Clinical, histological and immunohistochemical study of feline viral plaques and bowenoid *in situ* carcinomas

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What is known about the topic of this paper
- Reports of papillomavirus-induced dermatitis in cats are rare.
- Lesions of feline viral plaques have been described as feline hyperpigmented plaques and are clinically indistinguishable from lesions of bowenoid *in situ* carcinomas.
- Feline bowenoid *in situ* carcinoma could be, like feline viral plaques, papillomavirus-induced.

What this paper adds to the field of veterinary dermatology
- Clinically, feline viral plaques and feline bowenoid *in situ* carcinomas are indistinguishable.
- Feline viral plaques and feline bowenoid *in situ* carcinomas might have the same viral cause.
- Feline viral plaques could be a precursory lesion of feline bowenoid *in situ* carcinoma.

Abstract

Feline viral plaques (FVP) induced by papillomavirus (PV) are often hyperpigmented and flat warts. The fact that up to 47% of bowenoid *in situ* carcinomas (BISC), which also usually occur in the form of hyperpigmented plaques, are positive for PV antigen in immunochemistry suggests that BISC could evolve from FVP.

The relationship between the presence of PV antigens and the clinical and histological features of 26 cases of feline dermatoses (clinically described as pigmented plaques and with histological diagnosis of FVP and/or BISC) was therefore determined. The cases were classified into one of the three following groups: FVP, FVP + BISC or BISC. Immunohistological detection of papillomavirus group-specific antigen was performed using a polyclonal rabbit antibovine papillomavirus antiserum.

Of the seven cases in the FVP group, six were deemed positive by immunohistology as were all 10 cats in the FVP + BISC group. On the other hand, only one of the nine BISC cats was positive. The presence of both FVP and BISC lesions in some cats and the high detection rate of PV antigens in the FVP and FVP + BISC groups suggest that both conditions might have the same viral cause and that some BISC may evolve from FVP. The low rate of viral antigen detection in the BISC group indicates another cause or a loss of viral replication during the cancerogenesis.

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Introduction

Papillomaviruses (PV) are highly diverse viruses that usually induce benign skin or mucous membrane proliferation in mammals and birds but can also cause squamous cell carcinomas.[1] In humans, the PVs that induce benign hyperplasia and those that induce cancers are phylogenetically different.[1] Benign hyperplasias (warts) usually regress after a few months, a regression associated with the development of cell-mediated immunity.[2]

In contrast with dogs, where PV infections are frequently observed, reports of PV-induced dermatoses are rare in cats.[3–7] Lesions are usually flat and hyperpigmented, rather than exophytic and flesh colour warts, and spontaneous regression is rare.[3–7] These lesions are usually, but not always, multiple and have been described as feline viral plaques (FVP).[8]

Feline multicentric *in situ* squamous cell carcinomas also usually occur as multiple hyperpigmented plaques that resemble those of human Bowen's disease.[9,10] Gross and coworkers, however, recently remarked that there are major differences between the human and the feline diseases, and have coined the term 'bowenoid *in situ* carcinoma' (BISC) to describe the feline condition.[8] As FVP clinically resembles BISC, it was suggested that both conditions may have the same cause, and one report mentions the association of both FVP and BISC on the same cat.[11,12] Furthermore, it has been shown immunohistologically that up to 47% of feline BISC samples are positive for PV antigen, suggesting that BISC is virally induced and that FVP could be, at least in some instances, precursory lesions of feline BISC.[11]

Using records of the clinical, histological and immunohistological features of 26 cases of feline dermatoses clinically described as pigmented plaques and with an initial histological diagnosis of FVP and/or BISC, the hypotheses that both lesions are often associated in the same samples, and that PV antigens are present in the majority of these lesions, were tested.

Materials and methods

Animals

History and clinical information was obtained from 26 cats with hyperpigmented plaques. Cats were included, provided that a histological diagnosis of FVP and/or BISC had been made previously, and clinical data (including concurrent diseases, immunosuppressive therapy and evolution of the lesions, when available) were subsequently analysed for each of the three histological groups: FVP, FVP + BISC and BISC.

Statistical analysis

Data were analysed using nonparametric statistical methods (GraphPad PRISM® for Windows, version 4.0; GraphPad Software, Inc., San Diego, CA, USA). Kruskal–Wallis one-way ANOVA by ranks and the Dunn's post-test for multiple comparisons were used to compare ages among the three histological groups.

