DOWNLOAD PDF CHAPTER 7: RETINA AND VITREOUS

Chapter 1 : Chapter 4: Beginner's guide to the Retina - calendrierdelascience.com

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The iris is the pigmented diaphragm separating the anterior and posterior chambers. It is joined to the ciliary body at the iris root. The anterior surface is composed of a condensed layer of fibroblasts, melanocytes, and collagen fibrils. The iris stroma contains melanocyts, fibroblasts, and blood vessels arranged in a loose network. The posterior surface of the iris is composed of two pigmented epithelial layers Figure The two layers have interwoven microvilli and are attached to each other laterally by desmosomes. The more anterior iris epithelial cells are fusiform in shape and extend myofilamentous cytoplasmic processes into the iris stroma the dilator muscle. The posterior epithelial cells are columnar in shape. Both layers are densely pigmented The iris is rarely sampled by cytologic techniques. However, normal iris may appear in intraocular washings from incidental ocutome cutting of the iris in an attempt to remove vitreous or lens fragments in the anterior chamber Figure Normal iris also may appear in fine needle aspiration specimens of iris neoplasms. In general, normal iris epithelium is so densely pigmented that cellular details are obscured Figure Iris stroma is characterized by the fine reticular meshwork of very cohesive and vascularized stroma. The lens is an encapsulated, biconvex structure that is suspended by thin zonules that are attached to the ciliary body. The lens epithelium is located on the internal surface of the capsule. The interior of the lens is composed of cortical and nuclear cells. These hexagonally shaped anucleate cells are joined by interdigitations Figure Because lens cells migrate anteriorly during embryogenesis, the posterior surface of the normal adult lens has no epithelium. Thus, the posterior surface of the lens covered only by a capsule Figure The lens is generally sampled during vitrectomy or lensectomy. The lens capsule can be identified on cytology preparations as a translucent glass membrane Figure This appears light green with Papanicolaou stain. Cortical fragments taken from the lens periphery or the bow sometimes demonstrate nucleated cells. Lens fragments appear as eosinophilic hexagonal structures by hemtoxylin and eosin, and light green with Papanicolaou stain Figure About 70 radially arranged ciliary processes form the pars plicata anteriorly and are joined posteriorly with the smooth portion of the ciliary body, the pars plana Figure The pars plana joins the retina and choroid at the ora serrata. The ciliary body is covered by two nonpigmented layer and an outer pigmented layer Figure Under normal circumstances, ciliary body structures will not appear in vitrectomy specimens. However, ciliary epithelium may be sampled by fine needle aspiration of adjacent tumors. It is important to recognize the two-layered structure of the epithelium with abundant cytoplasm and large pigmented granules Figure The vitreous normally contains anteroposterior oriented collagen fibrils and occasional macrophages or hyalocytes The presence of even small numbers of acute or chronic inflammatory cells within the vitreous is distinctly abnormal. The vitreous has distinct attachments to ocular structures It is attached anteriorly in a circumferential band extending from the posterior pars plana to a few millimeters behind the ora serrata in what has been termed the vitreous base. Traction exerted by the vitreous body at the base results in hyperpigmentation of the underlying pigment epithelium and is evident grossly 16 Figure below. Below-transillumination highlights the pigmentation of the vitreous base as it straddles the ora serrata. The vitreous is also attached to the retina over retinal blood vessels and at the optic disc These attachments are important to understanding vitreous traction, retinal tears, and retinal detachment, for which vitrectomies are sometimes performed. These include the layer of outer and inner segments of the photoreceptor cells, the outer nuclear layer cell bodies of photoreceptor cells , the outer plexiform layer, the inner nuclear layer, the inner plexiform layer, the ganglion cell layer, the nerve fiber layer, and the inner limiting lamina membrane. Normal and abnormal retina and pigment epithelium may be sampled in both vitrectomy and fine needle aspiration. In vitrectomy, the retina may be removed inadvertently. In cytologic preparations, the retina usually appears as a plexiform pattern of cells with round nuclei and characteristic organoid architecture Figure Often only small fragments of retinal tissue will be present, but can be recognized by the organoid architecture and distinctive

nuclear halos Figure Occasionally, ganglion cells may be sampled Figure It is important for the cytologist to report retinal fragments discovered in intraocular washings because full thickness breaks in the retina may lead to retinal detachment. If the surgeon is made aware, the breaks may be closed with cryotherapy, laser, gas injection, or scleral buckle. Fragments of partial-thickness retina that have been stripped in the process of peeling membranes from the retinal surface are not uncommon in intraocular washings and are not regarded presently as clinically significant.

