

Keratoconus Etiopatogenesis and True Cure : Modern Concepts

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Introduction

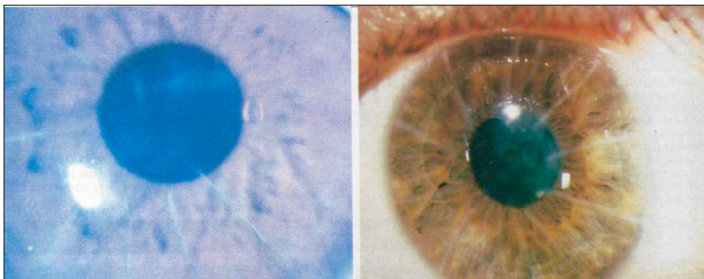
A kind advise for the skeptics and denying persons, at any cost, please reflect on what has been written, two Centuries ago, by a great French Physiologist: Claude Bernard (1813-1878): “ what we know it is the main obstacle to acquire what we do not yet know”

I have been dealing with the Study of the Epidemiology of the pathology known as KERATOCO-NUS for more than 35 years. We are witnessing an exponential growth in the number of cases showing symptoms ascribable to this disease.

This increase it is also the result of a better ability of today Ophthalmologists and more refined diagnostic electronic apparatus in use to make an earlier and more precise diagnose than in the past. From my side I could refine in this 35 years my understanding of the Corneal maps to individuate the very early stages of the Sickness. Also using:

- a. a1) “ clear definition of the Origin of the disease” and a 2) “ better and more accurate understand-ing of the underlying biomechanical mechanism “ has given us the possibility 3) to ask more precise questions to our patients to verify exactly the very first subjective symptoms in medical history
- b. More accurate diagnostic instruments (keratoscope, Corneal-map and scanners, Corneal Scheimpflug Cameras, electronic pachymeter, etc.).
- c. a more in-depth clinical-case study experience and over 20.000 eyes examined.

Some of these patients have choosed and positively undergone Ark-Microsurgery before, and Mini-ASRK-MICROSURGERY in the last 20 years collecting a number of 3.500 successfully op-erated eyes and about 2000 happy and healed patients.



Keratoconus

Asymmetrical Radial Keratotomy

Over the years, we have noticed a significant statistical change in our surgical practice as regards the number of patients who were referred to us for refractive surgery and those who were aware of having a keratoconus.

As a matter of fact, 20 to 30 years ago, in 100 patients coming to us for refractive surgery, 95 were selected for refractive surgery and 5 were unaware of having a keratoconus. Now a days this ratio has been totally reversed: in 100 patients coming to us for a possible refractive surgery, approximately 60 to 70 %, in fact, are unaware of having an early keratoconus and only 30 % are real and simply refractive surgery patients.

It is therefore necessary to explain to 70% of our patients, who are unaware of having a keratoconus and most of the times are borderline cases, what their actual pathology is, and the difference between their sight impairment before the keratoconus and the actual impairment with it, as detected through biometry, and the pre- and post- estasia pachymeter difference, and comparison of the dif-ferences in Corneal Maps and Keratometric differences.

If we wish to recognize, also in the early stages, a keratoconus whose origin it is not yet been offi-cially designed (see Wikipedia and Pub-med) and given as “unknown origin associated to atopic Disease” some researchers consider that it could be due to a “not better defined Genetic Disease”, theory not agreed by most of the “researchers”, also considering the indisputable fact that statistical-ly the “Familiarity” has been found “only” in the 12% of the investigated cases.

Then we could also think, about the origin, to the possibility of an “INFECTIOUS-DISEASE” as, in fact, I have discovered to be caused by a “CORNEAL- ASPERGILLOSIS”, that is manifested through a new definition of the Pathology as a: “Chronic Subacute Kerato-Conjunctivitis”

We should ask our patient to recall any episodes of tingling, irritation, rare redness (intermittent conjunctival hyperemia) very rare transparent secretion and feeling of having a foreign body or sand in the eye/s, accompanied by a desire, sometimes very strong, to rub the eyes.

These episodes which have certainly preceded, many times and for a variable long period (most probably years), with short or longer periods of apparent “CLINICAL REMITTANCE”, the first clinical subjective signs of changes in the vision.

These symptoms of irritation and discomfort are due to the: “Reproductive Activity of the “FUN-GUS”. After having taken due note of: medical history, the Corneal thickness should be measured using an electronic pachymeter with a minimum of 3 to 5/6 measurements to be done along the axis of the Corneal map asymmetrical ectasia.

