Trauma to the Posterior Segment

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Traumatic Cataracts

Usually bright white in color

Hypermature cataract is swollen in a capsular bag of fluid

Perforating injury

Residual capsular bag

Shrunken lens due to loss of lens material

Rosette cataract

Caused by pressure shock wave

Fresh has feathery clear area that may have fluid pocket nodes along the feathery extensions later it becomes more defined with sharp edges and white in cool

Unusual presentations

Subluxed lens – partial dislocation

Zonular ruptures and cataract formation

Prolapsed vitreous move past the zonules into the AC

Luxated lens – total dislocation

AC or vitreous cavity. If it effects vision, removal is likely to be warranted.

Iris

Pupil often points to the site of the injury

Slight elevation of the anterior surface may have pigment specks deposited on them
Black spot in iris will indicate the entrance wound and a cataract may be seen opposite the dark spot

Check the fundus in the region opposite the wound site for possible retinal involvement

Blunt trauma injury

Iridodialysis

Separation of the iris root from the ciliary body

“D” shaped pupil

Anterior chamber bleed is usually from the iris

Traumatic PVD

May produce a retinal bleed, retinal tear

Avulsion of vitreous base

Buckle handle or garland shape

Requires extreme blunt trauma (explosions, bungee cords, high velocity objects, etc)

Vitreous FBs

Metallic or nonmetallic

Reactive or nonreactive

Metalllic nonreactive FBs can be left inside the globe as long as there is no associated infection or chance of damaging internal structures.

Vitreous hemorrhage

Red and dispersed

Subhyloidal and intravitreal

Blood tract down retinal surface

Old blood fibrotic and white to yellow in color

Double wall perforation anterior and posterior wall involvement
Commtio retinae

Fresh – blood and edema

Old – fibrosis, reactive RPE hyperplasia, C-R atrophy

Juxta papillary trauma

Fresh well show retinal elevation on OCT scans

Old well show retinal thinning on OCT scans

Retinitis scolopteria

Injury due to high velocity object without contact with the globe. Caused by the shock wave produced by the missile.

Post traumatic epiretinal membranes

Retinal surface break leads to glial and other retinal cells migration thru the break onto the retinal surface to seal the break but it has contraction properties

Post traumatic macular holes

Concussion shock wave blow can cause tissue separation by ischemia and edema and vitreous traction

RPE hyperplasia

RPE cells proliferate and migrate. The severity of the trauma will determine the extent of the hyperplasia

Solar retinitis

Depends on the brightness of the light source and length of the exposure. Sun gazing

Retinal dialysis

Separation of the retina from the ora serrate. Usually causes a RD

Giant retinal tear

Retinal dialysis greater than 3 clock hours. Always causes a RD. RD may require extensive laserpexy, intraocular expandable gas, and silicone oil
Purtscher’s retinopathy

Extensive crushing trauma to the chest and long bones that liberates fat emboli that result in vascular occlusions

Breaks in Bruch’s membrane and choroidal ruptures

Globe expansion breaks. See in high velocity impacts or less velocity but thin scleral walls (high myopia)

Vascular insults

CRAO, CRVO, choroidal hemorrhage

Either due to rupture of the vessel walls or intense vascular contraction (spasms)

Post traumatic optic atrophy

Flat optic atrophy

Avulsion of the optic nerve

Fresh - partial or total tearing of the nerve from the optic canal with hemorrhaging and possible vitreous hemorrhaging

Old – fibrosis, RPE hyperplasia, C-R atrophy

Phthisis bulbi

Shrinkage and loss of transparency due to severe trauma

Squaring of the globe due to contraction of the recti muscles on the soft globe

Hyperplasia of the Retinal Pigment Epithelium

Clinical Description

The retinal pigment epithelium (RPE) of the retina and ciliary body is composed of a monolayer on cuboidal and columnar cells containing numerous pigment granules. They derive from the outer layer of the primitive optic cup. These cells are located between the photoreceptors and the basal lamina (Bruch’s membrane). Embryologically, the RPE has
a different tissue origin than do choroidal melanocytes, which gives rise to different acquired disease manifestations. The RPE cells have the capability of undergoing reactive proliferation and migration, and they rarely display neoplastic capabilities. The choroidal melanocytes are branching, dendritic cells interspersed throughout the uveal space and are believed to originate from the neurocrest.