Histological evaluation

Archival specimens of all 26 cats were compiled. These samples have been previously collected by biopsy from all 26 cats, fixed in formalin, and processed routinely to paraffin wax for histological assessment. Sections (5 μm) were cut, routinely processed and stained with haematoxylin and eosin. The following criteria were systematically assessed: severity and nature of the acanthosis, hypergranulosis and size of the keratohyalin granules, premature keratinization, involvement of the hair follicle in the pathological process, disorderly or abnormal maturation of the epidermis, atypia (pleomorphic or abnormally large nuclei, multinucleate cells), mitoses more than three cell layers above the basal cell layer, koilocytosis, clear cells and presence of intracytoplasmic pseudo-inclusions and intranuclear inclusions. Koilocytes were defined as keratinocytes with swollen cytoplasms and shrunken nuclei.[8] Clear cells were defined as keratinocytes with swollen cytoplasm but rather enlarged, vesicular nuclei. These modified keratinocytes (clear cells and koilocytes) have been reported to be also regularly associated with human PV infection.[13] When observed, the margins of the lesions were checked for changes suggestive of viral infection such as koilocytes and clear cells, pseudo-inclusions, and clumped keratohyalin granules.

Samples were subsequently classified into one of three groups: FVP, FVP + BISC (when both lesions were present on the same cat or on the same section) or BISC in accordance with standard criteria (Table 1) for the diagnosis of FVP and BISC.[8] When changes overlapping typical FVP and BISC lesions were observed, lesions were designated as FVP, provided that the acanthosis remained moderate and atypia was absent. Lesions were classified as BISC if the acanthosis was marked and loss of polarity as well as atypia was evident.

Immunohistochemical analysis

Papillomavirus antigen was detected (at the Immunology Laboratory of Prairie Diagnostic Services, Saskatoon, Saskatchewan, Canada) using an avidin–biotin complex technique adapted for an automated slide stainer (Codon Histomatic Stainer, Fisher Scientific, Edmonton, AB, Canada) as previously described.[14] This method has already been validated for the detection of feline PV antigens.[7] Briefly, sections from each tissue block were mounted on slides (Codon Slides, Fisher Scientific, Edmonton, AB, Canada) coated with 0.1% poly-D-lysine, digested with protease XIV (Sigma Chemical Co., St. Louis, MO, USA) for 20 min at 42 °C and treated with a 1 : 2000 dilution of rabbit antibovine papillomavirus type-1 antibody (Dako Diagnostics Canada Inc., Missisauga, ON, Canada). A goat-biotinylated antirabbit IgG (Vector Laboratories Inc., Burlington, ON, Canada) was used at a 1 : 400 dilution as the secondary antibody. Replicate sections were stained as above without protease digestion, and additional sections were stained with a normal rabbit antiserum as the primary antibody to provide negative control. A positive control tissue, canine cutaneous papilloma, was included in each assay run.

Both diaminobenzidine (DAB) (Electron Microscopy Sciences, Fort Washington, PA, USA) and Nova Red (Vector Laboratories Inc., Burlington, ON, Canada) were used as chromogens on two different sections for each sample.

Results

Clinical information

The clinical data are summarized in Table 2. Differences between ages of cats in FVP, FVP + BISC and BISC groups (median 11.5, 12 and 13, respectively) were not statistically significant. The sizes of the groups did not allow a proper evaluation of potential breed or sex predispositions.

On clinical examination, FVP and BISC lesions were often indistinguishable and usually presented as solitary or multiple grey, tan to black papules or small flat plaques (Figs 1 and 2). Some, more frequently the BISC, appeared ulcerated (Fig. 2). Solitary lesions were observed in only three of the 26 cats. The face, neck and limbs were mostly affected by BISC. FVP occurred mostly on the trunk, even if other areas, including face and neck, were also affected. Cats with both conditions usually presented lesions on more than one body area and all body regions could be affected. Very little follow-up information was available but cases of transformation of FVP into BISC after the initial histological diagnosis were not recorded. None of the affected cats had a known history of immunosuppressive drug administration or concurrent disease.

Histological examination

The results are summarized in Table 3.

FVP

The diagnosis of FVP was made in seven cases (Table 3). Lesions consisted of well-demarcated epidermal hyperplasia with acanthosis, hyperpigmentation, hypergranulosis with clumped keratohyalin granules and numerous koilocytes (Fig. 3). Some of these keratinocytes contained blue-grey fibrillar pseudo-inclusions (one of seven). Larger and compact amphophilic intracytoplasmic pseudo-inclusions were present in four cases (Fig. 3). In one case, both pseudo-inclusion types were present in the same sample and compact ones (present in the stratum granulosum)

Table 1. Histological features of feline viral plaque and bowenoid *in situ* carcinoma

	Feline viral plaque	Bowenoid *in situ* carcinoma
Acanthosis	Mild to moderate	Moderate to severe
Follicular involvement	Sometimes	Yes
Differentiation	Normal	Dysplastic epidermis, loss of polarity
Clumped keratohyalin granules	Yes	Yes
Koilocytes	Yes	Yes
Intracytoplasmic pseudo-inclusions	Yes	Yes
Atypia	No	Yes
Mitotic activity	No	Moderate

