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Case report

Bulky naevocytoma of the perineum: a singular variant of congenital giant pigmented naevus

Miguel Reyes-Mugica[1], Frank Gonzalez-Crussi[1], Bruce S. Bauer[2], and Gilberto Medina-Escobedo[3]

Departments of [1] Pathology, and [2] Plastic Surgery, Children's Memorial Hospital, Northwestern University, Chicago, Illinois, USA
[3] Department of Pathology, National Institute of Paediatrics, Mexico City, Mexico

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Summary. We describe two instances of a previously unrecognized variant of congenital giant pigmented naevus (GPN), presenting as a bulky naevocytic tumour in the perineal region. In both cases the lesion was present at birth and attained massive dimensions. In addition to the characteristic histological patterns found in GPN, which included extensive areas with a neural appearance, these tumours presented an uncommon tendency to form pseudo-follicular structures lined by naevus cells. No features suggestive of malignant transformation were found. Because GPN may associate with an underlying malignancy, accurate diagnosis of this lesion is important in clinical practice.

Key words: Giant pigmented naevi – Naevocytoma – Congenital tumours

Introduction

Giant pigmented naevi (GPN) occurring at birth are striking lesions, apt to attract attention. Accordingly, a voluminous literature has evolved in which many of their clinical and biological features are described. Thus, it is known that one or more segments of the body may be involved, often with a dermatome distribution (Reed et al. 1965); that satellite lesions may or may not be present (Castilla et al. 1981; Greeley et al. 1965); and that associated conditions, such as limb maldevelopment in GPN covering the extremities, or meningeal melanosis, mainly in those covering the head and neck, may complicate the course of the disease (Reed et al. 1965). Other facts generally appreciated are: the lack of obvious hereditary transmission (Solomon et al. 1980) but tendency to familial aggregation (Reed et al. 1965; Rhodes 1987) and the potential for malignant transformation, even though accurate estimates of the latter occurrence are far from agreed (Gari et al. 1988; Greeley et al. 1965; Hendrickson and Ross 1981; Kaplan 1974; Lanier et al. 1971; Reed et al. 1965; Russell and Reyes 1959). Considering the abundant information and very numerous reports on GPN, it is surprising that a congenital, highly cellular naevocytic tumour of the perineum with apparently distinctive clinical and pathological presentation has not been described. In this communication, we record two examples of such a lesion.

Offprint requests to: Miguel Reyes-Mugica, Department of Pathology, Children's Memorial Hospital, 2300 Children's Plaza, Box 17, Chicago, IL 60614, USA

