Management of Chronic Peripheral Arterial Occlusion Disease

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ABSTRACT

The chief complaint of chronic peripheral arterial occlusion disease is intermittent claudication, or calf pain during exercise that is relieved by resting. As the disease advances, pain occurs even during resting. The main cause for this disease is atherosclerosis.

Clinical evaluation of patients comprise of questionnaire, exploration of atherosclerosis risk factors such as smoking, hyperlipidemia, diabetes mellitus, hypertension, etc. Pulse examination, ankle-brachial pressure index (ABPI) measurements and leg segmental blood pressure assessments are integral examinations in such patients. Another important test is functional assessments using the treadmill, or other tests to cause hyperemia.

The chief management is to prevent common morbidity and mortality due to atherosclersosis such as coronary heart disease and its complications, as well as cerebrovascular disease and its complications. Management is also targeted towards preventing the progress of peripheral arterial occlusion disease.

Specific management for peripheral arterial occlusion disease that is clearly beneficial to improve symptoms is physical exercise. Drugs such as pentoxyphylline, naftidrofuryl, buflomedil, and cilostazol are of little benefit only. Aspirin is useful to prevent cardiovascular, but is useless to improve symptoms.

Prior to offering interventional procedures (angioplasty and/or stent insertion) or surgery, the following issues should be contemplated: 1) the patient does not respond to physical exercise and risk factor modification, b) there is severe disability, c) there is no longer other disease that limits activity, and d) the morphology of the lesion is in line with the intervention, with a low risk and high probability for initial and long-term success.

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INTRODUCTION

It is estimated that peripheral arterial disease as a manifestation of systemic atherosclerosis occurs among 12% of the population. The prevalence increases to 20% among those above 70 years of age. Since peripheral arterial disease and coronary heart disease occurs simultaneously, it is not surprising that 80% of mortality due to peripheral arterial disease is caused by cardiovascular complications, 63% due to coronary heart disease, 9% due to cerebrovascular disease, and 8% due to other cardiovascular complications such as rupture of aneurisms.[1,2,3]

The main symptom of peripheral arterial occlusion disease is intermittent claudication, with an incidence rate of 2% per year among populations above 65 years of age.[3] Intermittent claudication is diagnosed based on a history of calf pain during exercise that is alleviated by resting. Other diagnostic methods include interview (questionnaire), clinical assessment, and non-invasive examinations. The degree of symptom is difficult to assess. Patients with severe peripheral arterial occlusion disease may not demonstrate symptoms if another cause is limiting the patient's physical activity. Patients with mild peripheral arterial occlusion disease may demonstrate symptoms due to a high level of physical activity.[4]

CLINICAL EVALUATION OF CHRONIC PERIPHERAL ARTERIAL OCCLUSION DISEASE

Chronic arterial insufficiency of the lower extremities causes two types of unique pain: intermittent claudication and ischemic resting pain. Intermittent claudication refers to exercise associated pain or discomfort. The degree and extent of pain determines the location of pain and the level of exercise required to cause it. Femoral, popliteal, or tibial lesions cause cramps in the calf muscles. More proximal occlusions (at the aortoiliac arteries) cause pain associated sensation of

