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ORIGINAL ARTICLE

The significance of crystalline/chrysalis structures in the diagnosis of melanocytic and nonmelanocytic lesions

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Background: Crystalline/chrysalis structures (CS) are white shiny streaks that can only be seen with polarized dermatoscopy.

Objectives: We sought to estimate the prevalence and assess the clinical significance of CS in melanocytic and nonmelanocytic lesions.

Methods: This was a prospective observational study in which dermatoscopic assessment of lesions was recorded in consecutive patients examined during a 6-month period. In addition, a data set of biopsy-proven melanomas was retrospectively analyzed.

Results: In all, 11,225 lesions in 881 patients were prospectively examined. Retrospectively, 229 melanomas imaged with polarized dermatoscopy were analyzed. In the prospective data set, a median of 12.7 lesions (range, 1-54) were evaluated per patient. None of clinically diagnosed Clark nevi (n = 9750, 86.8%) demonstrated CS. Overall, CS were observed in 206 (1.8%) lesions, most commonly dermatofibromas and scars among nonbiopsied lesions. A total of 265 (2.4%) lesions were biopsied, including 20 melanomas and 36 nevi. Among biopsied malignant lesions, CS were most commonly observed in basal cell carcinoma (47.6%) and invasive melanomas (84.6%). Melanomas were more likely to have CS than biopsied nevi (odds ratio = 9.7, 95% confidence interval 2.7-34.1). In the retrospective data set, CS were more commonly observed among invasive melanomas (41%) compared with in situ melanomas (17%) (odds ratio = 3.4, 95% confidence interval 1.9-6.3, $P < .001$). The prevalence of CS correlated with increased melanoma thickness ($P = .001$).

Limitations: Biopsied lesions represent a small percentage of the total number of lesions evaluated.

Conclusion: Among biopsied malignant lesions, CS are most commonly observed in basal cell carcinoma and invasive melanomas and rarely seen in nevi. In melanoma, CS may reflect increased tumor thickness and progression. (J Am Acad Dermatol 10.1016/j.jaad.2011.04.039.)

Key words: basal cell carcinoma; crystalline/chrysalis structures; dermatofibroma; invasive melanoma; matrix remodeling; polarized dermatoscopy.

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Identification of melanoma-specific dermatoscopic structures has improved the ability of clinicians to recognize melanoma.[1-4] In recent years, dermatoscopes that use cross-polarization technology have been introduced and are increasingly used.[5-7] However, there are clinically relevant and notable differences between lesion appearance under polarized dermatoscopy (PD) and non-PD.[8,9] In particular, PD has allowed identification of new dermatoscopic structures not previously seen with non-PD.[10] Shiny, bright white, orthogonally oriented linear streaks, detected exclusively with PD, represent one example of such structures (Fig 1).[8,11,12] These bright white lines are also metaphorically known as crystalline or chrysalis structures (CS). The CS are observed in skin lesions with increased amount of dermal collagen, and are visible as a result of the interaction of polarized light with the birefringent properties of collagen.[12] The CS are frequently visualized in dermatofibromas (DFs) and scars but can also be seen in basal cell carcinomas (BCC), Spitz nevi, and melanomas.[8,12] The prevalence of CS among melanocytic neoplasms and among various nonmelanocytic lesions is currently unknown. More importantly, the clinical significance of this novel dermatoscopic structure has not been established.

CAPSULE SUMMARY

- Crystalline/chrysalis structures have been observed under polarized dermatoscopy in a variety of lesions including melanomas, basal cell carcinomas, dermatofibromas, and Spitz nevi.
- Our study demonstrates the prevalence of crystalline/chrysalis structures in a wide spectrum of melanocytic and nonmelanocytic lesions.
- Crystalline/chrysalis structures may serve as a diagnostically useful dermatoscopic feature. In the diagnosis of melanomas they may serve as a clue to differentiate them from nevi and identify a subset of more advanced lesions.

