Central Retinal Artery Occlusion following Macular Hole Surgery

Dr. Valsa Stephan MS, Dr. Meena Chakrabarti MS, Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS

Introduction

Macular hole surgery has been found to be associated with certain complications such as iatrogenic retinal breaks, retinal detachment visual field defects, glaucoma and cataract. However, cases of central retinal artery occlusion following macular hole surgery have rarely been described. We present a case of central retinal artery occlusion occurring immediately following macular hole surgery, on the first postoperative day.

A 51 year old female was referred to our OPD with a history of defective vision in the right eye of 2 months duration. She was a diabetic and hypertensive of 8 years duration and under good control. On examination, best corrected visual acuity was 6/60 N₃₆ in the right eye, and 6/6, N₈ in the left. Her anterior segment examination was within normal limits. Applanation tonometry was 20 mm in both eyes. A dilated fundus examination revealed a macular hole with subretinal fluid in the right eye (Fig.1).

The left fundus was within normal limits. FFA was done which showed a window defect corresponding to the full thickness macular hole in the right eye (Fig. 2).

An optical coherence tomography revealed a full thickness operculated stage 2 macular hole in the right eye (Fig. 3).
The left eye was normal. She underwent a pars plana vitrectomy with internal limiting membrane peeling in the right eye under retrobulbar anesthesia.

The routine retrobulbar block with facial block using a 5:2 combination of lignocaine with adrenaline and bupivacaine to which an ampule of hylase was added was given. A 23 gauge three port pars plana vitrectomy route was employed. Preservative free triamcinolone acetonide was injected into the vitreous cavity to delineate the posterior hyaloid face and facilitate easy PVD induction. After inducing posterior vitreous detachment, a vitrectomy was done. The internal limiting membrane was then stained with a drop of Brilliant Blue G and ILM peel was performed. A blunt spatula was used to stroke the retinal surface and once the ILM edge was obtained, a maculorhexis was done and peeling completed with an ILM forceps. The intraoperative period was uneventful. Gas tamponade was not used taking into consideration the patients' physical inability to maintain prone positioning.

The patient was examined on the first post operative day. The eye was quite, with an intra ocular pressure of 14mm, no evidence of anterior chamber inflammation, a clear media and an attached retina. The posterior pole of the eye was pale and edematous with a cherry red spot (Fig.4).

The patient was re evaluated for any thromboembolic risks and a cardiology consultation was also performed both of which were noncontributory. The visual outcome was discussed with the patient and relatives and she was discharged on the second post operative day. Subsequent review at 1 month postoperative period showed evidence of gross arterial attenuation and consecutive optic atrophy. The macular hole appeared closed (Fig.5).

Discussion

Macular hole surgery has been found to be associated with several complications. The most important intraoperative complication is an iatrogenic break. Undetected or improperly managed intraoperative breaks can lead to postoperative retinal detachments that may require additional surgery or lead to further visual loss. Incidence of retinal break after macular hole surgery is reported to be 5.5 %, an incidence that is similar to that of vitrectomy for other indications. They may be caused by vitreous traction on the retina during surgical maneuvers, including instrument insertion and withdrawal. It is essential to inspect the entire retinal surface by indirect ophthalmoscopy for iatrogenic retinal breaks before fluid – air exchange. If present, breaks are treated with intraoperative retinopexy and postoperative intravitreal gas tamponade.

Intraoperative light toxicity has been reported in less than 1 % of all patients from the fibre optic endoilluminators.

Complications of orbital regional anesthesia can occur following MHS with the same frequency as in other intra ocular procedures. Table1: gives a list of complication due to regional anaesthesia.

CRAO has been reported following retrobulbar hematoma or an optic nerve sheath hematoma. A high
incidence of CRAO has also been reported in patients having intraocular gas tamponade when they undergo other non ophthalmic procedure in the immediate postoperative period under general anesthesia using nitrous oxide. Acute intraoperative increase in intraocular pressure can result in central retinal artery occlusion and optic atrophy. Hence it is necessary to have a tag on the patient stating the gas filled status of his operated eye.