Table 2. Clinical findings

Case	Breed	Sex	Age (years)	Lesions	Multiple/Solitary	Localization
1	DSH	M	14	Crusty plaques	Multiple	Thorax, face, shoulder
2	Cornish Rex	M	12	Papillary plaques	Multiple	Shoulder, paw, trunk, abdomen
3	DSH	F	11	Plaque	Solitary	Neck
4	DSH	F	15	Plaques, papules	Multiple	Face, feet, abdomen
5	DSH		18	Papules	Multiple	Flank
6	DSH	M	12	Crusts, plaques	Multiple	Face and digits
7	DSH	F	13	Crusts, plaques	Multiple	Eyelids, face
8	American curl	M	14	Crusts, plaques	Multiple	Face, neck
9	DSH	F	13	Papules	Multiple	Unknown
10	DSH	F	12	Papules	Multiple	Flank
11	Sphynx	F	6	Macules	Multiple	Neck
12	DSH	M	3	Ventral papules	Multiple	Abdomen
13	DSH	M	16	Plaque	Solitary	Abdomen
14	DSH	F	8	Papules	Solitary	Dorsum
15	DSH	F	13	Crusts, erythema, erosion	Multiple	Axilla, feet
16	DSH	M	13	Papules, crusts	Multiple	Face, neck
17	DLH	F	9	Scaly papules	Multiple	Face
18	DSH	F	12	Erythematous papules	Multiple	Neck, face
19	DLH	M	11	Erythematous plaques	Multiple	Neck, face
20	Himalayan	F	8	Crusty plaque	Multiple	Dorsum, neck
21	DSH	F	15	Crusty plaque	Multiple	Leg, toe
22	DSH	F	11	Crusty plaques	Multiple	Face, lip
23	DLH	M	17	Plaques	Multiple	Dorsum
24	DSH	F	11	Crusty plaques	Multiple	Face, lip
25	DSH	M	16	Crusty plaques	Multiple	Face, neck, shoulders, foot pads
26	DSH	M	12	Crust papules	Multiple	Face, neck

DSH, domestic shorthair cat; DLH, domestic longhair cat.

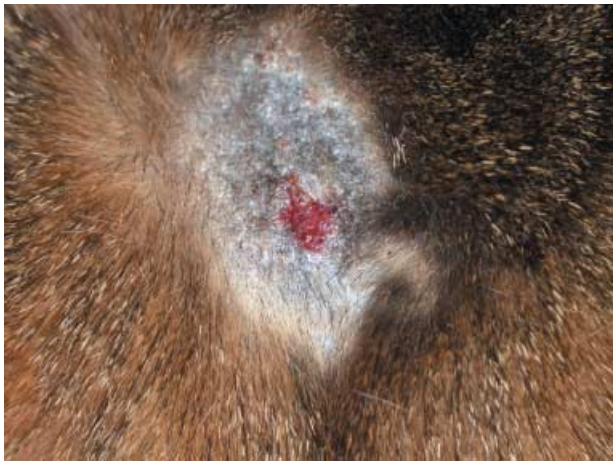

Figure 1. Cat no. 6. Pigmented plaque on the head diagnosed as feline viral plaque: Note the slightly raised and hyperpigmented lesion with a small central ulceration. Courtesy of Catherine Mège.

seemed to result from the condensation of fibrillar ones (more prevalent in the stratum spinosum) (Fig. 4). Intranuclear inclusions were not observed.

FVP + BISC

Interestingly, both BISC and FVP changes were present in 10 cats, sometimes in the same, sometimes in different, skin samples (Fig. 5a,b). Transition lesions exhibiting both FVP and BISC features were also sometimes observed.

BISC

The diagnosis of BISC was made on nine cases. These lesions consisted of sharply demarcated expansion of the epidermis with irregular acanthosis and broad rete ridges. Irregular acanthosis frequently descended around hair follicles. The epidermis was disorganized with a marked loss of cellular polarity and loss of normal stratification of the stratum basale and spinosum in all cases (wind-blown appearance). Keratinocytes with a hyperchromatic nucleus were present throughout the whole epidermis. Atypia was variable in nature and intensity (anisocytosis, anisocryosis and rare binucleated keratinocytes). Rare mitotic figures were present in all samples. Scattered apoptotic keratinocytes were present in four BISC samples. Koilocytes were present in all of them (Fig. 6). Other clear cells with rather enlarged vesicular nuclei were also observed. The cells (koilocytes and clear cells) contained sometimes intracytoplasmic additional blue-grey fibrillar pseudo-inclusions (three of nine cases). Clumped keratohyalin granules were seen in one of nine BISC cases. Erosions or ulcerations were present in five of nine cases.

Immunohistochemical examination

Results are summarized in Table 3. Of the seven cases of the FVP group, six were positive for PV antigen. Interestingly, all of the 10 samples with BISC and FVP lesion types were positive (Fig. 7). Only one of the nine BISC cases was deemed positive (11%). PV antigens were always visualized in the nucleus of the koilocytes; intracytoplasmic pseudo-inclusions remained unstained (Fig. 4).