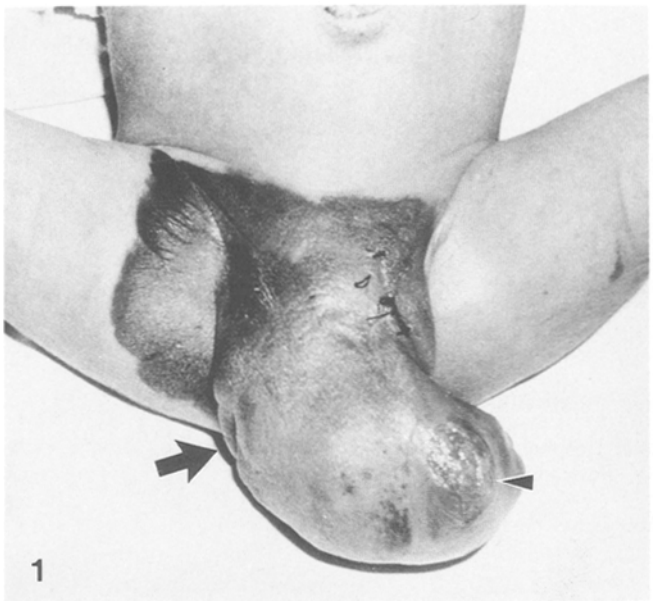
Case reports

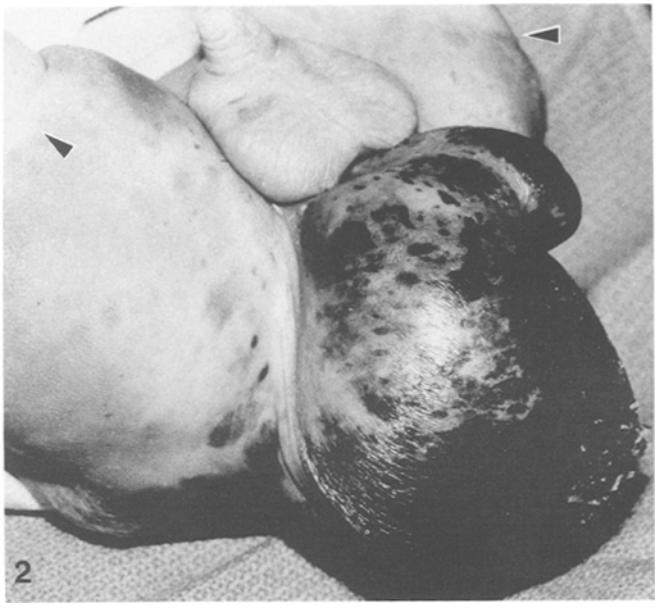
Case 1

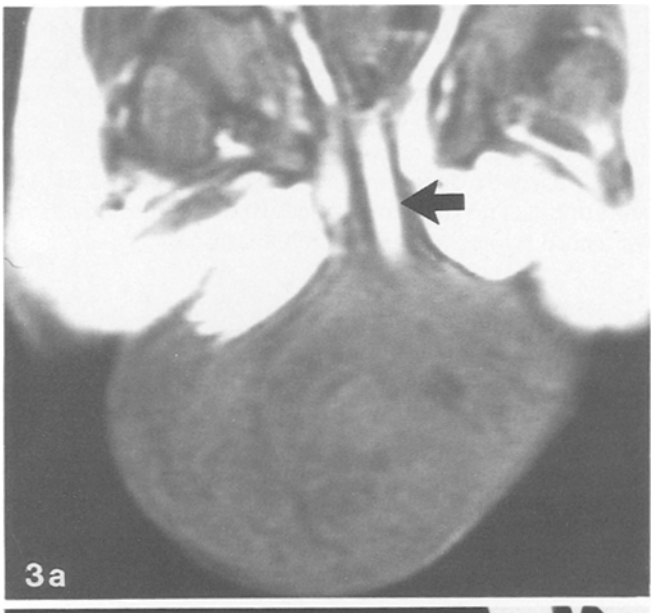
A male newborn was admitted to the National Institute of Paediatrics, Mexico City, at the age of 2 days, presenting with a large tumour in the genital and perineal areas. The family history was non-contributory and the pregnancy was unremarkable. On physical examination the tumour appeared as a hyperpigmented pedunculated cylindrical mass measuring 10×6.5 cm. It was impossible to recognize the patient's external genitalia because of the bulky deformation of the area. The anus was unaffected. The skin surrounding the tumour was hyperpigmented for a rim of 2–3 cm (Fig. 1). The lesion had a soft rubbery consistency with some nodular areas, and the overlying skin showed variable pigmentation. There were a few satellite hyperpigmented skin lesions, between 3 and 5 cm in diameter, located on the lumbar region and lower extremities. Radiography of thorax and vertebral column, venocavography and pyelography were normal. A cystourethrogram disclosed a male urethra abnormally positioned in its membranous and penile portions, but of normal calibre. After an incisional biopsy, the tumour was resected surgically. A second surgical procedure was performed 1 year later, to remove part of the remaining hyperpigmented skin lesion. At the age of 2.5 years, the patient is asymptomatic, with a smaller persistent area of hyperpigmentation in the genital and perineal region.

Case 2

A male newborn was admitted to the Children's Memorial Hospital in Chicago, at the age of 9 weeks. On physical examination there was a large, sessile tumour in the perineum, covered by a heavily