Chapter 2 : Chapter â€“ Retinal Breaks | Free Medical Textbook

An overview of the structure and function of the retina and its relationship to the pigment epithelium, choroid and vitreous is provided. This section describes the major vitreoretinal disorders and appropriate diagnostic methods and treatment principles.

Binocular indirect ophthalmoscopy with scleral depression to see the retina in relief, supplemented by Goldmann three-mirror examination, remains the standard method by which to differentiate these lesions. The differential diagnosis of retinal breaks is given in Box See Chapter for a more detailed discussion. TREATMENT Upon the discovery of a retinal break, the initial decision is whether the benefits of treatment to prevent retinal detachment outweigh the risks and cost of treatment. In each case many factors should be considered and the risks and benefits of treatment discussed with the patient. The factors under consideration in each case include the presence or absence of symptoms; age and systemic health of the patient; refractive error of the eye; location, age, type, and size of the break; status of the fellow eye; and whether the patient is aphakic, pseudophakic, or will soon undergo cataract surgery. The typical symptoms associated with an acute retinal break are new floaters and flashes. These symptoms occur secondary to an acute PVD. Studies have shown that the presence or absence of symptoms in association with the onset of the break is the most important prognostic criterion for progression to retinal detachment. In phakic patients who have no previous history of retinal disease or of high myopia and who develop asymptomatic horseshoe tears, atrophic holes, or holes with opercula, prophylactic treatment is rarely indicated. In each case, the patient should be made aware of the symptoms of vitreous traction and retinal detachment and should be instructed on how to assess the peripheral visual field. Age and systemic health status of the patient are other variables to be considered in the management of a retinal break. As an example, a superotemporal horseshoe tear in a year-old patient is more likely to cause a subsequent retinal detachment than is one in an year-old patient who has metastatic lung cancer. Refractive error is another variable to consider in the management of retinal breaks. The increased incidence of retinal detachment in patients who have greater than 6D of myopia may increase the likelihood for treatment of an asymptomatic retinal tear. The age, location, and size of a retinal break are also considered when its management is determined. Long-standing tears often have retinal pigment epithelial changes adjacent to them. These changes indicate to the clinician the decreased likelihood of retinal detachment. Although no increased incidence of retinal detachment occurs with a retinal break in any particular quadrant, a greater likelihood of a macula-off retinal detachment is present as a result of superotemporal breaks than of either inferior breaks or nasal breaks. Although small retinal breaks can lead to retinal detachment, most ophthalmologists agree that in general larger breaks are more likely to cause a retinal detachment. The type of break should also be a consideration in whether prophylactic treatment is offered. A horseshoe tear with persistent traction or a retinal dialysis is much more likely to result in a detachment than is an atrophic hole. Some controversy exists about the management of asymptomatic horseshoe tears in patients who need cataract surgery, in aphakic or pseudophakic patients, and in patients who have retinal detachments in their fellow eye. Retinal dialyses, whether traumatic or idiopathic, have a high association with the development of retinal detachment. In these cases, prophylaxis is usually indicated. Asymptomatic holes in lattice degeneration rarely lead to detachment and usually receive no prophylaxis. Retinopexy Two main modalities are utilized in the treatment of retinal breaksâ€”cryopexy and laser photocoagulation. Cryotherapy is delivered transconjunctivally. It destroys the choriocapillaris, retinal pigment epithelium RPE , and outer retina to provide a chorioretinal adhesion between the tear and the adjacent retina, which prevents liquid vitreous access through the hole and into the subretinal space. The adhesion with cryotherapy is not immediate; 1 week is required to achieve partial adhesion and up to 3 weeks for the full adhesive effects to occur. Laser photocoagulation treatment of retinal breaks typically utilizes the argon green, argon blue-green, krypton red, or diode laser. No evidence exists that one wavelength is better than another. Two main delivery systems are used, the slit lamp and the indirect ophthalmoscope. In contrast to cryotherapy, chorioretinal adhesion occurs the instant that the laser photocoagulation is applied, but maximal adhesion occurs 7â€”10 days later. Often,

