Photosensitization: A Case Report

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History and Physical Examination

A yearling black-baldy steer was presented to the Veterinary Medical Teaching Hospital on August 4, 1986. The owner reported that the steer had been depressed, losing weight, anorexic and not drinking. This steer was one of 220 calves purchased by the owner to be used as stockers. Of the 220 calves there were four currently sick and five dead. The steer presented was representative of the herd problem.

These calves had been on the present location for thirty days and the first calf noticed sick was fourteen days ago. The calves were on a pasture containing mixed bermuda and native grass. Eleven acres of the pasture contained alfalfa with *Kochia* mixed in. There was also a pasture of millet which the owner reported the calves would not eat.

Upon arrival the steer had a temperature of 103.9° F, heart rate 72 and regular and respiratory rate was 24. The steer was thin, weak and had a rough hair coat. The head was swollen and there was excess lacrimal and nasal discharge. The mucous membranes were pale and yellow and mucosal and nasal ulcers were present. The underside of the tongue was ulcerated and necrotic.

Diagnostic Plan

A complete blood count on August 5, 1986 revealed a WBC of 6000 cmm, a total protein of 9.7 and a fibrinogen of 1200. Urinalysis was normal.

Blood Chemistry results were:

	8/5/86	8/6/86
T. Bili	12.52	12.76
D. Bili	—	8.78
BUN	36.80	—
CREAT	2.20	—
T.P.	8.00	—
SGGT	89.00	114.00
SGOT	230.00	—
CPK	—	100.00
SAP	—	76.00
SDH	—	7.50
LDH	—	1185.00

Photosensitivity due to *Kochia* was suspected based on history and clinical signs and the steer was submitted for necropsy on August 7, 1986.

Necropsy Findings:

Skin: dermatitis, photodynamic
Liver: hepatitis, interstitual (portal, mild)
Esophagus: esophagitis
Omasum: ulceration
Kidney: nephritis, interstitial (acute)
necrosis, acute tubular
glomerulitis, acute

Discussion

Under certain circumstances animals become hypersensitive to light. Hypersensitivity can be traced to the presence of a pigment (photodynamic agent) in the peripheral circulation which is not normally found there. Photosensitization is a condition caused by the sensitization of the superficial layers of lightly pigmented skin to sunlight. This must be differentiated from sunburn in which lightly pigmented skin slowly becomes inflamed following exposure to rays of sunlight in the ultraviolet range.

The exact biochemical mechanism by which photosensitization is produced is not yet clear. The basic reaction is probably an oxidation enhanced by energy from incident light trapped and made chemically available by the photodynamic agent. Both light and the presence of a reactant substance in the peripheral circulation are required. There is some evidence to suggest that proteins such as histidine, tryptophan and tyrosine form the substrate in which the light enhanced oxidations take place and the observed changes are due to the resulting cellular damage or alterations in permeability of the cell membrane.

The reaction produced occurs in areas of unpigmented or lightly pigmented skin which is not covered by a dense light-screening coat of hair.[1,2]

Photosensitizing agents cover a broad class of chemicals, most of which are from plant material. They reach the skin by contact, ingestion or from aberrant metabolism of hemoglobin. Photosensitization is classified as primary, hepatogenous or aberrant pigment metabolism.

Primary Photosensitization: Primary photosensitization is due to the ingestion of exogenous photodynamic agents. The photodynamic agent is absorbed from the digestive tract and reaches the skin unchanged. The plant must be eaten in large quantities to produce clinical signs so is usually the dominant plant on the pasture. Livestock are usually affected four to five days after going onto pasture. The entire disease process is that of photosensitization.

Hepatogenous Photosensitivity: This class of photosensitivity diseases develops secondarily to and as a direct result of hepatic function. Hepatogenous photosensitivity is due to the photosensitizing agent phylloerythrin (a porphyrin) which is derived from the anaerobic breakdown of

chlorophyll by the microorganisms in the forestomachs of ruminants. Phylloerythrin is normally effectively excreted by the liver into the bile. Any condition which markedly interferes with the excretion of bile and thus phylloerythrin may lead to photosensitivity. Hepatogenous photosensitization is more common in animals grazing green pasture, but can occur in animals fed entirely on hay or other stored feeds.

Aberrant Pigment Metabolism: Aberrant pigment metabolism produces photosensitivity as a result of defects in the synthesis of hemoglobin. This results in an accumulation of the photodynamic agent in the body. The only known example of this is Congenital Porphyria (Pink Tooth).

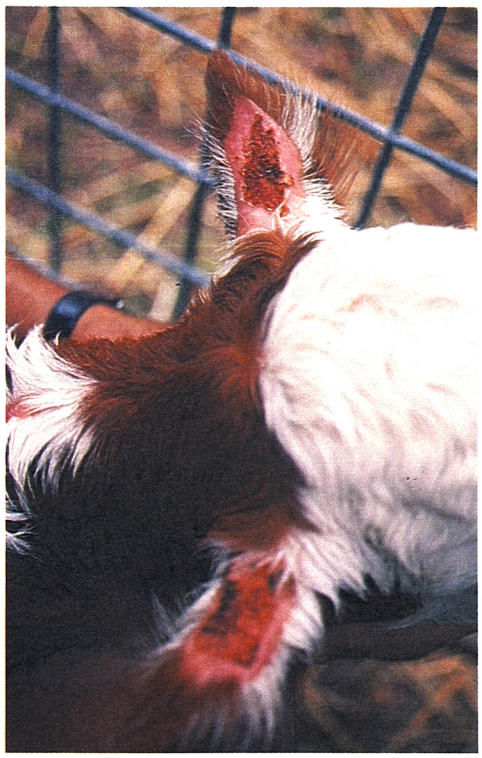
Substances Causing Photosensitization[2]

