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INVITED LECTURE

TREATMENT OF PATIENTS WITH PERIPHERAL ARTERIAL DISEASE

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ABSTRACT

Many risk which factors lead to development of arteriosclerotic arterial disease , on one hand and significant possibilities for development of collateral circulation, on the other hand, confirm the possibilities of prevention and need for timely treatments of patients with peripheral arterial disease (PAD). PAD prevention should start as early as possible, through education o population about healthy nutrition and appropriate physical activities, which is programmed and supervised.
In treatment of asymptomatic and mild symptoms cases as well as post revascularisation cases, along with drug therapy, physical therapy has an important role: it leads to the subjective improvement, it enhances development of collateral circulation, increases claudication distance. By all these, physical therapy contributes to the limb preservation and to the quality of life. Hyperbaric oxygenotherapy (HBOT) is important in patients with arterial occlusion followed by critical ischemia when oxygen enters the tissue in only way possible – dissolved in plasma.

Keywords: *peripheral arterial disease, treatment*

INTRODUCTION

Peripheral arterial disease primarily refers to atherosclerotic arterial disease, which is underlying cause in 90 % of diseased.

Atherosclerosis is systemic, chronic, progressive, metabolism-degenerative, inflammatory disease of arterial wall. It is a diseases with highest incidence and prevalence and, at the same time, the most researched pathogenetic and clinical entity on the beginning of third millennium.[1]

Atherogenesis is a dynamic patho-physiological group of multifactor initiated degenerative and regenerative processes in the arterial wall which lead to the following:

- Increase in thickness and rigidity of the intima media complex,
- Reduction of the lumen (stenosis) and finally
- Total obstruction of artery (occlusion) with distal ischemia[1]

Coronary, carotidal, peripheral atherosclerotic disease (PAD) is the main cause of morbidity and mortality in the industrialized countries. Recently has the WHO statistics shown that such tendency is to continue and progress in the new millennium, with younger and younger population group being affected. [1,2]

PERIPHERAL ARTERIAL DISEASE (PAD)

Most commonly, atherosclerosis is manifested as coronary, carotidal, peripheral arterial disease (PAD) and aneurismatic disease of the abdominal aorta.

Changes of arterial trunk, in form of stenoses and occlusions, inevitably lead to slower and reduced blood flow. Laminar blood flow is disturbed and turbulence can be caused y any local substrate : plaque, ulcers, dilatation and aneurismatic expansions, arterial wall rigidity etc. Total blood flow interruption makes the limb dependant on the collateral circulation. Arterial pressure bellow the occlusion site is reduced as the flow itself.

Peripheral arterial disease (PAD) is most commonly caused by atherosclerosis. In a high percentage it is asymptomatic and detected by chance. If manifested, it is most often in the form of intermittent pain-claudiacation pain. As disease progresses, pain in present in rest, which indicates critical ischemia and limb is in danger (1-2% diseased from PAD). Emboli and thrombosis are rare in arterial blood vessels.

Prognosis and survival of patients suffering PAD are greatly influenced by existence of coronary and cerebral arteries.[3]

COLLATERAL CIRCULATION

Collateral circulation (vasa collateralia) are side branches which present an alternative path for the blood flow and, when needed, these enable collateral circulation.[4] Collateral blood vessels can be already existing blood vessels or newl formed arterial channels. According to the type of the obstacle by pass and filling of postoccluisve part of chief arteria, collateral circulation can be: primary and secondary; native or functional; direct and indirect. [4,5,6]

The first type of collateral circulation presents anastomosis between branches of the same arteria, which origin from the arteria above and bellow the occlusion site and join together.

The second type of collateral circulation is the anastomosis of the other arteria with branches of the occlued one, which are derived from it bellow the occluded segment.

Coomunitations and by passses are created from both visceral and parietal arteria of abdomen and pelvis as well as conductive and nutrient arteries of the lower limb.

When blood flow through the chief blood vessel is compromized by significant stenosis or occlusion, the limb function and surrvival will depend entirely from the collateral circulation. [7,8,9]

Degree of functional deficit which develops after occlusion will depend on: occlusion localisation, bellow occlusion tissue metabolic needs and extent of the remaining blood flow through the collateral vessels. [7]

For development of collateral vessels and preservation of limb, more favourable are situations of short segment occlusions and gradualy developed occlusions. With gradual narrowing of arterial lumen and in activities, gradient pressure develops and distal flow is insifficient so it could ensure metabolic reserves of the tissue, collateral circulation develops and proessure in collateral blood vessles becomes higher than in blood vessles distal to stenosis. This process of collatral blood flow development is not purely mechanical then autoregulation process with involvement of several factors. These factors are attributed various level of importance:

1. oppening and development of collateral circulation are initiated by hipoxia and intermediar roducts of anaerobic metabolism and lately, more importance is given to release of potasium ions, hyperosmolarity and anorganic phosphates.
2. ground for autoregulation mechanism whihc starts collateral blood vessels opening are haemodynamic factors (pressure above the occlusion site, pressure gradient and perihperal resistance)
3. ischaemia of the limb bellow occlusion by reflex causes vasodilatation and development of collateral vessels.

