The nose knows (nasal planum diseases)

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Anatomy

The nasal planum has a very thick epidermis. It contains sweat (eccrine) glands and does not contain hair follicles. It is designed to withstand mechanical trauma.

Diagnostic approach

Diseases restricted to haired skin will not affect the nasal planum, allowing many diseases to be excluded from the outset, e.g. demodicosis. Take some time to consider the structures present and the process that is unfolding on the nasal planum and the reason for it – this will help narrow things down.

Clinical presentations can include depigmentation, erosions, ulceration, crusting, loss of the normal cobblestone architecture, hyperkeratosis and swelling.

During the physical examination think about whether the nasal planum is the only area affected or whether there are other dermatological or systemic signs that can give you clues as to aetiopathogenesis.

Cytology from the nasal planum may be useful in some cases (hyphae in dermatophytosis, acantholytic cells in pemphigus), but in other cases may be of limited use as inflammatory cells and cocci are commonly seen. Staphylococcal bacteria which colonise or infect the nasal planum may exacerbate clinical symptoms and can make histological interpretation difficult. If cocci are seen in significant numbers or are intracellular antibiotic therapy with an appropriate antibiotic (cephalexin or clindamycin) for at least two weeks prior to biopsy will allow you to get a better picture of underlying disease if present and will assist the pathologist with interpretation.

The nasal planum is well innervated and vascularised, so ideally biopsies should be taken under general anaesthesia rather than sedation.

Depigmentation

The normal process of skin pigmentation occurs when melanocytes transfer melanin to epithelial cells located in the basal cell layer of the epidermis. Depigmentation occurs when melanin transfer is disrupted. This can occur due to melanocyte injury, loss of melanocytes, or basal cell injury.

When there are alterations to the pigment of the nasal planum we need to consider whether:
1. disease is targeting melanocytes (loss of pigment without loss of cobblestone architecture) or
2. disease is targeting basal cells (pigment change with loss of cobblestone architecture or erosions), or
3. basal cells are being disrupted due to infiltration with bacteria, inflammation or a neoplastic process.

Infiltration will often lead to swelling or changes in the shape of the nasal planum.

Erosions and ulcers

An erosion is a partial thickness loss of the epithelium while an ulcer is a full thickness loss (deeper). They may be covered by a crust. Haemorrhagic crusts (blood) are dark due to a deeper involvement including blood

vessels, while neutrophilic crusts are yellow and often more superficial.

Loss of cobblestone architecture

Diseases damaging the basal cell layer interfere with cell turnover and cause thinning of the nasal planum. As the skin thins more and more and pigment is lost, the nose looks pinker due to the blood vessels being seen through the thin epidermis. Thinning of the nasal planum generally precedes erosion and ulceration.

Hyperkeratosis

Epithelial turnover is controlled by genetic factors as well as hydration. Dry skin is a major driver of increased skin turnover. When hyperkeratosis is present, diseases of keratinisation, viral diseases and diseases that affect hydration of the nasal planum, e.g. sweat gland dysfunction need to be considered.

Swelling

If the dermis is invaded by a population of cells (infectious, inflammatory or neoplastic), the shape of the nasal planum will be altered. Erosion or ulceration may occur if the swelling effects nutrition of the basal cells or if there is bystander injury to the basal cells.

Non inflammatory depigmenting conditions

Idiopathic nasal depigmentation

Dogs with a “Dudley nose” have less nasal pigmentation than normal, usually present from birth, with no loss of architecture. It may wax and wane. Common in huskies and Labrador and golden retrievers. Snow nose is a seasonal decrease in nasal pigmentation (lighter coloured nose in winter months) with no architectural change, common in huskies, Labrador and golden retrievers and Bernese mountain dogs. Neither of these conditions are associated with inflammation. They are cosmetic problems only and do not require treatment.

Vitiligo

Loss of melanocytes with normal architecture. Rare disease characterised by symmetric macular or patchy leukoderma and leukotrichia that commonly affects multiple areas simultaneously, including the nasal planum. Pathogenesis is unknown, but there may be a hereditary predisposition as it is most commonly seen in rottweilers, Belgian Tervuren dogs and old English sheepdogs. Considered a cosmetic disease in dogs with no consistently successful intervention reported, although spontaneous resolution has been observed.

Hyperkeratotic conditions

Nasal parakeratosis of Labrador retrievers

Hyperkeratosis of dorsal nasal planum of young adult Labrador retrievers (6– 12 months old).

Nasodigital hyperkeratosis

Keratinisation defect occurring in older dogs. Dry skin which may become fissured.

Infectious diseases

Mucocutaneous pyoderma

Superficial toxic reaction +/- immunological targeting of keratinocytes triggered by bacteria. Crusting, depigmentation, more common in German Shepherds and crosses. Often lateral nares or alar wing fissures. Intracellular cocci may be seen. Antibiotic therapy is indicated in any case where MCP is a primary or secondary possibility.

Dermatophytosis

Crusting, depigmentation of the haired skin of the muzzle and face also commonly affected with scaly or crusty lesions. Fungal culture using scale and crusts should be performed.

Environmental conditions

Solar (actinic) dermatitis

UVA and UVB injury to normal non pigmented skin and eyelids. More common in collies, border collies, Shetland sheepdogs, bull terriers, Dalmations.

Contact dermatitis

Inflammatory disruption of epidermis, lips also commonly affected, history of plastic or rubber food dishes.

Trauma

Acute onset, history compatible.

Metabolic conditions

Zinc responsive dermatosis

Metabolic disruption of epidermal turnover/repair. Alaskan malamutes and Siberian huskies. Also look for lesions around eyes, on footpads and on bony prominences.

Auto-immune conditions

Pemphigus foliaceus

Immunological attack on intracellular connections in subcorneal epithelium. Genetic UV and other factors. Neutrophilic crusting with or without depigmentation. Usually affects follicular (haired) skin of the face limbs and trunk, unusual to just affect nasal planum.

Discoid lupus erythematosus

Immunological attack directed against basal cells (due to genetics, UV light and other triggers). Haemorrhagic crusting, depigmentation, ulceration and architectural change. Eyelids and haired skin may be affected. More common in collies, border collies, Shetland sheepdogs, kelpies.

Uveodermatologic syndrome

Melanocyte destruction. Skin may be normal other than the pigment loss, i.e. normal architecture. Extensive depigmentation affecting lips, eyelids, footpads, anus, hard palate +/- leukotrichia, dermatologic disease may precede uveitis. Akitas, chows, samoyeds, Siberian huskies. Check for uveitis.

Dermal arteritis

Ischaemia due to vascular targeting. Ulceration of the philtrum between the nostrils, young adult onset in St Bernards and Newfoundlands.

Cutaneous vasculitis

Ischaemia. Haemorrhagic crusting, erosion, ulceration in JRT, Labradors. Usually lesions also seen on other extremities, eg. nails, tail tip, footpads and ear margins.

Neoplastic conditions

Cutaneous histiocytosis

'Clown nose' Inflammatory sterile infiltrate. Papules, nodules, infiltration of the planum or nares. Collies, shelties, golden retrievers.

Epitheliotrophic T cell lymphoma

Infiltrate in epidermis, depigmentation, ulceration, +/- nodules, haired skin also likely to be affected.

Squamous cell carcinoma

UV light affects non pigmented skin. Proliferative and or ulcerative.

Mast cell tumour

Infiltrate, swelling. Depigmentation. Disruption of melanin transfer and down regulation of melanin synthesis.