either of the techniques can be used for successful prophylaxis of retinal breaks. However, certain circumstances dictate which modality is easiest and has the best chance of success. Cryopexy has the advantage of not requiring a perfectly clear media; it can be delivered adequately despite the presence of extensive cataract, anterior or posterior capsular opacity, or relatively dense vitreous hemorrhage. Media opacity can make adequate treatment of retinal breaks by laser nearly impossible. In general, retinal cryopexy and indirect ophthalmoscopic laser photocoagulation are preferred for anterior retinal breaks because of difficulty in treatment of the anterior margin at the slit lamp. Similarly, posterior breaks are difficult to reach with the cryoprobe unless a conjunctival incision is made. These breaks can be managed more easily with the slit lamp or an indirect laser delivery system. Occasionally, breaks with a large anteroposterior extent require both cryopexy to the anterior and photocoagulation to the posterior margins of the break. Patients who have a retinal tear and no detachment may have an avulsed retinal vessel with persistent traction and recurrent vitreous hemorrhage. In these cases, scleral buckling or vitrectomy may be necessary to relieve traction and prevent further hemorrhage. If multiple large breaks are present and the patient is unable to tolerate the treatment, retrobulbar anesthesia may facilitate completion of the procedure. Cryopexy Under indirect ophthalmoscopic visualization, the cryoprobe is placed on the conjunctiva that overlies the break and cryotherapy is delivered until the retina adjacent to the tear becomes gray-white. Multiple applications are placed until the break is surrounded completely with confluent treatment see Figs. An attempt should be made not to treat the choroid and RPE directly beneath the break, especially in large tears, because of disruption and displacement of RPE cells into the vitreous cavity and concerns of macular pucker and proliferative vitreoretinopathy. In horseshoe tears, the anterior retina between the tear and the ora serrata should be treated, as anterior extension of the tear secondary to continuous vitreous traction can lead to retinal detachment. Photocoagulation The Goldmann three-mirror lens or panfundoscope lens is used when treatment is with the slit-lamp delivery system. The tear should be surrounded completely by three to four rows of laser burns. Although the spots need not be confluent, there should be no more than half a spot size of untreated retina between burns. Typically, the settings are â€”? The indirect laser delivery system can also be used to treat retinal breaks. An advantage of this technique is that simultaneous scleral depression allows treatment of anterior tears and even dialysis. As with cryopexy, care should be taken to treat thoroughly the anterior margin of horseshoe tears to prevent anterior traction that reopens the break. If a subconjunctival injection has been utilized, a topical antibiotic corticosteroid preparation may be used for the first 2â€”3 days. Subsequently, the eye is reexamined after approximately 7 days. Although vigorous patient activity is often discouraged initially, no clinical study has suggested that diminished activity improves treatment results. A firm chorioretinal adhesion is present by 3 weeks after either technique. Failure rates for prophylactically treated retinal breaks depend on many factors, which include the type of retinal break, indications for treatment, length of follow-up, and definition of failure. Risk factors for failure in this series included aphakic or pseudophakic status, acute symptoms, retinal detachment in the fellow eye, and male gender. An exceedingly rare, but potentially devastating, complication in patients who have staphylomatous sclera and eyes treated with cryotherapy is scleral rupture. In eyes that fail prophylactic therapy, retinal detachment repair by pneumatic retinopexy, scleral buckling, or vitrectomy is usually successful in the anatomic reattachment of the retina. Gross and microscopic pathology in autopsy eyes. Retinal breaks without detachment. Retinal tears and lesser lesions of the peripheral retina in autopsy eyes. Clinical study of retinal breaks. Trans Am Acad Ophthalmol Otolaryngol. Rutnin U, Schepens CL. Fundus appearance in normal eyes. Retinal breaks and other findings. Epidemiology of retinal detachment. The incidence of retinal detachment in Rochester Minnesota, â€” Hyams SW, Neumann E. Peripheral retina in myopia with particular reference to retinal breaks. Aphakic and phakic retinal detachment. Lattice degeneration of the retina. Retinal detachment due to ocular contusion. Retinal breaks caused by blunt nonperforating trauma at the point of impact. Retinal dialyses and retinal detachment. The natural history of asymptomatic retinal breaks. Natural history of retinal breaks without detachment. Management of the fellow eye. Management of the fellow eyes of patients with rhegmatogenous retinal detachment. Long-term natural history of lattice degeneration of the retina. Avulsed retinal vessels with retinal breaks. Prophylactic cryoretinopexy of retinal breaks. Results and complications in treated retinal breaks.

Long-term follow-up of treated retinal breaks.

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Chapter 3 : About JRVS : JRVS - Japanese Retina and Vitreous Society

CHAPTER STUDY. PLAY. sensory cells in the retina that are sensitive to bring light and provide color vision. AQUEOUS VITREOUS-eyeball with a hollow.

