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Decrease of Vision in Senescent

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ABSTRACT

Vision loss in the elderly is caused by: physiological aging of the eye, eye disease specific in the elderly (cataract, DMLV, NO impairments), non-age specific eye disease similar to adult disease and multiple general pathology determination given by aging. Vision in the elderly declines over time, through an inevitable evolutionary process, aggravated by the involution of senescence. Ocular change with age affected: cornea, the trabecular meshwork lens, retina, choroid, vitreous, sclera and NO. Vision loss in elderly patients is often chronic, binocular, asymmetric. Presbyopia represents the physiological disorder of the eye, with on onset around the age of 45 which is manifested by the progressive decrease of the accommodative amplitude in near vision (the accommodation deficit by emmetrope is +0,5D at 45 years old, and 70 years the accommodation power of the lens is 0, needs +3,5 +4D for near vision.

Acute vision loss unilateral in amaurosis fugax, vitreous hemorrhage, AMD, ACR, VCR obst DRR, bilateral in: trachoma, AMD, diabetic hypertensive, retinopathy, infiltrative optic, neuropathy, cortical infarction, hemorrhage. Chronic vision loss in elderly unilateral in: cataract, DMLV, RD, keratopathies, uveitis, DR, glaucoma, trauma macular disease, affects of NO, and bilateral in: GPUD decompensated, secondary glaucoma, maculopathy, papillary edema in HIC. Decreased vision in age related eye pathology in: cataract (senile, pathological, complicated), glaucoma (GPUD, GSUD), DMLV atrophic, exudative), pathological change of NO (NOIA), artetitic (Horton's disease), nonarteritic. The general cause of AV decrease cause: systemic diseases: DM, HTN, ASC, stroke, mental illnesses (depression, dementia).

The management of vision loss must begin with the earliest possible diagnosis of the eye disease and the establishment of preventive and curative therapeutic opportunities – each age decade being evolutionary and nonregressive.

Keywords

Vision loss (acute, chronic, uni/bilateral), Decrease of vision, Presbyopia, Refractive error cataract (age related, pathological, complicated), GPUD, GSUD, AMD, Dry eye, Optic nerve damage (NO).

Introduction

- The increase in life expectancy causes an increase in the number of people over 65 years of age, which represents around 20% of the total population [1].
- Elderly people accuse the progressive decrease of vision with the passage of years.
- The progressive decline of vision in elderly people changes the quality of life for millions of people, especially through: cataracts, glaucoma, macular degeneration, diabetic retinopathy, hypertensive retinopathy, hypercholesterolemia, dry eye.
- Vision loss in the elderly is caused by: [2]
  - physiological aging of the eye
  - eye diseases specific to the elderly - cataracts, DMLV, NO impairments
  - non-age-specific eye diseases, similar to adult diseases
  - multiple general pathological determinations given by aging
- Vision in the elderly declines over time, through an inevitable evolutionary process, aggravated by the involution of senescence.

- Eye diseases with progressive evolution in the elderly cause vision loss and variable visual field amputations, many of which are detected late (when visual function and CV are significantly affected), when the ability to perform daily activities such as reading, writing, watching TV, professional work (if any), driving a car.
- The progressive decrease in vision (sometimes alarming) parallel to the decrease in physical performance, increases the risk of the elderly patient losing independence and amplifies cognitive impairment [3].
- Visual deficit must be identified early and treated appropriately to reduce morbidity through related eye and general conditions.
- The management of vision loss must begin with the earliest possible diagnosis of the eye disease and the establishment of preventive and curative therapeutic opportunities - each age decade being evolutionary and non-regressive.

Evolutionary Ocular Changes with Age [4-6]

- ***CORNEA***
  - The senile arc with lipid deposits near the limbus in the periphery of the cornea.
  - Thinning of the Bowman's membrane, with calcium deposits in the periphery of the Bowman's layer.
  - Reducing the density of stromal keratocytes.
  - Thinning of Descemet's membrane.
  - Uneven decrease in the density of corneal endothelial cells from 5000 cells/mm2 at birth to 3000 cells/mm2 in the elderly, with an increase in the density of paracentral and peripheral corneal endothelial cells.

- ***TRABECULAR MESHWORK***
  - the trabecular network thins progressively and changes its structure parallel to the decrease in cellularity
  - age-related trabecular changes reduce the humor's outflow facility
  - watery.

- ***LENS***
  - The shape of the lens changes with age
  - the lens increases in volume from 90 mg at birth to 240 mg at 80 years
  - the age-related cataract sets in, manifested by a change in color of the crystalline lens as a result of the oxidation of the crystalline proteins
  - the most frequent cataractogenic changes are located equatorial and posterior cortical
    - the proliferation and posterior migration of cells of the crystalline epithelium
    - cells Wedd - which give rise to posterior subcapsular cataract
    - liquefaction of the cortex.