METHODS

The aim of this observational study was to estimate the prevalence of CS seen with PD. The study consisted of two parts. In the initial prospective analysis, the lesions of 881 consecutive patients from the practices of two dermatologists (A. A. M. and R. P. B.) were evaluated. The two physicians specialize in pigmented lesions and are experts in dermatoscopy. All lesions assessed dermatoscopically as part of the routine total body skin examination of patients who were screened for skin cancer were included in this study. Dermatoscopic evaluations were performed during the clinical encounter using a DL-3 dermatoscope (3Gen, LLC, Dana Point, CA) and were recorded on a designated study sheet. For each lesion, the clinical diagnosis and the presence or absence of CS on dermatoscopic evaluation were recorded. For lesions that were biopsied, histopathologic diagnosis was collected. In the retrospective portion of the study, contact PD (DermLite, 3Gen, LLC) images of histopathology-proven melanomas, consecutively recorded between 2004 and 2009, were evaluated for the presence of CS. All photographs were taken with a digital camera (Nikon Coolpix 4500, Nikon USA Inc, Melville, NY). Each lesion was analyzed for the presence of the following

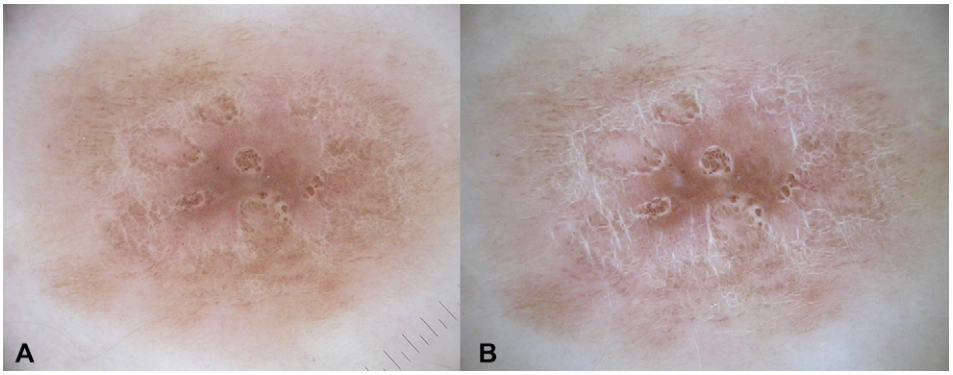

Fig 1. **A**, Contact nonpolarized image of invasive melanoma displaying negative network and irregular globules. **B**, Polarized contact dermatoscopic image demonstrating conspicuous crystalline/chrysalis structures (CS) throughout lesion. Note: This lesion has both negative network and CS. These two structures are not the same. Negative network can be seen with either polarized or nonpolarized dermatoscopy and crystalline/chrysalis can only be seen with polarized dermatoscopy. Whereas some melanomas may manifest both structures, others will manifest one or the other.

Abbreviations used:

BCC: basal cell carcinoma
bFGF: basic fibroblast growth factor
CI: confidence interval
CS: crystalline/chrysalis structures
DFs: dermatofibromas
OR: odds ratio
PD: polarized dermatoscopy
SK: seborrheic keratoses

vascular structures: linear-irregular, dotted, comma, hairpin, coiled vessels, and pink veil, with the goal of investigating their potential association with CS. The presence of two or more vascular structures, excluding the pink veil, constituted a polymorphous vascular pattern. Similarly, melanoma thickness and the presence of histologic regression were extracted from the pathology reports to investigate their relationship with CS. Because CS are frequently observed in biopsy scars, melanomas that have been previously biopsied were specifically excluded from the retrospective analysis to avoid a misclassification bias.

Statistical analysis

The goal of the analysis was to determine the prevalence of CS in lesions of patients undergoing cutaneous skin cancer surveillance. Study lesions were classified by their clinical diagnosis if not biopsied, and by the histopathologic diagnosis if biopsied. The distributional characteristics of all study variables were examined graphically. Descriptive frequencies, means, and medians were used to describe the study variables. For both prospective and retrospective aspects of the study, the main outcome was the presence or absence of CS. To evaluate the association of CS with other study variables, χ^2 statistics and logistic regression models were used. All analyses were performed with Stata SE v.10.1, Stata Corp, College Station, TX.

Results

In the prospective part of the study, a total of 11,225 lesions were dermatoscopically evaluated in 881 patients. The median number of lesions evaluated per patient was 12.7 (range 1-54). The clinical diagnoses for nonbiopsied lesions and the histopathologic diagnoses for biopsied lesions are presented (Tables I and II). The majority (86.8%, n = 9750) of lesions that were not biopsied were clinically diagnosed as Clark nevi followed by seborrheic keratoses (SK) and solar lentigines (5.8%). Of the 11,225 lesions, 265 (2.4%) were biopsied (Table II). Nonmelanocytic lesions comprised the majority (n = 209, 78.8%) of biopsied lesions, BCC being the most common diagnosis (n = 82, 31%). A total of 56 (21.1%) of the 265 biopsied lesions were melanocytic neoplasms, 20 of which were melanomas (13 invasive melanoma, 3 in situ melanomas, and 4 cutaneous melanoma metastases).

