Biopsies of Facial Dermatoses Made Simple

Faisal Al-Mohammedi, MD, FRCPC; Richard I. Crawford, MD, FRCPC; Magdalena Martinka, MD, FRCPC

• ***Context.***—**Biopsy of the face is rarely done for inflammatory skin diseases, unless the entire process is confined to the face.**

Objective.—**We hypothesized that facial dermatitis has a differential diagnosis that is more limited than the differential diagnosis of inflammatory skin diseases that affect other parts of the body. To our knowledge, the classification of inflammatory skin diseases occurring on the face has never been conducted before in the English literature.**

Design.—**The most-recent 100 facial biopsies of inflammatory skin conditions were retrieved from our files, and the cases were categorized into the main inflammatory skin patterns.**

Results.—**Forty-seven cases (47%) were categorized as interface dermatitis, 2 cases (2%) as psoriasiform dermatitis, 11 cases (11%) as spongiotic dermatitis, 16 cases (16%) as diffuse and nodular dermatitis, 8 cases (8%) as perivascular dermatitis, 14 cases (14%) as folliculitis and perifolliculitis, 1 case (1%) as panniculitis, and 1 case (1%) as fibrosing dermatitis. The number of diagnostic entities represented within each of these patterns was small.**

Conclusions.—**We believe that facial dermatitis should have its own more-circumscribed differential diagnosis. From a practical viewpoint, many of the inflammatory skin diseases that affect other parts of the body should be excluded from the differential diagnosis after the tissue is determined to be from a facial skin biopsy, and others should not be considered unless the biopsy is from the face.**

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Physicians and scientists interested in the skin have microscopically examined every visible abnormality on our exterior for more than a century, before surgical pathology emerged as a medical specialty and before endoscopic techniques facilitated histopathologic examination of other organ systems. This has resulted in a plethora of clinicopathologic correlation, and the result has been the clinicopathologic definition of literally hundreds of nonneoplastic skin conditions. Therefore, the critical task in assisting pathologists in practicing the dermatopathology of inflammatory skin conditions largely consists of prioritizing that wealth of information, rather than presenting recent discoveries.

There is a particular opportunity for the distillation of information when biopsies of facial dermatoses are considered. Most patients affected by an inflammatory dermatosis do not have facial involvement, and sites other than the face are chosen for biopsy. Of those that do occur on the face, most occur synchronously elsewhere on the body. Patients and clinicians prefer that a site other than the face be biopsied whenever possible; therefore, facial biopsies usually come from dermatoses that are confined to the face at the time they present. Therefore, it was our hypothesis that, of the hundreds of inflammatory dermatoses, only a few would regularly appear in facial biopsies, and an analysis of the most recent 100 facial dermatoses submitted to our dermatopathology practice would support that hypothesis.

MATERIALS AND METHODS

The most recent 100 facial biopsies of inflammatory skin conditions were retrieved from our files, and the cases were categorized into the main inflammatory skin patterns.

RESULTS

Forty-seven of the 100 patients (47%) had interface changes (Table). Of those forty-seven patients, 27 (54%) had benign lichenoid keratosis, 17 (36%) had lupus erythematosus, and 3 (6%) had lichen planus. Two of the 100 patients (2%) had psoriasiform dermatitis. Eleven patients (11%) had eczematous dermatitis. Eighteen patients (18%) had nodular and diffuse dermatitis divided as follows: 10 of the 18 (56%) were consistent with foreign body granulomatous reaction; 3 (17%), lymphoid hyperplasia; 2 (11%), granuloma faciale; 1 (6%), sarcoidosis; 1 (6%), orofacial granulomatosis; and 1 (6%), Sweet syndrome. Six of the 100 patients (6%) had perivascular dermatitis divided as follows: 3 of the 6 (50%), postinflammatory pigmentary alteration; 2 (33%), superficial perivascular lymphocytic infiltrate, and 1 (17%), superficial and deep perivascular lymphocytic infiltrate. Fourteen of the 100 patients (14%) had follicular and perifollicular dermatitis divided as follows: 9 of the 14 patients (64%) had rosacea, and 5 of the 14 patients (36%) had folliculitis. One patient of the 100 (1%) had panniculitis compatible with lupus panniculitis, and 1 patient (1%) had fibrosing dermatitis compatible with linear scleroderma.

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From the Department of Pathology, University of Toronto, Toronto, Ontario, Canada (Dr Al-Mohammedi); and the Department of Pathology, University of British Columbia, Vancouver, Canada (Drs Crawford and Martinka).

