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Journal of Clinical Research and Reports

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Review Article

Clinical and Therapeutic Considerations in Retinal Vascular Occlusion

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Received date: March 06, 2024;; **Accepted date:** March 25, 2024; **Published date:** April 16, 2024

Citation: Marieta Dumitrache, (2024), Clinical and Therapeutic Considerations in Retinal Vascular Occlusion, *J Clinical Research and Reports*, 15(4); **DOI:10.31579/2690-1919/361**

Abstract

As young adults enter college, many seem to increase their alcohol consumption. Whether this behavior change is solely due to environmental factors, such as peer pressure or lack of supervision, has not been fully separated from developing or emerging intrapersonal traits that might facilitate the behavior. This trend underscores the importance of investigating the relationship between excessive drinking habits and deviant personality traits. This study, aimed at investigating the relationship between "dark triad" personality traits and harmful alcohol behaviors, may provide valuable insights into the social cultures on college campuses that promote maladaptive drinking habits. Based on the trait theory of personality, we hypothesized that dark triad traits would be associated with negative alcohol behaviors. To test our hypothesis, we administered the Young Adult Alcohol Consequences Questionnaire (YAACQ) and Short Dark Triad scale (SD3) to 800 participants. After cleaning the data, we analyzed 799 responses to study measures and demographic items. Our findings revealed that, of the dark personality traits, Machiavellianism and psychopathy had small positive associations with harmful alcohol consumption. Moreover, we found that both personality and Greek life participation were significant predictors of alcohol misuse. This study may serve as a stepping-stone for future research into the interplay between the behavioral tendencies and cultural settings in which alcohol abuse occurs among college students. Thus, provides additional insight into a complex problem plaguing college communities.

Keywords: alcohol abuse; deviance; personality; psychopathy; alcohol consumption; trait theory

Introduction

Central retinal arterial occlusion – CRAO is on ophthalmic emergency, analogous to a central stroke, with higher incidence to 60 years males

Central retinal artery occlusion – CRAO is an acute retinal ischemic stroke; it is an absolute, devastating eye emergency, with the need for immediate evaluation and the institution of emergency treatment in a specialized center.

Mechanism of retinal vascular occlusion are: arterial embolism (cholesterol plaque, fibrinoplatelet) hypoperfusion and vasospasm – CRAO major risk factors for CRAO are atheroma and emboligenic cardiac anomalies.

Causes of CRAO are frequent general – vascular obliteration (embolus, thrombosis) in obstructive and/or emboligenic cardiovascular pathology, hypertension, atherosclerosis, valvular heart disease cardiac arrhythmia, carotid atheroma plaques, inflammatory (Horton's arteritis, SLE, Wegener, Churg Strauss disease – and locale rare – ocular (foveolar drusen, HIV), orbital (cellulitis, tumors).

CRAO occurs when the artery is blocked by an embolus (generally), is frequently unilateral, painless and severe, and is accompanied by a sudden, severe decrease in vision that in most cases remains permanent if the occlusion of the central retinal artery (CRA) lasts for more than 90 minutes.

Ocular sign are: ***retinal ischemic signs*** – pale milky white retina, edematous macula with cheery/red center and disruption of the retinal vascularization; if cilioretinal artery is preserved the interpapillo macular space is perfused and is normal.

CRAO is a rare condition occurring in 1.9/100,000 patients on average, with an incidence that increases with age, after 60 years old with a frequency of 10/100,000 patients, more common in men.

The retina vascularized by the CRA in conditions of prolonged occlusion over 90' is accompanied by retinal ischemia followed by definitive retinal atrophy. If the patient comes within the first hours after the arterial occlusion, it is possible to try to unblock the embolus by thrombolytic

medication with selective injection after the carotid/ophthalmic artery catheterization.

Late ocular complications are: retinal and iris neovascularization with rubeosis iridis and secondary glaucoma neovascular. In the presence of iris neovascularization, intravitreal hemorrhage may occur after vascular occlusion.

