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Admission Blood Glucose Level is a Risk Predictor in Acute Myocardial Infarction in Non-Diabetic Patients

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ارتفاع مستوى السكر في الدم عند التنويم لمرضى جلطة القلب الغير مصابين بالسكر مؤشر يدلل على زيادة نسبة المخاطرة

لمعـــرفة إمكانية استخدام مستوى السكر في الدم عند التنويم لمرضى جلطة القلب الغير مصابين بالسكري كعامل تنبئي خطر، تمت دراسة جميـــع حـــالات جلطة القلب عند المرضى الغير مصابين بالسكري الذين تم تنويمهم في مستشفى جامعة الملك عبد العزيز بجدة من يناير ١٩٩٥م إلـــى ديسمبر ١٩٩٩م. كان مجموع الحالات ٨٧ حالة و كان متوسط الأعمار ٥٣ سنة و نسبة الذكور للإناث ٧،٧:١وجد ارتفاع مســـتوى السكر في الدم عند التنويم لدى المرضى الذين أصيبوا بهبوط في القلب، جلطة متكررة، و المتوفين، وأيضا ارتفاع نسبة حالا ت الوفـــاة لدى المتقدمين في السن. لم توجد علاقة بين مستوى إنزيم القلب (كرياتنين فسفوكاينيز) و الوفاة. نستنتج من هده الدراسة أنه يمكن استخدام مستوى السكر في الدم عند التنويم كعامل تنبئي خطر في حالات جلطة القلب عند المرضى الغير مصابين بالسكري

The aim of the study is to determine whether admission blood glucose level can be used as a risk predictor in acute myocardial infarction in non-diabetic patients. Analysis of all non-diabetic patient's files admitted to King Abdul Aziz University Hospital, Jeddah, with a definitive diagnosis of acute myocardial infarction between January 1995 to December 1999 was carried out. A total of 87 patient's charts were studied. The mean age was 53 years (range 35-87 years), and M:F ratio was 7.7:1. Admission blood glucose level was significantly higher in patients who developed heart failure, reinfarction and those who died, 8.6 mmol/l versus 7.4mmol/l, 8.1mmol/l versus 7.3 mmol/l and 9.3mmol/l versus 7.8 mmol/l (P= 0.01, 0.03, 0.003 respectively). Old age was also significantly associated with poor outcome while no significant relation was found between the peak cardiac enzyme (creatinine phosphokinase) level and worse outcome. Our data indicated that admission blood glucose level could be used as a risk predictor in non-diabetic patients with acute myocardial infarction.

Key Words: admission blood glucose, myocardial infarction, non-diabetic patients, risk predictor.

INTRODUCTION

Myocardial infarction (MI) is one of the most common diagnoses in hospitalized patients in industrialized countries. Although the mortality rate after admission for MI has declined by about 30% over the last two decades, approximately 1 of 25 patients who survive the initial hospitalization die in the first year after MI as reported by Antman & Braumwald (1998). Multiple risk factors have been found to be a predictor of poor outcome in patients with acute MI , such as age >70 years, prior MI, Killip class I, anterior site of infarction, and the combination of hypotension and tachycardia, (Becker *et al.* 1998). Many investigators (Fuller *et al.* 1980; Scheidt-Nave *et al.* 1991; Bjornholt *et al.*

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1999; Coutinho *et al.* 1999; Gersrein *et al.* 1999) have shown that a moderately elevated blood glucose level is a continuous risk factor for ischemic heart disease and cardiovascular mortality. The aim of this study is to determine whether admission blood glucose level can be used as a risk predictor in acute MI in non-diabetic patients.

METHOD

Medical charts of all patients admitted to King Abdulaziz University Hospital (KAUH), Jeddah, Kingdom of Saudi Arabia with a diagnosis of acute M.I in the period between January 1995 till December 1999 were reviewed. Definitive diagnosis of MI was confirmed by Winter & Eisenberg (1995) criteria: the presence of at least two of the following criteria: 1) a history of prolonged chest discomfort. 2) electrocardiographic changes consistent with ischemia or necrosis. 3) elevated cardiac enzymes. Non-diabetic patients were eligible for inclusion in this study while diabetic patients whether known or discovered on admission (those who had fasting blood glucose level after admission >7.8 mmol/l and 2-hours post prandial >11.1 mmol/l) were excluded. Relevant data were collected from medical charts including patient's age, sex, body mass index (BMI) (body weight in kilograms divided by the square height in meters), admission blood glucose level, peak cardiac enzyme level (creatinine phosphokinase), mode of treatment whether thrombolytic or surgical intervention, development of complications as heart failure and reinfarction (defined as infarct with an onset >72hours after the index infarct) were retrieved as well as duration of hospital stay and mortality. Blood glucose and cardiac enzyme (creatinine phosphokinase) analysis were performed on Hitachi autoanalyzer (Hoffmann-La Roche-BM, Switzerland). Analysis was according to the manufacturer instructions. Statistical analysis was done using computer software (SPSS 7.5).

