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Research Article

CURRENT VIEWS ON THE ETIOLOGY, DIAGNOSIS AND TREATMENT OF VERTEBROBASILAR ISCHEMIA

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ABSTRACT

In norm, the vertebral arteries branch from the subclavian arteries, passing cranially through the rib and transverse foramen of the sixth to second cervical vertebrae. They enter the skull through the greater occipital foramen, joining at the pontine and medulla oblongata to form the basilar artery, which divides afterwards into the two posterior cerebral arteries at the top of the pontine. At the base of the brain, the carotid and basilar systems connect to form the circle of villisia.

KEYWORDS

Vertebrobasillary insufficiency, ischemia, atherosclerosis Villizyev ring.

INTRODUCTION

Atherosclerosis is the most common cause of haemodynamic changes leading to the development of IBS. Other frequent causes are: embolism, large vessel atherosclerosis and arterial dissection. Less frequent causes are migraine, fibromuscular dysplasia, coagulopathies and drug abuse.

Review studies examining the epidemiology of IAP have shown that the prevalence of atherosclerosis and blockage of the vertebrobasilar circulation differs according to the sex and age of patients. It is more frequently observed in males after the fourth decade of life, arterial hypertension, obesity and smoking are considered major risk factors[1]. Vertebrobasilar insufficiency is defined as transient ischemia of the vertebrobasilar circulation. The most common symptoms are dizziness, vertigo, headaches, vomiting, diplopia, blindness, ataxia, balance problems, and weakness on both sides of the body. This arrangement of collateral circulation can ensure adequate cerebral perfusion even in the event of occlusion of the main

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vessel. The onset and duration of symptoms may vary depending on the aetiology. The etiology may vary: One common cause is basilar artery thrombosis which may precede a transient ischaemic attack several days or weeks before occlusion and are seen in half of patients who have suffered a vertebrobasilar stroke [2]. Embolic events causing sudden symptoms without a prodromal period or temporary occlusion of a single vertebral artery with collateral insufficiency due to progressive atherosclerosis.

Atherosclerosis: the most common vascular disease affecting the vertebrobasilar system[1]. Which affects the large vessels causing them to narrow and occlude, subsequently leading to stenosis at the origin of both vertebral arteries to cause vertebrobasilar ischaemia. Even with vertebral artery occlusion, collaterals (Circle of Villisia) can prevent ischaemia. And in this case, hindbrain ischaemia probably develops in combination with carotid artery lesions (often at the carotid bifurcation site), vertebral artery stenosis and intracerebral disease.On examination, it is found that the circle of villis is intact in only 50% of patients [9].

Lipohyalinosis: affects small vessels, eventually causing occlusion. It often occurs in association with hypertension. Lipohyalinosis also causes weakening of vessel walls and vessel rupture, leading to focal haemorrhages. Almost all intracerebral haemorrhages are from such rupture of small penetrating vessels [10].

Embolic occlusion of the vertebrobasilar system: rare, emboli usually originate from the aortic arch, subclavian artery and vertebral arteries. Emboli usually occlude the basilar artery.

- Dissection of the vertebrobasilar artery .

Pathophysiology of IAP and vestibular symptoms

Many studies have reported the complexity of the irrigation pattern of the vestibular system and suggested that this fact plays an important role in the pathophysiologic mechanism of IAP manifested by labyrinthine symptoms. The vestibular system is supplied by:

-very small penetrating vessels branching from the main artery supplying blood to the vestibular nuclei ;

