possible culprit and was stopped. She was afebrile and was not on antibiotics or G-CSF at the time. There was no improvement in the rash and a skin biopsy was performed. The skin biopsy revealed marked oedema of the upper dermis associated with a mild superficial perivascular chronic inflammatory infiltrate. The striking feature was the presence of a heavy infiltrate of neutrophils within the eccrine sweat glands in the deep dermis with focal gland destruction (Fig. 1b), consistent with the diagnosis of NEH. Stains for infectious agents were negative and no abnormal leukaemic infiltrates were seen. She was treated symptomatically with analgesia. The rash eventually resolved 10 days later upon recovery of her bone marrow function. The patient underwent four subsequent courses of consolidation treatment with high-dose cytarabine without recurrence of her rash.

NEH is a rare and benign dermatosis which is self-limiting and usually resolves after 1–3 weeks. Recurrence with subsequent therapy can occur. The pathogenesis is uncertain but may be the direct consequence of the cytotoxicity of drugs secreted in the sweat on the eccrine gland, leading to tissue necrosis. This phenomenon may then induce chemotaxis of neutrophils. The important differential diagnosis clinically is Sweet's syndrome, leukaemia cutis and cutaneous infection. In this case, skin biopsy findings excluded the latter two and the lack of fever and presence of severe neutropenia during the development of the rash makes Sweet's syndrome unlikely.

Although NEH developed in the classical setting in our patient, several features make this case unusual. NEH tends to occur in the setting of fever and is usually asymmetrical.² Our patient was afebrile and had a striking symmetrical rash around both breasts without involvement of other areas of the skin. In a review of 51 published cases of NEH, the anterior chest was affected in nine cases (16%). Five of the patients had AML, two had Hodgkin's disease, one had nephroblastoma and one had HIV infection. Five patients were men and four were women. The anterior chest is usually affected in the setting of more extensive involvement including other areas such as the trunk, limbs and face.² Only one other case solely involving the breast was reported, in a 45-year-old woman with AML who presented with papules on her breast when severely neutropenic.⁶ In our patient, the rash was erythematous and raised in a symmetrical, circumferential distribution around both breasts with sparing of the nipple and the periareolar region. The localization of the rash was postulated to be due to differences in the metabolic secretory activity of the eccrine glands and additional local trauma,² although this was not apparent in our patient. Although NEH is frequently asymptomatic, pain and discomfort can be a presenting symptom and was a striking feature in our patient. Furthermore, NEH is most often reported in caucasians. Only two Japanese patients⁷ and one Korean patient⁸ have been reported. Ours is the first Chinese patient reported. This case further highlights the polymorphic presentation of NEH. We suggest that NEH should be considered as a differential diagnosis in rashes that develop following chemotherapy for AML. Early recognition is

important to avoid unnecessary drug therapy or drug cessation. A skin biopsy is usually diagnostic.

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References

- 1 Harriot TJ, Fine JD, Berman RS *et al.* Neutrophilic eccrine hidradenitis. A distinctive type of neutrophilic dermatosis associated with myelogenous leukaemia and chemotherapy. *Arch Dermatol* 1982; **118**: 263–6.
- 2 Bachmeyer C, Aractingi S. Neutrophilic eccrine hidradenitis. *Clin Dermatol* 2000; **18**: 319–30.
- 3 Nijsten TEC, Muelemans L, Lambert T. Chronic pruritic neutrophilic eccrine hidradenitis in a patient with Behçet's disease. *Br J Dermatol* 2002; **147**: 797–800.
- 4 Pierson JC, Helm TN, Taylor JS *et al.* Neutrophilic eccrine hidradenitis heralding the onset of acute myelogenous leukaemia. *Arch Dermatol* 1993; **129**: 791–2.
- 5 Gomez Vasquez M, Peteiro C, Toribio J. Neutrophilic eccrine hidradenitis heralding the onset of chronic myelogenous leukaemia. *J Eur Acad Dermatol Venereol* 2003; **17**: 328–30.
- 6 D'Hondt L, Doyen C, Chatelain C *et al.* Neutrophilic eccrine hidradenitis. *Acta Clin Belg* 1994; **49**: 114.
- 7 Kanzaki H, Oono T, Makino E *et al.* Neutrophilic eccrine hidradenitis: report of two cases. *J Dermatol* 1995; **22**: 137–42.
- 8 Cho KH, Han JH, Sim SW *et al.* Neutrophilic dermatoses associated with myeloid malignancy. *Clin Exp Dermatol* 1997; **22**: 269–73.