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Reprints: Faisal Al-Mohammedi, MD, FRCPC, Department of Pathology, University of Toronto, 22 Greystone Court, Toronto, ON M8V4A5, Canada (e-mail:fahmd2002@hotmail.com).

Inflamatory Skin Conditions Found in 100 Facial Skin Biopsies		
Pattern of Inflammation	**Cases, No.**	**Diagnosis, No.**
Interface dermatitis	47	Benign lichenoid keratosis, 27 Lupus erythematosus, 17 Lichen planus, 3
Psoriasiform dermatitis	2	Psoriasis versus chronic eczematous dermatitis
Spongiotic dermatitis	11	Eczematous dermatitis
Nodular and diffuse dermatitis	18	Consistent with foreign body granulomatous reaction, 10 Lymphoid hyperplasia, 3 Granuloma faciale, 2 Sarcoidosis, 1 Orofacial granulomatosis, 1 Consistent with Sweet syndrome, 1
Perivascular dermatitis	6	Postinflammatory pigmentary alteration, 3 Superficial perivascular lymphocytic infiltrate, 2 Superficial and deep perivascular lymphocytic infiltrate, 1
Follicular and perifollicular dermatitis	14	Rosacea, 9 Folliculitis, 5
Panniculitis	1	Lupus panniculitis
Fibrosing dermatitis	1	Linear scleroderma

COMMENT

Interface Dermatitis

Approximately one-half of the biopsies of facial inflammatory dermatoses (47%) showed an interface pattern. The principal differential diagnosis in this category lies between lupus erythematosus and lichenoid keratosis. Although lichen planus and lupus are usually readily distinguishable from one another, lichenoid keratosis has intermediate features and can be more difficult to distinguish from lupus, requiring the evaluation of multiple criteria.[1–5] The presence of residual solar lentigo or seborrheic keratosis can be still seen around lichenoid keratosis.[2] Lichenoid keratosis is usually sampled because, clinically, it can mimic basal cell carcinoma or Bowen disease.[1] Careful examination of the tissue section should be conducted to exclude lichenoid actinic keratosis[6] and melanocytic neoplasms that have halo features.[7]

Follicular Inflammation

The most frequently biopsied cause of facial follicular inflammation is rosacea. Rosacea often presents in the middle-aged or elderly patient and is commonly biopsied because it can sometimes be difficult to distinguish clinically from lupus.[8–10] Histologically, rosacea demonstrates intrafollicular and perifollicular lymphocytic inflammation with or without granulomas (*granulomatous rosacea*) or neutrophils (*pustular rosacea*). The perifollicular granulomatous pattern is quite specific for rosacea, the other patterns, less so. Unlike lupus, in rosacea, the intrafollicular inflammation is spongiotic, rather than interface; there is no dermoepidermal inflammation; and there is no excess dermal mucin.[5,11,12] *Perioral dermatitis* is a clinical variant of rosacea with indistinguishable histology but with a perioral or periocular location, distinct triggers, and a high likelihood of cure.[13–15]

A pustular folliculitis affecting the face can be caused by bacteria (principally *Staphylococcus*), by dermatophyte fungi, by acne, and occasionally by rosacea. Gram and periodic acid–Schiff and diastase stains can be helpful in identifying an infectious cause if the lumen of the inflamed follicle can be captured in the specially stained section.[8,16–18]

The histology of follicular mucinosis differs from that of rosacea by the accumulation of mucin within empty spaces between follicular epithelial cells. Unlike most other examples of epithelial mucin, the mucin in follicular mucinosis is acidic and is best identified by Alcian blue or colloidal iron staining.[19]

The most common cause of follicular inflammation occurring on the face is, in fact, acne, but acne is rarely biopsied because it is readily identified clinically. In the setting of follicular inflammation, the histologic hallmark of acne is the dilation of the inflamed follicle by a plug of keratin and sebum, although that plug can become destroyed in intensely inflammatory lesions.[18]

Nodular Inflammation

The most common cause of a nodular inflammatory infiltrate on facial skin is a foreign body reaction, mainly secondary to a ruptured epidermoid cyst. The infiltrate is typically polymorphous and contains all inflammatory cell types, including foreign-body–type giant cells. If present, flakes of keratin or remnants of follicular epithelium in the center of the inflammatory nodule allows for a secure diagnosis.

