Frequency of Cataract in Atopic Dermatitis

Jens Dahl Christensen

Department of Dermatology, Copenhagen County Hospital, DK-1353, Copenhagen, Denmark

Received May 5, 1980

Abstract. Atopic cataract (AC) has been reported to occur as many as one patient in every five with atopic dermatitis (AD). The lenticular opacities may rapidly lead to blindness. To establish whether in Denmark screening of AD patients is justified especially for AC, 51 such patients underwent ophthalmological examination. None of them were found to have AC. Although the study does not exclude a certain incidence of AC, there is nothing to indicate that routine ophthalmological examination of patients with AD is required.

Key words: Atopic cataract; Atopic dermatitis

Atopic cataract (AC) associated with atopic dermatitis (AD) has been described in case reports and in larger materials in which AC has been found in up to 21% of the patients (6). The purpose of the present study has been to establish whether in Denmark routine ophthalmological screening of all AD patients under dermatological care is justified.

MATERIAL AND METHOD

Over a 3-month period 51 patients (33 females, 18 males) entered the study. All of them had had AD for at least 5 years and at least 10% of their skin surface was affected. The youngest patient was 16 and the oldest 39 years, mean age 24.6 years. All the patients underwent ophthalmological examination, especially for cataract.

RESULTS

AC was not demonstrated in any of the patients, but other ocular disorders were detected in 17. One had bilateral, probably congenital, cataract made up of small grey dot-like opacities in the middle between the anterior capsule of the lens and the central Y-shape coincident with the extent of same. Three had conjunctivitis, and 13 patients had refractive errors.

DISCUSSION

From 8 cases of AC and a review of the literature, Cowan & Klauder (2) stated that the characteristic changes of AC are usually bilateral and begin with flattened vacuoles and yellowish opacities at the posterior pole of the lens immediately in front of the capsule. At the same time, or soon afterwards, opacities are also seen in the anterior subcapsular zone. Later the whole cortex becomes opaque. In AC, shield-shaped opacities have been described in the anterior subcapsular zone (4), but according to Wardingburg et al. (11) these changes are not pathognomonic of AC.

Table I. *Frequency of occurrence of atopic cataract according to different authors*

	Examined AD patients	Frequency (%)	Comments
Brunsting et al., 1955 (1)	1 158	12	(*a*)
Cowan & Klauder, 1950 (2)	100	8	
Ingram, 1955 (3)	60	17	(*b*)
Karel et al., 1965 (5)	128	2	(*a*)
Katavisto, 1949 (6)	58	21	(*b*)
Kornerup & Lodin, 1959 (7)	100	3	(*a*)
Korting, 1960 (8)	509	0.4	(*c*)
Sprafke, 1966 (10)	1 414	0.4	(*c*)
Present data	51	0	

(*a*) Observed lens changes are mainly in agreement with the description of Cowan & Klauder (2).
(*b*) Not all ophthalmological information is available.
(*c*) Observed lens changes are mainly in agreement with the description of Janzen (4).

The statements with regard to the frequency of AC are varying and are given in Table I. Sautter (9) estimated AC to develop on average 5 years after the outbreak of the skin disease. Brunsting et al. (1) found the age of the patients at the time the cataract was detected or symptoms appeared to be as follows: about 30% were teenagers, about 50% were in their twenties, and about 15% in their thirties. Approximately 20% became blind within a rather short time, i.e. in the course of a period ranging from 12 days to one year. Another few per cent became blind within 10 years. Previous X-ray treatment was found to be unconnected with the development of the cataract. As this study was carried out on a very selected material at the Mayo Clinic, Minnesota, it does not allow generalization, however.

The present study revealed one case of cataract, but the lenticular changes observed in this patient did not resemble any of those previously described in atopic cataract cases.

Even though the result of the present study does not rule out a certain incidence of AC (5.7% with

0.95 and 8.6% with 0.99 confidence probability, respectively), it does not suggest either that routine ophthalmological screening of all patients with AD is indicated.

ACKNOWLEDGEMENTS

Thanks are due to Drs B. Albrectsen, R. Andersen, S. La Cour Andersen, H. Bisgaard, L. Bundgaard, T. Castellani, I. Grunnet, L. Hallinger, N. Hjorth, B. Borch Jørgensen, H. Kopp, H. Ekkert Knudsen, A.-M. Niordson, I. Borup Svendsen and J. Tissot for referring their patients.