In eyes with gas tamponade, expansion of the gas in the postoperative period can also result in acute rise of intraocular pressure which may result in severe pain, and can cause occlusion of the central retinal artery. Postoperative rhegmatogenous retinal detachment probably occur in 1-2 % to 14 % of all patients undergoing macular hole surgery. Detachments may occur soon after vitrectomy probably due to intraoperative unrecognized peripheral retinal breaks, or later due to contraction of the remaining peripheral vitreous or further separation of the peripheral vitreous. These are usually satisfactory treated by retinopexy and intravitreal gas tamponade. Peripheral visual field loss after pars plana vitrectomy with fluid gas exchange was first identified in patients undergoing macular hole surgery. The typical field defect is a temporal wedge defect often contiguous with the physiologic blind spot. The field defect has been associated with sectoral pallor of the optic nerve and loss of corresponding nerve fiber layer indicating damage of the inner retina, nerve fiber layer and or optic nerve. Its etiology though incompletely understood, is thought to be related to mechanical trauma during the surgical creation of posterior vitreous detachment, fluid–air exchange or postoperative tamponade with intravitreal gas. It is recognized by the patient within 24-48 hrs postoperatively even in the presence of a large intraocular gas bubble.

Ocular hypertension and glaucoma may also occur following macular hole surgery. Secondary open angle glaucoma may occur due to inflammation or steroid response. Incorrect mixing of gas concentrations intraoperatively may lead to an expansile gas bubble and elevated intraocular pressure.

Progressive opacification of the lens is another reported complication of vitrectomy and macular hole surgery. Other complication such as endophthalmitis/ proliferative vitreoretinopathy may occur infrequently.

Cases of central retinal artery occlusion have very rarely been reported. One case reported occurred 8 months following macular hole surgery in a highly myopic eye. In our patient on analysis of available data it seems likely that her arterial occlusion occurred as a complication of retrobulbar anesthesia. Although the surgery resulted in good macular hole closure, she was left with a vision worse than her preoperative status.

### TABLE 1: Complications of Orbital Regional Anaesthesia

<table>
<thead>
<tr>
<th>Sl.No</th>
<th>Complications</th>
<th>Signs and Symptoms</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Venous Haemorrhage</td>
<td>Retrobulbar hematoma</td>
<td>Tearing or puncture of Orbital Vein</td>
</tr>
<tr>
<td>2</td>
<td>Arterial Haemorrhage</td>
<td>Acute massive RBH with ischemia</td>
<td>Tearing or puncture of Orbital Artery</td>
</tr>
<tr>
<td>3</td>
<td>Vascular Occlusion</td>
<td>Occlusion of CRA</td>
<td>Retrobulbar hematoma or intra sheath hematoma</td>
</tr>
<tr>
<td>4</td>
<td>ON Conduction defect</td>
<td>Transient Visual loss and VF defects</td>
<td>Conduction block by anaesthetic</td>
</tr>
<tr>
<td>5</td>
<td>ON Penetration</td>
<td>Permanent visual loss and visual field defects, ONH swelling ,OA</td>
<td>Ischemic compression by hematoma, trauma to ciliary arteries, traumatic optic neuropathy</td>
</tr>
<tr>
<td>6</td>
<td>Globe Perforation</td>
<td>Pain, ↓ IOP, Intraocular haemorrhage, retinal tear, retinal detachment</td>
<td>Needle perforation with damage to choroid and retina</td>
</tr>
<tr>
<td>7</td>
<td>Needle penetration of Optic Nerve Sheath</td>
<td>Cardio vascular vital signs (↑↓), Respiratory arrest contralateral amaurosis, III N palsy, Hemiplegia, convulsions etc</td>
<td>Central spread of local anaesthetic along submeningeal pathway</td>
</tr>
<tr>
<td>8</td>
<td>Intra venous injection</td>
<td>Bradycardia, hypotension, cardiac arrest, drowsiness, switching</td>
<td>Increased systemic levels of local anaesthetic (CNS and CVS toxicity)</td>
</tr>
<tr>
<td>9</td>
<td>Intra arterial injection</td>
<td>A/c Grand mal convulsive state</td>
<td>Acutely increased cerebral levels of local anaesthetic</td>
</tr>
<tr>
<td>10</td>
<td>Oculo cardiac reflex</td>
<td>Slowing of pulse, nausea , ↓ BP</td>
<td></td>
</tr>
</tbody>
</table>

This complication was a totally unexpected one. We should take care to counsel all patients on the occurrence of complications during local anaesthesia administration also as apart of preoperative patient counselling.