Multiple therapeutic modalities have been tried in CRAO, all without spectacular results, not being obvious that the use of fibrinolytics in the treatment of acute CRAO is life-saving, the treatment not being able to regenerate the ischemic necrotic retina, so the therapeutic efficacy remains debatable.[1, 2, 31, 32]

Retinal Blood Flow

The central retinal artery - CRA - is the first branch of the ophthalmic artery, a branch of the internal carotid artery, which vascularizes the fibers of the optic nerve and the inner layer of the retina; after entering the eye the CRA is divided into 2 superior and inferior branches and each of these into the nasal and temporal branch. CRA occlusion occurs most frequently (90%) through embolus or thrombosis in which the risk factors are: diabetes, hypertension, coronary disease, atherosclerotic plaque, carotid artery, family history of vascular disease. [3, 4, 36]

The cilioretinal artery is present in 20% of patients and is a branch of the short posterior ciliary artery, a branch of the ophthalmic artery, a branch of the internal carotid artery.

The cilioretinal artery can be obstructed secondary to central retinal vein occlusion through increased outflow resistance.

Arterioles give birth to capillaries which are terminal vessels.

Ophthalmic artery occlusion may be secondary to giant cell arteritis.

- **Non-arteritic CRAO is:**
  - Permanent non-arteritic CRAO due to embolus or thrombosis
  - Transient non-arteritic CRAO – 15-17% with good visual prognosis, often with normal fundus.[9, 11, 45, 46]
- **Arteritic CRAO** is generally secondary to Horton's arteritis. 4% of patients are at risk of bilateral vision loss (initially, the eye is asymptomatic); it requires immediate corticosteroid treatment. [4, 39, 43]

CRAO is produced by cholesterol emboli (from Hollenhorst plaque), fibrinoplatelets (associated with atherosclerotic vessels) and calcium emboli (from heart valves). [5, 23, 28, 37]
Rarely, CRAO can be produced by cardiac myxoma, fat embolism, from long bone fractures, migraine (30-40 years old), arrhythmia, mitral valve prolapse, oral contraceptives, pregnancy, coagulation disorders, infectious inflammatory etiology (toxoplasmic retinochoroiditis, syphilis, giant cell arteritis).
CRAO - major ocular emergency has poor visual prognosis, less than 15% of patients with CRAO under conventional treatment can benefit from minimal vision improvement. [1, 27, 28, 44]

if the patient comes in the first hours after CRAO, it is possible to try to unblock the embolus by thrombolytic medication, by selective injection with catheterization of the internal carotid artery/ophthalmic artery.
Occlusion of retinal arterial branches produces edema in the distribution territory of the affected artery.
Precapillary arterial occlusion manifests as cotton-wool nodules which are superficial white ischemic retinal cotton wool spots, which in 5-7 weeks are followed by varying decrease in visual acuity depending on the extent and location of the occluded area.