Overall, CS were observed in 206 of the 11,225 study lesions (1.8%). Notably, none of the nonbiopsied Clark nevi and only 3 (0.5%) of SK and solar lentigines showed CS (Table I). CS were identified in a much higher proportion of biopsied lesions than nonbiopsied lesions (33.2% vs 1.1%, $P < .0001$). The distribution of CS by histopathologic diagnosis is presented (Table II). Of note, all of the biopsied SK were clinically inflamed SK. Overall, CS were 2.5 times more likely to be observed in malignant compared with benign lesions (odds ratio [OR] = 2.5, 95% confidence interval [CI] 1.5-4.3). CS were commonly observed among melanomas (n = 14, 70%); in fact, melanomas were more likely to harbor CS than biopsied nevi (OR = 9.7, 95% CI 2.7-34.1) (Fig 2 and Table III). If the nonbiopsied and biopsied primary melanocytic neoplasms are analyzed together, excluding melanoma metastases, these differences become even more striking; whereas CS were observed in 11 of 16 primary melanomas (68.8%), CS were observed in only 7 of 10,059 nevi examined (0.07%), 3 of the 7 nevi showing CS being Spitz nevi. Notably, in the prospective data set, CS were only observed among invasive melanomas and cutaneous metastases, and were absent among in situ melanomas. In addition, CS were more likely to be observed in BCC than all other biopsied nonmelanocytic lesions (OR = 3.5, 95% CI 1.8-6.7).

In the retrospective part of the study, contact PD images from a total of 229 melanomas were analyzed. The data set comprised 119 in situ and 110 invasive melanomas. CS were observed among 45 invasive melanomas (41%) and among 20 in situ melanomas (17%) (Fig 3). CS were 3.4 times more likely to be visualized among invasive compared with in situ melanomas (Table IV). Melanomas showing CS on dermatoscopy tended to be thicker on histopathology than those without CS (Table V). Controlling for the presence of histologic regression, invasive melanomas were significantly more likely to have CS (OR = 3.6, 95% CI 1.9-7.0, $P < .001$) compared with in situ melanomas. In contrast, no significant association was observed between CS and the presence of regression (OR = 1.3, 95% CI 0.7-2.4, $P = .49$). Polymorphous vessels were observed more frequently among invasive melanomas compared with in situ melanomas ($P < .001$) (Fig 4). Interestingly, CS were observed more frequently

Table I. Prevalence of crystalline/chrysalis structures for melanocytic and nonmelanocytic lesions that were not biopsied

Nonmelanocytic lesions				Melanocytic lesions			
	Crystalline/chrysalis				Crystalline/chrysalis		
	No	Yes			No	Yes	
DF	31 (24.8)	94 (75.2)	125 (100)	MM Met	8 (100)	0 (0)	8 (100)
SK/solar lentigines	648 (99.5)	3 (0.5)	651 (100)	Clark/DN	9750 (100)	0 (0)	9750 (100)
Hemangioma	48 (96)	2 (4)	50 (100)	CMN	90 (100)	0 (0)	90 (100)
SCC/Bowen/KA	2 (100)	0 (0)	2 (100)	Blue nevus	15 (100)	0 (0)	15 (100)
LPLK	6 (100)	0 (0)	6 (100)	IDN	168 (100)	0 (0)	168 (100)
AK	57 (98.3)	1 (1.7)	58 (100)	Total	10031 (100)	0 (0)	10031 (100)
Scar	2 (9.5)	19 (90.5)	21 (100)				
Other—nonmelanocytic	15 (93.8)	1 (6.3)	16 (100)				
Total	809 (87.1)	120 (12.9)	929 (100)				

AK, Actinic keratosis; *CMN*, congenital melanocytic nevus; *DF*, dermatofibroma; *DN*, dysplastic nevus; *IDN*, intradermal nevus; *KA*, keratoacanthoma; *LPLK*, lichen planuslike keratosis; *MM Met*, malignant melanoma cutaneous metastasis; *SCC*, squamous cell carcinoma; *SK*, seborrheic keratosis.