Reference:

Johann Gottfried Zinn

(Anatomist, Ophthalmologist, Botanist…………) & the Zinnia flowers

Prof. Padmaja Krishnan, Calicut

Johann Gottfried Zinn, was born in the German town of Schwabach on the 6th of December 1727. Not much is known of his early years.

He studied Medicine in the nearby city of Ansbach, the capital of Mittelfranken in the German state of Bavaria. He then went to the college town of Göttingen with its famous University and worked under Albrecht von Haller. He was one of Haller’s best students. Obtaining his doctorate in 1749, he went to Berlin where he did extensive research into the anatomy of the eye. Here he also devoted time to the study of his other favourite subject, Botany.

In 1753, Zinn was called back to Göttingen and made director of the Botanical garden in the University. Two years later in 1755 he became Professor of Medicine.

In 1765, Zinn published his masterpiece Descriptio anatomica oculi humani. This book gave the first detailed and comprehensive descriptions of the anatomy of the human eye.

Zinn’s contributions to our understanding of ocular anatomy have been immortalised in the zonules of Zinn and the arterial circle of Zinn-Haller.

His active interest in Botany led to his writing and publishing in this field also, including descriptions of the flora around Göttingen. He described the orchid genus *Epipactis* that belongs to the family Orchidaceae in 1757.

To honour Zinn and his contribution to Botany, Carolus Linnaeus, the father of modern taxonomy, designated as *Zinnia* a genus of annual and perennial flowers in the family Asteraceae, which was native to Mexico and Central America.

Zinnia are old favourites in gardens – in pots, along borders or as background. Their flowers last more than a week, have long stems, come in various bright colours and make excellent fresh cut flowers too.

Zinn died at the age of 32 on 6th April 1759 at Göttingen, probably of lung cancer.

Despite his short life, his contributions to Ophthalmology were great and continue to live after him……
Artificial Vision

Dr Meena Chakrabarti MS, Dr Sonia Rani John DNB, Dr Arup Chakrabarti MS

Introduction

The term artificial vision comprises approaches for restoring vision in blind individuals using device or implants, interfacing with neurons of the visual system \(^1,2\). These systems are based on the electrical stimulation of groups of neurons at several levels of the visual system with multielectrode arrays placed onto or underneath the retina, onto the visual cortex, around the optic nerve, on the sclera or in the suprachroidal space \(^1,2,3,\) and \(^4\).

The history of the artificial vision began when Brindley \(^3,4\) implanted several electrical stimulators close to the visual cortex in a woman who was blind due to the retinitis pigmentosa (RP). After surgery, this patient was able to see spots of lights – electrically evoked phosphenes. Efforts were made to characterize the kind of phosphenes that were elicited with this system \(^3,4,5\).

The Brindley approach was later continued by Dobelle, who implanted several patients with his cortical stimulator. The stimulator was connected to an external power source and to a visual processor with a cable. The information for the visual processor was taken from a camera chip mounted on one glass spectacles and from an ultrasound sensor giving distance information. The Dobelle group reported that the patients were able to see phosphenes, to identify obstacles and to recognize high contrast objects \(^5,6\).

As technology advanced, new concepts were considered. Much smaller devices were designed and fabricated, devices that were remotely controlled, and devices that could be much more efficient in terms of spatial and temporal resolution compared with the historic approach of Brindley. The final goal of artificial vision is not to elicit phosphenes, but to restore vision with spatial and temporal properties similar to natural vision, vision that can be used by blind individuals to improve their daily life and performance, not only to restore spatial and temporal resolution in a picture, but also to restore the emotional content of the vision, such as the recognition of a beautiful landscape, or the face of a beloved friend.