Mechanisms Of Retinal Vascular Occlusion

- Three mechanisms are involved in retinal vascular occlusion:
  - **Arterial embolism** originates from atheromatous plaque at the level of the carotid bifurcation, sometimes the ophthalmic artery, aortic arch, or embolic heart disease.
    - **Cholesterol plaque** – yellow-orange, refractive, completely occludes the arterial lumen, located at the arterial bifurcation, mobile from one examination/to another (ophthalmic carotid atheroma, optic arch).
    - **Fibrinoplatelet** - white/gray embolus, pale, mobile, superimposed in the vascular lumen - mural thrombus (carotid, aortic atheroma), intracardiac thrombus.
  - **Hypoperfusion** – the consequence of a stenosis in the ophthalmic artery, carotid artery.
  - **Vasospasm** – CRA
- **Causes Of Crao**[3, 5, 6, 35]
  - **General** – most frequent
    - embolus – starting from the heart in cardiovascular pathology is the main cause of retinal vein occlusion in atherosclerosis, hypercholesterolemia, endocarditis, fractures, trauma.
    - thrombosis
    - inflammatory arteritis in Horton's arteritis, collagen diseases, SLE, periarteritis nodosa, Takayaso disease, Churg Strauss syndrome, hypercoagulability, polycythemia, antiphospholipid syndrome
    - infectious: syphilis, leptospirosis, HIV.
    - vascular obliteration in:
      - obstructive and/or emboligenic cardiovascular pathology, hypertension, atheromatosis, atherosclerosis, valvular heart disease, cardiac arrhythmia, carotid atheroma plaques, hypercholesterolemia[7]
      - inflammatory arteritis – Horton's, collagenosis (SLE), Takayasu disease, Wegener's, Churg Strauss disease.
      - coagulopathies
      - infectious arteritis: syphilis, leptospira, viruses (HSV)
      - retinal migraine
      - carotid artery disease by atheromatous ulceration at the bifurcation of the internal and external carotid artery, associated with possible emboli: of cholesterol - Hollenhorst plaques at the bifurcation, yellowish, asymmetric, fibrinoplatelets that can produce transient retinal ischemia with decreased vision 2-10' or calcium emboli which are the most dangerous, producing permanent vision loss.
  - **Locale** – rare
    - ocular: foveolar drusen, arterial macroaneurysm, toxoplasmosis, HIV.
    - orbital: cellulitis, tumors, hematoma, trauma, RB injection.
    - CRAO has a 10% risk of bilateralization.
- **Major risk factors for CRAO are atheroma and emboligenic cardiac anomalies** [28, 27, 25, 37]
- Cases of CRAO in patients affected by COVID-19 are reported in the adult population and in pediatric (rare). Treatment with anticoagulants and steroids was tried but the visual was poor.

SYMPTOMS, CLINICAL SIGNS – IN CRAO[3, 5, 8, 24, 27]

Symptoms

- sudden, total, painless, unilateral vision loss
- decrease in vision is less if cilioretinal artery is present
- amaurosis fugax can precede vision loss
- RAPD-net or abolished

- absent visual field or major defect
- specific symptoms for Horton's disease

➢ temporal headache
➢ scalp hyperesthesia, anorexia, fever, fatigue.

Ocular signs

- ***retinal ischemia signs:***
  - ➢ pale, milky white retina
  - ➢ the papilla may be normal or pale with a blurred outline, with few hemorrhages on the optic disc
  - ➢ edematous macula with diffuse edema with cherry/red center by the contrast between the white macula and the red fovea (vascularized and perfused by the choroid)
- ***disruption of the retinal vascularization*** - distally to the arterial blockage, thin arteries (sometimes visible embolus), segmental flow

➢ **if cilioretinal artery is preserved, the interpapillomacular space is perfused and is perfectly normal, but surrounded by retinal edema**

Topographic Forms of Retinal Artery Occlusion

Retinal artery occlusion produces irreversible ischemia with retinal edema, stopping the transmission of information to the photoreceptor

- the first changes appear 15 minutes after the occlusion, and the definitive lesions occur in 90 minutes

- after a few months, the artery can re-permeabilize with total resorption of the edema, with a necrotic, non-viable retina, and profoundly altered vision.

CRAO

- CRAO is central retinal artery occlusion
- CRAO is interruption of blood flow through central retinal artery by thromboembolism with /or vasospasm with/or without retinal ischemia.
- CRAO is:
  - **CRAO Non-Arteritic** result of local thrombus formation or thromboembolism.
  - **CRAO Arteritic** occurring in the context of systemic inflammatory condition
- CRAO
  - **without the cilioretinal artery**, it produces unilateral blindness
  - **with cilioretinal** artery, it produces massive perimetric deficit, with preservation of vision

Branch retinal artery occlusion (BRAO)

