Blood Glucose Level Determination in Acute Myocardial Infarction

*NAHEED HANEEF**
*AZHAR FARUQUI***
*M. A. NAJEEB****

SUMMARY:

Hyperglycaemia is often associated with acute myocardial infarction. We observed anecdotally that routine dextrose-water infusion plays some part in raising the blood glucose level during initial treatment in non-diabetic patients with infarction. We tested our obervation in a group of 14 patients. In 5 out of 14 patients, I.V. line was maintained with heparin lock and in remaining 9, I.V. line was maintained with dextrose-water drip. Blood glucose level was estimated before and after I.V. line maintenance and then daily for 2 days and then on alternate days. It was observed that those patients who were not given dextrose infusion did not show any significant rise in blood glucose level, while 60% of those who were on I.V. dextrose infusion showed some rise in blood glucose level while I.V. line was maintained, and later when I.V. line was discontinued after 24-48 hrs, the blood glucose level fell down to within normal limits. These findings suggest that initial hyperglycaemia in non-diabetic patients may be due to I.V. dextrose infusion.

INTRODUCTION:

There are different theories regarding the cause of hyperglycaemia associated with infarction: (A) Increased adrenocortical hormone production as a response to stress and chest pain which causes increase gluconeogenesis, (B) A circulatory disturbance in the brain with functional changes in the hypothalamic region (Roab & Rabinowitz), (C) Hypersecretion of adrenaline due to stress causing mobilization of glycogen, (D) Liver cell necrosis due to shock upsetting CHO metabolism, (E) Suppression of insulin due to reduction in pancreatic blood flow as a consequence of Splanchic vaso-constriction following severe left ventricular failure and augmented circulating catacholamines due to high sympathetic nervous system activity and (F) Recently, it has been suggested that hyperglycaemia is due to pre-existing latent undiagnosed diabetes rather than "Stress Hyperglycaemia". We observed that dextrose-water infusion played some part in raising the blood glucose level during initial treatment in non-diabetics and the present pilot study was an attempt confirm this observation.

MATERIAL AND METHODS:

In this study a group of 14 consecutive patients with acute myocardial infarction were included, who fulfilled the WHO criteria of AMI. In addition the following requirements were also mandatory:

1) No prior history of ischaemic heart disease.
2) No known history of diabetes or other endocrine disorder.
3) No family history of diabetes.

Characteristies of 14 patients studied were as follows. All were male except one, their ages ranged from 30 to 60 years, the mean age being 45 years. All having no previous history of hospitalization. Two were suffering from hypertension of 3 years and of 6 months duration.

From: National Institute of Cardiovascular Diseases (Pakistan) Karachi.
* RMO Southward.
** Consultant Cardiologist.
*** Director

All were smokers except for one female. One had family history of heart attack and one for hypertension. All had no significant past history of any disease except one who had history of renal colic 20 years ago and one with atypical chest pain off and on since 1 year.

Of the 14, 4 had acute inferior wall infarction, 4 had anterior wall infarction, 4 had anterolateral infarction and 2 had anteroseptal infarction. All patients fulfilled all 3 criteria of WHO and all had evolutionary changes of transmural infarction.

When a patient came in emergency of NICVD who fulfilled the above criteria, before I.V. line maintenance, venous blood was drawn for blood glucose level. After taking sample of blood for glucose, patients were randomized into group I and group II.

GROUP – I :

Consisted of 9 patients in whom after taking blood sample for glucose I.V. line was maintained with dextrose-water drip while rest of care was standard.

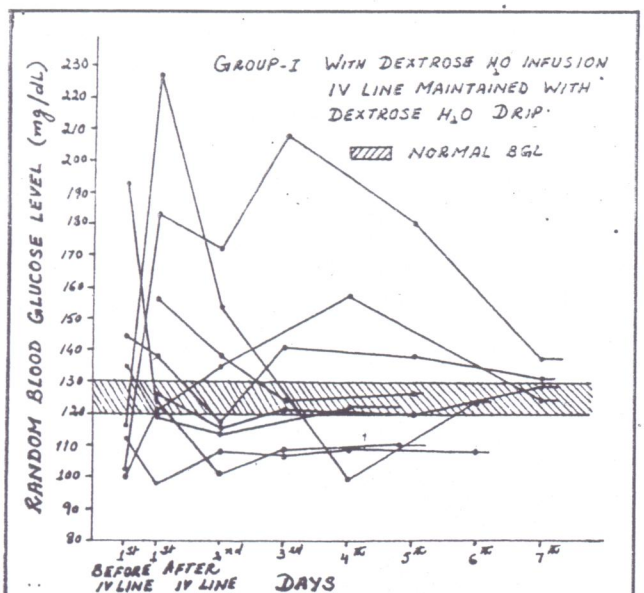

Fig 1. Blood Sugar levels in group I.

