



Max Ruiter (1900-1974)

Ruud Horlings, dermatoloog

*De gouden Mendes da Costa medaille, die is ingesteld door de Nederlandse vereniging van Dermatologen en die eens in de vijf jaar uitgereikt kan worden voor verdienstelijk werk op het gebied van de dermatologie is voor de eerste maal op 14 mei jl. uitgereikt aan Prof. Dr. M. Ruiter.
NTvG, 28 Mei 1960*

Prof. Maximiliaan Ruiter, stond bekend als een vooraanstaand en toegewijd vertegenwoordiger van de dermatologie, een wetenschapper die daarnaast over grote klinische begaafdheid beschikte. Geboren in Nijmegen, doorliep hij het gymnasium in Leeuwarden en volgde zijn geneeskunde en dermatologie opleiding aan de Universiteit van Groningen. In 1933 promoveerde hij cum laude op het onderwerp chronische pyodermie. Na een periode in Deventer keerde hij in 1947 terug in Groningen als hoogleraar dermatologie.

Ruiter had een opvallende interesse in ziektebeelden die niet tot de huid beperkt bleef. In zijn Deventer jaren publiceerde hij samen met Pompen (internist) en Wyers (patholoog) over angiokeratoma diffusum als marker van een fosfolipiden stapeling, een thesaurismosis. Internationale bekendheid verwierf hij met onderzoek naar vasculitis allergica, een tot dan toe nog weinig begrepen aandoening. Ruiter herkende dat de aandoening een allergische reactie is op diverse prikkels, zoals bacteriën, geneesmiddelen, voedingsstoffen of insectenbeten. In de nadagen van zijn ordinariaat was Ruiter de eerste die met behulp van een elektronenmicroscopie aantoonde dat epidermodysplasia verruciformis veroorzaakt wordt door het humane papillomavirus.

Naast de dermatologische zorg was Ruiter in Groningen betrokken bij de transitie van een naoorlogs houten noodgebouw naar een fraaie dermatologische kliniek. Toch meenden



sommigen van zijn toenmalige collega's dat het oude gebouw mogelijk beter paste bij de teruggetrokken en haast iets verlegen prof. Ruiter. Daar had hij dagelijks contact met zijn leerlingen en heerste de intieme sfeer waar hij zo'n behoefte aan had. Ruiter was een man met een brede interesse in kunst, geschiedenis en muziek. Na zijn emeritaat in 1969 portretteerde de bekende schilder Sierk Schröder hem in witte jas. Ruiter sleet zijn laatste jaren in het landelijke Norg. Hoewel collegiaal contact hierdoor veelal verbroken werd, bleef hij wetenschappelijk actief.

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Arteriolitis (Vasculitis) “Allergica” Cutis (Superficialis). A New Dermatological Concept

By M. RUITER

The designation arteriolitis (vasculitis) “allergica” cutis superficialis (a.a.c.) applies to a group of closely related skin eruptions showing variable and polymorphous cutaneous manifestations but falling under one heading, when using the vascular lesions as a common denominator [7–12]. These latter dominate the histological picture and by their typical histological changes, their location in the skin and the caliber of the affected vessels they show a pattern of their own to the effect that they can be satisfactorily delineated from related vascular conditions. This observation afforded the possibility to study the eruptions in question from one and the same point of view and has contributed to a better understanding of the cutaneous syndromes grouped under this heading. Both the theoretical aspects and the clinical usefulness of the concept a.a.c. will be discussed in this paper.

The Histological Features

The vascular changes, *predominantly confined* to the skin, have proved to represent the essential lesion. Histological investigation has shown that *superficial cutaneous blood vessels* (mainly arterioles, but also venules and capillaries) were particularly affected. The affected vessels (Fig. 1) showed endothelial swelling, fibrinoid changes in the vascular wall and inflammatory infiltrates both in the vascular wall proper and in surrounding parts. The often very dense inflammatory infiltrates consisted chiefly of polynuclear leucocytes, as a rule including some eosinophils; they showed remarkably pronounced nuclear disintegration (leucoclasia). In addition to polynuclear leucocytes lymphocytes and monohistiocytic cells were also encountered though in smaller number. The vascular alterations generally showed