Executive Board Members shall be elected by vote of Full Members from the candidates. Rules for Election Management Committee shall be stipulated separately. The right for voting shall be given to the official member who has made full payment of membership in the election year. This right shall be effective on the day when election date is officially announced in the election year. Candidates shall be under the age 65 as of the day for official election announcement and someone who has been a member of JRVS for more than six 6 consecutive years and has sufficient experience and enthusiasm in retina and vitreous studies. Each Candidate shall also have written ten 10 or more papers for publication regarding retina and vitreous studies whether in English or Japanese. In addition, the Candidate must be the lead author of at least two 2 of those papers. Each Candidate must fill out the required items on the prescribed application form, and submit the completed form to arrive at the Secretariat no later than two 2 months prior to the election date. In the event of a tie vote for the last position, the person who holds the longest JRVS membership among the Candidates shall be elected. Revised Rules shall take effect on December 1, Committee members shall be selected from among Full Members and be delegated duties by the Executive Board Chairperson within three 3 months of the election date. The Committee shall consist of four 4 members, and the Chairperson shall be selected by mutual vote from among the committee members. The Committee must keep members informed about the procedure to run for the position of Executive Board Member, the closing date, and the election date. If any question arises regarding the quality of an Executive Board Member candidate, the Committee shall investigate the matter. The Committee shall deliver ballots, and accept and count the votes. The Committee shall be in charge of electing the Executive Board Members. Revisions to these bylaws may be made by decision at the Executive Board Meeting and General Meeting. The original paper shall be written horizontally on a word processor; if handwritten, please write in standard style. Copying and writing in pencil shall not be accepted. Fill out the required items in the title page and copyright transfer consent form, and attach them to the front cover of the manuscript. As a rule, the manuscript shall be written in order of preface, cases or methods, results, discussion, and conclusion or summary. As a rule, the manuscript must consist of no more than 8, characters and no more than six 6 charts or graphs. Each chart or graph shall be attached to the manuscript paper, one chart or graph per sheet, each with a title, and with the insertion position of the chart or graph written in red in the margin. Write down five 5 or fewer keywords before the list of reference literatures. English abstracts may be attached. If the lead author of the paper is a JRVS member, the costs for publishing up to 2 pages , photo composition, and tracing are free. Non-JRVS members shall be responsible for all costs. If you wish to have your paper published in full color, please indicate this. A fee will be charged for this. Thirty 30 printed copies of the original paper will be given to the author.

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Chapter 4 : Ocular Cytopathology: Chapter 2 Anatomy- Iris, Pigment Epithelium, Retina, Vitreous

Chapter 7 Diseases of the Eye and Adnexa Presented by: HH36 Disorders of the choroid and retina HH42 Glaucoma HH44 Disorders of vitreous body and globe.

Other disease processes that involve the retina will be covered in other chapters. Diabetic Retinopathy Diabetes is a common disease and many affected patients have vision problems. In fact, diabetics are twenty times more likely to go blind than the general population. Diabetic retinopathy is the term used to describe the retinal damage causing this visual loss. Diabetics have a high prevalence of retinopathy, and one out of every five patients with newly diagnosed diabetes will also show signs of retinopathy on exam. Mechanism of Vessel Breakdown How are the eyes affected? Basically, diabetes is a disease of blood vessels. With large amounts of glucose coursing through the circulatory system, a glycosylation reaction occurs between the sugar and the proteins that make up the vessel walls. Over time, this glycosylation promotes denaturing of collagen protein within the walls, creating capillary thickening and eventually, wall breakdown. While this process occurs throughout the entire body, the microvasculature of certain organs, such as the kidneys and eyes, are more susceptible to damage. Along these lines, a good predictor of microvasculature damage in the diabetic eye is prior evidence of renal microvasculature disease as measured by proteinurea, elevated BUN, and creatinine. Because vessel damage accumulates over time, the most accurate predictor of retinopathy is duration of diabetes. The relative control of glucose during this time is also important, and studies have shown that patients who maintain lower hemoglobin A1C levels have delayed onset and slower progression of disease. Additional risk factors include smoking, hypertension, and pregnancy. Two Types of Retinopathy It is useful to divide patients into two categories of retinopathy. This is the earliest stage of retinopathy and it progresses slowly. With worsening retinopathy and vessel damage, the retina begins to show early signs of ischemia. As vessel damage progresses, you can also see beading of the larger retinal veins and other vascular anomalies. Proliferative Retinopathy With ongoing injury to the retinal vasculature, there eventually comes a time when the vessels occlude entirely, shutting down all blood supply to areas of the retina. In response, the ischemic retina sends out chemicals that stimulate growth of new vessels. This new vessel growth is called neovascularization, and is the defining characteristic of proliferative retinopathy. Far fewer patients have proliferative retinopathy, which is fortunate as this stage can advance rapidly with half of these patients going blind within five years if left untreated. The Mechanism of Neovascularization With complete vessel occlusion, parts of the retina become starved for nourishment. The ischemic retina responds by releasing angiogenic molecules like VEGF to promote new vessel growth. These new vessels serve to bypass the clogged arteries in order to resupply the starved retina. A collateral blood supply seems like a great idea, but unfortunately there is a problem. The newly formed vessels are abnormal in both appearance and function. The new vessels are friable and prone to leaking. They also grow in the wrong place, spreading and growing along the surface of the retina. They can even grow off the retina, sprouting up into the vitreous fluid. The vitreous is mostly water, but it also contains a lattice framework of proteins that the new vessels can adhere to. With vitreous movement or contraction, these new connections pull on the retina and the traction can lead to retinal detachment. Since the vessels are also weak, any vitreous traction can break the vessels and create sudden hemorrhaging with subsequent vision loss as the eye fills with blood. Finally, the new vessels can regress and scar down, creating massive traction on the retina underneath. NVI neovascularization of the iris is an ominous sign, as the new vessels can cover the trabecular meshwork and create a sudden neovascular glaucoma. Macular Edema Despite the neovascularization phenomenon and its potential for detachments and hemorrhage, the most common cause of blindness in diabetic patients is from macular edema. This occurs when diffuse capillary and microaneurysm leakage at the macula causes the macular retina to swell with fluid. These exudates are fatty lipids that are left behind after past macular swelling subsides, similar to a dirt ring in a bathtub. Treatment of DR diabetic retinopathy Preventative medicine with tighter control of glucose is the ideal treatment, but for worsening symptoms, surgical treatment is necessary. The two main surgeries are laser treatment and vitrectomy. Laser Treatment In cases of macular edema, an argon laser can be used to seal off

