RATIONALE FOR 2 HOUR POST – BLOOD GLUCOSE ESTIMATION FOR DIAGNOSIS OF DIABETES

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Hyperglycemia results from an imbalance between systemic glucose delivery (endogenous glucose production with or without absorption of exogenous glucose) and glucose utilisation. In Type 1 diabetes, insulin deficiency results in both endogenous over production of glucose and impaired glucose utilisation. Whereas Type 2 diabetes, is a heterogeneous disorder with defects both in insulin secretion and insulin sensitivity.

In the natural history of Type 2 diabetes, the earliest defect to occur is insulin resistance followed by insulin deficiency. The metabolic consequence of insulin resistance is defective peripheral glucose disposal in muscles and adipose tissues. In the early stages of Type 2 diabetes (with fasting plasma glucose <7.8mmol/l) systemic glucose production and glucose utilisation (GU) have been found to be normal, where as glucose clearance (calculated by dividing normal GU "near normal" plasma concentration) was found to be decreased [1]. This finding has been considered to demonstrate that muscle insulin resistance is a primary defect in Type 2 diabetes.

The aim in the management of diabetes is "early detection and intervention". The early detection depends upon the testing for plasma glucose at the time of occurrence of early abnormality in glucose metabolism. Insulin resistance is the primary defect that can be identified in the prediabetic subject, years before they develop diabetes. Estimating one hour or two hour post glucose sugar, identifies the person at risk of developing diabetes in the future, as the fasting plasma glucose is lower than the diagnostic criteria for diabetes at this stage.

Fasting hyperglycemia develops years later as the insulin sensitivity worsens and insulin deficiency starts appearing. Hence relying on the fasting plasma glucose for diagnosis would have exposed the system to abnormal glucose levels for many years and thus increased the risk of vascular complications.

Insulin resistance is a major cause of impaired glucose tolerance and diabetes. Beta cell dysfunction is invariable. Once fasting hyperglycemia develops. Upto 80% of beta cells' capacity must be lost before absolute insulin deficiency and significant fasting hyperglycemia appears. It is also worth noting that McCance et al in their Pima Indian study [2], found fasting plasma glucose to have slightly lower specificity and sensitivity that the 2 hour level. Nearly all those with a plasma glucose level 7.8 mmol/l or higher in NHANES survey had a diagnosis 2 hour value, but only 25% of those with a 2 hour value above 11.1 mmol/l (200mg/dl) had a raised fasting level [4].

We analysed 4000 GTT's of non-diabetics who underwent Master Health Check up at Apollo Hospitals, Chennai. Out of them 1002 persons had either fasting plasma glucose (FBS)>126mg/dl or 2 hour post glucose (PPBS) value between 140mg to 199mg/dl or 2 hour post glucose>200mg/dl. When we categorised these 1002 individuals on the basis of the plasma glucose values, 71 had FBS<126 but 2 hour post glucose<200mg/dl, 602 had FBS<126 and 2 hour post glucose between 140 and 199mg/dl, 219 had FBS>126 mg and 2 hour post glucose>200 mg/dl and the remaining 110 had FBS<126 mg/dl but 2h post glucose>200 mg/dl. The outcome of the study was that only 219 (about 22%) had both FBS and PPBS above the diagnostic cut off point for diabetes, whereas 712 (71%) had FBS<126 but had 2 hour post glucose value either in the impaired glucose tolerance (IGT) range (140 to 199mg/dl) or 2 hour post glucose value>200mg/dl. By considering only the fasting plasma glucose for diagnosis of diabetes, we would have missed a large segment of 71% who have either IGT or diabetes.

A clear distinction has to be made between diagnosis for clinical purposes and for epidemiological studies. It is al-right to rely on fasting plasma glucose (ADA recommendation) for epidemiological study purpose, as the procedure is simpler. The drawback of measuring fasting plasma glucose alone will under estimate true prevalence by upto 50% [4].

REFERENCES:

1. Defranzo RA, Bonadonna RC, Ferrnani: Pathogenesis of NIDDM A balanced overview. Diabetes Care 1992, 15:318-68
2. McCane D. R., Hansan RL, Charles et al Comparison of test for a Glycosylated Hemoglobin and fasting and two hour plasma glucose concentration as diagnostic method for diabetes Br Med J 1994, 308: 1323-8
3. Harris Ml, Haddon WC. Knowler WC, Bennett. P.H. Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in the US population aged 20-74 years Diabetes 1987; 36: 523-34.
4. Knoll. CP Editor. World book on Diabetes in Practice Vol 3, Elservier Publication. 1988.

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