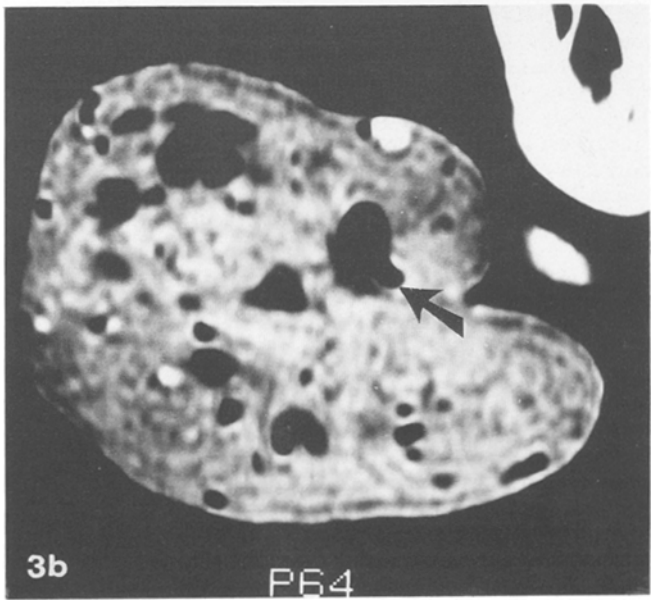


Fig. 1. Bulky tumour in the perineal and genital area (case 1). The skin surface was hyperpigmented and some irregular nodules could be seen (*arrow*). An ulcerated area was present at the tip of the mass (*arrowhead*). Note the hyperpigmented skin rim around the tumour implantation

Fig. 2. Hemispheric perineal tumour (case 2). The mass was covered by heavily hyperpigmented skin with multiple spots. Note the light hyperpigmented skin covering the proximal part of the thigh and the abrupt colour change to the normal-appearance skin (*arrowheads*). Some small satellite lesions can be identified

Fig. 3. a A coronal T1-weighted magnetic resonance image through the pelvis shows the relationship of the perineal mass to the rectum (*arrow*). **b** An axial T1-weighted magnetic resonance image through the mass after intravenous injection of gadolinium shows multiple small cystic spaces. The mass completely surrounds the rectum (*arrow*)

hyperpigmented skin with irregular surface, surrounded by an area of brown light hyperpigmented skin of smooth surface with "bathing-trunk" distribution (Fig. 2). The tumour mass was hemispheric, 10×8 cm, of soft rubbery consistency. The lesion infiltrated the perineum to the level of the deep fascia of perineal muscles and extended to the anal verge, which was distorted by the neoplasm. On radiographic examination no skeletal abnormalities were detected. Nuclear magnetic resonance study of the tumour demonstrated no anatomical connection with the coccyx and showed some small, cystic areas in the mass (Fig. 3). The tumour was surgically resected. Extensive infiltration was noted around the full circumference of the anus, extending into the base of the scrotum. The mass was excised sparing the external sphincter; peripheral buttock flaps were used to close the defect. At the age of 8 months, the patient is asymptomatic. Future surgery will be directed at excising the bathing trunk naevus.

Materials and methods

Tissue sections fixed in 10% buffered formalin were processed for conventional light microscopy, and stained with haematoxylin and eosin (H&E). Selected fragments were stained by the following methods: Fontana-Masson, Masson's trichrome, periodic acid-Schiff reagent (PAS), or alcian blue at pH 2.5.

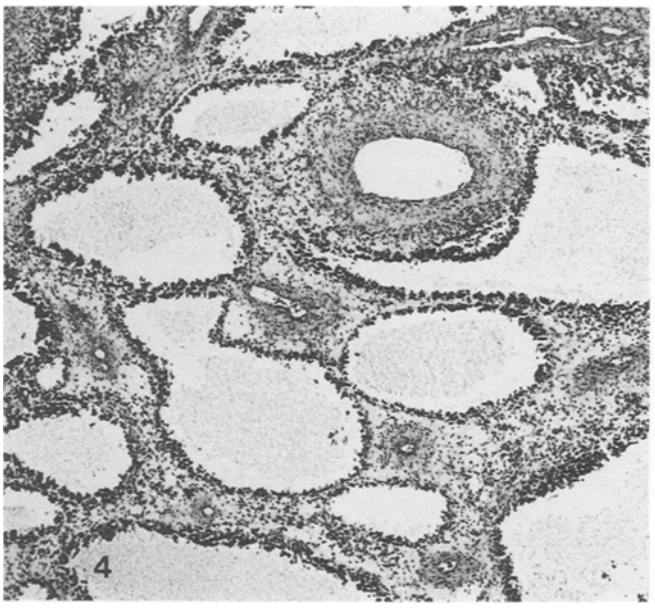


Fig. 4. Photomicrograph showing a low-power view of the pseudo-follicular areas lined by the naevo-follicular cells. Note the abnormal large vessel. Masson's trichrome. ×25