Table II. Prevalence of crystalline/chrysalis structures by histologic diagnosis for nonmelanocytic and melanocytic lesions

Nonmelanocytic				Melanocytic			
	Crystalline/chrysalis				Crystalline/chrysalis		
	No	Yes			No	Yes	
BCC*	43 (52.4)	39 (47.6)	82 (100)	Invasive melanoma	2 (15.4)	11 (84.6)	13 (100)
SK/solar lentigines	27 (84.4)	5 (15.6)	32 (100)	MIS	3 (100.0)	0 (0.0)	3 (100)
Hemangioma	1 (100.0)	0 (0.0)	1 (100)	MM Met	1 (25.0)	3 (75.0)	4 (100)
SCC/Bowen/KA	35 (87.5)	5 (12.5)	40 (100)	Spitz	0 (0.0)	3 (100.0)	3 (100)
LPLK	7 (46.7)	8 (53.3)	15 (100)	Clark/DN	19 (95.0)	1 (5.0)	20 (100)
AK	12 (70.6)	5 (29.4)	17 (100)	CMN	1 (100.0)	0 (0.0)	1 (100)
Scar	0 (0.0)	2 (100.0)	2 (100)	IDN	8 (80.0)	2 (20.0)	10 (100)
Other—nonmelanocytic	19 (95.0)	1 (5.0)†	20 (100)	Other—melanocytic	1 (50.0)	1 (50.0)‡	2 (100)
Total	144 (68.9)	65 (31.1)	209 (100)	Total	35 (62.5)	21 (37.5)	56 (100)

AK, Actinic keratosis; *BCC*, basal cell carcinoma; *CMN*, congenital melanocytic nevus; *DN*, dysplastic nevus; *IDN*, intradermal nevus; *KA*, keratoacanthoma; *LPLK*, lichen planuslike keratosis; *MIS*, melanoma in situ; *MM Met*, malignant melanoma cutaneous metastasis; *SCC*, squamous cell carcinoma; *SK*, seborrheic keratosis.

*5 BCCs were treated with Mohs and histologically confirmed to be BCCs without initial biopsy performed.

†Lesion observed with crystalline/chrysalis structures in this category was normal scalp from bald individual.

‡Lesion observed with crystalline/chrysalis structures in this category was traumatized nevus.

(63%) among melanomas with polymorphous vessels on dermatoscopy, and infrequently (12%) among melanomas without visible dermatoscopically identified vascular structures; thus, melanomas with polymorphous vascular pattern were 12.5 times more likely to display CS on dermatoscopy compared with melanomas lacking discernible vasculature (Table VI).

DISCUSSION

The use of PD has improved visualization of dermal structures such as collagen and microvasculature.[13] The different optical properties of PD have allowed identification of new dermatoscopic structures such as shiny, bright white, orthogonally oriented linear streaks, which have been termed "crystalline or chrysalis structures."[12] These structures were previously described in a variety of lesions including melanomas,[9,12] Spitz nevi,[12] BCC,[9] DFs,[8,14] scars,[12] and porokeratoses. They can also be frequently visualized on the extensively sun-damaged skin of the bald scalp. It has been postulated that CS correlate with dermal fibroplasias; the birefringent properties of the collagen bundles result in rapid randomization and increased backscatter of polarized light, which makes collagen apparent under PD.[13] The fact that CS are very commonly observed in biopsy scars and DFs, whose histopathologic

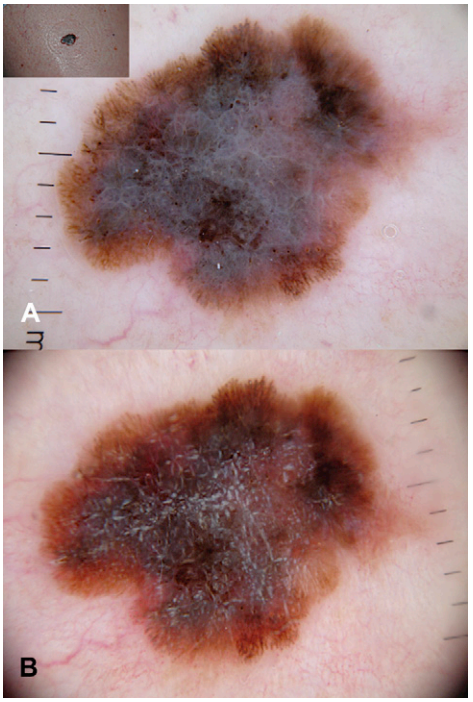

Fig 2. A, Contact nonpolarized image of heavily melanized invasive melanoma (0.85-mm thickness) displaying negative network, atypical pigment network, peripheral streaks, and irregular dots and globules. **B**, Corresponding polarized contact dermatoscopic image demonstrating crystalline/chrysalis structures. **Inset**, Clinical image.

hallmark is dermal fibrosis,[14,15] supports this notion. We have demonstrated that CS are encountered in a spectrum of melanocytic and nonmelanocytic lesions. Probably, all lesions showing CS harbor increased (scarlike) or altered collagen in the dermis.