Hyperplasia of the RPE is primarily caused by acquired lesions of the retina and underlying choroid. The acquired form is the result of some type of stimulus to the RPE and may denote a past episode of trauma (Figure 3-18 and 19) or post inflammatory (e.g., photocoagulation of the choroid or retina) or degenerative process that involves the retina or choroid (e.g., Toxoplasmosis gondii), but this condition has been seen in eyes with no pre-existing history of trauma, inflammation, hemorrhage, retinal detachment, and phthisis bulbi. Acquired hyperplasia of the RPE produce jet-black retinal lesions just like those seen in CHRPE, except that the lesions tend to be irregular in shape, may exhibit fibrosis or gliosis, and are not associated with depigmented rings. Because acquired RPE hyperplasia is often due to some type of post inflammatory event, there is almost always evidence of sensory retinal degeneration above it, which has a whitish appearance. Acquired hyperplasia can be found in any region of the fundus and can vary in size from very tiny to many disc diameters. Usually these pigmented lesions are stable over long periods of time. However, acquired hyperplasia may show rapid progressive growth and often signs of resolution with time.

Pigment clumping is a form of acquired hyperplasia of the RPE and aggregation of RPE cells in a small isolated area. Pigment clumping may be seen with C-R atrophy, may be produced by isolated vitreous traction on the retina and with other retinal lesions. When due to vitreous traction, the pigmentation slowly increases with time due to the continued traction by the vitreous in the involved area. Because of the time required to produce these lesions, they are most often found in adults. Pigment clumps are small areas of increased pigmentation of the retina, usually much less than one disc diameter in size. Condensed vitreous over the lesions can often be seen, particularly with scleral depression, and they represent the site of vitreoretinal traction. CHRPEs usually do not have condensed vitreous over the area of involvement. Due to the intermittent episodes of vitreous traction in various directions, these clumps are usually round with irregular margins. The area of hyperplasia may invade the overlying sensory retina and result in a slight elevation of the involved area. These lesions are generally located in the equatorial region of the retina, rarely posterior to the equator. They seem to occur in all quadrants of the fundus without predilection for a particular one. It is likely that myopes and patients with vitreoretinal disorders are more likely to acquire pigment clumping due to the increase in vitreous degeneration and subsequent traction. Sometimes, a large pigment clump may look like a round atypical lattice degeneration lesion (Table 3-10).

Another form of pigment clumping is known as clumped pigmented retinal degeneration (CPRD), which is seen as numerous pigment spots scattered throughout the retinal midperiphery, of various size ranging from barely visible to 1 disc diameter. Pigment clumps are commonly found on routine eye examinations; the prevalence rate is probably close to that of peripheral pigmentary degeneration. These lesions can be found at any age but are more likely to be detected in older adults. The pathologic findings are those of abnormal RPE cells laden with melanin granules. There is loss of photoreceptors
in areas of CPRD, which is the likely cause of functional deficit. The pigment spots do not usually show any “bone spicule” appearance. Studies show that the visual fields may be constricted, have elevated dark-adaptation thresholds, and reduced or delayed full-field ERGs. In more advanced cases the pigmentation can extend as far posterior as the temporal arcades. The incidence of CRPD is reported to be about 0.5%. People at risk are most likely those with myopia and other conditions causing vitreous degeneration. Some think that this condition may be inherited in a recessive mode.  

On fluorescein angiography, acquired hyperplasia of the RPE shows hypofluorescence throughout the dye study; areas of fibrosis and gliosis in the lesion display autofluorescence, progressive hyperfluorescence, and intense late staining.

The differential diagnosis of acquired hyperplasia of the RPE includes CHRPE, choroidal nevus, malignant melanoma, and benign and malignant tumors of the RPE.

**Histopathology**

Acquired hyperplasia of the RPE results from the benign proliferation of RPE cells, and often these cells migrate anteriorly into the sensory retina. On ocular coherence tomography (OCT), pigment clumps of proliferating RPE cells can be seen immediately under the internal limiting membrane. (Figure 3-20) The pathology of this hyperplasia depends on the type of response producing the reaction and on whether the RPE undergoes metaplasia. The possible responses are (1) simple proliferation, (2) proliferation with the formation of cuticular masses, (3) proliferation with fibrous metaplasia, (4) proliferation with calcification, (5) proliferation with ossification, (6) migration, (7) proliferation in response to demand for phagocytes, and (8) pseudoepitheliomatous hyperplasia. Pigment clumps are acquired hyperplasia and may be due to vitreous traction and, as such, demonstrate areas of condensed vitreous directly above the lesions. The reasons for the particular responses are poorly understood. Simple proliferation is a duplication of RPE cells uncomplicated by metaplasia or by the production of cuticular material.

**Clinical Significance**

Acquired hyperplasia of the RPE signifies that a stimulus to this layer was enough to cause a proliferation of the RPE cells. The most common types of stimulus are inflammation, trauma, and traction. The pigment epithelium needs time to respond in this proliferative process, which can vary depending on the strength of the stimulus. Following an episode of acute and intense stimulation, a reactive proliferation of RPE cells can be seen as a pigmented retinal lesion in 3-6 months. A longstanding, weak stimulus (e.g., mild vitreous traction) requires years to affect such a pigmented retinal lesion. Therefore, areas of retinal hyperplasia may indicate previous episodes of retinal or chorioretinal inflammation, previous episodes of ocular trauma, or sites of significant vitreous traction. It is often found associated with retinal detachments and chorioretinal inflammation.