Current Concepts for Restoring Vision Using Electrical Stimulation

Artificial vision uses electrical stimulation to drive neurons of the visual system, which are depleted of their natural input. Usually, electrical stimulation is provided in such concepts by implants consisting of an array of simulated electrodes and electronic components, e.g., for pulse generation. Two main concepts evolved, one is that the optic path of the eye is still used to transmit visual transformation. In the second concept visual information is obtained by a camera system. This information is then further processed depending on the level of the visual system where the stimulation is intended.

In the original idea of subretinal stimulation the implantation of thousands of very small microphotodiodes in the subretinal space was planned. These elements could transform light coming naturally through the optical path of the eye into electric current strong enough to drive postsynaptic cells. The microphotodiodes were intended to replace the photoreceptors. In this concept additional data processing or energy supply was not required. It was
thought that the postreceptoral retinal data processing would be done by postsynaptic neural network, which was thought to be more or less intact. Chow et al. implanted several patients with such a system, “an artificial silicon retina” (ASR) in the subretinal space. The surgery was carried out without complications and the patients reported visual sensations in the first year. Unfortunately after a longer follow-up the patients reported that the percepts disappeared, and they were as blind as before the implantation. It turned out the devices did not generate enough power to drive postsynaptic cells. Most likely, the primary percepts were the result of an unspecified effect of mediators and other cell signal molecules released after surgery.

In approaches interfacing with ganglion cells or cells in the visual cortex, camera systems and data processing algorithms with application-specific hardware are used to obtain visual information and to calculate optimal stimulation pulses. Furthermore, in such approaches data processing algorithms will be modified by the percepts of user in a training procedure.

Interfacing the Neurons

In RP the photoreceptors degenerate. However, postsynaptic neurons also show considerable changes in the degenerated retina with a loss of cell bodies and a chaotic disorganization. In advanced cases of RP a certain amounts of ganglion cells remain alive, but a huge amount of remodeling occurs in which new circuits are established and neurons migrate along glial structures forming microneuromas. The typical layered structure of the retina with known functional connections is destroyed (Fig 1). Electrical stimulation to restore neural function uses charge delivery from a stimulating electrode to adjacent cell membranes so that their membrane potential is considerably modulated. By changing this membrane potential a neuron may fire action potential or release neurotransmitters at its synaptic terminal, thus making the neuronal chain functioning again as a response to stimulating pulses emitted from electrodes of an implanted stimulation device. However making predictions as to which cell will be stimulated and which postsynaptic cells will be activated is nearly impossible because of the structural and functional remodeling of the degenerated retina and because in the clinical situation it cannot be exactly planned where stimulating electrodes will be placed with regards to the position of target cells. If specific activation of cells is the goal, then it is desirable to have as many electrodes as possible to contact as many neurons as possible in a 1:1 ratio. Electrodes should be placed as near to the target cell as possible. Charge delivery may include adverse events in the target issue or in the material of the electrode; therefore, certain safety ranges of charge delivery should be taken into account. As consequence electrodes cannot not be made as small as possible because the charge density would be enhanced, which is the main parameter in terms of electrode material stability and safety. Currently in approaches using electrodes on the retinal surface, electrodes are fabricated in diameters of 40 -200 mm. Electrode materials are platinum or gold with or without regular or sputtered iridium oxide. Surface modification of these electrodes is used to increase the surface area of the electrode without increasing the electrode diameter in order to reduce the charge density to protect both the material and also the tissue against side-effects of chronic electrical stimulation. These large electrodes could be placed close to the outer surface of the retina as well as underneath the retina. However, compared with the cell the electrodes are still very large and single
cell stimulation is not possible. Whole cell clusters will be activated with such large electrodes. However, by intelligent selection of stimulus parameters the activation of certain cell types may be possible even when the electrode is adjacent to a cell cluster. Technical difficulties are explained by the power needed to individually activate thousands of electrodes and by the electronics to transmit such a very high density signals within a biologically safe range of power.