The clinical significance of CS in melanomas has not yet been established. Herein, we found that the presence of CS among melanocytic neoplasms is highly suggestive of melanoma diagnosis. In our prospective analysis, among all primary melanocytic neoplasms, CS were observed in over two thirds of melanomas, while seen in merely 7 nevi among over 10,000 nevi examined, most frequently in Spitz nevi. Interestingly, CS were seen only among biopsied nevi, suggesting that CS are almost only seen among nevi that are clinically and dermatoscopically equivocal. Thus, CS may serve as an additional feature that can help distinguish melanomas from nevi; the dermatoscopic finding of CS when evaluating a melanocytic neoplasm should warrant extreme caution for the diagnosis of melanoma.

In the prospective data set, CS were exclusively observed in invasive melanomas and in cutaneous melanoma metastases and were not identified for in situ melanomas. Similarly, in the retrospective data set of 229 melanomas, CS were 3.4 times more commonly encountered among invasive (41%) than among in situ (17%) melanomas. Melanomas showing CS were also thicker than those without CS. Although we observed a significant association between histologic regression changes and CS, our analyses also demonstrated that the association between CS and lesion thickness was independent of the presence of regression. Taken together, these data suggest that CS is a dermatoscopic finding that in melanoma signifies a higher likelihood of dermal invasion and thicker tumors. Indeed, these findings are in line with those of a recent study that demonstrated a statistically significant correlation between the presence of CS and increased depth of invasion.[16]

Superficial spreading melanoma begins as in situ melanoma and progresses by dermal invasion with subsequent metastases. The functional interaction of melanoma cells with surrounding neoplastic cells, tumoral stoma, inflammatory milieu, and growth factors is critical, and fibroblasts serve as the major source of extracellular matrix and contribute to melanoma progression.[17] Basic fibroblast growth factor (bFGF) appears to play a critical role in the ability of melanoma cells to invade and proliferate in the papillary dermis.[18] In primary invasive melanomas, the greatest expression of bFGF is along the advancing front of the neoplasm, adjacent to the fibrotic changes in the dermis, and it is usually not detectable in definitive in situ lesions.[19] Melanocyte-fibroblast interactions and tumor-induced collagen turn-over appear to play a critical role in melanoma evolution through both radial and vertical growth phases.[20] Melanocytes in the radial and early vertical growth phases have been shown to induce de novo type I collagen synthesis by fibroblasts juxtaposed to microinvasive nests in the papillary dermis; staining with Sirius red can identify tightly woven collagen bundles encircling these melanoma nests at the dermoepidermal junction.[21] In contrast, this has not been demonstrated in dysplastic nevi.[21] Evidence of stromal remodeling can also be identified with reflectance confocal microscopy by visualizing bundles of collagen fibers that histopathologically correspond to fibroplasia and to regression structures on dermatoscopy.[22,23] Type I collagen with extracellular matrix glycoproteins, such as tenascin C and fibronectin, have been shown to facilitate dermal invasion during the more advanced vertical growth phase by forming tubular channels that ensheath and isolate invading melanoma cells.[24] Based on the available preclinical data, we hypothesize that CS may reflect de novo synthesis or remodeling of type I collagen in the papillary dermis, signifying changes

Table III. Association between lesion diagnosis and crystalline/chrysalis structures for histologically diagnosed melanomas and basal cell carcinomas

	Crystalline/chrysalis structures			Crystalline/chrysalis structures	
	Yes	No		Yes	No
Melanoma	14 (70.0)	6 (30.0)	Basal cell carcinoma	39 (60.0)	43 (30.1)
All other biopsied melanocytic lesions	7 (19.4)	29 (80.6)	All other biopsied nonmelanocytic lesions	26 (40.0)	100 (69.9)
Total	21 (100)	35 (100)	Total	65 (100)	143 (100)
OR = 9.7; 95% CI 2.7-34.1			OR = 3.5; 95% CI 1.8-6.7		

CI, Confidence interval; *OR*, odds ratio.

