VITREOUS HEMORRHAGE

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Abstract: *Vitreous hemorrhage (VH) is the accumulation of blood in the vitreous chamber or in the space produced by vitreous detachment; it can be preretinal (subhyaloid) or in the entire vitreous. VH is one of the most common causes of decreased visual acuity. VH may be located in the anterior, middle or posterior vitreous or in the entire vitreous. Clinical examination is very important as VH is an ocular emergency, due to the dramatic decrease of the visual acuity that can occur sometimes, which is related to the degree of bleeding and added injuries. VH treatment includes: conservative – medical treatment; causative etiologic treatment. In posttraumatic VH, invasive surgery with YAG laser is indicated; surgical treatment - pars plana vitrectomy; treatment of complications.*

Cuvinte cheie: *decolare posterioară de vitros, neovascularizaţie retiniană, transvazare sanguină, retinopatia diabetică proliferativă, ultrasonografia B, glaucom secundar hemolitic*

Rezumat: *Hemoragia vitreană (H.V.) este acumularea de sânge în camera vitreană sau în spaţiul produs prin detaşarea vitrosului, putând fi preretiniană (subhialoidiană) sau în tot vitrosul. H.V. este una din cele mai frecvente cauze de scădere a A.V. H.V. poate fi localizată în vitrosul anterior, mediu, posterior sau în tot corpul vitros. Examenul clinic este foarte important pentru că H.V. este o urgenţă oculară, prin scăderea uneori dramatică a A.V., scăderea vederii fiind legată de gradul hemoragiei şi leziunile adăugate. Tratamentul HV cuprinde: Tratamentul conservator – medical; Tratamentul cauzal, etiologic; În H.V. posttraumatică este indicată chirurgia neinvazivă cu laser YAG; Tratamentul chirurgical – vitrectomia în pars plana; Tratamentul complicaţiilor.*

Vitreous hemorrhage can occur through: bleeding from the normal retinal vessels that break: a. during the vitreous posterior detachment with rupture of retinal vessels. b. retinal detachment, 11-44%. c. injuries. d. diabetic retinopathy. E. Terson syndrome, bleeding from the abnormal retinal vessels, secondary to the retinal neovascularisation in ischemic retinopathy or retinal microaneurysms, blood decantation in the vitreous from other bleeding sources from the anterior pole or the subretinal space: in age-related macular degeneration or traumatic choroidal rupture, the blood can invade the vitreous if there is a rupture of the Bruch membrane or of the internal neighbouring one.

The most frequent causes of VH are: vitreous posterior detachment, with or without retinal detachment 38% (according to some authors), metabolic diseases – in proliferative diabetic retinopathy 32%, VH being among the most common complications of the proliferative diabetic retinopathy that impairs vision, vascular diseases – hypertensive retinopathy, central retinal vein occlusion (11%), Coats diseases, spontaneous VH due to retinal rupture associated with vitreo-retinal degeneration, contusion or perforated ocular trauma (12%), with or without intraocular foreign body retention, acute chorioretinal inflammatory diseases, periphlebitis, vascular diseases, primary or secondary to uveitis, retinal detachment with superior retinal detachment in cases with severe VH, VH secondary to eye surgery, blood diseases: retinopathy due to anemia, leukemia, polycythemia, sickle cell retinopathy, purpura, hemophilia, cancers in which VH occurs through the vessels rupture, by acute tumour necrosis / retinoblastoma, age-related macular degeneration, retinal arterial microaneurysms, rupture, Terson syndrome, subarachnoid hemorrhage, rapid intracranial hypertension in brain injuries brings about peripapillary VH unilaterally or bilaterally, the blood from the subarachnoid hemorrhage reaches the eye through the length of the optic nerves, retinoschisis, pars plana, abused child.

Clinically, the following can be noticed: mobile, floating opacities in the vitreous (under the form of black dots, fog, smoke, shadows, spider web) when the VH is small, significant, painless decrease of vision, sometimes dramatically up to p. 1., when VH is massive, red pupillary reflex absent, eye fundus being difficult to examine because of the blood flood of the vitreous. Anamnesis should be carefully performed in order to highlight previous history of trauma, diabetes, bleeding disorders, high myopia, ocular surgery.

Clinical examination requires: determining the visual acuity in both eyes, slit lamp careful examination of both eyes, Schafer's sign, the presence of pigment in the vitreous, red blood in the vitreous, gonioscopy for iris neovascularisation or intraocular pressure, that may be normal in small VH, or increased in high VH. Direct ophthalmoscopy reveals black shadows in the red ophthalmoscopic field in small VH and the total absence of the red reflex in important VH. Indirect

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ophthalmoscopy shows the presence of blood in the vitreous cavity.

