The FS or auriculotemporal syndrome or gustatory sweating results from the salivary stimulus during and immediately after eating, seeing, thinking about, or talking about certain foods. [2,3]

The hypothesis is that, after a parotid gland trauma, a lesion would occur to the auriculotemporal branch of the trigeminal nerve, followed by an anomalous and aberrant regeneration of nerve fibers, with the anastomosis of the parasympathetic fibers with sympathetic fibers of the subcutaneous sweat glands and surface blood vessels.[4] Consequently, not only is the salivary reflex stimulated during chewing, but also the production of sweat and the cutaneous vasodilation of the affected region.[5]

The symptoms generally arise about six months after the parotid gland trauma, the time necessary for the regeneration of the damaged nerve, but there are reports of medical conditions that began up to 14 years after the traumatic event.[4]

The incidence described for FS after parotidectomy is quite variable and depends on the criteria used to reach this diagnosis. One subjective incidence (based on the perception of the patient's symptoms) was identified between 12.5% and 62%, while an objective incidence (verified by the Minor test) was found between 22% and 98%.[3]

FS treatment can be challenging and involves clinical and surgical options. Some patients who complain of discomfort due to sweating can be benefitted by the use of topical antiperspirants applied to the affected area, such as aluminum chloride.[2]

Autologous fat grafts, temporoparietal fascia grafts, muscle flaps, and the use of artificial tissues are example of surgical techniques used in both the prevention and treatment of FS, whose objective is to construct a barrier between the skin and the auriculotemporal nerve in order to avoid anomalous regeneration. [3]

Botulinum toxin type A was proposed as a treatment of FS in 1995, and seeks to block the pre-synaptic release of acetylcholine in the neuromuscular and neuroglandular joint,[2] in turn provoking a chemical denervation. As an advantage, this is characterized as being a relatively non-invasive therapeutic measure that is safe, effective, and long-lasting. [1-3]

In general, the results of botulinum toxin type A for sweating are more prolonged than those obtained in treatments that focus on the reduction of muscular actions. In practice, the successive treatment with the toxin seems to promote a reduction in the severity of the symptoms and the extension of the treated area, as well as space out the period between recurrences. One possible explanation would be the atrophy of the eccrine glands, inhibited for long periods of time.[2,5]

Disadvantages that may occur include: dry mouth, weakening of the facial muscles, eyelid ptosis, facial paralysis, as well as short-term local reactions of pain, edema, erythema, and ecchymoses. Allergic reactions and the development of resistance to botulinum toxin type A can occur, and in these cases, the use of botulinum toxin type B would be a plausible alternative.[1,2] ❑

REFERENCES

1. Xie S, Wang K, Xu T, Guo XS, Shan XF, Cai ZG. Eficacy and safety of botulinum toxin type A for treatment of Frey's syndrome: evidence from 22 published articles. Cancer Med. 2015;4:1639-50.
2. Lopes T, Barroso L, Mesquita M, Bitoque S, Lopez D, Ferreira A. Síndrome de Frey - uma visão global do tema. Rev Port Estomal Med Dent Cir Maxilofac. 2013;54:103-14.
3. Hayashi A, Mochizuki M, Suda S, Natori Y, Ando E, Yoshizawa H, et al. Effectiveness of platysma muscle flap in preventing Frey syndrome and depressive deformities after parotidectomy. J Plast Reconstr Aesthet Surg. 2016;69:663-72.
4. Santos RC, Chagas JFS, Bezerra TFP, Baptistella JE, Pagani MA, Melo AR. Prevalência da Síndrome de Frey após parotidectomia parcial. Rev Bras Otorrinolaringol. 2006;72:112-5.
5. Martos Díaz P, Bances del Castillo R, Mancha de la Plata M, Naval Gías L, Martínez Nieto C, Lee GY, et al. Clinical results in the management of Frey's syndrome with injections of Botulinum toxin. Med Oral Patol Oral Cir Bucal. 2008;13:E248-52.

Mailing address:
Gustavo Vieira Gualberto
Avenida Álvares Cabral 344, sala 1410
Lourdes
30170-001 Belo Horizonte, MG.
Brazil
E-mail: gustavovieiragualberto@yahoo.com.br

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Pseudo "fringe sign" in frontal fibrosing alopecia*

Leticia Arsie Contin[1] Vanessa Barreto Rocha[1]

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Dear Editor,

Since its description in 1994 by Kossard, frontal fibrosing alopecia (FFA) has been intensively studied, with new features described at every moment.[1-4]

Recently, Pirmez *et al.* described the pseudo "fringe sign", an atypical presentation of the disease, which resembled traction alopecia (TA).[5] However, the patients presented with features of FFA, scarring alopecia and facial papules or lichen planus pigmentosum, as well as loss of eyebrows and body hairs. The true "fringe sign" is described in TA, in which the hairs of the implantation region are spared after traction. In principle, this difference would help in the differential diagnosis between TA and FFA.

