Table I. *Patients with atopic dermatitis and Hodgkin's disease*

Case no.	Sex	Age at onset: Atopic dermatitis	Age at onset: Hodgkin's disease (yr of age)	Other signs/symptoms	Previous treatment	Type of Hodgkin's disease	Outcome
1	f	Early childhood	68	Asthma	Steroids	Granuloma	Dead, 1 yr
2	f	Early childhood	32	Urticaria; hay fever		Granuloma	Dead, 1 yr
3	m	16 yr	30		X-ray to skin	Granuloma	Dead, 1 yr
4	f	Postnatal	24	Verrucae; thrombophlebitis		Granuloma	Dead, 8 yr
5	m	8 yr	24	Hay fever/nocturnal sweatings, loss of weight	X-ray to skin	Granuloma	Dead, 5 yr

DISCUSSION

The findings in our 5 patients were remarkably uniform. Only one had fever, loss of weight and nucturnal sweatings. Four of these patients were young adults in whom adenopathy developed in addition to their atopic dermatitis. The symptoms and dermatitis responded only to steroid therapy. Elevated sedimentation rates and eosinophilia were sporadic associated findings. All 5 patients had granulomatous Hodgkin's disease, equivalent to nodular sclerosis and mixed cellularity in the current literature (2).

The concurrence of atopic dermatitis and Hodgkin's disease is uncommon. In the series of Amlot & Green (1) of 15 patients with Hodgkin's disease, 8 had atopy and only 2 of these had atopic dermatitis. However, their and our quoted cases raise the question whether chronic (atopic) dermatitis may evolve into lymphoproliferative disease. Degos (3) showed a relationship of dermatitis to mycosis fungoides and we have demonstrated a possible relationship between atopic dermatitis and Seżary's syndrome (Rajka & Winkelmann, 5). Amlot & Green did not find any relationship between other forms of lymphoma and atopy, but they did not study cutaneous lymphoma (1).

There is no direct evidence that atopic dermatitis either shields from or predisposes to tumour proliferation. A connecting such factor may be the reduced cell-mediated immunity (4) or presence of immunodeficiency in severe atopic dermatitis (6). It may be speculated that in non-atopic patients with Hodgkin's disease, mycosis fungoides, or Seżary's syndrome, the elevated IgE values may represent a direct stimulation of the IgE antibody system, a loss of T suppressor cell effect upon it, or a T helper cell effect. These mechanisms, especially the first two mentioned, might be operative also in our cases of atopic dermatitis with Hodgkin's disease.

The practical consequence of our findings is that adenopathy in chronic atopic dermatitis should not be dismissed casually.

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Nevus Oligemicus with Sensory Changes

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Abstract. After a cold bath a 16-year-old man developed livid erythema with hot anesthesia on the trunk and arm, with unilateral topography. A similar case was previously

reported by Davies as pharmacological nevus under the name of nevus oligemicus. We believe that variations in capillary blood supply induced neurological abnormalities.

Key words: Livid erythema; Nevus oligemicus; Nevus anemicus; Pharmacological nevus

In 1981, Davies et al. (1) reported the case of an acquired, persistent, fixed area of livid erythema on the trunk. The authors suggested that the lesion was in fact a functional anomaly of the cutaneous vascularization: a stasis in superficial nutritional vascularization with a global decrease in cutaneous blood flow. This case was qualified as nevus oligemicus in reference to anemic type. In nevus anemicus, on the contrary, cutaneous blood flow is maintained, though with marked decrease in nutritional blood flow (2). Both types are considered as pharmacological nevus, due to local variations in sensitivity to sympathetic mediators. The case reported by Davies et al. was not accompanied by any neurological abnormalities.

We now report a new example of nevus oligemicus with two interesting points: (i) the precise onset after a cold bath, (ii) the association with a segmental loss of temperature sensation.

CASE REPORT

A 16-year-old boy was admitted because of a livid erythema on his right arm. The lesions had appeared 2 years earlier. While taking a prolonged cold bath, the patient was suddenly affected by a sensation of numbness in the arm. The erythema appeared immediately and had remained constant since that time. Examination revealed a double cutaneous and nervous symptomatology.

Dermatological examination revealed a large area of livid, pale, cyanotic erythema with irregular borders. It was located on the back of the hand (Fig. 1), the forearm, the upper arm, the adjacent part of the shoulder and the flank, with an absolute right unilateral topography. The skin was cold in the erythematous zone, especially at the extremity of the member. The distribution was fixed and blanched under light pressure. On comparison with healthy zones, sweating and hair distribution were the same in the affected zone.

