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The Serous Choroidal Detachment After Glaucoma Surgery: Demonstration of a Pathogenic Mechanism Denied by Modern Literature

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Abstract

Aim: Prove a mechanism of action in postoperative serous choroidal detachment (SCD), a mechanism denied by modern literature**.**

Materials and Methods: Ten cases with advanced trabecular glaucomas that developed SCD: 9 cases - after Vancea trabeculectomy and 1 case - after my technique named trabeculokeratencleizis. The methodology used in this study started from a series of observations: after the drainage of SCD through a scleral puncture - practiced after 7 days of ineffective medical treatment, and after the reformation of the anterior chamber (AC) with saline through a corneal puncture, the AC rapidly disappeared in parallel with SCD fluid outflow through the scleral puncture, when it was opened. This finding suggested the existence of a direct communication between the AC and the SCD lobes. In order to demonstrate its existence, Na and K ions concentration was measured by gas chromatography in the SCD fluid collected at the first evacuation and after several suprachoroidal space washings.

Results: The Na^+ concentration remained within normal limits both in the first sample and in the one collected after 3 - 6 suprachoroidal space washing cycles. The K^+ concentration showed a dramatic decrease. This can be explained by the fact that the washing fluid (the saline solution) contains only Na^+ and no K^+.

Conclusions:

1. In case of SCD after glaucoma surgery, a communication between AC and the supraciliary and suprachoroidal space existed in all tested cases after 7 days of athalamia/hypotony syndrome.
2. The communication was proven in a quasi-experimental manner, by spectrophotometric dosage of Na and K ions in the SCD fluid collected before and after several suprachoroidal space washings.
3. This communication might either be the cause of SCD or its consequence. Certainly, the presence of such communication prolongs the sickness.

Keywords: *Serous Choroidal Detachment; Pathogenic Mechanism; Glaucoma Surgery*

Introduction

The serous choroid detachment (SCD) is an accumulation of serous humor in the normally virtual suprachoroidal space. Usually, the complication is associated with hypotony and flat anterior chamber (AC) [1-10]. Glaucoma (G) surgery carries the highest risk for this

complication, whose rate can go as high as 3% - 34% of trabeculectomies and 3 - 35% of glaucoma implant procedures [1-10].

Synthesizing the knowledge, recent reviews [2,5,6] stated that the exact triggering mechanism of SCD is not known and suggested that it can be caused by transudation of serum into the suprachoroidal space due to increased transmural pressure (high blood pressure or excessive globe hypotony, or both), and/or by an increase in vascular permeability (trauma or inflammation). The resulting ultrafiltration through the capillary wall is driven by hydrostatic and oncotic pressure gradients according to the Sterling equation [11]. The possible role of unintentional cyclodialysis, suggested by Fuchs [12] is not even taken into consideration.

Entering into detail, the fact that SCD usually appears after incisional surgery (penetrating G surgery - with/without various implants, or classical cataract surgery with imperfect wound closure - when signs of overfiltration exist) suggests the role of transudation - caused by eye hypotony. The aggravating role of risk factors (hypertension, old age, elevated episcleral venous pressure as seen in Sturge-Weber syndrome and related conditions [14]) suggest the mechanism of transudation - caused by elevated arterial or venous pressure. The cases in which SCD appears without incisional surgery (after laser surgery [15], vitrectomy [16], topical and systemic medications such as sulfa drugs, prostaglandin analogs [17] or in posterior scleritis [18]) sustain the role of exudation caused by inflammation. As a result of these accepted mechanisms, the serum with large protein molecules passes into the suprachoroidal space: the oncotic pressure of this fluid is similar to plasma, so that its spontaneous reabsorption is possible only if the underlying cause (i.e. inflammation, hypotony) is cured [6].

With the above mentioned information, nothing has changed in the last half century, since the same mechanisms were used and accepted during the 7th - 8th decade of the last century [13,14] when I began G surgery (iridencleizis, Scheie operation, Cairns' trabeculectomy), and faced the same complication rate. I tried to understand the pathogenic mechanism and act in a pathogenic way, but the task was practically impossible because during and after the external drainage surgery of G, all predisposing factors coexist: the blood pressure may be high, the intraocular pressure (IOP) may be low, the surgery is a trauma for the eye, and the organism responds to trauma by inflammation. Which factor is more important? Which factor to address first? These questions remain unanswered until today [1-11,15-21].