leaking vessels and microaneurysm in the retina by burning them. If the leakage or microaneurysm is small and well-defined, it can be selectively sealed off. With advanced retinopathy and neovascularization, a different approach is taken. Instead of individually targeting vessels, PRP pan-retinal photocoagulation is performed. With PRP, the ophthalmologist burns thousands of spots around the peripheral retina. This destroys the ischemic retina, decreasing the angiogenic stimulus, and commonly leads to regression and even the complete disappearance of the new vessels. This treatment may seem drastic, but it has proven to be effective. Naturally, there are side effects, with peripheral vision loss and decreased night vision from the rod photoreceptor loss , but this is acceptable if the central vision is saved. Vitrectomy A vitrectomy may also need to be performed and is often done in conjunction with other surgeries. This surgery involves removing the vitreous humor from the eye and replacing it with saline. This allows removal of hemorrhaged blood, inflammatory cells, and other debris that may obscure the visual axis. While removing the vitreous, the surgeon also removes any fine strands of vitreous attached to the retina in order to relieve traction that might have, or will, cause a detachment. Conclusion As you can see, diabetic retinopathy is a big problem as a large percentage of our patients have diabetes. Retinal vessel damage leads to edema, and vessel occlusion stimulates neovascularization that can lead to trouble. Fortunately, better glucose control and surgical treatments have significantly decreased the incidence of visual loss in these patients. Retinal Detachments A retinal detachment is an abnormal separation between the sensory retina and the underlying RPE and choroid plexus. If you remember from the anatomy lecture, the outer third the part furthest from the vitreous of the retina gets its nourishment primarily from the underlying choroid â€" with a detachment, the photoreceptor layer loses its blood supply and becomes ischemic. The macular retina is especially susceptible to this damage. The prognosis for patients with retinal detachments depends upon the quickness to treatment; patients with detachments that involve the macula have much worse outcomes. Risk Factors and Epidemiology Up to six percent of the general population have retinal breaks of some kind, though most of these are benign atrophic holes. The actual incidence of retinal detachment is only 1 in every 10, people. Relative risk is equal between men and women, with higher rates in those of Jewish descent and decreased risk in black populations. When looking at patients who already have retinal detachments, you begin to see some interesting trends. Many of these patients are myopic near-sighted. Myopic eyes are physically larger and longer than normal eyes and have thinner retinas at the periphery. This thin retina is more likely to break, forming small holes and tears that may progress to a detachment. Up to 35 percent of patients with retinal detachments develop them after another eye surgery â€" typically a cataract extraction. Finally, traumatic sports such as boxing, football, and bungee-jumping predispose younger people to forming detachments. The Three Types of Detachment Retinal detachments generally occur by three different mechanisms. The most common detachment is the rhegmatogenous retinal detachment. This is an actual tear in the retina, with a full-thickness break through the retinal sensory layers. These tears can occur from trauma, surgery, or extend from preexisting retinal holes. Fluid from the vitreous chamber flows through the tear and collects in the sub-retinal space. Eventually, the retina tears away, peeling off the underlying RPE and choroid. Without treatment, a rhegmatogenous detachment can spread and eventually involve the entire retina. The second type of detachment is from traction on the retina. This is when the retina is pulled from its base. This can occur from vitreous pulling, or from diseases like diabetic retinopathy where neovascular membranes on the retinal surface contract and tug on the retina with great force. A less common mechanism for detachment is from hemorrhagic or exudative retinal detachment. This occurs when blood or fluid builds up under the retina, slowly pushing the retina upwards. This occurs with dysfunction of the RPE or choroid plexus and can be caused by ocular tumors, inflammatory diseases, or congenital abnormalities that create a breakdown of the blood-retina barrier. PVD posterior vitreous detachment One common cause of a retinal tear is secondary to a posterior vitreous separation. As we age, the vitreous liquefies and contracts in upon itself â€¦ if this occurs suddenly, the posterior vitreous face can suddenly peel off the retina. We check these patients very closely, and, assuming no tears are seen, check them again in a few weeks to insure none have developed. Symptoms With detachment, patients often report seeing flashes of light and floaters. Flashing lights, or photopsias, are often seen when a detachment first occurs. Photoreceptors are normally triggered by light, but severe mechanical disturbance can stimulate them