- the internal auditory artery originating from either (a) the anterior inferior cerebellar artery (80-85%) or a vascular loop from the posterior inferior cerebellar artery (PICA), which is a branch of the vertebral artery (15%) [7]. Given that the labyrinth branches are smaller and receive less collateral irrigation, it is possible that the labyrinth should be more affected by atherosclerotic blockade of the vertebrobasilar arterial system. On the other hand, the cochlea receives collateral irrigation from the carotid artery, which supplies blood to neighboring areas of the stony bone. This special feature protects the cochlea from ischemic symptoms in case of vascular insufficiency [8]. According to other scientistsi the prevalence of positional vertigo and imbalance, as well as clinical and electronystagmography data in two groups: one with changes in the vertebral arteries on magnetic resonance angiography (stenosed or hypoplastic vertebral arteries) and one without changes, where no differences were found between the two groups [4]. These results suggested that clinical symptoms were caused by occlusion or obstruction of small vessels in more distal regions. Individual episodes of vertigo could be a consequence of transient ischemia of the vestibular labyrinth due to the peculiarities of its terminal circulation without collateral vessels [12]. Thus, the vestibular system may be more sensitive in detecting differences in action potential secondary to ischemia. Ischemia can affect both peripheral and

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central structures of the vestibular system. Studies by other authors have described two mechanisms by which episodes of ischemia may cause isolated attacks of vertigo: First decreased blood flow to the vestibular nuclei or the root of the entrance zone of the prevertebral-cochlear nerve and second direct ischemia of the labyrinth[13].

Risk factors associated with stroke

- Age;
- Hypertension;
- Alcoholism;
- Coronary heart disease and other diseases;
- Diabetes mellitus;
- Substance abuse;
- Family history of stroke;
- Gigantocellular arteritis;
- Obesity and physical inactivity;
- Previous stroke history;
- Race;
- Smoking;

Clinical features: The most common symptoms are dizziness, vertigo, vertigo, headaches, vomiting, diplopia, blindness, ataxia, balance problems, and weakness on both sides of the body. Some authors state that lower extremity weakness associated with leg imbalance and ataxia, oculomotor nerve palsy, and/or oropharyngeal dysfunction are the most common symptoms of IBS. Ischemia in the posterior circulation rarely causes only one symptom at a time throughout the course of the disease; in most cases, there is a combination of different symptoms presenting simultaneously. Caused by disruption of the vertebrobasilar blood supply, the medulla oblongata, cerebellum, pontine, midbrain, thalamus, and occipital cortex are affected. This leads to a number of clinical syndromes and is primarily due to atherosclerosis [1,3].

- Diagnosis. The diagnosis can be made on the basis of a thorough clinical examination, but the complexity of the signs requires a thorough and detailed neurologic examination. General findings:
- Abnormal level of consciousness.
- Hemiparesis or quadriparesis (usually asymmetric).
- Bulbar manifestations (facial weakness, dysphagia, dysarthria, dysphonia).
- Pupillary and oculomotor abnormalities are also common.
- Physical findings can be very varied and complex depending on the exact location of the affected brain:
- The diverting nucleus, horizontal gaze center (located in the paramedian pontine reticular formation) and medial longitudinal fasciculus lesion cause oculomotor symptoms [10]. This can lead to:
- Ipsilateral lateral gaze palsy.
- Concomitant gaze palsy.
- Eye twitching.
- Midbrain syndromes:
- Vertical gaze paralysis.
- Paralysis of the third cranial nerve.
- Pontine syndromes:
- Tremor, ataxia, and mild hemiparesis.
- Paralysis of horizontal gaze.
- Paralysis of the VI and VII cranial nerves.
- Medullary syndromes:
- Loss of facial pain and temperature sensitivity (ipsilateral).
- Horner's syndrome.

Clinically identified by the following syndromes :

-Lateral medullary or Wallenberg syndrome(usually from occlusion of the vertebral artery, sometimes due

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to occlusion of the posterior inferior cerebellar artery; vestibular lesions cause nausea, vomiting, and dizziness. Ataxia due to cerebellar lesions[11];

- Horner's syndrome due to lesion of descending sympathetic fibers. which is accompanied by decreased corneal reflex due to damage to the descending spinal pathway, nystagmus, nystagmus, hypacusia, dysarthria, dysphagia and paralysis of the palate, pharynx and vocal cords,loss of taste on the posterior third of the tongue; early diagnosis is important as edema can cause compression of the brainstem.

