Peripheral Neuropathy

Pages with reference to book, From 1 To 2
Huma Qureshi (PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi.)

Peripheral Neuropathy is defined as deranged function and structure of peripheral motor, sensory and autonomic neurons involving either the entire neuron or selected levels (Dyck, 1982). Many potential sites and mechanisms are involved in neuropathy. In parenchymatous disorders (toxic, genetic or metabolic) the pathological process is intrinsic to neurons (axons) or schawann cells, and in intrestitial disorders, the derangement is from without (trauma, compressions or exudates). The symptoms of the patient, nerve conduction and electromyograms are helpful in detecting the neurons (axons) affected and the presumed pathological abnormality.

Axonal degeneration is pcccdcd by atrophy and secondary demyelination in most of the metabolic and inherited neui opathies.

The major types of neuropathies are entrapment, leprosy, diabetic and other metabolic disorders; inherited, deficiency (malnutri-. tion, alcohol) inflammatory demyelinating, toxic, ischaemic and paraneoplastic. In a recent analysis of 205 patients with undiagnosed neuropathies, inherited disorders accounted for 42%, inflammatory for 21% and those in association with other diseases 13% (Dyck et al., 1981). Patients with well developed neuropathy, often have relatives with asymptomic to mild neuropathy and their diagnosis can best be made with clinical, neurophysiologic tests, computer assisted sensory examination or biopsy of the nerve.

Industrial or medicinal poisons, deficiencies and infectious agents have been reported to produce neuropathies, for instance ingestion of fish contaminated with methyl mercury from Minamata Bay in Japan was responsible for neuropathy and other CNS abnormalities. Exposure to n-hexane (used in glue in shoe industry) and acrylamide (used in water proofing caissons) is also known to produce neuropathy. An outbreak of neuropathy in United States among plastic factory workers was attributed to methyl-n-butyl ketone (Allen et al., 1975). Ataxic neuropathy was observed with chlordecone (insecticide) contact in Virginia (Taylor et al., 1980). Workers at polyurethane manufacturing plant, in 1977 experienced difficulty in micturation, blddder distension and weakness of sexual erections due to dimethyl aminopropionitrile (Keogh et al., 1980). Swine influenza immunization programme in United States was associated with Guillain-Barre syndrome which appeared 3 days to 8 weeks after injection. Prolonged use of clioquinalone in Japan for gastrointestinal upsets, lead to subacute mvelo-optical neuro- pathy. An outbreak of wet beriberi in school boys in southern Japan was due to thiamine deficiency in the diet.

Treatment of primary disorders and symptoms is still inadequate. Surgery is useful for entrapped nerves. SuFone drugs are still effective in leprosy. Plasmapheresis is reported to be useful (Fowler et al., 1979; Levy et al., 1979; Sewver et al., 1979; Cook et al., 1980). Chronic inflammatorv demyelinating polyradiculo neuropathy e.g. Guillain Barre Syndrome responds well to prednholone (Dyck et al., 1975). Treatment with prednisolone must however be weighed against the risks, e.g. cataract, infection, gastrointestinal baemorrhage, hyperglycemic hyperosmolar coma and aseptic necrosis of hip. Controlled trials of azathioprine, cyclophosphamide and other immunotherapy in this disorder are not available. Myxedema neuropathy responds well to thyroid medication. Vitamin B deficiency due to any cause responds to specific therapy for it. Uraamic rzeuropathy is uncommon with the availability of haemodialysis, and renal transplantation. Diabetic neuropathy still remains a problem to be treated. Insulin pumps and supplimentation of diet with myo-inositol, a metabolite excessively excreted in uncontrolled diabetes are still on trial. The results of clinical trials with drugs that inhibit plasma cell proliferation in neuropathy and benign dysproteinemia are not available. Multiple neuropathy due to necrotizing angiopathy in periorteritis nodosa, rheumatoid arthritis, churg-strauss

syndrome and Wegener's granuromatosis is often suppressed by prednisolone nit the ultimate usefulness of this drug is still doubted. A few paraneoplastic neuropathies can be improved by the removal of ncoplasia.

Treatment of inherited neuropathy has been genetic counselling and symptomatic treatment, infusion of deficient enzymes in these patients have been unsuccessful. Reduction of phytols in patients with Refsum's disease is helpful. Attacks in acute intermittent porphyria can be controlled by avoiding certain drugs. A set of patients with inflammatory demyelinating inherited motor and sensory neuropathy responded well to prednisolone.

Pain in peripheral neuropathy occurs with the involvemont of small myelinated or un- myelinated affer nt flbers. Tt is described as "tight bend, like", "prickling" "burning", "Searing" and "seneitive with use'. Phenvt&n sodium and earbazepine are help ud. Their do se should be increased gradually in the early stages and then reduced. Transcutaneous stimulation sometimes gives relief, sedative and an ilgesics (salicylates, codcinc) are alr o helpful (Dyck, 1982). Patients with progressive mascu- lar atrophy or atrophy of large afferent fibers with degeneration (Friedreich's ataxia) however, do not experience pain.

References

1. Allen, N., Mendell, J.R., Billmaier, D.J., Fontaine, R.E. and O'Neill, J. (1975) Toxic Polyneuropathy due to methyln-hutyl ketone. Arch. Neurol., 32:209.
2. Cook, J.D., Tindall, R.A.S., Walker, J., Khan, A. and Rosenberg, R. (1980) Plasma exchange as a treatment of acute and chronic idiopathic autoimmune polyneuropathy; limited success. Neurology, 30:361.
3. Dyck, P.J., Thomas, P.K. and Lambert, E.H. Peripheralneuropathy. Philadelphia, Saunders, 1975.
4. Dyck, P.J., Oviatt, K.F. and Lambert, E.H.(1981)yelds Intensive evaluation of unclassified neuropathies improved diagnosis. Ann. Neurol., 10 :222.
5. Dyck, P.j., O'Brien, P.C., Oviatt, K.F. et al. Prednisolone improves chronic inflammatory demyelinating polyradiculoneuropathy more than no treatment. Ann. Neurol., (in press).
6. Dyck, P.j., Swanson, C.j., Low, P.A., Bartleson, J.D., Lambert, E.H. Prednisolone responsive hereditary motor and sensory neuropathy. Mayo Clin. Proc., (in press).
7. J)yck, P.J. (1982) The causes, classification and treatment of peripheral neuropathy. N. EngI. J. Med., 307:283.
8. Fowler, H., VulPe, M., Marks, G., Egolic, C. and Dau, P.C. (1979) Recovery from chronic progressive polyneuropathy after treatment with plasma exchange and cyclophosphamide. Lancet, 2:1193.
9. Keogh, J.P., Pestronk, A., Wertheimer, D. and Moreland, R. (1980) An epidemic of urinary retension caused by dimethylamino-propionitrile. JAMA., 243:746.
10. Levy, R.L., Newkrik, R. and Ochoa, J. (1979) Treating chronic relapsing Guillain-Barre syndrome by Plasma exchange. Lancet, 2:259.
11. Server, A.C., Lefkowith, j., Braine, H. and McKhann, G.M. (1979) Treatment of chronic relapsing inflammatory polyradiculoneuropathy by plasma exchange. Ann. Neurol., 6:258.
12. Taylor, J.R., Selhorst, J.B., Calabrese, V.P. Chordecone, in experimental and clinical neurotoxicology. Edited by Spencer, P.S. et al. Baltimore, Williams and Wilkins, 1980, pP. 407-21.