In an attempt to clarify these questions, I started from a personal fact of observation [22]: usually, after SCD lobe puncture I reformed the AC with air through a corneal incision. In one case, I reformed it with saline, and accidentally opened the scleral puncture again: I observed that the AC disappeared in 1 - 2 seconds, in parallel with SCD liquid outflow through the scleral puncture. The sign was observed anytime the AC was reformed with saline and the scleral puncture was opened, faster - when the puncture was widely opened. This suggested that an unwanted cyclodialysis was produced during trabeculectomy, or in the first days after trabeculectomy.

Materials and Methods

In an attempt to demonstrate this clinical conclusion by an objective method, I measured the Na^+ and K^+ concentration in SCD fluid collected at its first evacuation and after a various number of suprachoroidal space washings. After obtaining the informed consent of the patients and the approval from the Ethics Committee of the hospital, I practiced the following procedure:

1. Local anesthesia, transconjunctival traction sutures on two recti muscles (the inferior and one lateral - according to the site of the most prominent SCD lobe: (a and b in figure 1).

2. A 27 G cannula reopened the lateral corneal puncture always practiced at the beginning of any glaucoma surgery, in order to: (a) slowly reduce the IOP before opening the eye; and (b) have a small and easy to close accessory entrance into the eye, other than the main wound. This preexisting puncture was opened at the beginning of the SCD surgery because it would be more difficult to find the puncture path after the evacuation of SCD fluid, on an extremely hypotonic eye. The AC was reformed with air, in order to induce a tonus and to ease further maneuvers.

3. A 4-mm long radial incision of conjunctiva started at 2 mm from the limbus, at 4 or 8 o'clock, in the zone of most developed SCD (c in figure 1). The sclera was dissected with scissors under the inferior lip only, towards 6 o'clock.

4. A curved forceps entered under the inferior lip of conjunctiva incision, catched the Tenon capsule in the vicinity of the inferior rectus muscle insertion and a traction suture was passed, avoiding the perforation of conjunctival epithelium (c in Fig. 1). Two hemostatic forceps hanging on sutures a and b stabilized the eye, while a small Pean forceps hanging on filament c denuded the sclera and formed a sort of flat "sink" (d in Fig. 1), with sclera beneath, and conjunctiva wound edges as margins.

5. Thermal hemostasis of the denuded sclera.

6. The "sink" was carefully dried, and insulated at distance with PVA sponges, in order to prevent tears or saline to parasitize the results. A 2-mm long radial perforating incision of sclera was practiced at 5 or 7 o'clock meridian - according to the situation of the most prominent lobe, starting at 4 mm from the limbus (e in Figure 1), and slightly oblique on sclera surface, in order to form a short valve. The yellowish clear fluid began to evacuate when the lips were manipulated, but the evacuation stopped when the scleral lips were released. When the "sink" was full, the assistant collected the yellowish clear liquid in a specially prepared 2 ml glass syringe (washed in distilled water before sterilization and perfectly dried). When 0.2 - 0.3 ml fluid was collected, the syringe plunger was pulled out and the fluid was transferred into a sterile capillary glass tube, hermetically closed at both ends with sealing paste (sample No 1).

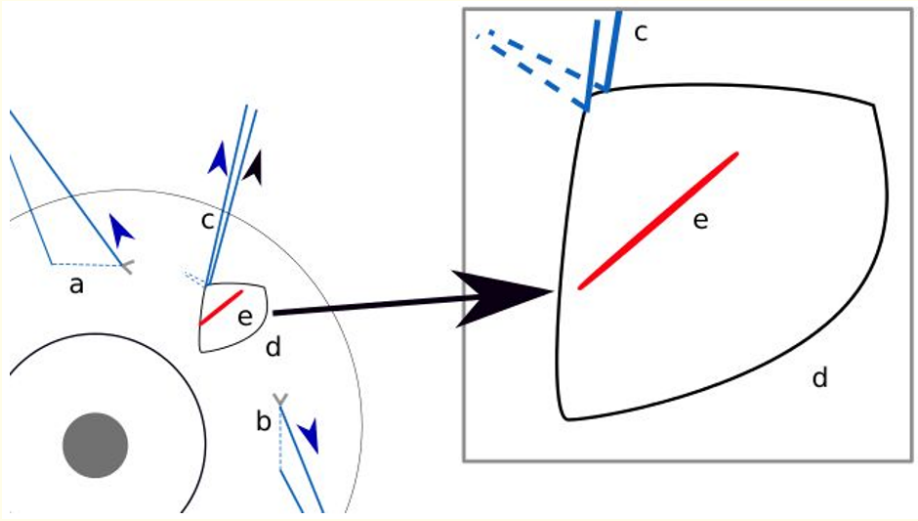

Figure 1: *The "sink" formation, in order to collect the SCD fluid.*
a and b: transconjunctival traction sutures under one horizontal rectus muscle and under the inferior one; c: one additional traction suture meant to increase the sclera exposure is passed through the deep tissue close to the inferior rectus insertion; d: the "sink" meant to collect the SCD fluid, kept open by c; e: the perforant scleral incision..

