DIABETIC NEUROPATHY

Pages with reference to book, From 123 To 125

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Diabetic neuropathy, a troublesome complication of Diabetes Mellitus is encountered in an incidence of 5 to 50 percent of cases.[1] According to the distribution it is classed as symmetric distal polyneuropathy, symmetric proximal motor neuropathy and focal asymmetric neuropathy[2-4]. In most cases the sensory, motor and autonomic nerves are involved. The small fibre type neuropathy manifests as pain and parasthesias in the lower extremities with less involvement of reflexes, position and vibration sensation. Autonomic sensation is usually present. In the large fibre type symmetric polyneuropathy there is loss of ankle reflexes decreased position and vibration sense and sensory ataxia. The cause for selective nerve fibre involvement is not known. The element of pain manifests itself due to acute axonal degeneration involving fibres of all sizes[5], and is associated with profound weight loss and depression. The cause of the pain is attributed to regeneration of small and unmyelinated fibres causing spontaneous nerve impulses,[6-7] or hypoglycaemia causing increased pain intensity[8]. A motor neuropathy may result from repeated episodes of hypoglycaemia due to spinal motor neuron loss[9]. Weakness and wasting of hand muscles and foot drop preceded by paraesthesias are usually observed. Focal neuropathy may affect any cranial or peripheral nerve. The most commonly involved nerves are median, ulnar, common peroneal and femoral with the cranial nerves being the ill, VI and IV in frequency. Painful diabetic thoracoabdominal neuropathy is another focal involvement seen in the fifth or sixth decade[10,11,12]. Weight loss, is significant and provides a suspicion of malignancy. Hyperesthesia and hypaesthesia in the appropriate thoracic segment is suggestive of the diagnosis. Autonomic neuropathy is encountered in 20 to 40 percent of the diabetics.[13-16] The cardiovascular and genitourinary systems have been largely assessed. The most commonly observed cardiovascular abnormalities are postural hypotension, resting tachycardia and painless myocardial infarction.[16] Orthostatic hypotension involves both systolic and diastolic valves and as there is a blunted catecholamine response in these cases, the compensatory tachycardia is lacking. It has also been observed that blood pressure does not rise progressively during the early morning hours in Type I diabetics due to dysfunction of the autonomic nervous system. Silent or less painful infarction was observed in 42 percent 9f diabetics in comparison to 6 to 15 percent of the non-diabetics[17]. This is attributed to lesions in the afferent fibres running through the cardiac sympathetic nerves which carry the pain impulses[18-20]. Despite recent advances, the exact pathogenesis of diabetic peripheral neuropathy remains unknown. Many theories have been proposed. Insulin deficiency may cause metabolic derangements but they fail to explain the development of a structural neuropathy. The sorbitol accumulation hypothesis is the most popular theory. Elevated glucose levels lead to saturation of hexokinase and collection of sorbitol causing direct toxicity by tissue swelling by osmotic effect[21]. Myoinositol, a precursor of polyphosphoinositides, an important element of nerve cellular functions, has been considered to play a role in diabetic neuropathy. Insulin deficiency and, hyperglycaemia cause reduced myoinositol concentration correlating with reduced nerve conduction velocity[22]. The hypoxia hypothesis[23] proposes that the diabetic state causes capillary abnormalities leading to decreased blood and oxygen delivery to nerves and consequent slowing of nerve conduction. + To maintain normal blood glucose and lipids level in conjunction with ideal body weight has been the aim of treatment of Diabetes Mellitus. A relationship between the duration of Diabetes and development of neuropathies before therapy, with improvement of motor nerve conduction velocity after therapy, is flow well established[24-26]. The improvement becomes evident until after six months of treatment. Lately aldose

reductase inhibitors have been tried as therapy for diabetic neuropathy. These act on the nerve metabolism and a favourable though small response has been achieved.[27,28] The substances used are Alrestatin, Sorbinil, toirestat which have been given multicentre trials but none have been approved for clinical use. Dietary myoinositol supplementation has given inconclusive and contradictory results. Vitamins, especially of the B group, do not show enough evidence to justify their use in diabetic neuropathy. Pyridoxine, used for over three months in a daily dose of 150 mg, proved to be ineffective in alleviating the symptoms of diabetic neuropathy.[29] Phenytoin commonly used in painful neuropathy.[30] should be avoided due to its inhibitory effect on insulin secretion. Carbamazepine has proved to be helpful after being tried in three controlled double blind studies[31-33] but should be reserved for refractory cases due to its potential toxicity. Patients with orthostatic hypotension should be instructed to sleep with head up tilt, use elastic stockings, increase salt in diet and to minimize sudden changes of posture. Drugs which have been suggested are Fluorohydrocortisone[34], indomethacin[35], a combination of . diphenhydramine and cimetidine[36] dihydroergotamine[37], metoclopramide,[38] and pindolol.[39] Bladder complications due to autonornic neuropathy can be very troublesome. Voidance every three hours with iritermittent catheterization is helpful. Cholinergic drugs as bethanechol may assist in triggering detrusor function. Urinary tract infection requires appropriate antibiotics and bladder neck resection is considered when all conservative measures fail. Male impotency requiiès psychological counselling before a penile prosthesis is considered.[40] Dysphagia or heart-burns due to oesophageal dysfunction may be managed with domperidone[41] or bethanechol[42]. Metoclopramide relieves delayed gastric emptying in a dose of 10 mg before each meal and at bed time.[43] Diarrhoea may be treated with tetracycline although it resolves spontaneously. Patients with diabetic neuropathy should be encouraged to stop the use of tobacco and alcohol. Drugs known to aggravate peripheral neuropathies as isoniazid, nitrofurantoin, hydralizine and dapsone should be avoided.

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