

Retinal Detachment: A Mini Review

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Abstract: Retinal detachments comprised of serious ocular conditions and can lead to permanent vision loss. When the retina, the neurosensory layer, detaches from the back of the eye, it loses its oxygen and nutrient supply leading to the death of the tissue. Immediate diagnosis and treatment are essential to avoid significant morbidity associated with this condition. In this mini review, we outline the evaluation and management of retinal detachments and highlights the role of the interprofessional team in evaluating and treating patients with this condition.

Keywords: Rhegmatogenous, Exudative, Tractional, Retinal Detachment

1. Introduction

The retina is the innermost, light-sensitive layer of the posterior eye portion [1]. It is composed of two parts - the outer retinal pigment epithelium (RPE) layer and the inner nine layers which comprise the neurosensory retina (Figure 1 and Figure 2). There is a potential space between the RPE and the neurosensory layers known as the subretinal space [2]. Retinal detachment (RD) happens when there is a separation of the neurosensory retina from the underlying RPE (Figure 3) [1].

The photoreceptors lie in the outer part of the neurosensory retina and are supplied with oxygen and nutrition by the choroid. The fovea is avascular and is supplied with its oxygen requirements entirely by the choroid [2]. Thus, macular detachment may result in permanent damage to the photoreceptors. If the macula is still on or attached, vision is potentially retained. However, if the macula is off, vision will be poor despite surgical intervention [1].

There are three various types of RD: rhegmatogenous, exudative, and tractional [1] and each of them can be managed in different ways [3, 4].

In this report, we have reviewed and summarised the different causes of retinal detachment, and the various treatment options, in particular vitrectomy, for ideal functional and anatomical outcomes.

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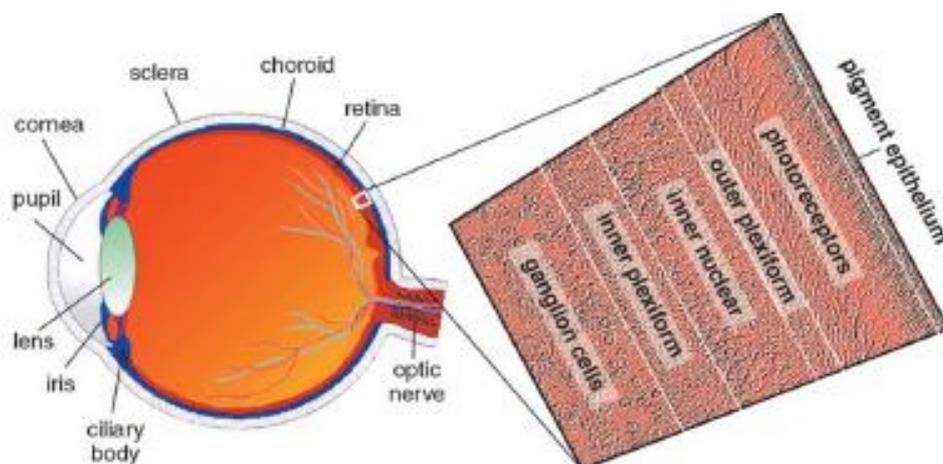


Figure 1. Illustration of eye anatomy and retinal layers [5].