Huge potential capacity and wide network of communication between arteries of abdomen, pelvis and lowerlimbs, explains the ability to compensate for occlusive changes on chief arteries and sometimes discrepancy between degree of occlusive lession and foot nutrition state. If the lession is more proximal, degree of ischaemia and vital endengerement are lower. However, flow reduction on some leves which Salmon calls „dangerous zones" can lead to critical ischaemia distal to occlusion site due to insufficient number of by passess. This is the case with poplitel arteria, especially if occlusion affects distal third of it.

DIAGNOSTICS

For assessment of patient with PAD, beside detailed history and clinical examination, the following diagnostic procedures are importants: lab tests (blood sugar, lipid status), blood pressure control, pedobrachial index, segmental pressure measurement, measurement of claudication distnace, (six minutes walk test), transcutaneous oxymetry, tissue flow, CDS. Uop completion of diagnostics every patient will fall into on of Fontaineu stages, whihc will help us to opt for further diagnostic and treatment. PAD stages are: I asymptomatic, II intermittent claudication, (-II a walk distance more than 200 m (100m); -II b walk distance more than 200 m (100m), III pain in rest, IV ulcer or gangrene.

Short claudication distance and chronic critical ischaemia require further diagnostics: contrast angiography, CT angiography, MR angiography.

PREVENTION, TREATMENT, REHABILITATION

Prevention, treatment and rehabilitation are by no means separate segments in treatment of the PAD. Depending on phase, some of the segments are dominant or more represented. World guide recommendations are giving advantage to prevention,[10] which is always more efficient, best for diseased (client) and cheapest .

Prevention of atherosclerotic disease understands work on all risk factors: cessation of smoking, blood pressure control (change in life style, drug therapy: beta blockers, tiasides, ACE inhibitors), lipid status control, (adequate nutrition, taking food rich in omega 3 fat acids, physical activity and body mass control, drugs against lipids), physical activity, (BMI and waist circumference control), body mass decrease, drug therapy in case of enormous BMI value), control of diabetes (life style change and drug therapy, life style change and drug therapy, risk factors management (inadequate nutrition, abdominal obesity, insufficient physical activity all precede type II diabetes).

Drug therapy which is used in prevention of PAD

- anti aggregation therapy: aspirin 75-325 mg daily, continuously (level of evidence I A), clopidogrel in combination with aspirin or as monotherapy,
- anticoagulant therapy in chronic atrial fibrillation and following heart attack, so that INR is between 2 and 3. (level of evidence I A)

Treatment of PAD through improvement of circulation we alleviate patient's subjective symptoms, prevent or postpone ischemic event and thus prevent limb amputation.

Conservative treatment is indicated when disease is in early stage, stages Fontaineu I and II a: claudiaction distance longer, Doppler index less than 0,9 but without symptoms. Cilostazol 2x100 mg daily is efficient in increasing claudication distance in diseased who have intermittent claudication (Level of evidence A). Pentoxifylline 400 mg 3 times daily, can be useful for claudication pain, as secondary line to cilostazol.

Physical therapy – most important are therapeutic exercises which are programmed to target muscle groups immediately bellow the occlusion, in order to support development of the collateral circulation. Interval training which patients with PAD conduct under supervision and following individual plan, walking 30-60 minutes once a day, at least 5 times a week, at least 12 weeks. (Level of evidence I A)

(The usefulness of unsupervised exercise programs is not well established as an effective initial treatment modality for patients with intermittent claudication. (Level of Evidence: B)

As for physical modalities, vacuum compression therapy is recommended, with predominantly negative pressure, interferential current for sympaticus blockage and locally, on affected limb, low frequency magnetic field.

As patient has short walking distance, chronic critical ischemia or pain in rest (Fontaine II b, III and IV), for further treatment is necessary to have morphological display of affected arteries which, beside CDS understands angiography and CT or NM angiography.

Based on findings, a treatment decision will be made about the revascularisation procedure (surgical intervention, percutaneous transluminal angioplasty).[11] Following the successful revascularisation procedure, treatment will continue with drug treatment and physical therapy which is individually programmed and includes a close follow up.

If there is no possibility for revascularisation procedure or the revascularisation procedure was not successful, the drug therapy will continue (vasoactive cocktails, parenteral administration of prostaglandins with limited effect,[11] physical therapy if indicated , depending on limb condition and general status of a patient.

HBOT is indicated in this phase (Fontaine II b, III and IV) prior and after surgical treatment or in situation when revascularisation procedure is not feasible, before the tissue suffers severe damage due to ischemia.

If the limb gangrene developed, it is necessary to perform limb amputation and then continue with drug treatment and prosthetic rehabilitation.

CONCLUSION

Timely prevention and treatment of PAD are precondition for prevention of complications and most common are lower limb amputation, decreased working and life ability and impaired quality of life of patients with PAD.

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