GROUP – II :

Consisted of 5 patients in whom after taking blood sample for glucose I.V. line was maintained with heparin lock only, while rest of care was standard.

After reaching the ward a second sample of blood for glucose from opposite arm was drawn

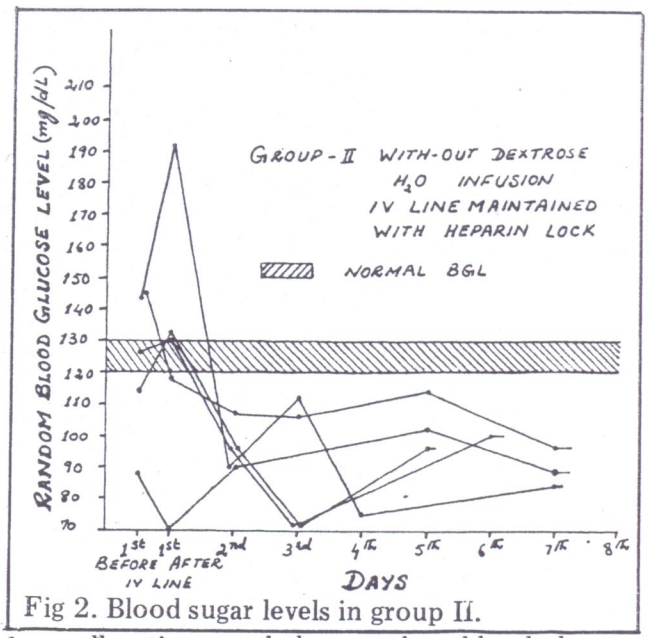

Fig 2. Blood sugar levels in group II.

from all patients and then random blood glucose levels were obtained daily for 2 days, and then on alternate days.

All these patients were on soft diet and were receiving standard treatments. Two samples of blood for enzyme were collected on each morning for 2 consecutive days.

Out of 14 patients only one patient developed heart failure and ventricular fibrillation from Group I, who was readmitted for 3 days in CCU and was discharged on 10th day at which time he was fully mobilized. The remaining patients were discharged without any complication on the 7th days. Results are summarized in Figs 1, 2 and 3.

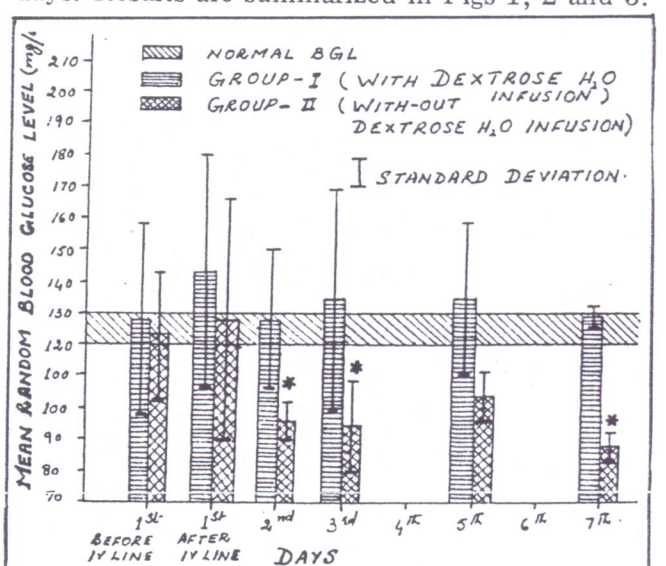

Fig 3. Bar diagrams combining the data in Fig 1 and Fig2. * p values <0.05

DISCUSSION :

