HBA1C IN 100 NON INSULIN DEPENDENT DIABETIC PATIENTS

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ABSTRACT

Background: Good glycemic control is associated with reduction in the development and progression of long term microvascular complications of both insulin dependent and non insulin dependent diabetes mellitus. Diabetic control is assessed by fastening, 2 hours post prandial glucose measurement but HBA1C is easy is to measure and gives good control of average glycemic control over the previous three months. We conducted a cross sectional study to assess HBAIC in 100 consecutive patients who had non insulin dependent diabetes for over 1 year from June 2009 to December 2009 who had no acute medical or surgical problems.

Material and Methods: 100 patients who had non insulin dependent diabetes over minimum of 1 year, stable with no acute medical or surgical emergency were selected from medical OPD and private clinics were selected after a verbal consent. HBA1C was measured and bio data was recorded on a written Performa. The grouped into three groups good, fair and poor control based on the HBA1C level of 6.5 or less, 6.5-7 and over 7 as poor recent IDF guidelines.

Results: Out 100, 54 patients (54%) were male and 46 (46%) were female. Mean age was 54.58 ± 11.77 years. 10 patients have good diabetic control having HBA1C of less than 6, 30% had fair (HBA1c 6-8) and 60% had poor diabetic control (HBA1C > 8). Taking both fair and poor diabetic control together, 90 % had inadequate diabetic control. There was no statistically significant association of HBA1C with gender P = 0.2.

Conclusion: According to our study majority of non insulin dependent patient has inadequate diabetic control.

Key words: Non insulin dependent diabetes mellitus, HBA1C.

INTRODUCTION

Diabetes mellitus (DM) is a syndrome of chronic hyperglycaemia due to relative insulin deficiency, resistance, or both[1]. It affects more than 120 million people world-wide, and it is estimated that it will affect 370 million by the year 2030 [2]. Diabetes is usually irreversible and, although patients can have a reasonably normal lifestyle, its late complications result in reduced life expectancy and major health costs. These include macrovascular disease, leading to an increased prevalence of coronary artery disease, peripheral vascular disease and stroke, and microvascular damage causing diabetic retinopathy, nephropathy and neuropathy[3].

Glycated haemoglobin provides an accurate and objective measure of glycaemic control over a period of weeks to months[4]. This can be utilized as an assessment of glycaemic control in a patient with known diabetes, but is not sufficiently sensitive to make a diagnosis of diabetes and is usually within the normal range in patients with impaired glucose tolerance. HbA_{1c} estimates may be erroneously diminished in anemia or during pregnancy, and may be difficult to interpret with some assay methods in patients who have uremia or a haemoglobinopathy[5]. In clinical practice, HbA_{1c} is usually measured once or twice yearly to assess glycaemic control, permitting appropriate changes in treatment and identifying inconsistency with the patient's record of home blood glucose monitoring. HbA_{1c} also provides an index of risk for developing diabetic complications. Prospective, randomized clinical trials such as the Diabetes Control and Complications Trial (DCCT)[6], the United Kingdom Prospective Diabetes Study (UKPDS)[7], and the Kumamoto study have demonstrated that intensive therapy aimed at lower levels of glycemia results in decreased rates of retinopathy, nephropathy, and neuropathy[8]. Every 1 percent drop in A1C was associated with improved outcomes and there was no threshold effect[9,10,11]. Although the goal of the intensive interventions in these studies was normoglycemia, with an A1C less than 6.1 percent, the average A1C achieved in the intensive therapy groups of these trials was around 7 percent[12,13]. We conducted a cross sectional study involving 100 NIDDM cases having diabetes for more than one year who had no acute medical or surgical problem and estimated their HBA1C to assess their diabetic control.

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MATERIAL AND METHODS

A total of 100 consecutive NIDDM patients who had diabetes for over 1 year were selected from medical OPD and private clinics who had no acute medical

or surgical problems after a verbal consent. Their HBA1C was estimated and was entered to a set proforma. They were categorized into thee.

1. Good diabetic control HBA1C <6.5
2. Fair HBA1C 6.5-7
3. Poor HBA1C >7

Exclusion criteria was age <16 years old, pregnancy, Urea more than 40mg/dl, diabetes <1 year duration or presence of acute medical or surgical problems.