7. After harvesting the first sample, I reformed the AC with saline, opened the scleral puncture again and pressed the cornea center with the finger, in order to "milk" as much fluid as possible, until the cornea became concave.

8. Then, I reformed the tonus and the AC with saline through the corneal puncture and opened the scleral puncture again. The AC became flatter and flatter until it disappeared in parallel with fluid evacuation through the scleral puncture. In order to evacuate more SCD fluid, I repeated the "milking maneuver" described above. When the cornea became concave, I reformed the tonus and AC with saline again. I repeated this veritable suprachoroidal space wash several times, observing that the fluid progressively lost its yellowish color.

9. After as many as six suprachoroidal space washes, I dried the "sink", replaced the "sink" insulation with new PVA sponges, dried the "sink" again, and opened the scleral puncture, while the assistant collected 0.2 - 0.3 ml from the fluid that filled the "sink", into a new, similarly prepared glass syringe. Then, I transferred the collected fluid in a new sterile glass capillary tube and sealed it at both ends with sealing paste. This tube represented the sample no. 2. Both tubes were marked with the patient's name and sample number, and were sent for gas chromatography.

10. In the end, I reformed the IOP with saline, and the AC with air, until the lens-iris plane remained slightly concave. Conjunctiva closure with 1-2 U-shape sutures.

The procedure was practiced on 10 cases with advanced or absolute trabecular [23,24] glaucoma (6 women and 4 men, aged 61 - 87 years), that developed SCD in the immediate postoperative period after Vancea's enlarged trabeculectomy [25] (Figure 2: 9 cases), or after trabeculo-keratencleizis [26], a procedure in which the trabecular strip is not excised, but is detached on 3 sides and reflected over the thinned scleral bed, covered by the scleral flap and caught between two flap sutures (Figure 3: 1 case). In the 2 first cases the second sample was collected after 3 suprachoroidal washes (study group "S3"); in 8 cases the second sample was harvested after 6 suprachoroidal washes (study group "S6").

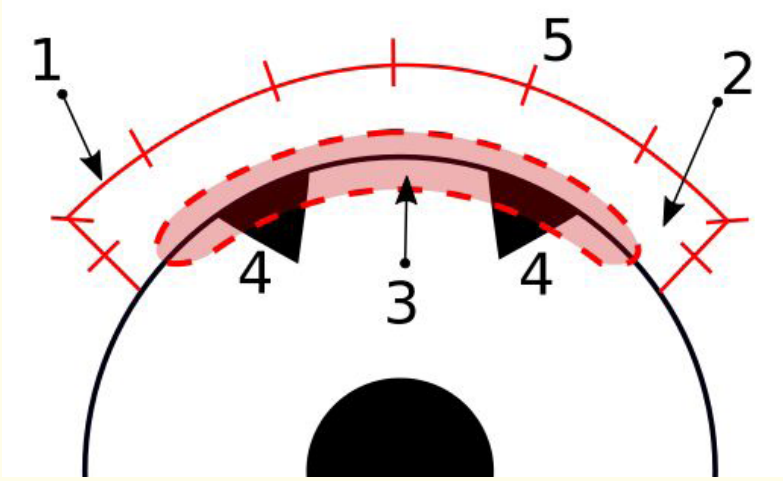

Figure 2: *Vancea enlarged trabeculectomy. The fornix based conjunctival flap and the traction suture under the superior rectus are not figured. 1: the incision line delineating the superficial scleral flap; 2: the superficial scleral flap; 3: the deep fistula after the excision of the scleral strip containing the trabeculum and Schlemm's canal; 4: two peripheral iridectomies; 5: 9-11 separate flap sutures. The conjunctival flap is closed with 2 sutures at the limbus: when radial incisions have been added at the extremities of the conjunctival flap, each incision needs 1-2 additional sutures.*

