**Title:** 18-year-old Thoroughbred gelding presented for more than one-year history of crusts on the coronary bands and pastern of the hindlimbs.

**Question:** Name the disease.

A. Staphylococcal pyoderma  
B. Pemphigus foliaceus  
C. Dermatophilosis  
D. Cutaneous vasculitis

**Signalment:** 18-year-old Thoroughbred gelding.

**Clinical history:**  
An 18-year-old Thoroughbred gelding was presented for chronic (more than 1 year) history of waxing and waning dermatitis affecting the coronary bands and pastern of the hindlimbs. On physical examination, there were multiple crusts with foul-smelling exudate on the skin of the pastern region and coronary band (Figure 1). No clinical response to antibiotics, antifungal shampoos, steroid creams, or systemic sulfameth/trimeth (SMZ).

**Histopathologic description:**  
Left coronary band: The epidermis is diffusely thickened (acanthosis) with multifocal segmental loss of epithelium (ulceration), covered by a thick layer of serocellular crust (Figure 2). Multifocally, the wall of the small superficial dermal venules and capillaries is expanded by homogeneously eosinophilic material with loss of vascular mural architecture (fibrinoid necrosis), and these vessels frequently contain fibrin thrombi (Figure 3). There are few neutrophils and karyorrhectic debris in the vascular wall, and the superficial and mid dermal vessels are surrounded by low numbers of neutrophils, lymphocytes, plasma cells, and red blood cells (hemorrhage) (Figure 4). The overlying epidermis demonstrates segments of swollen keratinocytes with cytoplasmic pallor (hydropic degeneration), widening of the intercellular spaces (spongiosis), occasional loss of differential staining (acute necrosis), and accumulation of compact laminated keratin without retention of the nuclei (orthokeratotic hyperkeratosis) (Figure 5). The serocellular crust is composed of degenerate neutrophils admixed with red blood cells, cellular debris, pale eosinophilic homogeneous fluid (serum), and abundant cocci (Figure 6).

**Morphologic diagnosis:**  
Haired skin: Moderate, multifocal, acute, superficial papillary dermal fibrinoid necrosis with fibrin thrombi, dermal hemorrhage, epidermal hyperplasia, ulceration, and necrosis with intracorneal cocci.
Comments:
The primary process represented in the current case is superficial dermal vasculitis with thrombosis. Cutaneous vasculitis is a reaction pattern which has been associated with a wide range of causes, including infectious agents (bacteria, fungi, and viruses), ultraviolet radiation, adverse drug reaction (e.g. penicillin, phenylbutazone, and acepromazine), and idiopathic vasculitis. The pathogenesis of cutaneous vasculitis in horses is not completely understood. A type III hypersensitivity reaction, potentially associated with an infection, with immune complex formation and deposition in vessel walls has been proposed. *Streptococcus equi*-induced purpura hemorrhagica is reported to be the most common associated cause of cutaneous vasculitis in horses. Other infectious causes for vasculitis include equine influenza virus, equine infectious anemia virus, equine viral arteritis virus, *Anaplasma phagocytophila*, *Burkholderia mallei*, *Corynebacterium pseudotuberculosis*, *Staphylococcus aureus*, *Staphylococcus pseudintermedius*, *Streptococcus zooepidemicus*, *Rhodococcus equi*, *Proteus* spp., and *Pseudomonas aeruginosa*. Cutaneous lesions such as purpura, wheals, edema, erythema, nodules, necrosis, “punched-out” ulcers, and crusts usually occur on the distal limbs, pinnae, lips, and periocular area.

Equine pastern leukocytoclastic vasculitis is a unique syndrome that predominantly affects the unpigmented skin of the distal extremities. This disease is commonly seen in summer with exposure to sunlight; therefore, ultraviolet radiation is suggested to play a role in the disease pathogenesis. However, this syndrome is also associated with other causes of vasculitis as abovementioned.

The cause for vasculitis in the present case is not apparent. Given that the dermatitis affects the pigmented skin, causes other than photo-aggravated dermatitis are considered more likely, and clinical testing for ruling out infectious and drug-related causes is warranted. The identity of the intralesional coccis is uncertain since a bacterial culture of the lesion was not performed. Although the association of the intralesional bacteria with vasculitis cannot be determined, the bacteria in the present case likely represents secondary infection since the organisms were only present on the surface of the lesion.

References:

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Figures:

Figure 1.
Figure 6.