CASE REPORT

The importance of histopathologic aspects in the diagnosis of dissecting cellulitis of the scalp

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Abstract
Dissecting cellulitis of the scalp or dissecting folliculitis also known as “perifoliculitis capitis abscedens et suffodiens” (PCAS), is a rare, severe and distinct dermatological disease. It most probably occurs because of follicular occlusion via hyperkeratosis, having the same mechanism of acne conglobata and hidradenitis suppurativa. These dermatoses may be associated or may have an isolated evolution. PCAS is one of the primitive cicatricial alopecias. This disease mainly affects young males, especially the black ones [2, 3].

Keywords: dissecting cellulitis of the scalp, dissecting folliculitis, cicatricial alopecia, acne conglobata.

Introduction

Denominated by Hoffman in 1908 “perifoliculitis capitis abscedens et suffodiens”, dissecting cellulitis of the scalp is a rare dermatosis, with rich and characteristic clinical semiology, with a long evolution and most of the times resistant to the administered therapies [1]. Baylor Hair Research Center considers that PCAS represents 1–2% of the primitive cicatricial alopecias. This disease mainly affects young males, especially the black ones [2, 3].

PCAS may evolve as an isolated disease or within “the follicular occlusion triad”, associated with hidradenitis suppurativa and acne conglobata. All these three diseases have the same pathogenesis and the same histological changes. From the clinical point of view, it is a profound, chronic and recurrent folliculitis, ending in abscesses and sinuous tracts and scars [1, 4].

A long topic in the specialty literature is the “tetrad of follicular occlusion”, the pilonidal disease being considered by many authors as a part of this group of diseases [4].

The main mechanism involved in the pathophysiology of the disease is the follicular hyperkeratosis and not the bacterial colonization, which interferes only secondarily to its evolution. The lack of diagnosis and an incorrect treatment of PCAS lead to permanent cicatricial alopecia, with a major aesthetic lesion and a psychological wrong on the patients. The evolution itself of the disease is long and teasing, being able to determine serious infectious complications.

Patient, Methods and Results

We present a case of a 24-year-old patient, without a significant heredo-collateral history, who was hospitali-
ized in the Clinic of Dermatology Iassy because of cicatrical alopecia lesions and inflammatory nodules, placed at the level of the scalp (vertex and occipital area), associated with face lesions of moderate acne vulgaris.

**History of disease**

The onset of the disease of gradual starting 3.5 years ago with acne conglobata lesions on the face and back thorax. At the scalp level follicular pustular lesions and inflammatory nodules, slightly painful appeared at the level of the vertex. The scalp nodules were initially small and firm, then started growing and became fluctuant, expanding in an effenter way and communicating among them via deep sinusous tracts. Either under pressure or spontaneously, suppuration with blood was coming out via multiple pores.

The healing of the nodular follicular lesions happened slowly with cicatrical alopecia surrounded by “en touffe” folliculitis lesions on the margin. The scalp lesions were slightly painful and the general status was not influenced. The patient came to the dermatologist, who chose the diagnosis of multiple furuncles of the scalp, treated with systemic antibiotics (azithromycin, claritromycin, doxycycline) and incised by the surgeon. The evolution of the lesions was favorable, but with ulterior recurrences and the installation of scalp cicatrical alopecia in the affected area.

The introduction by another dermatologist, at the beginning of 2008, of the systemic treatment with isotretinoin for severe acne, in variable, small doses (10 mg/day, then 40 mg/day and 20 mg/day) and in repeated therapies, had the effect of remission both for the acne lesions and for scalp nodular lesions. However, in short time the scalp lesions appeared again, evolving in outbreaks, with short times of remission, reason for which the patient was hospitalized in December 2008 in the Clinic of Dermatology Iassy, for diagnosis and treatment.

**General clinical examination**

General clinical examination pathologically showed only a bilateral submandibular and right lateral-cervical polyadenopathy, less than 1 cm in diameter, with an elastic consistency. The nodules were not painful both spontaneously and when touched and they were mobile in their deep areas.

**Local examination**

At the scalp level, in the vertex area but also expanded to the occipital area, there are some cicatrical alopecic plaques of small size (1–1.5 cm in diameter) (Figure 1). On their surface some variable-sized inflammatory lesions were visible (Figure 2), some of them hard when touched, others fluctuant. Pus was coming out via multiple pores when pressed. Between the nodules sinusous communication tracts were present, deeply placed. At the margin of the cicatrical area, slightly pruriginous follicular pustules were visible.

The associated subjective symptomatology was reduced in intensity (small pain). At the face level, the tegument was seborrheic, with closed and open comedones, rare papulo-pustules and numerous post-acne scars (Figure 3). On the trunk there were numerous, well-marginated, finely scaly, achromic, hyperchromic and erythematic macules, isolated and confluent in plaques with “geographic map” aspect (typical lesions of tinea versicolor).