Malignant deep sclerosing blue naevus presenting as a subcutaneous soft tissue mass

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SIR, Malignancy criteria are well characterized for junctional melanocytic lesions, but not for primary dermal melanocytic lesions.^{1,2} Numerous histological variants have been described for these uncommon lesions,^{1–4} impeding a reproducible diagnosis. We describe the first documented case of a deep sclerosing blue naevus (BN), initially presenting as a subcutaneous mass with histological criteria of malignant melanoma (MM).

A 30-year-old caucasian woman was seen by surgeons for a mobile subcutaneous mass in the back that was thought to be a lipoma/neuroma. A bluish plaque overlying the lesion was initially overlooked and was not reflected in the histopathology request form. The initial specimen revealed a 4-cm well-circumscribed lesion with a solid nodular growth pattern, surrounded by a minimal amount of mature adipose tissue without skin (Fig. 1a). The neoplasm showed necrotic foci, epithelioid cells with irregular nuclei and prominent

Figure 1. Cellular subcutaneous mass showing a nodular growth pattern with islands and strands of neoplastic cells, foci of necrosis and no peritumoral lymphocytic infiltrate (a). Higher magnification reveals coagulative necrosis (b, right side) and homogeneous proliferation of epithelioid cells. The neoplastic cells show eosinophilic cytoplasm, prominent nucleolus, and numerous mitotic figures (c, arrows), along with spindle cells containing melanin (d). Haematoxylin and eosin; original magnification: (a) $\times 16$; (b) $\times 200$; (c,d) $\times 400$.

nucleolus, numerous mitoses ($8\text{--}10\text{ mm}^{-2}$, including atypical ones; Fig. 1b,c) and cytoplasmic melanin (Fig. 1d). Serial sections revealed no lymphovascular invasion at the outer periphery of the lesion and no coexistent melanocytic lesion. With a diagnosis of MM deposit, investigations for primary sites were initiated.

Subsequent examinations demonstrated a long-standing bruise-like 'birthmark' on the left lower back and a 5-year history of a slowly enlarging lump. The lower back revealed

bluish plaques for 1–2 cm around the postsurgical scar (Fig. 2a). There was no metastatic disease elsewhere, as confirmed by computed tomographic (CT) scan, and no obvious primary (investigations included an ophthalmoscopic examination), lymphadenopathy, weight loss, or other masses were identified.

Serial sections of the whole wider excision showed a normal epidermis with no junctional melanocytic lesion. The dermis and subcutaneous soft tissue revealed noninfiltrative

Figure 2. Clinical appearance of the postsurgical scar revealing irregular bluish discoloration, but no nodules (a). The lesion shows irregular fibrous bands with melanin-containing spindle cells (b), positive for Melan-A (b, inset). Densely pigmented melanocytes, both spindle shaped and epithelioid, are revealed in the deepest area of the lesion with interstitial and nested growth patterns (c), positive for Melan-A (c, inset). These melanocytes show no cytological atypia (no nuclear enlargement, nucleolus or hyperchromatism), and no mitotic figures are present (d). Haematoxylin and eosin; original magnification: (b,d) $\times 400$; (c) $\times 160$; (c,d insets) avidin–biotin complex immunohistochemistry; original magnification $\times 160$.

broad fibrous bands containing prominent fibroblasts, melanin-containing dendritic cells (Fig. 2b) arranged in fascicles parallel to the fibrous bands, and aggregates of epithelioid and spindle-shaped melanocytes (S-100 and Melan-A immunoreactive; Fig. 2b,c, insets) along the deep subcutaneous fascia (Fig. 2c). These melanocytes did not reveal atypia, mitoses (Ki-67 index < 5%) or necrosis (Fig. 2d). Neither perineural/intraneural invasion nor residual MM was observed. These findings were diagnostic of deep sclerosing BN.

The diagnosis of subcutaneous MM requires the application of strict diagnostic criteria and investigation of the presence of coexistent lesions. Cytoarchitectural malignancy criteria include diffuse growth pattern, tumour necrosis, nuclear pleomorphism and hyperchromatism, prominent nucleoli and mitotic figures. Coexistent melanocytic lesions [congenital melanocytic naevus (MELN), BN, neurocristic hamartomas, dermal melanocytosis] and clear cell sarcomas are considered in Table 1.^{1-3,5,6} Owing to the previous surgery, sclerosing BN must be distinguished from postsurgical changes and persistent/recurrent BN.

