POST-OPERATIVE GANGRENE OF EYELID*

BY

ALY MORTADA

Department of Ophthalmology, Faculty of Medicine, Cairo University, Egypt

PROGRESSIVE post-operative gangrene is a rare but clinically typical phenomenon of secondary infection of a surgical wound. It is usually abdominal or thoracic (Sutton, 1956), and has occurred most often soon after the drainage of an appendix abscess or empyema (Illingworth and Dick, 1945). Post-operative gangrene has rarely been reported to affect the eyelids and the description of the following case is therefore of ophthalmological interest.

Case Report

A 45-year-old woman had a peculiar oedema of the right upper lid with brownish discoloration of the skin and ptosis (Fig. 1). The condition occurred 2 weeks after incising an abscess in the inner part of the affected lid.

FIG. 1.—Appearance of right upper lid 2 weeks after incising a lid abscess in a 45-year-old woman.

History.—There was no important past or family history. There was no history of dysentery or conjunctivitis. The affected eyelid had not been exposed to trauma, burn, caustics, insect bite, pressure, cold, heat, or radiation. There was no intermittent claudication, cyanosis, or sensation of cold fingers, brittle nails, or oedema of legs. No drugs such as ergot had been taken by mouth or by injection. There were no manifestations of allergy, neurological diseases, leprosy, syphilis or tuberculosis. The mouth and genitalia had normal skin and mucous membrane. Apart from the affected lid the skin of the body showed no signs of infection or ulceration.

Examination.—The patient was anaemic and debilitated. The blood pressure was normal, pulse 85, temperature 37.5°C. There were no septic foci in the body. The chest, cardiovascular system, and abdomen showed no abnormality. The urine was free of sugar and contained a trace of albumen. The blood sugar curve and plasma lipoids were normal. The blood Wassermann reaction, and tuberculin tests were negative. The blood count showed: red blood corpuscles 4,610,000; white blood corpuscles 6,800; basophils 0 per cent.; eosinophils 4 per cent.; staff nucleated 10 per cent., segmented 58 per cent. (total polymorphs 68 per cent.), lymphocytes 24 per cent., monocytes 4 per cent. Blood film negative for malaria. Haemoglobin 65 per cent.

The left eyelids and globe were normal. The fundus was normal. There was no arteriosclerosis of the retinal blood vessels. The visual acuity was 6/9. A conjunctival smear culture from the left eye was negative for organisms.

* Received for publication April 25, 1963.

The right upper lid was painful, showing severe oedema, brownish skin colour, and offensive odour. Necrosis affected the lid skin, subcutaneous tissue, and palpebral part of orbicularis oculi but not the underlying bone. The slowly spreading area of lid necrosis was surrounded by an inner zone of purplish skin and an outer red zone. Ptosis prevented examination of the right eye. The right lower lid was oedematous and red. The right pre-auricular lymph gland was enlarged, tender, soft, and not adherent to the surrounding structures. The submaxillary and cervical lymph glands were not enlarged. Cultures of the lesion swab showed infection by micro-aerophilic non-haemolytic streptococci and *Staphylococcus aureus*. Blood culture was negative for organisms.

Therapy.—With diathermy a deep “fire-break” was made in the normal skin around the edge of the gangrenous area. Treatment was continued by penicillin injections, sulpha tablets by mouth, and Terramycin ointment to the gangrenous area. As supportive measures vitamins A, B complex, and C, and iron were given.

Progress.—One week after this treatment a line of demarcation appeared between the living and necrosed tissues of the lid. Necrosed skin, subcutaneous tissue, and the palpebral part of the orbicularis oculi became mummified, showing as a black thick crust (Fig. 2). A marginal lid strip was not affected.

One week later the black crust separated leaving a large lid ulcer (Fig. 3), with an irregular edge, occupying an area 5 mm. from the lid margin to the orbito-palpebral fold and affecting the whole breadth of the lid. The base of the ulcer was covered by sloughs.

FIG. 2.—Line of demarcation between living and gangrenous skin one week after treatment.

FIG. 3.—Large skin ulcer after separation of gangrenous area of lid.

2 weeks later the ulcer showed epithelialization at the edges, and fresh granulation tissue in the base (Fig. 4). The oedema of the lid subsided and the pre-auricular lymph gland was not palpable. The body temperature was 37°C. The right eye was normal, with visual acuity 6/12.

FIG. 4.—Granulating lid ulcer 2 weeks after separation of gangrenous area.

Result.—After 2 more weeks the lid ulcer healed completely, but left a scar producing slight right upper lid cicatricial ectropion. This was later treated by temporary tarsorrhaphy, excision of the lid scar, and application of a skin dermo-epidermal graft taken from behind the ear.