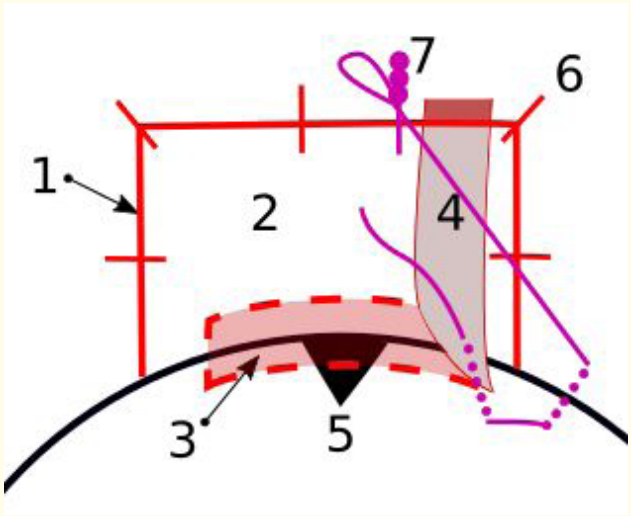

Figure 3: *The "trabeculo-keratencleizis". The fornix based conjunctival flap and the traction suture under the superior rectus are not figured. Usually the conjunctiva incision is "L" shaped, with one arm parallel with the limbus, and one arm - meridional. 1: the incision line delineating the superficial scleral flap; 2: the superficial scleral flap; 3: the deep fistula after tailoring on 3 sides the deep corneo-scleral strip containing the trabeculum and Schlemm's canal; 4: the deep corneo-scleral strip; 5: the peripheral iridectomy; 6: the flap sutures; 7: the releasable suture. Usually, the deep strip is caught between the left corner suture (6) and the releasable one (7). The conjunctiva is closed by one suture on flap corner and one suture on its meridional arm.*

A control group of 10 cases with SCD after G surgery, with similar distribution on sex, age, type and stage of G, and type of surgical procedure, have been selected. In this group, after harvesting the first sample, the AC was reformed with saline, and no SCD space wash and "milking" followed. The second sample was harvested after a pause of 90 seconds - in the first 2 cases ("C90": control group, 90 seconds pause), and of 180 seconds - in the last 8 cases ("C180": control group, 180 seconds pause). The length of the pause was established starting from the fact that in the study group each suprachoroidal wash and "milking" cycle took around 30 seconds to perform. Thus, 3 wash cycles lasted around 90 seconds, while 6 washing cycles lasted around 180 seconds.

In all cases, the SCD drainage surgery was performed in the 7^{th} postoperative day, after the failure of conservative attitude (bed rest, binocular bandage, cycloplegic and anti-inflammatory eye drops, vasotropic, anti-aggregating and anti-inflammatory general treatment). The chromatographic analysis was performed with Beckman spectrophotometer, the margin of error being ± 1.6 mEq/l for Na^+, and ± 0.12 mEq/l for K^+.

Results

In all 20 cases, the saline injected into the AC rapidly disappeared, with AC flattening - in parallel with the evacuation of SCD fluid through the scleral puncture, when opened.

Table 1 synthesizes the gas chromatography data in the study group, focused on Na^+ and K^+ concentrations. Table 2 synthesizes the gas chromatography data in the control group.

	Na^+ (mEq/liter) Serum normal values: 135 - 153 mEq/liter			K^+ (mEq/liter) Serum normal values: 3.5 - 5.3 mEq/liter		
Number of washes	Sample 1	Diff.	Sample 2	Sample 1	Diff.	Sample 2
3	146.0	+5.0	151.0	4.0	- 0.4	3.6
3	147.0	+3.0	150.0	4.1	-0.6	3.5
S3 m ± SD	Na1 146.500 ± 0.7071	p = 0.155958	Na2 150.500 ± 0.7071	K1 4.050 ± 0.0707	p = 0.125666	K2 3.550 ± 0.0707
6	148.0	+2.5	150.5	3.9	- 1.8	2.1
6	144.5	+6.0	151.5	4.1	-1.8	2.3
6	145.0	+5.0	150.0	3.7	-1.2	2.5
6	145.0	+5.0	150.0	4.0	-1.8	2.2
6	147.5	+2.5	150.0	4.2	-1.4	2.8
6	145.5	+4.5	151.0	4.0	-1.9	2.1
6	144.0	+6	150.5	4.3	-2.2	2.1
6	146.0	+6	152	4.1	-2.2	1.9
S6 m ± SD	Na1 145.687 ± 1.4126	p = 0.000272	Na2 150.687 ± 0.7529	K1 4.037 ± 0.1846	p = 0.000008*	K2 2.250 ± 0.2828

