Blood glucose in the CCU: time to measure

See page 1102 for the article to which this Editorial refers

For decades it has been known that patients with diabetes mellitus have an increased case fatality after acute myocardial infarction. Recently, this knowledge has been extended to unstable angina or non-Q wave myocardial infarction\(^1\). With the growing prevalence of type 2 diabetes and the continued higher risk of heart disease in diabetic subjects\(^2\) it can be anticipated that the impact of diabetes as a risk factor for coronary heart disease will increase. Secondary interventions with beta-blockers, ACE inhibitors and statins have been proven to reduce mortality after myocardial infarction in patients with diabetes to at least the same extent as in patients without diabetes. However, these interventions seem to be under utilized in diabetic subjects\(^3\), in whom besides conventional treatment modalities for myocardial infarction, intensive insulin treatment may further improve outcome in these patients\(^4\). As diabetes is a considerable risk factor and the potential benefit of conventional and other treatments is great, it is essential to know whether a myocardial infarction patient is diabetic or not.

The prevalence of diabetes in myocardial infarction populations is approximately 20%, but a regional variation is reported. When employing an oral glucose tolerance test in survivors of myocardial infarction of 2–3 months, the prevalence of undiagnosed diabetes was estimated at about 4.5%\(^5,6\). In one study this group of patients was found to have increased in-hospital mortality\(^5\). Diagnosing new cases of diabetes in the setting of acute myocardial infarction is problematic since raised blood glucose can, at least in part, be attributed to physiological ‘stress’, mediated by catecholamines or perhaps primarily by cortisol\(^7\). Thus, it has been suggested that glycylated haemoglobin (HbA\(_1c\)) be used, since it can distinguish between stress–hyperglycaemia and hyperglycaemia due to diabetes\(^5,6,8\).

In this issue Tenerz and colleagues\(^9\) report on the prevalence of undiagnosed diabetes in a contemporary unselected population with myocardial infarction. In addition, they sought to find out whether casual blood glucose and HbA\(_1c\) can be used to identify patients with previously unknown diabetes. Tenerz et al. sought to classify the patients according to WHO criteria from 1985, proposed WHO criteria from 1998 and criteria from the American Diabetes Association 1997, the two latter with a lower diagnostic blood glucose limit for diabetes. New cases of diabetes were recognized by an oral glucose tolerance test 2–3 months after discharge, but no tests were performed in patients with normal blood glucose and some were lost to follow-up. The main finding was a 4% prevalence of undiagnosed diabetes based on the 1985 WHO criteria, while 20% had previously diagnosed diabetes (the corresponding figures for the 1998 proposed WHO and the 1997 ADA criteria were 5% and 23%). It was confirmed that raised admission blood glucose is not a reliable measure of diabetes. Only 50% of those with admission blood glucose \(\geq 11.1\) mmol.l\(^{-1}\) turned out to have diabetes. With regard to HbA\(_1c\), there was significant overlapping between patients with known diabetes, with newly diagnosed diabetes and without diabetes. If the diagnosis of diabetes had been based on fasting blood glucose measured on day 5 after the myocardial infarction, three times as many would have been ‘diagnosed’ as diabetics. It is possible that some patients who were in fact diabetic on day five reverted their hyperglycaemia by losing weight during the first 2–3 months after the myocardial infarction. However, the raised blood glucose on day 5 may reflect prolonged stress–hyperglycaemia, since both urinary cortisol and blood glucose have been shown to be raised on day 4 compared to day 8 after myocardial infarction\(^10\).

With these and earlier data in mind we must accept that measuring blood glucose or HbA\(_1c\) in a patient with acute coronary syndrome in the CCU is not a valid tool for diagnosing diabetes. However, there are other reasons for performing these measurements. First of all, fasting blood glucose in particular, can be used to identify those with values in the normal range, who are therefore not diabetic, from those who have to be examined again 2–3 months later. The data of Tenerz et al.\(^9\) show that after excluding 61 patients with previously known diabetes and 123 with fasting blood glucose below the upper normal limit, 12 of 121 (10%) turned out to have diabetes on later
examination. Hence it seems worthwhile performing screening for diabetes in selected patients 2–3 months after they have been hospitalized for myocardial infarction. By 1975 it was demonstrated that fasting blood glucose measured within 72 h of myocardial infarction was a good guide to the prediction of the subsequent development of diabetes. Very few with a normal fasting blood glucose developed diabetes 6 years after the myocardial infarction, while diabetes did develop in most with elevated fasting blood glucose. When further increasing the selection of patients for oral glucose tolerance test by either confining the test to those with an elevated admission blood glucose or with a clearly abnormal HbA1c a very high specificity for diabetes is obtained.

Another reason for measuring either admission or fasting blood glucose, or even HbA1c, is the prognostic value of these measurements. Recently, a meta-analysis of 15 studies reporting in-hospital mortality or rates of congestive heart failure in relation to blood glucose on admission was published. Stress–hyperglycaemia was associated with an increased risk of in-hospital mortality in patients both with and without diabetes and in the latter group the risk of congestive heart failure was increased as well. In a study of 253 myocardial infarction patients without diabetes, HbA1c was found to be an independent predictor of 1-year mortality, probably reflecting a higher risk in patients with impaired glucose tolerance associated with the metabolic syndrome. More knowledge on pathophysiology and interventional possibilities is required for the group of patients with blood glucose and HbA1c values in the upper part of the normal range. At least part of their increased risk can be attributed to co-existing risk factors, but the importance of the relative insulin deficiency is emphasized by the effect on mortality of insulin administration to myocardial infarction patients without diabetes.

In conclusion, secondary intervention after myocardial infarction is an important task for cardiology departments. It is becoming common practice to measure lipid profiles on admission and arrange follow-up of these parameters. It appears to be of equal importance to recognize the glycometabolic state of the patient. Traditionally, diabetes is taken care of by the endocrinologist or the general practitioner, but since a substantial fraction of type 2 diabetic patients present with cardiovascular disease, screening for diabetes in the cardiology department is reasonable. For patients without known diabetes, who are admitted to the CCU for acute coronary syndrome, it is recommended that fasting blood glucose is measured before discharge. Follow-up 2–3 months after the acute event should be planned for those with elevated values. If further prognostic information is required, admission blood glucose and HbA1c may be measured. The usage of these simple tools will allow identification of a group of high-risk subjects in whom benefit from aggressive secondary intervention is well documented.

I. GUSTAFSSON
P. HILDEBRANDT
Department of Cardiology and Endocrinology, Frederiksborg University Hospital, Copenhagen, Denmark

References