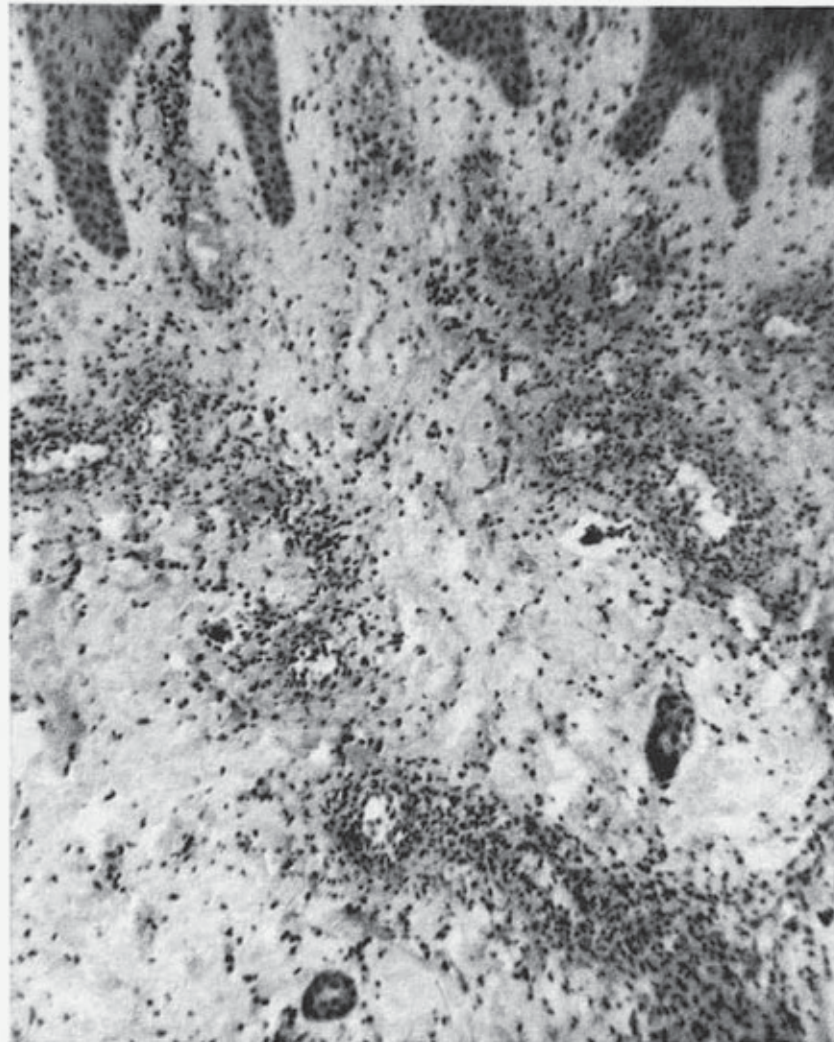


Fig. 1. Histopathological substrate of arteriolitis (vasculitis) "allergica" cutis superficialis. Panoramic view. H.E. $\times 95$.

an exudative character; as a rule the lesions were histologically of the same age. The fibrinoid changes found in the vascular wall and in the adjacent tissue often lent a characteristic aspect to the histological picture. Only in cases in which haemorrhages predominated were the fibrinoid changes less distinct and, in exceptional cases, even proved to be lacking (an attempt at a detailed analysis of the vascular fibrinoid in a. a. c. [13, 14] has been communicated elsewhere, (1962).

The location of the vascular changes (Fig. 2) was as follows: the vascular alterations were most pronounced in the stratum subpapillare



Fig. 2. Localization of vascular changes in arteriolitis (vasculitis) "allergica" cutis superficialis according to their occurrence.

and reticulare. Towards the deeper layers of the dermis the number of affected vessels decreased and so did the extent of the changes. At the junction between cutis and subcutis pathologically changed vessels were only occasionally found; as in the dermis only small vessels were involved.

From a histopathological point of view the vascular lesions found in a.a.c. seem to be closely related to those found in Zeek's hypersensitivity angiitis, although the latter represents a more generalized vascular disease with predominantly visceral involvement.

The Clinical Side of the Concept

A classification exclusively based on histopathological changes is of only limited value for clinical and diagnostic purposes. In the course of our observations, however, we succeeded in establishing a number of common clinical features. First to be mentioned is the rather typical intermittent course of these cutaneous syndromes and the

symmetrical distribution of the efflorescences observed. Secondly a number of common characteristics could be demonstrated with reference to the cutaneous pictures. Thus, the cutaneous lesions as a rule showed: (a) a tendency towards cutaneous haemorrhage; (b) the presence of an urticarial component; (c) inflammatory changes of a transient or more persistent character, sometimes followed by superficial scarring.

In the course of time a number of more or less regularly occurring combinations of cutaneous lesions could be distinguished in addition. These might be regarded as "landmarks" in the rather confusing field of cutaneous manifestations occurring in the arteriolitis group. Such *clinical variants* are best demonstrated with the help of a number of case histories.

Case 1

A man aged 59. Early in december 1959 sore throat and fever: some 6 days later painful swelling of the ankles soon spreading to other joints. Simultaneously pain in the upper abdominal region. On the following day a skin eruption developed. Since then there has been a recurrence twice. On 23rd December 1959 the patient was admitted to hospital.