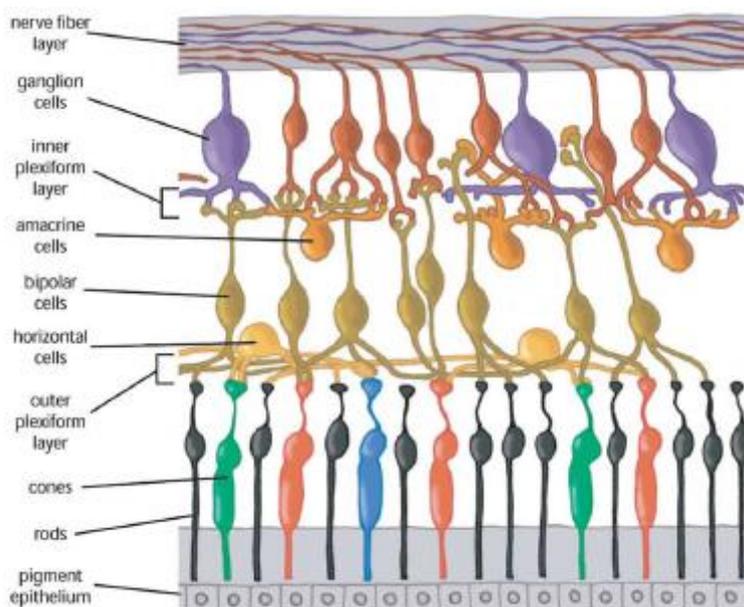


Figure 2. schematic illustration of retinal cellular layers [5].

2. Epidemiology

The incidence of RD is approximately 1 in 10000 [6]. It can occur at any age, but peaks in people between 60 to 69 years [7]. Males have a slightly higher chance of developing RRD than females [6, 8]. Southeastern Asians may have a higher risk of RRD than Caucasians as they have a higher risk of myopia [1].

Myopic patients over -3 dioptres have a 10-fold increased likelihood of developing RDD [6]. The fellow eye also has a higher risk of developing RRD, with 2% to 10% of RRDs being bilateral [6]. The incidence is also higher in patients with a family history of RD [6]. The risk increases by 1% after cataract surgery and 4% following NdYAG capsulotomy [8]. Patients with pre-existing peripheral retinal disease or degeneration, such as lattice degeneration, have a higher risk of developing RRD. All patients with lattice degeneration have a 1% chance of developing RRD. In contrast around 30% of phakic patients have a chance of developing RRD [8]. Other risk factors that may predispose RD are summarised in Table 1 [8].

3. Risk Factors

Table 1. Risk factors for various RD types.

RDD [1, 9-11]	Tractional RD [1, 12, 13]	Exudative RD [1]
Myopia	Proliferative vitreoretinopathy	Primary ocular tumours
Lattice degeneration	Proliferative diabetic retinopathy	Ocular metastases
Peripheral retinal excavations	Sickling hemoglobinopathies	Syphilis
Previous intraocular surgery	Retinal vein occlusion Trauma	Sarcoidosis
Previous RD in the other eye	Retinopathy of prematurity	Sympathetic ophthalmia
Trauma		Toxoplasmosis
Family history		Polypoidal choroidal vasculopathy
		Central serous chorioretinopathy
		Corticosteroid therapy
		Tuberculosis
		Pre-eclampsia, eclampsia
		Vogt-Koyanagi-Harada syndrome
		Acute retinal necrosis
		Organ transplantation
Optic nerve pit		
Coats disease		

4. Pathophysiology

Retinal detachment (RD) happens when there is a separation of the neurosensory retina from the underlying RPE (Figure 3) [1].

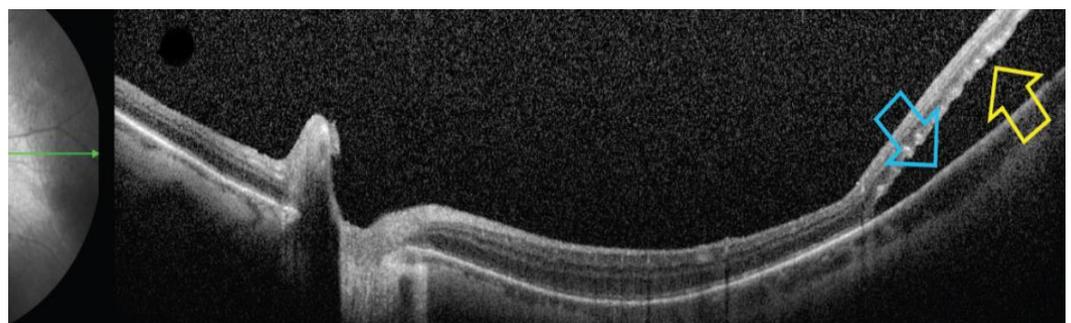


Figure 3. OCT exhibiting (a) neurosensory retina separation (yellow arrow) from the underlying RPE (blue arrow) [8].