Less frequently, a nodular infiltrate on facial skin will consist almost entirely of lymphocytes, raising the differential diagnosis of cutaneous lymphoid hyperplasia versus low-grade lymphoma. The principal lymphoma showing this pattern of infiltrate on facial skin is marginal-zone lymphoma. Similar histologic, immunophenotypic, and molecular genetic criteria can be used to identify marginal-zone lymphoma in the skin as used in other sites.[20,21]

Spongiotic Dermatitis—Eczema

On sites other than the face, many dermatoses show the histology of spongiotic dermatitis: pityriasis rosea, erythema annulare centrifugum, Gianotti-Crosti syndrome, and small-plaque parapsoriasis. Fortunately, these conditions do not occur on the face, and on the face, spongiotic dermatitis can be assumed to indicate an eczematous process. When faced with an eczematous process, the clinician needs to consider external causes (irritant contact and allergic contact dermatitis) and endogenous predisposition (atopic dermatitis). On any body site, it is necessary to use a periodic acid–Schiff stain to rule out dermatophyte infection as a cause for spongiotic dermatitis. Tinea faciei seems to represent less than 1% of facial dermatoses, but missing the diagnosis is to be avoided

because inappropriate treatment of tinea greatly magnifies the extent of the disease. Therefore, the face is no exception to the indication for a periodic acid–Schiff and diastase stain in spongiotic dermatitis.[1,2]

Psoriasiform Dermatoses—Psoriasis/Seborrheic Dermatitis

Many psoriasiform dermatoses, such as erythroderma, pityriasis rubra pilaris, acrokeratosis paraneoplastica, and the nutritional deficiency dermatitides, always affect other body sites when they affect the face, and thus, they do not appear in facial biopsies. A psoriasiform dermatosis strictly localized to the face represents psoriasis or seborrheic dermatitis. Psoriasis and seborrheic dermatitis cannot be distinguished from one another histologically on the face, and many dermatologists argue that they, in fact, represent the same disease. Tinea faciei can induce a psoriasiform as well as a spongiotic reaction pattern, and therefore, a periodic acid–Schiff stain is appropriate in this setting as well.[1,2,22]

Diffuse Dermatitis—Granuloma Faciale

Granuloma faciale is an uncommon facial dermatosis with a distinctive clinical presentation and diagnostic histology, demonstrating a diffuse, polymorphous dermal infiltrate, separated from the epidermis and adnexal epithelium by a grenz zone. The neutrophils and extravasated red cells tend to aggregate around dilated vessels, whereas the lymphocytes, histiocytes, eosinophils, and plasma cells lie farther away from the vessels. The histiocytes do not form granulomas.[23]

The Granulomatous Reaction Pattern

Granulomatous disorders that can arise exclusively to face include sarcoidosis, perioral dermatitis, lupus miliaris disseminatous faciei, infection (mycobacterial and deep fungal), foreign body, Crohn disease, and Melkerson-Rosenthal syndrome.

Granulomatous reaction patterns are usually a clinicopathologic diagnosis after excluding infections and foreign bodies.[1,2]

Postinflammatory Pigmentary Alteration

Postinflammatory hyperpigmentation is represented by melanophages in the superficial dermis. When they are seen, it is important to look for an underlying pattern of epidermal inflammation as the cause. Melanophages that are present deeper than the superficial vascular plexus indicate an alternate cause, such as drug-induced pigmentation or dermal melanocytic proliferation. Melasma ("the mask of pregnancy") is diagnosed clinically and generally, not biopsied but has an indistinguishable histology from postinflammatory hyperpigmentation.[1,2]

Panniculitis

Cold panniculitis in children and, rarely, lupus panniculitis can arise in the face. They are both characterized by lymphocytic lobular panniculitis. No epidermal or dermal changes are usually seen in either case, but lymphocytic aggregates and plasma cells are identified more often with lupus panniculitis.[1,2,24,25]

Fibrosing Dermatitis

Linear morphea in the frontoparietal area "en coup de sabre" is the most common fibrosing dermatitis to occur in the face.[26] Radiation dermatitis and lichen sclerosus are rarely seen in facial biopsies.

CONCLUSION

We believe that facial dermatitis should have its own more circumscribed differential diagnosis. From a practical viewpoint, many of the inflammatory skin diseases that affect different parts of the body should be excluded from the differential diagnosis after the material being examined is identified as a facial skin biopsy, and others should not be considered unless the biopsy is from the face. In general, some of the inflammatory dermatosis that are discussed in this article occur exclusively in the face, such as granuloma faciale and orofacial granulomatosis, and the rest can affect only the face but may sometimes affect other parts of the body as well (eg, sarcoidosis, linear scleroderma, among others). Reporting the main histologic pattern and providing a limited differential diagnosis is always a crucial step in patient care and management.

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