



# Association of British Clinical Diabetologists Spring 2009 Meeting Abstracts

**(P1): Body mass index, weight and insulin dose as selection criteria for islet transplantation recipients?**

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**Introduction.** Selection criteria for islet transplantation include body mass index (BMI), weight and insulin dose (ID) as clinical indicators of insulin resistance. Identification of patients with high levels of insulin resistance is essential as it is undesirable to transplant a small beta cell mass in the face of significant insulin resistance, this mismatch being likely to impact on transplant outcome. There are few data comparing these surrogate markers to the gold standard assessment of insulin resistance (euglycaemic hyperinsulinaemic clamp) in patients with type 1 diabetes mellitus (T1DM). We aimed to define the range of insulin resistance within this patient group as well as the validity of BMI, weight and insulin dose as measures for estimating insulin resistance.

**Materials and methods.** We assessed insulin resistance in 15 patients with T1DM awaiting islet or pancreas transplantation using the euglycaemic hyperinsulinaemic clamp method. We then correlated glucose disposal rates (GDRs) as estimated by the euglycaemic clamp to patient BMI, weight, insulin dose and daily insulin dose per kg.

**Results.** Patients demonstrated a wide range of insulin resistance with GDRs ranging from 149mg/m<sup>2</sup>/min-1 to 325mg/m<sup>2</sup>/min-1 comparable to the distribution of GDRs within a population of healthy subjects. We demonstrated no correlation between BMI and estimations of insulin sensitivity. Similarly, when GDRs were plotted against both total insulin dose per day (ID/day) or weight adjusted insulin dose (ID/kg/day), no significant correlation was demonstrated.

**Conclusions.** Islet transplantation in the face of high levels of insulin resistance may mean patients are less likely to achieve the same quality of metabolic control or maintain insulin independence for as long as their insulin sensitive counterparts. Thus, identification of patients with high levels of insulin resistance is clinically important in the context of clinical islet transplantation. Our data suggest that not all patients with T1DM are insulin sensitive and that measured insulin sensitivity fails to correlate reliably with BMI, weight and insulin dose. Simple assessments of weight/BMI and insulin dose, although shown to be important for islet transplant success, are perhaps not good clinical indicators of insulin sensitivity in patients with T1DM.

**(P2): Give me my pen back!**

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**Aim.** To assess satisfaction with treatment in diabetes inpatients allowed to self-manage their condition compared with controls.

**Method.** Patients with insulin-treated diabetes deemed able to self-manage their condition whilst an inpatient completed a standardised questionnaire (DTSQ-IP). Group 1 were allowed to self-manage; group 2 had their diabetes care delivered by ward nurses.

**Results.** In all, 22 patients were recruited over a 3-month period; 19/22 questionnaires were returned. Group demographics are shown in Table 1.

Patient satisfaction was greater in group 1 compared with group 2 (100% 'very satisfied' vs 0% respectively). Blood glucose control was perceived to be better and treatment more convenient and flexible in group 1. All patients in group 1 felt satisfied with their treatment plan compared with none in group 2. Including

Table 1. Group demographics (A Morgan *et al.*)

	Group 1 Self-managing	Group 2 Not self-managing
Size of sample	9	10
Years on insulin treatment: mean (range)	19.7 (1-52)	21.0 (5-39)
Age 16-40 years	2	4
Age 41-60 years	5	3
Age >60 years	2	3

meal appropriateness in this study has highlighted an interesting phenomenon: all patients had the same choice of meal and though dissatisfaction was expressed by group 1, there was greater dissatisfaction among group 2.

**Conclusion.** This study is limited by its size. However, there was overwhelming dissatisfaction with care in insulin-treated patients when the ability to self-manage was denied. This has been a powerful tool with which to recommend revision of trust policy.

**References**

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**(P3): The impact of the diabetes outreach team on effective implementation of the care pathway for diabetes management of patients admitted with acute myocardial infarction (AMI)**

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Wolverhampton diabetes outreach team (DOT) proactively reviews patients with diabetes admitted to all areas of the hospital. One of its important roles on the cardiac unit is to oversee the implementation of the care pathway for diabetes management in patients with AMI. We have audited 30 consecutive patients admitted with STEMI or NSTEMI and either established diabetes or an admission blood glucose  $\geq 11$ mmol/L. Mean age was 64 years, 67% of patients were males and 90% were previously known to have diabetes. In all, 93% of patients were reviewed by the DOT and median time to review was 1 day. During the first 24 hours of admission, overall management was appropriate in 44% of patients with non-commencement of intravenous insulin being the main contributor to non-adherence. Subsequent to the involvement of the DOT, the proportion of patients appropriately managed during the remainder of the stay went up to 75%. Structured review of diabetes care was undertaken in 72%, appropriate communication sent to the GP in 100% and a long-term management plan documented in 93% of patients. Mean blood glucose during the first 24 hours was 10.5mmol/L and during the remainder of the hospital stay was 9.8mmol/L.