Pigment clumping may occur as a result of vitreous traction and therefore, indicates an area of increased vitreoretinal adhesion (Figures 3-21 and 3-22). Pigment clumping becomes important when the overlying vitreous traction increases dramatically.
at these spots of vitreoretinal adhesion, such as after ocular trauma or a posterior vitreous detachment (PVD). An increase in traction may result in photopsia or a retinal tear, which is most often noted after a PVD; \cite{14, 15} therefore, pigment clumping is frequently seen next to operculated tears (sometimes on the operculum) or near the apex of a flap tear. The pigment is found next to the apex of the tear because that is the spot where the intense traction was initiated and thus, began the tearing process. Because vitreous traction also causes other phenomena, pigment clumping may have a surrounding ring of white-without-pressure. Pigment clumps in the equatorial region are more likely to produce a retinal break than those found close to the ora serrata.\cite{16} During ophthalmoscopy, the viewer should always be observant of areas of pigment clumping because retinal tears are most likely to occur there. So even if no retinal breaks are found, the clinician should locate the areas of pigment clumping on the retinal drawing before sending the patient to the retinal specialist. Patients with pigment clumping should have yearly eye examinations and given the symptoms of a retinal tear and detachment.

The visual prognosis of patients with acquired hyperplasia of the RPE varies with the cause and the extent of the condition. If the area of involvement is the optic disc or fovea, a permanent decrease in vision may result.

Occasionally RPE may assume tumorous appearances that can mimic a neoplasm.\cite{5-7, 17-22} In many of such incidences, clinically it may be difficult to differentiate an acquired neoplasm from typical RPE hyperplasia.\cite{6} On histopathological studies, even with adequate material, differentiation may be difficult.\cite{14, 20, 23}

Malignant neoplasms of the RPE are rare, with only a few well-documented cases described in the literature.\cite{9-11, 24-28} Proliferative conditions of the RPE include reactive hyperplasia, benign adenoma, and malignant adenocarcinoma. Transformation of RPE hyperplasia to acquired neoplasms is a slow progressive process that invades the sensory retina and attains a retinal blood supply, causes exudative retinopathy, retinal detachment and retinal traction.\cite{14, 23} Adenocarcinomas may be seen clinically as darkly pigmented\cite{9, 25-27} or nonpigmented.\cite{9-11, 24-28} These tumors have been found in the posterior pole,\cite{9-11, 26, 27} and anterior to the equator.\cite{24, 25} There are cases where this transformation occurred in RPE hyperplasia secondary to a photocoagulation treatment scar.\cite{14, 29} Tumors of the RPE tend to arise abruptly and perpendicular from the RPE and lack the adjacent base of pigmented choroidal tumor as is seen in most melanomas. Also, melanomas do not tend to invade the sensory retina or produce exudative retinopathy.\cite{11, 23} A fluorescein angiogram of RPE tumors tends to show hypofluorescence of the central position of the mass and a rim of hyperfluorescence (caused by the shallow subretinal fluid). Late hypofluorescence is not typical of a melanoma.\cite{14} Fine-needle biopsy is often successful in determining the diagnosis of a RPE tumor.\cite{11, 22, 28, 30} Most cases of RPE carcinomas have been misdiagnosed as choroidal melanomas. Tumors of the RPE have not been known to metastasize;\cite{23} but there is one case of extraocular extension.\cite{10} The best treatment for these growths has not been established.
Clinical Description

Chorioretinal atrophy usually appears as round, depigmented areas in the retina in which a clear view of the medium- and large-sized vessels of the choroid and melanocytes in the posterior pole. In the periphery where there are usually far fewer melanocytes, the vessels of the choroids are seen against the white background of the sclera. I refer to this as “window views” of the choroid, not to be confused with “window defects” in fluorescein angiography. Chorioretinal atrophy is believed to be the result of the occlusion of a single lobule of choroidal circulation that produces a focal postischemic atrophy of the pigment epithelium and outer layers of the sensory retina. They usually vary from 0.1 to 1.5 mm but occasionally are many disc diameters in size. Pigment accumulation may be found at the edges or in the centers of these lesions. Small lesions may coalesce into large areas with convex scalloped margins and incomplete septa. Single lesions are called chorioretinal atrophy, but when the lesions are aligned in a row parallel to the ora serrata, the condition is known as paving-stone or cobblestone degeneration (Figures 2-4, 3-65, 3-66, and 3-67). Sometimes the paving-stone lesions merge into what is termed as coalesced paving-stone and is seen as a curved band of chorioretinal atrophy. Chorioretinal atrophy occurs in congenital hypertrophy of the retinal pigment epithelium (CHRPE) and is known as lacunae. During scleral depression, chorioretinal atrophy appears flat and the overall appearance does not change upon rolling the lesions.

