Review Article

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Eye Diseases Affecting the Cerebral Vascular System

Marieta Dumitrache[1]*, Rodica Lascu[2] and Miruna Cioboată[2]

[1]*University of Medicine and Pharmacy Carol Davila, Bucharest, Romania.*

[2]*Lascu Rodica Ophthalmolgy Clinic IMED, Sibiu, Romania.*

***Correspondence:**

Marieta Dumitrache, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania.

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ABSTRACT

Carotid occlusive disease generated by the atheromatous process is responsible for transient or permanent ischemic stroke. Most ischemic attacks occur in the territory of the middle cerebral artery. Occlusions of the carotid axis are manifested by general ocular and cerebral signs. Management of carotid obstruction requires: control of risk factors, drug treatment (antihypertensive, statins, anticoagulant), surgical treatment if necessary.

Vertebrobasilar ischemia is accompanied by vestibular symptoms (vertigo), dizziness, vomiting, visual symptoms, less often, headache, mental confusion, dysarthria, tinnitus. Thrombosis of the cavernous sinus can be produced by the spread of infection from infectious foci of the cephalic extremity and is manifested by signs of generalized sepsis and inflammatory eye signs, initially unilateral that become bilateral.

Keywords

Occlusive carotid disease, Transient ischemic attack, Stroke, Middle cerebral artery syndrome, Posterior, Hemianopsia, Balint syndrome, Basilar artery occlusion, Vertebrobasilar insufficiency, Cavernous sinus thrombosis.

Introduction

- The arterial vascularization of the brain is given by the arterial **circle of Willis** which represents the complex anastomosis at the base of the brain between the internal carotid arteries (ICA) and the vertebral arteries (VA)
- internal carotid arteries and their collaterals irrigate the formations of the brain with the exception of the occipital lobe of the cerebral hemisphere
- vertebral arteries and their branches, provide vascularization of the occipital lobe, brain stem and cerebellum.

Occlusive Syndrome of the Carotid System

Common carotid artery (CCA) and internal carotid artery (ICA) pathology includes carotid obstructive disease, clinically manifested by:

- transient ischemic attack through the transient interruption of blood circulation
- stroke in the occlusion of an artery

Internal Carotid Artery Stenosis with Partial or Total Occlusion is Generated by the Atheromatous Process and is Responsible for Amaurosis Fugax, Transient or Permanent Ischemic Stroke, Ocular Ischemic Syndrome [1]

- Carotid stenosis is present in approximately 10% of the elderly, the rate of ICA occlusion being 6/100,000
- of people with carotid artery stenosis more than 5% will have a stroke in the next 5 years
- men older than 65 and physically inactive are more prone to stroke (as are blacks and Hispanics)
- Ischemic strokes are the most common strokes, in which atherothrombosis of large arteries including CA causes 15% of ischemic strokes
- **Stenosis (Occlusion) of the carotid axis – Common Carotid Artery – CCA and Internal Carotid Artery – ICA**
- represents 23% of all cerebral infarctions and 46% of strokes in the territory of the middle cerebral artery
- the main etiological factor of carotid axis stenoses - 60% is represented by atherosclerosis with multifocal intra and extracranial, uni, bilateral localization
- atherosclerosis is frequently localized at the level of the carotid sinus
- ¾ of carotid occlusions are generated by pre-existing arterial stenoses

- associated risk factors of carotid stenoses or occlusions are hypertension and diabetes.

- **Most carotid transient ischemic attacks (TIAs) and strokes occur in the middle cerebral artery (MCA) territory** and are associated with contralateral hemiplegia or sensory loss of the face or lower arm and contralateral homonymous hemianopsia [2].
- Stroke – left MCA can produce aphasia
- Carotid obstruction has fluctuating symptoms including recurrent transient ischemic attack, major or minor stroke depending on collateral circulation, vasoreactivity associated with hemodynamic factors
- **Stroke is the most serious complication of carotid occlusive disease**

Carotid Axis Stenoses or Occlusions Are Manifested by Polymorphic Signs: General, Ocular and Cerebral [3]

General warning signs:

- headache, especially in the form of hemicrania on the side of the damaged carotid with a continuous or migraine-like character
- mental disorders: confusional episodes, drowsiness, decreased intellectual performance, changes in affectivity
- different forms of lipothymia accompanied by tachycardia and arterial hypotension.