**Epiretinal Stimulation**

Based on the early experiments of Dawson and Radtke, but also on the consideration that in RP more damage is in the outer retina than in the inner retina, strategies were developed based on multielectrode arrays fixed onto the inner retinal surface. The aim of this concept is to stimulate ganglion cells. The electrodes are usually mounted on a flexible substrate; usually polyimide is used for this purpose. The electrodes are driven by power sources either outside the eye or inside the eye, then it has to be controlled remotely via inductive or optoelectronic signal end energy transfer. If the power source is outside the eye, it is necessary to connect the multielectrode array with a cable to the power source. The cable has to cross the wall of the globe, usually through the choroids and sclera. Such an implant with a transscleral cable connection to an epiretinal multielectrode array was fabricated by Mahadevappa and colleagues and has been evaluated in a pilot clinical trial. Theoretically, a trans retinal cable may be at risk of intraocular infection or the risk of shearing forces transmitted to the implant and its anatomical interface with the inner retinal surface when the eye is moved. However, no reports exist on such potentially adverse events. The approach being used by Horing et al. also consist of an epiretinal multielectrode array, a transscleral cable connection, and a data and energy system in which a receiver coil is mounted on to the scleral surface. The group of Walter and Mokwa fabricated an epiretinal device in which the transponder coil is implanted in the capsular bag, meaning that no cable passes the wall of the globe.

A crucial problem in epiretinal stimulation is the stimulus paradigm. Usually, ganglion cells receive preprocessed data from retinal interneuron and not only information on which receptor is activated by light. Therefore camera data resembling receptor activation have to be processed in an encoder simulating retinal data processing. Adaptive spatiotemporal filters are used to process the camera data and the output of this processing is then used to stimulate the ganglion cells.

Because it is previously not known which ganglion cells are stimulated, the encoder properties have to be modified in a learning procedure based on the percepts of the patient, which should be as near to the input signal as possible.

**Subretinal Approach**

In the subretinal approach micro photodiodes are implanted underneath the retina. The original idea of Chow et al and Zrenner et al was that thousands of
such photodiodes would act as artificial receptors and change the light into a current large enough to drive postsynaptic cells in the retina. However, it was found that the current generated by the currently available microphotodiodes was not strong enough to drive these cells. Therefore, implants are now fabricated with an additional power supply. Zrenner and his group was able to demonstrate in patients that direct subretinal electrical stimulation can elicit phosphenes.
in patients over a certain time period and that patients were able to detect some basic geometries\textsuperscript{34}. At present, it is not exactly clear what cells are activated in subretinal stimulation in advanced cases of RP because the retina shows a significant amount of destruction of the original layer structure making ganglion cells reach even as far as the outer retinal surface. It may be disclosed in the future that with both approaches ganglion cells and post synaptic cells were activated from the epiretinal or from the subretinal side. That may also mean that the processing of the input may be necessary for both approaches (Fig. 4 a-e).

In both the epiretinal and subretinal approaches major surgical steps have to be taken, meaning that both approaches have a certain risk profile. Therefore, approaches are considered to minimize the surgical risk. Electrode arrays may be placed with their basic structure onto the outer scleral surface or in a transcleral pocket\textsuperscript{35, 36}. Needle-type electrodes should penetrate into the suprachoroidal or into the subretinal space to get close to the target cells. Such approaches are not free of surgical risk because placement of such structures at the posterior pole may cause trouble with the ciliary arteries and sharp electrodes may penetrate deep within the retina (Fig. 5). In concepts in which the electrodes remain on the scleral surface or in the suprachoroidal space the main problem remains the distance between the electrode and the target issue. However, in a pilot trial Kanda was able to demonstrate in normal volunteers that they have phosphenes with stimulus intensities similar to those reported in epiretinal stimulation and that they were also able to differentiate different sizes of phosphenes, depending on stimulus parameters\textsuperscript{24}.