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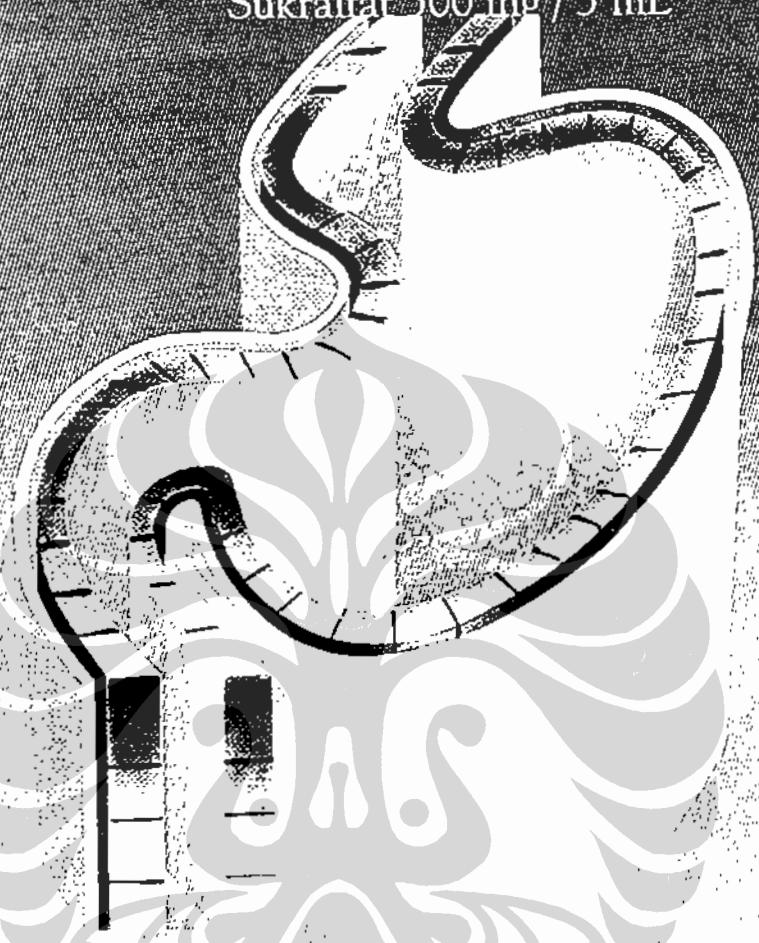

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weakness at the hip, buttocks, or the thighs. Severe claudication of the legs is usually accompanied with numbness, associated with severe infrapopliteal occlusion most commonly due to Buerger's disease (thromboangitis obliterans), and is rarely due to atherosclerosis.[5]

Generally, it is important to record:

1. The location of pain or discomfort
2. The duration of complaint
3. Whether the pain aggravates or improves with time and whether it responds to conservative treatment
4. How far the patient could walk before feeling complaints
5. How long it takes for the pain to subside after the patient stops walking
6. The position required to alleviate the pain (standing, sitting, or lying down during resting)
7. Whether the pain recurs after the same activity is conducted for the same time and distance.

Such information, in adjuvant with physical examination, is useful to localize lesions, eliminate differential diagnoses, and act as a basis for the evaluation of further changes.[5]

It is important to find risk factors associated with peripheral and systemic atherosclerosis. Peripheral arterial disease due to atherosclerosis rarely occurs without risk factors. On the other hand, it often occurs when there are one or more risk factors (smoking, diabetes mellitus, hypertension, hyperlipidemia, and hypercoagulable states). Age also acts as a risk factor, since peripheral arterial disease is often found among patients above 50 years of age.[5]

It is also important to determine the underlying disease that aggravates claudication, such as anemia, polycythemia, heart failure, or arrhythmia, and chronic pulmonary disease with hypoxemia. It should also be noted that atherosclerosis is found in several locations in one patient. Thus, peripheral arterial disease is often also accompanied by coronary heart disease or carotid artery disease or visa versa.[5]

Differential Diagnoses for Chronic Peripheral Arterial Occlusion Disease

1. Venous claudication occurs after iliofemoral thrombosis with poor recannalization and collaterals. The pain occurs during activity because of increased venous pressure.
2. Chronic compartment syndrome found in athletes with hypertrophy of the thigh muscles, disturbing venous flow, resulting in pain in the thighs. Complaints are alleviated by lifting the lower limbs.
3. Peripheral nerve pain may be caused by nerve root compression by herniated discs or the presence of osteophytes, and pain follows dermatomal distribution. Pain is felt on the back of the calf and not within the calf.
4. Secondary spinal cord compression due to osteoarthritis of the bony lumbar spine
5. Osteoarthritis of the upper limb
6. Other non-arterial causes, such as spasm of the thigh muscles
7. Other arterial causes: Buerger's disease, congenital coarctation, fibromatosis, or Takayasu's arteritis.[5]
8. Peripheral emboli due to heart disease (atrial fibrillation, mitral valve disease), aortal aneurysm.
9. Popliteal cyst

