

Study of Nerve Conduction in Peripheral Vascular Disease

Pandurang G. Chougule, Prakash Patankar and Mayur Nagvekar

Department of Surgery, Krishna Institute of Medical Sciences, 415110 Karad, Maharashtra, India

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Corresponding Author:

Prakash Patankar

Department of Surgery, Krishna Institute of Medical Sciences, 415110 Karad, Maharashtra, India

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Abstract: Peripheral vascular diseases is the most common cause of nerve weakness, so, we have to detect the nerve weakness at earlier stage to prevent the peripheral nerve damage. Since, nerve conduction is gold standard for the diagnosis of the peripheral nerve weakness. The disease is more common in smokers. The cause of Buerger's disease is not yet know, smoking is very-closely related with exacerbations and remission of the disease. The 40 patients of peripheral arterial disease constituted the present study. There is severe decrease in motor and sensory conduction velocity in Stage III patients of fontaine staging. Nerve conduction is significantly decreased in patients having peripheral vascular disease.

INTRODUCTION

Peripheral vascular diseases is the most common cause of nerve weakness, so, we have to detect the nerve weakness at earlier stage to prevent the peripheral nerve damage. However, nerve conduction is gold standard for diagnosis of the peripheral nerve weakness. So, it is necessary to study nerve conduction in peripheral vascular disease. Lower extremity peripheral vascular disease causes impaired lower extremity nerve function with limb threatening ischemia. So, it is necessary to laid down the association between lower limb ischemia and lower extremity neuropathy.

The disease is more common in smokers. The cause of Buerger's disease is not yet know, smoking is very-closely related with exacerbations and remission of the disease. Smoking progressively worsens the course of the disease. Almost all patients of Buerger's disease are smokers but as the incidence is low even among heavy smokers, an immunopathogenesis for the disease has been considered probable. Complement factor c4, antilastin and anticholugin antibodies and cellular sensitivity increase in human Type 1 or 3, Collagen suggest autoimmune

pathogenesis of the disease HLA analysis in patients with Buerger's disease show high frequencies of AW24, BW54, CW1 and DR2 antigens and a low frequency of DR9 and DRW52, however, the significance of these immunologic findings remains to be resolved. Tobacco smoking whether it is a direct etiologic factor or only a strongly contributory factor plays a vital role in disease development and progression. The pathological changes occurring in the early and late stage are described.

Acute ischemic limb: Acute arterial insufficiency is most often caused by intrinsic obstruction of major arteries due to embolus which most commonly occurs. In lower extremity embolus particularly lodge in the iliac, femoral and popliteal arteries. In upper extremity the embolus lodge in the brachial artery and less commonly in subclavian or axillary artery. In patients with atherosclerotic diseases, arterioarterial embolization may produce occlusion of small distal vessels. In feet and hand which is recognized as blue toe syndrome. In patients with atherosclerosis, arterial occlusion can also be caused by thrombosis. This occurs in areas where vessels are severely stenotic but due to stenotic lesion there is

development of collaterals and thus final occlusive lesion does not produce severe degree of arterial insufficiency as seen in other forms.

Intrinsic arterial obstruction may be caused by insertion of catheters or medical device that are employed in monitoring patients for e.g., arterial pressure monitoring, or blood gas analysis, transluminal angioplasty, administration of intra-arterial infusion. This leads to formation of local clots that may result in distal emboli. Arterial injuries either blunt or penetrating is the most common cause of extrinsic arterial obstruction. In some cases contusion to vessel may cause severe spasm and lead to acute vascular insufficiency. Secondary thrombosis is commonly seen in arterial injuries following significant periods of hypotension. Arterial injuries following fractures of long bones and dislocation may produce external compression or contusion, laceration or even transection. Arteries susceptible to such injuries are brachial, popliteal or axillary. Acute arterial insufficiency may also be seen rarely due to extrinsic compression by neoplastic masses or massive soft tissue swelling. This is most frequently seen in the lower leg due to rigid fascial sheath called as compartment syndrome. Acute vascular insufficiency may be seen in venous outflow obstruction. As in phlegmasiaceruleadoloris, due to ileo-femoral venous thrombosis this extensive obstruction of venous outflow from leg interferes with arterial inflow. Low flow states occurring in cardiac diseases, endotoxemia and debilitating diseases may lead to arterial obstruction due to thrombosis and sufficient ischemia to produce gangrene of the extremity. Several drugs including digitalis, corticosteroid and phenothiazines are thought to increase the risk of non-obstructive arterial occlusion. The exact mechanism is not known.

