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Rare localization of low-grade fibromyxoid sarcoma to the nail region

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SIR, Low-grade fibromyxoid sarcoma (LGFMS) is a rare soft tissue tumour. Its incidence is not known and it has not been universally established as a clinicopathological entity. It was originally described by Evans in 1987[1] as 'a metastasizing soft tissue tumour with a deceptively benign histological appearance'. Successive other descriptions[2–5] have appeared in the literature, to support the existence of this tumour as a distinct entity with emphasis on its indolent but malignant behaviour. Several cases showed metastatic spread to the lung. Nevertheless, the disease runs a protracted course in most patients. Most were still alive 10 years after excision of the primary lesion, and recurrences can be treated successfully by re-excision, chemotherapy and radiotherapy.

LGFMS typically arises in the deep soft tissue of young and middle-aged adults, and the most frequent sites reported in the literature are the trunk, neck and roots of the limbs. Other rare localizations are the retroperitoneum, mediastinum, falciform ligament and small bowel mesentery.[2,3,6,7,8] LGFMS has rarely been described in the hands and, to our knowledge, all such cases included aspects of 'hyalinizing spindle cell tumour with giant rosettes'.[6] To our knowledge, no cases of LGFMS have been described in the nail region.

We report two cases of digital soft tumours, located in the nail region, which we encountered between 2000 and 2003. Our two cases did not show any aspect of 'giant rosettes' and satisfied all Evans' histological criteria for LGFMS: the presence of spindle-shaped and stellate cells with relatively hyperchromatic but uniform nuclei, a predominantly whorled arrangement of these cells, the presence of alternating areas with a fibrous or a myxoid stroma (occasionally with an abrupt transition from one to the other) and a bland nodular pattern of growth (Fig. 1).

The immunohistochemical results were in agreement with those reported in the literature.[1–6] There was strong positivity for vimentin and CD99 and negativity for S100 and epithelial membrane antigen (EMA). Furthermore, our two cases were negative for actin, desmin, pankeratin and CD34. Although the full significance of the immunohistochemical profile is unclear, we know that CD99 has been demonstrated in subpopulations of fibroblasts.[9] We hypothesize that these findings in LGFMS are compatible with a CD34–/CD99 + subpopulation of fibroblasts.

Regarding malignant soft tissue tumours, the main differential diagnosis of LGFMS includes low-grade myxofibrosarcoma, myxoid malignant peripheral nerve sheath tumour and acral myxoinflammatory fibroblastic sarcoma.[10,11] We wish to emphasize that LGFMS, with its deceptively bland appearance, may be mistaken for a benign lesion. Thus the differential diagnosis with benign soft tissue tumours, e.g. myxoid neurofibroma, desmoid fibromatosis, superficial angiomyxoma and superficial acral fibromyxoma (SAFM)[10,11] is very important.

In relation to the particular localization, we consider that the most important differential diagnosis of our cases of LGFMS is with SAFM,[6] a distinctive soft tissue tumour that has a predilection for the hands and feet of middle-aged individuals. It is typically located in the dermis, sometimes involving the subcutis, and in a few instances extending to the fascia or to the periosteal surface of the bone. It is characterized by spindle-shaped and stellate fibroblast-like cells with random, loose storiform, and fascicular growth patterns. The lesional cells are embedded in myxoid, myxocollagenous or collagenous matrix, often with mildly to moderately accentuated vasculature (most notable in the highly myxoid examples). Mast cells are easily identified. SAFM is positive for CD34, EMA and CD99. Our two cases showed many similarities with SAFM because the tumours were from acral sites (nail region of the fingers) and, like SAFM, they had variable amounts of myxoid matrix, spindle-shaped cells with random and loose storiform growth patterns, and increased vascularity. In contrast to SAFM, in our cases the myxoid areas were circumscribed and delimited by fibrous bundles forming different sized nodules with an abrupt transition from one type of area to the other. Moreover, we did not detect the presence of mast cells, either with toluidine blue stain or with CD117 antibody. Finally, CD34 and EMA were negative.[9]