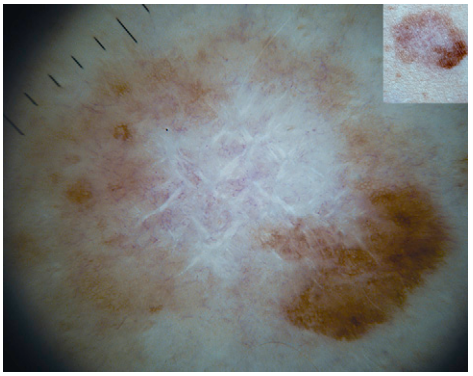

Fig 3. Polarized contact dermatoscopic image of hypomelanotic melanoma in situ with crystalline/chrysalis structures. **Inset**, Clinical image.

Table IV. Association between crystalline/chrysalis structures for in situ and invasive melanoma among retrospectively analyzed lesions

		PCD		
		MIS	Invasive	
Crystalline/chrysalis	Absent	99 (83.2)	65 (59.1)	164 (71.6)
	Present	20 (16.8)	45 (40.9)	65 (28.4)
	Total	119 (100)	110 (100)	229 (100)
		OR = 3.4 (CI 1.9-6.3), $P < .001$		

MIS, Melanoma in situ, *OR*, odds ratio; *PCD*, polarized contact dermatoscopy.

Table V. Differences in tumor thickness and presence of regression by presence or absence of crystalline/chrysalis structures for polarized contact dermatoscopy among retrospectively analyzed lesions

	PCD		
	Crystalline/chrysalis		
	Present	Absent	*P* value
Thickness, mean, (SD)*	0.68 (0.50)	0.43 (0.28)	.001
Thickness, median†	0.55	0.39	.001
Regression	n (%)	n (%)	
Absent	38 (53.5)	121 (67.2)	.04
Present	33 (46.5)	59 (32.8)	

PCD, Polarized contact dermatoscopy.
*Based on Student *t* test.
†Based on Wilcoxon sign rank test.

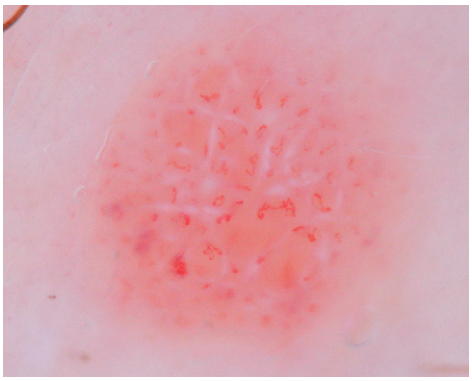

Fig 4. Polarized contact dermatoscopic image of amelanotic melanoma with crystalline/chrysalis structures and polymorphous vessels.

in the extracellular matrix induced by tumor progression and dermal invasion.[23]

Our retrospective analysis revealed that the presence of CS in melanomas was frequently associated with polymorphous vascular pattern (OR = 12.5) (Fig 5). Vascular structures were also significantly increased in invasive as compared with in situ melanomas. These findings support the preclinical evidence of intricate relationship between tumor-induced stromal response and neoangiogenesis. Early melanoma development is characterized by an inflammatory cell infiltrate including mast cells that secrete matrix metalloproteinases, which release matrix-sequestered vascular endothelial growth factor, thereby enhancing angiogenesis.[25] Subsequent influx of macrophages augments angiogenesis via the release of proangiogenic factors such as interleukin-8, vascular endothelial growth factor, and bFGF.[25] bFGF expression is positively correlated with increased intratumoral microvascular density.[26] In early melanomas, incompletely formed capillaries

Table VI. Association between crystalline/chrysalis structures and presence of vessels for lesions imaged with polarized contact dermatoscopy among retrospectively analyzed lesions

		Vascular structures			
		None	1	Polymorphous pattern	Total
Crystalline/chrysalis	Absent	117 (88.0)	26 (66.7)	21 (36.8)	164 (71.6)
	Present	16 (12.0)	13 (33.3)	36 (63.2)	65 (28.4)
	Total	133 (100)	39 (100)	57 (100)	229 (100)
		Referent	3.6 (1.6-8.5), $P = .003$	12.5 (5.9-26.5), $P < .001$	