PHYSICAL EXAMINATION FOR CHRONIC PERIPHERAL ARTERIAL OCCLUSION DISEASE

Examination is not limited to the lower extremities alone, but should be directed to the circulatory system as a whole. It should be able to assess hypertension, heart disease, carotid murmur, lung disease, anemia, or abdominal aortal aneurysm. The skin of the limbs, particularly the foot and the nails, should be examined to discover color and temperature changes and other abnormalities such as swelling, scar tissue from previous ulcers, and trophic changes associated with chronic ischemia such as dry and thin skin, hair loss, loss of subcutaneous fat, and thickened fingernails. Such abnormalities and signs of Buerger's disease are often mild or absent in claudication, which instead demonstrate muscle atrophy due to insufficient activity, reduced hair growth, and slow and thickened nails, due to atherosclerosis peripheral arterial disease.[5]

Table 1. Classification of Peripheral Arterial Disease: Fontaine Staging and Rutherford's Categories

Fontaine		Rutherford		
Stage	*Clinical condition*	*Degree*	*Category*	*Clinical condition*
I	*Asymptomatic*	*0*	*0*	*Asymptomatic*
IIa	*Mild*	*I*	*1*	*Mild claudication*
IIb	*claudication*	*I*	*2*	*Moderate*
	Moderate	*I*	*3*	*claudication*
III	*claudication-*	*II*	*4*	*Severe*
	berat	*III*	*5*	*claudication*
IV	*Resting ischemic pain Ulceration or gangrene*	*III*	*6*	*Resting ischemic pain Loss of minor tissue Loss of major tissue*

Palpation of pulse of the femoral, popliteal, posterior tibial, and dorsal pedis arteries are integral examinations for claudication. Pulse palpation is classified into 3 degrees, normal (score 2), reduced (score 1), not palpable (score 0). Auscultation should be used to determine the presence of bruit (murmur) proximal occlusion. When the pulse is absent, bruit is also absent.[5]

DIAGNOSING CHRONIC PERIPHERAL ARTERIAL OCCLUSION DISEASE

Several diagnostic methods that could be used are as follows:

Questionnaire

The first questionnaire was developed by Rose/WHO in 1962, with a 99.8% specificity and 67.5% sensitivity. Another questionnaire is the Edinburgh questionnaire, with a 99% specificity and 91% sensitivity to diagnosis intermittent claudication. However, the precision of questionnaires vary according to age, job, and specific characteristics of the population. Thus, epidemiological data that is based on questionnaire alone should be more carefully scrutinized.[4]

Physical Examination

Palpation of pulses may be able to determine the location of the lesion. However, absent pulses may be found in normal subjects due to anatomical variation.

Hematological and Biochemical Evaluation

Assessment should be made to establish risk factors and underlying disease that could aggravate claudication. The following items need to be examined: peripheral blood examination, platelet count, urinalysis, fasting and 2 hours post prandial blood glucose, HbA1c, blood fat (total cholesterol, LDL, HDL, and triglyceride), kidney function (blood urea nitrogen, serum creatinine).

Other Circulatory System Assessment Methods

Electrocardiography is routinely performed on new patients. Other examinations depend on clinical manifestations of patients (coronary heart disease, cerebrovascular disease, or renal arterial disease).

Ankle-Brachial Pressure Index (ABPI)

ABPI is measured on both legs in all new patients with intermittent claudication. The sphygmomanometer cuff is placed above the leg with a Doppler probe and on the posterior tibial or dorsal leg arteries. This examination could determine systolic blood pressure. The normal ABPI is over 1.0. Patients with peripheral arterial disease have an ABPI of less than 0.9.