Cutaneous picture: Skin lesions were found especially on the extremities, more particularly on the lower legs (Fig. 3). Efflorescences were seen also on the buttocks, flanks and periaxillary regions. Various stages of development were simultaneously found. The youngest elements consisted of *oval to round pale-red oedematous macules*.



Fig. 3. Haemorrhagic type of arteriolitis (vasculitis) "allergica" cutis superficialis (case 1).



Fig. 4. Nodular-haemorrhagic type of arteriolitis (vasculitis) "allergica" cutis superficialis (case 2) (from "Hautarzt").

Small *haemorrhagic spots* often occurred in the centre. During the later course of the disease the central haemorrhages became confluent and the inflammatory symptoms of the lesion decreased, until only the haemorrhagic part in the form of *purpura* remained. Smaller and larger greyish-brown pigmented patches were found among the efflorescences.

Physical examination: No anomalies. ESR after 1 hour 35 mm. Leucocyte count 4400. Protein pattern: total protein 6.3 g/100 ml; albumin 46.2%, globulins; α_1 5.3%, α_2 10.5%, β 15.6%, γ 22.4%. Platelets: 230,000. Clotting time 2 min 30 sec, bleeding time 4 min. Urine: no albumen or glucose; a few erythrocytes. Macroglob.: negative; Cryoglob.: negative. AST 500 E. C-reactive protein +++; L.E. phenomenon negative. Examination for focal infections: negative. Throat swab: pyogenic streptococci.

Histology: Typical vascular lesions in the upper and middle layers of the corium; slight fibrinoid changes.

Diagnose: *Haemorrhagic type of arteriolitis (vasculitis) "allergica" cutis superficialis.*

Course: In the past 2 years repeated remissions and exacerbations. Antistreptolysin titres determined during the course of the illness, showed varying though unmistakably elevated values. The articular manifestations did not recur.

Case 2

A 34-year-old Indonesian woman with a 5-day history of a cutaneous affection which had started on the ankles and lower legs. Tight feeling in the calves. No swelling of the joints, no abdominal pain. Two weeks ago sulfa drugs for acute tonsillitis.

Cutaneous picture: The lower legs, both on the flexor and on the extensor sides, showed a limited number of scattered, approximately cent-sized, pale red, round to oval *slightly elevated nodular infiltrates*, which were fairly sharply defined (Fig. 4). In the centre *disk-shaped haemorrhages or haemorrhagic crusts*, were observed. Some of these elements showed superficial necrosis. On the anterior aspect of the right foot there were a few bullae with a slightly haemorrhagic content. The ulnar sides of the forearms and the backs of the hands showed erythematous patches with a pale centre.

Physical examination showed no anomalies. ESR after 1 hour 44 mm. Protein pattern: total protein 6.7 g/100 ml; albumin 47.9%, globulins: α_1 4.9%, α_2 6.3%, β 13.6%, γ 27.3%. Urinalysis: no abnormalities. Cryoglob. negative; C-reactive protein + + + + +; AST 800 U.

From the throat pyogenic streptococci (group A) were recovered.

Histological examination of a haemorrhagic nodule: characteristic features with considerable vascular "fibrinoid"; occasionally "hyaline" thrombi in the small affected vessels of the dermis.

Diagnosis: Nodular haemorrhagic type of arteriolitis (vasculitis) "allergica" cutis superficialis.

Course: A few further efflorescences developed in the course of subsequent weeks. After this the patient recovered without medication.

Case 3

A boy aged 15 had suffered from bronchitis from his sixth year; later he coughed up large quantities of green sputum. The patient had shown a relapsing papular eruption for the past 3 years and had suffered recently from hay fever.

Cutaneous picture: Scattered over the body, but more frequent on the extremities than elsewhere, *numerous flat papular elements of a red or brownish-red colour*, sometimes surrounded by a reddish slightly oedematous border. Several papular elements showed *haemorrhagic crusts or haemorrhagic-necrotic plugs* over the summit (Fig. 5). Older elements showed some scaling as a rule. Various stages in the eruption were observed side by side, including small, superficial, varioliform scars. The efflorescences occurred on the neck, trunk, arms, thighs, lower legs and the dorsal aspect of the feet. A similar case is shown in Fig. 6.

Physical examination: Leucocyte count: 7900 per cmm; eosinophils 2%. ESR 20 mm in 1 hour. Thrombocytes and bleeding and clotting times normal. Urine: albumen —, glucose —. Pirquet reaction negative. X-ray examination of the chest revealed extensive bronchiectasis in both lungs.