**Optic Nerve approach**

There is some experience in connecting peripheral nerves with cuff electrodes. Therefore, such cuff electrodes were used to contact the optic nerve in two experiments by Delbeke et al\textsuperscript{37}. They found that they could elicit phosphenes in their patients. Patients were able to recognize objects after a long learning period and the object identification took several minutes for scanning.

The optic nerve was approached first in a neurosurgical approach at the level behind the orbit and in a second experiment within the orbit. Thresholds for electrical stimulation differed significantly and were much lower in the cranial approach than in the orbital approach\textsuperscript{38 39 37}.

From a theoretical point of view, a prosthesis using stimulating electrodes around the optic nerve fibres may have the problem of good spatial resolution because in the optic nerve the fibres are very densely packed and therefore a large amount of fibers could have been stimulated. Whether perforating electrodes are a solution to that theoretical problem remains unanswered at present.

**Cortical Prosthesis**

A large portion of the central nervous system is involved in the processing of visual information and the primary target of fibers from the lateral geniculate nucleus is layer 4 of area V1 at the occipital pole of the brain.

In this area good retinal topography is found with the large representation of the fovea at the most posterior parts and the peripheral representation in the more inwardly located, smaller areas of V1. Approaches in restoring vision in patients suffering from glaucoma or trauma to the optic nerve in contrast to retinal prostheses, may necessitate stimulation of neurons in V1 by passing other parts of the visual system\textsuperscript{40}.

Multielectrode arrays have been developed by Norman and his group based on silicon needle arrays\textsuperscript{41}. Such multielectrode arrays can be used for stimulation as
well as for recording. Animal experiments showed relatively little fibrotic response around the electrode tips. The electrodes could be placed very near to the somata of the neurons in V1. Silicon–based multielectrode arrays were also implanted in trial in 6 patients who underwent brain surgery. It was shown that the stimulators could be inserted with a pneumatic shooter. Excised brain tissue showed only minor alteration such as small bleeds and deformation.

**Pixel Vision and Filters**

The possible resolution of artificial vision by using retinal implants was evaluated in animal experiments in which cortical response were obtained from the cat’s visual cortex or by optical imaging. These data show a possible spatial resolution of about 1° in space and 25 images per second. However, relatively little data are known on the percepts of patients wearing the first available prototypes with regard to picture quality. In the pilot trials that are currently running threshold measurements are taken and standardized tests are used where the single electrodes or groups of electrodes were activated. Patients are asked if they can see something, or if they can see separate spots of light or a line. Patients are also asked if they can identify the orientation of a line, whether it is a horizontal or vertical line. A systematic analysis of the presentation of the real pictures has not yet been carried out. Patients who are implanted with the 16-electrode array used by Humayun’s group and who are already connected to a camera system reported that they can identify high contrast obstacles or that they can now find the door in the wall of a house. To learn more about this kind of artificial vision with only a few implanted electrodes, simulation were performed based on certain assumptions. The simplest simulation is pixel vision where each electrode is considered as a pixel in a rectangular montage. Such stimulation can be seen in Fig. 6. It becomes obvious that the percept depends on the complexity of the real picture. For very simple pictures, e.g., a dark door in a bright room, only a few electrodes are necessary to identify the door, for complex pictures such as the face of a person, many electrodes are necessary for face recognition (Fig. 7).

To point to a person 48 electrodes are necessary, but for face recognition in the example shown in Fig., 864 electrodes are necessary. In contrast, to identify the door as in Fig. 6, i.e. to move to the door, only 12 electrodes are necessary. To identify the obstacle right in front of the door to the right and to enable free movement to the door 48 electrodes are needed, but to find the door opener again 864 electrodes are necessary.

For paragraph reading Dagnelie and coworkers found that 256 electrodes placed on a 3 x 3 mm retinal implant are necessary.
Performance with such a low number of electrodes in similar test can be further improved by the design of multielectrode array. It may be useful to place electrodes with a high density in the centre of the device if it is implanted on to the macula and with a low density in the peripheral area of the multielectrode array. Performance will also depend on the size of the electrode array and the efficacy with which the electrodes make contact with retinal neurons.

