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Equine Dermatitis

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who had abused him, and beat him across the eye with a heavy, studded collar. The Humane Society took the dog from the trainer and gave him to the present owner. A similar wound around the right eye had healed, but the wound on the left eye failed to respond to treatment and remained in a raw condition for a period of one year.

A month of treatment with 5 per cent tannic acid, boric acid spray, mercurochrome dusting powder, pellitol, and boric acid-urea powder failed to bring about any indication of healing.

After these agents failed to get results, a new preparation was suggested. Twenty 5 grain sulfanilamide tablets containing sodium bicarbonate were ground in a mortar and 2 grains of urea were added. This powder was dusted on several times a day.

In three days the ulcer had become a dry wound and in a week the edges had begun to granulate. Then granulation tissue appeared in the center of the denuded area. Three weeks after this treatment was started, the area was completely covered with new skin, and the patient was sent home.

—C. H. Burnham, ’42

Equine Dermatitis. On the morning of June 14, 1941, Dr. L. E. Smith of Jefferson, Iowa, was called in consultation by a neighboring veterinarian to observe a rather unusual skin condition in a group of horses. The client had lost one animal, had four more with lesions, and had two animals that were not affected.

The lesions consisted of vesicular eruptions on the muzzle, eyelids, and legs which were developing toward necrosis and subsequent sloughing. The inflamed areas were swollen and traumatized due to an intense pruritis. The necropsy revealed a severe stomatitis, gastritis and hemorrhagic to necrotic enteritis.

After questioning the client it was discovered that the affected animals had been grazing in a different pasture than those that were not affected. An inspection of the pasture showed a short, half-dead growth of alsike clover that was covered with innumerable small brownish-black fungus colonies. This growth was later identified as the fungus, Uromyces trifolii, by a plant pathologist.

The lesions were treated with white lotion, and the owner was advised to keep his horses out of the clover pasture. When we saw the animals a few days later, they were recovering nicely.

In searching for the etiologic factor for the above condition, one finds two possible explanations. First, a luxurious growth of a fungus that has been previously suspected as being pathogenic was present on the clover. This suspected pathogenicity, however, has never been substantiated by experimental evidence. Secondly, alsike clover is known to contain a photosensitizing agent which, when ingested, will cause this type of lesion in the unpigmented areas of the skin if the animals are exposed to sunlight. It will also produce superficial sloughing of the mucous membranes of the lips and the tip of the tongue, vesicles on the mucous membranes within the mouth, and general digestive disturbances. This latter explanation would be quite plausible in grey horses or in dark horses with white points or markings, but this particular group were bays and blacks.

Whether this skin disease was due to fungus poisoning or to photosensitization is debatable, but in either case the treatment would be essentially the same, that of local wound treatment and a change of pasture.

—M. W. Karber, ’43

“Clay Pigeon” Poisoning in Swine. The latter part of June, 1941, one live and two dead pigs, weighing about thirty pounds each, were presented at the Diagnostic Laboratory, University of Minnesota, for the diagnosis of a herd problem.

A history of sporadic illness resulting in death in a short time was obtained. The pigs were permitted to run on pasture. The losses, in two weeks, had been fifteen pigs from a group of forty. Mature animals running with the pigs showed no signs of disease.