



## Case series of endophthalmitis phacoanaphylactic glaucoma

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### Abstract

**Introduction:** Phacolytic glaucoma (PG) was usually caused by hypermature lens and the principal mechanism was the obstructed trabecular meshwork with the leaky lens capsule proteins. The incidence of PG in our hospital where the backlog of cataract was relatively high, occupied three percent of operated glaucoma, one percent of cataract surgeries (by the year two thousand). The PG with rupture crystalline capsule (anterior chamber was not observed) exactly endophthalmitis phacoanaphylactic glaucoma (EPG) is commonly misdiagnosed and was therefore refused lens extraction by local eye doctors at district hospital level. With a new simple surgical procedure lens extraction is applied helping restoration the patient's vision.

**Objective:** To report results of six cases of endophthalmitis phacoanaphylactic glaucoma that were diagnosed and managed by a simple surgical procedure lens extraction.

**Materials/Patients:** Six cases of endophthalmitis phacoanaphylactic glaucoma were selected

**Methods:** Clinical intervention on series cases. A simple surgical procedure was applied for management both diagnosis and treatment after reducing IOP by carbonic anhydrase inhibitor. Paracentesis of anterior chamber in diagnosis and followed by nucleus lens extraction associated with local and systemic steroid treatment pre-surgery and post- surgery.

**Principal Measurements:** Visual acuity (VA): Snellen; Log MAR- Intraocular pressure (IOP): Schiötz, Maclakov.

**Results and Discussion:** Preoperation: VA: No light perception in three cases and light perception in three cases. IOP in all cases are equal and over thirty seven mmHg. Post operation: Increased VA in all cases: 0 point 0 five (0.05) to 0 point three (0.3) and normalized

**IOP:** fifteen to twenty mmHg

**Conclusion:** Patients suffering from EPG should be diagnosed and nucleus lens extraction. After medical treatment for reducing IOP, aspiration of anterior chamber fluid could be done first for both diagnosis and thereafter nucleus lens extraction is a radical treatment will help restored vision for patients. The results of six cases of EPG that were operated by the new simple surgical procedure lens extraction with satisfying results better than expected.

**Keywords:** phacolytic glaucoma, endophthalmitis phacoanaphylactic glaucoma, nucleus lens extraction

### 1. Introduction

In normal eye, the protein of crystalline lens which minimally escape from lens capsule and then the eye has an immunity response but a lot of these proteins excrete into anterior chamber will lead to severe inflammation. The leaky lens capsule proteins are modified, liquefied and liberated. In these condition macrophages phagocytosed these proteins and the trabecular meshwork became obstructed by this phenomenon. Accumulation of lens proteins coexisting macrophages over a short period of time may acutely obstruct the meshwork and result in transient elevation of intraocular pressure (IOP) in association with exercise of dilation of the pupil. Excessive of phagocytosis of lens proteins lead to migration of the trabecular cells spread over the denuded portion of the trabeculae to keep them cover. When the capacity of self-repair is lost the denuded trabecular beams degenerate which results in irreparable damage to the meshwork accompanied by PG. Inability of self-repair may represent a primary defect in the trabecular cells. PG is a severe complication of hypermature lens leading rupture capsule of crystalline lens and some reduce

IOP drugs have a little or no effect on it. The PG with rupture crystalline capsule (anterior chamber was not observed) exactly endophthalmitis phacoanaphylactic glaucoma (EPG) is commonly misdiagnosed and was therefore refused lens extraction by local eye doctors.

For clinical findings there are 3 forms of PG: 1. Phacolytic glaucoma, 2. Lens particle glaucoma (Phacotoxic, phacoallergic) and 3. Phacoanaphylactic glaucoma. The three forms do not separately but each form may also be integrated with another according to cases. PG caused by leaky lens capsules proteins from hypermature cataract but phacoallergic glaucoma did not excluded and it is also called phacolytic uveitis glaucoma. 2 Exam findings: IOP: high (41mmHg), conjunctiva and episclera: dilated vessels, cornea: epithelial and stroma oedema, anterior chamber: opacity. Crystalline lens and others components of posterior chamber: no observe. Tyndall's phenomenon: positive. Anterior chamber fluid: macrophages, polynuclears, lymphocytes... The functional signs included ocular pain, redness, blurred vision, nausea, vomiting...