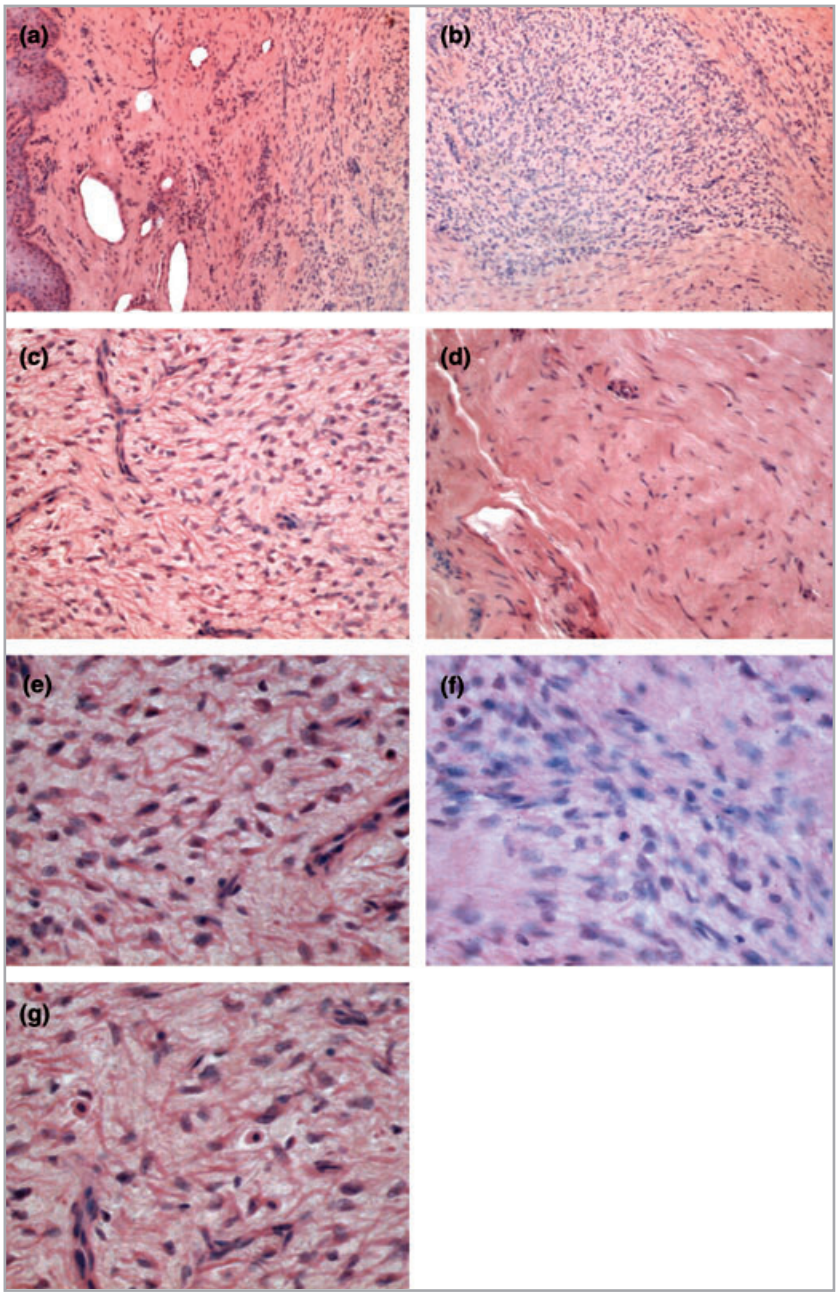

Fig 1. (a) Tumour in papillary dermis, with a Grenz zone, under the epithelium of the nail matrix. (b) Myxoid areas circumscribed and delimited by fibrous bundles forming nodules with an abrupt transition from one type of area to the other. (c) Cells in a whorled and partially storiform pattern of growth, with a network of curvilinear and branching capillary-sized vessels. (d) Extremely hypocellular areas with dense fibrous background; the swirling pattern is still discernible. (e) Stellate and spindle-shaped cells with poorly defined cytoplasm and small oval nuclei that were bent and wavy in appearance. (f) Pleomorphism was very slight and mitoses were very rare, fewer than 1 per 30 high-power fields (HPF) (1 HPF = 0·159 mm^2). (g) Apoptotic cells, with strongly eosinophilic cytoplasm and hyperchromic/condensed nuclei, more than 3 per HPF.