RESULTS

Out 100 54 (54%) were male and 46 (46%) female. Mean age was 54.58± 11.77 years. 10 patients hag good diabetic control having HBA1C of less than 6, 30% had fair (HBA1c 6-8) and 60% had poor diabetic control (HBA1C >8). Taking both fair and poor diabetic control together, 90% had inadequate diabetic control. There was no statistically significant association of HBA1C with gender P 0.2.

DISCUSSION

The goal of diabetes management is to keep blood glucose levels as close to normal as safely as possible, while avoiding blood glucose levels that are too high (hyperglycaemia) or too low (hypoglycaemia). The results of our study show that of the total study population, only 10% of the patients have acceptable HbA1c values of <6.5%. The remaining 90 % have values above the recommended cut off and hence an undesirable diabetic control status. These results are in agreement with the results of other studies previously conducted around the country. Using HbA1 c value of 7.5% as the cut-off for good diabetic control, studies conducted in Karachi and Rawalpindi found that 81.3% and 46.7% of the study population respectively, had values exceeding this cut-off indicating poor control of the disease.[14,15] Khalid Mehmood and A.H Amir in Peshawar used HbA1 c of 8.2% as the cut-off for poor control and reported 51.43% patients with poor control.[17] These results have important implications clinically. The Diabetes Control and Complications Trial (DCCT) in 1993, conclusively showed that intensive glucose control delayed the onset and progression of retinal, neural and nephropathic complications by 35% to more than 70%. In fact, it demonstrated that *any* sustained lowering of blood glucose was beneficial, irrespective of previous glycaemic control.[18] Undesirable control of the disease reflected in the majority of our patients can be attributed to a number of factors, the principle one being lack of adequate knowledge about the disease, as evidenced by a study conducted in Peshawar which demonstrated that 58% of the patients lacked appropriate education for disease management. This group also had the highest levels of HbA1c of 9.98%.[19] Financial burden imposed on the patients accounts for the majority of the patients with suboptimum HbA1c levels. Inaccessibility to health care facilities, psychosocial influences and non adherence to treatment are other limiting factors for developing countries like Pakistan. Similar trends have been observed in other south Asian countries. Chuang *et al* showed that 55% of the patients from South Asian countries had HbA1c values exceeding 8%.[20] This pattern has also been reflected in other parts of the world. A retrospective analysis of data from 1998-2002 in UK found that >60% of the patients had poorly controlled disease regardless of the cut-off used to determine good control[21], while a Canadian study demonstrated HbA1 c of 7.7% for 78% of the patients tested[22]. The average HbA1 c for patients in Australian population was 7.3±1.[23]

The ADVANCE study in 2008 has demonstrated that lowering the glycated Hb value to <6.5% leads to a 10% relative reduction in the risk for major micro and macro vascular events.[24] This is reasonable target for many but not all patients; more intensive treatment to bring the target HbA1c within the normal range may increase the mortality but nonetheless, maintains its beneficial effects.[25] Therefore the target value should be tailored according to every individual patient's requirements. The presence of a greater number of females in the poor control group points to another important aspect of the disease. Women in Pakistan have a lower literacy rate as compared to males and have been shown to be less aware of their disease.[26] They tend to spend less[27] and are less physically active with regard to their disease control than men.

REFERENCES

1. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005; 365: 14151428.
2. Kumar and Clark Clinical Medicine Seventh edition 1045-50.
3. Johnston D. NICE guidance on type 1 diabetes in adults. Clinical Medicine 2004; 4: 491–493.
4. Holman RR, Paul SK, Bethel MA et al. Long-term follow-up after tight control of blood pressure in type 2 diabetes. New England Journal of Medicine 2008; 359: 1565–1576.
5. Nico;as A Boon and Walker B, Davidson's practice and principles of medicine twenty edition. 808-810.
6. The Effect of Intensive Treatment of Diabetes on the Development and Progression of Long-Term Complications in Insulin-Dependent Diabetes Mellitus. N Engl J Med1993; 329:977–86.
7. DCCT/Epidemiology of Diabetes Interventions and Complications Research Group. Retinopathy and nephropathy in patients with type 1 diabetes four years after a trial of intensive insulin therapy. New England Journal of Medicine 2000; 342: 381–389.
8. Klein R ,Klein BE ,Moss SE. Relation of glycemic control to diabetic microvascular complications in

diabetes mellitus. Ann Intern Med 1996; 124(1): 90–6.