**Conclusions.** Proactive inpatient specialist review of all diabetes patients with an AMI has a positive impact on the implementation of cardiac care pathway and on the overall care of these patients. Further education of the cardiac team is required to improve care during the initial 24 hours of admission.



## ABSTRACTS



## Spring 2009 Meeting of the Association of British Clinical Diabetologists

**(P4): Metabolic measures of graft function after pancreas transplantation**

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**Introduction.** At present there is no simple means of monitoring metabolic function after pancreas transplantation. Random or fasting glucose and HbA<sub>1c</sub> are all used. However, these measures may remain within the normal range despite significant changes in insulin resistance and beta cell mass. In many centres, patients are stratified according to glucose tolerance determined by an oral glucose tolerance test (OGTT). These simple measures do not, however, interrogate beta cell function or beta cell mass and may be confounded by different levels of insulin resistance. We aim to determine a rate of beta cell decline as a measure of graft function post-transplantation and adjust for individual variation in insulin resistance.

**Materials and methods.** We performed metabolic assessments in 2 simultaneous pancreas and kidney patients, 2 pancreas alone and 2 pancreas after kidney transplant patients. Metabolic assessments included a frequently sampled 75g OGTT, an arginine and intravenous glucose tolerance test (Arg IVGTT) to allow estimation of beta cell mass, and a euglycaemic hyperinsulinaemic clamp study to estimate insulin sensitivity. The assessments were completed at 3 months post-transplant in all patients, and at 3 and 12 months after transplantation in 4 patients.

**Results.** On OGTT, 4 patients exhibited normal glucose tolerance (NGT), 1 patient had impaired glucose tolerance (IGT) and 1 had diabetic glucose tolerance (DGT) at 3 months post-transplant. At 12 months the response to a glucose load was equivalent to that seen at 3 months in 3 patients (2 NGT and 1 IGT) despite significant increases in both insulin sensitivity and acute insulin response to arginine and glucose. The patient who demonstrated DGT at 3 months showed IGT at 12 months despite deterioration in both insulin sensitivity and acute insulin response to glucose.

**Conclusions.** Detailed assessment of glucose homeostasis reveals abnormalities in a significant proportion of patients receiving whole pancreas transplants. Recognition of these abnormalities may allow targeted interventions which are able to prolong graft function.

**(P5): Is intervention after acute myocardial infarction effective in managing patients with abnormal glucose metabolism and cardiovascular disease?**John S Kane<sup>1</sup>, Kathryn J Kelly<sup>1</sup>, Jane Close<sup>2</sup>, Paul Daniels<sup>3</sup>, Robert West<sup>1</sup>, Lucinda KM Summers<sup>1</sup><sup>1</sup>University of Leeds; <sup>2</sup>Leeds Teaching Hospitals Trust; <sup>3</sup>Takeda UK

**Background.** Type 2 diabetes mellitus and impaired glucose tolerance (IGT) are major risk factors for cardiovascular disease (CVD) and affected patients have poor outcome after acute myocardial infarction (AMI).

**Aims.** To work with the regional cardiology tertiary referral centre to identify known or undiagnosed diabetes or IGT in AMI patients, and target these patients to decrease cardiovascular risk.

**Methods.** We audited the effectiveness of a new referral process and one-stop hospital cardiovascular risk clinic in providing evidence-based lifestyle advice and pharmacological intervention to patients region-wide with existing or newly diagnosed diabetes or IGT after AMI. Data were analysed at baseline and 6 months over a year.

**Results.** Only 0.02% of referrals failed to attend clinic; 92 attended: mean age 60.5 years, 85.4% white, 75.3% male; 9 had IGT. Patients were overweight/obese; mean baseline BMI (standard deviation) and waist circumference were 30.6(4.6)kg/m<sup>2</sup> and 104(12.7)cm. Baseline mean HbA<sub>1c</sub> was 7.6% (in those with diabetes), total cholesterol 4.1mmol/L, blood pressure 134/74. Waist circumference decreased by 11cm (p=0.18). HbA<sub>1c</sub> decreased by 0.3% (p=0.13) with no change in BMI, lipid profile or blood pressure.

**Conclusion.** The referral process worked well and the clinic was popular. Baseline measurements were reasonable, especially as

they included patients newly diagnosed with diabetes. BMI, waist circumference, HbA<sub>1c</sub>, blood pressure and lipids deteriorate over time, so the decrease in waist circumference and HbA<sub>1c</sub> and maintenance of BMI, cholesterol and blood pressure are clinically significant and the intervention effective. Further analysis will determine how effective this intervention is long term.