The initial lesion fulfilled criteria for MM:^{1,3} solid growth, tumour necrosis, nuclear pleomorphism and frequent mitoses. Even in the absence of coexistent melanocytic lesions, metastatic MM of unknown primary site is unlikely in young patients.¹⁻³ Metastatic MMs mimicking MELN and

BN are frequently located in the same anatomical region as the primary tumour or near the scar of a dissected lymph node metastasis, and show atypical epithelioid melanocytes, mitoses, and an inflammatory infiltrate at the periphery of the lesion. Although such features facilitate metastasis recognition, clinical correlation is essential for a definitive diagnosis. In our patient, ophthalmoscopic examination and CT scanning identified no neoplasm.

Malignancies arising in dermal melanocytic lesions are rare.^{1-3,7} Most documented malignant BNs have been large (≥ 2.5 cm), affect males more often than females, and are present in the scalp from birth or childhood.⁸ Normally enlarged before excision, they show a proliferation of spindle and epithelioid atypical melanocytes, numerous mitoses, necrotic foci and BN remnants (Table 1).⁸ There is current disagreement about the exact prognosis of malignant BN, indicating the need to improve the reliability of diagnostic criteria and to collect data for these patients.⁸ A complete excision and close follow-up is recommended for these lesions.^{7,8}

Common and cellular BNs have been reported in skin and extracutaneous locations,^{2,3} appearing as acquired (single) or familial (multiple) naevi.⁴ A fibroblastic reaction well beyond the scar and the absence of foreign body-type granulomas suggest no aetiological role for the previous

Table 1. Differential diagnosis of soft tissue malignancies with melanocytic immunophenotype

Categories	Conditions	Key diagnostic features
Without coexistent melanocytic lesion	Metastasis of MM	Near to scar of primary lesion of metastatic lymph node Well circumscribed, no epidermal component Atypical epithelioid melanocytes Mitotic figures Peritumoral lymphocytic infiltrate Deeply located in soft tissue, especially extremities Infiltrative margins Spindle and epithelioid clear cells Heterogeneous growth patterns
	Soft tissue clear cell sarcoma	Involvement of reticular and periadnexal dermis Fibrous matrix reaction Variable cytological atypia Two malignant categories: tumorous dysplasia-blastoma and lentiginous/junctional transformation
With coexistent melanocytic lesion	MM in congenital melanocytic naevus	No junctional component (except for combined naevus) Dendritic, spindle-shaped, oval-shaped or polyhedral melanocytes in variable proportions Dense melanin pigmentation Tumour growth with malignancy criteria
	Malignant blue naevus	Complex proliferation of naevomelanocytes, Schwann cells, and pigmented dendritic and spindle cells No junctional component. Deep dermis or subcutaneous tissue Bland, small, round to spindle-shaped melanin-containing cells Trabecular or nested growth pattern, nuclear palisading and perivascular pseudorosettes
	Malignant melanotic neurocristic tumour	No junctional component Scattered dendritic melanocytes in the reticular dermis Little or no stromal alteration Tumour growth with malignancy criteria
	MM in dermal melanocytosis	

MM, malignant melanoma.

surgery, while the association of stromal reaction and dendritic melanocytes supports the diagnosis of sclerosing BN. This variant must be distinguished from recurrent BN and desmoplastic/neurotropic MM. Persistent/recurrent BN tends to show a spindle-fascicular pattern with dendritic-sclerotic and senescent/atypical changes suggestive of malignancy, extending occasionally beyond the original excision scar.⁹ Although recurrence may suggest malignant transformation of BN,² follow-up supports a benign biology and a reactive nature for these atypical changes.⁹ Our case revealed confluent necrosis, marked atypia and frequent mitoses, supporting the original malignant diagnosis. The absence of an asymmetrical infiltrative pattern with perineural invasion, hyperchromatic/pleomorphic nuclei, mitoses and junctional lentiginous component ruled out desmoplastic MM.

Incomplete specimens make diagnosing malignant BN almost impossible, although the diagnosis of malignancy may be confirmed by demonstrating metastatic deposits during examination of sentinel lymph node biopsies.¹⁰ However, synchronous massive lymph node enlargement can accompany cellular BN,⁷ and small melanocyte groups are occasionally found in lymph nodes draining sites of cellular BN.^{3,7} True metastases cannot be definitively distinguished from MM mimicking cellular BN or nodal primary MM with concomitant cutaneous cellular BN.