- ***RETINA***
  - diffuse thinning of the internal limiting membrane of the retina
  - diminution of neural elements with gliosis in the peripheral retina
  - disorganization at the serrata ora
  - reduction of the nuclear layer
  - involutional lesions in the fiber layer of the optic nerve, NO head, NO with intracellular accumulations macular decline in scotopic, with cystoid degeneration
  - changes in retinal vessels with age
    - decreased cellularity in peripheral capillaries
    - decreasing the number of capillaries around the fovea
    - arteriosclerotic vascular changes with fibrinoid hyperplasia and necrosis
  - peripheral retinal degenerations with peripheral cystoid reticular degeneration, paving stone (cobblestone) and lattice (reticular) degeneration
  - EPR atrophy through retinal degenerative lesions and choroidal vascular insufficiency, with the appearance of nodular drusen under the EPR (excrescences of Bruch's membrane containing granular substances, lipids, proteins, calcium deposits)
  - with age, Bruch's membrane thins focally or diffusely and can be calcified.

- ***CHOROID***
  - OCT sometimes highlights the thinning of the choriocapillaris layer; other times, in DMLV, its increase.

- ***VITREOUS***
  - over time, posterior vitreous detachment may occur, the space between the retina and the detached vitreous being occupied by liquefied vitreous.
  - contraction of the vitreous base by posterior vitreous detachment (DPV) can produce traction in the periphery of the retina and sometimes retinal tears.

- ***SCLERA***
  - the sclera may become stiffer with relative dehydration especially anteriorly, at rectus muscle insertion (possibly calcified plaques).

- ***NO***
  - supporting tissue in the fibrovascular pial septum becomes more abundant and is associated with cellular and extracellular accumulation in the meninges and NO fibers
  - NO degeneration by chronic vascular occlusive disease.

Vision Loss in Elderly Patients is Often Chronic, Binocular, Asymmetric

- The history of general or ocular disease is essential for the identification and eventual treatment of vision loss (if possible) in the elderly patient (even in patients with profound cognitive impairment)
- The AV measurement must be done for near and distance with each eye and if necessary, with pinhole point
- the CV examination by confrontation can highlight

campimetric defects that direct the eye diagnosis towards an evolving disease: cataract, GPUD, DMLV, RD or identify a hemianopsia secondary to AVC

- In elderly people there is often combined pathology - cataract, DMLV, progressive GPUD and the primary cause of AV decrease or the different proportion influencing the decrease of vision of each eye disease must be identified.
- Loss of sight in elderly people favors the accentuation and acceleration of other comorbidities pre-existing the loss (or loss) of sight and can produce isolation, depression, anxiety.
- The loss of sight in the elderly changes the quality of life, limiting the patient's ability to participate in an active life in daily activities, limits physical and mental comfort, walks, leaving the house and over time immobilizes the patient in bed or in a chair, reducing to a vegetative life.
- Elderly patients with low AV have chronic cardiovascular disease, hypertension, hearing loss, fall fractures, stroke that accentuate visual difficulties.
- Various conditions can interact, they can have a chronic evolution, and various clinical manifestations (some asymptomatic) that add up to the involution process emphasizing:
  - the worsening of the functional deficit through the involution of senescence
  - decreased immune response
  - the presence of complex decompensated pathologies
  - adverse drug reactions through the clinical need to treat multiple conditions

- **Ocular aging is influenced by: [1,2]**
  - external factors: UV radiation, nutritional factors, smoking, stress
  - internal factors represented by metabolic biochemical processes accompanied by necrosis and apoptosis
    - necrosis and apoptosis of retinal ganglion cells and optic fibers secondary to ischemic cerebrovascular damage present in patients in age
      - ischemia and acute hypoxia, generate cell necrosis
      - chronic ischemia generates apoptosis with a process of slow, silent cell death, associated with progressive involution of cellular structures
  - capillary ischemia reduces oxygen and glucose in cells with the appearance of lactic acid and membrane depolarization followed by the accumulation of free radicals: superoxide anion O2, hydrogen peroxide H2O2, hydroxyl radical - OH
    - free radicals are very toxic for phospholipids in the cell membrane, proteins (nucleic acid) and produce cell necrosis or apoptosis
    - this whole assembly produces apoptosis at the level of ganglion cells of the retina and NO
  - enzymes such as: superoxide dismutase, catalase and peroxidase capture free radicals and reduce their negative effect on cells.

Vitamins (From Food or Administered Therapeutically), Vitamin E, Vitamin C, Provitamin A Would Have a Beneficial Effect in The Physiological Aging Process

  - with age, changes occur in the eye, which can be considered a slow process of ocular aging.
    - with advancing age, the crystalline lens progressively loses its transparency with the appearance of crystalline opacities and cataracts
    - at the level of the retina, the vascular endothelium of the capillaries and the nerve cells are subject to the action of free radicals; ischemia is established which favors DMLV
    - NO is sensitive to ischemia and optic disc atrophy can occur
  - currently, life expectancy has increased for the entire population (by about 5-6 years): at women at 82 years and men at 74 years.
- Many parameters related to AV are affected with age
  - vision is significantly reduced in mesopic conditions or in excessive lighting
  - contrast sensitivity and color vision decrease with age, especially for yellow and blue
  - there are difficulties in adapting to changes in light level.
- In physiological conditions, with advancing age, it occurs [10]
  - decrease in the differential sensitivity of the retina
  - decreased visual recovery after glare
  - progressive sclerosis of the lens
  - retinal senescence with ischemia and ganglion cell degradation
  - senile miosis
- AV quality is modified by senescence phenomena located at the pupil level
  - reduction of pupillary diameter
    - dark = 20 years - 7 mm
      = 80 years - 5 mm
    - light = 20 years - 4 mm
      = 80 years - 3 mm