Histological examination: Affected in particular were the small vessels in the stratum papillare and reticulare. There were fibrinoid vascular changes. Mono-

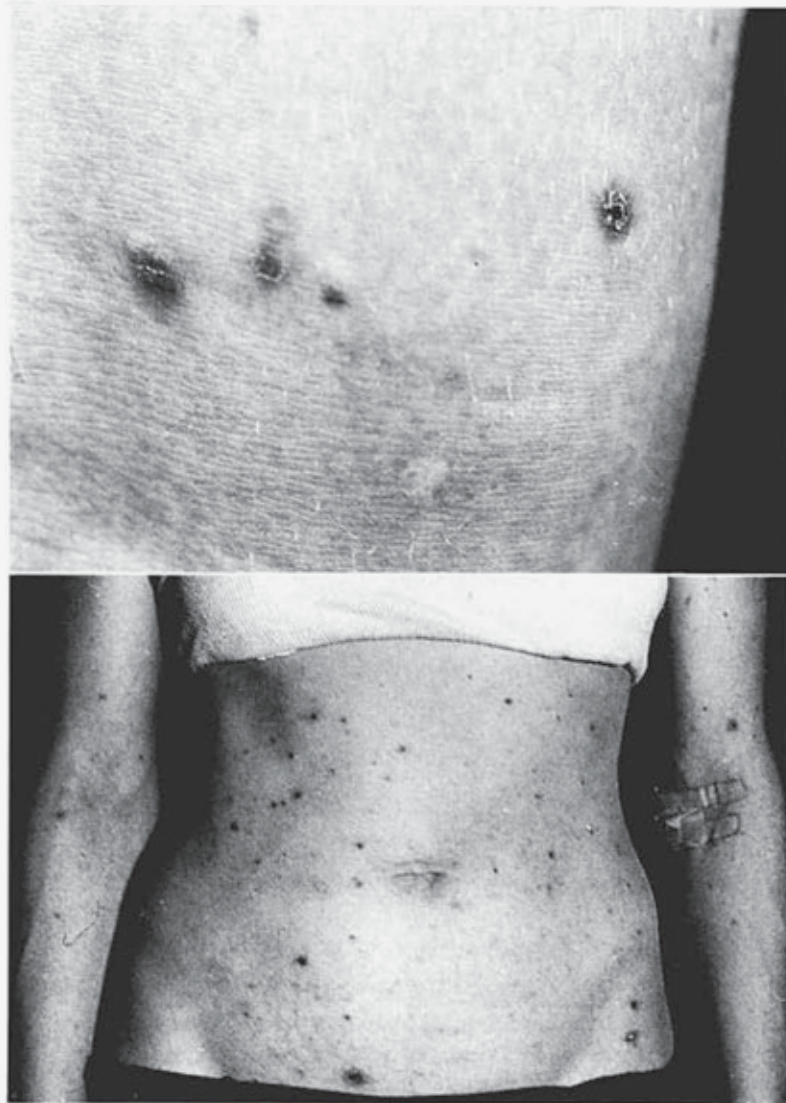


Fig. 5. Papulo-necrotic type of arteriolitis (vasculitis) "allergica" cutis superficialis (case 3) (from Acta dermat.-vener.).

Fig. 6. Distribution of lesions on trunk and arms in a case of the papulo necrotic type of arteriolitis (vasculitis) "allergica" cutis superficialis originally diagnosed as Mucha's disease.

nuclear and reticulo-histiocytic cells formed a larger part of the perivascular infiltrates than is usually seen. Among others a (terminal) arteriole supplying a hair follicle was affected. The larger vessels especially at the cutaneous-subcutaneous border or those in the fatty tissue proved to be unimpaired.

Diagnosis: Papulo-necrotic type of arteriolitis (vasculitis) "allergica" cutis superficialis.