Ocular signs:

- transient monocular blindness - which precedes the appearance of cerebral signs by months or years
- embolisms of the retinal arteries
- significant decrease in blood flow in the ophthalmic artery (ocular signs appear in at most 1/3 of cases with carotid lesions)
- lateral homonymous hemianopsia
- miosis or Claude-Bernard-Horner syndrome on the side opposite the hemiplegia when pericarotid sympathetic fibres are also involved
- oculomotor nerve palsies

Cerebral signs:

- hemiparesis or hemiplegia - predominantly faciobrachial, global (capsular), with crural predominance
- sensitivity disorders – total hemihypoesthesia or cortical type with predominance of deep hypoesthesia
- aphasia is constant in lesions of the dominant hemisphere
- convulsive seizures of focal type
- exploration of the carotid arteries and their branches can be done with: ophthalmodynamometry, ophthalmoscopy, Doppler echoarteriography, angioscintigraphy, cerebral arteriography

Transient Ischemic Attack– TIA and Amaurosis Fugax – FA [4]

- TIA is the most important sign and is frequently early in carotid stenoses or occlusions

- The brain injury frequently occurs in the same territory where the ICA occurred
- ***Amaurosis fugax***
- monocular decrease in second vision typically rarely prolonged total or partial (blurring of vision) unprovoked, unpredictable, isolated sometimes accentuated in evolution (over time)
- **there are no neurological symptoms in AF**
- ***Transient ischemic attack*** (15 minutes, rarely hours)
- decrease in monocular or total vision, painless or without ocular involvement
- possible neurological signs: dysphagia, contralateral hemiparesis and paresthesia
- temporary paresis of the contralateral arm, leg or both
- numbness in hand, foot, face, tongue, contralateral
- **Progressive Ischemic Attack**
- clinical manifestations progressively aggravated in hours ending with:
- severe and persistent neurological deficits
- cerebral infarction
- regressive ischemic attack
- possible complications: intracranial hypertension with disturbances of consciousness, motor deficit, papillary stasis.
- **Complete ischemic attack – cerebral infarction**
- acute onset
- major and persistent neurological signs
- 74% preceded by TIA
- middle cerebral artery syndrome is most frequently present with:
- hemiplegia
- hemihypoesthesia
- lateral homonymous hemianopsia
- aphasia if the dominant hemisphere is affected

Carotid Stenoses or Occlusions Can Also Develop without Cerebral Symptoms

In relation to the localization of arterial occlusions, several syndromes have been described: [5]

Common Carotid Artery occlusive syndrome – CCA is rare < 1% of ischemic stroke is:

- ***proximal type***
- obstruction in the aortic arch (left) or brachiocephalic trunk (right) similar to distal CCA without temporal artery asphyxia
- clinical aortic arch syndrome
- ***distal type***
- superior obstruction also affects bifurcation and carotid syndrome
- symptoms similar to TIA occlusion

Internal Carotid Artery occlusive syndrome - ICA - unilateral and bilateral

- **stroke symptomatology by affecting the ICA varies according to the vascular involvement of the Willis polygon through extra and intracranial collaterals and have elements of stroke in the territory of the middle or anterior cerebral artery (4)**
- Onset by TIA, by complete ischemic attacks, by progressive ischemic attacks
- arterial lesions discovered by chance during arteriography.
- The most frequent occlusions are found at the level of the origin of the ICA, most of the time they also include the carotid siphon

Middle Cerebral Artery Syndrome

- The middle cerebral artery (MCA) is the largest of the cerebral arteries and is the most affected by stroke.
- Infarcts that occur within the vast distribution of this blood vessel lead to neurological sequelae [6].
- MCA cerebral stroke is characterized by the sudden onset of focal neurological deficit, resulting from cerebral infarction or ischemia in the territory irrigated by MCA.
- Infarcts in the territory of the MCA are the most frequent in relation to all types of cerebral infarcts and those in the carotid system.
- Men are affected by MCA strokes more frequently than women with a ratio of 3/1.
- The risk of MCA strokes increases with age. The highest incidence is at the age of 70-80 years.
- Clinically, patients with MCA stroke syndrome may present: [7]
- sudden onset with focal neurological deficit with polymorphic and severe clinical picture
- global hemiplegia
- hemihyperesthesia
- mixed aphasia
- vegetative respiratory and cardiovascular disorders
- cerebral edema
- death in ½ cases
- those who survive experience severe motor sequelae and aphasia
- at ocular level:
- contralateral homonymous hemianopsia
- deviation of the head and eyeballs towards the injured side