Segmental Limb Pressure

Segmental limb pressure could be used to determine the location of the lesion and the severity of occlusion. Note that in diabetic patients with severe arterial calcification, the pressure may be very high (for example 300 mmHg). A solution would be to take the pressure on the first or second toe. The Doppler probe could be replaced by a digital phletismograph.

Functional Tests (Treadmill Tests or Other Tests That Induces Hyperemia)

During rest, the circulation of the arteries of the lower extremities is a prototype of a highly resistive circulation with low flow. Physical exercise changes it into a low resistive hemodynamic with high flow, causes an increased pressure gradient in patients with peripheral arterial occlusion, which may be undetected during resting. Measuring the pressure gradient (specifically ABPI) after exercise or using other protocols to induce hyperemia is the best way to quantify the function and disability of patients with intermittent claudication.[5]

The treadmill test is used to discover coronary heart disease. Patients with peripheral arterial disease are asked to continuously perform the test with a standard speed and degree until intermittent claudication occurs (for example: walk 5 minutes at a speed of 3.5 km/hour at an inclination of 12%, which is equivalent to walking 600 meters with average walking speed). Blood pressure should be taken immediately, usually 1 minute after the exercise test. In cases of leg ischemia, blood pressure reduction could reach 50 mmHg. In severe cases, it may be impossible to take the blood pressure of the leg at all for several minutes.[5]

If no treadmill facility is available, the patient is required to walk along the adjacent corridor until claudication ensues, and then the leg and arm blood pressure is measured, as well as the distance that the patient has walked. In elderly patients who are unable to perform the treadmill test, a sphygmomanometer could be positioned on the thigh, increasing the pressure just above the systolic blood pressure and maintaining it at that point for 3 to 5 minutes until reactive hyperemia ensues. A reduction in leg blood pressure at 30 seconds after the sphygmomanometer is deflated is equivalent to 1 minute after walking (at the initiation of claudication) on the treadmill test. Another way is by flexing the dorsal foot while elevating the leg 30 degrees, but this does not produce the same reduction in leg blood pressure as compared to the previous test.

Imaging

Imaging is needed to localize and determine the severity of the occlusive arterial lesion. The examination is done only in patients that require intervention (angioplasty or surgery). The examination may be non-invasive (duplex ultrasonography and contrast angiography or magnetic resonance imaging/angiography) or invasive (arteriography).

For patients that are scheduled for angioplasty, duplex ultrasound may be the choice. Arteriography is not performed prior to the procedure, because it can be performed simultaneously. Duplex ultrasound may give detailed anatomical information and a certain extent of functional information such as the blood flow rate gradient in stenosis.

Vascular surgeons usually require arteriography prior to performing vascular reconstruction.[5]

MORTALITY IN PERIPHERAL ARTERIAL DISEASE

Since peripheral arterial disease and coronary heart disease occurs simultaneously, it is not surprising that 80% of mortality due to peripheral arterial disease is caused by cardiovascular complications, 63% due to coronary heart disease, 9% due to cerebrovascular disease, and 8% due to other cardiovascular complications such as rupture of aneurisms.[3]

The following table portrays the mortality risks attributed to cardiovascular and other causes.

MANAGEMENT OF CHRONIC PERIPHERAL ARTERIAL OCCLUSION DISEASE

Patients with chronic peripheral arterial occlusion disease (CPAOD) have a high risk of suffering from severe and often fatal cardiovascular complications. Thus, the main problem is not their limitations in walking. Patients with intermittent claudication has a 2-4 times risk of dying due to general complications of atherosclerosis compared to patients who do not suffer from intermittent claudication.[13]

Thus, the main focus in treating such patients is changing risk factors of atherosclerosis progression and preventing the development of atherothrombotic complications. Steps that may be taken require quitting smoking, a low fat diet, weight loss for obese patients, low fat diet for hypertensive patients, etc. While beneficial drugs include anti-platelets such as low dose aspirin, statin for hyperlipidemia, etc. These problems will be discussed separately under the topic of Coronary heart disease.[13]

The next step in intermittent claudication treatment is aimed at preventing progress of atherosclerosis of limb artery as well as improvement in the quality of waling. The next discussion in this paper will be on this second step.