Pathophysiology of acute ischaemia: The response of the extremity to acute ischaemia depends upon the susceptibility of different cells to anoxia. This difference reflects the oxygen requirement for that particular type of cell. The skin and the subcutaneous tissue may, survive durations of hypoxia that may not be tolerated by skeletal muscles and peripheral nerves. The outcome following a period of ischaemia depends not only upon the specific tissue tolerance to hypoxia and period of circulatory interruption but also upon the local changes that impair restoration of normal flow after the initial cause has been removed or corrected. This is called as impaired reflow phenomenon. Following a period of hypoxia there is cellular swelling which plays important role in irreversibility. Following ischemia there is release of oxygen 'derived free radicals which cause damage to cell membrane with decrease in cell permeability and swelling.

Definition is a localised dilatation of a segment of the arterial wall. The 90% of peripheral aneurysms occur in either femoral or popliteal arteries with popliteal artery aneurysms predominating. Etiologically traumatic,

mycotic, syphilitic and anastomotic aneurysms are reported in these two locations. Atherosclerosis is the major cause of aneurysm formation in -peripheral arteries. The term arteritis includes any arterial inflammatory response induced by infection, radiation, direct or embolic phenomenon. The term arteritis is usually applied to a diverse group of diseases of unknown or immunologic origin (Pokrovsky, 1989).

Aim and objectives: To grade peripheral vascular disease according to severity to of nerve conduction studies.

Literature review: The term "ischemic neuritis" or "ischemic neuropathy" is utilized to depict peripheral nerve injuries brought about by atherosclerotic occlusive PVD which is a typical issue among the elderly. The ongoing edinburgh artery study conducted that, notwithstanding 4.5% of normal matured aged in the range of 55 and 74 having Intermittent claudication, twice that number (8.0%) had strange vascular research facility tests (Ruckley, 1991).

Mufson (1952) exhibited that 37% of 145 PVD subjects had neuropathic harmful in the ischaemic legs. The most prominent observations were sensory symptoms and signals but reflex shifts and muscle fatigue were also reported. Hutchinson and Liversedge (1996). indicated that the existence of neuropathy was directly linked to the severity of PVD while the most notable thing was the mildness of neurological abnormalities despite advanced PVD.

In 1908 Leo Buerger published clinical and pathologic observations on young men with severe ischemia of the extremities. These patients were addicted to cigarette smoking and had migratory superficial phlebitis. He called the syndrome thromboangiitis obliterans characterized by thrombosis in both arteries and veins. The condition became more commonly known as Buerger's disease. The episodic vasospasm of the small arteries and arterioles of the distal parts of the extremities was described by Raynaud's who suggested that the episodes represented sympathetic nervous system hyperactivity. This was later on disproved by Lewis in 1920's and 1930 who postulated that the condition arises from local vascular fault exclusive of sympathetic innervations. Chronic occlusion of the aortic bifurcation by thrombosis was significantly described by Leriche in France in 1923. Takayasu disease was described by a Japanese Ophthalmologist in 1908.

Earlier studies with respect to PAD and lower limit nerve function have been adversar (McDermott *et al.*, 2006). Chopra and Hurwitz (1968, 1969) considered nerved conduction in the middle, ulnar and peroneal and femoral nerves in people with and without PAD. Compared with non-PAD members, just the middle tangible nerve abundancy was marginally diminished in the PAD members. Weber and Ziegler (2002) studied electrodiagnostic testing of peroneal, posterior tibial and

sural nerves was conducted in 44 members with PAD and 37 members without PAD. Peripheral nervous dysfunction of ischemic limbs of atherosclerotic Peripheral Vascular Disorder (PVD) is challenging to diagnose owing to a small number of studies according to Nukada *et al.* (1996). Nerve conduction and EMG defects have been well known in chronic PVD subjects (Miglietta and Lowenthal, 1962; Miglietta, 1966; Chopra and Hurwitz, 1969; D'Amour *et al.*, 1987; Hunter *et al.*, 1988; England *et al.*, 1995) and in acute PVD (Lachance and Daube, 1991). Such anomalies involve sluggish conduction velocity and low amplitude in the common peroneal, posterior tibial and sural nerves and signs of distal denervation.

Priestley (1931, 1932) was the first to portray morphological changes of peripheral nerves in ischemic legs with non-diabetic PVD in the English literature. The range of nerve pathology was legitimately corresponding to the level of pathologically observed arteriosclerotic changes in vessels.