**Paraclinical investigations**

The bacteriologic examination of a scalp nodule secretion was negative. The microbiologic drawing was also negative, as well as the Sabouraud medium culture. The HIV serology was also negative. The inflammatory tests (VSH, fibrinogen, C-reactive protein) and the immunologic ones (CIC, IgG, IgA, IgM) were between normal limits, as well as the hemoleukogram, the hepatic tests and the ones for renal function exploring.

Bacteriological culture of the nose yields *Staphylococcus aureus* – a pathogen sensitive for the majority of the anti-staphyloccoci (ciproxoflaxacin, chloramphenicol, neomycin, gentamycin, oxacylin, doxycycline, kanamycin, tobramycin).

A cutaneous biopsy was also made from a recent nodular lesion from the parieto-occipital area.

The histological examination established the certain diagnosis based on the specific optical microscopy elements, correlated to the immunohistochemistry data obtained. The biopsy fragment contained hair with normal morphology, but with reduced numeric density in the anagenic or catagenic stage, with prominent sebaceous follicles. At the level of the infundibular area, a lot of hair contains keratin corks (Figure 4).
The dermo-epidermic junction presents a vacuole degenerative burning point, which associates aspects of exocytosis (Figure 5). In the reticular derma, there is a moderate inflammatory infiltrate mainly lymphomonocytic, which associates rare PMNs (Figure 6) such as a granuloma with macrophages and multinucleate giant cells (perifolkularly placed) (Figures 7 and 8). Part of the hair has the perifollicular fibrosis marked with areas of destruction of the pilosebaceous follicle and replaced with granulation tissue (Figure 9).

The dermis itself has colagenisation, containing dense collagen fibers (Figure 10). The prominent sebaceous follicles present a moderate mononuclear inflammatory infiltrate (Figure 11) and reduced fibrosis. Some sudoriferous glands present abundant acute inflammatory infiltrate in the area of the excretory duct – acute hidradenitis (Figure 12).

The histopathologic aspects identified correspond to...
a dissecting cellulitis of the scalp where the dermis colagenization aspects and the pilosebaceous follicles damage are predominant.

The immunohistochemical reactions demonstrate the presence of the predominance of the chronic, mononuclear, lymphomonocyte inflammatory infiltrate. The lymphoid population is mainly constituted out of T-lymphocytes (CD3-positive), representing over 90% of the lymphoid component (Figure 13).

T-lymphocytes are perivascular and perianexially placed. The population of T-lymphocytes contains rare CD8-positive lymphocytes (suppressant T-lymphocytes) (Figure 14), the majority of the T-lymphocytes being with helper phenotype (over 95% from the lymphocyte component). B-lymphocytes are isolated (CD20-positive), having perivascular predisposition (Figure 15). The macrophagic component (CD68-positive), in moderate quantity, presents the same perifollicular and periglandular predisposition (Figure 16).

The mainly inflammatory component represented by lymphocytes sustains the long-term evolution of the lesions.
The lymphoid elements contain as main component helper T-lymphocytes, indicating a possible lack of balance between the helper/suppressant components, which would explain a possible immune mechanism involved in the pathogenesis and evolution of the disease.

**Treatment**

Our patient was administered antibiotic therapy per bone with Doxycycline 100 mg/day – three months, both for the nose colonization with *Staphylococcus aureus* (according to the antibiogram) and for the acne vulgaris lesions. Topically fusidic acid 2% cream (*Fucidin*®) was administered, and 4% benzoil-peroxide cream (*Brevoxyl®*) alternating with *Isotrexin®* gel. For the tinea versicolor lesions, we have used anti-fungi dermato-cosmetic shampoo *Selegel®*.

**Evolution and prognosis**

Under treatment the evolution of the disease was favorable, with the inflammatory nodules resorption, but with the persistence of the cicatricial alopecia. The clinical and biologic monitoring is compulsory. In case of the disease recurrence, isoretinoin *per os* in doses of at least 0.5 mg/kg/day will be administered, associated with small and short doses of prednisone or methylprednisolone.

**Discussion**

PCAS etiology is still incompletely elucidated, but its frequent association with acne conglobata, hidradenitis suppurativa and sometimes with the pilonidal disease suggests the involvement of the follicular occlusion in the etiopathogenesis of the disease. The increased frequency of the disease in black people allowed the specialists to suspect a pathogenic mechanism similar with the one from incarnated pili [2–4].

The incipient lesion common and characteristic for all the diseases mentioned before is the follicular hyperkeratosis, which will lead afterwards to occlusion and dilatation of the hair follicle. The follicular epithelium may proliferate or may be destroyed. In the incipient stages of the disease, the cutaneous biopsy shows the presence of numerous intact or apparently non-affected follicles, with a high rate of hair in the catagen and telogen stages (situation also valid for our patient). At the same time a moderate dense lymphocyte perifollicular inflammation produces, the perifollicular inflammatory infiltrate being formed out of neutrophils, lymphocytes and histiocytes. Thus, the deep abscess, which will destroy the pilosebaceous follicle, is born. The granulomatous structure, which appears in the evolution of the disease, is a consequence of the follicular destruction process. The foreign body granuloma thus constituted, which contains plasmocytes and lymphocytes, rests of keratin and hair, will sit around the outstanding follicles. The abscess develops in depth, favoring the formation of sinuous tracts. The healing of the lesions is done with scars [4–6].