There are three various types of RD: rhegmatogenous, exudative, and tractional [1]. Rhegmatogenous retinal detachment is the most frequent RD and is caused by retinal tears, which allow fluid passing from the vitreous cavity to the subretinal space [14]. Retinal tears are retinal breaks that form from a tear usually at the same time of posterior vitreous detachment (PVD). Atrophic holes are formed because of lattice degeneration that forms circular retinal holes. Retinal breaks can occur because of inflammation and trauma to the eye, for example, retinal dialysis which has developed as a result of blunt

trauma [6]. Progression time from tear to detachment is unforeseeable as it can vary from weeks, months, or even years [8].

Tractional RD occurs when proliferative membranes exert forces elevating the retina [1]. Exudative RDs result from fluid collection due to inflammatory disease or tumours in the subretinal space [15].

5. History

Asymptomatic RD patients are often detected during their regular eye care checks [8]. These often present as temporal or inferior chronic RDs initiating from atrophic holes [8]. Patients with a non-central RD may be asymptomatic as they may not be as sensitive, and gradually suffer from peripheral loss of vision (Figure 4) [8]. This is related more to the temporal and inferior locations as nasal and superior fields of view are less sensitive [8].

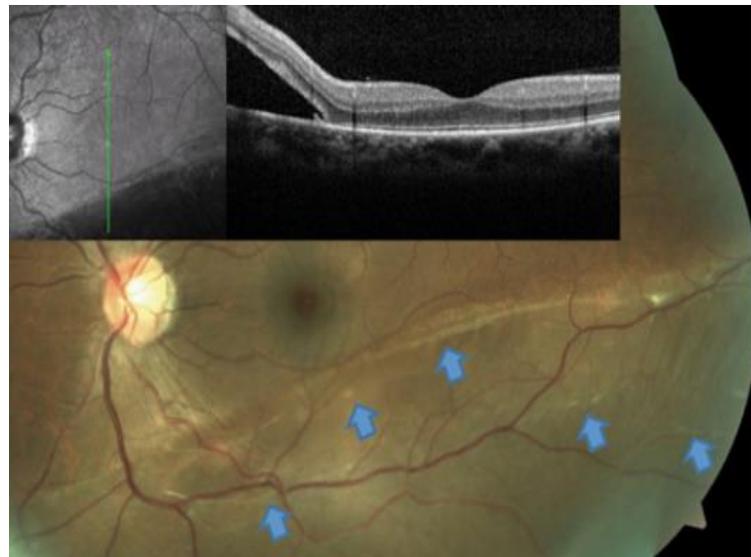


Figure 4. OCT showing RRD with partial involvement of the macula despite the patient being completely asymptomatic. Blue arrows indicate SRF and partial demarcation line, indicating the condition's chronicity [8].

RD symptoms can be nonspecific as floaters are a common issue among all age groups and are usually benign [8]. Most patients present with new-onset visual phenomenon such as floaters although the RD's onset may have occurred weeks to months before the presenting symptoms [1]. This can occur if the patient is sleeping and is unaware of the flashes or if the RD started more peripherally and took time to affect the central vision [1]. Patients with RD may also have photopsia (flashes of light) [16], a progressive, gradual loss in visual field, usually originating in the periphery and then moving centrally [1].

Patients complaining of flashes or floaters require a detailed examination as these symptoms can be associated with RD.

It is crucial to take a thorough history of the patient, including the time of the onset of the symptoms, if the symptoms are limited to one eye or both eyes, if there is central vision defect, if they have had any intraocular operation, or if they have suffered from any trauma before [1].