REFERENCES

1. Brunsting, L. A., Reed, W. B. & Bair, H. L.: Occurrence of cataracts and keratoconus with atopic dermatitis. Arch Dermatol *72:* 237, 1955.
2. Cowan, A. & Klauder, J. V.: Frequency of occurrence of cataract in atopic dermatitis. Arch Ophthalmol *43:* 759, 1950.
3. Ingram, J. T.: Besnier's prurigo: An ectodermal defect. Br J Dermatol *67:* 43, 1955.
4. Janzen, I.: Die Cataracta juvenilis bei endogenem Ekzem. Inaugural dissertation, p. 17. Breslau, 1940.
5. Karel, I., Myška, V. & Kvičalová, E.: Ophthalmological changes in atopic dermatitis. Acta Dermatovener (Stockholm) *45:* 381, 1965.
6. Katavisto, M.: Prurigo diathésique Besnier and cataract. Acta Ophthalmol *27:* 581, 1949.
7. Kornerup, T. & Lodin, A.: Ocular changes in 100 cases of Besnier's prurigo (atopic dermatitis). Acta Ophthalmol *37:* 508, 1959.
8. Korting, G. W.: Einige Wesenszüge des endogenen Ekzematikers. Dtsch Med Wochenschr *85:* 417, 1960.
9. Sautter, H.: Die Trübungsformen der menschlichen Linse, p. 88. Georg Thieme Verlag, Stuttgart, 1951.
10. Sprafke, H.: Cataracta iuvenilis syndermotica. Dermatol Wochenschr *152:* 928, 1966.
11. Waardenburg, P. J., Franceschetti, A. & Klein, D.: Genetics and Ophthalmology, vol. I, 902 pp. Van Gorcum, Assen, Netherlands; Blackwell Scientific Publications, Oxford; Charles C. Thomas, 1961.

Pyoderma Gangrenosum Associated with Transient Acantholytic Dermatosis (Pemphigus Erythematosus-like) and Paraproteinemia

Bo Lasthein Andersen

Department of Dermatology, the Finsen Institute, Copenhagen, Denmark

Received March 21, 1980

Abstract. A 69-year-old man with recurrent eruptions of pyoderma gangrenosum for 4 years is described. The patient also suffered from paraproteinemia (increased IgA with M-component) and transient acantholytic dermatosis resembling pemphigus erythematosus. He had no intestinal symptoms.

Key words: Pyoderma gangrenosum; Paraproteinemia; Subcorneal acantholytic bulla formation; Pemphigus erythematosus-like eruption; Transient acantholytic dermatosis

Pyoderma gangrenosum is an ulcerating inflammatory skin condition of unknown origin, but with typical clinical characteristics. Pyoderma gangrenosum is considered to be an auto-immune disease and is often accompanied by chronic ulcerative colitis, rheumatoid arthritis and paraproteinemia.

Pemphigus erythematosus is also regarded as an auto-immune disorder. It is extremely rare in Denmark.

This case report presents a patient suffering from pyoderma gangrenosum, paraproteinemia, and transient acantholytic dermatosis resembling pemphigus erythematosus.

CASE REPORT

A 69-year-old man with no family history of skin disorders. At the age of 65 years the patient developed a painful bluish discoloration on his left lower leg without any preceding trauma. Treatment with systemic and topical antibiotics was instituted, but he developed a putrid, necrotic ulceration measuring 14×16 cm. The edges were violaceous, elevated and undermined (Fig. 1). The patient had no intestinal symptoms.

Histopathological examination showed pseudoepithelial hyperplasia and dermal abscesses with inflammatory infiltrate of neutrophil granulocytes and lymphocytes. Many fibroblasts and blood vessels were present. Fibrinoid necrosis was present in the vascular wall.

The patient was treated with salicylazosulphapyridine 3 grams daily and topical antibiotics. As this treatment was without effect, a split-skin transplantation was performed, and the ulcer healed.

One year later the patient developed non-pruritic erythematous crusted skin lesions, mainly on the trunk, and for a short time there were facial lesions too. In some places small bullae were present, but no papules. The lesions healed spontaneously, forming brownish crusts.

Histopathological examination revealed an acanthotic epidermis, subcorneal bulla formation with neutrophil granulocytes and acantholytic cells, and in the dermis perivascular infiltration with lymphocytes and granulocytes was found. Direct immunofluorescence microscopy (from several lesions on the trunk) demonstrated only fluorescence of complement C3 at the dermo-epidermal junction. Indirect immunofluorescence microscopy was negative. Fluorescence could not be demonstrated in skin biopsy specimens from normal skin.