Table 1: *Spectrophotometric results in study the study group.*

S3: study group, 3 suprachoroidal washes; S6: study group, 6 washes.

p = ...: Bonferroni correction.*

	Na^+ (mEq/liter) Serum normal values: 135 - 153 mEq/liter			K^+ (mEq/liter) Serum normal values: 3.5 - 5.3 mEq/liter		
Waiting period	Sample I	Diff.	Sample 2	Sample I	Diff.	Sample 2
90 sec.	145.0	+1.5	146.5	4.0	- 0.1	3.9
90 sec.	145.5	+1.0	146.5	4.2	-0.1	4.1
C90 m ± SD	Na1 145.2500 ± 03535	p = 0.090544*	Na2 146.500 ± 0.0000	K1 4.100 ± 0.1414	-	K2 4.000 ± 0.1414
180 sec.	146.0	+0.5	146.5	4.2	0.0	4.2
180 sec.	145.0	+1.0	146.0	3.9	-0.1	3.8
180 sec.	147.0	+0.5	147.5	3.8	-0.1	3.7
180 sec.	144.5	+1.0	145.5	4.1	0.0	4.1
180 sec.	144.0	+1.0	145.0	3.9	-0.1	3.8
180 sec.	146.0	+1.0	146.5	4.1	0.0	4.1
180 sec.	147.5	+0.5	148.0	3.9	-0.2	3.7
180 sec.	147.0	+0.5	147.5	4.0	-0.1	3.9
C180 m ± SD	Na1 145.875 ± 1.2747	p = 0.125666*	Na2 146.562 ± 1.0500	K1 3.987±0.1356	p = 0.079768*	K2 3.912 ± 0.1959

Table 2: *Spectrophotometric results in the control group.*

C90: Control Group, 90 seconds interval between samples; C180: Control Group, 180 seconds interval

p = ...: Bonferroni correction.*

Table 3 facilitates the statistical analysis. The p-value for the comparison of results between the sample 1 and 2 of the same group (S3; S6; C90; C180) was calculated using the Paired Samples T Test, and is written with italic characters between the results of sample 1 and 2. The p-value for the comparison between the groups (S3 - S6; C90 - C180) of the same sample (1 or 2) was calculated with Independent Sample T Test, and is written with normal characters, between the results of the groups belonging to the same sample. When needed, the Bonferroni correction was applied. The highly significant values are written with bold characters.

S3 m ± SD	Na1 146.500 ± 0.7071	p = 0.155958	Na2 150.500 ± 0.7071	K1 4.050 ± 0.0707	p = 0.125666	K2 3.550 ± 0.0707
	p = 0.466682		p = 0.759118	p = 0.930052		p = 0.01052*
S6 m ± SD	Na1 145.687 ± 1.4126	p = 0.000272*	Na2 150.687 ± 0.7529	K1 4.037 ± 0.1846	p = 0.000008*	K2 2.250 ± 0.2828
C90 m ± SD	Na1 145.2500 ± 03535	p = 0.090544*	Na2 146.500 ± 0.0000	K1 4.100 ± 0.1414	-	K2 4.000 ± 0.1414
	p = 0.528172		p = 0937829	p = 0.327183		p = 0.576227
C180 m ± SD	Na1 145.875 ± 1.2747	p = 0.125666*	Na2 146.562 ± 1.0500	K1 3.987 ± 0.1356	p = 0.079768*	K2 3.912 ± 0.1959

Table 3: *Statistical analysis of spectrophotometric results.*

p=…..: Bonferroni correction.*

Discussion

The clinical results demonstrate the contribution of a mechanism denied by modern literature: the cyclodialysis. This mechanism was clinically suggested by the fact that in all tested cases, after seven days of athalamia/hypotony syndrome, the saline infused within AC drained through the puncture in pars planum.

The biochemical results confirm the direct communication between AC and SCD lobes:

A. In sample # 1, the Na^+ and K^+ concentrations are within the normal range found in normal human serum. In sample # 2, the Na^+ concentration increases, but remains within normal limits. This slight increase is explained by the fact that we washed the suprachoroidal space with saline, whose Na^+ ion concentration is 154 mEq/l. The fact that saline does not contain K^+ explains the diminution of K^+ ion concentration in the second sample, more dramatic in cases with more numerous suprachoroidal washing cycles.