as well giving the sensation of light like a camera flash. Floaters look like dark specks that obscure vision, and patients say they look like a swarm of flies. They are created by objects blood cells or pigment floating in the vitreous fluid that cast shadows on the retina. While the presence of a few floaters is normal, the sudden appearance of hundred of floaters may indicate a vitreous hemorrhage. The combination of flashing lights and floaters should be considered a retinal detachment until proven otherwise. Findings The definitive way to diagnose a retinal detachment is to actually see it with the indirect ophthalmoscope. If the tear is large enough, it will be obvious as the floating retina contains blood vessels and undulates with eye movement. An ultrasound can also pick up other pathology such as tumors. This illustration shows an ultrasound of a patient with a complete retinal detachment. The retina looks like a letter V in this picture, because it is still attached at two places â€" the optic disk and at the peripheral ora seratta. Treatment Options The treatment for retinal detachment varies. The primary treatment for the majority of retinal tears and traction detachments is surgical. How fast a patient needs surgery depends upon whether the central macula has detached or not.

Chapter 5 : OKAPs Reading Schedule - American Academy of Ophthalmology

detatchment of vitreous from retina; prompt eye consultation is indicated Diplopia lesion in brainstem or cerebellum, weakness or paralysis of extraocular muscles (horizontal diplopia from palsy of CNIII or VI, or vertical diplopia from palsy of CNIII or IV).

The numerous ophthalmoscopically visible features are anomalies attributable either to structural changes, such as the floaters of syneresis and the ring-like form associated with posterior vitreous detachment Figure , or to invasive elements, such as blood, white blood cell masses, or fibrovascular proliferations from adjacent tissues. Normal vitreous in situ and many important anomalies eg, the retraction, condensation, and shrinkage of vitreous characteristic of diabetes or injury can be viewed only with a slitlamp. The slitlamp biomicroscope is a microscope with a specialized illuminating system that make transparent and near-transparent ocular fluids and tissues visible. Although slitlamp examination of the vitreous is quite easy to learn and plays an important role in the management of vitreous disease, too few ophthalmologists make optimal use of this instrument. Contact Lenses as Aid in Vitreous Examination The anterior central vitreous is the only part of the inner eye behind the lens that can be seen with the slitlamp alone. A relatively thin contact lens with a flat front surface allows stereoscopic examination of tissues on and near the visual axis of the eye-the optic disk, the posterior retina and choroid, and the axial vitreous. Much thicker contact lenses with built-in mirrors and a flat front surface allow examination of the nonaxial retina and vitreous. These special contact lenses are also used in therapeutic procedures. Fundus contact lenses with built-in mirrors are widely used in laser photocoagulation of the peripheral retina, such as in the management of retinal neovascularization due to diabetic retinopathy, retinal vein occlusion, or more rarely sickle cell anemia. The thinner contact lenses are used in ablation of macular lesions associated with diabetic retinopathy, age-related macular degeneration, and histoplasmosis. Use of special contact lenses, whether for diagnostic or therapeutic procedures, requires maximum dilation of the pupil with a combination of mydriatic and cycloplegic solutions; use of a topical anesthetic to make the patient more comfortable; and use of a clear viscous solution of methylcellulose to prevent air from entering the lens-cornea interface. B-Scan Ultrasonography B-scan ultrasonography is an important diagnostic tool used in many posterior segment problems associated with gross vitreous opacification Figure Where light-dependent ophthalmoscopes and slitlamps provide insufficient information, skillful use of B-scan ultrasonography can provide much information about the vitreous and adjacent structures. For example, it is possible to identify and locate vitreous membranes Figure , vitreoretinal relationships and retinal detachments greater than 1 mm in depth Figures , and , scleral ruptures, and intraocular foreign bodies even nonlucent plastic and glass. Vitreous hemorrhage limited to posterior vitreous region in aphakic eye. Reproduced, with permission, from Coleman DJ: Ultrasound in vitreous surgery. Trans Am Acad Ophthalmol Otolaryngol ; Total retinal detachment viewed horizontally below iris plane. A vitreous membrane connecting two leaves of retina is clearly demonstrated. Vitreous membrane extending along posterior limiting membrane of vitreous from ora to ora. Retina is in place. Vitreous membrane connecting two leaves of detached retina. The patient is aware of a localized "light," "glow," "streak of light," or "flashing" as of a neon tube in the field of vision in the absence of a corresponding light source in the environment. The patient can usually point to the area of the disturbance and often describes an arc-shaped flicker in the periphery of one or two quadrants. The light seldom persists for more than a fraction of a second. It frequently recurs at short intervals for a few minutes and then disappears for hours, days, or even weeks. It is most readily identified on moving the eye and when illumination is dim or absent. Bilateral episodes may occur simultaneously but more commonly are separated by an interval of days to many years. The light represents a cerebral awareness of the initial physical traction on and excitation of the sensory retina by abnormal vitreous. It is most commonly associated with recent collapse and detachment of the vitreous due to syneresis with focal vitreous traction on vitreoretinal lesions such as lattice degeneration, meridional folds, congenital rosettes, and other vitreoretinal adhesions. A careful history will readily distinguish the light from the scintillating scotoma of migraine, which is characterized by a symmetric quivering scotoma usually in both eyes, of predictable configuration and progression, accompanied

