Hist. Ophthal. intern. 3. 273 – 286 (1985)

HILARIO DE GOUVÊA (1843 — 1923)

(OPHTHALMIA BRASILIANA — XEROPHTHALMIA CACHECTICA)

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Well before CELSUS, who said: "Est etiam genus aridae lippitudinis: xerophtalmia Graeci appelant", the Greeks spoke of xerophtalmia. HIPPOCRATES (460 B.C.) advised eating raw liver to cure nightblindness, and there are references to this in an Egyptian papyrus (150 B.C.). Hepatotherapy is, indeed, very old.

It is not necessary here to follow the evolution of the literature on this subject to the beginning of the 20th century when the vitamins — a name suggested by FUNK (1911 – 1914) — were described. But we must mention BITOT's spots because of their significance to our subject. In 1863, this French ophthalmologist described these spots in 29 debilitated orphans in Bordeaux, although HUBBENET had already recorded them in 1860.

Works were published demonstrating the concomitance of night blindness and xerophthalmia as well as its association with a precarious state of nutrition. There were also experiments such as the one conducted by MAGENDIE (1816), who produced ulcerated corneas in dogs by giving them a ration low in carbohydrates.

Still, the subject was obscure. It suffices here to recall BESOLD's (1874) description of the xerosis bacillus (Corynebacterium xerosis), which was considered responsible for xerophthalmia. Today, this bacillus is known to be a non-pathogenic saprophyte that, at the most, can participate in Bitot's spots.

This introduction is intended to justify the discussion of a memorable work of HILARIO DE GOUVÊA, the illustrious Brazilian ophthalmologist, that was presented at a European congress and was published in VON GRAEFE's Arch. Ophthal. 29 (1):167; 1883. This work, which had widespread repercussions, associated xerophthalmia, Bitot's spots, and night blindness with malnutrition (ophthalmia brasiliana, according to GAMA LOBO).

We will present here the prinicipal points of this historic communication, though we should prefer to reproduce the integral text, if its size did not make this impracticable. We will consider in turn:

1. The bibliographical data of HILARIO DE GOUVÊA *(fig. 1)*.
2. Comments and a partial transcription of his publication of 1883.

The Bibliographical Data (1843 – 1923)

HILARIO DE GOUVÊA was an extraordinary personality. His name was recently listed by BLODI (Vol. I, 1979, Part. II of Historia Ophthalmologica Internationalis) among the ophthalmologists whose pictures were engraved for postage stamps *(fig. 2)*. The stamp that Blodi reproduced, and which we reproduce here, commemorates the "Ligue Paulista contre la Tuberculose". The stamp represents the homage rendered by another specialty of medicine to a great ophthalmologist. His contribution to phthisiology — as an author of a major work on tuberculosis and as a founder of the Brazilian Anti-Tuberculosis League — was great.

HILARIO DE GOUVÊA was born in Caetè (Minas Gerais, Brazil) on 23th September 1843 and died in Rio de Janeiro on 25th October 1923. A graduate of the Faculty of Medicine of Rio de Janairo (1866), he submitted a thesis on glaucoma. He was Professor of the ophthalmological clinic (1883 – 1895) and, a few years later, of the otorhino-laryngological clinic (1911). He was one of the organizers of the first Brazilian Medical Congress (1887), and a founder of the "*Revue d'Ophtalmologie*" (1888). He was involved in an important reformation of medical education in Brazil (1881). After four years at the University Clinic at Heidelberg, he was invited to lecture, but he refused in order to return to Brazil.

His political life was eventful. He fought intensely for the adhesion of Brazil to the International Convention of the Red Cross at Geneva. He was accused of helping insurgents and of working for the monarchy. For this reason, he was


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HILARIO DE GOUVÊA

sentenced to prison. He felt that his life was then in danger so, with friends, he prepared his escape for 18th October 1893. The escape was tumultous and fraught with danger, his life being at risk at every instant. He was given refuge on the frigate "Arethuse" under the protection of the French flag before transferring to the steamer, "Brésil", which took him to Paris.

Arriving in Paris on the first of December, GOUVÊA set out to practice medicine and succeeded in having his diploma validated with honors, with masters such as PANAS and POTAIN among his examiners. He then defended a thesis entitled: "La distomatose pulmonaire par la douve du foie" (Pulmonary distomatosis caused by liver flukes).