6. Evaluation

A thorough ocular examination should be performed for any patient with suspected RD [1]. It is recommended that both a slit-lamp and an indirect ophthalmoscope should be used to evaluate the fundus in a case of RD [17]. Visual acuity (VA) should be assessed

as patients with RD can have vision ranges from excellent to poor [1]. Relative afferent pupillary defect (RAPD) should be checked for any pupillary defect [18]. A confrontational visual field testing must be done to look for any visual field loss. Checking the intraocular pressure (IOP) is also very helpful as high IOP usually occurs with chronic RD [1]. The anterior segment (AS) should be examined for any abnormality [1].

A thorough dilated fundoscopic examination should be performed in any patient with RD (Figure 5) [1]. The retina must be extensively examined to determine the extent of the detachment and to identify all the associated retinal breaks or tears. Findings in the vitreous includes Schaffer sign (pigmentation in the anterior vitreous) or haemorrhage [1, 8]. The retinal signs associated with RD depends on the duration of the detachment [19]. An acute or fresh retinal detachment will typically have a convex shape with an opaque and corrugated appearance with loss of the choroid pattern underneath [19]. A chronic or long-standing RD may have the following retinal features thinning of the detached retina, presence of intraretinal cysts, and demarcation lines ('high water' or 'tide' marks) [19].

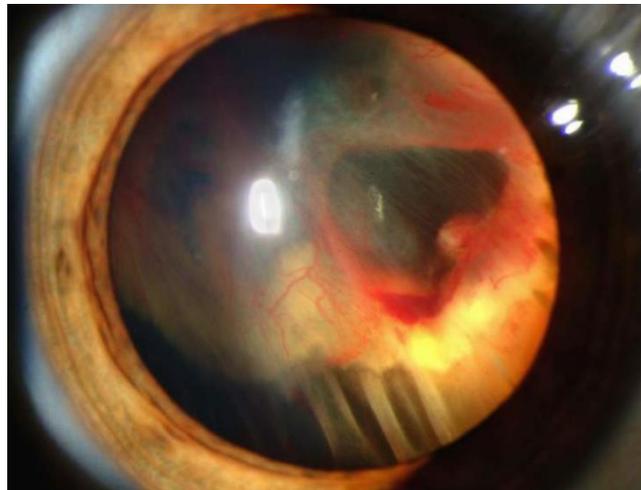


Figure 5. Slit lamp photograph showing RD [1].

RD can be quite challenging to identify, especially if located in the far periphery. To help in RD diagnosis a scleral depression as well as a three-mirror contact lens may be used to eliminate small peripheral tears [8, 20].

Linkoff's rules should be used to allocate the retinal break if the suspected detachment is RRD:

- If the RD is located superior nasally or superior temporally, the main break is located 1.5 clock hours of the highest border (98% of cases).
- In superior or total detachments that cross the 12 o'clock meridian, there are 2 likelihoods. One is that the main break is present at 12 o'clock. The other is that the break is a triangle, with the base extending from 11 to 1 o'clock and the apex pointing at the ora serrata (93% of cases).
- In inferior detachments, the higher side of RD specifies the side of the disc where the main break locates, and the break is present inferiorly (95% of cases).
- In inferior bullous detachments, the main break originates superiorly [1].

Wide-field imaging of the fundus such as Optos imaging is used for documenting the detachment and localising any areas of traction or retinal breaks. It can be used for monitoring patients postoperatively by comparing their post-op images to those pre-operatively [21].

A B-scan ultrasound is the quickest and least invasive imaging modality that can be used to diagnose retinal detachments. US can be performed by the patient's bedside and

is extremely useful to visualise the posterior segment in RD cases associated with opacities such as vitreous haemorrhage or dense cataracts [22]. US can help in distinguishing RD from PVD or choroidal detachment and aid in determining if the RD is fresh or chronic depending on the motility of the vitreous and retina [19, 22].

CT or MRI of the orbit are useful in cases of RD caused by trauma to check for any penetrating injuries and to rule out any intraocular foreign bodies. MRI should not be performed if a metallic FB is suspected [17].

