PHOTOSENSITIZATION

Photosensitization is sensitivity of skin to sunlight which causes dermatitis by the action of sunlight on certain photodynamic substances that may be present in the skin. Necrosis and edema are produced in the exposed areas of skin of animals. The cellular damage by photosensitization is due to release of reactive oxygen species leading to mast cell degranulation and production of chemical mediators of inflammation.

In sheep: face, muzzle, ears and back are affected whereas in cattle: teats, udder, vulva and other unpigmented parts are affected.

Factors necessary for photosensitization in animals

- Oxygen
- Sunlight
- Photodynamic chemicals
- Susceptible animals which have skin devoid of hair or wool and lacking pigments

Pathogenesis

- Radiant energy after penetrating into small cutaneous blood vessels, catalyses the oxidation of photodynamic substances. This produces the toxic substances which affects the vascular endothelium.
- Chemical mediators of inflammation released due to increased permeability of lysosomes of endothelial cells.
- Congestion, edema and thrombosis results. Edematous fluid may even oozes out of the skin.
- Necrosis and sloughing of skin is produced in the exposed areas of skin of animals.
- Infection of the wound by secondary organisms may occur.

Symptoms

- In sheep, affected edematous ears droop. Dysponea may occur due to swelling of muzzle. There may be swelling and closure of eyelids. Face appears swollen due to edema hence condition known as “Big Head”. Affected animals seek shady places.
- In cattle, mastitis can also found when teats are severely affected.
- Urine may be brown in colour.

Types of Photosensitization

- Type I: Primary photosensitization
- Type II: Photosensitization due to abnormal endogenous pigments
- Type III: Hepatogenous photosensitization

Type I: Primary Photosensitization

- Photosensitivity is due to exogenous materials which are absorbed directly by the intestinal mucosa and reach the skin. On exposure to sunlight, these substances in the skin produce lesions.

  Causes

  - Plants containing helianthrones (e.g. hypericine in *Hypericumperforatum*; fagopyrin in *Fagopyrumesculentum*) and furocoumarin pigments (e.g. *Cymopteruswatsonii* and *Ammimajus*), tetracyclines and sulphonamides.

  Pathogenesis

  - Phenothiazine is converted into sulfoxide in the intestine from where it reaches to the liver which is not able to convert all of it into harmless compounds. So, sulfoxide enters the circulation and reaches the skin causing photodermatitis on exposure to sunlight.
  - Urine is red in colour due to presence of dye.

Type II: Photosensitization due to abnormal endogenous pigments

It occurs due to excess of uroporphyrins which are basic pigments in the formation of haemoglobin.Due to inherited enzyme deficiency, abnormal porphyrin photodynamic metabolic products like uroporphyrin accumulate in blood and tissues. The uroporphyrin also causes discolouration of bone known as “osteohaemochromatosis” and teeth turns to pink on exposure to sunlight called “pink teeth”.

Causes

- Bovine congenital porphyria
- Bovine haematopoeticprotoporphyria

Type III: Hepatogenous photosensitization

- Hepatogenous photosensitization is caused by impaired hepatic capacity to excrete phylloerythrin derived from chlorophyll degradation in the alimentary tract by protozoa.
- Phylloerythrin accumulates in blood and produces photosensitivity.

Causes

- Hepatocellular damage or injury by hepatotoxic plants like*Lantana camara*, Calatrops and fungus Pithomyceschartarum.
- Inherited hepatic defects
- Biliary obstruction
- Infection: Leptospirosis
- Chemicals: CCl_4 poisoning

Pathogenesis

- Hepatotoxin directly acts on the liver cells and epithelium of bile ducts. This causes swelling of cells resulting in prevention of passage of bile causes jaundice.

Gross pathology

- Hairless, non-pigmented skin exposed to sun light shows erythema, edema, blisters, exudation, necrosis and sloughing of necrotic tissue.

Microscopic appearance

- Cells are swollen and manifest fatty degeneration.Coagulative necrosis of epidermis, subepidermalvesiculation, swelling of endothelial cells, fibrinoid degeneration and thrombosis of blood vessels leading to edema.