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Photosensitisation in Domestic Animals: Classification, Pathogenesis, Clinical Features, and Management

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Abstract:

Photosensitivity in animals is a severe dermatitis that affects dermal tissues upon exposure to sunlight or ingestion or contact with UV-reactive secondary plant-produced metabolites, such as those found in Lantana camara and Hypericum perforatum. This reaction is most severe in areas of the skin lacking protective hairs, fleece, or pigmentation. It generates free radicals and reactive oxygen species, leading to skin damage and ultimately DNA degradation. This is typically a biophysical phenomenon. Photosensitization occurs mostly in cattle, sheep, and horses worldwide, though its occurrence in buffaloes has been sporadic. Animals primarily develop secondary photosensitization due to fresh feed (71.8%) of normal quality (88.1%), indicating that the phototoxic agents originate from liver-toxic plants like the grass Brachiaria and the herb Froelichia. It predominantly affects regions near the equator, especially in India and South America. Photosensitization is seasonal, with the highest incidence during periods of intense sunlight combined with the availability of certain plants—mainly in summer and early rainy seasons. The condition not only impacts animal welfare but also results in significant economic losses due to reduced productivity, treatment costs, and sometimes mortality. Sporadic outbreaks of idiopathic photosensitization, such as spring eczema in calves and dairy cows, which occur countrywide during spring, and in lambs on unimproved pastures in the South Island, have frustrated farmers and veterinarians for many years. This review aims to provide an overview of the current approaches in managing photosensitization in animals, highlighting recent developments, challenges, and future perspectives in both conventional and traditional veterinary practices. Effective management of photosensitization requires a comprehensive understanding of its etiology, pathogenesis, clinical manifestations, and risk factors.

Keywords: Photosensitivity, dermatitis, phylloerythrin, hepatogenous toxicity

Introduction:

Photosensitization is a skin disorder in animals marked by an abnormal reaction to sunlight, particularly ultraviolet (UV) rays. This condition arises when phototoxic or photodynamic pigments (light-reactive substances) accumulate in the skin and, upon exposure to sunlight, cause tissue damage. These chromophores are energized by photons within the sunlight wavelength range of 280-790 nm, especially

in the ultraviolet spectrum. Once energized, the molecules return to their initial state, releasing energy that transfers to receptor molecules in the skin, leading to tissue injury. This occurs either through the formation of free radicals, which alter cell membrane permeability and damage adjacent proteins, nucleic acids, and membranes, or by directly damaging molecules or generating reactive molecules that initiate or continue chemical reactions. Most chromophores are not cytotoxic until photoactivated. Unlike sunburn and photodermatitis, photosensitization does not cause pathological skin changes without a photodynamic agent. Radiation-induced dermatitis typically affects the muzzle, ears, eyelids, face, tail, vulva, udder, and coronary band. However, with severe exposure, even black, heavily haired animals can develop photosensitivity. The term photodermatitis is broader, encompassing photosensitization and photo-aggravated dermatitis (increased UV sensitivity without a phototoxin) due to underlying diseases such as dermatomyositis, discoid lupus erythematosus, pemphigus erythematosus, and erythema multiforme. The time between exposure to a photodynamic agent and the onset of clinical signs of photosensitization depends on the agent type, its dose, and the duration of sunlight exposure. The syndrome of forage-associated photosensitization in livestock is sporadic worldwide, but in some countries, it holds significant economic and animal welfare importance. Photoallergic dermatitis in farm animals includes reactions to gluten (Yeruham et al., 1999) and cocoa shells (Yeruham and Avidar, 2003) in the diets of horses and cattle, respectively.

Classification:

Photosensitisation is classified based on the source of the photodynamic agent, liver involvement, and the underlying cause, which can be toxic, metabolic, or unknown. There are four categories of photosensitisation disease classification:

1. Type 1 - Primary form,
2. Type 2 - Congenital form due to aberrant endogenous pigment synthesis,
3. Type 3 - Hepatogenous form, and
4. Type 4 - Photosensitivity of unknown etiology.