Senile Miosis Reduces Retinal Illumination by 35%

  - diminution of the photomotor reflex
- AV evaluation and appropriate optical correction should be correlated with:
  - AV measurement for near and far
  - CV by confrontation
  - TIO
  - motility (many elderly patients have or have had a stroke)
  - external examination of the eyeballs and accessory structures, slit-lamp exam
  - FO exam - Retina, macula, NO

PRESBYOPIA [4,7-9]

The physiological decrease in visual function with age defines presbyopia and is the consequence of the decrease in the accommodative power of the crystalline lens so that presbyopia is the first sign of physiological aging of the eye

- AV at 50 is 10/10
- 70 years - 7/10
- 85 years - 5/10

In parallel with the decrease in AV, in the physiological aging process of the eye, changes occur in the pupil, lens, retina.

- It represents the physiological disorder of the eye with an onset around the age of 45, which is manifested by the progressive decrease of the accommodative amplitude in near vision
  - the accommodative amplitude decreases after 45 years due to the normal aging of the lens, which slowly loses its elasticity; this is also associated with slow sclerosis of the ciliary body
  - we read and write at 25-30 cm, so we need +3.5 +4D for reading, but the lens at 45 offers an accommodative power of +3+3.5D.
  - so, to see clearly at 30 cm (close up) at 45 years old, we have to remove the book, or optically correct the accommodation deficit by +0.5D
  - at 70 the accommodation power of the lens is 0, so an emmetrope needs +3.5 +4D near vision at 70 =>
    - in myopia, presbyopia sets in later and is added algebraically to the value of myopia; the patient with myopia of -4D at the age of 70 takes off the close-up glasses
    - in hypermetropia - presbyopia sets in earlier and adds algebraically to the value of hypermetropia; patient with hypermetropia of +2 dsf, requires glasses of almost +6 dsf at the age of 70
- **Uncorrected presbyopia** affects many individuals, especially in rural areas
  - the functional impact of uncorrected presbyopia produces [11]
    - difficulty reading
    - not recognizing small objects
    - limits visual comfort
  - the lack of optical correction in the individual with presbyopia can be caused by:
    - economic causes
    - the impossibility or difficulty of movement of elderly people due to their biological condition
    - too long a distance to a specialized service

Uncorrected presbyopia in patients with refractive error [12,13]

- after 45 years, in the presbyopic hypermetropic patient, the optical value of the near lens increases and it is necessary to supplement the optical correction with the value of the hypermetropia at a distance
- the myopic eye requires the algebraic subtraction of the optical correction by the amount of presbyopia
- astigmatic eye - the astigmatic axis remains in the same dioptric value and the same meridian, but adds algebraically the value of the spherical refractive error if it exists and the presbyopic value by adding hypermetropia and subtracting myopia.

- Refractive errors due to insufficient correction are possible in elderly people, due to lack of address, ignorance, diminished physical and mental status
  - improving near and far vision (reading, TV) has favorable effects on the elderly patient, improving daily life, degree of independence, desire to socialize
  - the insufficient correction will be brought as close as possible to a correction adapted to the age (therefore to presbyopia) and the refractive error through corrective lenses for near and distance
  - optical correction will be done with aerial lenses with two pairs of glasses, for close and distance, with bifocal lenses or progressive lenses depending on the patient's desire and his general and local condition, as well as financial possibilities.

Refractive Errors Are Possible in Eye and General Diseases in the Elderly Patient, In Evolutionary Cataracts, or in Diabetes, and it is Necessary to Recognize and Treat These Conditions

- the treatment of eye and general conditions in the geriatric patient is necessary because a worsening of the decrease (loss) of vision limits a normal and free life of the elderly patient.

- In order to ensure the most accurate optical correction of refractive errors, it is necessary to systematically examine the refraction as well as the clinical eye status under the biomicroscope and ophthalmoscope.
- Optical correction of refractive errors in elderly people is done after careful, individual general and ocular exploration; it is a prolonged process, which requires patience and attention from the examiner, but also from the patient, but the social benefit regarding eye rehabilitation through adequate correction is extremely important, especially for dependent people.
- Uncorrected refractive errors accentuate near and distance vision loss.
  - Bilateral AV decrease has much greater negative effects on the quality of life in elderly people; patients no longer participate in daily activities and over time they isolated
  - The family and the community of which the assisted patients are a part have the obligation to detect the cause of the isolation and help the patient with the appointment for an ophthalmological check-up
  - The lack or decrease of AV accentuates emotional stress, depression, can favor fractures due to falls and finally even death, especially in the case of a fall bilateral AV.