Optical coherence tomography (OCT) is an effective imaging modality to diagnose RD and helps in differentiating an RD from other retinal conditions (Figure 3) [23].

Visual field testing and fluorescein angiography (FA) are not crucial in diagnosing RD, however, they can help rule out other differential diagnoses [8].

7. Differential Diagnosis

Type OF RD	DDX
<p>Rhegmatogenous RD [1, 19, 24]</p>	<ul style="list-style-type: none"> • Migraine • Posterior uveitis • Posterior vitreous detachment (PVD) • Retinal Artery Occlusion (RAO) • Retinal vein occlusion (RVO) • Retinoschisis • Choroidal detachment • Uveal effusion syndrome • Suprachoroidal haemorrhage
<p>Exudative RD [15]</p>	<ul style="list-style-type: none"> • Choroidal masses • Ciliary Body Melanoma • Coat’s Disease • Age-Related Macular Degeneration (AMD) • Retinal vasoproliferative Tumour • Vitreoretinal lymphoma • Retinal astrocytic hamartoma • HELLP syndrome • Diabetic macular edema • Hemolytic uremic syndrome • Thrombotic thrombocytopenic purpura • Familial exudative vitreoretinopathy (FEVR) • Retinal Vein Occlusion (RVO) • Paraneoplastic retinopathy • Bilateral diffuse uveal melanocytic proliferation (BDUMP) • Acute paraneoplastic polymorphous vitelliform, • Retinoschisis • Retinoblastoma • ROP • Scleritis

Tractional RD [25, 26]	<ul style="list-style-type: none"> • Retinal Vein Occlusion (RVO) • Proliferative diabetic retinopathy • Epiretinal Membrane • Eales's disease • Sickle cell Retinopathy • Traumatic or Postoperative Retinal Detachment • Retinopathy of Prematurity • FEVR • Inherited vitreoretinal degenerations
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8. Management of Rhegmatogenous RD

Timely surgical reattachment of retinal breaks is needed to treat RDD. Commonly used procedures include scleral buckling, pars plana vitrectomy, and pneumatic retinopexy or a combination of these procedures into one [3, 27, 28].

Procedure to use depends on the surgeon’s familiarity with the procedure, availability of the right equipment, lens status, the capacity for the right head position following surgery, location and size of the retinal break [29].

8.1. Pars Plana Vitrectomy (PPV)

PPV aims to reattach the retinal breaks and relieve any traction by the vitreous. Vitrectomy is indicated when media and lens opacities obscure retinal tears from being visualized or when scleral buckle is inadequate to repair the retina breaks [3, 19, 30].

8.2. Technique

Under anaesthesia, an infusion cannula is introduced into the vitreous through sclerotomy ports at 3.5mm. The vitreous base is gently peeled off to remove traction on any retinal tear and allow access to the subretinal fluid. Triamcinolone is injected into the vitreous for clear retinal tear visualization. Endodrainage is followed by direct fluid exchange. It is done by draining out subretinal fluid through the retinal tear or infusing perfluorocarbon liquid to flush out the subretinal fluid to achieve a flat retina. Retinopexy is then done by using either endolaser around the periphery of the tear to stick it to the retina and prevent retinal detachment. Fluid air exchange is done. Finally, intraocular tamponade is introduced to fill the vitreous cavity to hold the retina in place and to keep the tear closed. Sclerotomies are closed. The intraocular pressure is checked. Antibiotics and steroids are applied to the eye. The eye is taped, and an eye shield applied [3, 29, 31].

8.3. Postop Management

Facedown position allows for gas bubble press against the hole to seal it. Post-op follow up with a visual exam is scheduled at 1,7- and 14-days and 1,3,6 and 12 months. Axial length is measured at 6 months post-op visit. Silicone oil is removed 3-6 months post-op. Gas tamponade if used is taken out after two weeks [32].

8.4. Intra-operative complications

8.4.1. Suprachoroidal hemorrhage

Suprachoroidal hemorrhage is a complication of vitrectomy. The risk increases with increasing age, increased intraocular pressure, high myopia, aphakia, and so on. This occurs because of ocular hypotony during the intraoperative period. Additionally, trauma to choroid during the operation can cause bleeding [33].

8.4.2. Injury to optic nerve and retina

Injury to the optic nerve may lead to blindness. The retina can also be traumatized during the shaving of the vitreous base. Therefore, it is important to make sure the nerve is persevered with gently peeling of the vitreous [29].

8.4.3. Post-op complications

Cataract arises from damage to the lens from the gas bubble or silicone oil leading to impaired vision and surgery for removal [19, 29].

8.4.4. Raised intraocular pressure and Glaucoma

This is from gas expanding and causing pupillary block. The presence of silicone oil in the AC could lead to obstruction of trabecular meshwork thereby causing raised IOP, which may damage the optic nerve to cause blindness [19, 31].

8.4.5. Retinal tears

This occurs when epiretinal membrane contracts and pulls on the retina. Injury to the retina and contraction of cortical vitreous is another mechanism [29, 33].

8.4.6. Endophthalmitis

It may occur as a result of contamination of the surgical instruments and other equipment for the surgery leading to bacterial infection and subsequent blindness. Strict sterilization can prevent endophthalmitis [33].

8.4.7. Iatrogenic phototoxicity

This may be caused by the endoilluminator or the operating microscope. The degree of ocular lesion corresponds to the power of the wavelength used, the length of exposure, and the power of the light source. Phototoxicity can be reduced by reducing the exposure to the operating microscope light, and the intensity of the light for the endoilluminator [34].

8.4.8. Sympathetic Ophthalmia

This manifests post-surgically after about three weeks to six months. It is bilateral anterior chamber inflammation, vitritis, Dalen-Fuchs nodules, and exudative serous retinal detachment. Sympathetic ophthalmia can be treated with enucleation and immunosuppression such as prednisolone and methotrexate [35].

8.5. Scleral Buckling

8.5.1. Aim of scleral buckling

The purpose of scleral buckling (SB) is to close a retinal break by indenting the retina inwards using a buckle element so that the neuroretina and the RPE are opposed and the vitreous traction is decreased [19, 36].

8.5.2. Use of scleral buckling

- Young age,
- Phakic patients,
- Rhegmatogenous RD with no PVD and/or less vitreous liquefaction,
- RD cases with small atrophic holes, shallow detachment, or slow progressive RD's,
- Retinal dialysis cases,
- Surgeon's preference [36, 37].

8.5.3. Preoperative assessment

A detailed evaluation of the fundus with an accurate location of the retinal breaks, depicted with the use of a diagram, must be available before the surgery [38].

As scleral buckling is usually done under general anaesthesia or peribulbar blocks, the patient must be evaluated by the anaesthetic team to assess the patient's health and select the appropriate anaesthesia for the procedure [38].

The pre-op checklist must be checked including a signed consent form and make sure the appropriate patient and the appropriate eye are labelled and marked before the procedure begins.

8.5.4. Operative steps [38-40]

1. Clean and drape the eye.
2. Place lid speculum.
3. Conjunctival peritomy is performed. Either a segmental or 360-degree peritomy is done depending on the buckle element used.
4. The rectus muscles are identified and secured using sling sutures.
5. The retina is once again examined using an indirect ophthalmoscope and condensing lens with indentation to verify the location of the retinal breaks and mark their position on the sclera.
6. Retinopexy: The breaks are sealed using either trans-scleral cryopexy, laser coagulation, or diathermy.
7. The sterile buckle element is placed underneath the muscles and sutured to the sclera.
8. Confirm the placement of the buckle using indirect ophthalmoscopy.
9. Drainage of SRF and intravitreal gas or air can be done if required.
10. The conjunctiva and tenon is sutured using absorbable sutures.
11. Subconjunctival injection of antibiotic plus corticosteroid.
12. Antibiotic ointment plus eye patch.

