TANGEMAN (C. W.)

Tangeman bullosa

TANGEMAN (C. W.)

KERATITIS BULLOSA.

By C. W. TANGEMAN, Cincinnati, Ohio,

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY AT THE MEDICAL COLLEGE OF OHIO.

THIS disease, though not of very frequent occurrence, has been described by a number of observers in the last century. Cases have been observed and reported from time to time, and the subject has received considerable attention, experimentally, at the hands of Prof. Leber, and yet the etiology of this disease is quite obscure. The scarcity of clinical material has made it difficult to study the affection with any degree of satisfaction. Experiments on the eyes of animals have yielded but few data, except to disprove the erroneous theory of Landesberg, who advanced the idea that the formation of vesicles on the cornea is purely mechanical, caused by a force from behind, as in increased intra-ocular pressure. It is now definitely fixed that, no matter how great the intra-ocular pressure, the fluid in the anterior chamber does not cause œdema of the cornea, or is not permitted to pass through the membrane of "Descemet" unless the surface be broken. In a large number of the reported cases of "keratitis bullosa" there was neither increased intra-ocular pressure nor inflammation of the uveal tract recorded.[1] Saemisch makes an infiltration of the parenchyma of the cornea the first stage in this disease. He says that all the difference there is between parenchymatous infiltration of the cornea and keratitis bullosa is that in the latter, some time during the course of the disease, there is a formation of large vesicles, larger than phlyctenular vesicles, and not so round or regular in shape. Graefe,[2] when making a microscopic examination of the anterior wall of the described vesicle, finds that not only the epithelial layer, but also the anterior elastic membrane and some fibres from

[1] Graefe and Saemisch: *Handb. d. Ges. Augenhk.*
[2] Graefe: *Arch. Ophth.*, ii., 1, 206.

Reprinted from the Archives of Ophthalmology, Vol. xvii., No. 1, 1888.

the parenchymatous layer of the cornea, is separated to make up the covering of the vesicle. Possibly the smallest number of cases would show an involvement of the cornea to that depth. Usually, as Schweigger[1] states, he never *found* that more than the anterior epithelial layer was separated in this disease. Bullous keratitis must be differentiated from the various forms of vesicular keratitis or herpes cornea, which may occur without any previous inflammatory disturbance. It is safe to say that we have to deal with a separation of the superficial layer only in all of these various forms of inflammation; the contents are the same, but the condition of the cornea and the circumstances under which each form is developed are different. Warlemont, McKenzie, and others have described this disease as dropsy of the cornea,—a formation of vesicles or blebs, due to an escape of fluid from the anterior chamber through the corneal tissue. While this theory has been disproven, nothing satisfactory has been substituted. Since a few cases have been reported of keratitis bullosa in glaucomatous eyes, the tendency has been to connect the etiology of the two diseases. Degenerative changes of the epithelial cells, causing an enlargement of the intercellular spaces, has most recently been advocated as the chief process that is going on in the corneal tissue, which looks so much like a parenchymatous inflammation of the cornea. Michel reports cases where he observed the formation of vesicles quite large on a cornea otherwise perfectly healthy.

The patient that came under the writer's observation at Prof. W. W. Seely's clinic is of special interest, since the vesicles appeared on both eyes simultaneously, and on an otherwise perfectly healthy cornea.

Mr. B., æt. thirty, laborer, white, robust and healthy, complained of a disturbance of his sight. Since the past week he had suffered acute attacks of pain, which seemed to get better one day and worse the next. A careful examination of his eyes showed three or four vesicles on each cornea, quite large, filled with a clear fluid. The patient said he had had previous attacks of a similar nature, and he had seen "blisters" form on the sight,

[1] Schweigger: Handbuch, 1873.

that would pass away again. The cornea showed distinct traces of an apparently superficial abrasion. Aside from these changes nothing abnormal could be found about the cornea. The chief complaint was an intense burning sensation and some redness of the ocular conjunctiva. The tension was normal, and the depth and contents of the anterior chamber looked natural. The patient was not suffering from any constitutional affection, but gave a distinct malarial history or tendency.

On the second day later, he presented himself again, and some new bullæ had formed. The patient was now put on large doses of quinine, with the effect of removing all symptoms of irritation, and preventing a recurrence of the vesicles on the cornea. The patient was kept under observation for some time, but there was not even a tendency to a return of the symptoms above recorded.

Here, now, we have a case of a rare disease, carefully watched, with an intermittent tendency, being absolutely cured upon the administration of an antiperiodic. Does this indicate any thing? Graefe, Schultze, Saemisch, and others have recorded cases with distinct periods of intermission, but fail to speak of the possibility of malaria having any etiological connection with this affection. In the above case all of the diseases usually given as causative of vesicular keratitis, viz., glaucoma, iritis, ichthyosis, inflammation of the uveal tract, degeneration of the corneal epithelium, corneal infiltration and enlargement of the intercellular spaces, engorgement of the lymph spaces, increased intra-ocular pressure, or a mechanical force, were absent. Hansen some time since described a case of intermittent vesicular keratitis, but insists that it was caused by trauma to the corneal epithelium.

How these vesicles should occur on the cornea due to malaria is difficult to say, yet it would be no more impossible than when they follow vaso-motor disturbances (Michel), or when we have a serous effusion into the vitreous (Seely[1]), or, finally, in the case reported by Perlia,[2] where the formation of the vesicles was due to vaso-motor changes[3] caused by migraine.

[1] Transactions of the Amer. Ophth. Soc., 1883.
[2] Zehender: *Monats. Bl.*, 1888.