MCA bilateral syndromes (rarely)

- tetraparesis predominates in a hemibody
- pseudobulbar syndrome
- parkinsonian phenomena
- mental disorders of dementia type

Anterior choroidal artery syndrome

- Anterior choroidal artery syndrome occurs very rarely, sometimes evolving asymptomatically. The complete clinical syndrome of anterior choroidal artery occlusion consists of:
- a contralateral hemiplegia, usually with crural predominance
- a global hemihypoesthesia
- at ocular level
- homonymous hemianopsia on the side opposite the lesion

Ischemic syndromes of the posterior cerebral artery (PCA)

- PCA strokes syndromes are manifested by:
- Pure hemisensory loss
- Loss of visual field
- **Anton syndrome**, a condition where they think they can see when they can't. Patients describe objects (although they do not see) erroneously and are not aware of this error.
- Visual agnosia - apperceptive and associative
- This refers to a lack of recognition or understanding of visual objects.
- **Balint syndrome** – it is a simultaneous triad: visual agnosia, optic ataxia, and gaze apraxia [8].

Ocular manifestations of obstructive carotid disease

- ***Transient loss of monocular vision***
- is frequently due to embolic disease and vasospasm of the ophthalmic artery and/or central retinal artery (CRA)
- transient vision loss increases stroke risk
- age >75 years old
- male gender
- history of transient ischemic attack or stroke
- intermittent claudication
- when the degree of ICA stenosis is 80-90%
- lack of collateral circulation
- ***Retinal arterial occlusion***
  - RCA can be more frequently stenosed at the level of the lamina cribrosa, RCAO being produced by: embolism, atheromatous stenosis, arteritic obliteration, hypoperfusion, vasospasm
- It is manifested by a sudden, total, painless decrease in vision, with amaurotic mydriasis.
- ***Ischemic ocular syndrome***
- Occurs through chronic hypoperfusion of the eyeball in ipsilateral or bilateral occlusive disease and manifests as anterior pole and posterior pole ischemia with venous stasis retinopathy.
- Requires treatment of ocular neovascularization with antiVEGF agent, panretinal photocoagulation.

Management of carotid obstruction [1,3]

- Control of risk factors for atherosclerosis: smoking cessation, treatment of hypertension, DM, dyslipidemia
- General measures
- Lifestyle changes: diet control, weight loss, exercise 30 minutes a day, at least 5 days a week
- Quit smoking to reduce the risk of progression of atherosclerosis and stroke
- BP control with antihypertensive drugs with maintenance of BP<140/90 mmHg, <150 mmHg in the elderly
- Drug treatment indicated for hemodynamically stable and compensated patients and patients with surgical decline for asymptomatic or symptomatic carotid stenosis: antiplatelet agents, statins
- Antihypertensive medication: statins, aspirin 75 to 325 mg/day
- If the patient had TIA or ischemic stroke - antiplatelet treatment: anticoagulants - Eliquis
- Surgical treatment – is indicated after the selection of patients after intracranial Doppler, which allows the detection of microemboli alongside the stenosis

Management in symptomatic carotid stenosis

- ***Carotid endarterectomy (CAE) in symptomatic patients with 70-99% stenosis without occlusion***
- The long-term benefit for patients with carotid occlusion is uncertain.
- EAC is recommended for patients with a life expectancy of at least 5 years
- ***Carotid artery stenting (CAS) –*** *considering age:*

Stroke Management

- ***Pharmacological prophylaxis*** of the attack with:
- oral anticoagulant drugs, for the prophylaxis of ischemic thromboembolic attack - Eliquis
- **Anticoagulants (heparin and low-weight heparin)** are used in the acute stroke patient to prevent a recurrent stroke and improve neurologic outcome.
- ***Surgical Treatment***
- Endarterectomy in transient ischemic attack, reduces the risk of stroke in patients with symptomatic or asymptomatic carotid artery atherosclerosis.
- Stenting for MCA stenosis presenting transient ischemic deficits or stroke
- Carotid stenting is a less invasive procedure than carotid endarterectomy for the treatment of carotid stenosis.
- ***Post-attack patient care in the medical unit requires:***
- Meticulous management of hypertension, diabetes, atrial fibrillation, congestive heart attack and pulmonary disease
- Thrombolysis with intravenous ALTEPLASE, a recombinant plasminogen activator. It appears to improve functional outcome at three months if given within three hours of symptom onset and if the patient meets rigorous criteria for treatment.