Type 1-Primary form:

This photosensitisation reaction is induced by the ingestion of various synthetic compounds, including antibiotics, antihistamines, anti-inflammatory agents, antimalarials, cardiovascular drugs, central nervous system-acting pharmaceuticals, and medications used in managing multisystemic disorders, as well as naturally occurring plant-derived bioactive substances. These compounds typically contain aromatic chromophore groups or photolabile functional bonds capable of absorbing light energy, generating free radicals, and initiating light-induced cellular damage. Plant compounds responsible for photosensitising effects in animals are secondary metabolites that play a functional role in plant physiology and etiology. Their concentration and distribution within plant tissue show substantial

variability and are not governed by the regulatory dynamics of primary metabolic processes essential for basic survival. Plants in the families Apiaceae (also known as Umbelliferae) and Rutaceae contain photoactive furocoumarins, which lead to photosensitisation in animals and poultry. Ammi majus and Cymopterus watsonii can produce photosensitisation in cattle and sheep, respectively. Ingestion of A. majus and Ammi visnaga seeds can cause severe photosensitisation in poultry. Plant species in the genera Trifolium, Medicago, Erodium, Polygonum, and Brassica have been implicated in primary photosensitisation.

Type 2-Congenital form:

This form is often hereditary, originating from enzymatic abnormalities or inadequate production of enzymes like catalase, glucose-6-phosphate dehydrogenase, etc., in RBCs, and disruption of the heme biosynthesis pathway. As a result, porphyrin or its derivatives accumulate in the body, including the skin. When exposed to bright sunlight, these substances become excited and interact with cellular macromolecules or molecular oxygen to generate toxic free radicals. Congenital erythropoietic porphyria and congenital erythropoietic protoporphyrin have both been reported in domestic cattle. Accumulation of uroporphyrin I and coproporphyrin I results in type 2 photosensitisation.

Type 3-Hepatogenous form:

Hepatogenous photosensitisation, also known as secondary or type 3 photosensitisation, is the most common type observed in animals. It is caused by impaired biliary excretion of phylloerythrin, a porphyrin that is a normal by-product of chlorophyll metabolism. Failure or partial failure of the liver to excrete pigments normally present in bile leads to this condition. Plant or microbial products associated with hepatogenic photosensitisation include lantadenes, steroidals, lithogenic saponins, mycotoxins, and certain tannins. Phylloerythrin is normally absorbed into the circulation and is effectively excreted by the liver into bile, similar to bilirubin metabolism. Failure to excrete phylloerythrin increases its circulating concentrations. When phylloerythrin reaches the skin, it can absorb the released light energy, initiating a phototoxic reaction. Phylloerythrin is recognized as the phototoxic agent responsible for poisoning by various plants, including *Tribulus terrestris*, *Lippia rehmanni*, *Lanthana camara*, *Cynodon dactylon*, and *Myoporum laetum*. Photosensitization linked to phylloerythrin accumulation can occur in animals with liver damage caused by various toxic substances such as pyrrolizidine alkaloids, cyanobacteria, Nolina spp, Agave lechuguilla, Holocalyx glaziovii, Brassica napus, Brachiaria brizantha, phosphorus, and carbon tetrachloride. Photosensitization was reported in a cow with cholestasis and Anaplasma centrale infection, hypothesized to result from intrahepatic cholestasis secondary to excessive RBC breakdown and bilirubin metabolism within the liver.

Type 4-Photosensitivity of unknown etiology:

Classified when the pathogenesis is unclear or a photodynamic agent is not identified. Outbreaks of

photosensitization have been reported in cattle exposed to water-damaged alfalfa hay, moldy straw, and orchard grass hay containing foxtails, suspected to be hepatogenous in origin. Ranunculus bulbosus is also presumed to cause hepatogenous photosensitization. Additionally, sporadic outbreaks of idiopathic photosensitization, such as spring eczema in calves and dairy cows during spring, and in lambs on unimproved pasture, have long frustrated farmers and veterinarians. Evidence suggests that most cases are primarily associated with ingested phototoxic plants.