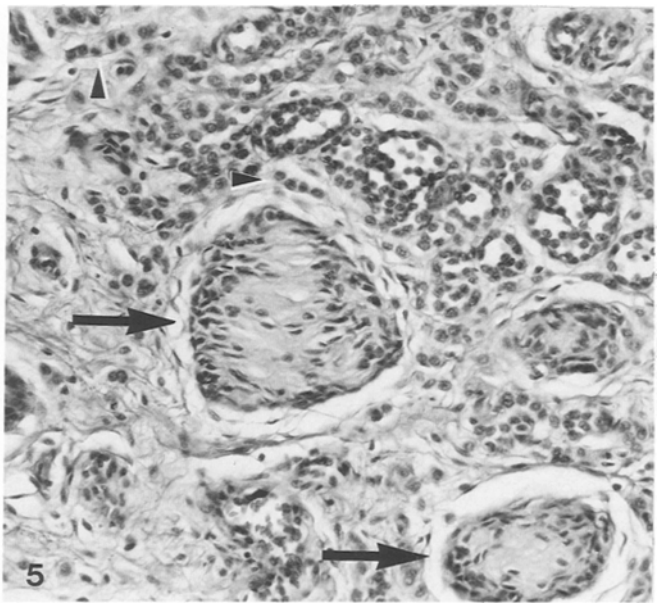


Fig. 5. Photomicrograph showing some nests of naevo-follicular cells in close relation to some Wagner-Meissner bodies (*arrows*). The "Indian file" pattern of naevus cells is present (*arrowheads*). H&E, ×250

For immunohistochemistry, we used an avidin-biotin complex immunoperoxidase-linked kit (Vectastain ABC kit, Vector Laboratories, Burlingame, Calif.) and followed the manufacturer's instructions. The following primary antibodies were used: anti-S-100 protein (Biomeda, Foster City, Calif.), anti-vimentin (Dakopatts, Glostrup, Denmark), HMB45 antibody (Enzo, New York, N.Y.), anti-epithelial membrane antigen (Dakopatts) and anti-factor XIIIa (Calbiochem, La Jolla, Calif.).

For electron microscopy, multiple tissue blocks were fixed in 3% glutaraldehyde, processed by a standard technique, embedded in Epon, cut at 1 μm thickness, and stained with toluidine blue to select representative areas for ultrastructural study. Ultra-thin sections were obtained with a Sorvall MT 6000 ultratome, stained with lead citrate and uranyl acetate, and examined with a Zeiss M-10 electron microscope.

Results

Macroscopically, case 1 showed a cylindrical tumour 11.5 × 7 × 7 cm, partly covered by a heterogeneously hyperpigmented skin with a slightly nodular surface and an irregular ulcerated zone of 2.5 cm in largest diameter. The cut surface showed pink, fleshy tissue, with cystic cavities 1–3 mm in diameter in some areas, some of them filled with blood. A pigmented zone 1–2 mm in width was identified only at the periphery, close to the epidermis.

In case 2, the specimen was an oval tumour 12 × 8 × 5 cm, partly covered by rough skin with multiple light and dark, brown and black spots. The cut surface was very similar to that of case 1, with more prominent cystic spaces and areas of honeycombing; the pigmented zones were present only beneath the epidermis.

Since the microscopic features are virtually identical in both cases, a combined description is given. A very complex morphology is observed. The epidermis is atrophic over most of the lesion. The basal layer of the epidermis is moderately pigmented. Tiny nests of naevus cells are present at the dermo-epidermal junction, thought to be consistent with minor junctional activity. Heavier pigmentation occurs in clusters of naevus cells arrayed along the upper dermis, where they constitute a thin layer parallel to the epidermis. This layer of naevus cells is the only element of the lesion that contains melanin, apart from the basal epidermis; no melanin is evident histologically in any other component of these massive lesions.

Cells with abundant clear or eosinophilic cytoplasm, rounded or cuboidal, about 15 μm in diameter, are found in the upper dermis, but also in deeper portions of the lesion. They are thought to correspond to so-called **A cells** (Lever and Schaumburg-Lever 1990; Meischer and Von Albertini 1935) except that they show no visible melanin. The nucleus is vesicular, folded, indented or irregular, with very fine chromatin and inconspicuous or absent nucleoli. These cells are disposed in the patterns termed "Indian files, cords, nests, and sheets", according to Mark et al. (1973).