For medical treatment there are: 1. Therapy of glaucoma: Over the past several years, a number of new medicines have become available that have changed physician's prescribing patterns for both mono and adjunctive therapy of glaucoma [1]. 2. Therapy of lens induced uveitis are local steroids: topical, injection sub conjunctiva, periocular and general steroids. For the surgical treatments, lens extraction with or without intraocular lens is suitable chosen.

Three of six cases of EPG were misdiagnosed because the anterior chamber was not observed especially no perception of light in visual acuity (Figure attach) and were refused lens extraction by local eye doctors at district hospital level. A simple surgical procedure was applied for management both diagnosis and treatment on these patients with some satisfying results were report herein [2, 3].

## 2. Case Series

A simple surgical procedure for both diagnosis and treatment is applied in 6 cases:

- A clear corneal incision at 12 o'clock for aspirating anterior chamber fluid for examining polynuclear, macrophage... in diagnosis as well as for inspecting of expulsive hemorrhage which rarely occurs in these cases. This is the same paracentesis.
- The anterior chamber and iris were then being seen clearer; lens extraction will be performed for radical treatment.

Six eyes of 6 patients of EPG (endophthalmitis phacoanaphylactic glaucoma) were reported in our hospital in the table I below.

**Table 1:** Six cases of endophthalmitis anaphylactic glaucoma

| N Patient | Sex    | Age | Days before Operation | Visual Acuity (Snellen) |        | Intraocular Pressure (Schiort) |        |
|-----------|--------|-----|-----------------------|-------------------------|--------|--------------------------------|--------|
|           |        |     |                       | Preop                   | Postop | Preop                          | Postop |
| 1         | Female | 68  | 9                     | PL(-)                   | 0,2    | 41                             | 15     |
| 2         | Male   | 57  | 5                     | PL(+)                   | 0,3    | 40                             | 17     |
| 3         | Female | 76  | 10                    | PL(+)                   | 0,1    | 37                             | 20     |
| 4         | Female | 67  | 12                    | PL(+)                   | 0,1    | 39                             | 20     |
| 5         | Male   | 78  | 21                    | PL(-)                   | 0,05   | 37                             | 17     |
| 6         | Female | 82  | 19                    | PL(-)                   | 0,05   | 41                             | 20     |

Note (Table I):

-Mean age (years):  $73.33 \pm 9.40$ , range: 57-82.

-Mean of days from onset of disease to surgery:  $12.66 \pm 6.15$ .

-Visual acuity Reoperation: No Perception of Light PL(-): 3 cases, PL(+): 3 cases.

Visual acuity (Snellen) postoperation 3 months: 0.05: 2 cases;

0.1: 2 cases; 0.2: 1 case; 0.3: 1 case. The Spearman rank correlation between the time from onset of disease to operated day with the visual outcome 3 months after surgery: early operation, higher vision ( $r \text{ Spearman} = 0.957, p < 0.05$ )

-Intraocular pressure (Schiozt) preoperation:  $38.83 \text{mmHg} \pm 7.88 \text{mmHg}$  and postoperation 3 months:  $18.16 \text{mmHg} \pm 5.19 \text{mmHg}$  ( $t = 8.26, p < 0.05$ )

## 3. Discussion

**3.1 The incidence:** The incidence of PG in our hospital where the backlog of cataract was relatively high was 3% of operated glaucoma, 1% of cataract surgeries [4]. According to Julia Song, and R. Rand Allingham, special to EyeNet (AAO 2014 Chicago): Will the increase in the number of under- and uninsured patients lead to an increase in this condition? [5].

