Vitreous Disorders

Vitreous Hemorrhage

Etiology
1) Retinal neovascularization - neovascular fragile vessels (e.g., diabetic retinopathy, retinal vein occlusion, retinopathy of prematurity)
2) Posterior vitreous detachment with retinal vascular tear, retinal tears, ocular trauma
3) Subretinal bleeding with secondary extension into vitreous
4) Terson syndrome: SAH → acutely elevated ICP transmitted to retinal venules via optic nerve; 33% SAH patients have associated intracranial hemorrhage!!!
N.B. coagulation disorders/anticoagulant therapy does not cause vitreous hemorrhage!

Clinical features - large floaters, painless monocular visual loss.
* Hemorrhage tends to absorb slowly.

Complications:
1) Hemosiderosis bulbi with photoreceptor toxicity
2) Glaucoma secondary to blockade of trabecular meshwork by formed ghost cells (develop from long-standing blood cells in vitreous)
3) Severe floaters
4) Myopic shift and amblyopia in infants.

Diagnosis - black reflex on ophthalmoscopy; blood impedes complete fundus examination (H: B-scan ultrasonography).
N.B. Examine carefully to rule out possible retinal detachment!

Old Vitreous Hemorrhage: diffuse yellowish opacity through which one may get orange reflex; it consists of breakdown products of hemoglobin.

![Old Vitreous Hemorrhage](source_of_picture)

TREATMENT
- Bed rest with head elevated 30°-45° with occasional bilateral patching (to allow blood to settle inferiorly).
- Treat cause (e.g., photocoagulation of new vessels).
- Localized bleeding → photocoagulation.
- Nonclearing vitreous hemorrhage → pars plana vitrectomy.

Posterior Vitreous Detachment

- Contraction of vitreous gel → separation from retina → macroscopic opaque aggregates of vitreous fibers floating in vitreous → floaters (become less noticeable with time).
- More prevalent in highly myopic and older persons.
Posterior vitreous detachment without Weiss ring: