GAVIN PUBLISHERS

Journal of Diabetes and Treatment

Knott RM. J Diabetes Treat 2017: J114.

Editorial

Diabetes and Vascular Complications: A Recognition of the Outlier

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Citation: KnottRM (2017) Diabetes and Vascular Complications: A Recognition of the Outlier. J Diabetes Treat 2017: J114.

Received Date: 27 February, 2017; **Accepted Date:** 28 February, 2017; **Published Date:** 7 March, 2017

Editorial

Anyone with even a peripheral interest in diabetes cannot fail to recognise the importance of the effective clinical management of the vascular complications associated with this condition. The predicted global increase in the incidence of diabetes is a reminder of the ever-increasing demand on our clinical resources and also for the corresponding loss of life and reduced quality of life that they represent.

A significant amount of work has been carried out in the form of clinical trials, animal studies and in vitro experimentation that form the evidence base for clinical guidelines and public health recommendations. However, despite the significant advances in knowledge there are numerous examples for whom it is inappropriate to apply these in a generalised way: the poor glucose-control of an individual who seems to have 'escaped' major vascular complications; the obese, sedentary, hypertensive individual who does do not develop type 2 diabetes; those who seem unable to manage their diabetes despite excellent compliance to all aspects of the clinical recommendations; and those whose unique genetics and/or socio-economic status fall out-with the rubric of general recommendations and guidelines. It is clear that while population based studies are important in identifying trends, they can never meet the needs of all affected individuals. There is therefore an urgent need for not only a greater embracement of the heterogeneity of diabetic aetiology, but also for the development oftreatment regimens that encompass the uniqueness of an individual'ssymptomatic experience.

Evidence Based Practice is defined as "the conscientious, explicit and judicious use of current best evidence in making decisions about the care of the individual patient. It means integrating individual clinical expertise with the best available external clinical evidence from systematic research." (Sackett D, 1996) [1]. Sackett goes on to describe how the patient brings his or her own personal preferences and unique concerns, expectations, and values and that this forms the basis of individualized care. The trio of evidence derived from scientific research, the clinical expertise and the patient preferences/experiences combine to inform evidence-based practice; and yet it is also the unique presentation of the condition itself that can reveal much about the mechanism of the disease.

So often with the constraints of time, energy, training and finance,standard models are developed based on a whole population approachwhich are then applieduniversally. Individuals who do not conform to the standard presentation of the disease, or who deviate from the 'norm' in terms of their response to medication are often treated as aberrationsrather than valid sources of information that may lead us to a new understanding of the disease that has hitherto been masked as a result of an "average" or typified approach to diagnosis and treatment.

In considering the vascular complications of Diabetes we must recognise the complexity of the vasculature.The role of the vascular system is to carry nutrients and oxygen to tissues but the tissues themselves are not uniformly dependent on receiving these essential supplies. There may also be a varied demand and response to the rate of removal of waste products. Thus, our understanding of how the body responds to conditions identified as 'risk factors' needs to be linked with our understanding of both the specific vascular function and also the impact of the loss and/or compromised function that results when a tissue is deprived of a functional vasculature. The absence of disease in the presence of risk factors can reveal much about identifying protective factors that may be utilised in facilitating our understanding and the treatment of this debilitating condition.

In seeking to define a unifying theory of vascular disease we arelimiting the impact of our own research. Brownlee2 purports that the intracellular events of glucose excess and the subsequent activation of protein kinase C beta is central to all complications. It presents a challenging thesis on unifying factors: elevation of aldose reductase, enhanced flux through the hexosamine pathway and increased production of diacylglycerol to name but a few [2]. But it does not extend our thinking beyond the mechanistic questions. It still leaves us with no rational understanding of the 'exceptions' to the theories that we present. While clinical understanding may embrace the existence of heterogeneity, the detailed investigation of those failing to conform to our defined norms could and should be acknowledged and developed.

In conclusion, if this editorial serves no other purpose that this, I would like to encourage us all to look beyond our normalised framework of reference: to challenge our own pre-conceptions about our respective areas of interest, and to embrace individual pathologiesthat do not conform to the 'average'.In so doing we will extend our scope for an informed management and understanding of diabetes-mediated vascular dysfunction.

Reference

1. Sackett D (1996) Evidence based medicine: what it is and what it isn't. BMJ 312:71.

2. Nishikawa T, Edelstein D, Du XL, Yamagishi S, Matsumura T, et al. (2000) Normalizing mitochondrial superoxide production blocks three pathways of hyperglycaemic damage. Nature404: 787-790