He practiced medicine in Paris until he returned to Rio de Janeiro in 1899. Though he had been a refugee, he retained no rancor against his former enemies, and the memory of his country sublimated his exile.

He was a true genius. He was the first professor of the ophthalmological clinic at Rio de Janeiro (1881) and, on his return to Brazil, he organized and occupied the first chair of otorhino-laryngology at the same school (1911).

A few days before his death, still practicing medicine, one of his legs had to be amputated at the thigh because of diabetes. He died on 25th October 1923.

He published innumerable works in Brazil and elsewhere. Today, we shall present the one concerned with xerophthalmia and night blindness, which noted clearly the involvement of nutrition. With this work, he has left us the mark of genius.

Beiträge zur Kenntnis der Hemeralopie und Xeroophthalmie aus Ernährungsstörungen (Contribution to the study of night blindnesss and xerophthalmia as a result of nutritional difficulties), von Graefe's Arch. Ophth. 29, 167 – 200, 1883. DE GOUVÊA was able to observe especially among slaves, a clear relationship between nutritional disorders, xerophthalmia, and night blindness. He was also able to observe tragic cases in which the cornea had been destroyed (keratomalacia), and, with a richness of detail, the clinical picture already described by Bitot (whitish, foamy or grease-like, dried out, lustreless, dull spots). The spots were located close to the cornea. He also noted the insensitivity of the opacity of the corneas, which are the precursors of softening.

We shall now turn to the most expressive and documented points of his work. In 1869, DE GOUVÊA observed his first case of xerosis conjunctivae bulbi, which ended fatally. He was already familiar with this disease because of a report published by GAMA LOBO (Annaes Brasileiros de Medicina), who called it ophthalmia brasiliana because he considered it to be related to the climate.

We shall summarize as well as we can the first case De Gouvêa observed, because this case was a point of departure for him. Thereafter, he could combine a great number of other cases that reflected different phases of the disease, thus giving us a global idea of the evolution of the disease and allowing valuable conclusions to be drawn (1883).

De Gouvêa's First Case

The patient was a negress, 4 years old, a slave, and from Rio de Janeiro. At first, for several months, the child could not orient herself after sundown. At the first examination, the little girl showed complete indifference to everything around her. Her skin, particularly that of the extremities, was desiccated, insensitive, glossy and scaly, and wrinkled by a great number of fine folds like the skin of an old person; the subcutaneous tissue was atrophici; the muscles were flabby, and the temperature was 39° C; the abdomen was bulging and very sensitive; bronchial catarrh; pale buccal mucosal membrane.

However, the patient was brought to the hospital because of eye trouble. Her mother stated that several children of the farm were blind and had died with the same clinical symptome displayed by her daughter, thus giving rise to the suspicion that an epidemic was occurring.

The patient had a vague and indifferent look, and blinked only rarely and slowly. Tears mixed with a white viscous secretion flowed down her face. The bulbar conjunctiva was dry and dull and had a greasy appearance like fine parchment upon which one had placed a drop of oil; there was no remnant of conjunctival vessels. There was a total loss of the elasticity of the conjunctiva, which folded easily under a simple contraction of the rectus muscles or a movement of the eyelids. On the exposed surfaces of the bulbar conjunctiva, there were deposits on each side of the cornea of a substance resembling white beaten egg that occupied the triangular space from the bulbar conjunctiva to the corneal limbus. When this substance was moved, which was easy with a dry brush, the same xerosis picture could be observed.

On the level of the rectus muscles, there were some subconjunctival veins filled with black blood forming a crown around the corneas that presented diffuse opacities though without hampering the visibility of the iris. The pupils were slightly dilated and reacted poorly to light.

Removed from the conjunctiva, the foamy substance resembled tallow and, under the microscope, it was seen to be constituted of numerous small, greasy spheres of varying size. There were also large pavement epithelial cells present, the pronounced granular content of which showed advanced degeneration.

The disease process worsened rapidly. The corneal opacity became more pronounced, the epithelium became duller, and there was a slight loss of the central substance with adjacent infiltration. There was a larger volume of tears, though they did not make the conjunctiva shiny. The corneas disintegrated, and the child soon died because of bronchial pneumonia. An autopsy was impossible.