Discussion

The clinical resemblance between BISC and FVP and the presence of both lesions in some cats suggest that some BISC evolve from FVP. Furthermore, despite the absence

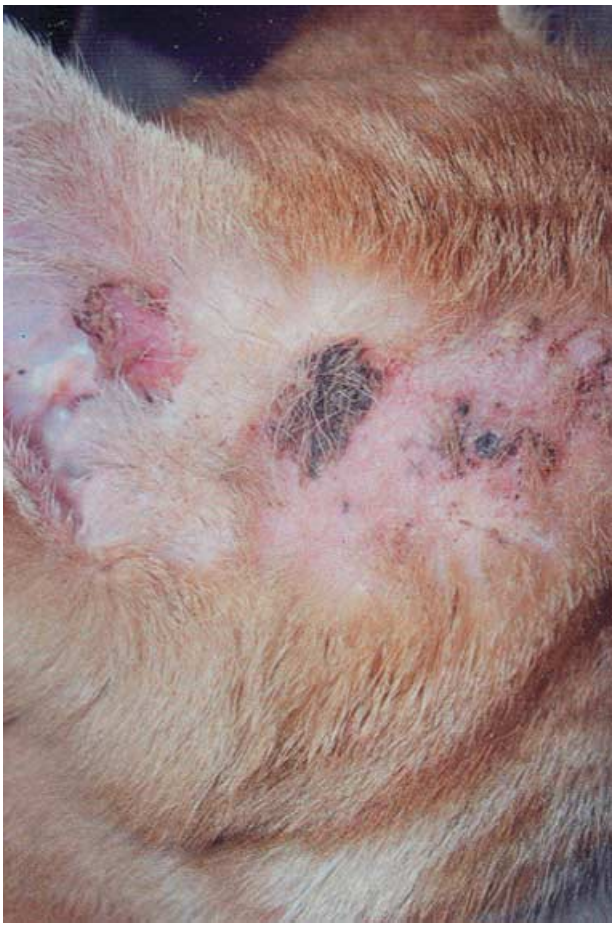

Figure 2. Cat no. 26. Pigmented plaques at the base of the ear and the pinna diagnosed as feline bowenoid *in situ* carcinoma. Note the slightly raised and ulcerated lesions partially covered by crusts.

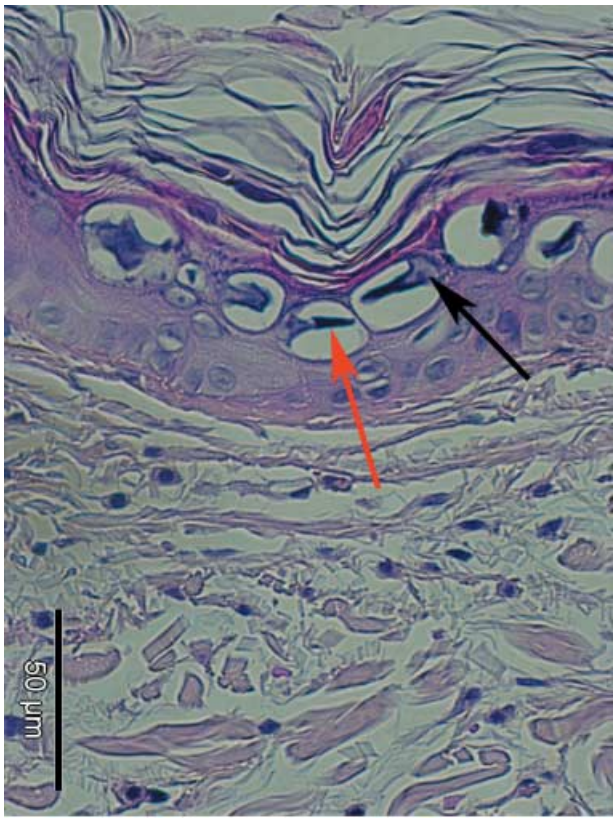

Figure 3. Cat no. 10. Histology of a feline viral plaque. Note the presence of clear cells (ballooned cytoplasm, rather swollen nucleus (black arrow) with intracytoplasmic pseudo-inclusions (red arrow). Haematoxylin and eosin. Magnification ×10. Bar = 200 µm.