Uni- Or Bilateral Decrease in Vision Due to Uncorrected Refractive Errors Can Increase Over Time, It Can Be Perceived or Not by The Patient and Family, but it Can Produce Social Limitation, Emotional Difficulties and Even Social Dependence

VISION LOSS [9,14,15]

Acute Vision Loss

Unilateral

- amaurosis fugax

- vitreous hemorrhage
- AMD
- ACR, VCR obstruction
- DRR
- ischemic optic neuritis

Bilateral
- trachoma
- AMD
- diabetic, hypertensive retinopathy
- infiltrative optic neuropathy
- papillary drusen
- cortical: infarction, hemorrhage

Chronic Vision Loss in the Elderly

Unilateral
- cataract, DMLV, RD can evolve initially and unilaterally
- secondary to keratopathy, vitreous opacities, EMC
- uveitis with posterior synechiae and keratic precipitates
- asteroid hyalosis
- synchysis scintillans
- an old DR with compromised macular function
- compressive lesions in sphenoid wing meningioma with optic atrophy
- confirmed asymmetric glaucoma
- pseudoexfoliation syndrome
- traumatic etiology
- macular diseases: DMLV
- affects of NO: unilateral optic atrophy in space-replacing lesions: aneurysm, meningioma, NO head drusen, optic neuropathy

Bilateral
- cataract: senile
- an advanced, decompensated GPUD
- secondary glaucoma: pigmentary glaucoma, by pseudoexfoliation, induced by the crystalline lens
- maculopathies: DZ, DMLV, pigmentary maculopathy (chloroquine, thioridazine, tamoxifen)
- primary optic atrophy: compressive, toxic, Foster-Kennedy syndrome
- bilateral chronic papillary edema in HIC
- a benign HIC

DIPLOPIA ASSOCIATED WITH DECREASED VISION
- refractive errors with early monocular diplopia in cataract that can give polyopia (multiple images) depending on illumination [5,7,16]
- chalazion by compression on the eyelids
- horizontal diplopia: paralysis NC VI, brain diseases: (internuclear ophthalmoplegia)
- vertical diplopia: paralysis NC IV, Parinaud syndrome
- horizontal diplopia: NC III paralysis

TRANSIENT VISION LOSS
- Transient loss of vision - amaurosis fugace - can be benign or can be caused by a severe disease [4,13,17].
- It is clinically manifested by transient episodes of decreased monocular vision, lasting 1-5 minutes, produced by embolus or retinal arterial circulatory insufficiency (or prolonged), in HTA, migraine, blood diseases with recurrent attacks
  - bilateral amaurosis fugitive may be present in homonymous disorders of occipital ischemia, visual migraine (cephalalgic migraine)
- Risk factors:
  - HTA, DM, hematological diseases
  - vascular diseases in the elderly
  - myocardial diseases
  - aortic or mitral valve diseases, arrhythmias in intermittent atrial fibrillation
  - carotid diseases stroke
- Treatment - prevention of further episodes and prevention of cerebral ischemic attack.

Decreased Vision in Age-Related Eye Pathology
- A percentage of 18% of patients over 70 have vision disorders, but their absolute number can be much higher (due to lack of addressability, physical and/or mental degradation, etc.) [2-4,10,18]
  - ocular and general pathology increases with age when ocular aging processes occur: cataracts, AMD, glaucoma, but also general - ASC, DM, HTA
  - variable decreases in AV, undiagnosed or neglected refractive errors may occur, as elderly patients with difficulty moving or with general disorders and sometimes serious mental disorders.
- Both the elderly and the family must recognize and be aware physically, socially, mentally, of the eye disease.

CATARACT
- The clear lens provides a third of the eye's refractive power.

Cataract is the opacification of the lens that can appear in the nucleus, cortex or/and both, with evolution towards total cataract, with a higher frequency in the female sex.
- The evolving cataract is accompanied by progressive vision loss [10,18].
  - the population over 80 has cataracts in 50% of cases, cataracts being an important cause of decrease, even loss of vision in the elderly.
  - cataracts require surgical treatment (cataract surgery is the most common eye surgery).
- Cataract prevention (minimum possible) requires reducing risk factors with:
  - limiting UV exposure by using protective lenses in the elderly
  - exclusion of smoking, alcohol
  - antioxidants
- Cataract is manifested by:
  - progressive, painless loss of vision
  - loss of sight
  - decrease in color brightness

- decreased night vision
- sometimes monocular diplopia

Age-related cataract - senile cataract [11,12,14]

- has slow evolution - several years
- it is bilateral, asymmetric
- it is more frequently corticonuclear, being able to take a purely nuclear form, cortical, posterior subcapsular

General **pathological cataract** represents opacification of the lens secondary to a cause general (bilateral) [**12,14]**

- ***Cataract and diabetes***
  - senile cataract is 6 times more common in patients with diabetes and is accompanied by diabetic retinopathy that conditions the postoperative visual functional result.
  - cataracts in diabetics appear earlier around 60 years
- ***Cortisone cataract***
  - posterior subcapsular crystalline opacities present in people under local cortisone treatment, general.
  - there is an individual susceptibility to cataract formation under cortisone treatment
  - cortisone cataract in elderly people is located posterior subcapsular, develops after a latency period and can have a slow evolution
  - is amenable to surgical treatment
  - any long-term corticosteroid therapy requires ophthalmological supervision of the lens

Complicated Cataract

- it is the opacification of the lens secondary to an eye pathology, frequently in eye diseases with chronic evolution
- cataract-generating eye diseases provide the postoperative functional prognosis after lens extraction
  - ***Complicated cataract in recurrent chronic uveitis,*** Fuchs heterochromic cyclitis (Irian heterochromia associated with unilateral torpid anterior uveitis and cataract)
  - Strong myopia is accompanied by early cataract
  - Retinopathy pigmentosa can accompany cataracts
  - DR