9. Hoerger TJ, Segel JE, Gregg EW, Saadine JB. Is Glycemic control improving in US adults? Diabetes Care 2008;31(1):81–6.

10. Stratton IM, Adler AI, Neil HAW, Matthews DR, Manley

11. SE, A Cull CA, *et al*. Association of glycemia with microvascular and macro vascular complications of type 2 diabetes mellitus (UKPDS 35): Prospective observational study. BMJ 2000; 321:405–12.

12. Qaseem A, Vijan S, Snow V, Cross JT, Weiss KB, Owens DK. Glycemic Control and Type 2 Diabetes Mellitus: The Optimal Haemoglobin A1c Targets. A Guidance Statement from the American College of Physicians. Annals of Internal Medicine 2007; 147(18):417–22.

13. IDF Clinical Guidelines Task Force. Global guideline for Type 2 diabetes. Brussels: International Diabetes Federation, 2005.

14. Nishtar S, Shera S. Diabetes prevention and control as a part of an integrated non-communicable disease strategy: the Pakistan approach. Pract Diabetes Int 2006; 23(8):332–4.

15. Habib SS, Aslam M. Risk factors, knowledge and health status in diabetic patients. Saudi Med J 2003; 24:1219–24.

16. Mahmood K, Amir AH. Glycemic control status in patients with type-2 diabetes. J Coll Physicians Surg Pak 2005;15(6):323–5.

17. DCCT/Epidemiology of Diabetes Interventions and Complications Research Group. Retinopathy and nephropathy in patients with type 1 diabetes four years after a trial of intensive insulin therapy. New England Journal of Medicine 2000; 342: 381–389.

18. The Effect of Intensive Treatment of Diabetes on the Development and Progression of Long-Term Complications in Insulin-Dependent Diabetes Mellitus. N Engl J Med 1993; 329:977–86.

19. Afridi MA, Khan MN. Role of health education in the management of diabetes mellitus. J Coll Physician Surg Pak 2003; 13:558–61.

20. Chuang LM, Tsai ST, Huang BY, Tai TY. The status of diabetes control in Asia-a cross sectional survey of 24317 patients with diabetes mellitus in 1998. Diabetic Med 2002; 19:978–85.

21. Fox KM, Gerber RA, Bolinder B, Chen J, Kumar S. Prevalence of inadequate Glycemic control among patients with diabetes in the United Kingdom general practice research data base: a series of retrospective analysis of data from 1998–2002. Clinl Ther 2006; 28(3):388–95.

22. Harris SB, Worrall G, Macaulay A, Norton P, Webster- Bogaert S, Donner A, *et al*. Diabetes Management in Canada: Baseline Results of the Group Practice Diabetes Management Study. Can J Diabetes 2006; 30(2):131–7.

23. Mark T, Andrew J, Merlin C. The management of diabetes in Indigenous Australians from primary care. BMC Public Health 2007; 7:303.

24. Patel A, MacMahon S, Chalmers J, Neal B, Billot L, Woodward M, *et al*. Intensive blood glucose control and vascular outcome in patients with type 2 diabetes. N Engl J Med 2008; 358(24):2560–72.

25. Gerstein HC, Miller EM, Byington RP, Goff DC, Bigger JT, Buse JB, *et al*. Effects of intensive glucose lowering in type 2 diabetes. N Engl J Med 2008; 358(24):2545–59.

26. Sabri A, Qayyum M, Saigol N, Zafar K, Aslam F. Comparing knowledge of diabetes mellitus among rural and urban diabetics. Mcgill J Med 2007; 10(2):87–9.

27. Khowaja AL, Khuwaja AK, Cosgrove P. Cost of diabetes care in out-patient clinics of Karachi, Pakistan. BMC Health Serv Res 2007; 7:189.