Zinc-Responsive Dermatitis

Features
The characteristic dermatitis may be seen with true zinc deficiency or as an idiopathic zinc-responsive condition. Causes of deficiency include diets deficient in zinc; diets with excessive calcium, iron, phytates, and other chelating agents; drinking water with excessive iron and other chelating agents; and genetic abnormality in zinc absorption (see Chapter 1.6). Zinc-responsive dermatoses are uncommon to rare. These are no apparent breed, sex, or age predilections.

More or less symmetrical erythema and scaling progress to crusting and alopecia. The face, pinnae, mucocutaneous junctions, pressure points, distal legs, flanks, and tail head are typically affected (Figs. 1.8-1 and 1.8-2). Some animals have a dull, rough, brittle hair coat. Pruritus may be intense or absent. Secondary bacterial skin infections are common. Truly zinc-deficient animals have accompanying systemic signs (decreased appetite and growth rate, weight loss, decreased milk production, depression, stiff joints, diarrhea), whereas animals with the idiopathic condition do not. With true zinc deficiency, multiple animals are often affected. With the idiopathic condition, a single animal is typically affected.

Differential Diagnosis
Dermatophytosis, dermatophilosis, staphylococcal folliculitis, demodicosis, stephanofilariasis, sterile eosinophilic folliculitis and furunculosis, and sarcoptic mange (when pruritic).
Section 1: Bovine

Diagnosis

1) Dermatohistopathology: Hyperplastic to spongiotic superficial perivascular-to-interstitial dermatitis with marked diffuse parakeratotic hyperkeratosis and a lympho-eosinophilic inflammatory infiltrate.
2) Analysis of diet.
3) Response to therapy.

Vitamin C–Responsive Dermatosis

Features

Vitamin C–responsive dermatosis is an uncommon cosmopolitan disorder. It has been theorized that the condition may represent a temporary vitamin C deficiency in growing calves. The condition is most often seen in the fall and winter in temperate climates. The disorder is seen in dairy calves, 2 to 10 weeks of age, with no apparent sex predilection.

Moderate to severe scaling, alopecia, occasional crusts, and easy epilation of hairs begin on the head and/or limbs (Figs. 1.8-3 to 1.8-7). Extremities are erythematous, and petechiae and ecchymoses are seen. Severely affected calves have widespread disease and may be depressed and grow slowly. Pruritus is absent.

Differential Diagnosis

Anagen defluxion.

Diagnosis

1) Dermatohistopathology: Diffuse orthokeratotic hyperkeratosis, curlicue hairs, vascular dilatation and congestion, and periadnexal hemorrhage.
2) Response to therapy.
## Miscellaneous Nutritional Disorders

**Table 1.8-1** Miscellaneous Nutritional Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A deficiency</td>
<td>Very rare; deficient diet; rough, dry, faded hair coat and generalized seborrhea; systemic signs (night blindness, excessive lacrimation, corneal changes, neurologic disorders, skeletal abnormalities); serum and liver concentration of vitamin A</td>
</tr>
<tr>
<td>Riboflavin deficiency</td>
<td>Very rare; deficient diet; generalized alopecia; systemic signs (anorexia, poor growth, excessive lacrimation, hypersalivation, diarrhea); riboflavin levels in diet</td>
</tr>
<tr>
<td>Cobalt deficiency</td>
<td>Very rare; deficient diet; rough, brittle, faded hair coat; systemic signs (decreased growth and lactation); serum or liver concentration of cobalt and vitamin B₁₂</td>
</tr>
<tr>
<td>Copper deficiency</td>
<td>Rare; primary (deficient diet) or secondary (excess cadmium, molybdenum, or zinc in diet); rough, brittle, faded hair coat with variable excessive licking; periocular hair coat fade and hair loss (“spectacles”); systemic signs (poor growth, diarrhea, anemia, bone disorders, infertility); serum and liver concentrations of copper</td>
</tr>
<tr>
<td>Iodine deficiency</td>
<td>Very rare; maternal dietary deficiency; newborn calves; generalized alopecia and thick, puffy skin (myxedema); systemic signs (weakness, neurologic disorders); serum concentration of thyroid hormone and thyroid gland pathology</td>
</tr>
<tr>
<td>Selenium deficiency</td>
<td>Rare and poorly documented; deficient diet; dermatitis over rump and tail base; systemic signs (infertility, retained placenta, respiratory disease, foot and leg problems); serum and liver concentrations of selenium</td>
</tr>
<tr>
<td>High-fat milk replacer dermatosis</td>
<td>Rare; calves; alopecia and scaling, especially on muzzle, periocular area, base of pinnae, and limbs</td>
</tr>
<tr>
<td>General nutritional deficiency</td>
<td>Rare to uncommon; generalized dull, dry, thin hair coat and scaling; nonpruritic</td>
</tr>
</tbody>
</table>

**Figure 1.8-6** Vitamin C–responsive dermatosis. Alopecia and scaling of pinnae.

**Figure 1.8-7** Vitamin C–responsive dermatosis. Alopecia and scaling over thorax.

**Figure 1.8-8** Copper deficiency. Faded hairs on face and around eyes (“spectacles”).

**Figure 1.8-9** Copper deficiency. Faded hairs in normally black hair coat. *Source:* Courtesy of E. Meissonnier, coll. J. Gourreau, AFSSA.
Figure 1.8-10 High-fat milk replacer dermatosis. Alopecia and scaling of muzzle and ventral neck.

Figure 1.8-11 General nutritional deficiency. Dull, dry, thin hair coat and diffuse scaling over rump.

Figure 1.8-12 General nutritional deficiency. Thin, dry hair coat and scaling on pinnae.

References