Table 2. Risk of Death from All Causes and Cardiovascular Death in Patients with Peripheral Arterial Disease.

				Death Due To All Causes			Death Due To Cardiovascular Disease	
Researcher	Age (years)	Sex	Number of subjects	Control	Peripheral arterial disease	Relative risk (RR) (95 % CI)	All patients	Patients Without Cardiovascular disease at the start of the research
				Percentage per year			RR (95 % CI)	
Criqui et al	*38-82*	*Male*	*256*	*1.7*	*6.2*	*3.3(1.9-6.0)*	*5.1(2.4-10.8)*	*3.9(1.5-10.6)*
		Female	*309*	*1.2*	*3.3*	*2.5(1.2-5.3)*	*4.8(1.6-14.7)*	*5.7(1.4-23.2)*
Vogt et al	*≥ 65*	*Female*	*1492*	*1.1*	*5.4*	*3.1(1.7-5.5)*	*4.0(1.3-8.5)*	*4.5(1.5-6.7)*
Leng et al	*55-74*	*Both*	*1592*	*2.0*	*3.8 (claudication)*	*1.6(0.9-2.89)*	*2.7(1.3-5.3)*	-
				2.0	*6.1 (without)*	*2.4(1.6-3.7)*	*2.1(1..1-3.8)*	-
Newman et al	*≥ 65*	*Both*	*5714*	*4.5*	*7.8*	*1.5(1.2-1.9)*	*2.0(1.1-2.8)*	*2.9(1.8-4.6)*
Newman et al	*≥ 60*	*Male*	*669*	*1.5*	*5.3*	*3.0(2.8-5.3)*		*3.4(1.3-8.9)*
		Female	*868*	*1.3*	*3.8*	*2.7(1.6-4.6*		*3.3(1.3-8.6)*
Kornitzer et al	*40-55*	*Male*	*2023*	*0.4*	*1.0 (without complaint)*	*2.8(1.4-5.5)*		*4.2(1.7-10.5)*

GENERAL TREATMENT FOR PATIENTS WITH CHRONIC PERIPHERAL ARTERIAL OCCLUSION DISEASE

Smoking is associated with a 2 to 3 times increase in chronic peripheral arterial occlusion disease and is the closest risk factor to chronic peripheral arterial occlusion disease compared to other heart disease risk factors. Smoking is also associated with the risk of losing a limb. It also increases the progress of vascular diseases, heart attacks, stroke, and death. Thus, quitting smoking is an important factor in the successful treatment of chronic peripheral arterial occlusion disease.[13]

Bodyweight may be directly associated with the distance that patients with intermittent claudication can reach. Weight loss increases the distance such patients could walk. In addition, obesity is also a risk factor for the development of fatal and non-fatal cardiovascular complications.

Diabetes mellitus is closely correlated with leg arterial disease and its progress. The United Kingdom Prospective Diabetes Study (UKPDS) demonstrated that reduced blood sugar also reduces the complications of all types of diabetes mellitus (particularly microvascular complications). Thus, in order to reduce macrovascular complications (coronary heart disease and infarct) other coronary risk factors (smoking, hypertension, and hyperlipidemia) should be controlled in addition to blood sugar.[13]

Patients with diabetes mellitus also have a risk of suffering from foot ulcer due to sensory neuropathy. Motor neuropathy also causes limb deformity that could result in ulcer. Thus, it is important to take heed of limb health by using leg support (socks) at all times, avoiding excessive pressure, and regularly visiting a diabetic limb specialty clinic.[13]