**(P6): Case report of hyperglycaemia-associated hemiballismus-hemichorea in a type 2 diabetic man**YS Cheah<sup>1</sup>, R Jain<sup>2</sup>, D Hopkins<sup>1</sup>, L Nashef<sup>2</sup>, NA Sibtain<sup>3</sup><sup>1</sup>Department of Diabetes, <sup>2</sup>Department of Neurology, <sup>3</sup>Department of Neuroradiology, King's College Hospital, London

We report a case of hyperglycaemia-associated hemiballismus-hemichorea (HBHC) in a 61-year-old Caucasian man with long-standing type 2 diabetes mellitus (T2DM). This had previously been well controlled with oral hypoglycaemic agents until 2006, when his HbA<sub>1c</sub> had risen sharply to 11.4%. Despite the introduction of insulin, glycaemic control remained poor. He had previously developed microalbuminuria in 2004, and proliferative diabetic retinopathy, necessitating pan-retinal photocoagulation, in 2000.

He was admitted in February 2009 with a 15-day history of sudden onset, progressive involuntary jerky right leg movements, associated with similar, but intermittent movements in his right arm. Clinical examination revealed predominant choreic movements of the right leg, with intermittent athetoid and ballistic movements. Fundoscopic examination revealed no new retinopathy. Admission HbA<sub>1c</sub> was 13.8%, but there was no evidence of a hyperosmotic hyperglycaemic non-ketotic state. An MRI scan of his brain revealed hyperintense signals on the T1- and T2-weighted images in the left putamen, suggestive of a hyperglycaemia-related putaminal lesion. Improvement in his glycaemic control and commencement of haloperidol improved these involuntary movements.

Chorea-ballism is an uncommon hyperglycaemia-associated movement disorder. Published case reports of patients with hyperglycaemia-associated chorea-ballism reveal a predominance of elderly female patients of East Asian origin, with non-ketotic hyperglycaemia, with mainly unilateral chorea. Our case of HBHC is unusual in that he is Caucasian. Normalisation of glycaemia often rapidly improves HBHC; however, our case highlights the importance of ophthalmological examination prior to achieving a euglycaemic state quickly, in patients who often have other diabetes-related complications.

**(P7): Survey of blood glucose monitoring and referral to specialist diabetes team in a district general hospital**Amalia Iliopoulou, Kathryn Jolly, Caroline Stroud, Ryan D'Costa  
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**Aim.** To assess the adequacy of blood glucose monitoring among diabetic patients, action undertaken and referral for specialist advice.

**Methods.** One-day survey of all inpatients with diabetes using a standardised questionnaire.

Fifty patients were identified with diabetes, 44 type 2, 4 type 1 and 2 indeterminate, accounting for 13.7% of inpatients. Thirty-six were under medical and 14 under surgical care. Information was obtained from examination of case notes, glucose monitoring and prescription charts.

**Results.** Except 1 patient, all had appropriate monitoring charts. Insulin type and dose were prescribed correctly. The abbreviation U for units was used twice. The frequency of capillary blood glucose testing was adequate as per hospital guidelines, apart from 3 cases (6%).

Twenty-six (52%) patients required adjustment of hypoglycaemic treatment; 9 had changes instigated by the team, 7 were referred to the diabetes team and 10 had no intervention.





Physicians were more likely to make appropriate changes to diabetes treatment than surgeons who tended to refer to the diabetes team.

The diabetes team had been consulted in 14% of patients with diabetes, but only in 27% of those requiring treatment changes.

**Conclusions.** Although blood glucose monitoring was generally appropriate, we were concerned that appropriate changes to medication are not being made in most cases. The diabetes team is not involved in a majority of cases. While education of medical and nursing staff will help, a dedicated diabetes inpatient team actively seeking out and sorting out issues on a daily basis will positively impact on inpatient diabetes care, potentially reducing hospital stay and re-admission rates.

### **(P8): Where has all the insulin gone? True insulin resistance or resistance to taking insulin?**

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High prescribed doses of insulin imply insulin resistance but this is not necessarily true, as the following cases demonstrate.

**Case 1.** A 28-year-old female presented with hyperglycaemia and severe hypertriglyceridaemia. She had Cushingoid habitus and acanthosis nigricans. Investigations for Cushing's syndrome were negative. She was given a basal bolus regimen of 300 units of insulin and metformin. She had very low adiponectin and high leptin levels. On the basis that she could have a PPAR gamma gene mutation, she was started on pioglitazone with marked improvement of her diabetes control and a sharp reduction in insulin doses.