After meticulously consulting the literature, DE GOUVÊA commented: "In the history of xerophthalmia resulting from nutritional disturbances, all the authors who described it believed they were the first ones to do so."

We shall now summarize DE GOUVÊA's interesting review of the literature:

1. The first mention must be attributed to RAU, professor in Berne. He published two cases in von Ammon's *Monatsschrift* (1841) which were later reproduced on page 166 of the *Revue ophthalmologique de la littérature médicale* (Paris, 1842). Rau gave no explanation of the etiology of xerophthalmia, and he was content with asserting that there were no obstructions of excretory passages of the lacrimaL glands, as was then believed because of the works of SCHMIDT, MACKENZIE, F. JAEGER, AMMON, KLINGSOHR, STOBER, TRAVERS, JEANSELINE, DUPREZ and others.

2. BITOT, professor of anatomy at Bordeaux, 22 years after Rau's publication, read an interesting study before the Academy of Medicine of Paris on 29 cases of xerophthalmia observed in the Orphans Hospital at Bordeaux (1859 – 1861). It was published in the *Gazette Hebdomadaire* of 1st May 1863 under the title *"Mémoire sur une lésion conjonctivale non encore décrite, coincidant avec l'héméralopie"* (Memoire on a not yet described conjunctival lesion coincident with nyctalopia).

BITOT was the first to call attention to the concomitance of night blindness and xerophthalmia. In his opinion, these conjunctival deposits were pathognomonic for the night blindness. This concomitance was later confirmed by VILLEMIN, of the School of Medicine at Strasbourg, who believed like BITOT that night blindness was a result of the xerosis itself. The detritus on the cornea formed a veil that was not sufficient to disturb day vision (Gazette Hebdomadaire, 22th March 1863) but was sufficient to disturb night vision.

A. NETTER, also from Strasbourg, published in 1863, his "Memoire sur les taches blanches des sclérotiques dans l'hèmèralopie" (Memoire on the white spots on the sclera in night blindness). (Gazette mèd., 1863, p. 505). He criticized his two predecessors and asserted that the night blindness cannot be caused by the conjunctival lesion but that it must be considered a collateral manifestation. Netter was the first to admit implicitly that the two manifestations could be subordinated to one single cause.

3. Two years later, GAMA LOBO published in Rio de Janeiro 4 cases of the same disease, which we called "ophthalmia brasiliana" (Annaes Brasiliensis de Medicina, Vol. XXIII, 1865). GAMA LOBO believed that his descriptions were the first published. His 4 cases were in the last stage of the disease.

These were the first fatal cases of xerophthalmia reported in the litarature. This work was summarized by ULLERSPEGER in the Klinische Monatsblätter für Augenheilkunde (1866). We refer here also to an interesting publication by BIESSIG (1866) on xerosis of the conjunctival epithelium and night blindness (Centralbl. f. d. med. Wissenschaft, 1867: pp. 424 – 436, Leber).

4. A. VON GRAEFE (1866) described a disease affecting infants and noted corneal complications (Hornhautverschwärung bei infantiler Encephalitis — Arch. f. Ophthal. Vol. XII, Part II, p. 250). Von Graefe was interested primarily in two cases that resulted in death and for which KLEBS had performed autopsies. Both cases presented a pale complexion, muscular hypotony, poor nutrition, anorexia, greenish diarrhea alternating with constipation, and no apparent neurological symptoms. Moreover, the autopsies did not reveal clear neurological findings except for fatty degeneration of the neuroglia in the white matter. KLEBS also found fatty livers in the two autopsies.

VON GRAEFE was the first to focus attention on the general manifestations and to point to the poor nutritional state of these patients. The cases described by von Graefe were the severest, as were GAMA LOBO's two cases and DE GOUVÊA's first case.

5. M.S. SNELL published observations identical to those of BITOT (The Lancet, 1876, p. 45), and he, too, thought that it was a new disease. This was the first time that recuperation was reported of all the patients after tonic treatment.

6. HOCQUARD, who also cited a case of WARLOMONT, described in the Archives d'Ophtalmologie (1881) what he called "epithelial spots of the cornea". This was, perhaps, the initial stage of the disease. The white spots of the cornea were not produced "in loco" (Hocquard), but were a substance produced in the conjunctiva and disposed on irregular areas of the cornea (DE GOUVÊA).

