A brief review of the “vulgaris” lesions of the skin

M Chen, E Bruno, E Santos, D Sarma

Citation

M Chen, E Bruno, E Santos, D Sarma. *A brief review of the “vulgaris” lesions of the skin*. The Internet Journal of Dermatology. 2006 Volume 6 Number 1.

Abstract

Medical students and the trainees in dermatology and pathology have a curiosity about the names, such as, acne vulgaris or verruca vulgaris. The word “vulgaris” sounds like vulgar, commonly meaning crudely indecent, coarse, or obscene. However, the word “vulgar” is derived from the Latin word “vulgaris”, meaning “of or belonging to the common people”. In medical terminology, the word “vulgaris” means common or ordinary.

A search through the indexes in Dermatology and Pathology text books revealed seven dermatologic conditions with “vulgaris” in their names. These included very common diseases, such as, acne vulgaris and verruca vulgaris as well as now rarely diagnosed disease, such as, lupus vulgaris. We are briefly reviewing the seven “vulgaris” diseases of the skin.

ACNE VULGARIS

The term acne originates from the Greek word acne, which Aëtius Amidenus used to describe a skin eruption. The most common form of acne is “acne vulgaris”, meaning “common acne”. Acne vulgaris is an inflammatory disease of the skin, affecting a large percentage of people, especially during puberty. The typical acne lesions can be categorized into comedones, papules, pustules, nodule and inflammatory cysts.

The mechanism of acne formation involves the excessive secretion of oils from the sebaceous glands due to hormone over-production as well as the shedding of dead skin cells. This likely obstructs the pores of hair follicle providing an ideal environment for microorganism growth, which initiates an inflammatory response. This leads to the formation of a visible lesion [1]. The remaining unsightly scars after acne resolution may have a significant psychosocial impact on the affected adolescent; early and aggressive treatment is therefore advocated.

Microscopically, acute folliculitis and peri-follicular neutrophil accumulation (pus) are evident. During the resolving stage, the hair follicle is completely destroyed and inflammatory cells are replaced by fibrous tissue leading to scar formation.

VERRUCA VULGARIS

Verruca vulgaris, commonly known as a wart, is one of the most recognizable skin growths. Common warts can occur at any age but generally occur during childhood and adolescence. Frequently, warts occur on the fingers and hands; however, warts may grow on any epidermal surface. The causative agent of verruca vulgaris is human papilloma virus (HPV), which is also responsible for genital warts, cervical dysplasia, and cervical cancer [2]. When HPV invades the skin, it incorporates into the human genome and interferes with the cell cycle, leading to unregulated cell growth and wart formation. HPV typically invades individuals with limited immunity. Children generally have less immunity to HPV than adults, and are more commonly infected. To date, no single treatment is perfect for verruca vulgaris. The treatment of choice depends on both the location and the size of the lesion. Genital warts require special consideration due to the precancerous risk.

Microscopically, the appearance of verruca vulgaris is quite eye-catching. The lesion has a centrally raised spiky undulation due to the upward proliferation of epidermis and subepidermal papillae and usually shows marked hyperkeratosis. The rete ridges at the edge of the lesion converge toward a central point. In the acute phase, keratinocytes undergo noticeable viral change (koilocytosis), which tends to be less obvious during the relapsing phase.

LUPUS VULGARIS

The term lupus, Latin for wolf, implies the rapacity and virulence of this disease. It has been used since the late

thirteenth century to describe ulcerative skin disease. However, it was not until the mid-nineteenth century that two specific skin diseases were separated as lupus erythematosus and lupus vulgaris.

Lupus vulgaris is a progressive form of cutaneous tuberculosis [3]. It is twice as common in women as in men, and occurs in patients with immunity produced by previous tuberculosis exposure. The pathogen may reach the skin by two mechanisms: 1) exogenous inoculation, via the blood or lymph, spreading from other tuberculous internal organs, or 2) by direct extension from underlying infected glands or joints. The disease often affects the face around nose and ears, and ultimately leads to skin disfigurement due to the ulcerative destruction. The characteristic lesion has a nodular appearance with a reddish-brown plaque, and shows an "apple-jelly" color when pressed with a glass spatula (diascopy).