Discussion

Ingram and Brain (1957) wrote that cutaneous gangrene might be due to the following causes:

- (1) Severe trauma.
- (2) Physical causes, such as intense heat and cold, prolonged exposure to x rays and radium, or a powerful electric current.
- (3) Chemicals such as strong acids and alkalis.
- (4) In rare cases, carbon monoxide poisoning, chloral hydrate, iodides, and arsenic.
- (5) Virulent bacterial infection, as in dermatitis gangrenosa pyogenica or infantum.
- (6) Diabetes.
- (7) Nervous disorders such as syringomyelia, nerve leprosy, and in association with pressure.
- (8) Interference with or suppression of the blood supply, by pressure on the vessels by neoplasms or exudate, contraction with occlusion of arteries in ergotism and Raynaud's disease, diseases of the intima or vessel walls as in endarteritis obliterans, syphilitic endarteritis, atheroma, periarteritis nodosa, or as obstruction of the lumen of vessels by thrombus or embolus.

Dry Gangrene.—This is rare in the lids (Duke-Elder, 1952). It occurs in absence of infection when the tissues are drained of fluids before their death, as is seen in an interruption of the circulation by trauma or complete thrombosis, in crushing injuries, burning, or freezing.

Moist Gangrene.—This is the more common manifestation in the lids and occurs as a septic or putrefying process, in debilitated persons, cases of diabetes, and children suffering from infectious diseases such as measles or scarlet fever (Axenfeld, 1904). It may follow infected wounds, abscess, erysipelas, or eczema of the lids (Possek, 1907). It may be a complication of smallpox. It may follow severe diphtheritic or gonococcal conjunctivitis (Elschnig, 1893). The most common infecting organisms are the streptococcus, staphylococcus, pneumococcus, diphtheria bacillus, and as a rarity *B. proteus* (Mohamed, 1934) but in many instances no definite aetiology has been determined and bacteriological investigation has proved negative. Much more rarely gangrene may follow an insect bite (Pes, 1904).

Egyptian Gangrene.—This is a characteristic form reported from North Africa from time to time. It appears to develop spontaneously and without apparent cause at any age period in either sex. Nine cases were collected in Egypt by El-Seesy (1937). Organisms of the proteus group have been found in some of these cases (Barrada and Mohamed, 1935), but it may be that the disease is of a virus origin.

Palpebral Gangrene of the Newborn.—This has been reported in cases of diphtheria, purulent ophthalmitis, and syphilis, and has invariably been progressive and fatal (Pereira and Conti, 1954).

Progressive Post-operative Bacterial Synergistic Gangrene.—This usually begins around some surgical drainage wound (Marshall 1960), and may spread for many months.

Summary

A rare case of post-operative gangrene of the upper lid followed the incision of a lid abscess. Cultures showed the causal organisms to be symbiotic microaerophilic non-haemolytic streptococci and *Staphylococcus aureus*.

REFERENCES

- AXENFELD, T. (1904). *Klin. Mbl. Augenheilk.*, **42** (1), 576.
 BARRADA, M. A., and MOHAMED, I. H. (1935). *Bull. ophthal. Soc. Egypt.*, **28**, 46.
 DUKE-ELDER, S. (1952). "Text-book of Ophthalmology", vol. 5, p. 4807. Kimpton, London.
 ELSCHNIG, A. (1893). *Klin. Mbl. Augenheilk.*, **31**, 191.
 EL-SEESY, A. M. (1937). *Bull. ophthal. Soc. Egypt.*, **30**, 120.
 ILLINGWORTH, C. F. W., and DICK, B. M. (1945). "A Text-book of Surgical Pathology", 5th ed., p. 23. Churchill, London.
 INGRAM, J. T., and BRAIN, R. T. (1957). "Sequeira's Diseases of the Skin", 6th ed., p. 149. Churchill, London.
 MARSHALL, J. (1960). "Diseases of the Skin", p. 187. Livingstone, Edinburgh.
 MOHAMED, I. A. (1934). *Rep. Giza mem. ophthal. Lab.*, **9**, 142.
 PES, O. (1904). *Z. Augenheilk.*, **12**, 438.
 PEREIRA, R. F., and CONTI, A. (1954). *Arch. Ofal. B. Aires*, **29**, 503.
 POSSEK, R. (1907). *Klin. Mbl. Augenheilk.*, n.s. **3**, 211.
 SUTTON, R. L. (1956). "Diseases of the Skin", 11th ed., p. 294. Kimpton, London.