The bacterial infection appears as a secondary phenomenon and the cultures in the closed abscesses are frequently negative. The benefic effect of systemic corticosteroids administration suggests that these three diseases of “the occlusional triad” represent antigen-antibody type reaction, resulted from the tissue destruction. The family character of the disease was reported in the specialty literature only in two cases (in two pairs of brothers) [7, 8].

The positive diagnosis is established based on a clinical picture only in exceptional cases, in the typical forms of the disease. The one establishing the certain diagnosis is the histopathologic examination of the cutaneous biopsy.

The differential diagnosis, both clinically and histopathologically, is done first of all with the other neutrophilic cicatricial alopecias (decalvant folliculitis – DF, “tufted” folliculitis), with the lymphocyte alopecias (plano-pilaris lichen, classic pseudopelade of Brocq, central centrifugal cicatricial alopecia, mucinous alopecia, decalvant spirillos follicular keratosis), with the mix forms of cicatricial alopecia (keloidian acne, necrotic acne and erosive pustulous dermatosis) [5, 9–12].

Although PCAS etiology is not bacterial, its differential diagnosis must take into consideration the profound, bacterial folliculitis as well. At the same time, in extremely rare cases, an inflammatory trichophytosis (trichophytic sycosis or Kerion Celsi) may mime PCAS [1]. Nevertheless, it is obvious that the differential diagnosis most difficult to establish is the one with decalvant folliculitis (Quinquaud).

Unlike PCAS, in decalvant folliculitis (DF) many authors sustain the absolute role of the golden staphylococcus in the pathogenesis of the disease, the production mechanism being either through the bacterial superantigens stimulating the immune system, without cellular involvement, or through an abnormal response of the host to the *Staphylococcus aureus* toxins. Other authors consider that the occurrence of pustules is secondary to the lesions supra-infection with *S. aureus* or that the occurrence of DF lesions would be the immune response to the follicular destruction phenomenon [1, 13].

Clinically, DF is characterized by the presence of alopecic plaques with pustules on the surface or at the margin, the inflammatory nodules occurring only exceptionally. The pustules cultures almost always render evident *S. aureus*. The PCAS inflammatory nodules cultures are most frequently negative, rarely being able to

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**Figure 16** – *The macrophagic component (CD68-positive), ×100.*
isolate opportunist germs (S. aureus or coagulase negative) or commensal germs. Frequently, “en touffe” aspect hair is noticed in DF. When the pustules are absent, DF lesions get the aspect of Brocq’s pseudopelade [13].

The histopathologic difference between these two entities is difficult. In DF initial pustulous lesions form a micro- abscess centered by hair and placed at the pilosebaceous infundibulum, from the inferior to the superior part, the area then formed wearing the aspect of a comedon-type dilatation. In evolution, the inflammatory infiltrate is placed as in PCAS, at the perifollicular level. What is specific for DF is its content mainly out of lymphocytes, together with very few neutrophils, plasmocytes, eosinophils and giant cells [4].

“Tufted” folliculitis is not considered anymore today a different entity, the denomination being abandoned. It can lately occur in the evolution of all clinical forms of cicatricial alopecia, being interpreted only in a semilogic context (polytrichia) [14–16].

The actual treatment of PCAS is with isotretinoin 0.5 mg/kg/day. Its efficiency is not yet proven à la longue after stopping administering it. Sometimes it is necessary to prolong the treatment up to four months after the lesions disappear [1, 17]. The long-pulse Nd:YAG laser is used for stopping the PCAS evolution, after the lesions disappear [1, 17]. The long-pulse Nd:YAG laser is used for stopping the PCAS evolution, after the lesions disappear [1, 17].

The evolution of the disease is chronic, with outbreaks interrupted by longer or shorter remission times. Sometimes, the lesions healing results in hypertrophic or keloidal scars [1].

The case presented here is a description of the disease in the incipient stage, difficult to clinically diagnose it. Other difficulties related to diagnosis were because the patient is Caucasian. The confusions the doctors made between PCAS and furuncles and the incision of the lesions, as well as the administration of isotretinoin in insufficient doses and in repeated and too short therapies for acne conglobata changed the clinical picture of PCAS and forced us to have a conservatory therapeutic approach. The good therapeutic results obtained allowed us to temporize the systemic administration of isotretinoin in the correct doses until the occurrence of an eventual recurrence of the disease.

§ Conclusions

Dissecting cellulitis of the scalp is an exceptional dermatological disease in male Caucasians. The clinical diagnosis is difficult in the initial stage of the disease and often creates confusions with other profound folliculitis or with different cicatricial alopecias. The histopathologic examination brings decisive arguments for the positive diagnosis of PCAS, allowing as well establishing the differential diagnosis. The diagnosis of one of the “occlusion triad” diseases forces the dermatologist to clinically monitoring the patient in order to fond out the other possible associated diseases, as it was the case for our patient, who had as a first manifestation acne conglobata.

References

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