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References

- 1 Barnhill RL. Tumors of melanocytes. In: *Textbook of Dermatopathology* (Barnhill RL, ed.), 1st edn. New York: McGraw-Hill, 1998; 561.
- 2 Elder DE, Murphy GE. Melanocytic tumors of the skin. In: *Atlas of Tumor Pathology* (Rosai J, Sobin L, eds), 3rd edn, Vol. 1. Washington DC: Armed Forces Institute of Pathology, 1991.
- 3 Gonzalez-Campora R, Galera-Davidson H, Vazquez-Ramirez FJ, Diaz-Cano SJ. Blue nevus: classical types and new related entities. A differential diagnostic review. *Pathol Res Pract* 1994; **190**: 627–35.
- 4 Carney JA, Ferreiro JA. The epithelioid blue nevus. A multicentric familial tumor with important associations, including cardiac myxoma and psammomatous melanotic schwannoma. *Am J Surg Pathol* 1996; **20**: 259–72.
- 5 Pearson JP, Weiss SW, Headington JT. Cutaneous malignant melanotic neurocristic tumors arising in neurocristic hamartomas. A melanocytic tumor morphologically and biologically distinct from common melanoma. *Am J Surg Pathol* 1996; **20**: 665–77.
- 6 Sciort R, Speleman F. Clear cell sarcoma of soft tissue. In: *World Health Organization Classification of Tumours. Pathology and Genetics of Tumours of Soft Tissue and Bone* (Fletcher CDM, Unni KK, Mertens F, eds). Lyon: International Agency for Research on Cancer Press, 2002; 211–12.
- 7 Gonzalez-Campora R, Diaz-Cano SJ, Vazquez-Ramirez F *et al.* Cellular blue nevus with massive regional lymph node metastases. *Dermatol Surg* 1996; **22**: 83–7.
- 8 Connelly J, Smith JL Jr. Malignant blue nevus. *Cancer* 1991; **67**: 2653–7.
- 9 Harvell JD, White WL. Persistent and recurrent blue nevi. *Am J Dermatopathol* 1999; **21**: 506–17.
- 10 Durrani AJ, Moir GC, Diaz-Cano SJ, Cerio R. Malignant melanoma in an 8-year-old Caribbean girl: diagnostic criteria and utility of sentinel lymph node biopsy. *Br J Dermatol* 2003; **148**: 569–72.

Pattern analysis, not simplified algorithms, is the most reliable method for teaching dermoscopy for melanoma diagnosis to residents in dermatology

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SIR, I read with great interest the paper by Carli *et al.*¹ The authors compared simplified algorithms with pattern analysis in dermoscopic diagnosis of melanocytic skin tumours. The conclusion of the authors was that pattern analysis was the most reliable method for teaching dermoscopy for melanoma diagnosis.

In this paper the authors mixed the terms 'sensitivity' and 'diagnostic accuracy'. The best result for melanoma diagnosis, as represented by the sensitivity, was given by the seven-point check-list, not by pattern analysis as indicated in the title and summary.^{2–4} Concerning the diagnostic accuracy I agree that pattern analysis gave the best results in the classification by the five residents.

Also, as mentioned in this paper, the residents had different skills in dermoscopy. Resident M.S. had previously taken part in a study using pattern analysis and the ABCD rule.⁵ Therefore there may well have been a bias due to his/her higher skills compared with the other residents. Did M.S. correspond to Resident 1 in the paper? If so, the results revealed that the simplified algorithms are better for diagnosing melanomas by residents who are beginners in the method of dermoscopy (e.g. Residents 2, 3 and 5). I agree that for advanced users pattern analysis is more effective in the diagnosis of malignant and benign melanocytic or non-melanocytic tumours of the skin, as stated in the virtual Consensus Net Meeting on Dermoscopy.⁶ The disadvantage of pattern analysis for beginners in dermoscopy is that there is no clear-cut differentiation between malignant and benign lesions.

Finally, I agree with Carli *et al.* that the scoring systems of the simplified algorithms are too complex.¹ Therefore we developed a modified ABC-point list⁷ that was published at the same time as their paper. This is a simplified algorithm with a high sensitivity (90.5%), specificity (87%) and