The standard measurement should not only be done at classical “6 o clock” axis also to control the possible presence of an eventual worse degeneration as the” Pellucid Degeneration.” In this regard, please take note that there can be “Early-keratoconus cases” with central corneal thickness up to 650 microns, thereby way beyond the “standard thickness of 500 microns”.

In such “early stages”, corneal thickness in both eyes could also be initially the same in both eyes, thereby showing no change whatsoever, but this “early anomaly” should not discharge the possibility to consider this case as a potential: early keratoconus, the Corneal Map that I still take with an Eysis Corneal Map Cheratometer will show an asymmetric image also with an initial small difference in ectasia on one side of the astigmatic Corneal image, that it is the only initial sign of the sychness that always start from the endothelium and Descemet membrane initial ectasia in the central, or paracentral Corneal area that in the books that I have studied in 1973-78 was defined as an early internal erosion of the central cornea without explaining who was causing the erosion.

We rely on the accurate examination of corneal maps, bearing in mind, however, that in the early stages of the disease, as I have just explained in the last paragraph, there is often a slight localised increase in the internal curvature of the central or paracentral-cornea, which is not considered nor usually recognized to be a sign of an early keratoconus by some electronic devices, (such as the Orbscan).

Such minimum change in the internal curvature, which can still seem to be ‘spherical’, leads to a subsequent light change in the external curvature that the Corneal Map can catch, that we consider “an indicator of an” early or borderline keratoconus “.

As a matter of fact, we will see a map shaped like a lower, central, nasal or temporal ampulla, the-reby quite seldom lateral or superior and anyway always asymmetrical considering the center of the cornea as the center of an hourglass, with the asymmetrical portion 3.4-times larger than the oppo-site area of the same eye.

Examining with great attention the Corneal Maps I could notice that the shape of right Corneal Map was in the 90% of the cases specular to that of the left one, the question that raised in my mind was “Why ? and How can be possible ?”.

I decided to study back again the Corneal Eye Embriology and I could find easily the right answers.

The development of” the Primary Corneal Ophthalmic Embriogenic Vesicles” starts from a common point with two branches that grow in the opposite directions one go left the other in the right direction describing , in the 3 Cartesian dimensions , a circle like a reverse U shaped life belt this Circle normally close, welding the two opposite branches at 6 o clock as looking a glass Clock Face.

This welding point represents the most delicate and critical point of the Corneal structure, consequently the weakest point. The ASPERGILLUS will,start its attack in this point.

This discover took me to redefine in a new way the characteristics of the KERATOCONUS, because the different locations of the Corneal welding weakest point could determine, for a certain range, a different evolution of the K.

Per example a KERATOCONUS with a welding point at 6 o clock will determine a complete different evolution from one with welding point at 12 o Clock.

The last will grow very slowly because the upper position has the advantage of the upper lid restraint that mechanically press on the upper part of the Cornea containing much of the Progressive Ectasia Growth, milding the progressive development of the KERATOCONUS.

This last it is a biomechanical explanation of the possible rational explanations for the presence of the so defined : “KERATOCONUS Frusto” a bio mechanical one. Others rational explanations will be described later in the book.

This Embriogenetic orientation of the Corneal Map, on the plane, of the 2 Cartesian dimensions X,Y took me to describe the Phenomenon as in the following paragraph.

IKERATOCONUS has been classified by me, with the exact point of closure of “embriologica oph-thalmic Vesicles” as looking to a “Clock’Face” indicating the “hour” referred to the point of natural welding of the two extremity of embriologica vesicle (like a semicircular life belt), I am use to say : K. At hour: 2-4-8 etc.

This closure it is normally positioned in the two Corneas (right and left) as opposite and specular like in a mirror, so that : hour 4 in the right map it is 8 in the left one, with the two only exceptions for the KERATOCONUS orientated at hour 6 and 12.

The initial color of the corneal map vary from a pale yellow to a small central or para-central area, which is usually from green to pink-reddish showing with the progressive color variation the parallel variation of the more elevated central Corneal area.