7. CAPELLO and IVENS, who were Portuguese investigators, recorded observations related to our subject in referring to a disease that terminated in blindness and death (Vol. I, p. 209 of their memoirs, 1881). A remark made by Capello and Ivens is curious: "The habit of sleeping in the sun is dangerous and favors the progress of the disease."

DE GOUVÊA then studied the different phases of the disease.

I. NIGHT BLINDNESS

He tells us that night blindness always preceded the changes of the bulbar conjunctiva. The incidence of the disease was greater in the interior of the country, particularly among the slaves who worked on the coffee plantations and during summertime when the work was exceptionally arduous. The young were particularly susceptible.

Frequently associated were anemia, intestinal parasitosis (Ancylostoma duodenale), haemorrhages, and long-term febrile diseases. The most common cause among the Brazilian slaves working in agriculture was insufficient nutrition for such debilitating labor in the scorching heat. The negro resisted the disease better. The common foods were beans, pork fat, and a corn flour paste called "angu".

In summary: too much work, poor nutrition, precarious hygiene, and, not rarely, vice. The night blindness often occurred in epidemic proportions, as other authors also noted (BITOT, A. VON GRAEFE, POULLAIN, GREMARS and others). The cases of night blindness were rare on the farms where the slaves were well taken care of.

DE GOUVÊA attempted to explain the night blindness with a substrate of anemia set off by the glare of the sun's rays. These were, according to De Gouvêa, "the only certain factors in the night blindness of our agricultural slaves. There is no night blindness without exposition to bright light."

DE GOUVEA noted that the night blindness diminished (or even disappeard) when the patient remained in a dark room. One night of rest was sufficient for the recovery of retinal sensitivity. This he called simple night blindness and described 30 cases of it. None of these cases presented a noticeable alteration of the fundus of the eye. All the patients were cured with the aid of a compound of iron, strychnine, visual rest, and a rich diet of animal products.

The retinal torpor associated with night blindness is a natural consequence of the nutritive deficiency of this membrane. There is a reduction of corneal and cutaneal sensitivity, loss of hair, the beginnings of a ring resembling an arcus senilis, weakening of the intelligence, and sleepiness. The patients seek out warmth and the sun's rays and devour everything that they can get their hands on. And simultaneously, if not earlier, xerophthalmia appears.

II. XEROPHTHALMIA

The conjunctiva, which was bloodless since the onset of the night blindness, now presents no trace at all of blood vessels. Near the cornea, the conjunctiva immediately loses its shiny aspect. The foamy substance, already noted, forms;

the mucous membrane becomes drier and drier and resembles skin. This is what von Graefe called "xerosis acuta".

Corneal sensitivity diminishes. RAU had already observed that the patient did not blink when his cornea was touched with the finger. Conjunctival and pericorneal congestion are present, and the opaque ring extends over the entire cornea.

Flat, red follicles appear in the conjunctiva of the eyelid and of the inferior cul-de-sacs. The foamy substance forms true patches alongside the cornea and even on it.

The xerosis continues to expand on the bulbar conjunctiva, and bronchial catarrh appears in both lungs accompanied by tachycardia and hyperthermia. Sometimes, bronchitis and diarrhea are present.

Thus, DE GOUVÊA includes reports and comments concerning systemic problems (pulse, temperature) primarily to establish that these disturbances are due to problems of the nervous system.

III. THE CORNEA AND THE LETHAL CONCLUSION

With the onset of the fever, the disease becomes more serious. There is a diffuse opacity of the cornea, and suddenly there appears in its center (or close to it) an inflammatory greyish or yellowish infiltrate with epithelial erosion. In a few hours, the corneas are destroyed. The sub-conjunctival congestion increases and the sclera becomes dark blue. The sensitivity no longer returns. The patient remains indifferent and dies because of complications (asphyxiation because of the accumulation of bronchial secretions or exhaustion because of diarrhea).

VON GRAEFE and GAMA LOBO observed the disease only in its final stage, while RAU, VILLEMIN, NETTER, SNELL and others saw it in the initial phases when the conjunctival changes were only slightly visible. This is the reason for the differences in the evaluation of the disease and its clinical picture.

Comments

With all the information that is provided by DE GOUVÊA, one can readily deduce that xerophthalmia, along with its other manifestations, is the natural consequence of a generalized nutritional disturbance caused by progressive anemia. This is why I would propose "xerophthalmia cachectica" as the designation for this ocular disease.