Paving-stone degeneration is present in 22-27% of adults and its prevalence increases markedly with age, from 10% in persons in their twenties to more than 30% of those greater than 70 years of age. There is a higher incidence of chorioretinal atrophy in myopes where it has been reported to have an incidence of 27.1% and at 40% in myopic individuals over 40 years of age. There seems to be a preference for the inferior half of the fundus, and more than half of these lesions are located between the 5 and 7 o’clock positions. They are bilateral in 38.0-41.4% of eyes and in 57% of myopic eyes. The majority are found just posterior to the ora serrata; rarely, paving-stone degeneration can occur as far posterior as the equator. It can, however, occasionally be seen in the posterior pole and pars plana. Chorioretinal atrophy may be associated with lattice retinal degeneration. Chorioretinal atrophy in the macula is one form of geographic atrophy (GA). Paving-stone degeneration is also found anterior to peripheral intraocular tumors, such as choroidal malignant melanoma and nevi, and metastatic choroidal tumors. It is believed that the association of this entity and intraocular tumors has to do with peripheral choroidal insufficiency induced by the space-occupying masses. Chorioretinal degeneration and atrophy can affect the same areas of the peripheral retina.

Histopathology

The pathology appears to result from the closure of small areas of the choriocapillaris, which produces subsequent atrophy of the overlying pigment epithelium and outer layers of the sensory retina (Figure 3-68). Ocular coherence tomography (OCT) shows the window effect from the loss of the pigment epithelial and choriocapillaris layers by allowing the light to penetrate deeper into the choroid (Figures 3-69 and 3-70). There is loss of photoreceptors and the external limiting membrane. All these retinal layers are nourished by the choriocapillaris. The inner layers of the retina are essentially intact.
because the retinal circulation supplies these layers. Thus, there is a clear window-like view of the choroid or choroid and sclera through the intact inner retinal layers. The inner surface of these lesions is slightly depressed as a result of tissue loss in the choroid and retina, but this depression is not detectable on routine ophthalmoscopy but can be seen on histopathology studies and ocular coherence tomography (OCT). The pigmented borders and septa are the result of the proliferation of pigment epithelial cells at the edges of the lesions. Other pigmented areas may be the result of pigment migration from the degenerated pigment epithelial cells into the sensory retina within the boundaries of the chorioretinal atrophy. The remaining inner retinal layers become tightly adherent to the basal lamina (Bruch’s membrane), which may show damage in its inner layers. Histopathologically, the areas of chorioretinal atrophy look similar to the scars produced by retinal cryopexy.9

Clinical Significance
Because the inner retinal layers are spared in the degenerative process, there are no breaks through which fluid can penetrate into the subretinal region to produce a retinal detachment. Therefore, these lesions do not predispose to retinal detachment. If a retinal detachment involves an area of chorioretinal atrophy, however, the tight adherence to the basal lamina may produce an additional tear in the retina at the edge of the lesion. These breaks are often small and irregular in shape, and close examination may be necessary to detect them. Often, small round chorioretinal atrophy lesions are mistaken for retinal holes because they are round with sharp margins (Table 3-6). This is especially true if a choroidal vessel is immediately beneath the lesion and gives it a red color, as is found in a retinal hole. However, on close examination, seeing a choroidal vessel in the small chorioretinal atrophy can make the differential diagnosis of a hole versus a “window view.” Occasionally, an advancing retinal detachment has been halted along a line of paving-stone degeneration.9

Vitreous Hemorrhage
The symptom of a vitreous bleed is the sudden onset of numerous tiny black floaters—the “swarm of gnats” or “black pepper specks”—that are best seen against a bright background, such as the blue sky or a white wall. They are the result of the shadow cast by RBCs close to the retinal surface. The RBCs are very small and must be positioned just above the retinal surface to cast a shadow significant enough to be detected by the photoreceptors. Because the retrovitreal space is filled with a fluid media, the RBCs can float next to the surface of the retina and therefore, be seen as numerous small floaters moving across the visual field. The patient may complain of blurry or smoky vision with a red tinge if a substantial amount of blood is found in the visual axis of the vitreous body (Figures 4-20 and 4-21). Ultrasonography can also be used to judge the extent of vitreous hemorrhage, especially if the view of the vitreous is poor (Figure 4-22). If there is a large vitreous hemorrhage, the patient may complain of very poor vision. Vitreous hemorrhage may occur as the result of trauma, a retinal tear, or from any type of hemorrhagic retinopathy (especially diabetes). One study of the records of 253 consecutive patients with newly diagnosed vitreous hemorrhage found that spontaneous vitreous hemorrhages
occurred in diabetic retinopathy (35.2%), trauma (18.3%), retinal vein occlusion (7.4%), retinal tear without detachment (7.0%), PVD (6.5%), proliferative sickle cell retinopathy (6.5%), retinal tear with a detachment (4.8%), subretinal neovascularization from age-related macular degeneration (2.2%), hypertensive retinopathy (1.7%), unknown (2.5%), and other causes (8.7%). The valsalva maneuver can result in vitreous hemorrhage.\(^1\)