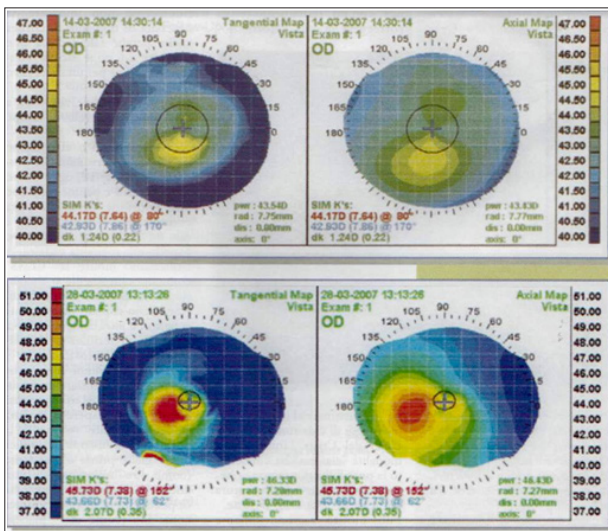
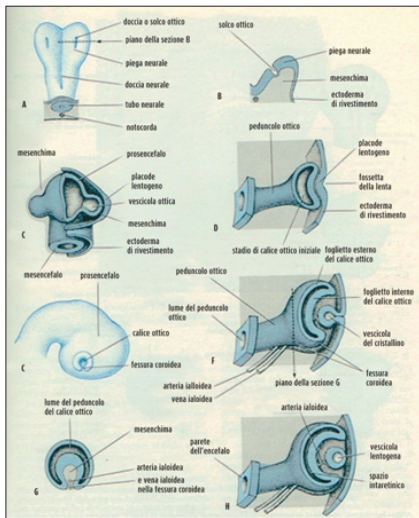


Figure: Embryological formation of optical vesicle

The reason for the “infero-nasal-temporal initially slight modification in the map shape” is due to early internal and external ectasia related to the natural Primary Ophthalmic Vesicle physiological welding point in embryogenesis of the eye. (The primary optical ectomesodermal vesicle at the end of its embryogenetic development).

We consider this “precise welding point” as the weaker point of the whole “Vesicle” and as normal in Nature the ASPERGILLUS will find more easy to start its infection in that point defined as: “Locus minori resistentia”=“in the weakest point of undermined resistance of the corneal “rima””.

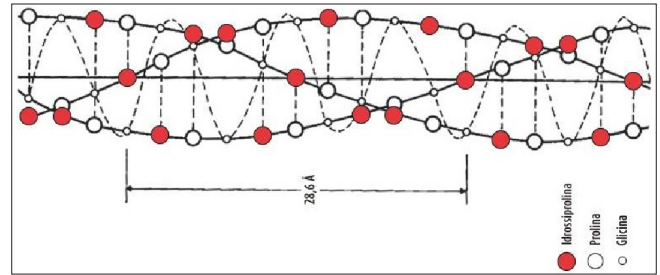
The Vesicle is usually closed centrally in the bottom part of the Cornea, sometimes asymmetrically in the nasal or temporal area, but it is usually closed in 90% of the corneal-maps in the lower area right at 6 o'clock. Or slightly on left or right as 7 or 5 o'clock.

The “Aspergillois Infection” starts very slow, at beginning and then spreads “Centrifugally” invading the lower part first and in the worse stages the whole cornea.

As it happens with all” infectious processes “who are” circular in

shape and starting from an initial point of insemination (infection) in centrifugal expansion “ as it can be observed in cultivation of: “virus, bacteria and fungus” as in a” Petri dish or in any other culture medium “.

The bio mechanical modification that will rappresent the main damage caused by the ASPERGILLUS infection it is the lysis of the “bonds (bridges) fibers” of the ”Collagen Helix”



which will make the very same cornea “extremely weak, friable and flexible and easy to be stretched“.

Now we need a particular “Power” to explain the progressive (dilatation) “Ectasia” of the K. In a linear progression with parallel reduction of the Corneal thickness like what happens to a stretched “Rubber Band”.

This Power is the “Intra-ocular pressure, even if normal”, that can lead, with its continous pressure, to a progressive weakening of the corneal wall.

The reason because also througha “NORMAL INTRA-OCULAR PRESSURE” the Cornea will be stretched, more or less, slowly but continuously it is because a normal tissue has been transformed from a relatively strong Collagen structure in a very weak one, soft and extensible.

The “Pressure” will induce the well-known progressive ectasia (like when you inflate a rubber bal-loon) in a Tissue that is become so “weak and unable to resist also to a normal hydrodynamic stress” produced by the continous pulsationof the central Retinal artery of the Eye “in tune with cardiac systolic contraction“.in the number of “518.400” pulses into the 24 hours (considering as a standard “60 pulses every 10”seconds).