VON GRAEFE believed he could attribute this ocular disease to a specific inflammatory process of the brain (infantile encephalitis), because of the results of two autopsies made by KLEBS.

"In our two cases", VON GRAEFE wrote, "there was in addition to the encephalitis, a fatty liver. This gives rise to the suspicion that the cerebral involvement represents more a partial manifestation of general nutritional difficulties than a point of departure for the entire disease."

A number of reasons and among them the age of his patients and his ignorance of previously published observations, led VON GRAEFE to believe that an encephalitis was involved, and that the xerophthalmia was merely a sign. In fact, however, notwithstandig von Graefe's authority, KLEBS' verifications indicated that the process was regressive and that it harmed the functions of all the sensory organs. Nevertheless, the subject still remained controverted.

SNELL did not know what was the cause of the disease, and noted that all the patients were apparently robust. One must state, however, that all these patients were cured with cod-liver oil.

As far as BITOT is concerned, all of his patients were lymphatic and scrofulous, even though the disease was more common among seemingly healthy children, thus giving rise to ambiguity.

DE GOUVÊA stressed and gathered documentary evidence to support the correctness of his concept. The treatment must restore the severely compromised general physical condition of the patient. It is necessary that the diet be controlled rigorously, especially with the young and in severe cases. When the diarrhea regresses, cod-liver oil, strychnine, and iron must be prescribed.

To prevent or cure xerophthalmia and the corneal disease (keratitis neuroparalytica), DE GOUVÊA recommended humid warmth (vaporizations at 40° C), and described an apparatus similar to LOURENÇO's vaporizer. He stressed the high value of this therapy, even for the corneal ulcers. Vaporizations are to be applied for 15 minutes, once or several times a day. "Vaporization as I use it", said DE GOUVÊA, "can be done by anyone; it is pleasant for the patient, and children soon get used to it. There are no complications at all, which cannot be said for cataplasms and hot compresses." With this procedure, De Gouvêa obtained an organ recuperation in all of his cases.

We will now summarize six of DE GOUVÊA's cases, which he considered the most interesting. Five of them presented corneal involvement.

Case reports

Case 1

Negro, 20 years old, transitory attacks of amaurosis, nightblindness, pronounced anemia, pale conjunctiva, juxtalimbal xerotic patches, arcus senilis, sluggish pupils. Good vision. Cured after 50 days of a rigorous diet and vaporizations. Final visual acuity: 20/30.

Case 2

Negro, 28 years old, nightblindness, anemia, discrete conjunctival xerosis, arcus senilies, sluggish pupils. Cured after 20 days of a nutritional diet and vaporizations.

Case 3

Negro, 16 years old, initial nightblindness, disease epidemic on the farm where he lived, pronounced anemia, initial alopecia, malleolar ulcers, generalized bronchitis, xerophthalmia of the bulbar conjunctiva, foamy patches, the cornea of the left eye completely destroyed, the right eye with great alterations of the conjunctiva and diffuse opacity of the cornea. Treatment: cod-liver oil, phosphorus, diet of animal products and vaporizations. After 3 months, he was cured and had a final visual acuity of 20/30 in his right eye. The left eye was atrophic.

Case 4

Negro, 14 years old, pronounced anemia, atonic muscles, apathy, initial bronchitis, malleolar ulcers, alopecia, conjunctival xerosis, peri-limbal follicles, opaque ring in the cornea, diffuse opacity throughout the cornea, corneal and conjunctival hyposensitivity, visual acuity severely reduced (finger counting at 10 feet). Visual acuity 20/30 after 4 months, when he was cured.

Case 5

Negro, 13 years old, nightblindness for 6 months, ocular inflammation for 20 days, very poor general physical condition, characteristic xerosis, conjunctival follicles, beginning corneal ulcer with deep infiltration and hypopyon. The left eye resembled the right eye, and the corneas were infiltrated without ulceration. The patient was treated with cod-liver oil, iron, strychnine, a rigorous diet, and vaporizations. There was an obvious improvement after 40 days; the ulcers healed, the corneas became clear, and the xerosis disappeared. Some time later, the patient died of bronchial pneumonia.