• **Medical treatment of cataracts has no significant effect on cataracts, it could prepare the patient for surgery.**

• **Surgical treatment is the only treatment for senile cataracts**

- the extraction of the cataractous lens is performed by phacoexeresis, followed by the correction of aphakia by an intraocular lens implant
- the operative indication for lens extraction is made when the opacification of the lens creates visual disability, which limits the autonomy of the elderly patient in daily personal or professional activities (in depending on the patient's age, type of activity)
- In cataract operated patients, vision loss improves significantly in up to 95% of cases and functional and cognitive ability improves
  - postoperatively, patients can move much more easily, the risk of accidents (domestic, traffic) is reduced, the risk of fractures is reduced, it improves the patient's psychosocial relationship
- vision and social integration improve even more after cataract surgery on the second eye
  - among cataract surgery patients there are also individuals whose vision does not improve or improves very little postoperatively due to multiple causes, which must be diagnosed and treated if possible:
    - ocular general comorbidities
    - postoperative astigmatism
    - complications of cataract surgery

GLAUCOMA

- Glaucoma comprises a group of eye diseases characterized by glaucomatous excavation at the level of the optic disc, visual field changes produced by increased IOP [2,4,5,10,16,17].
- Glaucoma is a leading cause of vision loss after age 40
- Glaucoma is present in 2% of the general population over the age of 65, with the percentage increasing after the age of 80
- In the elderly, many clinical forms of glaucoma are encountered, similar to those of adults, but in advanced evolutionary stages, decompensated, which require curative medical and even surgical treatment, it being known that primitive open-angle glaucoma - **GPUD (the most common form of glaucoma) has onset after 40 years and progressive chronic evolution**

Primitive open-angle glaucoma – GPUD [2,5,10,16]

- GPUD has minimal symptoms and it is possible to be initially detected in advanced stages, at an advanced age, by the family doctor, family members, institution (for institutionalized patients)
  - all glaucomatous patients must undergo strict periodic controls, to preserve visual function in order to ensure the quality of life and to limit the clinical decompensation of the disease
- Glaucoma screening includes:
  - early detection of glaucoma
  - complete eye clinical examination to detect the evolutionary stage of glaucoma and ensure the
  - indications of local, general medical treatment, if necessary surgical treatment
  - general clinical examination
  - tracking AV, CV, TIO
- The goals of glaucoma treatment in the elderly are:
  - maximum optical correction
  - prevention of glaucoma decompensation
  - ensuring eye and social comfort.
- Elderly patients have comorbidities, which must be identified and treated appropriately, and for glaucoma they receive topically administered drugs that are systemically resorbed

and can interfere with general medication
- Theoretically, the patient and the family of the elderly must recognize and be aware socially, physically and mentally of the eye disease, its stage of decompensation, associated or not with decompensation of general conditions and follow the treatment indications.
- It is essential - to ensure that vision loss is limited as much as possible - to carry out periodic eye checks, appropriate treatments and to have good communication between the doctor and the elderly glaucomatous patient

Secondary open-angle glaucoma - GSUD

- Clinical forms of GSUD: [4,10,15]
  - secondary glaucoma in senile cataract and aphakia
  - pseudoexfoliative glaucoma
  - cortisone glaucoma
  - neovascular glaucoma - GNV
  - glaucoma in tumors
  - post-traumatic glaucoma
- In all forms of glaucoma present in the elderly it is necessary:
  - performing glaucoma screening
  - documentation of eye disease
  - initiation or change of treatment depending on the clinical aspect, diagnosis and treatment of comorbidities associated with HTN, DM
  - control and exclusion of risk factors
  - control of psychosocial factors
- The decrease (or loss) of vision due to glaucoma in the elderly is an important factor in reducing the patient's quality of life and independence.
- Vision loss in elderly people with glaucoma must be quantified through periodic controls with clinical and therapeutic follow-up.
- **It would be necessary to have adequate ophthalmological and/or geriatric services to detect the cause of vision loss in the elderly patient and provide the necessary eye care; if these services exist, they are insufficient for careful control of visual function in all elderly patients who know (or do not know) the degree of decreased AV.**

AGE-RELATED MACULAR DEGENERATION - AMD

- AMD is related to the degree of retinal alteration at the level of the macula with the destruction of the neuro-sensory cells of the macular retina in the aging process accompanied by secondary functional alterations, with the progressive decrease of AV and CV alterations [9,16,20].
- AMD in the elderly is an important cause of vision loss after age 55, with age being an important risk factor.
- AMD = 1% - 50-55 years
- = 30% - after 70 years
- **The evaluation of AMD is irreversible after choroidal neovascularization appear under the action of VEGF angioforming factors**
- Elderly people with DMLV associated with cataracts require good patient/doctor communication to follow the evolution. Many elderly patients consider the vision loss to be caused by cataracts and believe that the AV loss may improve postoperatively, but the AMD patient may have total vision loss (difficult to accept for uninformed patients).
- Any elderly person with AMD should be informed of the possible progression of the disease to neovascularization accompanied by progressive or sudden loss of visual function.
- **AMD symptomatology is dominated by the macular syndrome**:
  - decreased central vision
  - metamorphopsia
  - central scotoma
  - difficult reading, recognizes details with difficulty

Atrophic AMD

  - progressive atrophy of EPR and alteration of photoreceptors
  - progressive decrease in AV
  - disabling central scotoma
  - FO- polycyclic bleached beach

AMD with subretinal neovascularization - the exudative form

  - is accompanied by significant serous and/or hemorrhagic exudate with macular detachment, with the extension of the neovas towards the fovea
  - rapid decrease in AV
  - metamorphopsia

AV self-monitoring recommended to the patient by the ophthalmologist is the best means to quickly identify the unfavorable evolution of AMD, but it is debatable in AMD patients with psychiatric disorders.