Figuratively imagine a small hammer that continuously heats the central Corneal second after second it remains me the Latin proverb: “Gutta Cavat Lapidem”.

Now you can realize the incredible efficacy of a continous central hammering mostly on a tessute that has no more the strength that was calculated by Our Creator in the original project where Aspergillois Infection was not took in consideration as many other Sicknesses.

We have to understand as a logic consequence of the concept exposed previously that the “Worse Complication”for a KERATOCONUS Patient it is the Glaucoma!

I could notice that many patients with a fast growth of the Ectasia had developed a Glaucoma some times also in relatively jung patients, any way this complication, also if rarely, can happen.

To be sure that this initial Corneal maps modifications are not due to an excessive mucous or greasy secretion of the eye, or as a Corneal-stamp due to the use of hard Corneal lenses it is extremely important to visit and take the test 5-7 days after removing contacts and having the patient rinse his/her eyes very well with plenty of cold water several times a day, not eating fat-oily-food or milk and derivatives and sweet food or sugar the 7 days before.

As a matter of fact, any oblique position of the irregular imagine of the "Corneal-Maps" in the asymmetric portion of the Clock' Face, and sometimes commonly referred to the peculiar shape of "Tears of Allah" (typical of Oriental fabrics and carpets very similar to the blood drop of the Wodaphone Symbol), are, for my "experience and knowledge" a clear sign of an early KERATOCONUS.

There are also keratoconus cases, which are increasing in number, also with "Pellucid Degeneration of the cornea", where besides the "Tears of Allah" an apparently flatter area can be observed between the two opposite "tips" of the "tear of Allah" approximately at half of inferior radius of the Cornea and almost always oriented to the inferior Center or paracentral part of the Cornea at 4 to 8 of the Clock'Face taking the 6 o'clock as the main axis of most of the "Pellucid Degeneration".

The associated pellucid degeneration is given by the measurement of corneal thickness, which will be lower in the central-para-central area been more thin than the central thickness along the inferior axis, sometimes, till the extremely peripheral area of the Cornea.

Therefore, we consider all those cases (except Pellucid Degeneration) to be an early keratoconus showing the above-mentioned changes in the corneal map, even if at an early stage.

We should take in consideration also a bio mechanical particular condition where the orientation of KERATOCONUS axis it is at 12 o'clock. We consider this a possible case of KERATOCONUS "Frusto" where the upper lid provokes a restraint to the progressive ectasia reducing consistently the effect of intraocular pressure vector.

Other explanations of a different nature for the same type of K. "Frusto" will be given later in the text after having introduced the explanation of how and why the ASPERGILLUS decide to infect that specific person.

For early diagnosis, there are also many cases showing clinical-subjective symptoms related to a worsening vision mostly at night, or when looking at TV, Hi-fi LEDs, spot lights or point lights.

I could also find few patients who confirmed me, when specifically requested by my "anamnestic" interrogations, about the possible direct experience, to have noticed the pulsing and slightly enlarging effect of pin pointed tv or computer leds lights synchronized with the hearth pulsation transmitted till central retina artery, confirming my discover of the mechanism capable to produce the progressing ectasia by the hearth pulsing provoking a continuous hammering effect inside the center of Cornea with the direct consequence of enflating and enlarging the same Cornea resulting in a progressive Ectasia growth and evolution of the Sickness known as KERATOCONUS.

Such phenomena could indicate the possibility of an early keratoconus in patients aged 10 to 50, who have noticed a change

in their normal vision in the distance and especially at night or watching, as previously described, pointed lights or led sources.

Of course, it is important to verify the presence of possible real refractive errors who are not the results of a secondary sight defect due to the development of an early KERATOCONUS. (latent hypermetropia or early myopia and de-compensated astigmatism that it is statistically possible).

The accuracy of such an early diagnosis, especially in borderline patients, is corroborated by our "Research Centre" that can confirm with a precision close to 99% the presence of a starting KERATOCONUS caused by a Corneal Aspergillosis by means of biological laboratory exams and with specific tests through electronic machineries capable to give a "BIORESONANCE DIAGNOSIS before and THERAPY after" which "PREVENT" the pathology from "further DEVELOPMENT".

And consequently having also the possibility, in many selected clinical cases, to "RESTORE FULL VISION" within 6 months to 1 year only through a simple "Biological-Natural-Therapy" with "no need of surgery" but only "in preclinical cases".

Many of these cases are closed parents or children who have been initially infected by the ASPERGILLUS because they were living in the same house or in other houses with similar specific characteristics.

