

Photosensitization in Livestock

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Photosensitization in animals refers to a skin condition where the skin becomes abnormally reactive to sunlight. Photosensitivity is an aberrant reaction of the body when exposed to the direct sunlight, owing to the abnormal presence of photodynamic pigments/compounds in the skin cells, cornea or mucus membranes (Mauldin and Peters-Kennedy, 2016). Photosensitization occurs when skin (especially areas exposed to light and lacking substantial protective hair, wool, or pigmentation) becomes more susceptible to ultraviolet (UV) light because of the presence of photodynamic agents. Unlike sunburn and photodermatitis, photosensitization does not result in pathological skin changes in the absence of a photodynamic agent. Upon being energized by the photons of the certain wavelength of sunlight, particularly in ultraviolet range of spectrum; the molecules get energized and when these return to their initial stage, the released energy transfers to the receptor molecules in skin and cause tissue injury either by formation of reactive oxygen intermediates or by changing the permeability of cell membranes. Thus, this reaction is typically a biophysical phenomenon. Photosensitization occurs mostly in cattle, sheep and horses worldwide. (Kumar and Patel, 2013).

Classification: The four-category classification of photosensitisation diseases (Clare, 1953), namely

Type I: Primary photosensitization

Type II: Aberrant pigment metabolism photosensitization

Type III: Secondary (hepatogenous) photosensitization.

Type IV: Idiopathic Photosensitization

A wide range of chemicals, including some that are fungal and bacterial in origin, may act as photosensitizing agents. However, most compounds that are implicated in photosensitization in animals are plant derived.

Primary Photosensitization (Type I): Primary photosensitization occurs when a photodynamic agent is ingested, injected, or absorbed through the skin. The agent enters the systemic circulation in its native form, where it results in skin cell membrane damage after the animal is exposed to UV light. Plants in the families Apiaceae (also known as Umbelliferae) and Rutaceae contain photoactive furocoumarins (psoralens), which lead to photosensitization in production animals and poultry. For example, Ammi majus (bishop's-weed) and Cymopterus watsonii (spring parsley) can produce photosensitization in cattle and sheep, respectively. Ingestion of A majus and Ammi visnaga seeds can produce severe photosensitization in poultry. Plant species in the genera Trifolium, Medicago (clovers and alfalfa), Erodium, Polygonum, and Brassica (mustards) have been incriminated in primary photosensitization. In addition, exposure to coal tar derivatives, such as polycyclic aromatic hydrocarbons, tetracyclines, and some sulfonamides, can lead to primary photosensitization. Phenothiazine anthelmintics have been reported to lead to primary photosensitization in cattle, sheep, goats, and swine (Gupta *et al.*, 2006).

Aberrant Pigment Metabolism Photosensitization (Type II): Type II photosensitization, due to aberrant pigment metabolism, occurs in both cattle and cats. In this syndrome, the photosensitizing porphyrin agents are endogenous pigments that arise from inherited or acquired defective functions of enzymes involved in heme synthesis. Bovine congenital erythropoietic porphyria and bovine erythropoietic protoporphyria are the most commonly reported diseases in this category. In this syndrome, the photosensitizing porphyrin agents are endogenous pigments that arise from inherited or acquired defective function of enzymes involved in haem synthesis like Glucose-6-phosphate dehydrogenase, which on deficit in RBC leads to disruption of haem biosynthetic pathway. As a result, porphyrins accumulate in body (Including skin) and when exposed to sunlight, oxygen free radicals formed due to interaction with cellular macromolecules, which ultimately damage the cell and get released into circulation (Barrington, 2017). Congenital erythropoietic porphyria (BCEP) and congenital erythropoietic protoporphyria (BCEPP) have both been reported in domestic cattle. Accumulation of uroporphyrin I and coproporphyrin I results in type-II photosensitization in BCEP. Bovine Congenital Erythropoietic Proto Porphyria (BCEPP), which is caused by a deficiency in the activity of ferrochelatase. This enzyme is involved in the final stage of the 8-step haem biosynthesis pathway, catalysing the chelation of ferrous iron to protoporphyrin in the production of haem. Accumulation of protoporphyrin is the cause of photosensitization in BCEPP (McAloon *et al.*, 2015).