Table 3. Histopathological findings

Case	Margins?	Hyperpig.	Koilocytes/clear cells	Dyskerat.	Comp. ps. incl.	Fibr. ps. incl.	KH Gran.	Diagnosis	PV-Ag
1	Yes	Yes	Yes	No	No	Yes	No	BISC	Neg
2	Yes	Yes	Yes	No	No	No	Yes	BISC + FVP	Pos
3	Yes	Yes	Yes	Yes	No	No	Yes	FVP	Pos
4	Yes	Yes	Yes	Yes	Yes	No	Yes	BISC + FVP	Pos
5	No	Yes	Yes	No	No	No	No	BISC	Neg
6	Yes	Yes	Yes	Yes	Yes	No	Yes	BISC + FVP	Pos
7	Yes	Yes	Yes	No	No	No	No	BISC	Neg
8	Yes	Yes	Yes	Yes	No	Yes	No	BISC	Neg
9	Yes	Yes	Yes	No	Yes	No	Yes	FVP	Pos
10	Yes	Yes	Yes	Yes	Yes	No	Yes	FVP	Neg
11	Yes	Yes	Yes	No	Yes	No	Yes	FVP	Pos
12	Yes	Yes	Yes	No	Yes	No	Yes	FVP	Pos
13	Yes	Yes	Yes	No	No	No	Yes	FVP	Pos
14	Yes	Yes	Yes	No	No	No	Yes	FVP	Pos
15	No	Yes	Yes	No	No	No	No	BISC	Neg
16	Yes	Yes	Yes	Yes	No	Yes	Yes	BISC + FVP	Pos
17	Yes	Yes	Yes	No	No	No	No	BISC	Neg
18	No	Yes	Yes	Yes	No	No	Yes	BISC	Pos
19	Yes	Yes	Yes	Yes	No	No	Yes	BISC + FVP	Pos
20	Yes	Yes	Yes	Yes	Yes	No	Yes	BISC + FVP	Pos
21	Yes	Yes	Yes	Yes	No	No	No	BISC	Neg
22	Yes	Yes	Yes	Yes	No	Yes	No	BISC	Neg
23	Yes	Yes	Yes	Yes	No	No	Yes	BISC + FVP	Pos
24	Yes	Yes	Yes	No	Yes	No	Yes	BISC + FVP	Pos
25	Yes	Yes	Yes	No	No	No	Yes	BISC + FVP	Pos
26	Yes	Yes	Yes	Yes	No	No	Yes	BISC + FVP	Pos

Margins?, presence of lesional margins; Hyperpig., hyperpigmentation; Dyskerat., dyskeratosis; Comp. ps. incl., compact pseudo-inclusions; Fibr. ps. inclus., fibrillar pseudo-inclusions; KH Gran., clumped keratohyalin granules; PV-Ag, papillomavirus antigen.
Pos, positive; Neg, negative.

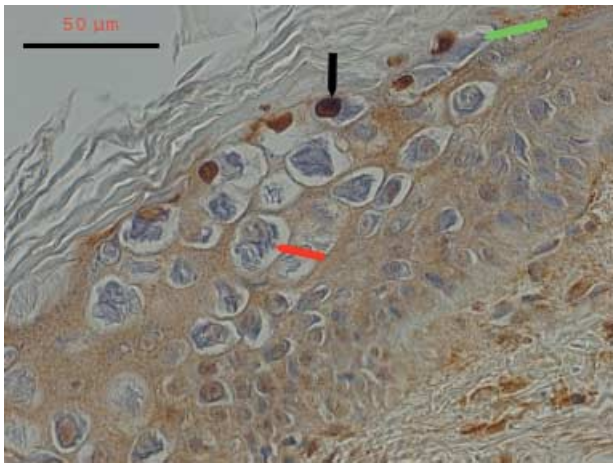

Figure 4. Cat no. 9. Immunohistochemical analysis of a feline viral plaque. Note the presence of positive nuclei (black arrow). The fibrillar (red arrow) and the solid (green arrow) intracytoplasmic inclusions remained unstained. Diaminobenzidine. Magnification ×40. Bar = 50 µm.

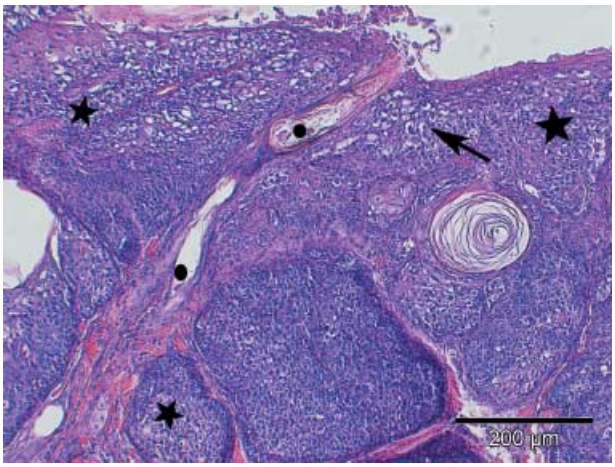

Figure 6. Cat no. 21. Histology of a feline bowenoid *in situ* carcinoma. Note the marked acanthosis (black stars: acanthotic epidermis), the follicular involvement (black points), the loss of polarity and the presence of numerous koilocytes (arrow). Haematoxylin and eosin. Magnification ×10. Bar = 200 µm.

of statistically significant difference, cats affected by FVP tended to be younger than those affected by BISC: this could imply that FVP are precursor lesions of BISC. However, while BISC affected the face, neck or the limbs in most cases, FVP lesions were more often present on the trunk even if other areas, including neck and face, were affected. This finding does not seem to support the hypothesis that BISC evolve from FVP but the discrepancy could be explained by a higher cancerization rate of lesions located on the face and neck, for example as a result of increased ultraviolet radiations exposure, compared to those in other regions of the body.