**3.2 The surgery:** Surgical options in the management of coincidental cataract and glaucoma continue to evolve and improve. Visual recovery is better and more rapid than with expected patients. Combined cataract and glaucoma surgery is the favor approach for all patients with lens induced glaucoma or phacolytic glaucoma [6]. In this report, some cases of the PG with rupture crystalline capsule (anterior chamber was not observed) exactly endophthalmitis phacoanaphylactic glaucoma (EPG) is commonly misdiagnosed and was therefore refused lens extraction by local eye doctors. (District hospital level). No perception of light in vision of three cases were transferred from district hospital with endophthalmitis. With this preferred technique a clear corneal incision from 6 to 9 mm at 12 o'clock according to nucleus lens diameter was done easily. If the lens capsules remained extracapsular cataract with or without intraocular lens was done, but our 6 cases the only nucleus lens, so that nucleus lens was extracted. One advantage of this technique was safe with no bleeding from congestional conjunctiva glaucoma. More recently, the mid- 1999s has witness an increase in the use of clear corneal incision for cataract surgery. I preferred this technique for these cases which may easily observe anterior chamber after aspiration. In our experience, five expulsive hemorrhage patients were seen, included the incidence 0, 1% (2/2,000 cases) of coincident of cataract and glaucoma operation, the incidence 0.03% (3/8,000 cases) of cataract extraction alone.

## 3.3 Some considerations on medical glaucoma treatment

- Autoregulation of ocular blood flow in glaucoma: It is evident that blood flow in the optic nerve head depends on: the mean blood pressure in the optic nerve head capillaries; the intraocular pressure; the vascular resistance in the blood vessels of the optic nerve head; the efficacy of autoregulation of blood flow in the optic nerve head. Altered blood flow to the optic nerve head has a role in the pathogenesis of glaucoma either directly or by increasing the susceptibility of the nerve head to raised intraocular pressure [7].
- The death of retinal ganglion cells in glaucoma: Do retinal ganglion cells commit suicide in glaucoma? The final outcome of glaucomatous damage in the eye is the death of retinal ganglion cells. Retinal ganglion cells die in glaucoma by the normal, way for cells to die, apoptosis, and the violent

death seen with necrosis. Six phases of apoptosis: 1) Apoptosis can be triggered in cells by a variety of different stimuli; 2) Partial chromatin condensation on the inner surface of nuclei; 3) Total chromatin condensation; 4) The pyknic nucleus begins to fragment; 5) The cell has completely fragmented into apoptotic bodies; 6) Apoptotic bodies are phagocytosed by macrophages.

It is important to note that the efficacy of any treatment directed at blocking apoptosis is depend on the primary stimuli of ganglion cell death and the exact genetic pathway [8]

- Retinal ischemia and exitoxicity in glaucoma: As a part of the central nervous system, the retina depends on glucose metabolism for its on metabolic need and is particularly sensitive to ischemic or hypoglycemic insults.

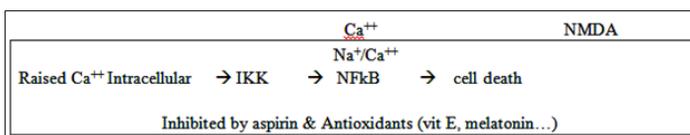
The aminoacids glutamate and aspartate are the most widespread and important excitatory neurotransmitters in the central nervous system.

The physiological actions of excitatory aminoacids are mediated by the activation of specific membrane receptors: the ionotropic (the N methyl D-aspartate, NMDA) and metatropic receptors. NMDA receptor complex showing activated receptor and sites of potential pharmacological modulation :( Figure1) [9].

|                      |   |                      |
|----------------------|---|----------------------|
| <u>Extracellular</u> | Glycine<br>Glutamate<br>Noncompetitive binding site<br>Mg <sup>++</sup> binding site<br>Polyamines site<br>Phosphorylation site<br>Ca <sup>++</sup> → | <u>Intracellular</u> |
| Na <sup>+</sup>      |   | K <sup>+</sup>       |

**Fig 1:** The physiological actions of excitatory aminoacids and sites of potential pharmacological modulation

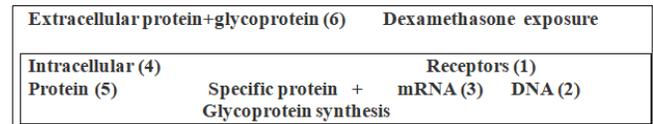
It is theorized that the increase in intracellular calcium post levels that can be controlled by normal cellular homeostatic mechanisms will activate the nuclear transcription factors, nuclear k B (NF-kB) which lead to cell death: (Figure2) [10].