Hyperlipidemia Risk factors for chronic peripheral arterial occlusion disease are increased LDL and triglyceride and reduced HDL. Lp(a) is also associated with increased risk for chronic peripheral arterial occlusion disease. Studies show that correction of the lipid abnormality stabilizes and may even cause the regression of femoral atherosclerosis. Probucol can prevent progress, but does not aggravate atherosclerosis. Pravastatin reduces carotid artery plague, but does not affect the femoral artery. What is more important is their effect on vascular mortality. Simvastatin reduces cardiovascular complication. The drug also reduces the risk for the development or the advancement of intermittent claudication by 38%.[13] The most recent recommendation from the National Cholesterol Educaion Program (NCEP) is to reduced LDL cholesterol to less than 100 mg% (2.6 mmol/L).[13]

Patients with lower HDL cholesterol levels may be considered for Niacin treatment. Patients with low HDL levels and increased triglyceride levels that fail to respond to dietary modification may receive fibres.[13]

Hypertension the effects of hypertension treatment on chronic obstructive peripheral arterial disease have not been evaluated. In patients with intermittent claudication, hypertension treatment is aimed to reduce the risks of stroke, myocardiac infarct, and cardiovascular death in accordance with the advise from WHO-ISH 1999,14 JNC VI 1997,15 and JNC VII 2003.[16]

A hypercoagulable state should be detected and treated using anticoagulants.[13]

Homocysteine Recent reports demonstrate a strong correlation between homocysteine and chronic obstructive peripheral arterial disease. This is an important risk factor for patients with intermittent claudication who are under 50 years of age. Meta-analysis demonstrates a risk factor for coronary heart disease in patients with homocysteine levels over 5 mmol/L. In patients with coronary heart disease, an elevated homocysteine level is an independent mortality risk factor. Treatment with folic acid, vitamin B12, and vitamin B6 is effective to reduce homocysteine levels, but its effect on the severity of vascular disease has not been evaluated.[13]

Antiplatelet therapy aspirin is used as a secondary prevention effort for coronary heart disease, and produces a 25% reduction in the risk of fatal and non-fatal myocardiac infarct, stroke and vascular death. Another effective drug is clopidogrel, which produces a 23.8% relative risk for ischemic stroke, myocardiac infarct, or vascular death, compared to aspirin. There needs to be further studies to evaluate the effects of these drugs on chronic obstructive peripheral arterial disease.[13]

Physical Exercise

Clinical evidence demonstrates that physical exercise programs, with or without supervision using the treadmill test increases the physical performance of patients with claudication. A 3-month supervised exercise increases the time performance on the treadmill test and reduces the degree of claudication during exercise. The mean increase in physical performance is 179% of the distance from the initial claudication and 122% on the treadmill test.[13]

Physical rehabilitation also has a great effect on other coronary risk factors, by improving glucose metabolism,

reducing cholesterol and triglyceride, increasing HDL cholesterol and helping patients quit smoking. The most important aspect of the program is the patient's acceptance and compliance to the physical exercise program.[13]

TREATMENT FOR SYMPTOMS OF INTERMITTENT CLAUDICATION

There is still no single drug that is fully effective in improving the patient's physical performance. Drugs are classified as vasoactive, reological, and prostanoid, whose mechanism of action is still uncertain. These drugs are only adjuvants and cannot replace the clearly beneficial physical exercise program.

