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各種湿疹皮膚炎群、蕁麻疹、血管炎皮疹の因数分解・ロジック診断： 内科医が診ても絶対間違えない方法

Logical diagnosis by factorizing skin eruptions of common skin diseases, as such a variety of eczema, urticaria, vasculitis: An easy understanding approach for non-dermatologist physicians to make solid diagnoses of skin diseases



北島 康雄
Yasuo Kitajima

木沢記念病院 北島 康雄

Yasuo Kitajima, Kizawa Memorial Hospital

対象者 医師, 後期研修医 (卒後3年目以上), 初期研修医 (卒後1-2年目)

Target Doctor, Senior Resident (3+years after graduation), Resident (1-2 years after graduation)


皮膚科医は、人の顔を見た瞬間に誰と認識できるように皮疹を診た瞬間に大抵の皮膚疾患を診断できる。この能力は、正しく診断された膨大な症例経験数によって獲得される。しかし、正しく診断された多数の症例経験がない若い皮膚科医や非皮膚科医にとって、このようなパターン認識診断（暗黙知診断）は、多様なバリエーションや修飾のため、たとえ湿疹や白癬のようなありふれた疾患でも容易でない。これを克服するには、皮膚症状を形成する皮疹（丘疹、紅斑、鱗屑、膨疹など）の臨床的構成要素と病理組織要素についてロジックを持って解析・判断し、皮疹の形成される病態メカニズムを理解することが近道であるとする。

このような視点で、発疹の診方のロジックを参加者の頭の中に動的な臨床と病理像がストーリー性を持って浮かび上がるように解説したい。なぜそのような発疹ができるか（本質的病態）を把握できれば、湿疹/蕁麻疹などのコモディージェズをその多様なバリエーションや重複疾患があっても確信を持って診断出来る。すなわち、逆にこれらのコモディージェズに混じってまれに出現する全身の疾患の皮膚症状を見落とさないことになる。

この視点から、アレルギー性接触皮膚炎(ACD)、脂漏性皮膚炎(SD)、皮脂欠乏性皮膚炎(ASD)、酒さ様皮膚炎(RD)、蕁麻疹(Ur)、蕁麻疹様血管炎(UrV)を例に挙げ解説する。ACD(表皮内IV型アレルギーで樹状細胞/リンパ球による炎症)はなぜ点状病変に成るか、SD(刺激性脂肪酸による直接的表皮角化細胞性の炎症)、ASD(角層バリアー不全による機序不明の表皮角化細胞刺激性炎症)では紅斑落屑性変化のみで点状病変は絶対作らないか、あるいはRD(グルココルチコイド刺激による毛嚢脂腺系、毛細血管の反応が関与した炎症)では紅斑、毛細血管拡張と毛嚢性丘疹のみで漿液性丘疹や鱗屑は作らないか、また、Ur(IgE/肥満細胞の脱顆粒-膨疹が機序)は数時間で膨疹が消失するが、UrV(IgG免疫複合体の表皮直下毛細血管壁沈着と補体による膨疹・炎症)はなぜ2日以上皮疹が続くのか、その本質的病態が全て皮疹に表現されている。そこで、その皮疹の因数分解と診断ロジックを解説したい。この様な視点の皮膚科解説書は未だないので、北島康雄著「皮疹の因数分解、ロジック診断」秀潤社2018年11月発行を参照されたい。

Dermatologists can diagnose any skin diseases in an instant as they look at the skin eruptions in the same manner as we can tell who is who in an instant as we see a certain person. We can obtain this capability through a large number of experiences of cases, which had been correctly diagnosed. By the pattern-recognition or implicit diagnosis like this, however, to diagnose skin diseases should be difficult for young dermatologists and non-dermatologists who have no such experiences, even as for common diseases like a variety of eczema (allergic contact dermatitis, seborrheic dermatitis, asteatotic dermatitis), rosacea-like dermatitis, tinea, urticaria, urticarial vasculitis and so on. In order to overcome this difficulty, it would be a short cut that we analyze logically constituent elements of skin eruptions such as papules, erythema, scales, wheal, by a logical understanding how these elements of these skin eruptions are generated in terms of scientific logics, i.e., being based on pathologic and dynamic molecular interaction mechanisms in the skin.

I would like to talk about this proposition, presenting how dynamic clinical and pathologic reactions generate individual specific elements of skin eruptions, by which we can understand how logically observe skin eruptions to make a doubtless diagnosis. When we master the logical observation-analysis of skin-eruption elements, we can make a solid diagnosis with confidence, even if these eruptions were masked by a variety of variations. This is expected to lead us to avoid oversight in systemic disease-related skin eruptions during routine medical practice.



To make it clearer how logically observe and find elements of skin eruptions, I would like to talk on examples as such allergic contact dermatitis (ACD), seborrheic dermatitis (SD), asteatotic dermatitis (AsD), rosacea-like dermatitis (RD), prurigo (Pg), urticaria (Ur), urticarial vasculitis (UrV). I will illustrate why ACD (inflammation by type IV allergy with lymphocytes in the epidermis) generate papules with crusts surrounded with micro-circular scales, why SD (inflammation by direct stimulation to keratinocytes with free fatty acids, breakdown products of sebum) and AsD (inflammation by direct stimulation to keratinocytes by unknown mechanisms due to impaired barrier function of stratum corneum) cannot show papules, but scaly erythema only, and why RD (inflammation related with proliferation of sebaceous and hair organs due to over/long-term glucocorticoid treatments) cause follicular papules, erythema and telangiectasia, but no serous papules and scales. In addition, I will do also how differs Pg (inflammation basically of type IV allergy in superficial dermis) from reactive perforating collagenosis often found in patients with diabetes mellitus or collagen diseases, and why Ur (IgE/mast cell-induced acute edema in superficial dermis) shows quick disappearance of wheal within several hours, while in case of UrV (IgG-immune complex-induced complement dependent wheal) eruptions last a few days long. It would be very important to logically understand, even if partially, how these differences exert different clinical signs in skin eruptions rather than to make an implicit diagnosis without thinking. All of these differences above-mentioned are illustrated in the clinical signs of skin eruptions, and we can distinguish them logically obtaining solid answers. There have been no textbooks describing skin eruptions in insights similar to my proposition mentioned here. I hope you could find a time to read my textbook before my talk; Yasuo Kitajima “Factorization of skin eruptions: logical diagnosis” Syujunsha, 2018, Nov (in Japanese).