Morphologically very similar, and perhaps a subtype of the **A cells**, are cells resembling large lymphoid cells. Distinction from the **A cells** is possible by virtue of their smaller size, thicker nuclear membrane, coarser chromatin, and scantier cytoplasm, often reduced to a thin, perinuclear rim or inapparent. We cannot positively identify this cell type with **B naevus cells** (Lever and Schaumburg-Lever 1990; Meischer and Von Albertini 1935) because of their seemingly "active" nuclei, lack of tendency to adopt a spindly shape, and certain immunophenotypical differences from **A cells** (see below). Moreover, these cells adopt architectural patterns not common to **B cells**. Chief among these is a pseudo-follicular pattern in which

Table 1. Results of immunohistochemical staining

Tumour component	S-100	Vimentin	HMB45	EMA	FXIIIa
A cells	++	+++	+++	−	−
Naevo-follicular cells	+	++	+/−	−	−
C cells	+++	+	−	−	−
N corpuscles	+++	+	−	−	−
Stroma	−	+	−	−	+

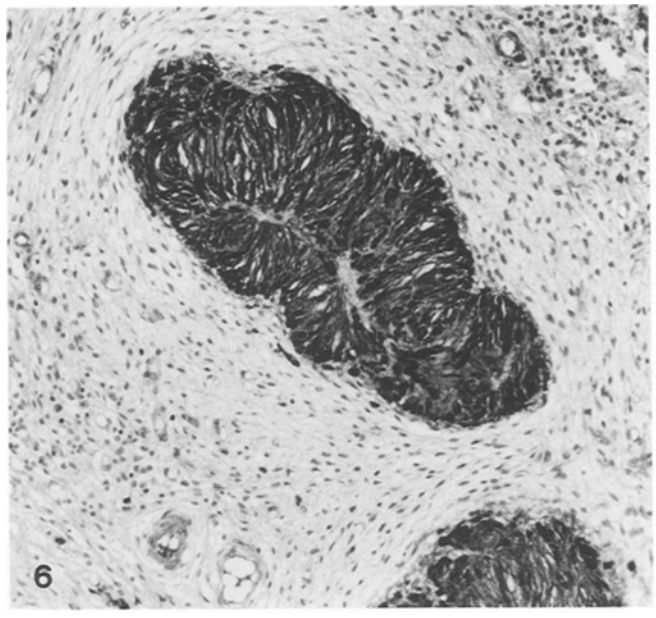
+, Weakly positive; ++, moderately positive; +++, intensively positive; −, negative; EMA, epithelial membrane antigen; FXIIIa, factor XIIIa; N corpuscles, naevus corpuscles

the cells pile up in multiple ill-formed layers around a central space (Fig. 4) that enclose a proteinaceous, PAS-negative, alcian blue-negative substance. We have tentatively referred to these cells as naevo-follicular by virtue of their cytological features and peculiar pattern of growth. Lastly, **C cells**, that is to say spindly cells arranged in bundles with greater or lesser collagenization, are prominent throughout. As in some GPN, these cells reproduce the morphology of neurofibroma fibres or form the discrete naevus corpuscles evocative of Wagner-Meissner's tactile bodies that are sometimes called **lames foliacées** (Fig. 5). These areas of the tumour with a neural appearance make up a very high proportion of its massive bulk, and extend to all levels of the lesion, up to the upper dermis.

Contributing to the complexity of histological appearances is a vascular component made up of abnormal, large, gaping vessels with ill-formed walls in which the subintimal tissue contains abundant matrix positive to alcian blue and to PAS stains. These vessels are difficult to identify as arteries or veins. **A cells** or naevo-follicular cells sometimes are concentrically arranged around the vessels. In some areas, a heamangiomatous appearance is produced by numerous sinusoidal or capillary type vessels that anastomose widely in a background of tumour cells.