Lower limb innervation

Cutaneous innervation: The skin of the buttock receives fibres that run down from the subcostal and iliohypogastric nerves, the posterior rami of the first three lumbar and first three sacral nerves and the perforating cutaneous nerve, with an upward contribution from the posterior femoral cutaneous nerve. The latter supplies a long strip down the back of the limb to below the popliteal fossa, with lateral and medial femoral cutaneous nerves on either side or a contribution of variable extent from the obturator nerve on the medial side. On the front of the thigh, the ilioinguinal nerve extends below the inguinal ligament with subcostal and genitofemoral elements on either side or the intermediate femoral cutaneous nerve down the middle overlapping with its medial and lateral namesakes and the obturator.

Muscular innervation: In the thigh the anterior compartment is supplied by the femoral nerve and the adductor group by the obturator nerve. The tibial portion of the sciatic nerve is the posterior compartment nerve, with only the short head of biceps supplied by the normal peroneal part. In the gluteal region, the gluteus nerve infects the gluteus maximus along with the other two glutes receiving their supply from the superior gluteal which also supplies tensor fasciae lata. The short lateral rotator muscles behind the hip have their own nerves with the obturatorem externus supplied by the obturator nerve which also supplies part of adductor magnus (hence, it has a double innervation with the sciatic). The tibial nerve is the nerve of the flexor of the leg and the plantar branches of the leg supply the muscles of the foot. The common peroneal nerve divides into the superficial peroneal for the peroneal compartment and the deep peroneal for the anterior or extensor compartment.

Sympathetic innervation: As with the brachial plexus, a grey ramus communicans joins each nerve root of the lumbar and sacral plexuses to the appropriate ganglion of the sympathetic trunk, so that, postganglionic fibres can be distributed to each nerve. The preganglionic fibres for the lower limb have come from cell bodies in the lateral horn of spinal cord segments T11-L2, for the ultimate supply of blood vessels and (as far as skin is concerned) sweat glands and erector spinae muscles.

MATERIALS AND METHODS

This proposed study was carried out after approval from the ethical committee as a prospective randomized clinical trial in 40 patients identified as peripheral vascular disease in the Surgery Department, KIMS, Karad.

All patients diagnosed as peripheral vascular disease were sent for nerve conduction study after obtaining their written informed consent. Three nerves were studied; two motor nerves, common peroneal and tibial nerves and one purely sensory nerve, sural nerve. After obtaining their nerve conduction study values, their results of nerve conduction study were tabulated according to the Fontaine staging.

RESULTS AND DISCUSSION

The 40 patients having peripheral vascular disease constituted the present study, maximum number of patients were in age group between 71-75 & 36-40 years (Table 1).

Male sex is slightly higher when compared to female sex in the ratio 1.5:1. Male sex is considered to be predominant in the development of peripheral vascular disease (Table 2).

The common presenting symptoms Peripheral Vascular diseases are Intermittent claudication and rest pain (Table 3).

In the studies carried out by Campbell, etc., it was observed that mean age of the patients affected by PVD was 75 years. In the present series it was observed that the

Table 1: Patients according to age group

Age groups	No. of patients
31-35	1
36-40	6
41-45	4
46-50	4
51-55	0
56-60	0
61-65	5
66-70	4
71-75	10
76-80	3
81-85	2
86-90	0
91-96	1

Table 2: Sex wise distribution

Gender	No. of patients
Male	24
Female	16

Table 3: Symptoms in No. of patients

Symptoms	No. of patients
Mild intermittent claudication	3
Moderate to severe claudication	6
Rest pain	30
Asymptomatic	1

patients affected were ranging in the age form 71-75 and the mean age is 72 years. Gibbons *et al.* (1979) and Nicholas *et al.* (1982) it was observed that the M:F ratio was 1.7:1,1.8:1, respectively with males affected more among the Indian population and females affected almost as equally as males among the western population. Cole *et al.* in his study had found that cigarette smoking is the important risk factor for the development of PVD. The risk was significantly elevated in patients who smokes 20 cigarettes per day and highest in patients who smokes 41+cigarettes per day.

The importance of cigarette smoking in etiology of PAD is well known. Tobacco smoking is associated with Buerger's disease. The present series show that cigarette smoking is a possible risk factor for development of PAD.

CONCLUSION

There is severe decrease in motor and sensory conduction velocity in Stage III patients of fontaine staging. Nerve conduction is significantly decreased in patients having peripheral vascular disease.

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