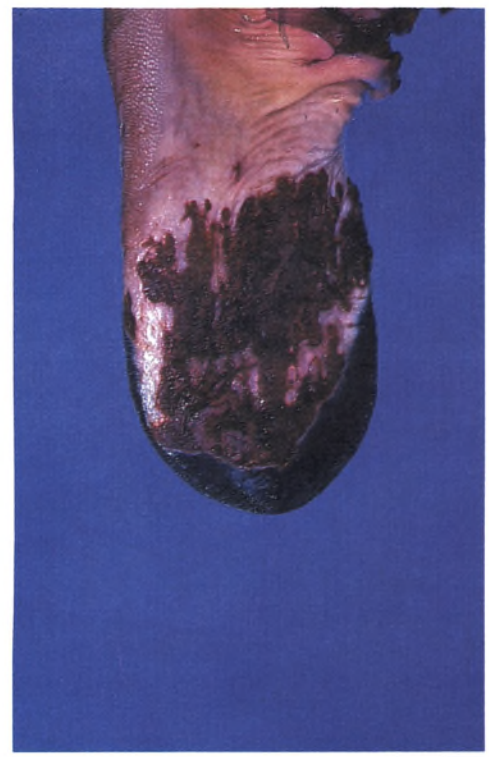
Primary:

Polygonum fagopyrum (Buckwheat)
Hypericum perforatum (St. Johnswort, Klamath weed)
Lolium perenne (perennial ryegrass)
Cynopterus sp. (wild carrot)
Phenothiazine
Rose bengal
Acridine dyes

Hepatogenous:

Agave lecheguilla (lechuguilla)





Brassica napus (cultivated rape)
Kochia
Panicum sp. (panic and millet grasses)
Lantana camara (lantana)
Carbon tetrachloride poisoning
Corticosteroids used systemically to terminate parturition in cows.
Leptospirosis

Onset of clinical signs varies with the causative agent. Photosensitive animals show photophobia when exposed to sunlight. The affected animals seek out shade and if none is available they turn their backs to the sun. Lesions are usually confined to lightly pigmented, exposed areas of skin, and are more severe on the dorsal parts of the body and on those underparts exposed to sunlight. Predilection sites include the ears, eyelids, muzzle, face, lateral aspects of the teats and the vulva and perineum.

Erythema develops rapidly and is followed by edema. Irritation at this stage is intense and the animal will rub the affected parts. Rubbing trauma can result in secondary invasion of the necrotic areas with bacterial infections. Prolonged exposure to sunlight results in marked serum exudation with matting of the hair and in severe cases closure of the eyelids. The edema if severe, may cause drooping of the ears, dyspnea due to nasal obstruction and dysphagia due to swelling of the lips. Necrosis and gangrene with sloughing of affected parts may occur.

With severe phylloerythrinemia and bright sunlight, even black-coated cattle can develop lesions.

In cattle, exposure of the tongue when licking can result in glossitis with ulceration and deep necrosis. Icterus may be present if the photosensitizing agent is hepatogenous in origin.

Severe skin lesions may cause shock and there will be an increased pulse rate, ataxia and weakness. A marked temperature increase may be noticed. Nervous signs including blindness, posterior paralysis, depression or excitement are often observed.

The photodynamic action by itself is rarely lethal, but deaths are frequent from starvation, other secondary effects or liver damage.[3 4 5]

Diagnosis of photosensitivity is based on history of access to the toxic plant or substance and distribution of skin lesions. If the photosensitivity is hepatogenous in origin there may be secondary signs of liver dysfunction.

Treatment of photosensitivity includes removal of animals from direct sunlight, prevention of ingestion of further toxic material and administration of laxatives to eliminate toxic materials already present. Corticosteroids and antihistimines administered early may be helpful. Secondary skin infections and fly strike should be prevented.[6]

Prognosis depends on severity of the lesions and the degree of resolution of the lesions.

Conclusion

Photosensitizing agents cover a broad class of chemicals. Photosensitization is classified as primary, hepatogenous or aberrant pigment metabolism. Diagnosis is based on history of access to the toxic material and clinical signs. General and local treatment should be initiated immediately in order to control the disease process.

References

1. Kingsbury, J.M.: Poisonous Plants of the United States and Canada. Englewood Cliffs, N.J.: Prentice-Hall Inc. 1964, pp 52-57. 2. Blood, D.C.; Radostits, O.M. and Henderson, J.A.: Veterinary Medicine 6th edition. Bailliere Tindall, 1983, pp 437-439. 3. Dickie, C.W. and James, L.F.: Kochia Scoparia poisoning in Cattle. J Am Vet Med Assoc 183:765-768, 1983. 4. Glenn, B.L.; Monlux, A.W. and Panciera, R.J.: A hepatogenous photosensitivity disease of cattle: I. Experimental production and clinical aspects of the disease. Path. Vet. 1:469-484. 1964. 5. Glenn, B.L.; Monlux, A.W. and Panciera, R.J.: A hepatogenous photosensitivity disease of cattle: II. Histopathology and pathogenesis of the hepatic lesions. Path. Vet. 2:49-67, 1965. 6. Black, L.: Photosensitization in Howard: Current Veterinary Therapy I, Food Animal Practice. W.B. Saunders Co., 1981, pp 1125.