Pathogenesis:

It involves sequence of events with photodynamic agents, sunlight, and skin tissue reaction. Photosensitizing chemicals can enter the skin directly or systemically through the bloodstream, causing harm by a cytotoxic mechanism (photocytotoxicity) or, less frequently, by triggering an immunological reaction (photoallergic reaction). Photodynamic agents circulate in the blood and deposit in the skin, especially in non-pigmented areas and areas with less hair coverage. Photocytotoxicity occurs minutes after direct contact with the toxic material, hours after the primary photosensitizing agent is deposited (by contact or ingestion), or days after exposure. In photosensitization, sunlight activates photodynamic agents in the skin, producing reactive oxygen species (free radicals) that damage cell and lysosomal membranes, causing leakage of cell contents and release of lytic enzymes, leading to inflammation, edema, necrosis, and skin ulceration. The severity depends on the type and amount of agent and the level of sunlight exposure.

Figure 1: Horse and Sheep exhibiting clinical signs of Photosensitization.

Pathological Lesions:

Pathological Lesions are characterized by cutaneous hyperesthesia and varying degrees of dermatitis and/or keratoconjunctivitis, edema, and necrosis in skin and/or eye membranes unprotected by melanin pigmentation, epidermis thickness, or hair, wool, or clothing coverage. Local lymph node enlargement, widespread subcutaneous edema, pink-brownish discoloration of teeth and bones, intense yellow discoloration in liver parenchyma, hydropic degeneration and necrosis in hepatocytes, cholangitis, and a distended, enlarged, edematous gall bladder is observed.

Prevention:

involves immediate veterinary care after the onset of photosensitization, appropriate pasture management, and control of toxic plants. Measures should be implemented to minimize direct sunlight exposure by housing animals in kachcha, semi-pakka, or pakka shelters according to the farmer's economic condition, preferably under a stall-fed rearing system. Ensuring an adequate and safe feed supply during drought is crucial to prevent photosensitization.

Clinical Signs:

Clinical Signs of photosensitisation in animals are consistent, regardless of the type. Photosensitive animals exhibit photophobia and discomfort, often scratching or rubbing lightly pigmented, exposed skin areas. Additional clinical signs include progressive weight loss, anorexia, oedema, and necrotic tissue in non-pigmented, exposed regions. Erythema associated with photosensitisation develops rapidly, soon followed by oedema. If light exposure ceases, lesions resolve quickly; however, prolonged exposure can lead to vesicle and bulla formation, serum exudation, ulceration, scab formation, and skin necrosis. In cattle, the udder and teats are most affected, while in small ruminants, the ears, nose, muzzle, and ear tips are primarily impacted. In bovine congenital erythropoietic porphyria, bone and urine discoloration accompany skin lesions. In birds, blistering occurs on the beak, feet, and legs, with sloughing of the comb and wattle also observed.

Diagnosis:

Diagnosis is primarily based on clinical evaluation, detailed history, and laboratory investigation. Lesions are mainly confined to non-pigmented skin. A thorough history helps identify exposure to photodynamic agents, hepatotoxic plants, and drugs. Laboratory diagnosis aids in differentiating types of photosensitisations. Evaluation of liver enzymes such as gamma-glutamyl transferase, aspartate aminotransferase, and increased bilirubin levels indicate hepatic dysfunction, often accompanied by phylloerythrin in circulation. High-performance liquid chromatography and spectrophotometric assays are employed for the quantitative identification of photodynamic compounds. An integrated diagnostic approach combining clinical, biochemical, and environmental assessment is essential for identifying and classifying photosensitisation in animals.

Treatment and Control:

Treatment and control involve a symptomatic approach, as no specific antidote for phototoxins is available. Animals should be moved away from photodynamic plants and substances and placed in shaded areas, away from direct sunlight, for 5 to 7 days. Fluid or electrolyte therapy should be introduced to prevent electrolyte imbalance and dehydration. Ingesta can be eliminated from the stomach or rumen using laxatives or saline purgatives. Antihistamines and antibiotics can be administered intramuscularly. It is important to keep flies and ectoparasites away from skin lesions. Hepatoprotectants like liver tonics and stimulants can be administered to prevent liver damage.

Conclusion:

Photosensitisation is a management-related serious condition that requires early identification and prompt treatment to prevent severe skin damage and production losses. It significantly affects hide quality and overall productivity. Therefore, veterinary extension programs are essential to create awareness among farmers. Animals with pigmented skin are less susceptible, whereas young, sick, or non-pigmented

animals are at higher risk. Understanding its mechanisms, types, and preventive measures is important for effective control, though further research is needed to improve management and treatment strategies.

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