RESULTS

A total of 87 patients fulfilled the inclusion criteria and they were included in the study. The mean age was 52.63 years (range 30-87 years) with M: F ratio 77:10 (7.7: 1) and mean BMI 26.21 (range 20-36). The mean admission blood glucose level was 7.91 ± 2.38 mmol/l and the mean peak cardiac enzyme level was 1289 iu/l (range 227-5319 iu/l) (normal range 0-195 iu/l). Fifty eight patients (66.7%) received thrombolytic therapy while 14 (16%) had surgical intervention. Heart failure developed in 38 patients (43.7%) while reinfarction in 13 (15%) and 2 (2.3%) died. The duration of hospital stay was ranging between 7-14 days. As shown in Tables (1 & 2), admission blood glucose level and old age were significantly associated with poor outcome i.e. development of heart failure, reinfarction and mortality after acute MI while infarct size measured by enzyme release (peak creatinine phosphokinase) was not related to worse outcome (Table 3).

Table 1. Relation of admission plasma glucose level to outcome following acute myocardial infarction

Variable	Plasma glucose (mmol/l)				P-value
	Yes	n	No	n	
Heart failure	8.59 ± 2.9	38	7.37 ± 1.7	49	0.01
Recurrent myocardial Infarction	8.1 ± 2.4	13	7.3 ±1.8	74	0.03
Mortality	9.3 ± 0.2	2	7.8 ± 2.4	85	0.003

Table 2. Relation of age to outcome following acute myocardial infarction

Variable	Mean age (years)				P-value
	Yes	n	No	n	
Heart failure	57.9	38	48.8	49	0.003
Recurrent myocardial Infarction	59.1	13	51.5	74	0.05
Mortality	52.8	2	46.5	85	0.04

Table 3. Relation between peak cardiac enzyme (creatinine phosphokinase) and outcome following acute myocardial infarction

Variable	Mean peak cardiac Enzyme (iu/l)*				P-value
	Yes	N	No	n	
Heart failure	1490 ± 602	38	1128 ± 386	49	0.1
Recurrent myocardial Infarction	1216 ± 64	13	1301 ± 52	74	0.5
Mortality	2707 ± 700	2	2131 ± 641	85	0.3

* Normal range for creatinine phosphokinase = 0-195 iu/l

DISCUSSION

It has been reported that the magnitude of the rise in the blood glucose during the early phase of an acute MI is attributed to the degree of left ventricular failure (Bellodi *et al.* 1989), which is mainly determined by a raised concentration of catecholamines and cortisol as a response to infarct extension and myocardial dysfunction as discribed by Oswald *et al.* (1986). We found a significant relation between admission blood glucose level and the development of heart failure. Mak *et al.* (1993) has found that a relationship exists between admission blood glucose level and hospital mortality in patients with acute MI, which is in agreement with that found in our study. This higher mortality could be explained by the altered protection afforded by ischemic preconditioning due to raised blood glucose level as suggested by Kersten *et al.* (1998). A significant association between admission blood glucose level and the development of recurrent MI has been described by Norhammar *et al.* (1999), which is similar to our findings. The relationship between admission blood glucose level and poor outcome after acute MI in non-diabetic patients is a metabolically caused mechanism and it may identify subjects with compromised glucose metabolism that is not solely a manifestation of acute stress. A 22-year follow up study conducted by Bjornholt *et al.* (1999), showed that non-diabetic men with fasting glucose level >4.7 mmol/l had 1.4 fold higher risk of cardiovascular mortality

than men with lower glucose values. Another epidemiological study done by Scheidt-Nave *et al.* (1991), with data from a follow up period of 14 years demonstrated that men with fasting blood glucose levels of 6.1-6.6 mmol/l and 6.7-7.2 mmol/l had ~1.3 and ~ 1.5 fold higher adjusted risk of ischemic heart disease mortality than men with glucose levels <5.5 mmol/l.. Results from the U.K. Prospective Diabetes Study (1998), reported that intensive blood glucose control decrease the risk of MI in diabetics. The retrospective design of our study eliminated the possibility to know if admission blood glucose was fasting or random. Therefore, if there is a dysglycemic level above which individuals are at a higher risk for ischemic heart disease mortality, it may be as low as 5.5 mmol/l for fasting and 6.5 mmol/l for 2 hours values. So, it may be possible to use the same levels in non-diabetic patients with acute MI as a predictor of poor outcome. Age was found to be a predictor of poor outcome in acute MI, a finding also reported by Becker *et al* (1998). In our study as well as in the DIGAMI study conducted by Malmberg *et al.* (1997) and other study conducted by Norhammar *et al.* (1999), infarct size measured by enzyme release was not related to poor outcome.

In conclusion, admission blood glucose level can be added to the other risk predictors in non-diabetic patients with acute MI. Further prospective studies are needed to determine the blood glucose level that can be used as risk predictor. Although no cardiovascular studies of interventions that lower blood glucose in non-diabetics people have been reported, this may be important especially for patients with history of previous MI. Also randomized trials designed to test the impact of specific management strategies on outcome according to initial risk classification are warranted. Follow up studies on patients with high admission blood glucose are required to find out if these patients are at a higher risk to develop diabetes mellitus if so, screening at more frequent intervals and application of secondary preventive measures, such as exercise, weight loss and smoking cessation, are needed.

REFERENCES

Antman, E.M. & Braumwald, E. 1998. Acute myocardial infarction. In: Harrison's principles of internal medicine, 14th edn. (Edited by Fauci, A. Braunwald, E., Isselbacher, K. Wilson, J., Martin, J.B., Kasper, D.L., Hauser, S.L. & Longo, D.L.) Vol. 1, pp1352- 1365. McGraw-Hill, USA.

Bellodi, G. Manicardi, V., Malvasi, V., Veneri, L., Bernini, G., Bossini, P., Distefano, S., Magnanini, G., Muratori, L. & Rossi, G. 1989. Hyperglycemia and prognosis of acute myocardial infarction in patients without diabetes mellitus. American Journal of Cardiology 64:885-888.

Becker, R.C., Burns, M., Gore, J.M., Spencer, F.A., Ball, S.P., French, W., Lambrew, C., Bowlby, L., Hilbe, J. & Rogers, W.J. 1998. Early assessment and in-hospital

management of patients with acute myocardial infarction at increased risk for adverse outcome: a nationwide perspective of current clinical practice. The national registry of myocardial infarction (NRMI-2) participants. American Heart Journal 135(5Pt 1): 786-796.

Bjornholt, J.V., Erikssen, G., Aaser, E., Sandvik, L., Nitter-Hange, S., Jervell, J., Erikseen, J. & Thaulow, E. 1999. Fasting blood glucose:an underestimated risk factor for cardiovascular death: results from a 22-year follow-up of healthy non-diabetic men. Diabetes Care 22:45-49.

Coutinho, M., Gerstein, H.C., Wang, Y. & Yusuf, S. 1999. The relation between glucose and incident cardiovascular events: a metaregression analysis of published data from 20 studies of 95,783 individuals followed for 12.4 years. Diabetes Care 22 : 233 - 240.

Fuller, J.H., Shipley, M.J., Rose, G., Jarrett, R.J. & Keen, H. 1980. Coronary heart disease risk and impaired glucose tolerance : the Whitehall study. Lancet 8183:1373 - 1376.

Gersrein, H.C., Pais, P., Pogue, J. & Yusuf, S. 1999. Relationship of glucose and insulin levels to the risk of myocardial infarction : a case-control study. Journal of American College of Cardiology 33(3): 612-619.

Kersten, J.R., Schemeling, T.J., Orth, K.G., Pagel, P.S. & Warltier, D.C 1998. Acute hyperglycemia abolish ischemic preconditioning in vivo. American Journal of Physiology 275 (2pt2): H721-725.

Mak, K., Mah, P., Tey, B., Sin, F. & Chia, G 1993. Fasting blood sugar level : a determinant for in hospital outcome in patients with first myocardial infarction and without glucose intolerance. Annals of the Academy of Medicine, Singapore 22: 291-295.

Malmberg, K., Ryden, L., Hamsten, A., Herlitz, J., Waldenstrom, A. & Wedel, H. 1997. Mortality prediction in diabetic patients with myocardial infarction: experiences from the DIGAMI study. Cardiovascular Research 34:248-253.

Norhammar, A.M., Ryden, L. & Malmber, K. 1999. Admission plasma glucose: independent risk factor for long term prognosis after myocardial infarction even in non-diabetic patients. Diabetes Care 22(11): 1827-1831.

Oswald, G.A., Smith, C.C.T., Betteridge, D.J. & Yudkin, J.S. 1986. Determinants and importance of stress hyperglycemia in non-diabetic patients with myocardial infarction. British Medical Journal 293:917-922.

Scheidt-Nave, C., Barrett-Connor, E., Wingard, D.L., Cohn, B.A. & Edelstein, S.L. 1991. Sex differences in fasting glycemia as a factor for ischemic heart disease death. American Journal of Epidemiology 133:565-576.

UK Prospective Diabetes Study Group (UKPDS). 1998. Intense blood glucose control with sulphonylurea or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet 352:837-853.

Winter, K.J. & Eisenberg, P.R. 1995. Ischemic heart disease. In: The Washington Manual, 28th edn. (Edited by Ewald, G.A. & McKenzie, C.R.), pp 94-97. Little Brown, Boston, Massachusetts.

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