  - The appearance of metamorphopsias in parallel with the sharp decrease in AV can constitute an emergency element in AMD.
- AFG is important for the diagnosis of neovascular membranes and for the therapeutic indication.
- ***Preventive medical treatment*** has uncertain efficacy.
  - antioxidants - Vitamin C, E, carotenoids
  - vasodilators
  - trace elements - zinc, selenium
- ***Laser photocoagulation*** - for the occlusion of neovas by direct effect after the AFG landmark.
  - Argon laser - by direct photocoagulation of neovessels directed by AFG
  - Dynamic phototherapy
  - Transpupillary thermotherapy
- ***Surgical treatment*** by:
  - endocular
  - microsurgery with direct excision of neovascular membranes crossing the EPR opening and macular translocation
  - intravitreal antiVEGF treatment

- ***Orthoptic re-education (if needed)***
- The evolving AMD patient requires repeated treatment with

intravitreal anti-VGF, to which multivitamin and lutein supplements can be associated, which could reduce the risk of progression of AMD.

- Therapeutic intervention in the middle stage of AMD will be done as a matter of urgency to preserve central vision for as long as possible, to allow the patient good near vision, necessary for daily activity.
- Monitoring the stage of neovascularization in AMD
  - The clinical examination and AFG confirm the diagnosis of NVC, guide the indication for treatment, and evaluate the response to treatment [5,12,17].
  - OCT monitors NVC development and response to treatment
  - The predominant treatment in DMLV with NVC is represented by the use of intravitreal anti-VEGF that can influence the recent progression of lesions with improvement of AV
    - side effects - bevacizumab can cause stroke, myocardial infarction.

DRY EYE

- Dry eye is accompanied by reduced tear production, instability of the tear film, rapid evaporation (21)
- Patients with dry eye present: burning sensation, red eyes, tearing, decreased vision initially episodic, which over time can become permanent
- Causes of dry eye: aging, autoimmune diseases, Sjogreen's syndrome, antihistamine, decongestant medication, poor eyelid hygiene, chemical irritants
- Dry eye patients require: identification and treatment of the underlying condition, hygiene measures, artificial tears, omega-3 diet, anti-inflammatories, heat

OPTIC NERVE DAMAGE (NO)

Physiological aging of NO is accompanied by axonal loss, with increases in supporting tissue, deposits and vascular changes [22].

- axonal losses occur due to chronic ischemic processes, with ganglion cell death by apoptosis

- Visual function changes through these processes of involution with:
  - partial or total decrease in AV
  - decrease in contrast sensitivity
  - variable CV alterations
  - increasing the latency time at PEV

FO - accentuation of physiological excavation with evolution towards optic atrophy

- ***Pathological changes of NO***
  - acute anterior ischemic optic neuropathy - NOIA (the most common acute NO disorder)
  - chronic optic neuropathy - rare
  - chronic glaucomatous optic neuropathy - NOG in GPUD

- **Acute anterior ischemic optic neuropathy – NOIA**
  - ***NOIA arteritic – Horton disease -*** is caused by obstruction of the posterior ciliary arteries by atherosclerosis or arteritis [10,18]
    - a debut after the age of 60
    - a supervised cortisone treatment
    - recurrence on the second eye the second eye it is possible, but it is more common in arteritic NOIA Horton's disease, where to avoid bilateralization and blindness, corticosteroid therapy must be instituted quickly
  - ***NOIA Nonarteritic in a vascular context,*** DM, HTA, atherosclerosis 80%
    - a sudden onset in elderly patients
      - symptoms – sudden drop in AV, unilateral, CV deficit, fascicular, altitudinal, severe
    - An FO initially shows a partially or totally edematous papilla, with flaming hemorrhages on the papilla margin
  - an evolution the papillary edema completely resorbs in 6-8 weeks and is followed by optic atrophy in the sector or in total, depending on the extent of the papillary edema in the acute phase
    - **functional recovery is not possible**

