cellular migration) in epidermal keratinocytes following treatment with C225. This is related to a change in the in vivo regulation of follicular and epidermal homeostasis mediated by EGFR, leading to the appearance of the acneiform eruption.[5,12,13]

Cultures fail to show an infectious agent as the cause of the eruption, a factor which supports the diagnosis.[3-5,8,9] The histopathologic study of the lesions shows a follicular reaction consisting of an intense neutrophilic inflammatory infiltrate surrounding the infundibuli,[5,12] which sometimes appear hyperkeratotic.[6] A differential diagnosis should be established with rapid-onset follicular eruptions, whether they are established entities or drug reactions; among the most common drug reactions are those produced by vitamin B_{12}, corticosteroids, androgens, lithium, tuberculostatic drugs, halogens, some tricyclic antidepressants, anticonvulsive drugs, and immunosuppressors. The eruption usually responds to tetracyclines such as minocycline at 100 mg/d, or doxycycline at the same dose. Currently no consensus exists as to the duration of treatment.[3,9,10,14] Recurrence is relatively frequent, although less intense than the initial episode. In view of the increased use of biological therapy in a variety of specialties, we can expect to see this entity with increased frequency in everyday dermatological practice. Dermatologists should therefore consider it as soon as the patient's history is taken, thus avoiding invasive tests such as biopsy in many cases. Tetracyclines are currently the treatment of choice and there have been few reports of therapeutic success with alternatives such as metronidazole, used in the present case, or other systemic antibiotics.

References

1. Reynolds NA, Wagstaff AJ. Cetuximab: in the treatment of metastatic colorectal cancer. Drugs. 2004;64: 109-18.
2. Molinari E, De Quatrebarbes J, Andre T, Aractingi S. Cetuximab-induced acne. Dermatology. 2005;211:330-3.
3. Herrera-Acosta E, Martín-Ezquerra G, Iglesias M, Umbert P. Erupción acneiforme secundaria a cetuximab. Actas Dermosifiliogr. 2005;96:252-4.
4. Fernández-Galar M, España A, López-Picazo JM. Acneiform lesions secondary to ZD1839, an inhibitor of the epidermal growth factor receptor. Clin Exp Dermatol. 2004;29:138-40.
5. Jacot W, Bessis D, Jorda E, Ychou M, Fabbro M, Pujol JL, et al. Acneiform eruption induced by epidermal growth factor receptor inhibitors in patients with solid tumors. Br J Dermatol. 2004; 151:238-41.
6. Busam KJ, Capodieci P, Motzer R, Kiehn T, Phelan D, Halpern AC. Cutaneous side-effects in patients treated with the antiepidermal growth factor receptor antibody C225. Br J Dermatol. 2001;144:1169-76.
7. Segaert S, Van Cutsem E. Clinical signs, pathophysiology and management of skin toxicity during therapy with epidermal growth factor receptor inhibitors. Ann Oncol. 2005;16: 1425-33.
8. Robert C, Soria JC, Spatz A, Le Cesne A, Malka D, Pautier P, et al. Cutaneous side-effects of kinase inhibitors and blocking antibodies. Lancet Oncol. 2005;6:491-500.
9. Lenz HJ. Anti-EGFR mechanism of action: antitumor effect and underlying cause of adverse events. Oncology. 2006;20: 5-13.
10. Van Doorn R, Kirtschig G, Scheffer E, Stoof TJ, Giaccone G. Follicular and epidermal alterations in patients treated with ZD1839 (Iressa), an inhibitor of the epidermal growth factor receptor. Br J Dermatol. 2002;147:598-601.
11. Lee MW, Seo CW, Kim SW, Yang HJ,Lee HW, Choi JH, et al. Cutaneous side effects in non-small cell lung cancer patients treated with Iressa (ZD1839), an inhibitor of epidermal growth factor receptor. Acta Derm Venereol. 2004;84:23-6.
12. Kimyai-Asadi A. Follicular toxic effects of chimeric anti-epidermal growth factor receptor antibody cetuximab used to treat human solid tumors. Arch Dermatol. 2002;138:129-31.
13. Hansen LA, Alexander N, Hogan ME, Sundberg JP, Dlugosz A, Threadgill DW, et al. Genetically null mice reveal a central role for epidermal growth factor receptor in the differentiation of the hair follicle and normal hair development. Am J Pathol. 1997; 150:1959-75.
14. Shah NT, Kris MG, Pao W, Tyson LB, Pizzo BM, Heinemann MH, et al. Practical management of patients with non-small-cell lung cancer treated with gefitinib. J Clin Oncol. 2005; 23:165-74.