B. Before discussing the statistical analysis, one must recognize that the number of cases is too small, so that the results must be taken "with a grain of salt": these results may be used only as indicators for the direction of the change, to be confirmed or denied by further studies on larger groups.

C. Referring to both control groups (C90 and C180), we find another limitation: in all these cases, the differences between sample 1 and 2 are situated within the margins of error of the apparatus. However, the slight increase in Na^+ ion concentration and the slight decrease in K^+ ion concentration in the second sample, after one single AC reformation with saline, suggest the huge capacity of SCD space compared with the content of AC (0.17 ml[14]- 0.25 ml[15]), and the importance of "milking" menuver - if we want to significantly modify the chemical composition of SCD fluid. This aspect could be important in case further research would find that SCD fluid contains substances that oppose communication closure.

D. In the group S3, the effect is more important than in control groups, but still not significant, both for the Na^+ and for the K^+ ($p > 0.05$).

E. The differences between samples 1 and 2 are highly significant only in group S6, both for Na^+ and for K^+. A significant difference exists also in K^+ measurements in sample 2 between group S3 and S6. All these findings prove that a direct communication between AC and SCD lobes exists in the 7th day of the sickness: through this communication, the AC liquid content (AH or the injected saline) may freely circulate towards the supraciliary/suprachoroidal space, prolonging the sickness. Indirectly, they confirm the huge difference in capacity between SCD and AC, as only after 6 washing cycles associated with SCD space "milking" the concentration changes became significant.

The form of communication: The lack of means for investigation prevented me from clarifying this aspect, and this happened because the professional promotion during the communist and postcommunist Romania generally depended on the degree of obedience toward the political leadership of each period. This resulted in the awkward situation that the university clinics were relatively well equipped, but the university staff members did not have original ideas to investigate. Conversely, some of us - provincial doctors, had original ideas, but no possibility to work on them.

As my hospital did not have high resolution ultrabiomicroscopy (HRUBM), an apparatus able to visualize choroidals [27], I tried to visualize the communication on 7 cases, using an original manner of intraoperative dynamic gonioscopy: (i) I reformed the AC with air through the paracentesis, practiced a new puncture for ACM and inserted it, with the line clamped. (ii) I practiced the radial perforant incision of SCD lobe slightly oblique on sclera - to form a short valve and favor its spontaneous closure and passed one traction suture (10/0 nylon) through the incision lip that covered the bevel of the other lip - to control the moment of fluid egress. (iii) I replaced the air in AC with saline, lowered the bottle level until the fluid in it was 3 cm higher than the eye, opened the line and slowly raised the bottle, until the AC remained deep, but not too deep, to avoid excessive zonule stretch. (iv) In 2 cases the dimensions of the filtering bleb increased rapidly, proving that the mechanism is overfiltration: in these cases I opened the conjunctiva approach again, identified the overfiltering site, closed it with a releasable suture, and closed the conjunctiva. (v) In all cases, I placed the surgical direct gonioprism and examined the angle all around with binocular magnifiers, while the scleral puncture was intermittently opened by pulling the traction suture. The results were inconclusive, the more that I had no possibility to record images. However, in one case I believe I saw a cleft in the trabeculectomy zone. Of course, practiced under surgical microscope, with modern gonio lenses, with devices to record the images, the procedure could reveal the shape, number and position of the communication points (clefts, pores or aspects that remain to be described). Unfortunately, I retired and I cannot pursue the research.

The timing and location of the communication are important, as they would indicate the mechanism. A communication identified in G surgery zone from the first day of athalamia, would point toward the iatrogenic mechanism. A communication situated outside the surgery zone, or one that would not exist from the first day of athalamia, would point toward other mechanism, discussed under #5 ("Pathogenic mechanism of communication"), point b. The communication timing will certainly influence the timing of SCD surgery, practiced according to an original procedure which will be described in my next paper.

The pathogenic mechanism of communication:

In my opinion, the communication between AC and SCD lobes could be the cause of SCD, the consequence of SCD lobes development, or a combination of these two mechanisms.

The communication - cause of SCD

I cannot sustain that all SCDs occurring after G fistulizing surgery are caused by the surgeon. However, the fact that SCD appears mostly after G fistulizing surgery seems to suggest the iatrogenic mechanism: in his desire to completely ablate the trabeculum, the surgeon has

pushed the excision beyond ciliary spur and generated either a real cyclodialysis cleft, or a frail zone in iris root insertion. The frailness in iris root insertion could be generated also by iris tractions during the iridectomy, especially in cases with rigid irises or with genetic frailness in iris insertion. The frail zones could be transformed in frank communication sites by the nonsurgical pathway, described below.

