

Popular Article

Photosensitization In Domestic Animals

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Photosensitization of livestock is a problem with many causes and is known throughout the world. Ruminants suffer from photosensitization most frequently; horses, swine, and poultry are affected less commonly. Photosensitization in livestock primarily originates from consumption of poisonous plants. Drugs, congenital diseases, and infectious diseases rarely induce photosensitization in livestock; outbreaks as a result of these causes generally involve relatively few animals. The greatest economic impact arises from frequently recurring problems that affect large numbers of animals.

Introduction

Photosensitization is a pathologic process or syndrome of the skin. The exposure of ultraviolet light or visible light mainly affects superficial layers of unpigmented or light skinned areas of the body *viz.* nose, face, back, udder, testes, teats, mucosa, cornea *etc.* due to the presence of photodynamic agents in the peripheral circulation. It is also defining a noncontagious clinical syndrome that develops when an animal or human becomes abnormally reactive to sunlight due to the presence of a phototoxin or photoallergen in or on the skin (Clare, 1952; Epstein, 1999). Photosensitization differs from sunburn in that; it requires the presence of a phototoxic agent; the onset of reaction is rapid, whereas onset of reaction for sunburn is delayed (Amstutz HE, 1970; Aplin TEH, 1975). It is not produced by sunburn but certain molecule which is normally present in the skin gets energized when sunlight falls on non-pigmented areas of the skin. When the molecules return into less energized state, the released energy causes chemical reaction in the skin. These abnormal reactions are due to high concentration of photodynamic agents. In peripheral circulation photosensitization may be observed after a few hours to few days of ingestion on exposure to strong sunlight.

Classification of the photosensitization according to their causative agents

1. Primary Photosensitization: The ingestion of photodynamic agent which may be present in drugs, dyes or/and plant developed primary Photosensitization.

Examples: -

- ✓ Drugs metabolite: Phenothiazine sulfoxide
- ✓ Dyes: Acridine, Rose Bengal
- ✓ Plants: *Hypericum perforatum* (hypericin), *Fagopyrum esculentum* (fagopyrin), *Fagopyrum sagittatum* (fagopyrin), *Polygonum fagopyrum* (fagopyrin), *Ammi majus* (furocoumarins), *Ammi visnaga* (furocoumarins), *Thamnosma texana* (furocoumarins), *Cymopterus watsoni* (furocoumarins), *Cymopterus longipes* (furocoumarins), *Lolium perenne* (perloline) etc.

2. Secondary Photosensitization: It developed due to hepatotoxicity and obstruction of bile duct resulting into defect in porphyrin metabolism.

Examples: -

- ✓ Plants: *Lantana camara*, *Agave lecheguilla*, *Lippia rehmanni*, *Myoporum laetum*, *Nartheicum ossifragum*, *Brachiaria brizantha* etc.
- ✓ Mycotoxin: *Pithomyces chartarum*
- ✓ Blue green algae: *Microcystis flos aquae*
- ✓ Chemicals: Carbanterachloride, Corticosteroids, phenanthridium, p-dehydroxybenzene

3. Congenital Photosensitization: It is hereditary in origin and occurs due to certain enzymatic abnormalities or inadequate production of enzymes like catalase, glucose-6-phosphate dehydrogenase etc. in RBCs and disruption of haeme biosynthetic pathway.

Mechanism of action

- Primary photosensitizers interact directly with cellular constituents particularly pyrimidine bases and nucleic acids and inhibit DNA synthesis and produce cellular damage.
- Secondary Photosensitization is the sequel of extensive hepatic damage and obstruction of bile duct. In the gastrointestinal tract, phylloerythrin (photodynamic agent) is produced by break down of chlorophyll in the presence of micro-organism. This is not excreted out from the body of animal. Results in accumulation of phylloerythrin in the body. Through peripheral circulation it comes in to capillaries of skin and on exposure to bright sunlight Photosensitization is produced. Hepatic damage is of serious consequence of ingestion of

plants toxins which produced toxicity in both ruminant and non-ruminant such as guinea pigs, rabbits and female rats are susceptible to the hepatotoxic action of *lantana camera*.