Solitary Congenital Plaque-Like Telangiectatic Glomangioma

B Monteagudo,[a] C de las Heras,[a] L Requena,[b] and M Ginarte[c]

[a]Servicio de Dermatología, Complejo Hospitalario Arquitecto Marcide-Novoa Santos, Ferrol, Spain
[b]Servicio de Dermatología, Fundación Jiménez Díaz, Universidad Autónoma, Madrid, Spain
[c]Servicio de Dermatología, Complejo Hospitalario Universitario, Facultad de Medicina, Santiago de Compostela, Spain

To the Editor:

Glomus tumors comprise a group of relatively rare neoplasms. They may be either solitary or multiple. The latter constitute less than 10% of all cases, and in the traditional classification they were divided into disseminated and localized forms.[1,2] In 1990, another form, called congenital multiple plaque-like glomus tumors, was described by Landthaler et al.[3] Subsequently, in 1998, Requena et al[4] described a rare variety

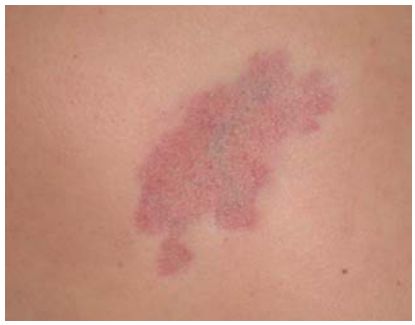

Figure 1. Telangiectatic lesion with plaque-like morphology located on the left scapula.

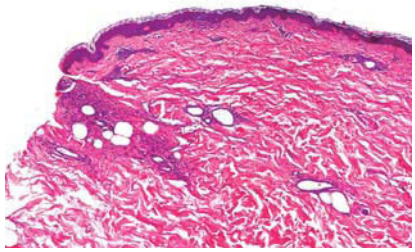

Figure 2. Dilated vascular spaces in the reticular dermis. (Hematoxylin-eosin, ×40.)

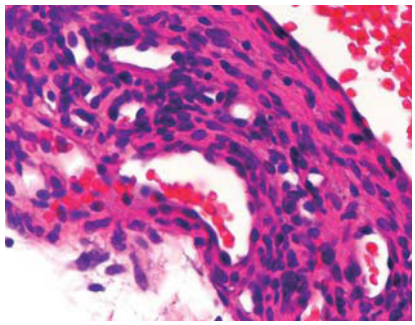

Figure 3. Vascular spaces filled with red blood cells surrounded by endothelial cells and several layers of glomus cells. (Hematoxylin-eosin, ×400.)