Neurological examination (Prof. Rascol, Department of Neurology) demonstrated sensitivity anomalies at the erythema: loss of hot sensation, especially at the extremity of the member; loss of cold sensation in the entire nevus zone. These sensitivity disorders appeared progressively one year after the appearance of the erythema. Both deep and tactile sensations were normal. Other peripheral neurological signs were negative: neither amyotrophy nor modifications of reflexes. There were no central neurological abnormalities. Electrical and radiological examinations were normal and the rest of the general examination was also normal.

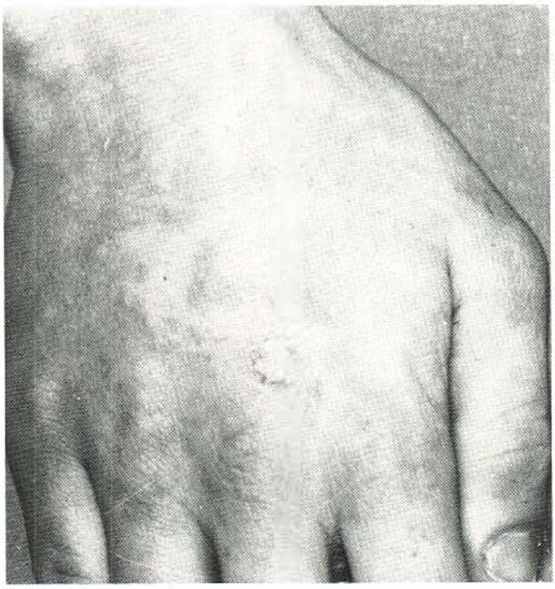

Fig. 1. Livid erythema on the back of the right hand, with a livedo-like appearance.

The histological examination (Fig. 2) of the erythematous zone revealed few anomalies. The number of dermal vessels was normal, but careful examination revealed the complete and paradoxal obstruction of the capillaries in the reticular dermis: the vessels were reduced to a mass of endothelial cells. Papillary vessels, on the other hand, were dilated, with a hyperplastic endothelium.

Capillaroscopy (Dr Boccalon, Vascular Hemodynamics Service) showed a normal density and morphology of vessels, which nevertheless appeared abnormally dilated. Cutaneous vascular blood flow was examined by plethysmography after inducing ischemia in the arm (Dr Boccalon): the hyperhemia reaction was reduced at the extremity of the member.

Pharmacological tests for vasomotricity estimation were performed with histamine (axon reflex inducer), phentolamine (α-blocking) and propanolol (β-blocking). Comparable results were obtained in the erythematous and healthy zones, except for phentolamine which caused a more pronounced rubefaction in the territory of the nevus. The lesions did not disappear after the application of corticosteroids. Local xylocaine injection had no effect on the erythema. Response to thermal stimuli was examined with cold (4°C) and hot (45°C) baths. When the pathological zone was immersed in cold water, there was no change, but the intensity of erythema increased in hot bath.

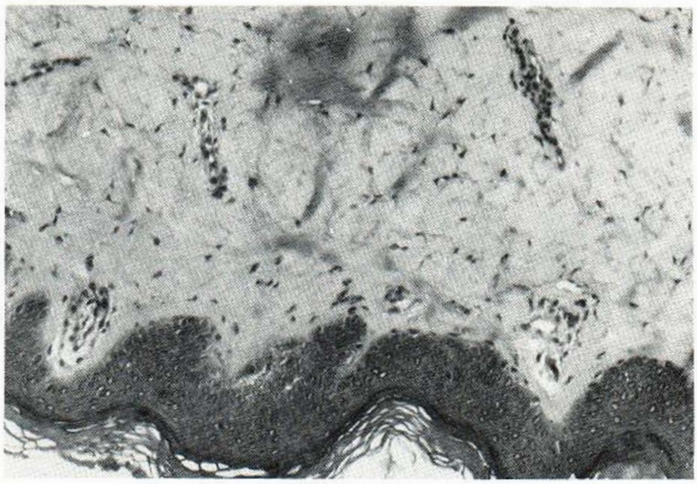

Fig. 2. Histopathologic features of livid erythema, showing dilated vessels in pars papillaris and a paradoxal constriction of the vessels in pars reticularis (×100).

DISCUSSION

Our case report corresponds perfectly to the description of nevus oligemicus of Davies et al. Clinically, this entity is an acquired cyanotic livid erythema. Histological and vascular examination revealed a vasodilation of the superficial nutritional vascularization, combined with reduced skin temperature and reduced blood flow following ischemia. All these findings correspond to a stasis at the level of nutritional circulation, with a pronounced vasoconstriction of the deep vessels.