Fig.1. Photosensitization in cattle



Fig.2. Photosensitization in Sheep

Secondary (Hepatogenous) Photosensitization (Type III): Secondary, or type III: photosensitization—also known as hepatogenous photosensitization—is by far the most common type

of photosensitization observed in production animals. It results from impaired biliary excretion of phylloerythrin (a porphyrin), which is a normal by-product of chlorophyll metabolism. Secondary photosensitization may be a sequela of any hepatocellular dysfunction or cholestasis; it is not related to phototoxin ingestion. Phylloerythrin, but not chlorophyll, is normally absorbed into the circulation and is effectively excreted by the liver into the bile, following the same general pathway as bilirubin metabolism. Failure to excrete phylloerythrin increases its circulating concentrations. When phylloerythrin reaches the skin, it can absorb and release light energy, initiating a phototoxic reaction. Phylloerythrin has been incriminated as the phototoxic agent in the following conditions: common bile duct occlusion; facial eczema; lupinosis; congenital photosensitization of Southdown and Corriedale sheep; and poisoning by numerous plants including *Tribulus terrestris* (puncture vine), *Lippia rehmannii*, *Lantana camara*, several *Panicum* spp (kleingrass, broomcorn millet, witchgrass). Photosensitization associated with phylloerythrin accumulation can occur in animals that have liver damage associated with various toxic substances (notably, these are hepatobiliary toxins, not direct phototoxins). Plant or microbial products associated with hepatogenous photosensitization include lantadenes, steroidal or lithogenic saponins, mycotoxins (sporidesmin and phomopsin) and certain tannins (Gupta, 2012).

Idiopathic Photosensitization (Type IV): Photosensitization in which the pathogenesis is unknown or the photodynamic agent is not identified is classified as idiopathic, or type IV.

Mechanisms of phototoxicity:

In photosensitization, unstable, high-energy molecules are formed when photons react with a photodynamic agent. These high-energy molecules initiate reactions with substrate molecules of the skin, leading to the release of free radicals that, in turn, result in increased permeability of cell and lysosomal membranes. Damage to a cell's outer membrane enables leakage of cellular potassium and cytoplasmic extrusion. Damage to lysosomal membranes within a cell releases lytic enzymes into the cell, potentially leading to skin ulceration, necrosis, and edema (Seawright, 1982). The time interval between exposure to a photodynamic agent and the onset of clinical signs of photosensitization

depends on the type of agent, its dose, and the length of time the animal is exposed to sunlight.

Clinical Findings:

Dermatological signs associated with photosensitization are similar regardless of the type of photosensitization. Photosensitive animals are photophobic immediately when exposed to sunlight and appear agitated and uncomfortable. They may scratch or rub lightly pigmented, exposed areas of skin (eg, ears, eyelids, muzzle). Clinical signs of photosensitization include progressive weight loss and anorexia, oedema and necrotic tissue, crusting and sloughing of skin in non-pigmented and exposed areas such as ears, face, rump, flank and vulva regions in females as well as visible jaundice. Affected animals seek shade, grazing in the evening or early mornings or remaining under trees and shrubs rather than actively grazing (Low, 2015). Cloudiness in cornea and neurological signs like ataxia, dizziness and convulsions are also seen in affected sheep and goats. In birds erythema, blistering on beak, feet, legs and sloughing of comb and wattles are also seen in affected birds.

Lesions:

Lesions caused by photosensitization initially appear in white-haired, nonpigmented, or hairless areas such as the nose and udder. However, high concentrations of plant-derived photoactive toxins or phylloerythrin coupled with very bright sunlight can induce lesions on pigmented skin. Erythema associated with photosensitization develops rapidly and is soon followed by edema. If exposure to light stops at this stage, the lesions soon resolve. When exposure is prolonged, lesions may progress to include vesicle and bulla formation, serum exudation, ulceration, scab formation, and skin necrosis. Generalized icterus in body tissues, extensive subcutaneous edema along with enlargement of local lymph nodes is usually noticed. Teeth and bones show pink-brownish discoloration. Enlarged and granular liver with thin margin and distended gall bladder are usually noticed in affected animals. In the final stage of photosensitization, skin is sloughed (Fig.1 & 2). In cattle, and especially in deer, exposure of the tongue while licking may result in glossitis, characterized by ulceration and deep necrosis. Regardless of coat color, cattle may develop epiphora, corneal edema, and blindness.

Diagnosis:

Presumptive diagnosis is based on clinical signs evidence or history of exposure to photosensitizing agents or hepatotoxins, and characteristic lesions. Definitive diagnosis: measurement of porphyrin in blood, urine, and feces. Porphyrin concentrations are elevated only in cases of abnormal phylloerythrin excretion.

Treatment:

As far as now no specific antidote for the photo toxins is available. Treatment regimen follows symptomatic approach on removing the toxin from body. Animals should be kept in dark areas until the toxin is completely excreted (5 to 7 days) to avoid further complication. Topical application of demulcents, antibiotics and corticosteroid ointments can be used. Antihistamines and antibiotics can be administered intramuscularly. Keep fly and ectoparasites away from skin lesions. Laxatives or saline purgatives can be administered to remove the ingesta from stomach/rumen. Hepatoprotectants like liver tonics and stimulants can be administered to prevent further damage to liver.

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