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[1] Trichology ambulatory at Dermatology department, Hospital do Servidor Público Municipal de São Paulo (HSPM) – São Paulo (SP), Brazil

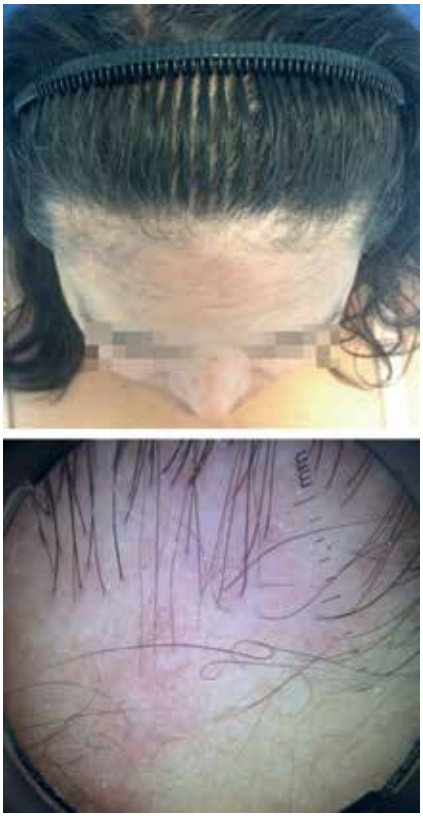

Figure 1: Clinical case 1 - Plaques with alopecia in the frontal region sparing the implantation hairline. Dermoscopy of the hairline evidencing the presence of vellus hair in the anterior region, absence of hairs with central erythema and posterior terminal hairline with discrete follicular hyperkeratosis

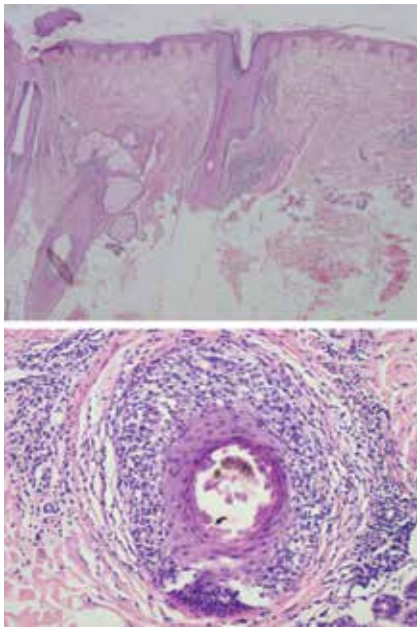

Figure 2: Vertical cut revealing lymphomononuclear and perifollicular inflammatory cell infiltration at the protuberance and infundibulum levels (Hematoxylin & eosin, X10). Horizontal cut showing lichenoid and lymphomononuclear inflammatory cell infiltration attacking the hair follicle (Hematoxylin & eosin, X100)

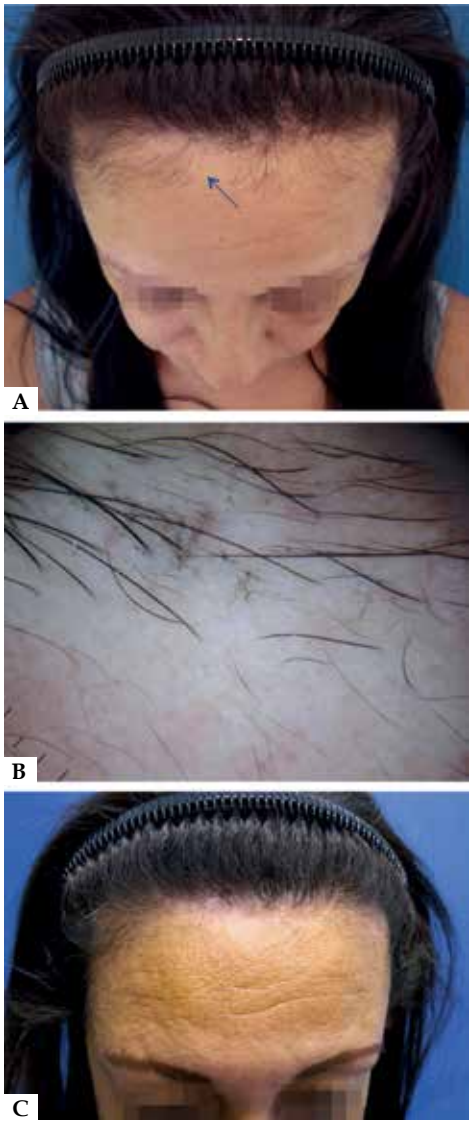

Figure 3: Clinical case 2 - Plaques with alopecia in the frontal region sparing the implantation hairline (**A**, arrow) and rarefaction of the eyebrows (**B**). Evolution of this case with increased alopecia areas two years later, already affecting the implantation line (**C**)

We describe here two cases of patients with FFA presented with pseudo "fringe sign".

The first case was a 54-year-old female patient with hair thinning in the frontotemporal region for about one year, with retained hairs along the frontal implantation line (Figure 1). She denied hair pulling. There was no superciliary alopecia at first, but the condition progressed to the loss of eyebrows and body hair. After a biopsy, we reached the clinical and histological diagnosis of FFA (Figure 2). Considering that lichen planopilaris (LPP) and FFA have indistinguishable histological features, the clinical evolution favored FFA.