The blood that spreads into the vitreous from a ruptured retinal vessel passes through channels and usually compartmentalizes in small vitreous lacuna. Therefore, it is common to see multiple blood clots in the vitreous during ophthalmoscopy or ultrasonography. Most of the blood in the vitreous settles inferiorly, where it clots and loses its red pigmentation over several weeks. The areas of clotted blood become yellowish white, round or teardrop-shaped vitreous scars with time and remain stationary in the attached formed vitreous (Figure 4-23). The clinical finding of yellowish white strands with blood clots intermixed within them is indicative of a rather recent vitreal bleed.

It may not be possible to see the fundus during a large vitreous bleed, even with the brightest power setting of the binocular indirect ophthalmoscope. Ultrasonography can be used to obtain a view of the vitreous cavity and the posterior structures and is done to determine whether a retinal tear, retinal detachment, or vitreoretinal mass is present.\(^6,7\) The patient is instructed to watch TV while sitting in a chair and to sleep with two pillows; this facilitates the gravitation of blood to the inferior vitreous cavity (Figure 4-24). The patient is told not to become involved in strenuous or rapid physical activity that may mix the settling blood higher up in the vitreous. Sometimes patients are placed on strict bed rest and bilateral eye patching to reduce head and eye movements to facilitate the gravitational effect further. The patient is instructed to return in a week for an evaluation to see if the vitreous blood has degenerated and settled enough to obtain a view of the fundus.

A pseudomembrane of the vitreous can result from the sheet-like formation of hazy vitreous gel secondary to vitreous hemorrhage. This pseudomembrane can be mistaken for a retinal detachment; unlike a retinal detachment, however, the pseudomembrane does not have blood vessels. Small, fresh blood clots in the vitreous close to the retina are bright red and can be mistaken for a retinal break, except that they tend to move a little on eye movements. These two entities are usually differentiated on careful examination by binocular indirect ophthalmoscopy with and without scleral depression or with a precorneal condensing lens or three-mirror lens.

Vitreous hemorrhages in infants is rare and has been reported to occur in birth trauma, shaken- baby syndrome, protein C deficiency, disseminated intravascular coagulation disorder, inflammation, Terson’s syndrome, retinopathy of prematurity, and *Toxocara* infection.\(^8-12\) Because the vitreous in infants is more formed and gelatinous, hemorrhages tend to remain concentrated and take longer to disperse than in adults. Vitreous hemorrhages can take as long as 2-13 weeks to dissipate in infants.\(^11,13\) A residual finding in vitreous hemorrhages in infants is the development of a pigmentary retinopathy after such a bleed.\(^8,14\)

A subretinal macular hemorrhage may be treated by causing a break in the ILM and allowing the blood to enter the vitreous cavity. A subretinal hemorrhage of great
enough volume and rapid development can break through the retina and enter the vitreous of vitreous cavity.\textsuperscript{15-21}

**Clinical Significance**

A vitreous hemorrhage can lead to vitreous gel degeneration, which is believed to be the result of the effects of the breakdown of hemoglobin from degenerating RBCs on the HA present in the vitreous. The by-products of degenerating hemoglobin are toxic to the vitreous, and the result is extensive loss of water from the vitreous gel. Lysis of red blood cells in the vitreous may result in the formation of scarring and vitreous membranes. Both these degenerative changes can produce substantial tractional forces that may result in a retinal break or detachment.\textsuperscript{22,23} Repeated vitreous hemorrhaging can produce extensive vitreous scarring, which can often result in vision restricted to hand motion and light perception. Treatment for scarred vitreous is vitrectomy with irrigation and infusion. A complication of this surgical procedure is retinal tears.