by variable nausea or headache. The vitreoretinal traction may require no treatment. However, as it can induce retinal tears, retinal detachment Figure , or vitreous hemorrhage, every new case requires a survey of the vitreoretinal relationship, especially in the periphery. Schematic representation of vitreous collapse causing the retina to tear and detach. The mind projects the corresponding dark form onto the appropriate area of the visual field. The term "vitreous floaters" denotes a common, potentially serious symptom that was formerly called muscae volitantes-Latin for flies that flit, flutter, or fly to and fro. The onset may be either insidious or acute and unilateral or bilateral. The patient is aware of one or more or even many fine, dark forms in the field of vision. Their configuration is usually so pronounced that the patient spontaneously classifies them as "spots," "soot," "particles," "spiders," "cobwebs," "threads," "worms," "dark streaks," "a ring," etc. Combinations are often reported. The objects continue to migrate after the eye comes to rest-hence the name "floaters. Peripheral ones are readily overlooked, as they are intermittent and require large eye motion or special positions merely to be seen. Unlike "flashing lights," they are most readily seen against bright lights or a uniform light background. They are extremely common in myopes and people with syneresis. Individual red cells are seen as small round black spots. Recent hemorrhages are often seen as black streaks or cobwebs that later break up into small round spots. White cell invasion of the vitreous gel associated with pars planitis may also cause "spots before the eyes. Vitreous floaters should never be dismissed as harmless or imaginary. A careful survey of the vitreous and retina is always indicated in order to identify the nature and origin of floaters and to decide on management. Failure to make such an examination not infrequently leads to missed diagnosis. In the absence of a serious causative pathologic process, the patient may be reassured that the condition is harmless. Unilateral cases are three times as common as bilateral cases. Hundreds of small yellow spheres consisting of calcium soaps are seen in the vitreous. These move when the eyes move but always return to their original positions because they are attached to interlacing fibers. There are no related ocular or systemic diseases. If there are enough asteroid bodies, the fundus is not viewable by ophthalmoscopy. Normal vitreous fills this cavity and remains firmly attached to the retina and pars plana near the ora serrata. All types of gels, whether vitreous or gelatin, become increasingly susceptible with the passage of time to a degenerative process known as syneresis, involving the drawing together of particles of the dispersed medium, separation of the medium, and shrinkage of the gel. Myopes are especially susceptible, even in childhood. With age, the center of the vitreous may undergo syneresis and become filled with liquid breakdown products of the degenerated gel Figure The liquid contents of the cavity can migrate into the preretinal space. The more solid, heavier vitreous gel collapses downward and forward to create a posterior vitreous detachment Figure The dynamic forces that accompany this collapse can rupture the last vestiges of the adhesions that once connected the vitreous to the disk, blood vessels, and sensory retina in childhood. Large intravitreal cavity filled with liquid breakdown products of syneresis. The patient and examiner can often see portions of the adhesions that remain attached to the collapsed vitreous as opacities. If they arise from the disk margin, the patient and examiner may note a ring-shaped opacity on the back of the vitreous. Since the front of the vitreous is attached to the globe and the back of the vitreous is collapsed in on itself, abrupt motions of the eye transmit a whip-like force to the back of the vitreous. The vitreous tends to fill out toward its normal configuration; liquid vitreous is drawn into the syneretic cavity, and the posterior separation tends to disappear Figure Liquid vitreous tends to be drawn into intravitreal cavity on abrupt eye motion left and expelled at rest right. The whip-like motions of the vitreous can give rise to photopsia by causing stimulation of the vitreoretinal juncture and may cause a characteristic floating motion of posterior vitreous opacities, or floaters. The floaters move with the eye and float to a resting position after the eye comes to rest. Since acute vitreous collapse can also cause asymptomatic retinal tears or detachment, it should be assumed that patients with new floaters or photopsia have retinal tears or detachment until proved otherwise by thorough examination of the peripheral retina with an indirect ophthalmoscope. While retinal tears can be caused by trauma, vitreous shrinkage, or proliferative vitreoretinopathy, most are caused by acute vitreous collapse. Tears following acute vitreous collapse are the result of a dynamic interaction between a focal vitreoretinal adhesion, collapsed mobile vitreous, and normal eye movement Figure Since the gel and liquid components of the collapsed vitreous are structurally relatively independent of the retina, they do not move synchronously with the retina.