The second case was a 46-year-old female patient with complaints of frontal hair loss for about one year and loss of eyebrows

hair in the last two years, as well as facial skin-colored papules for about six months (Figure 3). The patient had recently noticed a progressive decrease of axillary hairs. At the initial examination, we observed thinning eyebrows and alopecia in the frontal region, with sparing of the implantation hairline. Frontal biopsy was compatible with FFA, corroborating our clinical diagnosis. Figure 3C shows the evolution of the clinical features, compatible with FFA.

As already reported by Pirmez *et al*., although the pseudo "fringe sign" can occur in patients with FFA, biopsies show the characteristic pattern of LPP, which may make diagnosis challenging.[5] In the reported cases, the presence of facial papules and thinning eyebrows contributed to the diagnosis of FFA, in detriment of LPP with alopecia plaques. In addition, the loss of vellus hair in the frontal region was not observed initially, but a loss after the frontal implantation line, affecting the terminal hairs of that region. Unlike TA, this fringe slowly becomes more rarefied, eventually leading to some vellus hair loss in the region in a later phase, with a scarring, shiny appearance and absence of follicular ostia to trichoscopy.

A study with a greater number of reported cases of this clinical presentation could help explain this type of manifestation, its etiopathogenic implications, and the immune response involved, which would help in therapeutic decisions. ❑

REFERENCES

1. Kossard S. Postmenopausal frontal fibrosing alopecia. Scarring alopecia in a pattern distribution. Arch Dermatol. 1994;130:770-4.
2. Donati A, Molina L, Doche I, Valente NS, Romiti R. Facial papules in frontal fibrosing alopecia: evidence of vellus follicle involvement. Arch Dermatol. 2011;147:1424-7.
3. Pirmez R, Donati A, Valente NS, Sodré CT, Tosti A. Glabellar red dots in frontal fibrosing alopecia: a further clinical sign of vellus follicle involvement. Br J Dermatol. 2014;170:745-6.
4. Pirmez R, Duque-Estrada B, Donati A, Campos-do-Carmo G, Valente NS, Romiti R, et al. Clinical and dermoscopic features of lichen planus pigmentosus in 37 patients with frontal fibrosing alopecia. Br J Dermatol. 2016;175:1387-1390.
5. Pirmez R, Duque-Estrada B, Abraham LS, Pinto GM, de Farias DC, Kelly Y, et al. It's not all traction: the pseudo 'fringe sign' in frontal fibrosing alopecia. Br J Dermatol. 2015;173:1336-8.

Mailing address:
Leticia Arsie Contin
Rua Castro Alves, 60
Aclimação
01532-000 São Paulo, SP – Brazil
E-mail: lecontin@hotmail.com

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Dapsone-induced agranulocytosis in patients with Hansen's disease*

Tania Rita Moreno de Oliveira Fernandes[1,2]
Bruno Nascimento de Jesus[3]
Tathyane Trajano Barreto[3]
Anderson de Almeida Pereira[3]

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Dear Editor,

Agranulocytosis induced by sulphonamide or dapsone (44-diaminodiphenylsulphone – DDS) is characterized by a low concentration or absence of granulocytes due to sulfone cytotoxicity effects on bone marrow and mononuclear cells.[1]

DDS is a structural analogue of para-aminobenzoic acid (PABA) that acts as a competitive inhibitor of the enzyme dihydropteroate synthase in the folate pathway. It has anti-inflammatory, antibacterial, antiprotozoal, and antifungal activities. Used since 1943 to treat leprosy, it is also indicated for the treatment of malaria, rheumatoid arthritis, granuloma annulare, dermatitis herpetiformis, and other vesiculobullous diseases. DDS adverse effects include hemolytic anemia, methemoglobinemia, gastritis, headache, agranulocytosis, hepatitis, peripheral neuropathy, nephrotic syndrome, dapsone syndrome, among others.[1,2]

DDS is part of the multidrug therapy (MDT) used to treat leprosy. The regimen is a combination of rifampicin (supervised monthly dose of 600mg) and dapsone (supervised monthly dose of 100mg and 100mg/daily) for paucibacillary patients, with the addition of clofazimine (supervised monthly dose of 300mg and 50mg/daily) for multibacillar patients.[2]

We report a 61-year-old Caucasian female patient, resident in Juazeiro, state of Bahia, Brazil, complaining of a spot on the right elbow, which appeared 1 year before. Physical examination revealed a single hypochromic patch, approximately 1cm in diameter, with micropapular edges and absent thermal sensitivity. With a diagnosis of tuberculoid leprosy, we started a MDT regimen for paucibacillary leprosy. At day 14 after the first administration, the patient presented with adynamia, exertional dyspnea, normochromic normocyt-

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[1] Department of Medical and Surgical Pathology of the Integumentary System at Universidade Federal do Vale do São Francisco (Univasf) – Petrolina (PE), Brazil
[2] Leprosy and Tuberculosis Reference Center in Juazeiro (BA), Brazil
[3] Medical Student at Universidade Federal do Vale do São Francisco (Univasf) – Petrolina (PE), Brazil