Systemic fibrinolysis (thrombolysis) i.v. rt PA alteplase is effective in the first 4-5 hours after the onset of the first symptom in the absence of contraindications

- limitations
- small therapeutic window
- reduced efficiency in large vessel occlusions (ICA, middle cerebral artery initially, basilar artery)

 contraindicated in patients at risk of hemorrhagic complications

- ***Arterial recanalization by endovascular approach in selected patients.***

Rehabilitation Programme

- Physical therapy
- Psychological and social rehabilitation
- Occupational therapy
- Speech therapy
- Aphasia assessment and treatment is based on expressive and receptive language abilities.
- Expressive language treatment focuses on the ability to translate thoughts and ideas into verbal expressions, written language and gestures.
- Receptive language treatment addresses the ability to understand written verbal expressions and gestures.
- Recreational therapy

Ophthalmological Disorders in Cortical Syndromes
Cerebral lobe syndromes [9]

- Causes:
- heart attacks, hemorrhages, tumors, craniocerebral trauma.

Frontal Lobe Syndrome

- At ocular level - oculo-cephalogyric disorders with conjugate movements of the head and the contralateral eyeball
- conjugate horizontal oculo(cephalo)gyrus deviation on the same side of the destructive lesion (infarcts, hemorrhages)
- conjugate horizontal oculo(cephalo)gyrus deviation on the side opposite the irritating lesion (subdural hematomas)
- horizontal intermittent oculo(cephalo)gyrus conjugate deviations on the side opposite the irritating lesion (epileptic seizures)
- vertical intermittent oculo(cephalo)gyrus conjugate deviations (postencephalitic parkinsonism)
- other signs and symptoms of frontal lobe damage (right or left).

Parietal Lobe Syndromes

- ocular – at the level of parietal lobe from the dominant hemisphere (usually left):
- contralateral incongruent homonymous hemianopsia
- other signs and symptoms of damage to the dominant parietal lobe.
- ocular - parietal lobe from the non-dominant hemisphere:
- contralateral incongruent homonymous hemianopsia
- hemiagnosia of the hemibody and hemispace on the side opposite the lesion
- apraxia of eyelid opening
- other signs and symptoms of damage to the non-dominant parietal lobe.

Temporal Syndrome

- At ocular level
- changes in the visual field

 contralateral homonymous hemianopsia (initially in the quadrant) incongruent

- homonymous quadrantanopsia, usually incongruent
- homonymous onset incomplete or limited to the upper quadrants
- visual hallucinations present during temporal epileptic seizures
- distorted perceptions of objects
- objects can appear near/far or unreal.

Occipital Syndromes

- At ocular level:
- changes in the visual field
- total, incomplete or partial contralateral congruent homonymous hemianopsia in the upper quadrant with respect to macular vision in unilateral lesions of the occipital pole
- bilateral central hemianopsia in bilateral lesions
- temporal crescent in unilateral lesions of the anterior occipital lobe
- cortical blindness with preservation of pupillary reflexes and loss of the eye closure reflex to bright light in bilateral occipital lobe lesions
- visual anosognosia
- visual illusions with metamorphopsia, more common in occipitoparietal or occipitotemporal lesions

- elementary visual hallucinations, which occur in lesions of the calcarine cortex
- complex visual hallucinations, which appear in lesions of the temporal visual areas of association
- visual agnosias are associated with alexia and homonymous hemianopsia, in occipitotemporal lesions
- colour agnosia
- cortical blindness with tonic deviation of the eyes, oculoclonic seizures and nystagmus in occipital epilepsy.

Balint Syndrome
- at ocular level - simultaneous
- visual agnosia
- optic ataxia
- optic apraxia
- visuospatial disorientation
- the inability to make voluntary movements to visual stimuli
- paralysis of visual fixation.

Ocular pathology secondary to stroke in the cerebral vascular system is extensive and very serious, associated with decreased/ loss of vision and requires urgent diagnosis and treatment.

Ocular Manifestations in Comatos Syndrome
Eyelid changes
- In superficial coma, the muscle tone of the eyelids is preserved, so that after passive elevation of the upper eyelids, they tend to close quickly.
- In deep comas, the muscle tone of the eyelids is greatly reduced, so that after passive elevation of the upper eyelids, they tend to close slowly.
- Blinking movements when performing auropalpebral reflexes are maintained if the brainstem is not damaged and disappear if it is affected.
- Blinking when performing the corneal reflex is absent in ponto-mesencephalic lesions [10].