**Case 2.** A 36-year-old female is taking 1400 units of U500 Humulin R, with an HbA<sub>1c</sub> of 15%. She has polycystic ovarian syndrome, hypertriglyceridaemia, acanthosis nigricans, pancreatitis and ischaemic heart disease. She stops taking her insulin when depressed. It is noticeable that her diabetes control is little better, whether she takes insulin or not. We believe that her insulin requirements are even higher than that prescribed.

**Case 3.** A 42-year-old Asian man (BMI of 20kg/m<sup>2</sup>) is prescribed 270 units of insulin, with an HbA<sub>1c</sub> of 14% that has remained high since diagnosis. He does not have acanthosis nigricans. He is GADA negative. Insulin and C-peptide levels and insulin dispensing data suggest non-compliance as the cause of his poor control.

**Conclusions.** This highlights the importance of looking for cutaneous markers of insulin resistance, namely acanthosis nigricans and skin tags which are invariably present in cases of true insulin resistance.

### **(P9): Undiagnosed diabetes in hospital inpatients – the feasibility of using routine laboratory blood glucose measurements to identify cases**

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**Background and aims.** In 2008 an estimated 350 000 people in England had diabetes that was either undiagnosed or not recorded on diabetes registers.<sup>1</sup>

The early identification of diabetes is crucial so that people can receive the advice, support and treatment they need to manage their condition. If diabetes is diagnosed early, then action can be taken to prevent or delay its complications.<sup>2</sup>

Blood glucose values are regularly taken from hospital inpatients, both on admission and throughout their stay; the aim of this study was to audit these values in order to estimate the extent of potentially undiagnosed diabetes in the inpatient population of a busy city general hospital.

**Patients and methods.** A search of all blood glucose values for inpatients in Leicester General Hospital during May 2008 identified 322 patients with a laboratory blood glucose of >7.8mmol/L. From

this cohort a random sample of 50 patients was selected and the number already known to have diabetes was established through a thorough search of local diabetes databases, available clinical records and discharge summaries.

**Results.** A total of 45/50 (90%) patients were already known to have a diagnosis of diabetes and 1 patient had died since the beginning of the study. Therefore, 4 patients (8%) with potentially undiagnosed diabetes were identified.

**Conclusion.** There appeared to be a small but significant number of patients with potentially undiagnosed diabetes present in the inpatient population; further investigation of these patients by their GP to define their glucose tolerance status may be a feasible approach in identifying new cases of diabetes.

### **References**

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### **(P10): A symptomatic diabetic patient with hyporeninaemic hypoaldosteronism**

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**Case.** A 50-year-old diabetic man was admitted with 6 months' history of lethargy, exertional dyspnoea, anorexia and weight loss. He developed diabetes in 1987. His medications included Insulatard 20 units od, NovoRapid 12 units tds, and mirtazapine 15mg od. On examination he had significant postural drop in blood pressure, conjunctival pallor and was clinically euvoelaemic. Apart from evidence of proximal myopathy and peripheral neuropathy, the rest of his clinical examination was unremarkable.

**Investigations.** He had hyponatraemia (127mmol/L), and hyperkalaemia (5.1mmol/L). Urea, creatinine, liver function tests, short synacthen test and thyroid function tests were all within normal range. Arterial blood gases were consistent with compensated metabolic acidosis, and calculated anion gap was normal (13.7). Plasma renin and aldosterone levels were both low at (<0.2pmol/m, 240pmol/L) respectively. A calcium chloride loading test failed to reduce urine pH below 5.9 despite systemic acidosis. Nerve conduction studies confirmed severe diabetic polyneuropathy.

**Diagnosis.** A working diagnosis of hyporeninaemic hypoaldosteronism or type 4 renal tubular acidosis secondary to diabetes was made. He was started on fludrocortisone treatment with gradual resolution of his presenting symptoms, and normalisation of serum potassium and sodium levels. Nevertheless, the postural hypotension persisted, which is probably related to diabetic autonomic neuropathy.

**Discussion.** Hyporeninaemic hypoaldosteronism is commonly seen in long-term diabetic patients. The pathophysiology is likely to be multifactorial including damage to the juxtaglomerular apparatus, adrenal cortex and/or principal cells resulting in lack of production and sensitivity to aldosterone, hyperkalaemia and acidosis. Appropriate biochemical diagnosis and treatment with synthetic mineralocorticoids could result in symptomatic improvement and reduce long-term complications.

**Conclusion.** This case highlights the importance of considering hyporeninaemic hypoaldosteronism in symptomatic diabetic patients presenting with non-specific symptoms of debility and biochemical evidence of mineralocorticoid deficiency despite normal adrenocortical provocation tests.