The communication - consequence of SCD lobes development

If cyclodialysis was not generated by the surgeon, but represents a late complication of SCD, it can be explained by the change in choroid shape in the first days of SCD: on sagittal section, the normal choroid has the shape of an arc of circle with the centre inside the eye (Figure 4a). In the first stage of SCD, it becomes circle chord (Figure 4b) and it shortens its length by shrinking its structures. After several days of short choroid, when SCD lobes evolve toward either resorption or progression, the choroid length must increase, becoming arc of circle, again: with the centre inside the eye - in case of resorption (Figure 4a), or outside the eye - in case of progression (Figure 4c). In both situations, tensions would be generated within choroid, and transmitted to its points of fixation: vortex veins - posteriorly, and iris root - anteriorly. The posterior insertion is strong: otherwise, blood should have been frequently found in SCD lobes, which was not noted in literature. Only the anterior insertion can break without immediate disastrous consequences: the iris root disinsertion could be facilitated by its genetic frailness, or by a traumatically induced one (surgery included). The disinsertion could remain as small cyclodialysis pores or may confluate in frank cyclodialysis cleft. This is the only way to accommodate my findings with the transudation/exudation mechanism, which is accepted in literature.

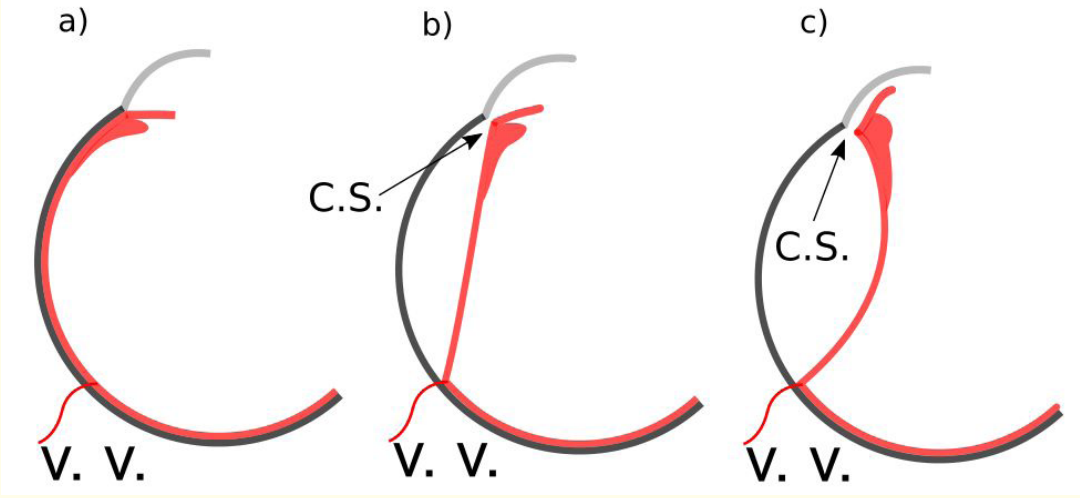

Figure 4: *The nonsurgical mechanism of cyclodialysis slit formation. CS: Cyclodialysis Slit; VV: Vorticous Veins.*

The communication is a factor of disease prolongation

After being produced directly by the surgeon, or indirectly, by hypotony/inflammation mechanisms, the communication will be maintained open by the above mentioned tensions induced in choroid by the evolving SCD lobes.

Summarizing my opinion: No matter which was the "primum movens", after seven days of athalamia with SCD, a direct communication between AC and SCD lobes exists. It might have been created by the surgeon, or it might have resulted from the evolution of SCD lobes.

What is important is that once produced, the communication is maintained open, possibly by the tensions generated in the choroid when passing from the shape of circle chord to that of an arc of circle. This communication might prolong the sickness, because as long as it exists, the SCD lobes exist, and the ciliary tissue is distorted: this could generate secretory shut down and hypotony, accentuating the pre-existing one - in case that the sickness was primarily generated by overfiltration and hypotony; as long as hypotony exists, transudation towards the supraciliary space will continue, accentuating the pre-existing one - in case that the sickness was primarily generated by transudation; as long as abnormal fluid exists within the supraciliary space, inflammation will increase the SCD content by exudation, accentuating the preexisting one - in case that the sickness was primarily generated by exudation.