In infants the complications following a vitreous hemorrhage are amblyopia exanopsia, anisometropia, epiretinal membranes, and retinal tear and detachment.\textsuperscript{8, 10, 11, 13, 14, 24} Due to the length of time necessary for vitreous hemorrhage to disperse in infants (2-13 weeks), the lack of visual sensory input may lead to amblyopia. It is estimated that in infants, occlusion amblyopia can develop as early as 6-8 weeks after monocular occlusion.\textsuperscript{25, 26} Anisometropia following a vitreous hemorrhage is possible after 6 months of visual occlusion. One study found that occlusion effects 3 months and 6 weeks after a vitreous hemorrhage in infants resulted in a unilateral myopic shift in refraction of 7.50 and 9.25 diopters, respectively.\textsuperscript{11}

**Avulsion of Vitreous Base**

The vitreous base is a condensation of cortical vitreous that adheres firmly to the peripheral retina and pars plana on either side of the ora serrata.\textsuperscript{1} The base extends 1.5-2 mm anterior to the ora serrata, 1-3 mm posterior to the ora serrata,\textsuperscript{2} and several millimeters into the vitreous body.\textsuperscript{3} After very severe ocular contusion, the base may become disinserted from the oral region. Avulsion of the vitreous base occurs when it is torn away from its anchorage to the base area (Figure 4-25). An avulsed vitreous base appears as a whitish translucent band floating in the vitreous cavity and is frequently twisted like a garland\textsuperscript{4} (Figure 4-26) or may have a bucket-handle appearance (Figure 4-27). It may or may not be associated with a retinal dialysis or peripheral retinal breaks and is most frequently seen superonasally, which is the same region in which traumatic retinal dialyses usually occur. It commonly occurs in young people. Inner retinal layers may be stripped away and adhere to the avulsed vitreous base.

**Clinical Significance**

The existence of an avulsed vitreous base confirms prior trauma. It has no clinical significance except as a cause of symptomatic floaters. Its clinical significance is in its association with blunt trauma and therefore may be seen in conjunction with vitreous hemorrhage, retinal tears, or retinal detachment. Simultaneous retinal dialysis and avulsion of the vitreous base are found in about 25% of patients with retinal detachment.
secondary to ocular contusion. Ocular trauma that is not severe enough to produce a retinal tear or avulsion of the vitreous base may result in a tenting-up of the peripheral retina and epithelium of the pars plana. Treatment is not necessary for an avulsed vitreous base without a concomitant retinal tear or detachment.

**Retinal Dialysis**

Retinal dialysis (disinsertion) was first described by Leber in the 1882, but it was Anderson who coined the term *dialysis*, *dia* meaning “apart” and *lysis* meaning “dissolution.” A retinal dialysis is a retinal tear that occurs at the ora serrata and is concentric with the ora (Figures 5-25 and 5-26). Most tears are less than 90 degrees, and they may occur bilaterally in 4% of the cases. Often the choroidal pattern becomes more visible in the area of the dialysis due to the loss of overlying retinal tissue. If the edge of the dialysis remains close to the underlying choroid, the break may not be discovered until scleral depression is performed. The condition is often asymptomatic. In a true dialysis, the posterior border of the vitreous base coincides with the ora serrata. If the posterior border lies slightly posterior to the ora serrata, a skirt of retinal tissue remains attached to the ora (Figures 5-25 and 5-27). Both types are usually regarded as retinal ora dialyses, but some consider the latter a retinal tear instead of a true dialysis. Also, because a true retinal dialysis is thought to be caused by separation of the retina from the ora serrata, dentate processes may pull away and remain attached to the torn edge of the retina. As the vitreous contracts, the tears become more elevated in conjunction with increasing retinal detachment. The edge of the dialysis is more scalloped in appearance nasally and smoother temporally due to the anatomic structure of these regions of the ora serrata. Retinal dialyses are usually asymptomatic, unless a clinically significant retinal detachment develops.

The nontraumatic spontaneous dialysis of the young is usually located inferotemporal and is characteristically asymptomatic. The location of the nontraumatic forms suggest a developmental abnormality of the inferotemporal retina and vitreous base region and are often bilateral. With trauma as a key factor in producing such a dialysis, some cases of spontaneous may actually be posttraumatic and be affected by patient recall bias, denial, and delayed onset. The incidence of in Hispanic, Asian, and Native Americans and familial pattern of inheritance reported has led to speculation of a genetic component. Studies have shown both a autosomal dominant and autosomal recessive patterns to spontaneous dialyses. The mean age of diagnosis of a bilateral nontraumatic dialysis is reported to be 28 years of age. Most nontraumatic and traumatic dialyses are found in the second decade of life. Ocular conditions that may lead to a retinal dialysis are: peripheral micro-cystoid degeneration, retinoschisis, retinal cysts, and peripheral uveitis.

