

Vitiligo in a Quarter Horse Filly: Clinicopathologic, Ultrastructural, and Nutritional Study

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ABSTRACT

Vitiligo is an autoimmune disorder characterized clinically by depigmentation of the skin—both epidermis and hairs—caused by destruction of melanocytes. Vitiliginous skin shows a T-lymphocyte inflammatory infiltrate, proliferation of Langerhans cells, epidermal vacuoles, and degenerative changes in the cutaneous nerve fibrils. Increased autoantibodies against melanin, tyrosinase, and various tissues are frequently present in vitiligo patients. Vitiligo is often associated with systemic abnormalities or nutritional deficiencies. A 2-year-old Quarter Horse filly developed facial vitiligo with depigmented areas that was strikingly similar to human vitiligo. Several innocuous small biopsies provided cutaneous specimens that were processed for light and electron microscopic studies. Many of the clinical and microscopic changes observed in human vitiligo were present in this equine patient. Marginal anemia was detected. Strengthening of the filly's nutritional and feeding conditions led to rapid and complete repigmentation. Equine practitioners as well as horse owners should be aware that vitiligo may not be simply a cosmetic problem; thus, a complete evaluation of horses affected by this condition should be performed.

Keywords: Vitiligo; Clinical features; Light microscopy; Ultrastructure; Management

INTRODUCTION

Vitiligo is an acquired autoimmune disorder characterized by whitening of the skin—epidermis and hairs—caused by destruction of melanocytes. In humans, vitiliginous skin shows a T-cell lymphocytic inflammatory infiltrate, proliferation of Langerhans cells, vacuole formation in the epidermis, and degenerative changes in the cutaneous nerve

fibrils. Systemic and nutritional abnormalities, as well as increased autoantibodies against melanin, tyrosinase, and various tissues are frequently present in human vitiligo patients.¹

A 2-year-old Quarter Horse filly developed multiple small depigmented facial areas exhibiting a striking similarity to human vitiligo. This report describes our clinicopathologic experience with this vitiligo patient, including light and electron microscopic observations, the latter to our knowledge not yet reported in equine vitiligo. This study was part of our continuing efforts in the field of vitiligo and is aimed to describe another similarity between human and equine dermatology, a task in which we have been involved during the past 30 years.²

MATERIALS AND METHODS

Case Report

A two-and-a-half-year-old bay Quarter Horse filly, unexpectedly and without any apparent reason, started to develop small depigmented macules on her muzzle (Fig. 1). These macules increased rapidly in number and size until they became quite noticeable, thus leading to great concern for the owner because of the striking change in the appearance of the filly. Neither the attending veterinarian nor the people at the stable boarding the horse had seen this skin condition before. Therefore, they sought our evaluation and advice.

Dermatologic Examination

Careful inspection of the entire skin on both sides of the muzzle revealed multiple depigmented macules, which were bilaterally distributed and very outstanding because of the darker color of the surrounding unaffected skin. A few depigmented areas also were seen near the eye (Fig. 1). The condition did not seem to cause any discomfort, such as itching or pain, but it became cosmetically disturbing.

Laboratory Studies

A complete equine panel was requested. Although no severe abnormalities were detected, the following marginal or abnormal values were noticed: hemoglobin, 11.0 g/dl (11.0–19.0); hematocrit, 32.9% (32.0–53.0); red blood

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Figure 1. Multiple depigmented macules symmetrically located on both sides of the muzzle.

cell count, $7.06 \times 10^6/\text{mm}^3$ (6.80–12.90); ratio of albumin to globulins, 1.06 (0.26–0.91); globulin, total, 2.9 g/dl (2.6–4.0); bilirubin, direct, 0.49 mg/dl (0.00–0.50).

Histopathology

Several punch-biopsy specimens obtained under local 1% Xylocaine injected intradermally² were processed for light and electron microscopy, using methods previously described.^{3,4}

RESULTS AND DISCUSSION

Clinical Features

This patient's vitiligo falls into Lerner's category of segmental vitiligo,¹ namely, a localized depigmentation corresponding to a dermatome. Furthermore, from the standpoint of its clinical course, this vitiligo was of an enlarging type, developing new lesions and thus falling also into Behl's¹ category of progressive vitiligo.

Histopathology

Light Microscopy. Comparison of skin from depigmented areas with uninvolved skin revealed striking differences (Fig. 2). The depigmented areas showed mild hyperkeratosis and almost complete disappearance of melanin in the stratum spinosum. There were cells with clear cytoplasm in a suprabasal location believed to be Langerhans cells.

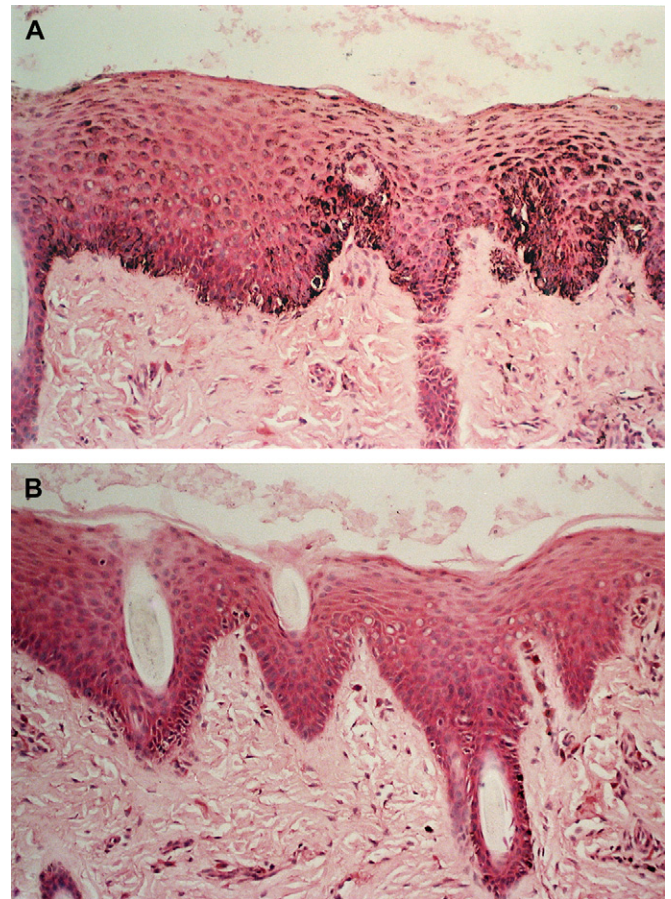


Figure 2. Epidermis and superficial dermis comparing uninvolved pigmented skin (A) with involved depigmented skin (B). Notice the normal amount of melanin in A and the diminished, almost absent, melanin in B. Methyl Green-Pyronin stain (original magnification $\times 50$).

Similar to human vitiligo,^{1,5,6} the superficial dermis displayed an inflammatory infiltrate, mostly in a perivascular location (Fig. 3).

Electron Microscopy. Transmission electron microscopy confirmed the findings of light microscopy. Langerhans cells were particularly prominent in the deeper layers of the stratum spinosum (Fig. 4). They were outstanding among keratinocytes because of their electron-dense lobulated nucleus and their more electron-translucent cytoplasm. They showed numerous mitochondria and only a few Langerhans cell granules or no granules. Schuler et al⁷ have described different subsets of Langerhans cells, emphasizing that Langerhans cells do not represent an entirely homogeneous population. For example, Langerhans cells in cultured murine epidermis or in the lung may lack Langerhans cell granules,⁷ as in this patient. In addition,

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