- BRAO is interruption of blood flow through a branch retinal artery by thromboembolism or vasospasm with or without retinal ischemia.
- decrease in VA
- perimetric deficit depending on the topography of the occlusion.
- of frequently embolic and sometimes periarteritic etiology; most frequently the **occlusion is localized on the superior temporal branch with macula involvement.**
- onset with decreased VA, altitudinal or sectoral amputation of the visual field
- retinal edema distally to the retinal occlusion corresponding to the ischemic retinal area, sometimes the embolus is visible
- soft exudates
- the obstructed artery can remain permanently occluded with the resorption of the edema in 30 days

Ophthalmic artery occlusion

- is accompanied by interruption of retinal and ciliary circulation
- VA – f.p.l.
- the clinical picture shows CRAO with the absence of the red/cherry macula and ocular hypotony
- FA – absence of vascular impregnation.

Transient retinal artery occlusion

- produces transient monocular blindness – Fugitive amaurosis with visual eclipses
- a few minutes with total functional recovery, progressive immediately.
- normal visual acuity and fundus outside the crisis
- cardiovascular, carotid, hematological balance is necessary

Cilioretinal artery occlusion (rarely)

- isolated or associated with Central retinal vein occlusion (CRVO), *Anterior ischemic optic neuropathy* (*AION*)[9, 31]

- eye fundus

  - ➢ retinal papilla edema in the area normally perfused by the cilioretinal artery
  - ➢ onset with sudden decrease in central vision.

Arteriolar occlusion

- cotton-wool nodules
- asymptomatic

A patient with arterial retinal vascular occlusion requires a complete multidisciplinary clinical examination: cardiologist, internist, neurologist, ophthalmologist, to determine the retinal arterial occlusion and its causes.

- identification of antecedents and cardiovascular risk factors
- clinical signs of Horton's disease (50 years old, headache, temporal artery anomalies, alteration of the general condition)
- BP, pulse, cardiac auscultation, carotid
- neurological examination
- EKG
- laboratory examination – ESR, CRP, leukocyte formula, blood sugar, lipids, creatinine
- cardio vascular balance, Doppler, Holter, ECG

Complete clinical examination, internal medicine, cardiology with ECG and echocardiography is mandatory.

Complementary Examinations[30, 31, 41]

- FA
  - ➢ determine the extent of the retinal vasculature and a variable amount of residual retinal circulation [31]
  - ➢ delayed filling of retinal vessels, increase in arm-to-retina time
  - ➢ non-filling of the arterial tract – "dead tree" appearance
  - ➢ associated choroidal ischemia
- Angiography with indocyanine green allows the study of choroidal and choriocapillary circulation
- OCT

  - May demonstrate an increase inner retinal layer thickness in the acute phase due to the retinal edema and optic nerve swelling [31]
  - macular – hyperreflectivity of the layers of the inner retina in the acute stage in relation to ischemic edema and atrophy in the chronic evolution
  - significant detachment of the internal limiting membrane significantly
  - maculopathy caused by ischemic injury through hypoperfusion of the retinal capillary plexus
- sequential clichés on eye fundus
- clichés in blue light to highlight the embolus
- identification of Horton's disease, ESR, CRP, temporal artery biopsy
- young people – transesophageal ultrasound to reveal chronic embolic pathology – left ventricular hypertrophy, atrial myxoma, mitral valve prolapse, tricuspid aortic valve
- in elderly patients:
  - evidence of ischemic stroke in the antecedents
  - research of cardiovascular risk factors by echo Doppler, cardiac ultrasound

Primary prevention

It is carried out by controlling risk factors and aggressive management of CRAO in specialized services.