Conclusion

Artificial vision comprises approaches to electrically stimulating the neurons of the visual system to bypass degenerated receptors or other neurons to restore vision in otherwise blind individuals. Stimulation can take place at the level of the retina with either subretinal or epiretinal electrodes, but also with the electrodes placed onto the scleral surface or electrodes, in the suprachoroidal space. Stimulation has also been effected at the level of the optic nerve and in the visual cortex. These concepts are currently being evaluated in pilot clinical trials providing safety data. Future developments will concentrate on increasing the number of implanted electrodes, on reducing the surgical risk, on optimizing stimulus paradigm strategies, and on modulating the degenerative process by electrical stimulation.

Reference:

Asteroid Hyalosis and Diabetic Retinopathy

Dr. Meena Chakrabarti MS, Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS

Asteroid hyalosis (asteroid hyalopathy, asteroid hyalitis) is a monocular non-inflammatory disorder of the vitreous characterized by an accumulation of minute white spherular particles within an otherwise apparently normal vitreous gel. The condition affects ~1% of the general population and occurs in the elderly. Men are mainly affected. The condition is rarely familial and it has been associated with diabetes mellitus, hypertension, atherosclerosis, gout and hyperopia. The particles, which move within the vitreous, are composed of calcium soaps (calcium stearate and calcium palmitate). They appear gray in hematoxylin and eosin stained preparations, but lipid and calcium can be detected histochemically in appropriately prepared specimens. The asteroid bodies are moderately positive with the periodic acid-Schiff reaction and they exhibit a vivid “Maltese cross” pattern of birefringence using polarization microscopy. Asteroid hyalosis does not affect vision and patients are asymptomatic. Examination of the eye reveals countless creamy white stellate opacity within the vitreous that resemble snowballs or Christmas ornaments. Asteroid hyalosis is occasionally confused clinically with synchisis scintillans and amyloidosis.

Signs and symptoms: Asteroid hyalosis is a primarily unilateral disorder that typically occurs in patients over 60 and in men twice as often as women. Usually asymptomatic, in severe cases asteroid hyalosis can mildly affect visual acuity. Complaints of floaters are a rarity.

Ophthalmoscopically, asteroid hyalosis appears as multiple, discrete, refractile yellow or yellow-white particles suspended in the vitreous. In early stages, there are fewer bodies and they accumulate in the inferior vitreous. Advanced cases can be so dense as to impair your view of the posterior fundus.

Asteroid hyalosis (AH) is a benign condition characterized by small white or yellow-white spherical or disc shaped opacities throughout the vitreous. The frequency of this condition in the general population is about 0.042 to 0.5% affecting all races with a male to female ratio of 2:1. In whites the prevalence of asteroid hyalosis is 1-2%, and it is bilateral in about 10% of cases and this prevalence seems to increase with age. Asteroid hyalosis is unilateral in 75% cases. The aetiology of asteroid hyalosis is not clearly understood. The association of asteroid hyalosis and diabetes mellitus has been a debatable issue in ophthalmology.

There have been reports which suggest an association between the two conditions while others dispute any such association.

- The controversy regarding an association between asteroid hyalosis and diabetes mellitus has been one of the longest disputes in ophthalmic literature. Multiple studies are present either indicating definite association between the two conditions or no association at all.

- Zinn reports 27% patients with asteroid hyalosis are diabetic, while Bergren reports that 29% of his asteroid hyalosis patients were also diabetic. Bilateral asteroid hyalosis was found in 37.5% of our patients. There are various and differing reports regarding involvement.
of both eyes. Moss\textsuperscript{11} reported approximately 9\% bilateral cases of AH, whereas according to Zinn\textsuperscript{9} it was 25\%. Jones\textsuperscript{12} has also documented a patient with acquired asteroid hyalosis in a case of early diagnosed diabetes mellitus which strongly supports association between the two conditions.