- Congenital Photosensitization is inherited defect in animals due to alteration in certain enzymatic abnormalities or inadequate production of enzymes in animal body resulting in to hypersensitivity reaction

Clinical signs

Photosensitization occurs worldwide and can affect any species but is most commonly seen in cattle, sheep, goats, and horses (Dietz O and Wiesner E, 1984; Mortimer PH, 1966).

In ruminant

- ❖ Anorexia, depression, diarrhoea, constipation and icterus are the first to be observed due to the liver damage.
- ❖ Hyperemia is the first change, followed by oedema and swelling.
- ❖ Serous fluid oozes out to the surface of skin followed by the necrosis and exfoliation of the superficial layer of the skin.
- ❖ The condition is characterised by clearly demarcated 'sunburn-like' lesions that are confined to unpigmented or hairless areas of the skin *viz.* ears, nose, muzzle, eyes, intermandibular region, udder and teat.
- ❖ Mandibular area is so distended and bulged with oedematous fluid that it gives a bottle jaw appearance and head becomes big, thus condition is also known as “Big head in sheep”.
- ❖ In sheep conjunctivitis is more pronounced while oedema is less marked in cattle.
- ❖ Increased lacrimation, initially watery which later becomes thick, swelling and closure of eyelids and nostrils.
- ❖ In sheep, cornea becomes clouded leading to blindness, break of wool and high mortality.

In poultry

- ❖ In young birds acute inflammatory effects include erythema and blistering on beaks and exposed skin followed by thick crust and scabs keratoconjunctivitis and closure of eyes with serous fluid which dries and seals the eyelids
- ❖ In chronic condition deformation of upper beak feet and legs, sloughing of comb and wattles and scarring of ocular opening.

In horse

- ❖ Symptoms are similar to that of cattle & sheep except colic show inflammation of the MM of mouth & brain.

Diagnosis

- ✓ On the basis of history and evidence of ingestion of photodynamic agent.
- ✓ Based on clinical signs:-
 - Icteric mucous membrane
 - Photosensitization lesion on non pigmented or light skinned areas
- ✓ By post mortem lesions.
- ✓ Lab diagnosis:-
 - Increased serum biochemical measurements, including sorbitol dehydrogenase, gamma glutamyltransferase, alkaline phosphatase, and direct bilirubin.
 - Increased serum phylloerythrin concentration
 - Examination of feces, and urine for porphyrins can also be performed.

Treatment

Treatment of photosensitization involves these major elements: termination of intake of the hepatotoxin or phototoxic agent, protection of animals from direct exposure to sunlight, provision of nutritious feed, treatment (or prevention) of secondary infection and fly strike and general supportive therapy as needed.

- Prevent further ingestion of the photodynamic agent.
- Remove the animal from sunlight and animal kept in dark or shaded place until the photodynamic agent has been excreted out.
- Flies and other ectoparasites are keeps away from the skin lesions to prevent further secondary infection.
- Administer laxative or saline purgative to remove the ingesta from the stomach/rumen.
- Systematically give antihistaminics, antibiotics and corticosteroids (early stages).
- Topical treatment of the dermatitis lesions with demulcent, antibiotic and corticosteroids ointment.
- In severe cases, dextrose saline therapy also given.
- No specific antidote is available.

Prevention and control

Strategy should be formulated for prevention and control of outbreaks by reducing or eliminating contributory factors.

- Toxic species of plants in pastures can be controlled satisfactorily by annual herbicide applications.
- Regular burning will reduce the number of poisonous plants. Fire is often used as a pre-treatment to herbicide.
- Biologic control of plants by predator moths, flea beetles and seeds flies is another tool to control lantana and other toxic plants.
- Animals, especially calves, treated with phenothiazine should be protected from direct sunlight for 48 hours.

Conclusion

Photosensitization is a sunburn-like condition caused by the presence of certain photoactive compounds (plant origin or drugs or chemicals) in the skin when it is exposed to the appropriate wavelength of light (Johnson AE, 1982; Mortimer PH and Ronaldson JW, 1983). Photosensitization primarily is a disorder of sheep and cattle, but all classes of livestock are susceptible. Clinical recognition of the syndrome usually presents no difficulty because of the restriction of lesions to areas of skin unprotected from sunlight. Prognosis generally depends on the extent of hepatic injury.

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