My study proves that SCD after G surgery is not determined by the double loop vicious circle accepted by modern literature: transudation - caused by hypotony or by increased arterial or venous pressure; exudation - caused by inflammation. In my opinion, the SCD fluid is generated by a triple loop mechanism (transudation/exudation and migration of AH through iris root discontinuities). In this combination, the third component could initiate the whole vicious circle: the hypotony and transudation may occur secondarily, after the secretory shut down - resulting from ciliary body distortion by the SCD lobes; the inflammation may be the answer to the presence of abnormal fluid in the suprachoroidal space. The third component could also intervene as a consequence, in the evolution of SCD lobes produced by other mechanisms. The combination of all these mechanisms may determine and certainly prolong the existence of SCD. If we add the possible genetic influences (genetic frailness in iris root insertion), as suggested by higher incidence of SCD in case of nanophthalmos [5], the result is a quadruple mechanism. Anyhow, my study proves that SCD after G surgery is not determined by a double loop vicious circle (transudation/exudation).

The present study has fulfilled its purpose, proving the intervention of a mechanism denied by modern literature: unwanted cyclodialysis. It was found in all cases after 7 days of athalamia. The practical consequences of this study (the suggestion of prophylactic and therapeutic measures) will be presented in my next paper.

Nevertheless, I must recognize that this study is incomplete: on one side, it was based only on the kindness of Mrs Chemist Dr. Dumitrescu Florica† from "Cablul Romanesc" factory in Ploiesti, who, at that time (1984-1987), was allowed to perform only Na^+ and K^+ analysis. On the other side, I cannot further pursue this research, because I retired.

As a consequence, many questions remain unanswered and some of these are listed below:

1. Is it possible to collect the SCD fluid directly from SCD lobes after placing a delicate catheter through sclera? Would the Na^+ and K^+ concentrations remain the same as when the fluid was collected from the "sink"?

2. What is the complete composition of SCD fluid? What role does the inflammation play in its genesis and what contribution does the anti-inflammatory treatment have in the healing process?

3. What are the results of daily HRUBM investigation as long as the athalamia-hypotony syndrome exists? When do the SCD lobes appear: from day 1, or after several days? Is HRUBM able to identify the AC-SCD communication, its site and shape? Does the communication appear from day one, or after several days?

4. What are the results of the dynamic intraoperative gonioscopy (#3 of "Discussions"), practiced with modern means in the 7^{th} day of athalamia/hypotony syndrome, when the communication AC-SCD usually exists?

5. What are the results of the dynamic intraoperative gonioscopy, practiced in the 6^{th} - 2^{nd} day of athalamia/hypotony syndrome? If the communication is identifiable, at what moment does it appear?

6. Does the indirect signs of communication (athalamia after opening the scleral puncture) exist in cases of SCD after other ocular surgeries, or in the late phases of SCD determined by nonsurgical causes?

7. The pathogenic interpretation of all these new developments: is the communication caused by the surgeon, or it represents the final consequence of an ample SCD lasting too long (or maybe both)? In other words, is cyclodialysis the cause or the consequence of SCD?

Each of these questions may justify an original research conducted by young and enthusiastic ophthalmologists. Of course, some of these projects need approval from the Ethics Committee of the hospital.

Conclusion

1. In the case of SCD after glaucoma surgery, a communication between AC and the supraciliary and suprachoroidal space existed in all tested cases after seven days of athalamia/hypotony syndrome.

2. This communication is clinically observable during the SCD drainage procedure, when the AC is reformed with serum and the scleral puncture is opened: the AC content disappears in parallel with the SCD fluid evacuation.

3. The communication may be proved in a quasi-experimental manner, by spectrophotometric dosage of Na and K ions in SCD fluid, collected before and after several suprachoroidal space washes.

4. This communication could be the cause of SCD or its consequence.

5. The dynamic intraoperative gonioscopy could identify the location, shape, number and timing of communication sites, every aspect influencing the prophylactic and therapeutic attitude.

6. The presence of such communication certainly prolongs the sickness: the hypotony and transudation may occur secondarily, after the secretory shut down - resulting from ciliary body distortion by SCD lobes; the inflammation may be the answer to the presence of abnormal fluid in the suprachoroidal space.

7. Young ophthalmologists could verify these results or may develop the research on at least 7 original directions stated in this paper.

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