Many studies have been done on hyperglycaemia following infarction. But no one exact cause of hyperglycaemia is recognized Roab & Rabinowitz (1936) found hyperglycaemia and glycosuria in all 12 cases of AMI studied by them within two weeks of the attack. According to these authors, hyperglycaemia is due to disturbance in the brain which causes functional changes in hypothalamic region and this hyperglycaemia and glycosuria is of transient nature. Their belief that glycosuria is of temporary significance was challenged by Gold-berger et. al. (1945) who found most of his patients became diabetic after the Acute MI. Ellenberg (1952) reviewing the literature has stated that liver cell necrosis following AMI, upset CHO metabolism and they also postulated that hyperglycaemia following AMI was due to mobilization of glycogen by increased adrenaline and increased gluconeogenesis under the influence of increased adrenocortical hormones. Sowton (1962) stated that hyperglycaemia is due to latent diabetes and the presence of latent diabetes in patients presenting with cardiac infarction is often not recognized, perhaps because it is rarely looked for. Reaven G (1965) stated that those with a previous MI have a higher fasting blood glucose concentration and do not dipose of an oral glucose load as efficiently as a comparable group without manifest coronary artery disease. Vallence-Queen & Ashlon (1963) found increased level of insulin antagonism (Synalbumen) that persisted for 3 months after the acute myocardial infarction.

Datty and Nanda (1967) in a follow-up study revealed that the incidence of hyperglycaemia was 70% at the onset of the attack and at the end of 7 to 10 days. The hyperglycaemia was transient in 38% of the cases and its duration being 11 to 30 days in 23% and between one month and one or 2 years in 15%. Those with initial normoglycaemia 35% did not show any tendency to diabetes. Volore C. Thomas (1967) stated that amount of nor-adrenaline and adrenaline excreted was commonly high during the initial period of AMI and causes suppression of insulin for a few days. S.P. Allison (1969) found in his study that suppression of insulin and hyperglycaemia occurs following AMI. N.J. Velter (1974) found in their study low plasm insulin level and blood glucose and cortisol concentration after AMI. Recently S.J. Husband (1983) in their study postulated that patients with suspected AMI, who are hyperglycaemic (> 10 mmol/L) are likely to be pre-existing undiagnosed diabetic rather than "Strees" Hyperglycaemics.

In our study following results were obtained. In Gp. I 5 patients (55.6%) out of 9 showed rise in blood glucose level after I.V. line maintenance with dextrose-water drip compared to before I.V. line maintenance. And later when I.V. line discontinued after 24-48 hrs, the blood glucose level came down within normal limits at the end of the week. In Gp. II as presented, 4 patients (80%) out of 5 showed no significant change in blood glucose level before and after I.V. line maintenance with heparin lock. Only one patient (20%) out of 5 showed rise in blood glucose level after I.V. line with heparin lock. We do not know the cause of hyperglycaemia in this patient and it may be due to stress and so-called "latent diabetes" or he may be a previously uniagnosed diabetic.

These results were plotted on a graph. Each graph (Fig. 1 and 2) showed variation in blood glucose level before and after I.V. line maintenance during a period of 7 days. For example, one patient with acute infarction having blood glucose level below normal, i.e., 102 mg/dl before his I.V. line was maintained, few hours after the I.V. line, his blood sugar was repeated which showed a rise of blood glucose to 183 mg/dl. This level remained raised on 2nd day, i.e., after 24 hours (172 mg/dl) and on 3rd day after 48 hours —208 ml/dl, later the I.V. was discontinued. On 5th day the blood glucose level came down to 180 mg/dl and on 7th day it reached to slightly above the upper limit of normal, i.e., 136 mg/dl.

Comparison of groups I and II is shown in Fig. 3. There was no significant difference ($P < 0.05$) before and after I.V. line maintenance. But there was statistically significant differences ($P<0.05$) in comparisons of blood glucose levels on 2nd, 3rd and 7th days.

These results support our previous observation that dextrose-water infusion plays some part in raising blood glucose level in patients with AMI, during the initial phase of infarction as has been discussed.

Thus there is in such patients an iatrogenic cause of hyperglycaemia created by giving them dextrose infusion which creates a new problem whether the patient is actually diabetic or not.

For this further investigation is necessary with wastage of time and resources. Therefore, we advocate using I.V. heparin lock to maintain I.V. access rather than I.V. dextrose - water slow infusion, unless a special reason to do so exists, e.g., nausea or vomiting or hypoglycaemia. It must be pointed out that this was a small pilot study and is in no way a large scale definitive study on the various causes or mechanism of post infarction hyperglycaemia. Our purpose was limited only to comparison of I.V. dextrose infusion versus simple I.V. heparin lock and blood sugar levels.

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