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Epidemiology of Diabetic Neuropathy

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INTRODUCTION

Peripheral neuropathy is a devastating complication of diabetes mellitus because of the debilitating symptoms it causes, or associated higher risk of other complications, in particular those involving the lower extremities. The epidemiology of diabetic neuropathy is not as well understood as other complications of this metabolic disorder, including retinal, renal, and coronary artery disease. Different peripheral nerves may be damaged through a variety of pathologic processes as described in other chapters of this book. This chapter will review the prevalence, incidence, and risk factors for different types of diabetic neuropathy. The natural history of diabetic neuropathy will be brie ly described with regard to foot complications. There are six major types of diabetic neuropathy: Distal symmetric polyneuropathy, autonomic neuropathy, nerve entrapment syndromes, proximal asymmetric mononeuropathy also known as diabetic amyotrophic and cranial mononeuropathy.

DESCRIPTION

This chapter will focus mainly on the irst two types of neuropathy. Little is known regarding the epidemiology of the remaining types, probably because, with the exception of nerve entrapment syndromes, these occur infrequently. In order to understand published research on the epidemiology of diabetic neuropathy, certain principles of epidemiologic study design must be taken into consideration. These principles guided this author in the selection of relevant citations and data presentation. Only cross sectional or case control studies conducted in a population based sample (such as a de ined community or health plan enrollment) were considered for this chapter based on review of medline citations using the keywords "epidemiology," "diabetes," and "neuropathy" from 1966 to March, 1997, review of bibliographies of the articles obtained from the Medline search for relevant citations, and review of the author's files. Nine published studies met this criterion. Clinic based cross sectional or case control studies have not been considered except in two instances, because of the potential problem of selection bias associated with these study designs. All 11 prospective studies were considered. Prospective research is less likely to be biased because of differences in probability of subject selection based on disease (neuropathy) and risk factor presence. Prospective research is a stronger study design with regard to inferring the possibility of causation, since the presence of risk factors may be determined prior to neuropathy onset. The problem of measurement error in the assessment of the presence or absence of diabetic neuropathy is well recognized. Nerve conduction velocity arguably the most mined prior to neuropathy onset. The problem of measurement error in the assessment of the presence or absence of diabetic neuropathy is well recognized. Nerve conduction velocity, arguably the most objective and accurate test available for the diagnosis of this complication, is known to sometimes result in erroneous classification. For example, nerve conduction velocity may be normal in diabetic subjects with symptoms of distal symmetric polyneuropathy. This misclassification problem becomes even more problematic when a test result is used to formulate a clinical plan for an individual patient, as compared to epidemiologic analysis where population statistics are the result of interest. When misclassification of neuropathy or risk factor status occurs no differentially (randomly), the net result is bias of any observed difference towards the value. Therefore, observed differences found in an epidemiologic analysis of risk factors for diabetic neuropathy validly reflect potential causative factors for this complication, but probably underestimate the magnitude of

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the risk increase. Epidemiologic studies may draw valid conclusions regarding risk factors for diabetic neuropathy even if the techniques used to measure either neuropathy or the potential risk factor is known to be inaccurate. Distal symmetric polyneuropathy incidence and risk factors (prospective research) the most important epidemiologic study of diabetic neuropathy performed to date is the Diabetes Control and Complications Trial (DCCT). Although designed to answer a therapeutic question, this trial provides much valuable information regarding the incidence of diabetic neuropathy and its relation to glycemic control. This clinical trial included 1161 patients with IDDM who were followed for 5 years for the development and progression of neuropathy. Subjects were randomized to intensive or control treatment groups, after being initially divided into a primary (diabetes for 5 years or less, no micro albuminuria, no retinopathy) or secondary prevention (diabetes for 15 years or less, moderate or less nonproliferative retinopathy, urinary albumin excretion less than 200 mg/24 hrs) subgroups, depending on the presence of end point complications at baseline. Clinical neuropathy was defined as two of the three following conditions: Neuropathic symptoms; sensory deficit to light touch, position, temperature, or pinprick; and abnormal deep tendon reflexes. Confirmed clinical neuropathy was defined as an abnormal clinical exam plus either abnormal nerve conduction in two or more nerves or abnormal response to autonomic testing. After 5 years follow up, the cumulative incidence of clinical neuropathy, confirmed clinical neuropathy, and abnormal nerve conduction was lower in the intensively treated through control groups, irrespective of presence of complications at baseline among controls, the cumulative incidence of clinical neuropathy was 15-21%, depending on presence of baseline complications. Cumulative incidence of abnormal nerve conduction was very high among controls (40-52%). These data demonstrate the crucial role of hyperglycemia in the development of distal symmetric polyneuropathy, but also suggest that neuropathy will continue to develop even in intent therapeutic question. This trial provides much valuable information regarding the incidence of diabetic neuropathy and its relation to glycemic control. This clinical trial included 1161 patients with 1DDM who were followed for 5 years for the development and progression of neuropathy. Subjects were randomized to intensive or control treatment groups, after being initially divided into a primary (diabetes for 5 years or less, no micro albuminuria, no retinopathy) or secondary prevention (diabetes for 15 years or less, moderate or less nonproliferative retinopathy, urinary albumin excretion less than 200 mg/24 h) subgroups, depending on the presence of end point complications at baseline. Clinical neuropathy was defined as two of the three following conditions: neuropathic symptoms; sensory deficit to light touch, position, temperature, or pinprick; and abnormal deep tendon reflexes.

CONCLUSION

Confirmed clinical neuropathy was defused as an abnormal clinical exam plus either abnormal nerve conduction in two or more nerves or abnormal response to autonomic testing. After 5 years follow up, the cumulative incidence of clinical neuropathy, confirmed clinical neuropathy, and abnormal nerve conduction was lower in the intensively treated through control groups, irrespective of presence of complications at baseline.