Drugs That are of Benefit, Be It Little, to Improve Claudication

- Pentoxyphylline (2 x 400 mg / day)
  Pentoxyphylline increases the deformability of red blood cells, reduces fibrinogen levels, reduces platelet aggregation, and increases the distance patients with peripheral arterial disease could walk for about 21% compared to placebo. Patients with over 1 year of symptoms and an ankle brachial pulse index of less than 0.80 demonstrate a better response to treatment.[13]
- Naftidrofuryl (3 x 200 mg / day).
  Naftidrofuryl is a 5-HT antagonist that improves aerobic metabolism in hypoxic tissue and may reduce red blood cell and platelet aggregation. Naftidrofuryl increases the distance that claudication patients could walk.[13]
- Buflomedil (2 x 150-300 mg / day)
  Buflomedil has an alpha-1 and alpha-2 adrenolytic effect, and thus reduces vasoconstriction. It also has an effect on platelets, red blood cell deformobility, and weak calcium antagonist. Studies on buflomedil demonstrate improved exercise capacity.[13]
- Cilostazol (2 x 100 mg / day).
  Cilostazol is a phosphodiesterase III inhibitor with vasodilator and antiplatelet activity. In studies, cilostazol increases the patient's capacity for physical performance.[13]

Drugs with Minimal or No Effect to Improve Intermittent Claudication

- Antiplatelet agents
  These drugs are greatly beneficial in preventing cardiovascular complications, but are of no use to increase the physical performance of claudication patients.[13]
- Vasodilators
  The drugs include alpha blockers, direct vasodilators (papaverin), beta-2 agonist (nylidrin), calcium antagonist (nifedipin), and angiotensin converting enzyme inhibitor. Why these drugs are not beneficial is as follows: because the blood vessels at the ischemic site has undergone maximum vasodilatation, it instead causes the steal phenomenon (vasodilatation of healthy blood vessels).[13]
- Ketanserin
  It is a serotonine selective (S2) antagonist that reduces blood viscosity in addition to having vasodilator and antiplatelet effects. Studies demonstrate that these drugs are ineffective for the treatment of claudication.[13]
- Other ineffective drugs: include aminophyllin, vitamin E, defibrotide, and other vasoactive agents such as cinnarizin, cyclandelate, and nicotinic acid derivatives.[13]

Drugs That May Be Beneficial, but Has Not Undergone Sufficient Studies

Carnitine, prostanoid, vascular endothelial growth factor (VEGF), and L-arginine may be beneficial, but has not been supported by adequate research.

ENDOVASCULAR PROCEDURE AND SURGERY FOR INTERMITTENT CLAUDICATION

Prior to offering interventional procedures (angioplasty and/or stent isertion) or surgery, the following issues should be contemplated:

1. Inadequate response was obtained from physical exercise and risk factor modification,
2. There is severe disability, the patient is unable to perform normal activity or there is a severe impediment to other activities that are important to the patient
3. There is no longer other disease that limits activity after claudication is alleviated (such as angina pectoris or chronic pulmonary disease, etc).
4. The morphology of the lesion is in line with the intervention, with a low risk and high probability for initial and long-term success.[13]

The type of the lesion determines the choice of either endovascular treatment or surgery, as can be found as follows:

Lesions at the Iliac artery;