In the present case the appearance of a livid circumscribed erythema after a prolonged cold bath suggests the existence of vascular receptors with low sensitivity threshold or the excessive circumscribed release of mediators. The following hypothetical sequence of events may be proposed: 1) the pre-existence of an abnormal "naevic" zone, clinically asymptomatic 14 years ago, fulfilling a latent pharmacological nevus with latent abnormality of pharmacological receptors; 2) the discovery of this pharmacologically abnormal zone under the effect of a particularly intense thermal stimulus (a cold bath), with appearance of livid erythema induced by rough vasoconstriction; 3) the vasoconstricted zone then becomes autonomous, being excluded from the normal regulating process. This latter hypothesis is favored by the fact that we found both hyperplasia of the endothelial cells and the impossibility of suppressing the livid erythema with pharmacological agents.

The associated neurological manifestations were clinically very remarkable, both by their dissociated symptomatology, since they involved only certain sensations, and by their topography which was strictly located to the erythematous zone.

These clinical features and the negative neuroradiological and electrical results enable us to distinguish this case from a neurocutaneous syndrome and from a nevus anemicus. The delayed occurrence of neurological manifestations in relation to the occurrence of erythema and vasoconstriction suggests a receptor response anomaly induced by the pharmacological nevus. The exact relationships between blood supply and neurological receptor activity remain to be established. With Waterston (3) we think that "the local fluctuations sensitivity to a stimulus may correspond to fluctuations in the activity of sensitive nerve endings, due to variations in capillary blood supply".

In conclusion, our observation must be included in the entity named nevus oligemicus, whose individuality appears to be unambiguous. It takes the form of a peculiar type of pharmacological nevus electively affecting the vascular response to sympathetic mediators and which may be compared to nevus anemicus. Our observation establishes two elements: the onset triggered by a cold bath and the association with sensitive disorders. This double origin has led us to propose an original pathogenic mechanism.

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Herpes Zoster in a 6-month-old Infant

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Abstract. A case of herpes zoster occurring in infancy is reported. The clinical picture was characteristic and the virological studies confirmed the diagnosis. The course was uneventful. The mother had varicella during the second trimester of pregnancy. This report is in accordance with the notes that herpes zoster in infancy is benign and the recovery is rapid and without sequelae.

Varicella and zoster are caused by the same virus, *Herpes varicellae*. Varicella is the primary infection with *H. varicellae*, whereas zoster is the result of reactivation of residual latent infection, usually of sensory neurones, infected by the viraemia of chicken pox. The virus can replicate and invade the sensory nerve and the skin around the sensory nerve endings. This mechanism explains the typical lesions, clusters of vesicles on an erythematous base of the area of the dermatome (7).

Maternal varicella may result in one of the following three clinical syndromes: early onset of postnatal varicella which may vary from a typical varicella to a fatal disseminated infection (3); intrauterine infection may rarely lead to severely affected infants who may display multiple congenital anomalies (3, 6); or herpes zoster, which may appear months or years after birth (1, 2, 4, 8, 9, 10).

We present a case of herpes zoster in a 6-month-old infant, whose mother had varicella during the second trimester of the gestation.

REPORT OF A CASE

A 6-month-old infant was brought to the Department of Dermatology of the University Central Hospital of Turku because of a rash characterized by groups of vesicles on an erythematous base situated along the distribution of the right dermatome C 2. The infant was otherwise asymptomatic. The typical zoster rash resolved in 2 weeks. The mother's sister and her son, who had visited the home at the beginning of the herpes zoster infection of our patient, showed the typical varicella infection 2 weeks later.

Virological findings

Virus isolation was attempted from a typical vesicle with fluid, but it was negative. Indirect immunofluorescence against varicella-zoster in the cells scraped from the bottom of the vesicles was positive (with some fluorescence against herpes simplex, probably due to cross-reaction).

In the complement-fixation test, no antibodies were measurable to VZ in the mother or the infant at the time of eruption, 6 months after parturition. However, in radioimmunoassay of VZ-specific IgG class antibodies both the mother and the child were seropositive, as also were the contacts. On the other hand, herpes simplex antibodies were found only in the mother's sister by radioimmunoassay.

COMMENT

Although transplacental passage of VZV from mother to fetus is probably a rather frequent event in cases of varicella during pregnancy (5), the low incidence of congenital varicella syndrome shows that the fetus is relatively resistant to infection. Some immunological disorder in the mother could therefore be assumed to be a co-factor for the development of the infection in the fetus.

Our patient was exposed to varicella *in utero*. The clinical picture, the spread of virus to relatives from the index case and the virological studies confirmed that the infant suffered from herpes zoster. The appearance of the zoster in the 6-month-old infant coincided with the disappearance of the transplacentally acquired VZ antibodies.

This report is in accordance with the notes that herpes zoster in infancy is benign and that the recovery is rapid and without sequelae. Our report further documents one aspect of the relationship between the maternal varicella and the occurrence of herpes zoster early in life.