**Introduction**

* The vitreous is a transparent extracellular gel consisting of colla­gen, soluble proteins, hyaluronic acid and water.
* Its total volume is approximately 4.0 ml.
* The few cells normally present in the gel are located predominantly in the cortex and include hyalocytes, astrocytes and glial cells.
* The vitreous provides structural support to the globe while allowing a clear and optically uniform path to the retina.
* Once liquefied or surgically removed it does not re-form.
* Vitreous opacities can be caused by a variety of patho­logical processes primarily involving other ocular sites; apart from vitreous haemorrhage, the conditions discussed below are those in which the vitreous gel is the primary site of pathology.

Muscae volitantes

* Muscae volitantes (Latin for ‘hovering flies’), commonly referred to as ‘floaters’, is an almost ubiquitous entoptic phenomenon of fly-, cobweb- or thread-like lesions best seen against a pale back­ground.
* It is thought to predominantly represent tiny embryologi­cal remnants in the vitreous gel.
* A sudden exacerbation can occur due to ***vitreous haemorrhage*** or, more commonly, a change in the conformation of the gel, such as a ***posterior vitreous detachment*** (see Fig. 16.22).

Vitreous haemorrhage

**Causes**

* Acute posterior vitreous detachment associated either with a retinal tear or avulsion of a peripheral vessel
* Proliferative retinopathy
* Diabetic
* Retinal vein occlusion
* Sickle cell disease
* Eales disease
* Vasculitis
* Miscellaneous retinal disorders
* Macroaneurysm
* Telangiectasia
* Capillary haemangioma
* Trauma
* Systemic
* Bleeding disorders
* Terson syndrome

**Symptoms**

* They vary according to severity.
* Mild haemor­rhage (Fig. 17.1A) causes sudden onset floaters and diffuse blurring of vision, but may not affect visual acuity
* A dense bleed (Fig. 17.1B) may result in very severe visual loss.

**Investigations**

* B-scan ultrasonography
* In unclotted vitreous haemorrhage, it generally shows a ***uniform appearance***
* Once cellular aggregates develop, ***small particulate echoes*** become visible (Fig. 17.1C);
* Ultrasonography is critical in the evaluation of eyes with dense vitreous haemorrhage to exclude an underlying retinal tear or detachment (Fig. 17.1D).

**Treatment**

* It is dictated by severity and cause, but an increasingly low threshold is being adopted for early vitrectomy (see Ch. 16) in cases of dense haemorrhage.

**Terson syndrome**

**Etiology**

* Terson syndrome refers to the combination of **intraocular** and **subarachnoid** haemorrhage secondary to ***aneurysmal rupture***, most commonly arising from *the anterior communicating artery*.
* However, intraocular haemorrhage may also occur with **subdural haematoma** and acute elevation of intracranial pressure from other causes.
* It is probable that intraocular bleeding is due to retinal venous stasis secondary to increase in cavernous sinus pressure.

**Signs**

* The haemorrhage is frequently bilateral and is typi­cally **intraretinal** and/or **preretinal** (Fig. 17.2), although occasion­ally **subhyaloid** blood may break into the vitreous.

**Management**

* Vitreous haemorrhage usually resolves spontaneously within a few months and the long-term visual prognosis is good in the majority.
* Early vitrectomy may be considered in some cases.

Asteroid hyalosis

**Etiology and Pathology**

* Asteroid hyalosis is a common degenerative process in which ***calcium pyrophosphate particles*** collect within the vitreous gel.
* An association with diabe­tes has been suggested, but is unproven.

**Prevalence**

* The prevalence of asteroid hyalosis increases with age and affects 3% of those aged 75–86 years.
* It is more common in men than in women.

**Clinical picture**

* It is seen clinically as numerous tiny round **yellow–white opacities** of varying size and density (Figs 17.3A and B). These move with the vitreous during eye movements but do **not** sediment inferiorly when the eye is immobile.
* Only one eye is affected in 75% of patients
* It rarely causes visual problems and the majority of patients are asymptomatic.

**Investigations**

* OCT (Figs 17.3C and D) and ultrasonography (Fig. 17.3E) show **high reflectivity foci**.

Synchysis scintillans

**Etiology & Pathogenesis**

* Synchysis (synchisis) scintillans occurs as a consequence of chronic vitreous haemorrhage, often in a blind eye. The condition is usually discovered when frank haemorrhage is no longer present.
* The crystals are composed of ***cholesterol*** and are derived from plasma cells or degraded products of erythrocytes, and lie either freely or engulfed within foreign body giant cells.

**Clinical picture**

* Numerous flat **golden-brown refractile particles** are seen; these tend to sediment inferiorly when the eye is immobile.
* Occasionally the anterior chamber may also be involved (Fig. 17.4).

Amyloidosis

**Pathogenesis**

* Amyloidosis is a localized or systemic condition in which there is extracellular deposition of fibrillary protein.
* Vitreous involvement typically occurs in familial amyloidosis, also characterized by polyneuropathy, prominent corneal nerves and pupillary light-near dissociation.

**Signs**

* Vitreous opacities may be unilateral or bilateral, and are initially *perivascular*.
* Later they involve *the anterior gel* and take on a characteristic **sheet-like (‘glass wool’) appearance** (Fig. 17.5A).
* The opacities may become attached to *the posterior lens* by thick footplates (Fig. 17.5B).

**Management**

* Dense opacification resulting in significant visual impairment may require vitrectomy.

Vitreous cyst

**Etiology**

Vitreous cysts can be congenital or acquired

* **Acquired cysts** are caused by a range of pathology such as trauma and inflammation. **They usually arise from** the ciliary body pigment epithelium
* **Congenital cysts** are pigmented or non-pigmented, **usually arising from** remnants of the primary hyaloid vascular system

**Signs**

* They are generally fixed – non-pigmented cysts are typically **attached** to the *optic disc* – but can be found **floating freely** in *the posterior (occa­sionally anterior) segment* (Fig. 17.6).

**Treatment**

* It is seldom required, but laser cystotomy or vitrectomy can be performed for troublesome symptoms.

Persistent fetal vasculature

* In addition to non-pigmented vitreous cysts, remnants of the hyaloid vessels can form:

1. A Bergmeister papilla, seen as a tuft at *the optic disc* (Fig. 17.7)
2. A Mittendorf dot on *the posterior lens surface*
3. The more marked manifestations for which the term persistent fetal vasculature is generally reserved, previously termed persistent hyperplastic primary vitreous (see Ch. 12).