A traumatic retinal dialysis is formed by a contusion blow producing an anterior-posterior compression of the globe, which results in a sudden equatorial expansion; thus, marked traction of the vitreous base area may result in a dialysis. Traumatic retinal dialyses are more commonly found superonasal and are unilateral; however, a blow to the eye from an angle to the temporal limbus can result in an inferior, temporal dialysis. Males are more frequently affected than females, probably because males have
a higher incidence of ocular trauma. Traumatic retinal dialyses probably occur at the time of the trauma and are most likely the result of equatorial scleral stretching. In patients with a traumatic hyphema, 4-18% of the eyes had either a tear along the posterior vitreous base or a dialysis as soon as the blood absorbed, but no additional breaks were found on follow-up examinations. 27, 28

Risk factors for a retinal dialysis are family history (especially for the idiopathic form), recent or past trauma, and a more minor association with refractive error, pseudophakia, and various retinal degenerations. A retinal dialysis may be associated with proliferative vitreoretinopathy, proliferative retinal vasculopathies, or chorioretinal inflammatory disease (especially pars planitis). 29

**Histopathology**

The retina is separated at or just posterior to the ora serrata (Figure 5-28). 2, 9 The retina itself may appear normal or dysplastic. The overlying vitreous is usually normal in appearance, without liquefaction.

**Clinical Significance**

A retinal dialysis often progresses to a retinal detachment, and generally the patient is asymptomatic until the macular area becomes involved. The retinal detachment caused by the nontraumatic form is usually slowly progressive and is often associated with multiple demarcation lines. 15, 30 Males are affected more than females, and the bilaterality of the congenital this condition stands in marked contrast to traumatic retinal dialyses.

Trauma is well recognized as cause of retinal dialysis. 26, 31-33 Post-traumatic dialysis may be associated with vitreous hemorrhage (Figure 5-26) and other stigmata of eye trauma. A traumatic dialysis may not be visible initially due to the presence of retinal or vitreal hemorrhage or retinal edema. Sometimes the trauma induces a chorioretinal inflammation at the site of the dialysis. The inflammation produces a scar and prevents formation of a retinal detachment. The retinal detachment associated with a traumatic dialysis is deceptive in that it often occurs weeks to months after the trauma (> 8 months in at least half of cases). 31 It is often asymptomatic and may be associated with a disinsertion of the vitreous base (vitreous base avulsion). The vitreous usually remains attached to the torn retina and thus, the posterior edge of a dialysis does not tend to form a roll, as is commonly seen in giant retinal tears. 15 The continued vitreous traction can cause the edge of the torn retina to become very elevated. One should suspect a retinal dialysis if the retinal detachment extends onto the pars plana.

The risk of a retinal detachment from a dialysis is reported to range from 8% to 33%. 3, 8, 14, 15, 20, 34, 35-39 Retinal dialyses are found in 75-84% of traumatic retinal detachments. 14 Because vitreous detachment is not commonly associated with a dialysis or resultant detachment, photopsia is usually not a symptom in these patients, however, typical signs of a retinal detachment may be present. 14, 40 However, given that these types of detachment are often chronic, it is not unusual to have an anterior chamber reaction or to find cellular debris in the anterior vitreous, which may be inflammatory products, blood, or pigment cells (tobacco dust). 3 The detachment is slowly progressive and is often characterized by successive demarcation lines, which are found in 50% of the cases. 3, 40, 41 Studies have suggested that the time between formation of the dialysis and its
detection is often greater than one year in 40% of the cases of non presenting retinal dialysis.\textsuperscript{14} The detachments may be thin and transparent and may be confused with a retinoschisis.\textsuperscript{42} Due to the chronicity of these detachments, intraretinal cysts (macrocysts) are present in 20% of the involved eyes.\textsuperscript{3, 26, 40} Shallow macular detachments may be associated with dialysis or detachment; these may be misdiagnosed as idiopathic central choroidopathy. Intraocular pressure may be elevated in eyes with a retinal dialysis, likely secondary to anterior chamber angle recession.\textsuperscript{21} Other retinal breaks, chorioretinal atrophy, or chorioretinal scars may appear in these eyes.\textsuperscript{43}

A post-traumatic retinal dialysis that is greater than 15 degrees in length, located above the horizontal, or is found in an aphakic eye should be evaluated for treatment.\textsuperscript{44} Some authorities say that they should always be treated.\textsuperscript{5} Treatment of a retinal dialysis is scleral buckling with cryopexy.\textsuperscript{45} The treatment should extend all the way to the ora serrata to completely seal each end of the break.\textsuperscript{5} The prognosis is generally favorable for surgical reattachment\textsuperscript{3} unless a giant tear develops.