Morphofunctional Vision Loss in the Elderly

- Slow vision loss in the elderly is caused by the morphofunctional aging of the visual analyzer [4]
  - elderly patients need bright light to read or perform various activities, and getting around at night can become increasingly difficult [12,22]
- Visual deficit in elderly people can be: [10,20]
  - blurred vision in recurrent vitreous hemorrhage, progressive cataract
  - central deficiency in:
    - AMD with difficulty in near vision, with lack of perception of central details, difficult or impossible to read, which can only be possible with the use of magnifiers.
    - RD in which the central visual deficit is related to macular invasion or extensive hemorrhages with DR.
  - peripheral deficit in glaucoma, vascular diseases of retina, cortical disorders of NO that can produce visual difficulties in different degrees of illumination
- The degree of AV decrease must be detected in the patient's medical context and methods that can help the elderly, individual, adapted to each patient, must be tried, through medical help, but very importantly also through familiar help so that the elderly do not end up in a situation of dependence.
- Eye and general physical and mental rehabilitation is attempted through specialized services.
- If the patient has a significant visual deficit and a degraded general condition, he needs permanent help to help him overcome his handicap
  - The blind must be helped, but not forced, because each person has his own personality and dignity, even if he is marked by a major handicap.
  - Patients with significant visual deficits must be escorted

outside the house (if necessary and inside), helped to eat, wash and carry out various maneuvers they need, without being abrupt, with tact, understanding, kindness, delicacy, for that any elderly person is a mentally unstable person, whose instability is accentuated by the weight in movement or the motor deficit (if any).

- It should be remembered that in elderly people there is a close connection between decreased vision, independence and depression.
- The patient can be trained in the community and in the family, for other activities, if his status allows.
- If the elderly person has to reshape his life, the family must do the same to help him, because any mentally intact person is willing to communicate. Any information provided to him must be adapted to his requirements.

Decreased Vision in Depression and Dementia

- The presence of depression in elderly people can accentuate vision loss, and vision loss in the elderly can be a determining risk factor in the development of depression, which over time affects the quality of life [1,3,21].
- Depression must be diagnosed and treated properly because over time it can contribute (depending on the individual and the degree of depression) to the accentuation of the biological and cognitive decline of the elderly.
- Any patient with reduced vision and depression must be monitored periodically and treated appropriately for each comorbidity.
- Patients with depression do not seek medical help and many of them do not notice the deterioration of vision, which can be caused by the unfavorable evolution of an eye condition.
- In order to establish a useful treatment for the eye and general disease, it is absolutely necessary to have a good collaboration between an ophthalmologist, a geriatrician, psychiatrist, to indicate the necessary treatment for each disordered component.
- Most often, the elderly do not recognize the existence of depression and often do not accept the indicated treatment.
  - a vicious circle is created in which the lack of antidepressant treatment accentuates the visual deficit, which decreases current daily activity and accentuates depression.
  - the doctor, the family, the social worker must identify the situation and immediately institute the necessary treatment and systematic periodic control.
  - antidepressant treatment must be done with great discernment, the doses will be used depending on the age, the clinical condition of the patient and other drugs used by the elderly for the multiple associated comorbidities.

Dementia

- Vision loss can be associated with dementia whose evolution is progressive towards major neurocognitive deficits, with memory loss.
- Elderly patients with dementia show a progressive cognitive deterioration, which causes the visual function, although present, to be poorly perceived by the mentally disabled patient and he behaves like an individual with major visual impairment
  - visual function in dementia progressively degrades and can constitute a major social problem because, every 5 years, the number of people with dementia between the ages of 65-90 increases alarmingly
  - it is difficult to establish visual maladjustment with or without actual visual deficit in dementia, as well as the fact that many patients with dementia on ophthalmological examination may have visual function within reasonable limits
  - in addition to the ophthalmological examination, a neurological and psychiatric check-up is necessary to assess the mental status to establish the existence of concentration problems, memory loss
- Visual symptoms in Alzheimer's disease precede memory loss or neurocognitive signs and include varying degrees of difficulty reading (alexia), difficulty writing (agraphia), homonymous hemianopsia, difficulty recognizing family objects (visual agnosia), difficulty perceiving colors, face recognition, etc.
- Often the visual function is normal (if the patient cooperates) the eye examination does not show pathological lesions, but the patient complains of difficulties in reading, walking, peripheral CV constriction
  - over time, the loss of vision increases, hemianopsia, unstable fixation, alexia, agnosia, optic apraxia appears
  - CT and MRI show cerebral atrophy in the posterior occipital and parietal lobe
  - PET (positron emission tomography) may show decreased function with hypometabolism in the parietal or occipital cortex
- The doctor and the family should be oriented in the situations of decreased vision, recognize the visual symptoms related to dementia and institute the appropriate treatment, the effectiveness of which is questionable.
- Dementia patients need permanent supervision by family members, geriatrician, neurologist and must be evaluated regularly, many times they become totally dependent because their visual-spatial orientation is finally compromised.

Screening for Comorbidities

- All elderly people who present vision loss due to eye diseases, vision loss accentuated and aggravated by the physical and psycho-social deficit related to the aging process require a clinical eye and general examination, which can sometimes be very difficult in uncooperative individuals for medical reasons or mental disorders related to aging.
- The rate of AV decrease in the elderly can be higher than in the adult because the eye diseases advance, and in old age blindness can occur in DMLV, glaucoma, cataracts.
- Need to look for:
  - ocular: refractive errors, uncorrected refraction, cataracts, glaucoma, DMLV, retinal vascular diseases.
  - the general causes of AV decrease can be: systemic

diseases DM, HTN, ASC, stroke, mental illnesses that can worsen over time. It is necessary for all patients to be permanently evaluated through annual checks (more frequently if necessary) to identify the functional and cognitive decline of the elderly person who blames the loss of vision, followed by diagnosis and treatment in which depression and dementia are important factors of emphasis of vision loss in the elderly.