- In diffuse VH, eye fundus viewing is blurred without the possibility of identifying details.
- Total VH does not allow eye fundus highlighting.

Bleeding in the vitreous adjacent tissues, in the retrohyaloid space, Berger, Petit space can produce a characteristic meniscus that can be seen on the slit lamp.

B ultrasonography helps setting the diagnosis if VH is severe and allows: to establish the integrity of the retina in the absence of the ophthalmoscopic visualization, highlighting the site of the VH volume, the possible presence of a retinal detachment, tumours, foreign intraocular body, angiography when needed.

Regarding the evolution of vitreous hemorrhage, VH absorption from the vitreous body is a long and very slow process. The clinical course of VH depends on: location; causes; severity of bleeding. The worst prognosis is for the patients with diabetes and macular degeneration.

Complete absorption of VH without vitreous organization with total vitreous clarity may take 6-8 weeks, the blood being removed at a rate of 1% per day; if bleeding persists over 6-8 weeks, spontaneous resorption is not possible. It is possible to organize the hemorrhage in white / yellowish flanges present in the persistent or recurrent bleeding. It can cause complications such as vitreous liquefaction, vitreous degeneration. Proliferative retinitis through the organization of the remaining flanges from the VH may produce tractional retinal detachment.

Patients with VH should be monitored periodically in order to determine the hemorrhage progress into the vitreous. If the patient also has systemic diseases (diabetes, blood), these disorders should be monitored, as well. B ultrasonography should be repeated at 2 to 3 weeks in order to exclude retinal detachment or tear. If VH is repeated, it is possible to need vitrectomy. any type of anticoagulants aspirin, warfarin clopidogrel are not risk factors for causing VH and the treatment with these drugs should be discontinued after the appearance of VH if the patient requires anticoagulation therapy for other reasons (prosthetic valve).

The most serious and frequent *complications* in long lasting, recurrent VH are the following:

- Secondary glaucoma through:
  - hemolytic glaucoma in which blood hemoglobin and red cell debris can block the trabecular meshwork;
  - ghost cell glaucoma (ghost) are spherical, rigid, khaki coloured cells that can reach into the anterior chamber and through their shape and rigidity can block the trabecular meshwork.
- hemosiderosis bulbs – the iron from the hemoglobin is toxic to photoreceptors
- in people with high myopia, there is the risk for retinal tear and retinal detachment.
- proliferative vitreoretinopathy can cause retinal detachment.

VH of traumatic nature is produced by: retinal, choroid, scleral rupture, lesions of the uveal tract, retinal choroidal, scleral rupture; uveal tract lesions; head avulsion optic nerve.

VH treatment includes:

If VH is reduced and there is no retinal detachment, the treatment is ambulatory, most of the patients being treated on an outpatient basis:

a) patients with VH are daily monitored for a period of 2-5 days, in order to exclude retinal detachment or tear, then periodically, at 1 – 2 weeks for following up the VH evolution (re-absorption or recurrence),

b) patients with VH should be carefully and periodically examined, at 2-3 months,

c) at 3 – 6 months, spontaneous resorption of VH is expected,

d) in children, the follow-up period of time is reduced to 3-4 weeks for the risk of amblyopia.

The patients who are not cooperating and present VH complicated with severe hyphaema require hospitalization for careful observation.

3. If VH persists and the primary cause is not known, B ultrasound should be performed regularly.

4. Conservative treatment - medical: bed rest, head elevation to 30-45 grades, binocular dressing, all these in order to favour the leaning position of the blood and blood cells sedimentation with the partial clarification of the vitreous, allowing the eye fundus examination in the upper part, there where retinal tear is most frequently encountered.

5. Causative etiologic treatment: indirect ophthalmoscopy shows the presence and the appearance of VH, as well as the lesions that bring about the occurrence of VH (if eye fundus is visible) retinal tear, proliferative retinal phlebitis.

If retina is accessible to the ophthalmoscopic examination, the treatment will be etiologic.

6. In posttraumatic VH, noninvasive surgery with YAG laser is indicated, which could reduce the duration of treatment in the traumatic VH. In proliferating diabetic retinopathy with retinal neo-vascularization (NVR), laser panphotocoagulation is carried out if possible through the residual bleeding in order to determine the regression of NVR: Krypton lasers cross the bleeding in a better way than the Argon laser and alternatively, intravitreal anti-VEGF agents in order to induce the regression of NVR until photocoagulation is possible.