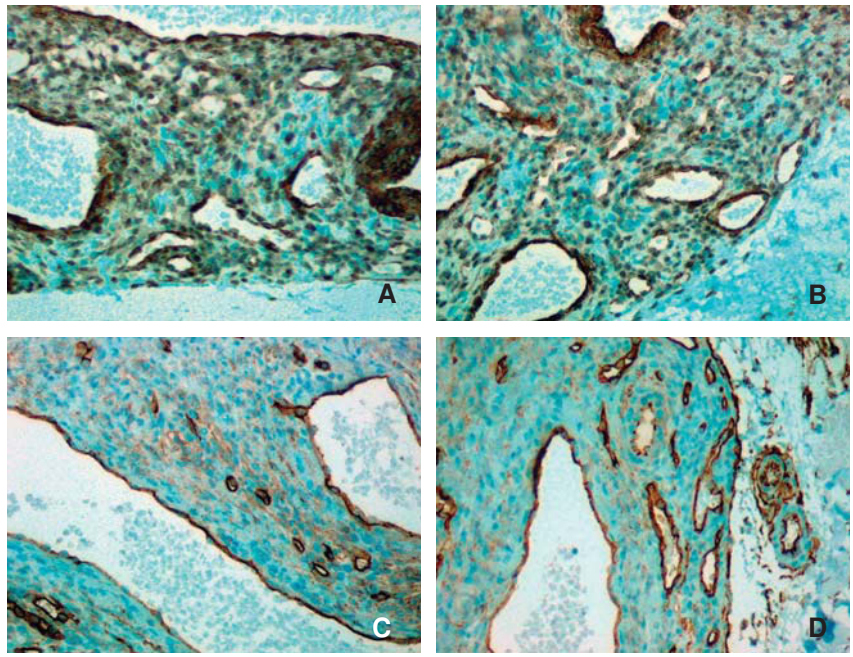

Figure 4. Immunohistochemical characteristics of glomus cells. (A) Positive for smooth muscle α-actin (×200). (B) Expression of vimentin (×200). (C) Negative immunostaining for desmin (×200). (D) Positive for CD34 in the endothelial cells covering the vascular spaces, but negative in the glomus cells (×200).

of solitary glomangioma consisting of an acquired telangiectatic plaque on a woman's shoulder. We present a case of solitary congenital plaque-like glomangioma with a telangiectatic surface recently seen in our department.

A 41-year-old woman with a history of lichen sclerosus in the genital area and cholecystectomy was referred to our department for evaluation of an asymptomatic lesion on the left scapula that had been present since birth. The patient reported that the size of the lesion had increased proportionally as she grew, without further change except for a slight "sinking" in the previous 4 years. She reported no family history of similar lesions.

Physical examination revealed a well-circumscribed telangiectatic plaque located on the left scapula; the plaque was 9 × 4 cm in diameter and depressed with respect to the adjacent skin (Figure 1). No other skin lesions were present.

An x-ray of the left scapula showed no significant changes, and a biopsy of the lesion was performed. The histopathologic study showed vessels in the reticular dermis that were dilated (Figure 2), with endothelial cells surrounding the lumen that were in turn covered by several layers of glomus cells (Figure 3). In the immunohistochemical study, the glomus cells were positive for smooth muscle α-actin and vimentin, and negative for expression of desmin and CD34 (Figure 4).

Glomus tumors are divided into 2 clinicopathological variants, solitary and multiple, which have different distribution patterns, gross morphology, and histopathology.

Solitary glomus tumors are the most common variety and often cause a sharp pain in response to pressure or exposure to cold. They usually appear in adulthood on the distal parts of the limbs, especially in the nail beds. Histopathology shows a well-circumscribed nodule composed of groups of glomus cells surrounding small vascular spaces covered with endothelial cells.[1,2]

Multiple glomus tumors, also called glomangiomas because of their angiomatous appearance or glomuvenous malformations, appear earlier, are usually painless, and are generally inherited as an autosomal dominant disease. In general, these lesions are less well demarcated and have far fewer glomus cells than solitary glomus tumors.[1,2,5] This variant is divided into 3 types: (1) multiple disseminated glomangiomas, characterized by lesions

distributed over the entire skin surface[6,7]; (2) localized multiple glomangiomas, in which glomus tumors are grouped together and limited to 1 region, for example a limb[8]; and (3) plaque-like congenital glomangioma, the rarest type of glomus tumor.[3,9,10]

The term congenital plaque-like glomangioma was coined in 1990 by Landthaler et al[3] in their description of poorly demarcated multiple plaques similar to hematomas located on the shoulders of 2 children. Subsequently, other cases were reported consisting of multiple bluish or reddish nodules grouped into 1 or several plaques, or in clusters of discrete nodules in a particular region of the body, which in some cases presented clinically with a morphology of venous malformation.[9,10] Plaque-like glomangioma is present from birth and may be painful. The lesions are generally flat at birth, pink or bluish in color, and increase in size as the child grows. During puberty, satellite lesions may appear at a distance from the initial lesion. There are descriptions of familial cases of autosomal dominant inheritance with incomplete penetrance and variable expressivity, in which family members have minor lesions. This should be differentiated from tufted angioma, venous malformations, or congenital plaque-like blue nevus[9,10].

In 1998, Requena et al[4] described a solitary plaque-like telangiectatic glomangioma, an entity distinct from congenital plaque-like glomangioma since it was solitary and acquired, with a telangiectatic surface. Our case substantially resembles this one (the patient's female sex and the localization, telangiectatic surface, and slight depression of the lesion), but differs from it in its congenital nature, and for this reason we consider it to be a solitary congenital plaque-like telangiectatic glomangioma.

References

1. Requena L, Sangueza OP. Cutaneous vascular proliferation. Part II. Hyperplasias and benign neoplasms. J Am Acad Dermatol. 1997;37:887-919; quiz 920-2.
2. Requena L, Requena C, Pichardo RO, Sangueza OP. Tumores glómicos. Monogr Dermatol. 2004;17:91-9.
3. Landthaler M, Braun-Falco O, Eckert F, Stolz W, Dorn M, Wolff HH. Congenital multiple plaquelike glomus tumors. Arch Dermatol. 1990;126: 1203-7.
4. Requena L, Galván C, Sánchez Yus E, Sangueza O, Kutzner H, Furio V. Solitary plaque-like telangiectatic glomangioma. Br J Dermatol. 1998;139: 902-5.
5. Boon LM, Mulliken JB, Enjolras O, Vikkula M. Glomuvenous malformation (glomangioma) and venous malformation: distinct clinicopathologic and genetic entities. Arch Dermatol. 2004;140:971-6.
6. Blume-Peytavi U, Adler YD, Geilen CC, Ahmad W, Christiano A, Goerdt S, et al. Multiple familial cutaneous glomangioma: a pedigree of 4 generations and critical analysis of histologic and genetic differences of glomus tumors. J Am Acad Dermatol. 2000;42: 633-9.
7. Requena Caballero L, Requena Caballero C, Sánchez López M, Vázquez López F, Coca Menchero S, Sánchez Yus E, et al. Glomangiomas múltiples hereditarios. Actas Dermosifiliogr. 1987;78:245-7.
8. Monteagudo B, León A, Durana C, de las Heras C, Used MM, Álvarez JC, et al. Manifestación segmentaria tipo 1 de glomangiomas múltiples. Actas Dermosifiliogr. 2006;97:358-9.
9. Mallory SB, Enjolras O, Boon LM, Rogers E, Berk DR, Blei F, et al. Congenital plaque-type glomuvenous malformations presenting in childhood. Arch Dermatol. 2006;142:892-6.
10. Glick SA, Markstein EA, Herreid P. Congenital glomangioma: case report and review of the world literature. Pediatr Dermatol. 1995;12:242-4.

Angiolipomas and Antiretroviral Therapy

A Ramírez-Santos, M Ginarte, and J Toribio

Servicio de Dermatología, Complejo Hospitalario Universitario, Facultad de Medicina, Santiago de Compostela, Spain

To the Editor:

In recent years, an association has been described between the use of protease inhibitors for antiretroviral therapy and the appearance of angiolipomas and lipomas, as well as with an increase in the number and size of those already present.[1-3]

A 40-year-old man consulted in 2005 for lesions that first appeared in 1998, and that had increased in number and size since then. Some were tender to pressure or spontaneously painful, while others were asymptomatic. The patient, a former intravenous drug user, tested positive for hepatitis B, C, and D, and for the human immunodeficiency virus (HIV). He had started antiretroviral treatment with lamivudine, zidovudine, and indinavir in 1998, and in 2001 indinavir was substituted with nelfinavir; however, the lesions continued to appear. He reported no family or personal history of similar lesions.

Physical examination revealed numerous subcutaneous tumors on the upper limbs and, to a lesser extent, on the trunk and lower limbs. These were clearly circumscribed, firm, and, in some instances, painful to the touch (Figure 1).