CCRH Quarterly Bulletin
Vol.17 (3&4) 1995

MANAGEMENT AND HOMOEOPATHIC TREATMENT OF PERIPHERAL VASCULAR DISEASE IN RELATION TO DIABETES MELLITUS*

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Abstract

Peripheral vascular disease (PVD) is highly prevalent in the patients suffering with Diabetes Mellitus. It generally occurs in the blood vessels of the peripheral parts of the body like arms and legs. Arterio- sclerosis of the arterioles of the periphery leads to ischaemia, neuropathy, infection and even to gangrene due to occlusion of blood vessels.

In the present study, the causes, pathogenesis, biochemical changes, complications and management of peripheral vascular disease in relation to Diabetes Mellitus are detailed.

Administration of Cephalandra indica Q in the treatment of Diabetes Mellitus was found efficacious in preventing peripheral vascular disease besides controlling the sugar levels. Other drugs like Apis mellifica, Arsenicum album, Cantharis, Carbo animalis, Carbo vegetabilis, Cinchona officinalis, Lachesis, Lycopodium, Phosphorus, Rhus tox, Silicea, Sulphur etc., were also found effective in certain cases. Administration of one drop/kg. body weight dosage of Cephalandra indica Q was found appropriate, whereas fifty millesimal potencies in cases of other drugs were found effective.

Introduction

Today many diabetics suffer from the same disease as King ASA - "Peripheral Vascular Disease" (PVD). Technically the name translates to the blood vessel disease at the periphery i.e. in the arms or legs. In diabetics the disease almost always strikes in the legs or feet and may involve in the order of incidence the tibial and common perennial arteries and their branches, the muscles, the pedal vessels and occasionally the small digital vessels. Symptoms depend upon the vessels that are narrowed or thrombosed, the suddenness and extent of the occlusion and the status of the proximal and collateral vessels. The clinical picture may thus vary from a rather stable or a slowly progressive form of vascular insufficiency that over a period of months or years may ultimately result in atrophy, ischaemic pain and occasionally gangrene.

A person with poor circulation and peripheral neuropathy when injures his foot or perhaps develops a blister but feels no pain nor can spot the trouble is supposed to be suffering from peripheral vascular disease. His blood supply to the injured areas is insufficient due to which the bacteria at the wound grows unchecked. The infection becomes so bad that even on treatment with specific antibiotics it resists healing. Eventually tissue in the area dies leading to the amputation of the limb to prevent gangrene from spreading. It is important to note that the seriousness of infection leading to gangrene and amputation can be prevented if causes and symptomatology are properly understood and effective management and treatment is provided to the individual.

Parameters for Evolving Peripheral Vascular Disease in Relation to Diabetes Mellitus

Age	:	Under 40 years	:	0.5%
		40 to 60 years	:	0.55%
		60 years and above	:	10%
Sex	:	Under 70 years	:	men = women (Equally affected)
		Over 70 years	:	Women > men

Occurrence of PVD in Diabetic and Non-Diabetic Individuals

Diabetic	*Non-Diabetic*
1. Men = Women (Equally affected)	Men > Women
2. Both legs	Mostly one leg.
3. Diffused	Localised
4. Younger age (20-30 years older than non-diabetic)	Little older age
5. Advances rapidly	Slowly
6. Arterial calcification markedly present	Less marked
7. Thickening of capillary basement membrane rapid	Less rapid.

* Paper presented at 16th National Homoeopathic Congress held from 17th to 19th November,1995 at New Delhi.

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Causes

The causes are uknown, suggestive rather than conclusive.

1. **Smoking** : Although the mechanism by which it produces atherosclerosis is uncertain, it is thought that it is the high level of CO in the form of carboxy-haemoglobin which is responsible. In an experimental animal it has been observed that due to damage occurring in endothelium of the blood vessel, the prostacyclin production gets depressed allowing platelet deposition at the site of the damage leading to formation of a fibrotic clot.

2. **Hypertension** : Just as hypertension is an important risk factor in cardiovascular and cerebrovascular diseases so it is in the development of peripheral vascular disease.

3. **Hypercholestrolaemia** :
4. **Hypertriglyceridaemia** : It is observed that certain patients suffering from PVD have raised cholesterol and triglycerides which contribute to the formation of atherosclerosis.