Pupillary Signs
- **Equal and reactive pupils** indicate coma from metabolic, toxic causes, except for anoxia, anticholinergics, and botulinum toxin which cause fixed mydriatic pupils, and narcotics produce miotic, slowly reactive pupils.
- **Equal, non-reactive pupils** – post-traumatic or severe brain process
- **Unequal pupils:**
- mydriatic pupil + absence of pupillary constriction to light in: head trauma with subdural, extradural hematoma, intracerebral hemorrhage, intracranial hypertension
- Horner's syndrome
- bilateral mydriatic pupils, fixed, 7-10 mm: bulbar lesions, anoxic encephalopathies, drug overdose (barbiturates, atropine), hypothermia, transtentorial cerebral hernia, cerebellar tonsil hernia.
- **Punctiform pupils:** in severe pontine lesions.

Occlusive Syndrome of the Vertebro-Basilar Arterial System
The vertebrobasilar system includes:
- the vertebral artery starting from the subclavian artery
- the basilar artery formed by the union of the two vertebral arteries
- right-left posterior cerebral arteries (PCA) - are terminal branches of the basilar artery
- The PCA makes the junction with the posterior communicating artery through which the vertebrobasilar circulatory system communicates with the carotid one.

Insufficiency of Vertebrobasilar Circulation
Vertebrobasilar insufficiency - VBI - or moderate, reversible vertebrobasilar circulatory ischemia is caused by a decrease in blood flow (with secondary ischemia) in the posterior circulation of the brain at the level of the spinal bulb, cerebellum, pons, midbrain, thalamus and occipital cortex.
- **IVB is defined as transient ischemia of the vertebrobasilar circulation**
- symptoms due to IVB vary depending on the part of the brain affected by the ischemia
- the incidence of IVB increases with age and affects men 2 times more than women [11,12].

General signs and symptoms
- vestibular symptoms: vertigo, can in many cases be the only symptom of low blood flow in the VB territory
- dizziness and chronic imbalance
- vomiting
- nystagmus
- less often: mental confusion, headache, hearing loss, paresthesias, dysarthria, tinnitus
- Neurological manifestations:
- recurrent alternating hemiplegia, paroxysmal headaches, dizziness, vertigo
- dysarthria, anartria
- cardiorespiratory vegetative phenomena, thermal dysregulation
- mental disorders: apathy, amnesia, intellectual deficit accompanying motor, sensory disorders, aphasia depending on the extent of the focus of ischemia
- possible symptoms secondary to a lateral medullary syndrome by stroke in the vertebral arteries or the irrigating territory in the brainstem: difficulty in swallowing and hoarseness, dizziness, nausea, vomiting, nystagmus, imbalance and incoordination of walking.
- Ocular manifestations
- diplopia and signs of damage to CN III, IV, VI with oculomotor paralysis associated with conjugate paralysis of laterality or verticality
- "blurry vision" seconds, minutes or persistent for days/weeks up to permanent decrease in vision which can progress to amaurosis
- visual field defects
- visual gnostic disorders
- pure-rare cortical blindness (+ visual hallucinations, sign of thrombosis), spatial-temporal disorientation, visual anosognosia

- transient loss of vision and visual hallucinations are susceptible to thrombosis
- lateral deviation of the gaze, downward deviation of the eyes, vertical deviation may suspect brain stem suffering
- internuclear ophthalmoplegia
- see-saw nystagmus
- fixed gaze
- visual agnosia (apperceptive and associative)
- the patient ignores the deficiency
- Balint syndrome with visual agnosia, optic ataxia, gaze apraxia
- colour perception disorders
- visual field defects - altitudinal hemianopsia
- contralateral homonymous hemianopsia congruent with sparing of the macula
- palpebral disorders; reflex blepharospasm, bilateral lagophthalmos
- pupillary disorders
- bilateral mydriasis in coma, bilateral miosis (in coma it means diencephalic or pontine damage, coma with barbiturates)
- correctopia (mesencephalic lesion)
- anisocoria (expansive process or hematoma)
- corneal reflex abolished or absent (suspect brainstem disease) [13]
- Treatment
- treatment of risk factors: diabetes, hypertension, atherosclerosis
- stop smoking, alcohol
- to prevent occlusion - Eliquis oral anticoagulant treatment (if bleeding is excluded on imaging)
- for the treatment of vertebrobasilar stenosis - intracranial angioplasty

Basilar Artery Occlusion - BAO

- The basilar artery is the most important artery in the posterior circulation. It is formed at the pontomedullary junction by the confluence of both vertebral arteries
- The terminal branch of the basilar artery is the posterior cerebral artery (PCA), which supplies the midbrain, the thalamus, and the medial part of the temporal and occipital lobes.