The histopathologic features of this disease are rather nonspecific. Besides the skin ulceration, tuberculoid granulomas in the deep dermis may be suggestive of the diagnosis. Special stain and tissue culture for acid-fast bacilli are confirmative but often yield a negative result.

The key for diagnosis of lupus vulgaris is the awareness of tuberculous etiology in any chronic ulcers. If the patient has clear evidence of tuberculosis, standard antitubercular treatment should be started to ensure a favorable prognosis.

ICHTHYOSIS VULGARIS

Ichthyosis vulgaris refers to a group of cutaneous disorders of keratinization. The term ichthyosis is derived from the ancient Greek root ichthys, meaning fish, and describes the resemblance of the affected skin to the fish scales. Ichthyosis vulgaris can be further classified into hereditary and acquired forms.

Hereditary ichthyosis vulgaris is an autosomal dominant genetic disorder first evident in early childhood. It is the most common form of ichthyosis, accounting for more than 95% of cases. It is caused by altered profilaggrin expression leading to scaling and desquamation [4]. Visible scales are retained for long periods and are sloughed off in clumps. On the other hand, acquired ichthyosis is a nonhereditary condition associated with internal disease, and usually appears for the first time in adulthood. Acquired ichthyosis is rare and must be viewed as a marker of systemic disease, including malignancies.

Ichthyosis vulgaris is classified as a retention hyperkeratosis. The histological appearance of both hereditary and acquired ichthyosis is practically identical. The characteristic finding is a moderate degree of hyperkeratosis with a thin or absent granular layer. The hyperkeratosis often extends into the hair follicles, resulting in large keratotic follicular plugs. The dermis is usually normal.

PEMPHIGUS VULGARIS

Pemphigus vulgaris is an autoimmune skin disorder characterized by blistering of the skin and mucous membrane. Pemphigus is derived from the Greek word pemphix meaning bubble or blister. This condition develops primarily in middle-aged or older individuals, and is a potentially life-threatening disease. Before corticosteroids became available, the mortality of this disease was high because of fluid loss and superinfection.

Blisters in pemphigus vulgaris are associated with the binding of IgG autoantibodies to keratinocyte surface molecules such as desmoglein 1 and desmoglein 3 [5]. The binding of autoantibodies results acantholysis, the loss of cell-cell adhesion. The antibody is capable of causing blistering without complement or inflammatory cells.

The earliest recognizable histopathologic change is either eosinophilic spongiosis or spongiosis of the lower epidermis. The subsequent acantholysis first leads to the formation of clefts, and then to predominantly suprabasal blisters. There is little inflammation in the early phase of blister formation. If, however, eosinophilic spongiosis is apparent, numerous eosinophils may infiltrate into the dermis. As the lesion ages, several changes ensue, including inflammatory cell reaction, blister erosion or ulceration, and keratinocyte proliferation.

Direct immunofluorescence (DIF) is a very sensitive and specific diagnostic test for pemphigus vulgaris. IgG is demonstrated in the squamous intercellular substance with a lacelike appearance in 80% to 95% of the cases. The positivity remains many years after the disease has regressed [6]. Negative DIF findings when the patient is in remission may be a good prognostic indicator.

PSORIASIS VULGARIS

Psoriasis vulgaris is a common chronic inflammation skin disorder that affects approximately 1.5% to 2% of the population. The term psoriasis is derived from the Greek word psora that means itch. Psoriasis vulgaris is also referred to plaque psoriasis because of the characteristic plaque formation on the skin. The well-defined patches of red raised

skin, usually involves the knees, elbows, scalp, trunk and nails. The flaky silvery white buildup composed of dead skin cells on top of the plaques is called scale. Affected skin is generally very dry, with possible superimposed symptoms of aching, itching, and cracking.

The pathogenesis of psoriasis is not fully understood. There are two main hypotheses regarding development of the disease. The first considers psoriasis primarily as a disorder of excessive growth and reproduction of skin cells. The second hypothesis believes that psoriasis is an immune-mediated disorder. It is thought that T cells become active, migrate to the dermis and trigger the release of certain cytokines especially tumor necrosis factor-alpha (TNF?), which causes inflammation and the rapid production of skin cells [7]. However, it is not known what initiates the activation of the T cells.