5. **Obesity** : Risk of increase in obesity in PVD individuals is as follows: Blood pressure increased, Plasma cholesterol increased, HDL decreased, Physical activity decreased, Triglycerides increased, Insulin production decreased.

6. **Physical inactivity** : Hastens the infiltration of fat and early formation of fibrotic plaque.

7. **Stress** : Hastens the process of development of ischaemic condition of arteries depriving nutrients and oxygen to the tissue leading to cramps and intermittent claudication in the legs and feet.

8. **Family history** : It is observed that family history is an important predisposing factor.

9. **Alcohol intake** : It increases the cholesterol and predisposes for early PVD.

According to Aughson's risk factors :

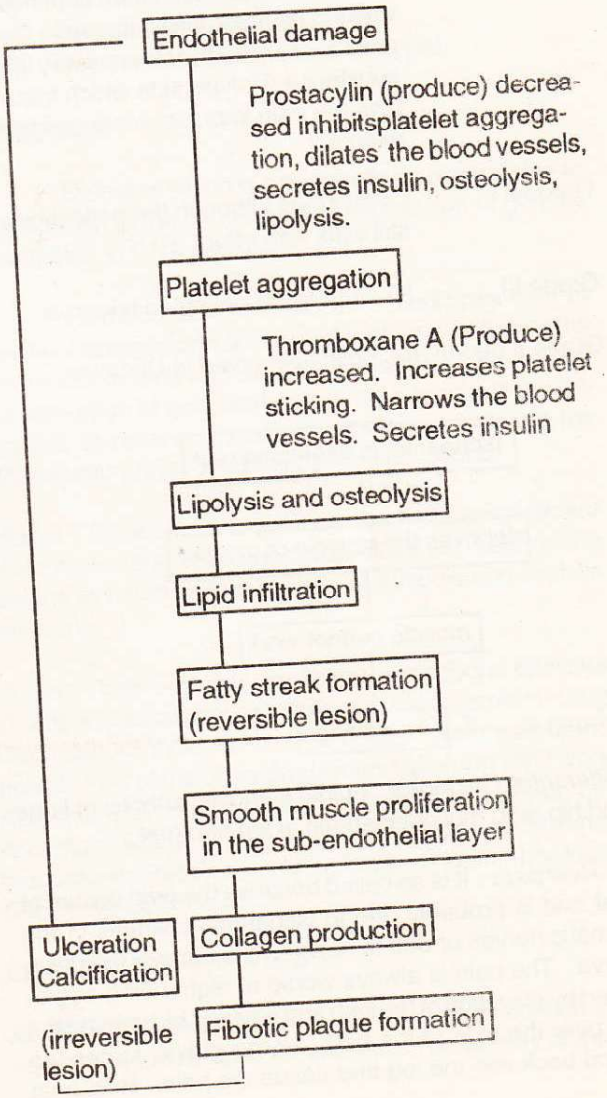
Risk factors	Relative risk of Intermittent claudication.
a) Systolic > 160 mm/Hg	3.4 %
b) Diastolic > 90 mm/Hg	3.2 %
c) Serum triglyceride increased	2 %
d) Smoking > 15 cigarettes/day	8.8 %

Biochemical Changes in the Peripheral Blood Vessels

Biochemical changes start from intima and progresses to media and adventitia.

Experimental model : Hypertension, Hypercholestrolaemia, CO, Immune complex disease (ICD)

Schematic representation



Clinical Manifestations

A. Symptoms

1. *Intermittent claudication* : The term claudication has originated from Latin word "Claudicare" which means "to limp". It is a cramp like pain occurring most commonly in the calf muscles, on walking or climbing. The site of pain depends on the level of the arterial occlusion and pain typically subsides when the person stops walking or climbing only to reappear again at the same distance as he walks again.

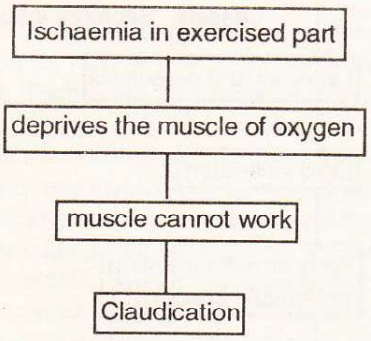
Boyd's classification

Grade I : Claudication passes off on continued walking as metabolites increase the muscle blood flow to sweep away the P. substance (Polypeptide which transmits the pain impulse) produced by exercise.

Grade II : Continuous although the patient can still walk with effort.