- The ranking of the degree of regression of the elderly must also be correlated with the degree of AV decrease, and if possible, the specific treatment of the visual deficit must be carried out through optical correction, medical and surgical treatment.
- The decrease in vision with age limits the patient's independence, accentuates physical disabilities, mental health and social function, effects that appear in variable time for each individual.

Conclusion

Elderly people accuse the progressive decrease of vision with the passage of years. The progressive decrease in vision (sometimes alarming) parallel to the decrease in physical performance, increase the risk of the elderly patient losing independence and amplifies cognitive impairment at the ocular level, multiple anatomical and secondary functional changes occur with age. Vision loss in elderly patients is often chronic, binocular, asymptomatic. 18% of patients over 70 years have vision disorders, but their absolute member can be much higher (due to lock of addressability, physical and/or mental degradations, etc.

Slow vision loss in the elderly is caused by morphofunctional aging of the visual analyzer. Visual deficit in elderly can be: blurred vision in vitreous hemorrhage, progressive cataract, central deficiency in AMD, DM, peripheral deficit in: glaucoma, vascular disease of the retina, cortical disorders of NO.

Ocular and pathology increase with age, when ocular aging processes occur: cataracts, AMD glaucoma, but also general: HTA, ASC, DM. Cataract (senile, pathological, complicated) is the opacification of the lens with evolution towards total cataract accompanied by progressive vision loss. The population over 80 years has cataracts in 50% od cases, cataracts being an important cause of decrease even loss of vision in the elderly. Cataract require surgical treatment. Glaucoma is a leading cause of vision loss after age 50. In the elderly, many clinical forms of glaucoma are encountered, similar to those of adults, but in advanced evolutionary stages, decompensated, with require curative medical and even surgical treatment. GPUD (the most common form of glaucoma) has onset after 40 years and progressive chronic evolution. Vision loss in elderly people with glaucoma must be quantified through periodic controls with clinical and therapeutic follow-up. AMD (age related macular degeneration) in the elderly is an important cause of vision loss after age 55 *30% after 70 years). AV self – monitoring recommended to the patient by the ophthalmologist. The predominant treatment in AMD with NVC is represented by the use of intravitreal anti-VEGF, that can influence the recent progression of lesion with improvement of AV. NOIA (acute anterior ischemic optic neuropathy) – Horton's disease (arteritis) initially with partially or totally edematous flaming hemorrhage on the papilla margin an evolution edema complete resorbs in 6-8 weeks and is followed by optic atrophy.

The presence of depression in elderly people can accentuate visual loss, and vision loss in the elderly can be a determining risk factor in the development of depression. Vision loss can be associated with dementia whose evolution is progressive towards major neurocognitive deficits, with memory loss the decrease in vision with age, limits the patient's independence, accentuates physical disabilities mental health and social function.

References

1. Prada GI Geriatrics and gerontology. University Press. 2014.
2. Dumitrache Marieta. Compendium of Ophthalmological Pathology. Academy Publishing House. 2022.
3. Capizisu A. Boli patologice și principii terapeutice în geriatrie. Editura Medicală. 2014.
4. Dumitrache Marieta. Treatise on ophthalmology. University Publishing House. 2012.
5. American Academy of Ophtalmology. 2013.
6. Lee AG, Beaver HA. Geriatric ophthalmology. Springer. 2009.
7. Agarwal A, Agarwal A, Jacob S. Refractive Surgery. Jaypee Med Publish. 2009.
8. Faik Orucoglu, Mehmet Akman, Sumru Onal. Analysis of age, refractive error and gender related changes of the cornea and the anterior segment of the eye with Scheimpflug imaging. Cont Lens Anterior Eye. 2015; 38: 345-350.
9. Yanoff Myron, Duker S Jay. Ophthalmology. Mosby. 2004.
10. Kaiser PK, Friedmann NJ, Pineda R. Ophthalmology. Elsevier Saunders. 2014.
11. Chern KC, Zegans ME. Ophthalmology review manual. Lippincott Williams. 2000.
12. Friedmann K Trattler. Review of ophthalmology. Elsevier. 2005.
13. Justis E, Chirag S. The Eye Willis Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. Lippincott Williams. 2015.
14. Artal Pablo. Handbook of Visual Optics. CRC Press. 2017.
15. Gaudric A. Ophthalmology. Elsevier Masson. 2013.
16. Kanski JJ. Précis d'Ophthalmologie Clinique. Elsevier. 2005.
17. Gupta AK, Vinod Kumar Aggarwal, Neha Goel. Handbook of Clinical Trials in Ophthalmology. Jaypee. 2014.
18. Kanski J. Clinical Ophthalmology. Butterworth-Heinemann.
19. Mark A Rudberg. The Merck Manual of geriatrics. JAMA. 2000.

20. Kanski JJ, Milewski SA, Disease of the macula. Mosby. 2002.
21. Samar Kumar Basak. Dry eye disease. All India Ophthalmological Society. 2012.
22. Bernard J. (coordinator) Patologie du Fo. Elsevier Masson. 2006.
23. Bălăceanu Stolnici. Geriatrie practice. Editura Amaltea. 1998.