- Locked-in syndrome, which will be caused by an infarction of the upper ventral pontine. Usually dramatic and sudden, causing quadriplegia with preserved consciousness, internuclear ophthalmoplegia where horizontal gaze paralysis will be seen. In younger patients it may be caused by multiple sclerosis;

- Hemiparetic syndrome is ipsilateral gaze palsy and internuclear ophthalmoplegia. which causes inability to move the ipsilateral eye and retraction of only the contralateral eye with nystagmus, as well as Millard-Gubler syndrome (ventral pontine syndrome, diplopia with facial paresis and contralateral hemiparesis). Upper basilar syndrome(sudden confusion and amnesia, visual symptoms, usual embolism);

-Raymond-Sestan syndrome where the upper dorsal bridges are affected, ipsilateral ataxia and tremor, weakness of mastication. contralateral loss of sensory modalities, may be accompanied by facial weakness and hemiparesis;

- Fauville syndrome (Inferior dorsal pontine lesion. Ipsilateral paresis of the entire face. Loss of ipsilateral horizontal gaze. Contralateral hemiplegia with preservation of the face;

- Weber syndrome (Ventral midbrain affected, ipsilateral mydriasis, III cranial nerve palsy and ptosis. contralateral hemiplegia)

- Benedict's syndrome(dorsal midbrain lesion. Ipsilateral oculomotor effects as in Weber syndrome. Contralateral tremor, ataxia, or chorea. Occlusion of the posterior cerebral artery, most commonly causing occipital lobe infarction with hemianopsia and preservation of the yellow spot. Variable effects from thalamic syndrome to variant cortical visual disturbances.

METHODS: Diagnosis of IBS by neuroimaging. Patients with suspected transient ischemic attack or vertebrobasilar stroke should undergo neuroimaging studies. Although arteriography is considered the most important study for this purpose, the risks associated with this study in patients with IAP should be considered. Many patients choose not to undergo this test after reading about the risks of the procedure in the informed consent. The major complications of arteriography are: localized arterial catheter complications; regional low blood flow; and stroke.

- Basic blood tests including OAC, COE, blood chemistry, coagulation, and lipid profile.

- Screening for hypercoagulability before age 45 years, for example:

- Lupus anticoagulant and anticardiolipin antibodies.

- Antithrombin III deficiency.

Visual examinations:

o Immediate brain imaging with unenhanced CT is recommended for all people with suspected acute

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stroke if they have an indication for thrombectomy (including patients with stroke involving both the basilar and posterior cerebral arteries, provided that brain tissue salvage is available.)), and with known bleeding tendency, depressed level of consciousness, unexplained progressive or fluctuating symptoms, or with anticoagulant therapy.

o - In patients with suspected stroke in whom there is no indication for immediate brain imaging, unenhanced CT should be performed within 24 hours.

o - MRI is better than CT, especially for ischemia, demyelination, tumors, and vascular disease.

o - Diffusion-weighted MR sequences can be used to show the volume of the infarct focus to assess suitability for thrombectomy;

o - MR angiography is usually performed in conjunction with MR imaging.

o - Newer MRI techniques allow even better definition of pathology.

- ECG:Mandatory in all patients with stroke.

- 20% of stroke patients have arrhythmias and about 2% have had previous myocardial ischemia (indicative of cardiovascular disease).

- Echocardiography:

- Identifies valve defects, vegetations, and other sources of emboli, especially in younger patients with main artery occlusion.

- Cerebral angiography:

- Used less because of noninvasive visualization.

Prevention depends on the cause; however, prevention strategies include and supportive skin, nutrition, and patient safety. More socialization with people and support from significant others and family members. Specifically includes physical therapy, nursing, speech and language, and safety skills, swallowing assessment, and caregiver and family education. Warfarin for atrial fibrillation. Better adherence to recommendations will prevent stroke. Treatment of hypertension, hyperlipidemia.

CONCLUSIONS

Thus, IAP should be suspected in patients with risk factors for ischemia and vestibular symptoms. Each diagnostic test has its pros and cons, so each patient should be treated individually to avoid possible complications.

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