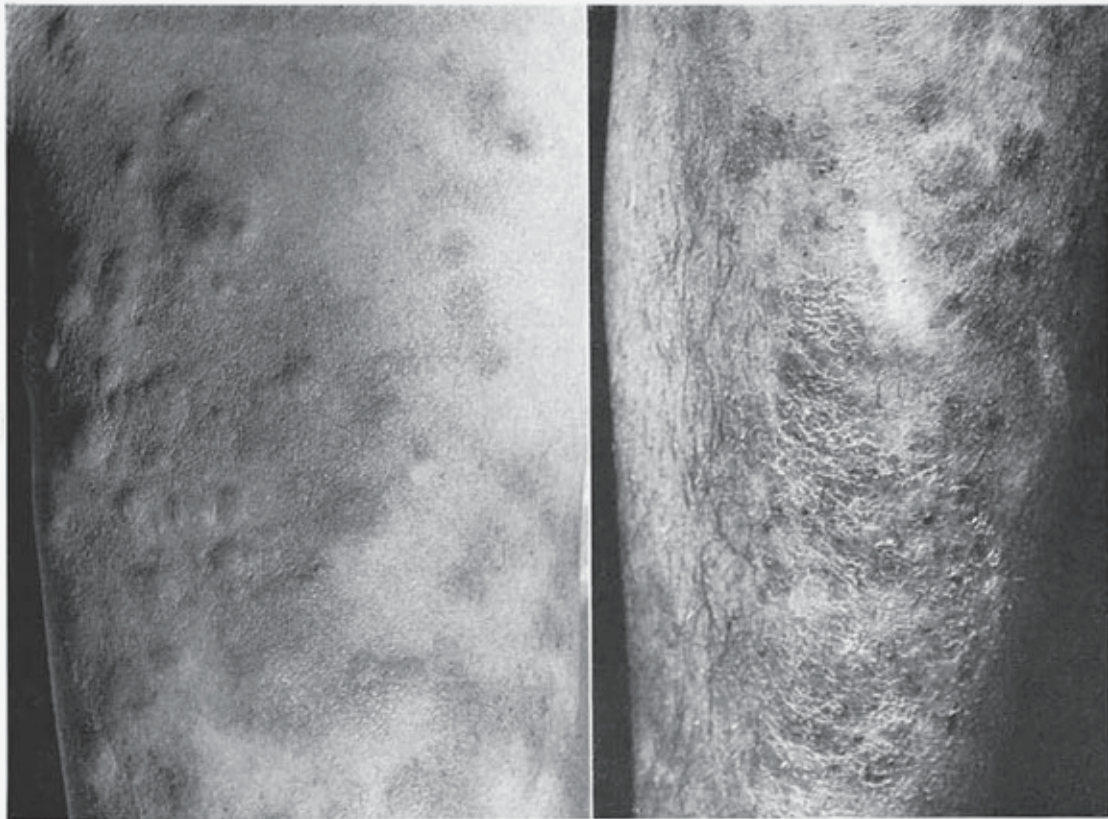


Fig. 7. Polymorphous-nodular type of arteriolitis (vasculitis) "allergica" cutis superficialis. Detail (shoulder) (case 4).

Fig. 8. Polymorphous-nodular type of arteriolitis (vasculitis) "allergica" cutis superficialis. Lesions on the calf (case 4) (from Brit. J. Derm.).

Course: Follow-up examination after 6 months showed that the skin changes had for the greater part receded (on account of antibiotic medication for bronchiectasis?).

Case 4

A woman aged 38. The eruption characterized by remissions and exacerbations had existed for 2 years. It was believed (?) to have developed following medication "for the gall-bladder". The cutaneous lesions were chiefly seen on the extremities, with some on the shoulders and the face. The presence of *firm nodules, the size of a pea or larger* was a conspicuous feature (Fig. 7). They lasted for several days and were somewhat *reminiscent of urticaria*, but too firm and not sufficiently transient for this type of lesion. Young specimens were more brownish-yellow of colour. Some of them showed a pinpoint-size vesicle or a haemorrhage at the top. These papular elements were most marked on the knees and elbows, and some were present over the shoulderblades. In addition to the nodular elements, *irregular erythematous macular lesions with a livid centre and superficially resembling erythema multiforme* were seen. They showed sometimes a pinpoint papule, vesicle or pustule in the centre and

frequently merged into larger patches. On the lower legs they were often associated with *purpuric spots* and occasionally with an annular haemorrhage at the border. Various brownish-red crusts and scabs were also observed. Extensive changes were present particularly on the calves (Fig. 8).

Physical examination: No anomalies. ESR 37 mm in one hour. Leucocyte count: 8000. Protein pattern: total protein 8.1 g/100 ml; albumin 35.7%, globulins: α_1 .4%, α_2 12.3%, β 17.2%, γ 28.4%. Macroglob. negative; Cryoglob. negative. Pirquet reaction ++. AST 50 U/ml. L.E. phenomenon repeatedly negative. Urinalysis: no anomalies. X-ray of gall-bladder: normal. Skiagram of the teeth showed a few retained roots. Culture from dental roots: streptococcus viridans. Throat swab: streptococcus viridans.

Histological examination (papular lesion): characteristic histological features with "fibrinoid" vascular changes.

Diagnosis: *Polymorphous nodular type of arteriolitis (vasculitis) "allergica" cutis superficialis.*

Course: During the next 5 years virtually unchanged, with remissions and exacerbations. Extraction of the retained dental roots no effect. Slight improvement by administration of prednisone (30 mg daily). Tolerance to this therapy, however, was poor.