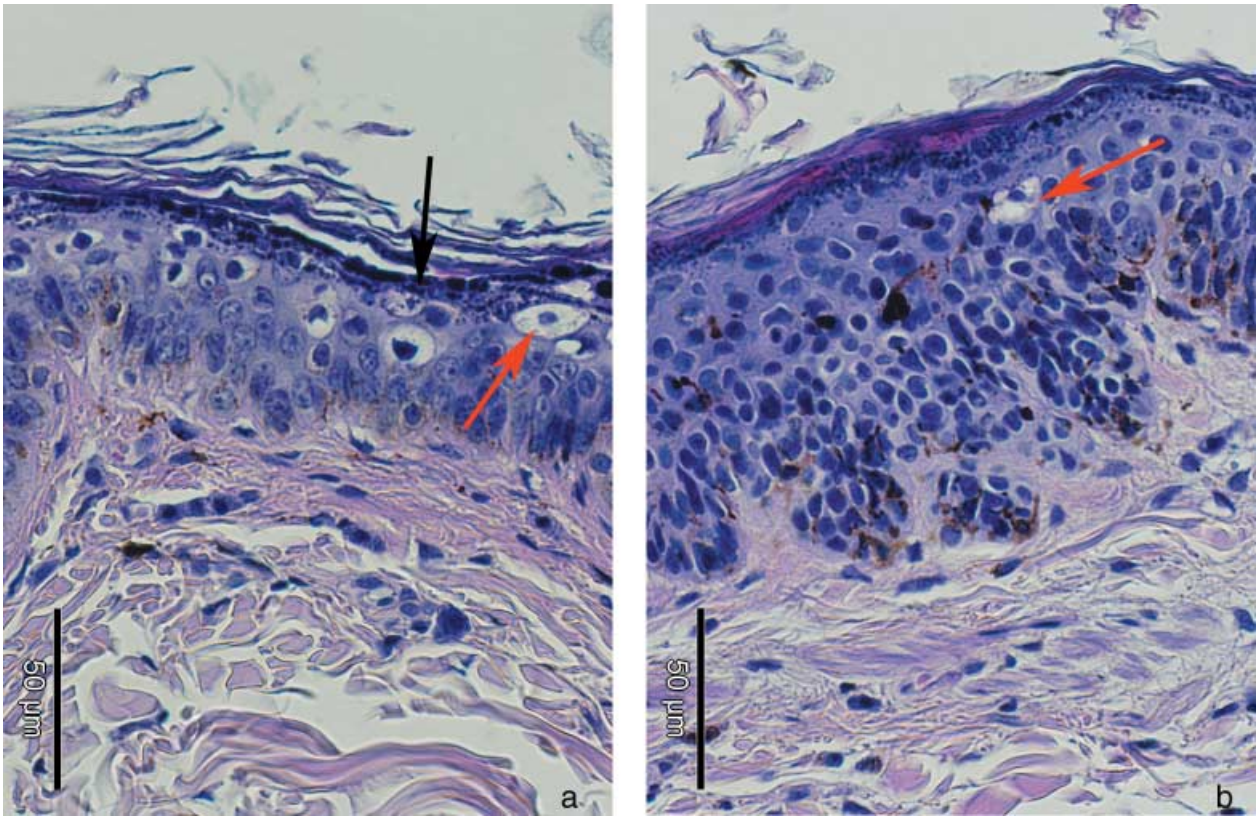

Figure 5. Cat no. 16. Histology of two lesions present on the same biopsy sample. Haematoxylin and eosin. Magnification ×40. Bar = 50 µm. (a) Feline viral plaque. Note the moderate acanthosis. The stratification and the differentiation of the epidermis are conserved. Koilocytes and clumped keratohyalin granules are the most obvious papillomaviruses' cytopathic characteristics on this lesion. (b). Early bowenoid *in situ* carcinoma. Note the acanthosis, the obvious disorganization of the epidermis and the abnormal differentiation of most keratinocytes. Clumped keratohyalin granules and one single koilocyte are the only papillomavirus cytopathic effects noticed on this lesion.