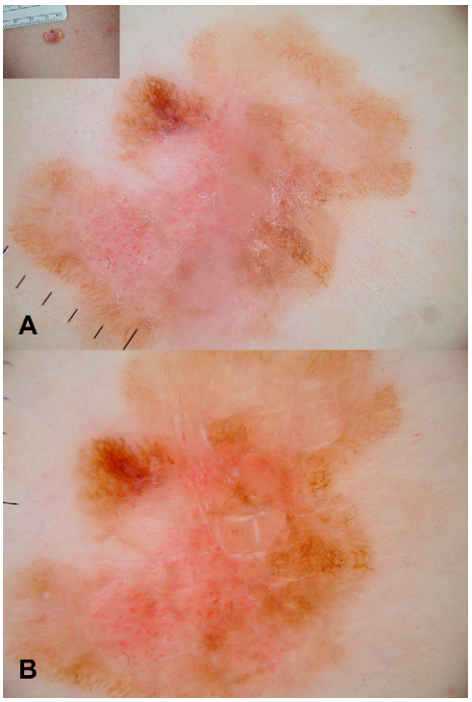

Fig 5. A, Contact nonpolarized image of invasive melanoma without negative network that displays atypical pigment network and polymorphous vascular pattern. **B**, Corresponding polarized contact dermatoscopic image of invasive melanoma (0.83-mm thickness) with crystalline/chrysalis structures, pink veil, and polymorphous vasculature. **Inset**, Clinical image.

can be visualized, and vascular density correlates with increased depth of invasion.[27,28] Both degradation and synthesis of new collagen fibrils are necessary for the development of the vascular network.[20] De novo type I collagen synthesis has been shown to serve as an angiogenic trigger in early melanoma,[21] with vascular network forming in the papillary dermis, where high levels of collagen expression are found.[21]

CS have been previously identified in BCC[12] but their prevalence and clinical significance are not known. In our study, CS were observed in approximately 48% of BCCs. Thus, CS may serve as an additional dermatoscopic feature in their diagnosis. A fibroplastic stroma is typical of BCC, particularly the morpheaform and infiltrative types,[29] but further studies may demonstrate different rates of CS across the various BCC histopathologic subtypes.

CS are very frequently observed in DFs.[8,10] In our study, they were seen in 75% of DFs and likely reflect the pronounced dermal fibrosis.[30] Increased prevalence of CS among biopsy-proven lichen planuslike keratoses (53.3%) was also noted, in contrast to their low prevalence among clinically diagnosed SK/solar lentigines (0.5%). As lichen planuslike keratosis represent SK/solar lentigines that have undergone regression,[31,32] the dermatoscopically observed CS probably reflect the papillary dermal fibrosis.

The main limitation of our study is that only a small percentage of lesions evaluated were biopsied. However, with the exception of cutaneous melanoma metastases, SCC/Bowen/keratoacanthoma, and actinic keratoses, the nonbiopsied lesions were deemed to be completely benign in appearance (eg, SK) and a confirmatory biopsy specimen was not necessary. Secondly, CS incidence was analyzed among nonbiopsied lesions whereby diagnosis was based solely on clinical and dermatoscopic assessments. This was done to avoid a selection bias intrinsic to studies that include only biopsied lesions. Of note, many of the lesions with CS were biopsied (46 times more frequently than lesions without CS), allowing histopathologic confirmation of the diagnosis. Our retrospective analysis also included some PD photographs taken with a flash, which may partially obscure the appearance of CS and potentially underestimate their incidence among melanomas.

In conclusion, we have systematically demonstrated the incidence of CS among melanocytic and nonmelanocytic lesions. CS may serve as a diagnostically useful dermatoscopic feature. They may be particularly important in the diagnosis of melanomas and serve as a clue to differentiate them from nevi and identify a subset of more advanced lesions. Like any other dermatoscopic clue, CS should be interpreted in the context of other dermatoscopic structures and the overall pattern. The precise

histopathologic correlates of CS need to be further studied. Nevertheless, our study reinforces the idea that dermatoscopy can be used as a powerful research tool that provides further insights into our understanding of malignant and benign neoplasms. Because morphologic features reflect underlying biological changes that can be correlated with specific molecular and cellular-level findings, dermatoscopy can serve as an ideal instrument to facilitate transitional research between the clinic and basic science investigations.

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