**Giant Retinal Tear**

Giant retinal tears are an extreme form of retinal dialysis involving more than 90 degrees in circumferential length. They are thought to favor the posterior margin of the vitreous base (Figure 5-29). An important feature of a giant tear is the mobility of the posterior flap, which makes surgical repair such a challenge.\textsuperscript{1} One report states that 70% are idiopathic, 20% traumatic, and 10% occur in an area of chorioretinal degeneration.\textsuperscript{2} Other reports estimate that ocular trauma is responsible for 25% of cases.\textsuperscript{3-5} It occurs four times as often in males as females and most often in young males.\textsuperscript{6} Giant retinal tears occur more often in younger patients on average than does the spontaneous form.\textsuperscript{7} Myopia, especially over 8 diopters, is a major associated finding. Since myopia and trauma are associated with giant tears, it is not surprising that keratomileusis (LASIK) may be associated with such tears.\textsuperscript{8} The risk of bilaterality of giant retinal tears is reported to be as high as 75%, and sometimes, the fellow eye has extensive lattice degeneration but often appears normal in the periphery.\textsuperscript{9} Even giant retinal tears have been the result of PVD.\textsuperscript{10} The tears are usually inferotemporal or superonasal, probably because of the greater exposure of the inferotemporal region to trauma and the effect of equatorial expansion from blunt trauma.\textsuperscript{7, 11} Tears from penetrating trauma generally tend to be superior in location. There is often a latent period from the time of the trauma to the diagnosis of the tear.\textsuperscript{7} This latent period is greater with penetrating trauma, which may result from secondary or delayed vitreous detachment associated with vitreous basal gel incarceration.\textsuperscript{12} Giant tears tend to be rapidly symptomatic. The anterior edge of the detaching retina is folded over by vitreous traction. There is extensive liquefaction and collapse of the vitreous, except for anterior adherence to the rolled edge of the torn retina. The retinal detachment frequently progresses, folding over on itself like a taco shell (Figure 5-30). A giant tear may involve 360 degrees.

Idiopathic giant tears often occur along the posterior edge of white-with-pressure. Giant tears may occur along the edge of extensive areas of lattice degeneration or along areas of the retina that have been over treated with cryotherapy or photocoagulation.
They may also occur from a PVD and can be associated with congenital colobomas of the lens and zonules.\textsuperscript{13}

**Histopathology**

The histopathology is similar to that found in retinal dialyses but more extensive (see section on Retinal Dialysis).

**Clinical Significance**

Giant retinal tears have a poor prognosis due to their tendency to increase in size from both ends. Immediate surgical intervention is indicated to limit their progression.

Management of giant retinal tears often involves of placing the patient in the prone positioning to unfold the retina. Then there is the introduction of heavy liquids to allow better manipulations of the retina to virtually assure closure of the break. Surgical correction involves vitrectomy, which is required to separate the vitreous traction from the rolled edge of the tear. Scleral buckling in conjunction with vitrectomy, internal fluid gas exchange, expanding gases, low-viscosity liquid fluorochemicals, silicone injection, sodium hyaluronate injection, trans-scleral suturing, retinal incarceration, and retinal tacks has improved the overall prognosis.\textsuperscript{3,4,14-29} The Silicone Study Group determined that silicone oil as compared to the expanding gas (SF6) to tamponade the retinal into position when complicated by proliferative vitreoretinopathy (PVR).\textsuperscript{30} Silicone oil is best removed after reattachment for optical reasons and long-term complications.\textsuperscript{31} However, the use of silicone oil can lead to the development of PVR. Macular complications that resulted in failure to regain visual acuity are maculopathy (retina or RPE changes) and epiretinal membranes.\textsuperscript{32}

Heavier-than-water vitreous substitutes can be used to unroll and flatten the retina, even in cases where PVR is present.\textsuperscript{27,33,34} Placing the patient in a prone position both operatively and postoperatively is required to unfold and maintain the retina in apposition to the pigment epithelium.\textsuperscript{35-37} Sometimes a posterior retinotomy is necessary to aspirate the subretinal fluid to enhance reattachment.\textsuperscript{3,21} Most giant retinal tears without PVR can be managed without scleral buckling.\textsuperscript{4,27,38} Proliferative vitreoretinopathy is a common postoperative complication.\textsuperscript{4} Another common postoperative complication is glaucoma (at least 32% of cases).\textsuperscript{3,7,39} Glaucoma may be due to the damage to the drainage angle or to the frequent occurrence of aphakia, with its known risk of glaucoma.\textsuperscript{31,40}

Previous successful reattachment of giant tears varied from 5% to 50%, and in cases where the tear was greater than 180 degrees, the success rate ranged from 11% to 25%.\textsuperscript{4,5,36} Newer surgical techniques are achieving higher repair rates (43-97%).\textsuperscript{3,7,18,19,22,33,41-43} The inability to unfold the detached retina accounts for half of the failures in surgical treatments.\textsuperscript{4} The success rate for reattachment and visual acuity outcome for giant tears due to penetrating trauma is less than that for blunt trauma or those of a spontaneous origin.\textsuperscript{7} One study found that 42% of patients with a giant retinal tear in one eye developed a subsequent retinal detachment in the fellow eye.\textsuperscript{5} Therefore, circumferential retinopexy to the follow eye may be considered,\textsuperscript{44} although such intensive treatment may lead to chronic iritis, cataract, fixed and dilated pupil, and macular pucker.\textsuperscript{45}