8.5.5. Postoperative care

Patients are seen the next day to check the status of the retina and if there are any complications [41]. Patients are advised not to lift heavy objects, avoid strenuous exercise or excessive coughing, to attend all their follow-ups and to seek medical attention if they have any eye pain, redness or swelling of the eyes or deterioration in their vision [42].

8.5.6. Complications of scleral buckling

They include diplopia, cystoid macular oedema, epiretinal membrane, anterior segment ischaemia, buckle extrusion, intrusion or infection, raised IOP, angle-closure, choroidal detachment and buckle failure [19].

8.5.7. PPV vs SB

A meta-analysis by Znaor *et al* found no important difference between PPV and SB in terms of primary reattachment rate, VA improvement postoperatively, and anatomical success rate [3].

They did however notice the difference in the complications of the two procedures. They found that cataract occurred more frequently in those who underwent PPV and choroidal detachments were more common in those who underwent SB [3].

8.6. Pneumatic Retinopexy

It is an outpatient procedure done under local anaesthesia. The choice of gas depends on the size, number, and location of retina breaks, size of the eye, and features of the retinal detachment. It is preferred for superior retinal tear within one clock hour [43, 44].

8.6.1. Technique

Eye is anaesthetized and under aseptic conditions a highly expanding gas bubble such as 100% sulfur hexafluoride is injected into the vitreous cavity. The gas rises to the detached retina area to close it. This is followed by retinopexy using cryotherapy or laser to seal up the tear [44, 45].

8.6.2. Post-op Management

IOP is checked and if raised paracentesis is done, right head posture is maintained and the patient is discouraged from being in high altitudes. Patients are given topical steroids and antibiotics and cycloplegic for two days [43-45].

8.6.3. Intra-operative complications

Raised IOP occurs from gas injected or misdirected. Subconjunctival gas may arise from leakage of injected gas into the subconjunctival space. This is prevented by the correct technique of injecting gas into the vitreous cavity [27, 45].

8.6.4. Post-op complications

They include failed surgery and need for another surgical procedure; new or missed tears and RD, gas migration into the AC and subretinal space, macula hole.

8.7. Types of Tamponades and when to use them

Tamponades are agents used to raise the surface tension across retinal breaks to stop further flow of fluid into subretinal space until the retinopexy. Tamponades commonly used are gases, Silicone oil, and heavy liquids(perfluorocarbons) [27, 45-49].

Gas tamponades include air, sulfur hexafluoride (SF₆), perfluoroethane (C₂F₆) and perfluoropropane(C₃F₈) [27, 46, 47].

The choice of tamponade agent depends on each case. Determinant factors include the location and characteristics of retinal detachment, anticipated compliance of postoperative position needs, and travel plans of the patient. Silicone oil is preferred in children, mentally challenged patients, and patients who require quicker rehabilitation time. Those who do not meet these criteria may opt to use gas tamponade [27, 47].

8.8. Prognosis and treatment outcome

Without treatment, RRD results in irreversible loss of vision [50]. With treatment, the prognosis is excellent with a 90% success rate regardless of the technique used [27].

9. Management of Exudative RD

Since exudative RD can be caused by numerous underlying pathologies, the underlying cause must be treated first [17].

Medical treatments are the first-line treatments considered for a case of exudative RD and numerous therapies can be used to treat underlying inflammatory or vascular causes of ERD which include corticosteroids (oral or injection), immunomodulatory drugs, biologics, antimicrobial drugs, and anti-VEGF treatments [15].

Various interventions can also be performed depending on the etiology including focal photocoagulation, PDT, plaque brachytherapy, and cryotherapy [15].

In several circumstances/cases, PPV or drainage of SRF via retinotomy may be considered [15, 17].

10. Management of Tractional RD

The most suitable treatment for tractional RD is surgical intervention. The surgery aims to relieve the traction and identify and treat any retinal breaks or holes [17]. Vitrectomy is the preferred surgery of choice which may be combined with scleral buckling or other treatment options [17, 26].

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