**Fig 2:** Cell death machanis

Steroid glaucoma: In Shiose’s study, the intraocular pressure of human eye was increased by administration of topical steroids after which the optic nerve became cupped and visual field loss developed. When steroids were stopped, the IOP fell, the cupping disappeared and visual field returned to normal [11]. The trabecular meshwork inducible glucocorticoid response gene (TIGR) for adult developed glaucoma. (Figure3) [12].

In our cases of EPG we have to use steroids for lens induced uveitis before and after lens extraction.



**Fig 3:** Human trabecular meshwork for glucocorticoid-effect on IOP



**4. Conclusion**

Patients suffering from endophthalmitis phacoanaphylactic glaucoma should be diagnosed and nucleus lens extraction. Medical treatment for reduced IOP and aspiration of anterior fluid could be done first for both diagnosis and treatment. Thereafter nucleus lens extraction is a radical treatment associated with steroids will help restored vision for patients. With new procedure mentioned above, the 6 cases of endophthalmitis phacoanaphylactic glaucoma were done for diagnosis and treatment having expected patients’ visual acuity.

**5. References**

1. William C Stewart, Perspectives in the medical treatment of glaucoma, Incurrent Opinion In Ophthalmology. 1999; 10(2):99-108
2. Duong Dieu. Endophthalmitis Phacoanaphytic Glaucoma: Diagnosis And Management, The World Ophthalmology Congress (WOC), APAO and JOS 118th in Tokyo-Japan, April 2-7, 2014.
3. <http://duongdieumd.blogspot.com/2014/12/six-cases-of-endophthalmitis.html>
4. Duong Dieu. New Simple Surgical Procedure for Management of Endophthalmitis Phacoanaphylactic Glaucoma. International Journal of Case Studies (Canada) ISSN (2305-509X). 2020; 9(2):1-3. <https://www.casestudiesjournal.com/Volume%209%20Issue%202%20Paper%201.pdf><http://www.aao.org/publications/eyenet/200407/glaucoma.cfm>
5. Duong Dieu, Some considerations on cataract surgery at the health stations of village in An Giang Province, VietNam, Current Pharmaceutical and Medical Information Journal of Ho Chi Minh City. 1995; 8:37-38
6. <http://www.aao.org/publications/eyenet/200407/glaucoma.cfm>
7. Thomas W Samuelson, Management of coincident cataract and glaucoma In: Current Opinion in Ophthalmology. 1998; 9(1):33-38.
8. Sinead Fenton, Colm O’Brien. Ocular blood flow in glaucoma, Glaucoma World. 1998; 17:9-12.

9. Robert W Nickells.: Do retinal ganglion cells commit suicide in glaucoma? *Glaucoma World*, no17, Dec 1998:3-5.
10. Filippo Drago: Retinal ischemia and exito-toxicity, *Glaucoma World*, 1998; 17:6-8.
11. Neville N Osborne, Glynchidlow, Mark S Nah & John PM Wood, The potential of neuroprotection in glaucoma treatment, In: *Current Opinion in Ophthalmology*. 1999; 10(2):82-92.
12. George L Spaeth, *Glaucoma in: Ophthalmology Secrets*, Hanley & Belfus, 1998, 111-116.
13. Polansky JR, Thai D Nguyen. The TIGR gene, pathogenic mechanisms, and other recent advances in glaucoma genetics. In: *Current Opinion in Ophthalmology*. 1998; 9(2):15-23.