stimulants like tea & coffee should be given
vii. Activated charcoal should be given to adsorb the poison

8. ATROPA BELLADONNA:

Scientific Name: Atropa belladonna, Atropa acuminata.

Common Name: Deadly night shade, Black berry, Mardum gaya (Persian) Angoor e shifa (Urdu), Tambakoo saag, Shah beezak, yabraj.

Family: Solanaceae

Toxic Parts: All parts of plants are toxic.

Geographical Source: Indigenous to Asia.

Plant Description:

- Plant is perennial herb, 0.2 – 2.5m tall
- Stem is branched
- Leaves are dark green, accuminate and petiolate.
- Flowers are solitary, clusters, dull green in colour.
- Fruit is purplish black berry.

CONSTITUENTS:

i. Atropine
ii. Hyoscyamine
iii. Belladonine

POISONING:

Causes of Poisoning:

a. The poisoning occurs mainly due to the over dosage of products containing the substances obtained from Atropa belladonna. It may be in the form of:

i. Eye drops

ii. Belladonna tincture

b. The poisoning also occurs by eating the flesh of rabbit that had eaten the Atropa belladonna.

Fatal Dose:

i. 125 mg of belladonna active constituents.

ii. Atropine have been proved fatal.

iii. It is safe in adults even in grams but

iv. Even 4 mg have caused death in children.

v. Fatal period is within 24 hours

SIGNS AND SYMPTOMS:

➢ Dysphasia (Difficulty in talking)

➢ Dysphagia

➢ Dilation of cutaneous blood vessels.

➢ Dilation of pupil

➢ Dry hot skin

➢ Drunken gait

➢ Delirium

➢ Urinary retention
Drowsiness

POST MORTEM APPEARANCE:
- Leaves, berries & seeds in GIT
- Mydriasis

TREATMENT:

General principles:
1. Gastric lavage
2. Symptomatic treatment
3. Physostigmine is absolutely necessary otherwise it is no more better than symptomatic treatment
4. Activated charcoal
5. Purgatives
6. Stimulants (Tea & Coffee)

9. COLCHICUM:

Scientific Name: Colchicum autumnale, Colchicum luteum.

Common Name: Suranjan e shireen, Gul e hasrat, Darkom.

Family: Liliaceae

Toxic Parts: Corm & seeds.

Geographical Source: Found in hilly areas of Pakistan.

Plant Description:
- Perennial herb which has underground bulbs or a woody corm.
- It is flattened at one side and convex on other.
- At the top of corm gray or greenish leaves emerge which fold each other up to mid and then spread.
- Flower is golden yellow/ pink
CONSTITUENTS:

i. Colchicine

ii. Gloriosine

iii. 3-desmethyl colchicine

iv. beta-lumicolchicine,

v. N-Formyl-desacetyl colchicine,

vi. 2-desmethyl colchicine

vii. Chelidonic acid

viii. Salicylic acid

POISONING:

Causes of Poisoning:

a. Although all parts of the plant are toxic but seeds are highly toxic.

b. Poisoning occurs due to over dosage of colchicine used for treatment of gout and cancer.

c. Also occurs by drinking the milk of goat fed on this plant.

Fatal Dose:

- 2 g of seeds are lethal for a child. 7 – 60 mg is lethal dose for adult and death occurs in 1 – 4 days.

SIGNS AND SYMPTOMS:

FIRST STAGE:

i. Burning pain in mouth, throat and oesophagus

ii. Locked jaws, difficulty in swallowing

iii. Intense thirst
iv. Nausea, vomiting & diarrhoea
v. Bloody stool
vi. Acute GIT syndrome resembling arsenic poisoning
vii. Vertigo
viii. Cold skin and pale face
ix. Irregular respiration
x. Dilated pupil
xi. Fever
xii. Alopecia
xiii. Liver damage
xiv. Tetanic convulsions
xv. Respiratory paralysis

TREATMENT:

GENERAL PRINCIPLES:

i. Gastric lavage using tannic acid

ii. Hypodermic injections of morphine & atropine to relive pain and inflammation.

iii. Artificial respiration to treat respiratory paralysis.

USES:

i. Used in gout & rheumatoid arthritis.

ii. In treatment of cancer.

10. NUX – VOMICA:

*Scientific Name: Strychnos nuxvomica.*
Common Name: Dog button, Kuchla.

Family: Loganeaceae

Toxic Parts: Dried ripe seeds.

Geographical Source: Found in India Ceylon & Pakistan.

Plant Description:

- It is a small tree with irregular branches and greyish bark.
- Leaves are simple, ovate & opposite.
- The flowers are greyish white.
- Fruit is orbicular berries. Each berry contains 5 orbicular seeds.
- Seeds are round, orbicular and greyish. They possess pressed hairs which give them silky appearance. Hilum is in the centre. Seeds are 10 – 30 mm in length and 3 – 5 mm thick.

Constituents:

i. Strychnine
ii. Brucine
iii. Loganine
iv. Strychnic acid
v. Proteins
vi. Fixed oils

Poisoning:

Causes of Poisoning:

i. Accidental poisoning occur if seeds are eaten by mistake
ii. Homicidal and suicidal poisoning

Fatal Dose:

✓ 1 seed or 15 – 30 mg strychnine.
✓ Death occurs within a day

Signs and Symptoms:
Appear Within 5 – 10 min:

i. Epigastric pain
ii. Choking sensation in throat
iii. Increased vomiting
iv. Stiffness of neck and face
v. Twitching of muscles
vi. Contraction of bronchial muscles
vii. Over excitement
viii. Convulsions
ix. Mydriasis

TREATMENT:

i. Short acting barbiturates
ii. Muscle relaxants
iii. Mephenesin (centrally acting muscle relaxant) is specific antidote.
iv. If convulsions are under control then stomach wash with KMnO₄, activated charcoal & 2% tannic acid or black tea.
v. Artificial respiration if required

USES: Killing of dogs