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REVIEW ARTICLE

EVALUATION OF CLINICAL FEATURES AND NERVE CONDUCTION STUDIES IN PATIENTS WITH IMPAIRED GLUCOSE TOLERANCE

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INTRODUCTION

Peripheral Neuropathy is a commonly encountered clinical problem. Gregg and his coworkers (Gregg et al., 2004) reported that more than 10% of their patients over the age of 40 years had Peripheral Neuropathy. Large studies have not been able to identify the cause for neuropathy in upto one third of the patients (Dyck et al 1981).When compared to the general population it has been observed that many of these patients of Idiopathic Neuropathy have pre-diabetes.

The commonly accepted diagnostic criteria for diabetes and pre-diabetes is as follows:

- Fasting plasma glucose 2-hour OGTT Hemoglobin A1CNormal <100 mg/dl (5.6 mmol/l) <140 mg/dl (7.8 mmol/l) <5.7%
- Pre-diabetes 100–125 mg/dl (5.6–6.9 mmol/l) 140–199 mg/dl (7.8–11.0 mmol/l) 5.7–6.4%
- Diabetes ≥126 mg/dl (7.0 mmol/l) ≥200 mg/dl (11.1 mmol/l) ≥6.5%

It is observed that a large number of subjects with Idiopathic neuropathy share the phenotype of patients with metabolic syndrome. This led to the theory that patients with pre-diabetes have an increased risk for neuropathy. Also screening of idiopathic neuropathy subjects with Oral Glucose Tolerance Test has shown that 40%-50% of this group have IGT (Novella et al., 2001). However investigator bias and the increasing prevalence of Obesity and the accompanying IGT may contribute to over reporting of this risk factor (Dyck et al 2007).

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Huges and his coworkers demonstrated that the prevalence of pre-diabetes in their patients with neuropathy was 30% as compared to controls (14%) (Hughes et al., 2004).This was not statistically significant. But this study demonstrated that these patients had hyperinsulinemia and hypertrigylceridemia when compared to controls. Though patients may not have features/symptoms of neuropathy, the diagnosis of neuropathy can be confirmed by Nerve Conduction Studies. Dyck and his coworkers demonstrated that 12% of Prediabetics and controls had neuropathy as compared to 17.4% diabetics.[6]However one has to remember that Nerve Conduction Studies give a picture of the involvement of large fibres, but Diabetes may involve small diameter fibres selectively. Idiopathic painful sensory neuropathy and neuropathy associated with pre diabetes share a similar clinical phenotype. Though patients have intense sensory symptoms, objective evidence of neuropathy including abnormal nerve conduction studies may be lacking. Hence Smith et al (2001) suggested that patients suspected to have small fibre neuropathy may be subjected to skin biopsy with assessment of intraepidermal nerve fibre density to document loss of small diameter fibres (Smith, 2001). There are several studies which showed that intense control of blood sugars did not influence the onset and progression of neuropathy in diabetics. This has led to the hypothesis that Obesity and associated metabolic derangement like hypertrigylceridemia may play an important role in the development of peripheral neuropathy. Even in the absence of glucose intolerance, Metabolic Syndrome itself has been shown by some authors to be a risk factor for neuropathy. Smith et al (2008) showed that 80% of patients with Metabolic syndrome and neuropathy had Lipid abnormalities despite having normal blood sugars (Smith et al., 2008). On the other hand 33% of population with normal blood glucose tolerance had dyslipedemia in a Finnish study. Hence lipid abnormalities may be a contributing risk factor for the development of neuropathy in pre diabetic state. Herman et al (2007) showed that morbidly obese subjects had nerve conducition abnormalities independent of blood sugar levels.

Hence it can be conjuctured that obesity may have a role in the development of peripheral neuropathy (Herman et al., 2007). Animal models in both diabetic and non diabetic Rats have substantiated the role obesity, high fat diet and lipid abnormalities in the development of nerve conduction abnormlaities.

Pathophysiology: Insulin resistance and dyslipidemia commonly seen in patients with obesity amplify each other in a visciouscycle. Loss of insulin sensitivity in the skeletal muscles results in increased glucose intake by the adipocytes. This results in stimulation and release of free fatty acids, triglycerides which in turn results in increase in adipocity and ectopic deposition of fat (muscle and liver). The ectopic deposition of fat in turn contributes to as increase in Insulin resistance. Ectopic adipose accumulation results in lipotoxic injury to multiple organs by various mechanisms including lipid peroxidation. Lipid peroxidation is an indirect marker for oxidative stress. Levels of TBARS (Thio Barbituric acid reactive substance) a commonly used marker for lipid peroxidation are elevated in Diabetics.[10] It is postulated that oxidative stress in lipid peroxidation may contribute to the development of neuropathy in non-diabetic patients. This is may be the reason for patients of prediabetes developing neuropathy.

Measures to assess neuropathy in pre diabetics: The early studies of neuropathy in diabetics were tilted towards detecting large fibre damage. A combination of symptom analysis, vibration testing/neurological examination with nerve conduction studies was used. With the passage of time it was recognized that small fibres bore the brunt of neuropathy in both pre diabetics and diabetics. Hence studies directed towards the assessment of small fibre functions like QSART (Quantitative sudomotor axonreflextest responses), SSR (Sympathetic Skin response) and Autonomic function tests came into vogue. In addition quantification of intra epidermal nerve fibre density (IENFD) has been attempted by a few investigators.

Importance of detecting neuropathy in pre diabetes: It is a accepted fact that early intervention strategies benefit patients of diabetic neuropathy. However because of the slow progression of neuropathy in this stage, it is difficult to document the benefits of the therapeutic modalities (Dyck et al., 2007b).[6]In the Impaired Glucose tolerance Neuropathy (IGTN) study, it was clearly shown that improvement in the metabolic parameters led to an improvement in the measures of small fiber function including intraepidermal nerve fiber density and quantitative sudomotor axon reflex testing.

We can conclude that intense life style modifications improve or reverse the changes of early neuropathy making it imperative to detect neuropathy as early as possible.

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