The results of immunostaining are summarized in Table 1. Epithelial membrane antigen is uniformly negative in all tumour cells. Factor XIIIa is expressed by fibroblast-like cells and cells with cytoplasmic extensions scattered in the stroma, but tends to concentrate around vessels. Vimentin is intensely positive in round or cuboidal tumour cells identified as **A cells**, but appreciably less so in naevo-follicular cells; the spindle-cell component of the tumour is only weakly positive. The pattern of S-100 protein expression is the reverse of vimentin's, in the sense that spindle (C) cells, particularly naeval corpuscles, are intensely positive (Fig. 6), whereas **A cells** react moderately and naevo-follicular cells weakly, to this antibody. Immunoreactivity to HMB45 antibody (Gown et al. 1986) is also non-uniform: the areas of the tumour with a neural appearance are completely negative, the naevo-follicular cells react weakly or ambiguously, whereas naevus **A cells** are intensely immunoreactive (Fig. 7).

On electron microscopy, rounded naevus cells, corresponding to **A cells**, have irregularly contoured nuclei



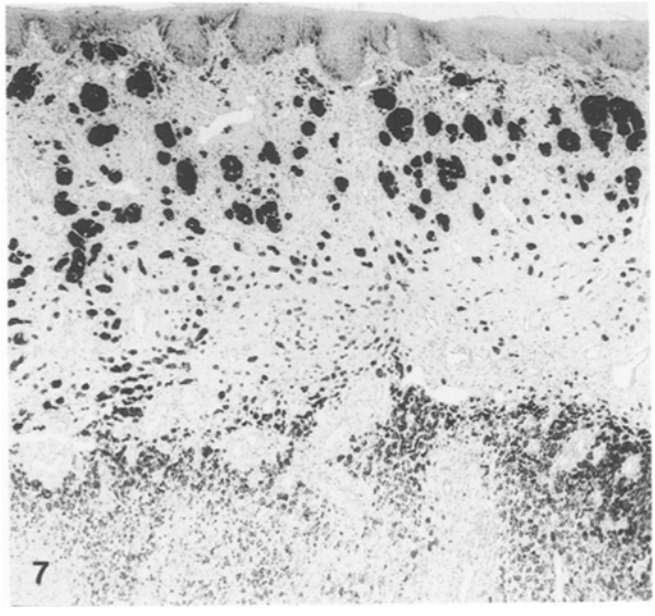
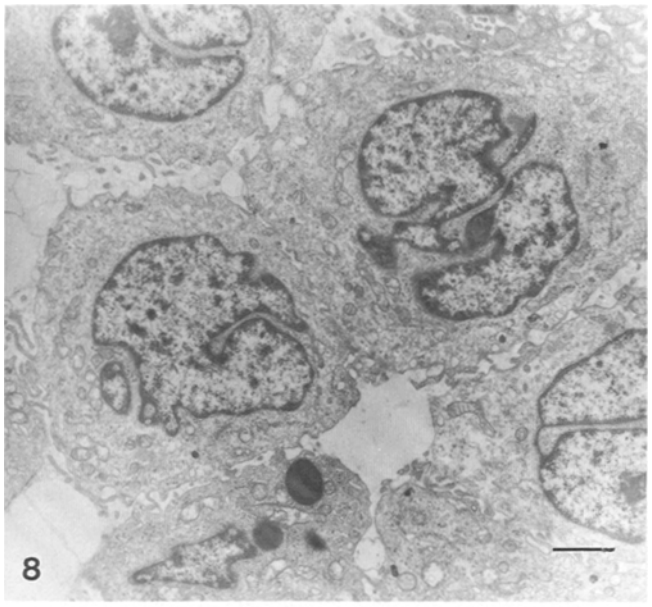


Fig. 6. Photomicrograph showing strong S-100 positivity of a naevus corpuscle in a area with neural appearance. Anti-S-100, ABC-complex procedure, ×250

Fig. 7. Low-power view showing the pattern of immunoreactivity obtained with the HMB45 antibody. HMB45 antibody, ABC-complex procedure, ×25

with many deep indentations and finely dispersed heterochromatin; nucleoli are generally inconspicuous, and when apparent in the plane of section tend to be placed against the nuclear envelope. The cytoplasm is abundant and rich in smooth or coated vesicles of varying size, many appearing "empty". Mitochondria are irregular, of clear matrix and transverse cristae. Despite numerous vesicles, Golgi profiles are infrequent and consist of few stacks. Likewise, the development of rough endoplasmic reticulum varies considerably from cell to cell, but in **A cells**, notwithstanding their ample cytoplasm, it is fre-



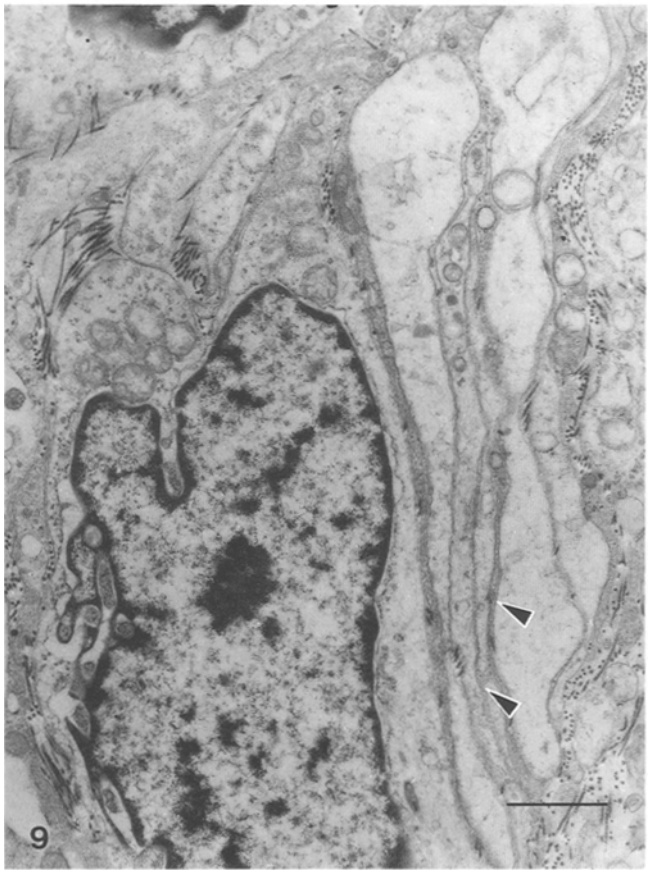


Fig. 8. Electron micrograph showing cells with extreme nuclear convolutions, and abundant vesicular cytoplasmic profiles, but poor development of rough endoplasmic reticulum. Note the presence of dense cytoplasmic inclusions, and lack of desmosomes; *bar*, 2 μm; ×4400

Fig. 9. Electron micrograph showing juxtaposition of cytoplasmic extensions (*arrowheads*) tending to reproduce the appearance of Wagner-Meissner corpuscles; *bar*, 1.7 μm; ×7000

quently represented by a few discontinuous, short cisternae. The previously reported observation that most naevus cells of GPN lack typical melanosomes (Mark et al. 1973) is confirmed in the present study. Rather, the majority of **A cells** contain electron-dense cytoplasmic granules of about 1–5 μm and heterogeneous morphology, compatible with the so-called melanin macroglobules described by Nakagawa et al. (1984), and interpreted by these authors as lysosomal-autophagic in nature. No consistent differences are found between **A cells** and naevo-follicular cells. The latter are identified by smaller size and higher nucleo-cytoplasmic ratio. In some of these cells the rough endoplasmic reticulum is highly developed, and co-exists with free ribosomal groups. **A cells** and naevo-follicular cells are often found closely intermixed. However, cell-to-cell junctions are scanty, poorly developed, and, as Gottlieb et al. (1965) have described, never show the morphology of typical desmosomes (Fig. 8). Spindle cells (C cells) show the structural features previously described by others (Mark et al. 1973). Where these cells extend laminated flaps of cytoplasm in close juxtaposition to each other, the resulting appearance is that of the "naeval corpuscle" (Fig. 9). We confirmed the observations of Sueki et al. (1984), that these "foliated" structures are composed exclusively of long cytoplasmic processes of naevus cells, but do not contain axons, nor are nerve fibres seen anywhere in the vicinity. This is in contrast to true Meissner corpuscles, in which an axonal component can be demonstrated ultrastructurally (Sueki et al. 1984).

Discussion

We know of no previous case directly comparable to the two tumours discussed here. The congenital tumour described by Jerdan et al. (1985) may be an exception, but it exhibited foci of chondroid differentiation and lacked follicular structures. Arao et al. (1989) described under the name of "proliferating giant pigmented naevus" a benign melanocytic lesion located in the vulva of a 32-year-old Japanese woman who also harboured a GPN of the torso. As in our patients, this lesion presented as a bulky pedunculated mass that suggested an underlying tumour: from its origin in labia majora the 4 kg tumour extended almost to the level of the knee with the patient standing, suggesting an underlying neoplasm of soft tissues. This possibility is of importance in children, since rhabdomyosarcoma has been known to associate with GPN (Zuniga et al. 1987). However, the tumour described by Arao et al. (1989) showed a simpler histological composition than our two examples. Neither neuroid areas nor truly follicular patterns were mentioned. Presentation in adult life, with slow growth over the 8 years that preceded the diagnosis, are further differences that set this case apart from the two lesions that form the subject of the present report. The same may be said of other occasional reports, such as that of Ito et al. (1988), who described a 47-year-old woman with a large, focally "neurotized" subcutaneous tumour in the scalp that grew over several decades.

Our two cases were remarkable for the consistency of their clinical and pathological manifestations. In both patients the tumour had attained bulky dimensions at birth. By virtue of their anatomical location, an important differential diagnosis was sacrococcygeal teratoma. Various pathological changes may affect the skin that overlies a sacrococcygeal teratoma, including naevi and haemangiomas (Gonzalez-Crussi 1982), thereby enhancing the importance of this distinction. The non-teratomatous nature of these masses could be suspected from the fact that the tumours were not contiguous with the coccyx, but diagnostic certainty was not possible before study of the histopathological features. Confusion with teratoma was enhanced due to the presence of cystic spaces on imaging studies. Likewise, the neural crest cells from which GPN originate are capable of embarking on multiple lines of differentiation, and therefore may give rise to multiple types of soft tissue tumours in association with GPN (Hendrickson and Ross 1981). As already described, soft tissue malignancy is a possibility (Zuniga et al. 1987).

Another important clinical differential diagnosis is neurofibromatosis, in view of the well-known association of this condition with large cutaneous masses, or so-called pachydermatous growths (Reed et al. 1965), which may co-exist with GPN. Lesions with neuroid appearance are common to GPN and neurofibromatosis (Bousema et al. 1989; Hendrickson and Ross 1981; Solomon et al. 1980). Two of 223 patients with von Recklinghausen's disease reported by Crowe et al. (cited by Solomon et al. 1980), and 2 of 26 patients in another study (Brasfield and Das Gupta 1972) had GPN. It has been said that 5% of the patients with neurofibromatosis have GPN (Solomon et al. 1980). Although neither of our patients has yet been investigated with the modern available tools of molecular biology, none was clinically thought to suffer from neurofibromatosis, because of the absence of a suggestive history, or stigmata of the disease. Furthermore, the immunostaining pattern observed with factor XIIIa in our two cases corresponds to the findings in neuroid naevi, and is opposed to the reactivity of neurofibromas described by Gray et al. (1990). The appearance of neuroid areas has been regarded by some authors as an age-related phenomenon, most pronounced at the base of acquired naevi, and likely to develop in old GPN (Mark et al. 1973). Its prominence in our two congenital cases belies the concept that neurofibromatous differentiation is a function of time, or that only conditions present in the postnatal period can foster its development.

A highly uncommon structural feature also present in the two cases reported here was the formation of pseudo-follicular structures. These areas of heaped up cellular growth might simply reflect active proliferation of poorly differentiated naevus cells. Despite the high cellularity, mitoses were very rare, and it does not seem probable that our cases had a high malignant potential. Indeed, it is extremely unlikely for a congenital pigmented lesion to be malignant in the neonatal period: according to Mancianti et al. (1990), only 16 cases are on record of malignant melanoma diagnosed at birth or in the neonatal period. Moreover, pigmented congenital lesions of alarming histology may not be truly malignant; the potential for malignant transformation increases with age, but it rarely manifests below the 6th month of life (Mancianti et al. 1990) and continues to exist throughout lifetime (Kaplan 1974). It is becoming increasingly apparent that features traditionally associated with malignancy in pigmented lesions, including high mitotic activity, pleomorphism, formation of nodules, ulceration, pagetoid invasion of the epidermis and inflammatory reaction – none of which were present in our cases – can coincide in neonatal naevi that behave as benign tumours when the patient is a neonate (Angelucci et al. 1991; Mancianti et al. 1990). Therefore, we feel justified in regarding our cases as benign lesions, without diminishing the importance of timely resection to stave off future complications.

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