In these case histories those combinations of skin lesions, which are more or less characteristic for the various clinical variants are indicated by italics. Typical for all clinical variants of a.a.c. are the symmetrical location of the eruptions and their intermittent character showing a distinct tendency to remissions and exacerbations. From the cutaneous pictures described it appears that these – with the possible exception of the papulo-necrotic type – are not infrequently accompanied by additional skin changes, which, however, from a diagnostical point of view, can be considered as being of secondary importance. Among these vesicles or pustules and (sometimes haemorrhagic) bullae are seen. The latter may be found a.o. on the lower legs (especially the lower third), and sometimes develop into superficial ulcers. Local maceration of the skin was also observed in some cases. Occasionally transient urticarial eruptions during the course of illness occurred.

Comment

Following the description of the a.a.c.-concept a short account should be given of the motives which have lead to its drawing-up.

Arteriolitis (vasculitis) "allergica" cutis superficialis includes among others a number of skin eruptions known in the dermatological literature under various designations. Among those is the so-called trisymptom complex of Gougerot. The history of this latter cutaneous

syndrome is not without interest as it clearly shows the difficulties in clinical defining such eruptions. *Gougerot*, in the long run, was compelled to extend his trisymptom complex by conceiving a whole series of symptom complexes ranging from a monosymptom to a pentasymptom. Later on this view was also abandoned and a new term (nodular allergids) was coined. As all these cutaneous conditions were classified along traditional dermatological lines, namely by means of a systematic analysis of their cutaneous manifestations, it is quite understandable that, in view of the polymorphy and variability of the cutaneous pictures in question, their definition was never quite satisfactory from a dermatological point of view. In our a.a.c.-concept a new approach to the whole group of eruptions under discussion, including those of *Gougerot*, is presented. Such rather ill-classified skin eruptions as rheumatic purpura, purpura Schoenlein, Finkelstein's acute haemorrhagic edema etc. also appeared to fall under the concept a.a.c. Moreover the a.a.c.-concept has enabled us to see for the first time the close interrelationship between the various cutaneous eruptions belonging to this group.

It should be stressed that, from the beginning, in drawing up the concept a.a.c. attention was focussed on the fact that these eruptions should be regarded essentially as a form of angiitis of the skin. In this way a link with internal medicine was also established. It also appeared that these cutaneous syndromes were well suited for a study of vascular allergy in general.

Probably of equal interest is the fact that the concept a.a.c. has also contributed to a better classification of a number of poorly understood skin eruptions and cutaneous conditions previously misclassified. Thus some cases of Mucha's disease have been identified a.o. by *Krüger and Weise* as papulo-necrotic variants of arteriolitis (vasculitis) "allergica" cutis superficialis [4]. At the same time the so-called Lucio phenomenon in diffuse lepra has been recognized as being due to the presence of an additional cutaneous allergic arteriolitis (*Kamp et al.* [3]). Apparently *Latapi's* view that in this phenomenon other micro-organisms than the leprosy bacillus play an important role, is supported by this observation (see aetio-pathogenesis of a.a.c.!). Finally *Binkley* [1] came to the conclusion, that a poorly understood dermatosis as dermatitis nodularis necroticans represented a form of cutaneous allergic vasculitis. *Orbaneja* and *Pujol* made a similar observation and wrote: "It is quite possible that in future dermatitis nodularis necroticans should have to be considered as "a more ex-

tensive" variant of the papular-necrotic type of Ruiter's allergic arteriolitis".

Clinical Course and Aetio-Pathogenesis

Clinical course. As to the clinical course the following findings can be reported from 30 personal observations (chiefly adults). Attention has already been drawn to the intermittent character of the eruptions. In our patients duration varied from a few weeks to a few months or years. Associated symptoms were general fatigue and tension or stiffness in the extremities. Sometimes these symptoms preceded the cutaneous manifestations. In a number of cases the appearance of the eruptions was associated with swelling of the joints (knees, elbows, ankles and hands). Vague abdominal symptoms, too, were not uncommon. Complications in the form of internal bleedings were occasionally observed in the haemorrhagic forms. In one of the cases of the haemorrhagic type violent abdominal pain was associated with haematemesis, melaena and haematuria; thrombophlebitis migrans also occurred in this case during the course of the illness. One patient with the nodular-haemorrhagic type showed transient haematuria. In a further case (haemorrhagic type) deviations in the electrocardiogram were found during the eruption. Episcleritis occurred in one of the patients. Rise of temperature was only occasionally observed.

Etiology. As to the etiology bacteria or bacterial products are primarily considered as causative factors [5, 6, 8, 12, 15, 17, 19]. In our experience [12, 15] the skin eruptions comparatively often occurred following local infections such as tonsillitis, pharyngitis, hidradenitis, etc. In some cases there seemed to be a connection with dental granulomata, cystitis or respiratory infections. Pyogenic streptococci were foremost among the micro-organisms recovered from suspected foci and local infections. Other bacteria cultured were staphylococcus aureus, pneumococci, Esch. coli and enterococci. In cases with a pronounced chronic course (several months to a few years), the role of bacteria or bacterial products was less apparent. Accordingly elimination of suspect foci in these cases was effective only exceptionally.

