Blood Glucose Monitoring in Hospitalized Patients

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Hyperglycemia is a frequently observed metabolic derangement, reported to be present in approximately one-third of hospitalized patients [1,2]. Diabetes care during hospitalization has received increasing attention during the last decade, and a number of recent studies have reported that glucose control during hospitalization in intensive care units is indeed clinically meaningful, both for individuals with known diabetes and for patients without this diagnosis [3-5]. Despite these reports, some clinicians resist attempts to increase glycemic control in the hospitalized diabetic patient, primarily due to anxiety on the part of the medical staff regarding increased risk of hypoglycemic events and even death [6,7]. These fears notwithstanding, many clinicians view the hospitalization period as a window of opportunity to improve diabetes care.

In recent years, the institutional glucometer has replaced the old personal glucometer still in use in many hospitals in Israel and abroad. But at hospitals such as Assaf Harofeh in Zerifin and Wolfson in Holon, for example, the new glucometer is in use and this instrument facilitates hospital care of the diabetic patient through processes such as documentation, follow-up, quality control, data analysis, etc. [8]. This enables identifying and tracking trends regarding the level of the individual patient and the patient population at large. These instruments are also more accurate and more reliable than older glucometers [9]. In Israel, the National Diabetes Council has recommended the use of these institutional glucometers to the Ministry of Health, noting that they facilitate diabetes care and improve glucose homeostasis during the hospitalization of diabetic patients.

The main purpose of implementing projects such as the Program for the Treatment of the Hospitalized Diabetic Patient (PTHDP) at Wolfson Medical Center is to utilize the diabetic patient’s hospitalization as an opportunity to empower patients and help them recognize their ability to control their disease [8].

In agreement with the findings of our report [8], the present study by Buchs and Rapaport in this issue of IMAJ [10] found that the glucometer was an effective tool to facilitate glucose homeostasis in the hospitalized diabetic patient. Nevertheless, a few methodologic questions emerge from the reported data, three of which deserve mention. First, blood glucose was recorded from a single daily capillary measure for most patients. Because insulin-treated patients are expected to monitor blood glucose two to four times daily at home, it would have been instructive, and certainly clinically important, to have done so during the hospitalization period. The average glucose level reported in this study’s population is 161 mg/dl, which corresponds to an HbA1c level of about 7.4%. This is a most impressive achievement in a diabetic patient population, particularly during the stress of hospitalization. HbA1c levels are typically around 8% in community-dwelling diabetic individuals, so such low levels during illness may reflect exceptional diabetes care in the community setting.

Third, glucose values on the first day of hospitalization were 132–155 mg/dl and remained stable throughout hospitalization until discharge. It is not clear whether these initial values were obtained in the emergency room, but they do seem lower than expected and are consistent with unusually good diabetes care prior to hospitalization.

Despite our questions regarding methodology, we agree with Buchs and co-authors that the time has come to adopt the electronic database. It is clearly superior to the current paper-based system. And we agree that the institutional glucometer is an important step in that direction, one that will improve patient care and facilitate patient autonomy.

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References

**Capsule**

**Pathogen recognition receptor gene NOD2 increases the risk of developing Crohn's disease**

Polymorphisms in the pathogen recognition receptor gene NOD2, found in 10–20% of North American individuals with Crohn's disease, increase the risk of developing the condition. Findings by Rahman et al. suggest that such polymorphisms result in dysregulated activation of regulatory T cells, cells that can shield the body from excess immune activation. Individuals with Crohn's disease homozygous for a disease-associated variant of NOD2 have decreased numbers of Treg cells in their intestines. Normally, stimulation of NOD2 protects Treg cells from Fas ligand-mediated apoptosis by upregulating anti-apoptotic machinery. The researchers found that this mechanism seems to go awry in human Treg cells either deficient in NOD2 or expressing disease-associated variants. Such cells showed increased susceptibility to apoptosis in cell culture. The findings suggest that activation of wild-type NOD2 is protective. Other researchers have observed increased expression of Fas ligand in the inflamed intestine, which could decrease Treg cell numbers in these individuals and lead to excess T cell activation.

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**Capsule**

**Muscle dysfunction caused by a K\textsubscript{ATP} channel mutation in neonatal diabetes is neuronal in origin**

Gain-of-function mutations in Kir6.2 (KCNJ11), the pore-forming subunit of the adenosine triphosphate (ATP)-sensitive potassium (K\textsubscript{ATP}) channel, cause neonatal diabetes. Many patients also suffer from hypotonia (weak and flaccid muscles) and balance problems. The diabetes arises from suppressed insulin secretion by overactive K\textsubscript{ATP} channels in pancreatic \(\beta\)-cells, but the source of the motor phenotype is unknown. By using mice carrying a human Kir6.2 mutation (Val\textsuperscript{59}Met\textsuperscript{59}) targeted to either muscle or nerve, Clark et al. showed that analogous motor impairments originate in the central nervous system rather than in muscle or peripheral nerves. The authors also identified locomotor hyperactivity as a feature of K\textsubscript{ATP} channel overactivity. These findings suggest that drugs targeted against neuronal, rather than muscle, K\textsubscript{ATP} channels are needed to treat the motor deficits and that such drugs require high blood-brain barrier permeability.

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**Capsule**

**Dangerous dengue provocation by antibody response promotes virus infection of cells**

One problem with dengue virus is that one infection does not protect against a subsequent infection; secondary infections can result in the severe immunopathology of dengue hemorrhagic fever. Dejnirattisai and team derived a panel of monoclonal antibodies specific for dengue viruses. These antibodies were mainly directed against the dengue virus precursor membrane protein (prM), and most cross-reacted with all four dengue serotypes. The antibodies were not capable of fully neutralizing the virus, but instead promoted immune responses over a wide range of concentrations. During virus production and virion assembly, maturation of prM is often incomplete, and, consequently, a major part of the host’s natural antibody response recognizes a component that is present in variable numbers on the virion. Thus, rather than resulting in complete neutralization, the antibody response promotes virus infection of cells that carry receptors for antibodies.

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