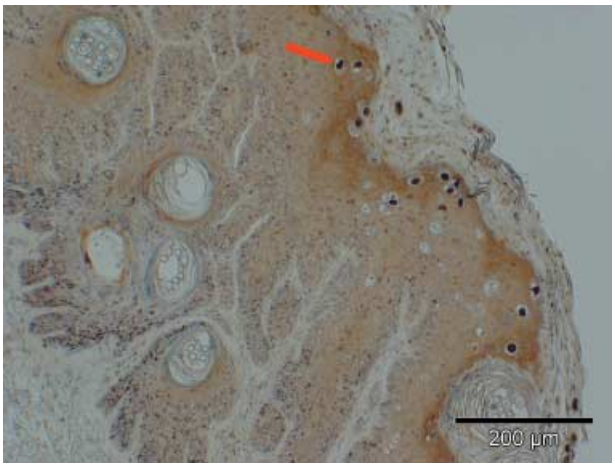

Figure 7. Cat no 19. Immunohistochemical analysis of a feline bowenoid *in situ* carcinoma. Note the presence of numerous koilocytes and clear cells (red arrow) with positive nuclei. Novared. Magnification ×10. Bar = 200 µm.

FVP usually conserved the general organization of the epidermis and atypia was absent, whereas BISC lesions were disorganized and abnormal keratinocytes were present throughout the epidermis. However, both conditions share numerous histological features: irregular acanthosis with rete ridges formations, presence of clumped keratohyalin granules, koilocytes and clear cells. The presence of koilocytes or clear cells in all BISC lesions (including IHC-negative ones) might be regarded as a proof of presence of the virus. These cells with vacuolated cytoplasm and shrunken, pycnotic nuclei are usually considered highly suggestive of PV infections.[8,13] All the authors who have studied feline BISC have recognized these cells, but two of three have not used the term 'koilocyte' to describe them.[8–10] *In situ* hybridization studies could be helpful to determine if these cells actually harbour PV nucleic acids and if the term 'koilocyte' is appropriate.

In both FVP and BISC samples, fibrillar and compact pseudo-inclusions were seen. In one case both were present in the same sample, and compact ones (more present in the stratum granulosum) seemed to result from the condensation of fibrillar ones (more prevalent in the stratum spinosum) (Fig. 4). This condensation has already been described by Carney and coworkers.[3]

Our study demonstrates that the association between FVP and BISC is frequent and occurs sometimes on the same skin lesion. Additionally, cases of overlapping BISC and FVP lesions have been detected. This association was already described before.[11,12] These similarities support the hypothesis that FVP could be precursory lesions of BISC.

All except one FVP and FVP + BISC cases were positive for PV antigen by immunohistochemistry (IHC). As pseudo-inclusions were present in the negative case, it can be considered that all these samples were infected by PV. Furthermore, as IHC detects capsid antigens, it can be concluded that productive infection occurred in all positive samples (all FVP lesions and positive BISC). These findings support the hypothesis that PVs play an active role in the development of such lesions. It must, however, be borne in mind that PVs are sometimes commensal, and nucleic acids are often uncovered in normal mammalian skin. However, genome copy number is usually very low and productive infection rarely occurs in such cases.[15,16] Establishing causality between the presence of viruses in skin lesions and oncogenesis remains problematic, and the presence of replicating viruses cannot be regarded as a sufficient proof. *In vitro* studies are mandatory to establish such causality.[17]

Almost all cats affected by BISC were deemed negative by IHC. These findings might suggest that BISC has two distinct causes and that only a subgroup of BISC is virally induced. A loss of viral replication during the cancerization process could also explain these findings. In fact latent PV infection or infection with minimal replication may remain undetected by IHC, because of the relatively low sensitivity of such techniques. The 'hit and run' model, which postulates an initial cellular transformation by the virus and a subsequent loss of viral genome, could account for the negative IHC in some BISC lesions.[18] Furthermore, it was recently demonstrated that PVs maintained productive infections in precursory lesions of cervical cancer but that capsid antigens were no longer produced in late cervical cancers.[19] In conclusion, a loss of viral protein expression in advanced cases of BISC seems likely.

Feline BISC has long been considered the counterpart of human Bowen's disease (BD) – an *in situ* squamous cell carcinoma that presents as solitary, well-circumscribed, erythematous plaques and occurs on the face, extremities and genitalia.[20,21] Koilocytes are usually not present in such lesions.[21] Human bowenoid papulosis is characterized by genital pigmented verrucous papules or plaques.[21] This condition is also histologically characterized by *in situ* SCC lesions but, in contrast to BD, bowenoid papulosis lacks full-thickness epidermal atypia. PV DNA is uncovered in virtually all samples of bowenoid papulosis but data concerning the presence of PV in human BD remain contradictory.[22–25] Furthermore, PVs that infect human bowenoid papulosis and BD are usually to mucosal and not to cutaneous strains.[23,24] These data show that feline BISC lesions display substantial differences from both human conditions and justify the use of a specific denomination, as emphasized by Gross and coworkers.[8]

The results of the present study support the hypothesis that some BISC evolve from FVP lesions and the causative role of PV. However, evidence that these PVs are able to induce cancerization in mammalian skin is lacking and further studies are warranted. Nucleic acids amplification techniques could establish which PVs are present in FVP and BISC lesions and whether BISC samples without FVP are really sterile or infected by dormant PV. As well, *in vitro* studies addressing the transforming potential of feline PV are required to better understand the role that these viruses play in this condition.