Case 6

Mulatto, female, 9 years old, diet almost exclusively vegetarian, digestive difficulties, nightblindness, anemia, emaciation, atonic muscles, edema of the

lower extremities, sleepiness, alopecia, bronchitis, tachycardia (132 beats), axillary temperature of 36.5° C, xerophthalmia in the last phase, beginning of infiltration of the central cornea of the left eye. She was treated with cod-liver oil, iron, vaporizations, and a diet of animal products. She was almost completely cured when she abandoned the treatment (30 days).

After a century, what can one conclude about the work of de GOUVÊA?

Obviously, xerophthalmia was known in Antiquity: HIPPOCRATES and CELSUS wrote about it, and there were even earlier reports. Still, it can be said that RAU (1841) was the first to describe it, though he did not clearly associate it with malnutrition. RAU did have the merit of dismissing the presumed relationship between xerosis and obstruction of the excretory canals of the lacrimal glands. RAU made no allusion to nightblindness, however, and treated only the alterations of the conjunctiva and the cornea (1866). The subject was so confused that the brilliant von Graefe (1866), the greatest ophthalmologist in the world, was led on the basis of histopathological observations to associate the corneal lesions with an infantile encephalitis. Nevertheless, in 1863, 22 years after Rau, BITOT reported 29 cases of xerophthalmia among the children of an orphanage in Bordeaux and established, for the first time, the relationship between xerophthalmia and nightblindness. The subsequent clinical work reinforced the relationship between xerophthalmia and nightblindness, even though the cause of the nightblindness was highly disputed. It was even admitted that it resulted from a veil of epithelial detritus on the surface of the cornea.

The merit of the work of DE GOUVÊA (1883), however, cannot be denied. By detailed examination and careful observation of his many cases, he succeeded in establishing a relationship between the disease and malnutrition. DE GOUVÊA even established a chronological sequence of the morbid manifestations that still holds today: 1) nightblindness, 2) xerosis, 3) severe corneal lesions (keratomalacia).

Nevertheless, even the most perspicacious observer is not exempt from error. DE GOUVÊA's error was that he stated that the foamy substance was not produced **in situ**, but that it was the result of a deposit. Today, we know that the foamy aspect is the result of disturbances in the superficial layers of the keratinized epithelium, perhaps reinforced by the gas produced by Corynebacterium xerosis.

Here we leave the synthesis of the magistral work of De Gouvêa. We would recommend the reading of the entire text to all those interested in the subject. Thus we render homage to the great Brazilian ophthalmologist:
HILARIO DE GOUVÊA.

Resumé

Hilário De Gouvêa (1843 – 1923) est un grand ophtalmologiste du Brésil, qui a particulièrement étudié la xérophtalmie, connue à cette époque sous le nom de ,,Opththalmia Brasiliana''. Bien que cette affection ait déjà été signalée dans l'antiquité, il faut cependant admettre que c'est Rau qui, en 1841, en a fait la première description. Le sujet est resté très confus au point que von Graefe a attribué les lésions cornéennes à une encéphalite infantile. C'est le mérite de De Gouvêa d'avoir établi la relation entre la maladie et la malnutrition, ainsi que d'avoir donné de l'huile de foie de morue comme traitement.

Summary

Hilário De Gouvêa (1843 – 1923) was a great Brazilian ophthalmologist, who studied particularly xerophthalmia, known at that time under the name of ''Ophthalmia Braziliana''. Although this disease was already mentioned in antiquity, it is a matter of fact that Rau, in 1841, gave its first description. The subject remained so confused that von Graefe was led to associate the corneal lesions with an infantile encephalitis. It was the merit of De Gouvêa to have established the relation between the disease and malnutrition, as well as to have given cod-liver oil as treatment.

Resumen

Hilario De Gouvêa (1843 – 1923) es un gran oftalmólogo del Brasil, quién ha estudiado particularmente la xéroftalmia, conocida en esta época bajo el nombre de ,,Oftalmia Brasiliana''. Aún cuando esta afección habia sido senalada en la antigüedad, hay sin embargo que admitir que es Rau, en 1841, quién hizo la primera descripción. El tema quedó muy confuso, a tal punto que von Graefe atribuyó las lesiones corneales a una encefalitis infantil. Es el mérito de De Gouvêa el haber establecido la relación entre la enfermedad y la malnutrición, así como de haber dado aceite de higado de bacalao como tratamiento.