In a fair number of cases the skin changes occurred following treatment of local infections with sulfa-drugs or antibiotics. As a rule these patients recovered within a short time after discontinuation of treatment. On the other hand we observed identical pictures in patients who had received no medication at all.

G. Miescher [5] is of opinion that the type of histological changes found in a.a.c. constitutes an argument in favour of bacterial products being the causative agent. Consequently he believes that chemotherapeutics or antibiotics cannot be responsible themselves, but merely act as "activators" of a focal infection in these cases. It is noteworthy, however, that *Winkelmann* [20] observed the cutaneous syndromes in patients following contact with insecticides.

Pathogenesis. As to the pathogenesis of cutaneous "allergic" vasculitis various authors assume an *allergic or related* mechanism [5, 8, 9, 12, 15, 17]. *G. Miescher* (chiefly on clinical grounds) regards the eruptions as allergic haematogenous dissemination reactions of the skin. According to *Storck* [17] the results of intracutaneous tests with bacterial filtrates also point to an allergic origin. The arguments in favour of an allergy in the more restricted sense (i.e. on the basis of an antigen-antibody reaction) on the other hand cannot be considered proved, though an association of a.a.c. with immunobiological processes does occur in a number of cases. So far, however, it has not been established to what extent these immune-biological processes can be considered causal, or whether they should be regarded as accompanying phenomena. In a number of cases [20] we found, in varying degrees and to some extent dependent on the clinical type, increased β -globulin and γ -globulin fractions in the blood proteins [12, 15]. Antibodies are known to occur preferably in these fractions. In a few of our patients genuine antibodies (Schultz-Dale technique applied according to the inverse anaphylactic principle) against leucocytes and thrombocytes were demonstrated in the serum [12, 15], although the blood cell count was normal. Moreover in several cases the Coombs' consumption test with leucocytes and/or thrombocytes was positive. The possible significance of the latter finding is studied at the moment. In periarteriitis nodosa – a vascular condition in our opinion pathogenetically related to a.a.c. – *Vorlaender* (1962) recently demonstrated (by a modified agar-gel diffusion method) precipitating serum factors against extract of homologous arterial vessels. *P. Miescher* 1956 [6]) believes to have succeeded in passive transmitting bacterial allergy in cases of leucoclastic microbids (haemorrhagic type of a.a.c.) by means of the patient's white blood cells. He regards the vascular changes as an unusual allergic tissue reaction of the delayed type, an opinion not shared by other investigators. Thus *Spier* and others suggested that the vascular alterations represent a clinical analogue of the Arthus

phenomenon. In some types also the so-called Shwartzman phenomenon might be involved. It should be pointed out that this phenomenon can occur on the basis of an antigen-antibody reaction.

As to the genesis of the vascular lesions attention should also be paid to *non-specific* stimuli. They might be called localizing resp. provocative factors. Histological examination of serial sections in our cases revealed that the most pronounced changes were found at the ramification of the dermal blood vessels affected. These findings show that local conditions of blood circulation play a part in the genesis of the vascular lesions – a fact also apparent from the clinical picture. Thus the observation that the limbs are sites of predilection for the cutaneous changes, might be explained on account of a greater risk of irregularities in the blood flow in these parts. In addition, various external stimuli appeared to promote the development of the cutaneous lesions. Thus we observed the occurrence of numerous efflorescences in the course of scratches and at pressure sites (caused by elastic bands in the form of belts, braces etc.). In one of our patients, in which skin tests with vaccines were performed shortly after the onset of a relapse, the intracutaneous injection of the control fluid (saline containing traces of carbolglycerin) caused a haemorrhagic lesion after 24 hours, in which histological changes were found identical with those of the spontaneous lesions. The same procedure, carried out in a normal subject, caused only a non-specific inflammatory reaction, histologically without any vascular changes.

Differential Diagnosis

The *differentiation* of a. a. c. includes various cutaneous conditions. As a rule no major difficulties will arise in this respect. As to an other form of “allergic” resp. necrotizing angiitis viz. the so-called cutaneous type of periarteriitis nodosa [15], which is also predominantly confined to the skin, the histopathological observations reveal that in contrast to a. a. c. more especially larger arterial branches of the muscular type situated at the cutaneous-subcutaneous border or lower in the fatty tissue are affected. Consequently the skin lesions occurring in the cutaneous type of periarteriitis nodosa for the greater part consist of deeply located nodosities and infiltrates, which may develop into ulcers. Allergic resp. necrotising angiitis with cutaneous manifestations and visceral involvement (hypersensitivity angiitis Zeek, allergic granulomatosis Churg and Strauss, “classic” peri-

arteriitis nodosa) can be distinguished from the arteriolitis group in as much as the patients are as a rule seriously ill, showing a more or less elevated temperature and the character of a multiple-organ symptom complex. At the same time among the cutaneous changes in these conditions more extensive lesions, based on involvement of deeper situated cutaneous blood vessels, like cutaneous – subcutaneous nodosities and infiltrates, disseminated patches of hemorrhagic gangrene, large tissue defects and so on may be seen.