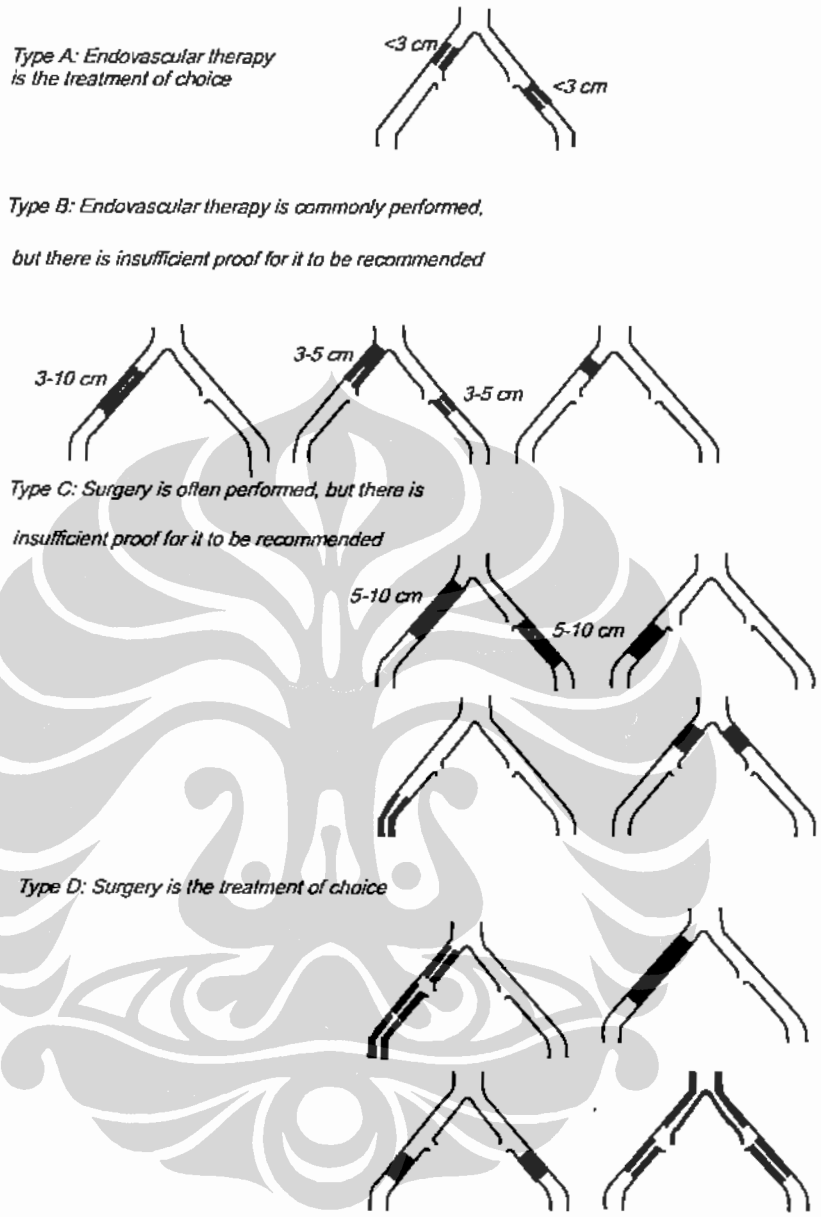

Figure 1. Summary of The Interventional Treatment Choices for Ilial Lesions

Type A iliac lesion is the lesion of choice to perform percutaneous angioplasty (PTA). Stenting improves initial clinical success, but if there is still a significant pressure gradient, dissection and elastic recoil will occur.[13]

Femoropopliteal Lesions

Patients with aortoileal disease alone tend to be younger, with less likelihood of accompanying coronary heart disease. On the other hand, patients with femoropopliteal and infragenicular disease or multilevel stenosis tend to have a lower ABPI and a higher chance for coronary heart disease. Thus, perform percutaneous angioplasty when possible to preserve the major saphenous vein if it is needed for coronary bypass surgery. Unless there is diabetes mellitus with severe limb ischemia, bypass surgery may be the treatment of choice.[13]

As with iliac lesions, femoropopliteal lesions are also stratified based on morphology, as follows:

Morphological stratification of Femoropopliteal lesions.[13]

TASC Type A:

- Single stenosis of < 3 cm (unilateral/bilateral)

TASC Type B:

- Single stenosis of 3-10 cm, not involving the distal

popliteal artery
- Stenosis of < 3 cm with severe calcification
- Multiple lesions (stenosis or occlusion), each < 3 cm
- Single or multiple lesions without the presence of continuous tibial runoff

TASC Type C:
- Single stenosis or occlusion of > 5 cm
- Multiple stenosis or occlusion, each 3-5 cm, with/ without severe calcification

TASC Type D:
- Total occlusion of the common femoral artery, the superficial femoral artery, or popliteal artery and proximal trifurcation

Endovascular procedure (Angioplasty and Stent) is the treatment of choice for TASC Type A and D. For TASC Type B and C, further evidence is required to determine the most appropriate intervention choice.[13]

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