Retinal tears are treated by cryotherapy or laser photocoagulation (which can shut the compromised vessels around the retinal breaks).

7. Surgery treatment - pars plana vitrectomy to clear the vitreous which is carried out 3 months later if VH has not been absorbed.

8. Vitrectomy is indicated in: VH that is not reabsorbed spontaneously in 2-3 months, recent VH less than 2-3 months in the patients with juvenile diabetes, infant with amblyopia risk, bilateral VH with retinal detachment and tear.

Emergency vitrectomy is carried out in the following cases:

- associated retinal detachment
- iris neovascularisation and / or in angle
- at one month in diabetes mellitus type I, subhyaloid haemorrhage
- at 2 – 3 months in diabetes mellitus type II, other causes

Table no. 1. Vitrectomy performance according to the type of disease

VITRECTOMY	
Retinal detachment	emergency
N.V.I.	emergency
Diabetes type I	1 month
Subhyaloid hemorrhage	1 month
Diabetes type II	2 – 3 months
Other causes	3 months and more

Vitrectomy is indicated in VH when VH is not spontaneously remitted/ within 6 weeks up 3 months.

Emergency vitrectomy is also indicated in bilateral VH or when ultrasound highlights a retinal tear through rhegmatogenous retinal detachment or tractional retinal detachment that endangers the macula.

If vitrectomy is indicated: pars plana vitrectomy will be accomplished through which the vitreous haemorrhage

excision is performed (initially, in the centre, posterior pole, anterior pole towards the vitreous base), hyaloid release from the flanges, epiretinal membrane excision, preretinal fibrosis, retinal reattachment if it is detached, followed by laser photocoagulation or applications of cryotherapy, if necessary, intraocular air, gas, silicone, oil tamponade. Early vitrectomy, in less than a few weeks is questionable although bleeding can handle spontaneously.

The advantages of the early vitrectomy are the following:

- Immediate visual rehabilitation,
- Treatment of retinal tear prior to the occurrence of retinal detachment,
- Prevention of complications related to bleeding,
- Recurrence of proliferative vitreoretinopathy, the most frequent cause of failure in the anatomic retinal reinsertion.

Treatment indications according to VH cause:

Proliferative diabetic retinopathy complicated with VH.

In case eye fundus can be examined:

- It may be retinal detachment requiring vitrectomy,
- It is not retinal detachment. The patient will be followed-up clinically. Diabetes and hypertension will be balanced; when needed photocoagulation with YAG laser will be accomplished.

If eye fundus is inaccessible, B ultrasound should be performed highlighting the gravity factors requiring vitrectomy:

- VH dense at monophthalmus,
- Rubeosis iridis,
- Neovascular glaucoma.

Proliferative diabetic retinopathy in an adelf eye, VH recurrence requires clinical and ultrasound weekly systematic check up if:

- Eye fundus is not visible, vitrectomy is necessary,
- Eye fundus is visible, YAG laser treatment can be indicated.

If there are not gravity factors for VH, the clinical and ultrasound check-up will be made monthly and when needed, in all cases when the eye fundus is visible, laser treatment will be performed.

Neodiabetic VH requires clinical examination: with the research of rubeosis iridis and intraocular pressure, eye fundus examination and the examination of the contralateral eye.

If the eye fundus is visible, the following can be highlighted: retinal tear that requires laser treatment or cryotherapy, DPV with isolated VH, sickle cell disease, thalassemia, Eales disease, vasculitis, carotid insufficiency, macro-aneurysms, age-related macular degeneration, tumours, hemopathies, retinal angiomatosis, infectious etiology. This type of VH can resolve and will require the supervision of the resorption for a period of 1-2 months, clinically and by ultrasound. Symptomatically, bed rest and fluid intake are recommended.

If eye fundus is not visible, B ultrasound will be performed that can highlight: the absence of retinal detachment, case in which symptomatic measures will be taken, such as rest and fluid intake, the presence of retinal detachment requiring surgical treatment.

Treatment of complications: For the risk of bleeding, capillaroprotectors, hemostatic agents will be used. It is important to check up and balance the blood pressure, diabetes, intraocular pressure through topic instillations and systemic therapy. Enzymatic treatment through intravitreal hyaluronidase injections has been administered in the patients with VH and diabetes mellitus with beneficial effects. Patients with hypertension and diabetes were treated with an antioxidant – histochrome, which proved to be effective in recent VH in the patients with nonproliferative diabetic retinopathy. Retinal circumferential cryocoagulation can be a useful treatment in diabetic VH by favouring of the reabsorbtion and decrease of vitrectomy rates.

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