Summary

A definition and an analysis of the concept arteriolitis (vasculitis) "allergica" cutis (superficialis) is given. Aetiology and pathogenesis of the eruptions headed under this designation are discussed.

Zusammenfassung

Es wird eine Analyse des Begriffes Arteriolitis (Vasculitis) «allergica» cutis superficialis gegeben. Die unter diesem Begriff aufgeführten Erscheinungen werden in ihrer Ätiologie und Pathogenese diskutiert.

Résumé

Une définition et une analyse du concept de l'arteriolitis (vasculitis) «allergica» cutis (superficialis) est donnée. L'étiologie et la pathogénèse des lésions désignées par ce terme sont discutées.

References

1. Binkley, G. W.: Dermatitis nodularis necrotica. Arch. Derm., Chicago 75: 387 (1957).
2. Jansz, A.: Thesis (Groningen 1960) with a summary in English.
3. Kamp, H.; Leiker, D. L. and Frenken, J. H.: The relation between the Lucio phenomenon and cutaneous allergic vasculitis (*Ruiter*). Int. J. Leprosy 30: 138 (1962).
4. Krüger, H. und Weise, H. J.: Über klinische und histologische Beziehungen bestimmter Formen der Parapsoriasis guttata zur allergischen Vaskulitis (*Ruiter*). Derm. Wschr. 140: 813 (1959).
5. Miescher, G.: Akut-entzündliche Gefäßkrankheiten und deren Auswirkung auf die Haut (vaskuläre Allergide). Arch. klin. exp. Derm. 206: 135 (1957).
6. Miescher, P.; Raymond, A. et Ritter, O.: Le rôle de l'allergie bactérienne dans la pathogénèse de certaines vasculites. Schweiz. med. Wschr. 86: 799 (1956).

7. *Ruiter, M. and Brandsma, C. H.*: Arteriolitis allergica. *Dermatologica* 97: 5/6 (1948).
8. *Ruiter, M.*: A case of allergic cutaneous vasculitis (arteriolitis allergica). *Brit. J. Derm.* 65: 77 (1953).
9. *Ruiter, M.*: Purpura rheumatica: a type of allergic cutaneous arteriolitis. *Brit. J. Derm.* 68: 16 (1956).
10. *Ruiter, M.*: Über die sogenannte Arteriolitis (Vasculitis) «allergica» cutis. *Hautarzt* 7: 293 (1957).
11. *Ruiter, M.*: Clinical and histological features of "allergic" necrotising angitiides with predominantly cutaneous localization. *Proc. 11th Int. Congr. Derm.* Stockholm 1957, vol. p. II, 172.
12. *Ruiter, M. und Oswald, F. H.*: Weiterer Beitrag zur Kenntnis der Arteriolitis (Vasculitis) «allergica» cutis (Purpura Schönlein, leukoklastische Mikrobide, anaphylactoide Purpura, Maladie trisymptomatique de Gougerot, Allergides nodulaires dermiques usw.). *Hautarzt* 14: 6 (1963).
13. *Ruiter, M.*: Vascular fibrinoid in cutaneous "allergic" arteriolitis. *J. invest. Derm.* 38: 85 (1962).
14. *Ruiter, M.*: Possible connection between an abnormal plasma fraction (H. P. F.) and vascular fibrinoid in arteriolitis (vasculitis) "allergica" cutis. *J. invest. Derm.* 38: 117 (1962).
15. *Ruiter, M.*: Cutaneous allergic vasculitis and its management. *Proc. 12th Int. Congr. Derm.*, Washington 1962, vol. II, p. 1061.
16. *Ruiter, M.*: The so-called cutaneous type of periarteriitis nodosa. *Brit. J. Derm.* 70: 102 (1958).
17. *Storck, H.*: Über hämorrhagische Phänomene in der Dermatologie. *Dermatologica* 102: 197 (1951).
18. *Vorlaender, K. O.*: Zur Klinik und Immunologie der entzündlichen und allergischen Gefäßerkrankungen. *V. European Congress of Allergy*, Basel 1962, p. 112.
19. *Wereide, K.*: Hypersensitive vasculitis of the skin. *Acta derm.-vener.* 43: 109 (1963).
20. *Winkelmann, R. K.*: Clinical and pathologic findings in the skin in anaphylactoid purpura (Allergic angitis). *Proc. Mayo Clin.* 33: 227 (1958).

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