Grade III : It compels the patient to take rest.

Grade II and III are mostly observed in Diabetes.



Differential Diagnosis : Spinal stenosis, arthritis of knee and hip, and muscular sprain in leg and feet.

2. *Rest pain* : It is so called because the pain occurs at rest and is probably due to ischaemic changes in the somatic nerves or due to sepsis. It is a cry of the dying nerve. The pain is always worse at night being aggravated by elevation of the limb and relieved by hanging the leg over the side of the bed, as the gravity forces the blood back into the leg and eases the pain. Rest pain often comes on in bed at night when the cardiac output is less. In more advanced stages the pain may be constant and so severe that even narcotics may not relieve it. It is in this state that amputation is needed.

3. *Muscle cramps* : Sudden painful contraction that lasts only a few minutes but leaves a feeling of soreness for minutes or days in a painless leg are usually related to the PVD.

B. Signs

1. *Absence of pulsation* : Careful palpation over the femoral, popliteal, dorsal pedis and posterior tibial arteries should be done to determine which pulsation is present.

2. *Colour changes in feet* : Defective blood supply causes anoxic paralysis of capillaries.

a) Pallor upon elevation : If pallor appears rapidly upon elevation of foot from the horizontal or if it appears when the leg is only slightly raised the circulation status is poor.

b) Flushing time : If flushing time is over 20 seconds and especially if it appears only after 45-60 seconds the arterial disease is extensive.

c) Depending rubor : Means the foot becoming red or purple when it hangs down and blood flow in the vessel is poor, the pressure may be too weak to pump the blood back up the legs.

d) Rubor stasis : If dependent rubor is persisting for a long time.

e) Patchy cyanosis and pallor : This indicates severe schaemia. They are seen frequently following acute thrombosis.

3. *Venous filling time* : If it takes the vein on the dorsum of the foot more than 30 seconds to fill when the leg is placed in a dependent position after having been elevated for a minute the circulation impairment in the leg is severe.

4. *Local tissue changes* : Diminished arterial flow causes wasting of the subcutaneous tissue of the foot and lower leg. Hair is lost over the area. The skin becomes smooth and shiny. The nail may become thick

and deformed. Infections are commoner following the minor injuries or even without injury. Once established infection may become indolent and chronic with the formation of an ischaemic ulcer that is often located over the pressure point of the foot or the infection may lead to localised or progressive gangrene.

5. *Skin temperature* : If the arterial circulation is inadequate the leg becomes quite cool.

6. *Sweating* : The feet are often dry but if the patient with PVD still gets sweating of the feet, some degree of sympathetic activity is present.

C. Other Methods

X- ray findings : Films of lower leg and foot may show calcification of the vessel. If there is a draining sinus at an ulcer close to the bone or joint, osteomyelitis may be apparent on the film.

D. Physiological testing

1. *Doppler measurement* :

   a) The Doppler instrument sends out a sound signal which bounces off the moving red blood cells in the blood vessel. When the vessels are narrowed or blood flow is decreased the Doppler will emit characteristic wave signal.
   b) We can also elicit by this instrument B.P. at the ankle which is same as in the arm. When the pressure is significantly lower in the ankle than in the arm the PVD is to be considered.

2. *Blood flow to the limb is also determined by* :

   a) Electromagnetic flow meter.
   b) Radioactive isotope clearance.
   c) Arteriography: This procedure gives information about the size and course of the arteries, their constriction and dilatation, and the condition of the collateral circulation.

Prevention

Although we cannot control all the factors that contribute to the PVD but to some extent it will be surely helpful.

1. Smoking : Avoiding will go a long way in saving high risk factor.
2. Cholesterol in the diet : Saturated fat (animal) are believed to contribute to cholesterol production leading to early PVD and hence polyunsaturated fats (vegetable fat) are advised.
3. High B.P. causes blood vessel damage and its control is needed.
4. Blood sugar should be controlled.
5. Taking good care of foot
   a) Wash and inspect toes and toe webs daily, check for blisters, cuts and scratches.
   b) Avoid extreme hot and cold. Do not apply hot water bottle or heated pads to your feet. Wear socks for keeping them warm.
   c) Do not use chemical agents to remove corns, calluses and do not cut them.
   d) Cut toe nails straight across.
   e) Do not walk bare foot.
   f) Use comfortable shoes or chappal.

Treatment

Diabetes which is a metabolic disorder is due to some defect in the content or the expression of genetic information in DNA molecule.

Homoeopathic potentised medicines due to their light isotopic form are capable of penetrating into the chromosome level and exert their chemical influence for the correction of genetical defect, thereby rendering the process of reverse mutation resulting in reducing the blood sugar and preventing the complications.

Miasm : Basically PVD is psoric due to the involvement of blood vessels. When it is complicated with neuropathy leading to necrosis and gangrene, sycosis and syphilis intervene.

Drugs : Homoeopathic medicines were found effective in the management of PVD. An indigenous drug Cephalandra indica in mother tincture form was found useful. Few of the other drugs found useful in PVD were Arsenicum album, Phosphorus, Lachesis, Sulphur, Lycopodium, Silicea, Carbo vegetabilis, Rhus tox, Calcarea carbonicum, Secale cor., Apis, Arnica, Crotalus horridus, Cantharis, China etc.

CASES BEING TREATED

A. Cephalandra indica Q It is found to contain an enzyme, hormone and an alkaloid. The activity of this

enzyme glucokinin was isolated by Dr. Kollip in 1923 and he stated its property of reducing blood and urine sugar. It has marked amylolytic property and rapidly hydrolyses starch. Dr. W.C. Dutta advocated the drug's usefulness in diabetes. Dr. R.N. Chopra has proclaimed in his book - "Indigenous Drugs of India" that Cephalandra indica is of less value in genuine diabetic patients. But however Dr. S.C. Ghose in his book "Drugs of Hindoosthan" has given us a elaborative clinical verification of this drug.

Duration of administration of drug : 2 years 6 months.

Dosage : 1 drop / kg. body weight.

Improvement in respect of signs and symptoms

	Prescribed Total No. of cases			Found effective after treatment		
	T	M	F	T	M	F
Symptoms						
1. Intermittent claudication	17	7	10	6	4	2
2. Muscle cramps	20	6	14	6	3	3
3. Rest pain	7	4	3	3	2	1
Signs						
1. Postural hypotension	7	3	4	2	1	1
2. Dependent oedema	5	1	1	2	1	1
3. Sepsis (Boils)	6	4	2	3	2	1

B. Other drugs

Sl. No.	Patient & disease particulars	Laboratory Investigations	Treatment given	Present status
1.	F-55, widow, DM since 4 years, pain calf muscle suddenly < walking, sleeping during. Dependent rubor of both feet + Irritable, constipation UTI	*April, 1991* PLBS : 280 mg/dl++ Alb : +++ Hb : 8.5 gm%	Apis 0/1 TDS for 10 days F.P. 6x K.M. 6x TDS for 10 days A* 1+1 Tab.	*June, 1991* PLBS:210 mg/dl+ Alb : Traces Hb : 10 gm% Apis 0/3 TDS for 10 days A 1+1 Tab. Dependent oedema reduced slightly. Calf muscle pain reduced slightly. *August, 1991* PLBS : 180 mg/dl+ Alb : Nil Hb. 10.5 gm% Apis 0/3 TDS for 10 days A 1 Tab. Oedema and pain reduced markedly.
				Patient was given Apis 0/6 in April, 1992 and December, 1992 with the recurrence of dependent rubor and found good relief.
2.	M-65, DM since 6 yrs., extreme weakness restless, depression+, thirst ++, skin dry & coldness of lower endof both legs with burning pain > by warmth, fever on and off with thirst for small quantity.	*Nov. 1991* FBS : 136 mg/dl+ PLBS : 290 mg/dl ++ Hb : 10.5 gm%	Ars. alb 0/3. TDS for 10 days Abroma augusta Q TDS A.	*Feb., 1992* PLBS : 260 mg/dl+ Hb : 10.5 gm% Ars.alb 0/6 TDS for 10 days Abroma augusta Q TDS A. Weakness+ skin dryness +. Burning slightly + No fever. *April, 1992* PLBS :202 mg/dl+ Hb : 10.5 mg% Ars.alb 0/6 TDS for 10 days. Abroma augusta Q TDS A. Burning reduced, weakness reduced. No fever
				Again in December, 1992 & April, 1993, Ars. 0/6 was repeated and found good result.
3.	F-45, DM since 4 years, UTI, Bloody urine, constant urge to urinate, polyneuritis, + dependent oedema of both legs and feet. +	Sept., 1991. PLBS : 216 mg/dl++ P.C. : Loaded Alb : Traces	Cantharis 0/1 TDS for 10 days Cephalandra Q TDS.	

A* = Allopathic drug.

	Nov., 1991 PLBS : 186 mg/ dl++ P.C. : 10-15/HPL Alb : Traces	Cantharis 0/3TDS for 10 days Cephalandra indica Q TDS	UTI reduced. bloody urine reduced, dependent oedema slightly+
UTI since 2 months, constant urge to urinate, evening rise of temperature from 4 P.M., oedema of both the legs & feet +.	*Dec., 1992.* FBS : 110 mg/dl+ PLBS : 196 mg/ dl ++ P.C.: Loaded Alb. : Traces.	Lyco. 0/3 TDS for 7 days. Cephalandra indica Q TDS	
	Jan., 1993. PLBS : 190 mg/ dl ++ P.C. : 4-6/HPL Alb. : Traces	Lyco. 0/3 TDS for 7 Cephalandra indica Q	UTI reduced. oedema reduced, gastritis +
4. M-60, DM since 3 years, pain in the legs < night; coldness of legs and feet, wants to uncover, digestion weak, varicose veins of both legs. Pain in the left leg (Sciatica) < night, < damp weather > continuous motion vericose veins of both legs present.	*Jan., 1991.* PLBS : 266 mg/ dl ++ Alb. : Traces	Carbo veg. 0/3 TDS for 10 days A. 1+1 Tab.	
	April, 1991. PLBS:210 mg/dl+ Alb : Nil A. 1+1 Tab.	Carbo veg. 0/3 TDS for 10 days	Pain and coldness of the foot slightly reduced.
	Sept., 1991. PLBS :190 mg/dl+ Alb : Nil	Rhus tox 0/3 TDS for 10 days A.1+1 Tab.	
	Nov., 1991. PLBS :130 mg/dl+ Alb : Nil	Rhus tox. 0/6 TDS for 10 days A. 1+1 Tab.	Pain in the leg slightly present
5. M-62, DM since 4 years, physically and mentally weak, trembling of hands + pain leg during sleep (rest pain), suspicious, talkative, cannot bear clothes on body and legs.	*Sept., 1991.* PLBS : 276 mg/ dl++ Alb.: Traces	Lach.0/3TDS for 10 days Abroma aug.Q TDS A.1/2+1/2Tab.	
	Dec., 1991. PLBS : 242 mg/ dl ++	Lach. 0/6 TDS for 1 week Abr. aug. Q A.1/2+1/2 Tab.	Rest pain slightly reduced, Trembling slightly reduced.
	March, 1992. PLBS : 264 mg/ dl ++ Alb. : Traces	Rhus tox 0/6 TDS for 7 days Abr. aug.Q TDS A.1/2+1/2 Tab.	Rest pain slightly present.
	June, 1992. PLBS : ++ Alb. : Traces	Rhus tox 0/6 TDS for 7 days Abr. aug. Q TDS A.1/2+1/2 Tab.	Rest pain reduced markedly.
6. M-49, DM since 2 years, varicose ulcer on left leg with itching, wakes up suddenly at night due to leg pain (rest pain), history of suppressed eczema of varicose ulcers, polyneuritis.	July, 1995. PLBS : 222 mg/ dl++ Alb. : Nil Hb.: 11.5 gm%	Sulph. 0/3 TDS for10 days Cephalandra Q TDS	
	August, 1995 RBS : 196 mg/ dl++ Alb : Nil	Sulph. 0/6 TDS for 10 days Cephalandra Q TDS	Itching slightly reduced. Polyneuritis slightly reduced, pain leg reduced slightly +
	Nov., 1995 PLBS:190 mg/dl+	Sulph. 0/6 TDS for 7 days Cephalandra indica Q	Feeling better with less suffering.

Acknowledgments

Thanks are due to the authorities of CCRH for facilities and encouragement and to Prof. T. Rajagopal, Project Officer, Drug Standardisation Unit, Hyderabad, for the help provided in preparation of this paper.

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"Homoeopathics treats of both the science and the art of healing by the law of similars, and if the art is to remain and progress among men the science must be better understood than at present. To apply the art without the science is merely a pretension, and such practice should be relegated to the domain of empiricism. To safely practice the art of curing sick people, the homoeopathic physician must know the science".

J.T. Kent
Preface to Lectures on Homoeopathic Philisophy