Secondary prevention

Through multidisciplinary collaboration - neurologist/ophthalmologist/internist

- treatment of hypertension, dyslipidemia, DM, obesity, sleep apnea

- diet, sports

- carotid revascularization, anticoagulation for cardioembolic source, steroid treatment in Horton's arteritis

Positive diagnosis:

- symptomatic clinical balance
  - significant, sudden, painless loss of vision within seconds
  - complete loss of vision in Horton's disease
- eye exam:
  - RAPD
  - early eye fundus – normal
  - rapid cherry red macula (prognosis reserved)
  - amaurotic mydriasis
  - in evolution, pale optic disc, thinning of the retinal layer, retinal pigment mobilizations, diminished vascular caliber, profoundly reduced vision
- FA – absence of perfusion in the affected artery
- Echo Doppler
- Carotid echocardiography can detect the source of the embolus
- ECG
- in Horton's arteritis – ESR, PCR, platelet count

Complications:

- Retinal and iris neovascularization with rubeosis iridis, secondary neovascular glaucoma is possible in 20% of patients within a few weeks
- In the presence of iris neovascularization, intravitreal hemorrhage may occur after vascular occlusion
- Increased risk of stroke after CRAO in the following weeks.
- Common causes of death in CRAO include: coronary events (acute myocardiac infarction) cardiovascular related (hypertension, heart valve disease) or stroke ischemic more frequent in CRAO. [29, 33, 34, 42]

Management in Retinal Artery Occlusion

- **CRAO** treatment is an **ABSOLUTE MEDICAL EMERGENCY** because the loss of vision is severe and irreversible. Clinical diagnosis and treatment must be started immediately (even before the necessary clinical and paraclinical investigations), because after 2 hours, the irreversible cellular changes related to anoxia by ischemia are set in, as well as optic and retinal atrophy occur. [10, 11, 12]
- The management of CRAO [24]
  - acute -restore ocular perfusion in the CRAO
  - subacute preventing secondary neovascular complications to the eye
  - long term – preventing others vascular ischemic events
- **The occluded central retinal artery generates cell death within 90' of ischemia, and the retina does not have the ability to regenerate and atrophies.**
- It is estimated that less than 15% of patients may have real benefit, the effectiveness of treatment in CRAO being debatable.
- **Treatment in CRAO is immediate, multidisciplinary –** ophthalmologist / neurologist / internal medicine, emergency medicine.
- **There is no treatment capable of regenerating the necrotic retina**. [11]
- Treatment in CRAO must be carried out immediately before retinal necrosis, the evaluation of the therapeutic potential in CRAO being related to the duration of ischemia. [12]
- ***Conservative treatment*** is a therapeutic alternative to reduce the risk of ischemic events.
  - Conservative therapies with: vasodilatation, reduction of intraocular pressure, ocular massage.
- Intraarterial, intravenous thrombolysis for reperfusion therapies in CRAO.
- Treatment with thrombolytics remains controversial.

Medical treatment[23, 24, 25, 29]
Treatment objectives in CRAO are[11, 13, 24, 27, 33, 34]

- ***increase in retinal oxygenation***
  - mask inhalation 95% O2 and 5% CO2 mixture, for 10 minutes every 2 hours for 24-48 hours → 48-72 hours
  - retinal dynamics after carbogen is controversial
- ***protection of the retinal cell from ischemia***
  - xanthine derivative reduces blood viscosity, increases microcirculatory flow and tissue perfusion
  - nitroglycerin relaxes the muscles of small vessels
  - vasodilators
  - systemically, in the ophthalmic artery through an arterial catheter - plasminogen activator administered by a competent person
- ***dislocation of the embolus***
  - external ocular massage that could mobilize the embolus to the periphery
  - anterior chamber paracentesis
  - ocular hypotonics
  - dopamine receptor agonist: Bromcriptine, Cabergoline
  - Dihydroergotamine, Ergotamine
  - Methysergide
  - phorfodiesterase inhibitors

- ***intraocular pressure lowering to perfusion pressures and dislodge obstruction:***
  - topical β blocker
  - carbonic anhydrase inhibitor, mannitol
  - anterior chamber paracentesis
- ***ocular massage for retinal artery dilatation*** and possible risk of embolus moving peripherally and worsening symptoms
- ***dissolution of the thrombus***
  - heparin therapy in curative doses in the absence of a contraindication until the embolic pathology is eliminated
  - anticoagulants can be effective 6-8 hours after VA decrease.
  - low molecular weight dextran - Reomacrodex i.v.
  - antiplatelet agents in the absence of an indication for anticoagulant treatment
  - fibrinolytic therapy
    - intravenous for dissolve clot with reduced efficacity after 4,5 h tPA (tissue – type plasminogen activator);
    - intraarterial trombolysis to patient selection because of increase risk of adverse events;
    - controversial; the risk/benefit ratio will be followed (if used) [14, 38]
- ***the treatment of the underlying disease that can generate CRAO***
  - Horton's disease – emergency corticotherapy; methylprednisolone i.v. 250-500 mg/day for 3 days followed by oral Prednisone 1 mg/kg/day.

There is no effective curative treatment in CRAO.

Immediate therapeutic protocol in CRAO[15, 16]

At the moment of vision loss, the patient is directed to the specialized center where the following will be performed:

- external eye massage with intermittent pressure with closed eyes

- ocular hypotensive drugs – mannitol, acetazolamide, antiglaucoma topical Timolol.

- thrombolytic medication (if indicated)

- in the acute form – the attempt to restore the occluded arterial flow

- prevention through systemic control of possible ischemic vascular events over time.

CRAO treatment - retinal tolerance 240 minutes.

Non-invasive therapy[16, 17, 20, 24, 27, 33, 34, 42]

- **vasodilators** – vascular dilatation can increase blood flow and could reduce CRA blockage (with potential risk on systemic circulation
  - pentoxifylline, sublingual isosorbide dinitrate relaxes the smooth muscles of the vessels
  - carbogen inhalation (95% O2, 5% CO2)
  - hyperbaric oxygen[18]
- **eye massage** – can mobilize (dislodge the clot), reduce IOP, increase CRA perfusion
  - combined with Acetozolamide can lower IOP by 5 mmHg
- **decrease in intraocular pressure**
  - Acetozolamide 500-250 mg, iv, increases retinal perfusion
  - Mannitol 1.5-2g/kg – 30-60 i.v – increases the perfusion gradient in the optic nerve head and decrease IOP
  - topical antiglaucomatous therapy
- **reduction of retinal edema**
  - Methylprednisolone – i.v. controversial; to reduce retinal edema, but mandatory in young people with vasculitis

Invasive therapy

- **Anterior chamber paracentesis – reduces IOP, improves retinal perfusion.**
- **Transluminal Nd Yag laser**
  - only visible intraluminal embolus can be treated, which is very rare
  - Yag laser with energy 0.5-1 mJ
  - visual prognosis variable below 50%
  - high rate of possible complications – vitreous hemorrhage
  - **The technique is not simple and can only be used for visible embolus (very rare).**

Thrombolytic treatment[25, 29, 31, 32, 34, 38, 39]

CRAO can be considered clinically as a cerebral ischemic stroke, in which thrombolytic treatment might be indicated.

- **Intravenous thrombolysis** with rTPA 0,9 mg/body weight – maxim 90 mg 10% i.v. bolus time maxim is given in patients with CRAO within 1,5-4,5 h of the onset of symptoms in acute stroke, exposes the patient to higher doses and systemic distribution with its consequences and risks.
  - recent studies report that there may be a small visual benefit for intravenous tPA.
  - intravenous thrombolytic treatment, as there are potential benefit in a time windows up to 4,5 h for NA-CRAO[23]
  - is necessary strict case selection for intravenous, treatment trombolytic
- **Intraarterial thrombolysis** by selective catheterization and thrombolytic infusion in the ophthalmic artery reduces systemic therapeutic load, but requires multidisciplinary team, radiology service, monitoring of vital functions during and after catheterization
  - the most serious complication is symptomatic intracranial hemorrhage 8% of which 4% are fatal (NINDS study cited by N. Hakim) [22].
  - cholesterol emboli CANNOT be dissolved, so the technique is eligible in 11% of patients. [19, 21, 22]
  - is possible an improvement in visual acuity in up to 60-70% of patients treated with tPA
  - the use of intra-arterial clot-busting tissue plasminogen activator (tPA) therapy in the Eagle study (cited by J.I. Lim) found no post-therapeutic visual benefit, and had adverse effects with significant intracranial haemorrhage[4, 19, 20, 21, 23].
  - new technique Kodonaso et al (cited N. Hakim) - vitrectomy and injection of rTPA directly into the retinal artery with a micro-needle of 200 μg found an improvement in vision, but the technique is difficult[22].

- **Strict case selection is required for thrombolytic treatment, as there are potential benefits (currently inconclusive) but also possible risks (some even lethal).**
- **There is no clear evidence for the effectiveness of the therapy.**
- **No treatment, up to this point, is sufficient to restore visual function.**
- Medical education is necessary for the patient.

Long-term management

CRAO is an ischemic stroke in a terminal vessel as is cerebrovascular accident (CVA)

- it is necessary to identify the triggering factors of ischemia and their correction (as much as possible)
- clinical follow-up of the case in the ophthalmology service
- treatment of complications
- sight is lost and requires professional reorientation when needed
- patients with retinal emboli have a current mortality rate 3 times higher.

Management of arterial branch occlusion

- CRAO treatment
- complete evaluation of the cardiovascular system, coagulopathy
- VA has a minimal/average decrease in these cases

Cilioretinal artery occlusion

Cilioretinal artery occlusion causes variable central vision loss.

The visual prognosis may be good if the macula has been spared.

If cilioretinal artery occlusion is combined with AION, the visual prognosis is reduced.

Inflammatory arterial occlusion[6, 13, 17, 39]

Rare condition related to Horton's inflammatory arteritis, SLE, syphilis, idiopathic.

- Single or multiple arterial branch occlusions without visible embolus, sometimes periarteritis sheaths are present.
- Treatment requires systemic corticosteroid therapy.

Discussion

In our study CRAO is an ophthalmological emergency, causing severe permanent vision loss of patients.

Most patients with CRAO, in our study has cardiovascular disease (hypertension ischemic hearts disease, atrial fibrillation), diabetes, dyslipidemia.

For patients with CRAO is necessary detection:

- common vascular risc factors – blood pressure, cholesterol, blood sugar level
- embolic sources – duplex carotid ultrasound echocardiogram
- young patients (< 50 ani) – hypercoagulable screen, vasculitis screen, blood analysis.

CRAO was usually a clinical diagnosis with funduscopic examination a cherry red spot and retinal pallor from retinal ischemia, associated with acute visual loss.

Patients suspected of having CRAO may be identified and then given higher priority. Is necessary detection CRAO patients within 4, 5h after symptoms onset, in „time therapeutic window" with intravenous thrombolysis the use of tissue plasminogen activator – tPA for treatment thrombolytic.

Current options for treatment was: isosorbide dinitrate, pentoxiphyline, inhalation of a carbogen hyperbaric, oxygen ocular massage, acetazolamide, mannitol intravenous, anterior chambers paracentesis and methylprednisolone. Recent has been in the use tPA for treatment of acute CRAO.

Conclusions

CRAO is an ophthalmological medical emergency accompanied by permanent vision loss. There is no treatment capable of regenerating the ischemic necrotic retina.

Treatment in our study was: vasodilator medication with pentoxifylline, isosorbide dinitrate, carbogen inhalation, hyperbaric oxygen provides vascular dilation that could reduce CRAO blockage. External ocular massage could mobilize the clot, lowering intraocular pressure increases retinal perfusion, and methylprednisolone is indicated in young vasculitis. Anterior chamber paracentesis improves retinal perfusion. If necessary, transluminal Nd Yag laser treatment is indicated in CRAO. Thrombolytic treatment with intra-arterial tissue plasminogen activator (tPA) is debatable. CRAO remains a serious disease with significant vision loss, with minimal therapeutic efficiency. Patient education, CRAO treatment, prevention of risk factors are necessary for the remaining healthy eye. Dispensarisation of patients with central retinal artery occlusion is very important.

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DOI:10.31579/2690-1919/361