• Symptoms present in BAO:

o motor deficits with hemiparesis or tetraparesis and facial paresis
o dysarthria and impaired speech
o dizziness, nausea and vomiting
o headache
o altered emotional reactions
o asymmetric hemiparesis or quadriparesis
o facial weakness, dysphonia, dysarthria, dysphagia
o at ocular level:

- pupillary abnormalities
- oculomotor signs: ipsilateral abducens paralysis, ipsilateral conjugate gaze paralysis
- internuclear ophthalmoplegia
- one-and-a-half syndrome

Ophthalmological Manifestations in Cerebral Venous Thrombopathies

Cavernous sinus thrombosis

- It can be produced by the spread of infection from acute or chronic foci of the cephalic extremity (furunculosis of the face, orbital, sinus, tonsillar, dental, ear infection) [1,14].
- They have also been described in facial trauma, head trauma, skull base tumours, polycythemia vera.
- Patients present

- headache
- nausea
- vomiting
- drowsiness
- possible fever, chills, tachycardia with a **picture of generalized sepsis**

- Initially unilateral eye signs may become bilateral

- **periorbital edema (can be one of the earliest signs)**

 palpebral edema mainly affects the upper eyelid; the eyelids become colored, and hard cords are felt on palpation (the palpebral veins are affected)

- ptosis
- conjunctival chemosis
- exophthalmia with serosanguineous chemosis, the eye being projected forward, with rapid lateral or vertical deviation with CN III, IV, VI damage
- movements of the eyeballs are limited, and its compression is painful
- complete internal and external ophthalmoplegia by involving the common oculomotor, external oculomotor and pathetic nerves
- initially, there is an excess of tears, but later their quantity decreases
- paresis of CN VII
- decreased visual acuity, as a result of increased intraocular pressure and pressure exerted on the optic nerve and central retinal artery
- increased intraocular pressure
- corneal anesthesia, corneal ulcerations
- diminished pupillary reflexes, Claude Bernard Horner syndrome
- papillary edema and venous stasis retinopathy or unilateral premature optic atrophy and blindness, dilatation of retinal veins, retinal hemorrhages
- hypoesthesia or hyperesthesia in the dermatome of the ophthalmic nerve
- **signs and symptoms in the contralateral eye** are highly suggestive of cavernous sinus thrombosis, but the process may remain localized in only one eye (the initially affected one)
- **signs of meningeal involvement (stiffness, neck stiffness** – inability to bend the head forward due to stiffness of the cervical muscles), Kernig and Brudzinski's sign (it is the involuntary movement of raising the legs when the patient's head is bent).
- **Signs and systemic conditions suggestive of sepsis:** chills, fever, delirium, shock, coma.

Conclusions

Carotid artery stenosis is present in 10% of people over 60 years of age, of which 5% may have a stroke in the next 5 years, with a higher frequency in men. Carotid occlusion presents multiple clinical signs: general, cerebral, ocular in relation to the location of the arterial obstruction. The most common strokes are in the territory of the middle cerebral artery and the posterior cerebral artery. Medical treatment of carotid obstruction requires prophylactic and curative treatment of hypertension, diabetes, atherosclerosis, anticoagulant medication. Surgical treatment (carotid endarterectomy, carotid artery stenting) is necessary in selected cases. Systemic fibrinolysis is effective in the first 4-6 hours after the onset of the first symptom. Rehabilitation program is necessary for stroke patients. In cortical syndromes, the ocular manifestations are variable depending on the location of the stroke in the frontal, parietal, temporal, occipital lobe. Vertebrobasilar ischemia caused by decreased blood flow in the posterior circulation of the brain has variable symptoms depending on the location of the ischemia. Thrombosis of the cavernous sinus is a serious condition produced by the spread of infection from infectious processes of the cephalic extremity and is manifested by symptomatology generated by the septic state associated with unilateral ocular inflammatory signs that rapidly expand bilaterally (early periorbital edema with serosanguineous chemosis, exophthalmia that rapidly becomes lateral or vertical, CN III, IV, VI, VII damage, papillary edema, signs of meningeal involvement.

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