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When the eye and hence the retina moves, the gel and liquid tend to lag behind the retina, and when the eye stops moving, the gel and liquid tend to continue in motion. The vitreous gel and liquid are said to exhibit inertial lag with respect to the retina. Inertial lag of the gel can cause the vitreous to tear the friable sensory retina at the point where they adhere to each other Figure The torn retina is seen to be pulled inward as a flap or a detached operculum Figure If retinal vessels are broken, they bleed briefly. A variable amount of blood accumulates in the vitreous cavity. Ophthalmoscopic view of retinal tear. Ophthalmoscopic view of retinal detachment. Some patients are not aware of the onset of retinal tears but often complain of photopsia and floaters. Some present with gross vitreous hemorrhage. Many retinal tears never lead to retinal detachment, but recent symptomatic tears, especially those with symptomatic vitreous hemorrhage, have a strong tendency to cause retinal detachment. Patients with symptoms of acute vitreous collapse or vitreous hemorrhage should therefore undergo careful examination of the retina from the optic disk to the ora serrata to rule out one or more tears. Management of tears by prophylactic laser therapy or cryopexy is relatively simple and very effective compared to the performance of silicone buckling once retinal detachment has occurred. Retinal tears are usually located anterior to the equator and are more often in the upper quadrants Figure left. Retinal detachment secondary to retinal tear formation is characterized as rhegmatogenous see Chapter 10 , particularly to differentiate it from serous retinal detachment such as that due to choroidal tumor, choroidal or scleral inflammation, or choroidal neovascularization, and from tractional retinal detachment such as that due to retinal neovascularization. Retinal neovascularization secondary to diabetic retinopathy, branch or central retinal vein occlusion, and hypertension are also frequent causes of vitreous hemorrhage. Acute collapse of the vitreous with posterior vitreous detachment will sometimes cause bleeding without tear formation. The patient often complains of floaters that suggest red blood cells, a sudden shower of small black dots, or even tiny ring-like forms with clear centers. Visual loss ranges from imperceptible to gross. The appearance of the retina and its visibility vary with the cause and amount of bleeding in the vitreous cavity see Chapter Fresh blood is red and tends to be located behind the vitreous gel or within a syneretic cavity Figure Within weeks to months, the blood tends to break down, becomes a pale color, and migrates into the gel Figure A retinal vessel ruptures due to vitreous traction.

Chapter 6 : Jaypee Brothers Medical Publishers (P) Ltd. RETINA: Medical and Surgical Manangement

I. The transparent vitreous body, or hyaloid (Fig.), is one of the most delicate connective tissues in the body. A. It occupies the posterior or largest compartment of the eye (~80% of the eye's volume), filling the globe between the internal limiting membrane of the neural retina and the posterior lens capsule.

Chapter 7 : Retina And Vitreous - ProProfs Quiz

E. sensory cells in the retina that are sensitive to bright light and provide color vision F. the watery fluid that fills the anterior and posterior chambers of the eye G. tiny structures that attach the lens of the eye to the ciliary body.

Chapter 8 : AccessLange: General Ophthalmology ; Chapter 9: Vitreous, Page 1

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Chapter 9 : Radiation retinopathy

Everything you want to know about eye allergies. Allergies affect hundreds of millions of people worldwide and ocular(eye) allergy affects approximately one in five people worldwide.