Asteroid hyalosis has been described in association with other systemic diseases such as systemic arterial hypertension and atherosclerotic vascular disease\textsuperscript{5}. Owing to association with systemic conditions, it has been suggested that asteroid hyalosis may be secondary to some form of vasculopathy in many frequencies and that diabetes mellitus is one of the conditions that may be associated with formation of asteroid hyalosis.

Pathophysiology: Asteroid bodies represent small calcium-laden lipids suspended within and attached to the hyaluronic acid framework of the vitreous body. While we understand the composition of the asteroid bodies, the exact genesis remains unclear. Current theories suggest that asteroid hyalosis results from aging collagen within the vitreous or a depolymerization of hyaluronic acid.

Management: Asteroid hyalosis is a benign condition in itself. Although it progresses, it never leads to severe vision loss, and the mild symptoms occur rarely. The vast majority of cases merely require documentation. More often than not, this disorder poses a greater challenge to the examining physician because it can obscure details of the underlying retina. Consider treatment only in patients who are also being managed for retinal disease (proliferative diabetic retinopathy, retinal tear or detachment). Vitrectomy is typically indicated in these instances. In vitrectomy for PDR with asteroid hyalosis, and in cases of simple vitreous hemorrhage, surgery should be performed with full understanding of the anatomic characteristics. Notably, if posterior vitreous detachment is not present, the occurrence of iatrogenic retinal breaks is more likely. Complete posterior vitreous detachment (PVD) occurred less often in eyes with asteroid bodies than in control eyes and partial PVD occurred more often in eyes with asteroid bodies after the age of 70 years, the prevalence of PVD, either complete or partial, was lower than in age-matched control eyes and the prevalence of liquifaction (19\%) was lower than has been reported in controls. The presence of asteroid bodies may arrest the process of vitreous collapse or contraction and that diabetes might influence the development of asteroid hyalosis.

Clinical Pearls:

- Asteroid hyalosis presents a picture akin to “stars in the night sky.” On eye movement, the asteroid bodies sway within the vitreous, but always return to their original position.
- Synchisis scintillans and amyloidosis are often confused with asteroid hyalosis.
- Synchisis scintillans (cholesterol bulbi) is an extremely rare condition that occurs in severely diseased eyes. This condition also presents with refractile crystals in the vitreous, although these particles are composed of cholesterol. They are not attached to the vitreal framework, so they tend to settle out inferiorly after eye movement. Because this condition occurs in end-stage eye disease, pathologists rather than clinical optometrists or ophthalmologists typically make the diagnosis of synchisis scintillans.
- Amyloidosis of the vitreous is also quite rare, and occurs typically after age 40. Patients characteristically demonstrate bilateral involvement with granular, strand-like opacities within the central vitreous. These membranes are anchored to the posterior lens surface in about half of patients. Small, yellow-white bodies dot the vitreal strands.
- Remember that the density of asteroid hyalosis does not correlate with visual dysfunction. If a patient presents with significantly diminished acuity, asteroid hyalosis is not to blame.
- Patients with asteroid hyalosis and an unknown medical status require evaluation for diabetes, hypertension, hyperlipidemia and atherosclerotic vascular disease.
- Filters used for performing fluorescein angiography can allow a better fundus view through the retinal camera in cases of severe asteroid hyalosis and may allow you to observe pathologies that conventional ophthalmoscopy does not reveal.
Photoessay of a 65 years old chronic diabetic with asteroid hyalosis and proliferative diabetic retinopathy

Fig. 1. (a & b): Color fundus photograph (a & b) showing an unclear view of the fundus details. The vitreous cavity shows specular refractable particles of asteroid hyalosis. Observe the difficulty in making out the fundus details in this 55 year old chronic diabetic with proliferative diabetic retinopathy in her right eye.

Fig. 2. (a & b): Fluoroscein fundus angiography pictures of the right eye of the same patient the posterior pole with tracking microaneurysms.

Fig. 3. (a& b): Postvitrectomy color fundus photograph of right eye on the 2nd postoperative day showing pigmented laser burns and a dry macula.
Reference: