

located and resembles hemangiopericytoma. Our case, however, did not show the typical hemangiopericytoma-like pattern. Immunohistochemically, the tumor cells are immunoreactive for vimentin and actin, but do not stain for desmin or S-100 protein, consistent with myofibroblastic differentiation [2].

There is some debate as to the histogenesis of the tumor. Benjamin *et al.* [3] concluded that it principally reflects a proliferation of myofibroblasts, based on ultrastructural and immunofluorescent studies, whereas the similar histological features between infantile myofibromatosis and infantile hemangiopericytoma led Mentzel *et al.* [4] to propose that these two conditions may represent different stages of maturation of a single entity. In our case, the tumor cells were widely positive for CD34, although such immunoreactivity in infantile myofibromatosis has not been discussed previously. The CD34 antigen is a 110-kDa heavily-glycosylated transmembrane protein of unknown function, which was originally described as a marker of human hematopoietic stem cells. It has been found to be expressed in a diverse group of neoplasms, including leukemias, vascular neoplasms, hemangiopericytomas, epithelioid sarcomas, dermatofibrosarcoma protuberans, and solitary fibrous tumors [5]. Most hemangiopericytomas express CD34, and it has been suggested that this expression might be related to a common origin of endothelial cells and pericytic cells, although it is equally possible that CD34 reactivity relates hemangiopericytomas to cells normally exhibiting smooth-muscle differentiation [6].

Our case suggests that infantile myofibromatosis is caused by a proliferation of myofibroblasts exhibiting smooth-muscle differentiation, and that this tumor may be regarded as a single entity as infantile hemangiopericytoma. Although the function of the CD34 molecule remains unknown and the explanation for its expression by such diverse neoplasms is unclear, CD34 may be a useful marker for diagnosis and histogenesis of infantile myofibromatosis. ■

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The “COMMA-flap”: a new technique for inner canthus reconstruction

Surgery is the preferred treatment of tumors in the inner canthus area; the excision of the neoplasia should be radical, with wide margins, because tumours in this region tend to recur and involve lachrymal ducts, the eyelid and the bulbar conjunctiva.

To avoid local recurrences the margins and the bottom of the lesion have to be subjected to extemporaneous histological examination and once the surgeon has obtained wide margins free from neoplasia, the flap may be harvested.

After tumour excision, the defects of the inner canthal area are generally repaired by full-thickness skin grafts, Limberg flaps, glabellar flaps or forehead flaps that sacrifice the supratrochlear pedicles [1]. All these techniques can cause some problems in restoring the functional and/or aesthetic features of this region [2-6].

The glabellar flap is a significantly thicker flap and is, therefore, often too bulky to maintain the natural concave shape normally found in the medial canthus; this flap also sacrifices the supratrochlear arteries, the vascular pedicle of the forehead flap, which would not be considered a safe procedure in case of tumor recurrence.

In 22 patients (aged from 55 to 91) the authors performed reconstruction of the medial canthal area after tumour excision by using a myocutaneous advancement-rotation flap from the glabellar area, sparing the supratrochlear pedicles: the “COMMA Flap”.

This flap, with its random pattern of vascularization, allows for covering a substance loss of the inner canthus area of up to 3.5 × 2.2 cm².

Furthermore, the wide excision of the tumours (related to the possibility of reconstruction) reduced the incidence of recurrences in our cases.

The name “COMMA-Flap” comes from the movement of the flap from the donor to the recipient site. During the preoperative stage in all patients, the surgeons localized the supratrochlear artery using a portable Doppler probe, in order to preserve its integrity during the harvesting of the flap. Once the loss of substance was delimited, a flap of adequate size was drawn in the glabellar area, between the two supratrochlear arteries, as a musculocutaneous advancement flap that included the procerus muscle.

The subperiosteum dissection allows for the flap to be advanced up to 1 cm.

The flap is positioned with a deep layer of 5-0 absorbable sutures and a cutaneous layer of interrupted 6-0 nylon. The donor site is closed with a simple suture.

After surgery, the surgeons controlled the integrity of the supratrochlear pedicle using a Doppler probe. In each case, the long term results were cosmetically very satisfying (*figure 1*). No immediate or delayed complications, related to this technique, were noted in the follow up period. There was no flap edema in the follow up period; this could be because of the preservation of the lymphatic vessels during the sub periosteum dissection of the comma- flap.

In 2 cases there was a tumor recurrence in the lateral aspect of the flap, 18 months after initial surgery. These cases needed only a simple excision.

The advantages of this flap include: good match of tissue thickness, no need for padding or immobilisation, no significant donor-site complications, residual scars hidden between the glabellar wrinkles, no sensory loss in the forehead area, and the possibility of harvesting a flap with

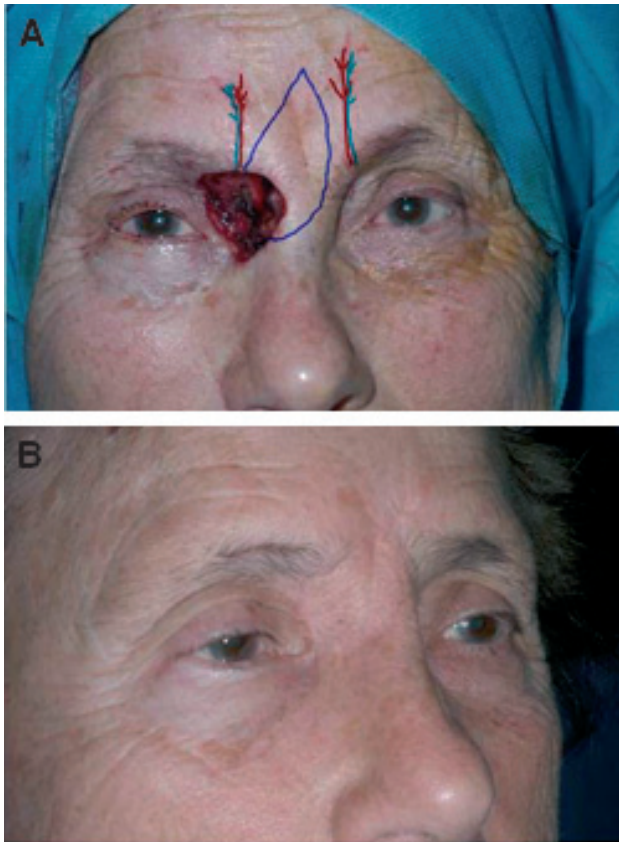


Figure 1. **A)** The tissue defect with the exposed periosteum after surgery. The loss of substance was 3 × 2 cm. **B)** Result after 1 year. Good medial canthal contour without evidence of donor site morbidity or recurrence.

an axial supply from the supratrochlear arteries. Using a Doppler probe to preserve the supratrochlear vessels is not a disadvantage because it is inexpensive and easy to use. We believe that the COMMA-flap is the preferred treatment, especially in patients with multiple basal cell carcinomas (BCC) of the face; it is the first step towards inner canthus reconstruction, since the integrity of both supratrochlear arteries allows for the use of other flaps in secondary surgery like forehead flaps, this being the only option for complete nose reconstruction. ■

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Acquired reactive perforating collagenosis induced by indinavir in 2 patients with HIV disease

Reactive perforating collagenosis (RPC) is a rare skin disorder in which altered collagen bundles of the superficial dermis are extruded through the overlying epidermis. It is clinically characterized by umbilicated pruritic papules or nodules with a central keratotic plug [1]. The disease can arise at any age. When it begins in childhood and in a familial setting, an autosomal inheritance has been suggested [1]. In adult patients with no family history, RPC has often been associated with chronic diseases, mainly diabetes or kidney failure [1-3]. The course of the disease is variable. Lesions may regress spontaneously in a few weeks, leaving hypopigmented areas and minor scars, or persist chronically for years. The clinical differential diagnosis includes Kyrle's disease, perforating folliculitis, elastosis perforans serpiginosa, perforating granuloma annulare and perforating osteoma [1-3]. We report two AIDS patients in whom RPC developed shortly after they started highly active antiretroviral therapy (HAART), which included indinavir as protease inhibitor.

A 40-year-old HIV positive man (stage IIIC) came to our observation in June 2002 complaining of widespread pruritic, ulcerated papules (*figure 1A*). The lesions appeared concurrently with diffuse xerosis, about three weeks after the patient started a treatment comprising indinavir, stavudine, and zidovudine. A skin biopsy showed a cup-shaped depression in the epidermis, containing necrotic debris and a mixed chronic inflammatory infiltrate. In the dermis, basophilic collagen bundles, surrounded by a mixed chronic inflammatory infiltrate, were oriented perpendicularly to the surface and perforated the epidermis (*figure 1B*). Glycemia was normal, as was glycosylated haemoglobin. The lesions had been treated with topical corticosteroids and UVB-NB for one month with poor results. As the patient had previously taken zidovudine and stavudine with no cutaneous problems, indinavir was suspected and replaced with nelfinavir. Shortly afterwards, the lesions began to regress and completely cleared 3 months later, leaving atrophic scars.

A 29-year-old man, diagnosed with AIDS in 1997, presented with multiple, ulcerated, painful papules on the 4 limbs (*figure 1C*). The patient's medical record was unremarkable: no history of diabetes or renal disease. He had already taken stavudine and lamivudine with no cutaneous side effects. Five weeks after starting combined therapy with indinavir, stavudine and lamivudine, he developed diffuse xerosis, followed by excoriated, pruritic papules. Histology was diagnostic for RPC (*figure 1D*). Indinavir was discontinued: 4 months later, most lesions had healed with slight atrophic scars.

Acquired RPC is a rare dermatosis that usually occurs in patients suffering from diabetes mellitus or chronic kidney