Fetsch *et al.*[6] found two of 23 cases of SAFM to be negative for CD34, and a further case to be recurrent. Five cases showed nuclear atypia and the authors suggested the possibility that SAFM may progress to a low-grade neoplasm (fibromyxoid tumour of low malignant potential). We consider that SAFM and LGFMS are two different entities, both histologically and immunohistochemically. Perhaps the two CD34– cases and/or the recurrent case reported by Fetsch *et al.* are LGFMS *ab initio*. In fact, the picture of the recurrent case shows an abrupt transition between fibrous and myxoid areas. Moreover, we consider that some 'cutaneous and subcutaneous low-grade myxoid neoplasms with recurrent potential' can represent cases of LGFMS, when they are negative for CD34 and S100.[12,13]

Finally, we hypothesize that the superficial localization of LGFMS, like other soft tissue tumours (i.e. low-grade lipomatous tumours, low-grade myxofibrosarcomas), could be a good prognostic factor. However, long-term follow-up of a greater number of cases is required to assess the real behaviour of these superficial low-grade fibromyxoid tumours.

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A novel type of human papillomavirus (HPV 95): comparison with infections of closely related human papillomavirus types

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SIR, Infection with certain human papillomavirus (HPV) types induces warts with specific macroscopic and microscopic features.[1] Clinical, histological and virological correlations have been established between myrmecia (deep palmoplantar warts), granular intracytoplasmic inclusion bodies (Gr-ICBs) and HPV 1, between pigmented warts, homogeneous (Hg)-ICBs and the related HPV 4, 60 and 65, and between punctate warts, filamentous (Fl)-ICBs and HPV 63.

We recently observed a distinct type of Hg-ICB in small wart-like lesions in a 16-year-old Japanese renal transplant recipient with a 2-year history of viral warts on her hands and feet (Fig. 1a). The asymptomatic small warty papules of normal skin colour shared some resemblance to those of HPV 2/27/57-induced common warts, HPV 4/60/65-induced pigmented warts, or HPV 88-associated small wart-like lesions.[2] Microscopically, the epidermis was acanthotic with hyperkeratosis and partial hypergranulosis, with basic histological features compatible with those of common warts. The additional histological feature of eosinophilic Hg-ICBs surrounding a vacuolated nucleus in each cell in the granular and upper spinous cell layers was characteristic for all lesions examined (Figs 1b,c). These microscopic features of the Hg-ICBs resembled those of HPV 4/65-associated Hg-ICBs (Figs 1d,e) to a certain extent, but were quite distinct from those of HPV 60-associated Hg-ICBs (Fig. 1f), as well as those of HPV 1-associated Gr-ICBs, HPV 63-associated Fl-ICBs and HPV 88-associated fibrillar ICBs. The subsequent comparative analysis of lesions displaying the different types of Hg-ICB revealed an abundance of the HPV 95-associated Hg-ICBs in the upper epidermal cell layers, similar to that seen in HPV 4/65-associated lesions, in contrast to a few scattered cells in the HPV 60-infected lesions. HPV 95-associated Hg-ICBs shared the feature of eosinophilic bodies with a crescent-like appearance with HPV 4/65-associated Hg-ICBs, in contrast to the round bodies in HPV 60-associated Hg-ICBs. The crescent-like eosinophilic bodies were, however, usually 'atrophic' and separated from an extremely vacuolated nucleus in HPV 4-induced warts, in contrast to that seen in HPV 65/95-induced warts where they filled out the entire cytoplasm and encased the nucleus. Tiny vesicular structures seen within HPV 4/65-associated Hg-ICBs were not seen in Hg-ICBs associated with HPV 95. Clear distinction between the different types of Hg-ICB was not always possible, despite the differences mentioned.

The full-length genome of a novel papillomavirus was cloned from these lesions and characterized. The DNA genome of HPV 95 consists of 7337 base pairs (accession number AJ620210). The genome shows a gene arrangement characteristic for all HPVs, except for the absence of a distinct E5 open reading frame between the early and late regions, similar to other cutaneous HPV types.[3] The nucleotide sequence of the HPV 95 L1 open reading frame shares 80% homology to HPV 65 L1 and 79% homology to HPV 4 L1, thereby defining this novel HPV type to species 1 of the genus Gamma-papillomavirus.[3] The percentage homology to HPV 60 L1 (species 4) is 62% and to HPV 88 L1 (species 5) is 61%.

The present case is the only patient with HPV 95-associated inclusion warts studied thus far, suggesting that HPV 95 is an extremely rare HPV type. It seems likely that the conditions accompanying the immunosuppression of this renal transplant recipient may have contributed to stimulate viral production of HPV 95, thus leading to wart formation. To ascertain the true nature of HPV 95 and its associated inclusion warts, we need to perform epidemiological, as well as further clinicopathological and virological studies on a larger