The histologic appearance varies considerably with the stage and severity of the disease. At the early stage, there are non-specific signs such as capillary dilation and edema in the papillary dermis with pericapillary lymphocyte infiltration. The diagnostic feature in this stage is the so-called Munro microabscesses, the infiltration of neutrophils into the area of parakeratosis. If there is excessive exocytosis of neutrophils, they may aggregate to form small spongioform pustules of Kogoj. The fully developed lesions are characterized by acanthosis with rete ridge elongation, thinning of the suprapapillary epidermis with small spongiform pustules, pallor of the upper epidermis, diminished granular layer, confluent parakeratosis, presence of Munro microabscesses, dermal papillary edema, and dilated capillaries. Of all these listed features, only the spongiform pustules of Kogoj and Munro microabscesses are truly diagnostic.

VITILIGO VULGARIS

Vitiligo vulgaris is a skin condition characterized by patches of skin losing their pigmentation, due to the destruction of the pigment producing melanocytes. This disease affects an estimated 1% of the world's population, and has a strong familial association. Half the patients first recognize vitiligo before 20 years of age, when it often appears in an area of minor injury or sunburn.

The central process in vitiligo is the destruction of melanocytes at the dermo-epidermal junction. Fully blossomed lesions are totally devoid of melanocytes. Rarely, a superficial perivascular lichenoid mononuclear infiltration may be observed at the border of the depigmented areas. The exact cause of vitiligo is unknown. Though autoimmune mechanisms with an underlying genetic predisposition are the most likely the cause. Antibodies to the melanocytes have been detected by immunoprecipitation in the sera of the patients with vitiligo. Also, sera from patients with vitiligo causes damage to melanocytes in cell culture, suggesting that the antibodies present in sera of affected patients are involved in the pathogenesis of vitiligo [8].

The diagnosis is usually straightforward, and requires no special testing. While vitiligo is a purely cosmetic concern, it is disfiguring and may be psychologically traumatic.

CORRESPONDENCE TO

Deba P Sarma, MD Department of Pathology Creighton University Medical Center Omaha, NE 68131 Tel: 402-449-4951 debasarma@creighton.edu

References

1. Thiboutot D, Gilliland K, Light J, Lookingbill D: Androgen metabolism in sebaceous glands from subjects with and without acne. Arch Dermatol 1999; 135:1041.
2. Carr J, Gyorfi T: Human papillomavirus. Epidemiology, transmission, and pathogenesis. Clin Lab Med 2000; 20:235-55.
3. Lipsker D, Grosshans E: What is lupus vulgaris in 2005? Dermatol 2005; 211:189.
4. Segre JA: Epidermal differentiation complex yields a secret: mutations in the cornification protein filaggrin underlie ichthyosis. J Invest Dermatol 2006; 126:1202.
5. Cozzani E, Dal Bello MG, Mastrogiacomo A, Drosera M, Parodi A: Antidesmoplakin antibodies in pemphigus vulgaris. Br J Dermatol 2006; 154:624.
6. Judd KP, Lever WF. Correlation of antibodies in skin and serum with disease severity in pemphigus. Arch Dermatol 1979; 115:428.
7. Griffiths CE, Iaccarino L, Naldi L, Olivieri I, Pipitome N, Salvarani C, Doria A: Psoriasis and psoriatic arthritis: immunological aspects and therapeutic guidelines. Clin Exp Rheumatol 2006; 24 (1 Suppl 40):S72.
8. Norris DA, Kissinger RM, Naughton GM, Bystryn JC: Evidence for immunologic mechanisms in human vitiligo: patients' sera induce damage to human melanocytes in vitro by complement-mediated damage and antibody-depedent cellular cytotoxicity. J Invest Dermatol 1988; 90:783.

Author Information

Mingkui Chen, M.D., Ph.D.
Department of Pathology, Creighton University Medical School

Erin Bruno, B.S.
Department of Pathology, Creighton University Medical School

Eric E. Santos, M.D.
Department of Pathology, Creighton University Medical School

Deba P. Sarma, M.D.
Department of Pathology, Creighton University Medical School