I have at this point to explain why I had to use an unusual system in the so said : Scientific Medicine Research Biological Systems to find out the Presence of ASPERGILLUS : the Bioelectronic Approach for the Physical presence of "Bioresonance Effect".

In fact the Classic Researches with Biological samples of affected Corneas has specific difficulties, mostly because it is very difficult to convince a living patient to have a Biopsy on his Corneas, and also with the Biological Cornea sample it is very difficult to find qualified research laboratories specialized in detecting and prove ASPERGILLUS Corneal infections, mostly when the infection has just started or it is in the early stages.

Having understood that Biological samples had too many difficulties to be used extensively, I have looked around and discovered that the Bioresonance was a much easier method of scientific investigation.

I did study the principles and the technique following specific courses. I made a systematic research before on all my patients already clinically classified as KERATOCONUS affected patients, and I found out that approximately the 99% of them were positive to the Bioelectronic test for ASPERGILLUS.

Later in the text I will also explain how I could "Eliminate-Or better and more precisely Erase the ASPERGILLUS" from their Corneas and Eyes.

The "BIOELECTRONIC-BIORESONANCE INSTRUMENT" used for DIAGNOSIS and THERAPEUTICAL purposes to find first and eliminate after the Aspergillosis, is the: MORA-SUPER, well known and reputed German made machinery. (to day there are many others similar machineries)

The technic of BIORESONANCE it is the same that by, many

years has been proved reliable, and normally used at airport : the electromagnetic “Arc”, under which pass every passenger before the Embarkment on the flight, to find out dangerous items as metals, eventual explosive, also synthetic, and other forbidden substances.

The “Arc” works exactly with the same fisic Principle of “Bioresonance” With the “Mora-Super” machine we could find out the presence also of very few Parassites, Virus, Bacterials, Fungus etc.

The presence of a Biological Entity it is recognized when the machine find the Specific Bioelectronic Freqence of that Biological Entity through “Bioresonance Effect”that it is normally a unique Freqence for that type of Biological Entity.

On the web you can find the list and files of the Bioresonance Frequencies. Always on the web, having previously individuated the Specific patologicous “parassite”, you can find the proper specific “Killer Contra-Frequency” to eliminate it through the body of the patient.

As soon as we have individuate the Specific Presence of that Biological Pathological Entity we can apply an other Fisical and Biological Principle sending, with specific Bioelectronic Apparatus as the over mentioned “Mora Super” (and many others) an identical “Wave” but with: “ half Phase of delay”.

The consequence it is the “Biological Electromagnetic Erase” of the Freqence that the Specific Entity issues that it is in fact the expression of its Biological Existence.

If you have a design of a “Wave” into a bidimensional Cartesian axis imagine to superimpose an identical “Wave” but slightly moved to the right till the two Waves will show only a sequence of zeros.

You have in fact Erased that Wave and in the Biological Reality you have just “Killed” that Biological Entity.

I have to advise you that the fast and simple killing of the ASPERGILLUS does not mean automatically “For Ever” but in that moment.

To Erase definivly the infection we have to “Expell” the Fungus from the “house or office” where the patient live and where the ASPERGILLUS live.

I have spent all together 7 years of continous researches to solve once and for ever the Biological problem of the infection and 34 years of perfecting to the maximum extent the microsurgical presidia known as “Mini-Asrk (asimmetrical selective Radial Keratotomy)” to repair and correct the deformed Cornea giving to the patient the best possible visual correction for the rest of his life.

I want to introduce now the concept of Electrobiological Preventive Therapy of very early infected patients who have taken the Corneal infection but not yet developed the Clinical substantial Corneal Modifications that will make him understand that something it is slowly but progressively changing in his eyes and sight.

Those patients, affected by early KERATOCONUS who needed a lens - usually no more than +/- 0,75-1 to improve their vision, tend to

spontaneously recover, after the “Bioelectronic Resonance Tretment” to full sight without any lens any more, even if they considered it to be essential before, because their subjective improvement brings them back to the “status quo ante”=(emmetropia or the refractive condition before the Sickness).

This result confirms even further the diagnosis of a borderline keratoconus and the accurate defini-tion of the Keratoconus Origin: Aspergillosis infectious disease, as reinforced by the continuous successful therapeutical results of our treatment in the last 12 years practiced on 2500 patients, and, where necessary, also in other members of the same family who, after examination, were found in a preclinical stage.

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