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Résumé Les plaques virales du chat (FVP) induites par les papillomavirus (PV) se présentent souvent comme des plaques hyperpigmentées. Le fait que jusqu'à 47% des carincomes in situ bowenoides (BISC), qui se présentent aussi sous la forme de plaques hyperpigmentées, sont positifs pour l'antigène de PV par immunohistochimie suggère que les BISC pourraient provenir de FVP. La relation entre la présence d'antigènes de PV et les données cliniques et histologiques de 26 cas de dermatoses félines cliniquement répertoriées comme des plaques hyperpigmentées avec un diagnostic histologique de FVP et/ou de BISC a été recherchée. Les cas ont été classés en trois groupes : FVP, FVP + BISC ou BISC. La recherche immunohistochimique de papillomavirus a été réalisée en utilisant un antisérum polyclonal de lapin anti-bovin. Sur les sept cas du groupe FVP, six étaient positifs à l'immunohistochimie, un seul des neuf BISC était positif. La présence de lésions de FVP et de BISC chez certains chats, et la fréquence importante de découverte d'antigènes de PV dans les groupes FVP et FVP + BISC suggère que ces deux maladies ont une même cause virale, et que certains BISC peuvent provenir de FVP. Le faible taux de détection d'antigène viral dans le groupe BISC indique une autre cause, ou la perte de la réplication virale pendant la cancérogénèse.

Resumen Las placas virales felinas (FVP) inducidas por el virus papiloma son a menudo verrugas hiperpigmentadas y planas. El hecho de que hasta un 47% de los carcinomas Bowenoides *in situ* (BISC), que también ocurren como placas hiperpigmentadas, son positivos al antígeno del virus papiloma mediante inmunohistoquímica sugiere que los BISC pueden evolucionar a partir de placas virales felinas. Se determinó la relación entre la presencia de antígenos del virus del papiloma y las características clínicas e histológicas de 26 casos de dermatosis (clínicamente descritas como placas pigmentadas y con diagnostico histológico de FVP y/o BISC). Los casos se clasificaron en uno de los tres grupos siguientes: FVP, FVP + BISC o BISC. La detección inmunohistológica de antígeno especifico del grupo del virus papiloma se realizó utilizando un antisuero policlonal de conejo frente al papiloma bovino. De los siete caso en el grupo FVP, seis fueron considerados positivos mediante inmunohistoquímica así como los diez gatos del grupo FVP + BISC. Por otro lado, solo uno de los nueve gatos con BISC fue positivo. La presencia de ambas lesiones FVP y BISC en algunos gatos y el elevado nivel de detección de antígenos del virus papiloma en los grupos FVP y FVP + BISC sugiere que ambas condiciones podrían tener la misma causa vírica y que algunos BISC podrían

progresar desde FVP. El bajo porcentaje de detección de antígeno vírico en el grupo BISC sugiere otra causa o una pérdida de replicación viral durante el proceso de carcinogénesis.

Zusammenfassung Feline virale Plaques (FVP), die von Papillomavirus (PV) verursacht werden, sind oft hyperpigmentierte und flache Warzen. Die Tatsache, dass bis zu 47% der 'Bowen'-ähnlichen *in situ* Karzinome (BISC), die normalerweise auch in Form von hyperpigmentierten Plaques erscheinen, mittels Immunchemie positiv sind für PV-Antigen, weist darauf hin, dass BISC sich aus FVP entwickeln könnte. Der Zusammenhang zwischen dem Auftreten von PV Antigenen und den klinischen und histologischen Erscheinungsbildern von 26 Fällen von felinen Dermatosen (die klinisch als pigmentierte Plaques beschrieben und histologisch als FVP und/oder BISC diagnostiziert wurden) wurde daher bestimmt. Die Fälle wurden in eine der drei folgenden Gruppen eingeteilt: FVP, FVP + BISC oder BISC. Die immunhistologische Bestimmung des gruppenspezifischen Papillomavirus Antigens wurde mit einem polyklonalen Kaninchen Antiserum gegen bovines Papillomavirus durchgeführt. Von den sieben Fällen in der FVP Gruppe wurden sechs mittels Immunhistologie als positiv angesehen, genauso wie alle 10 Katzen in der FVP + BISC Gruppe. Andererseits war nur eine der neun BISC Katzen positiv. Das Vorhandensein von beiden, FVP und BISC Läsionen bei manchen Katzen und das häufige Auftreten von PV-Antigenen in den FVP und FVP + BISC Gruppen ist ein Hinweis darauf, dass beide Formen dieselbe virale Ursache haben und einige BISC sich aus den FVP entwickeln könnten. Das seltene Auftreten von viralem Antigen in der BISC Gruppe bedeutet, dass eine andere Ursache vorliegt oder der Verlust von viraler Replikation während der Kanzerogenese besteht.

