An integrated diabetes and mental health intervention for people with type 1 diabetes and severe disordered eating: a prospective proof-of concept cohort study

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Disordered eating is a phenomenon that affects approximately 30% of people with type 1 diabetes, and is characterised by omission of some amount of insulin secondary to fear of weight gain.[1,2] Depending on the dose and type of insulin omitted, the subsequent persistent hyperglycaemia, polyuria, and osmotic diuresis results in a catabolic and dehydrated state, characterised by rapid weight loss. Rapid re-insulinisation after diabetic ketoacidosis can lead to oedema and rapid weight gain, often experienced as uncomfortable physical sensations, reinforcing the individual's negative belief that insulin is causing weight gain, promoting a recurring cycle of insulin omission. Type 1 diabetes with disordered eating is associated with earlier onset of microvascular complications and a three-times increased risk of mortality compared with type 1 diabetes alone.[1,2] Low-intensity psychological interventions have not been found to be effective in improving glycaemic control in this group of people.[3] We

developed diagnostic criteria and a risk score for type 1 diabetes and disordered eating (the T1DE risk score). We also developed an innovative hub-and-spoke service that integrates diabetes and mental health treatments. We aimed to determine whether use of the service would improve HbA_{1c} rates of hospital admissions for diabetic ketoacidosis, severe hypoglycaemia, T1DE risk score, and patient-reported outcomes, consisting of symptoms of depression, anxiety, disordered eating, and diabetes distress.

This prospective uncontrolled proof-of-concept cohort study included patients with type 1 diabetes and disordered eating referred to the service between March 28, 2019, and June 6, 2022. The service was set in Greater London, UK, which has an ethnically and socioeconomically diverse population of 8·8 million residents, with the hub of the service being based in one teaching hospital (King's College Hospital, London, UK)and the spokes comprising 25 hospital-based diabetes centres.

We defined severe type 1 diabetes and disordered eating as a diagnosis of type 1 diabetes, pervasive fear of insulin as a cause of weight gain, omission of insulin to control weight, and at least one severity indicator of either a HbA_{1c} concentration of 10% or higher (≥86 mmol/mol)in the past 12 months, recurrent diabetic ketoacidosis admissions (more than one in the past 2 years), or recurrent severe hypoglycaemia or BMI of 15 kg/m^2 or lower in the past 12 months (appendix pp 5–6).

Routine clinical data (ie, HbA_{1c}, BMI, number of diabetic ketoacidosis admissions, episodes of severe hypoglycaemia) and patient-reported outcome measures were collected at the first assessment (baseline; appendix p 6).

The service model applied the principles of collaborative care and consisted of three phrases to be delivered within 12 months.[4] The hub clinical team consisted of a psychiatrist, a psychoanalytical psychotherapist, a diabetologist, a diabetes specialist nurse also qualified in cognitive behaviour therapy (CBT), and a coordinator (appendix p 7). The first phase was a joint diabetes and psychiatry assessment followed by a psychoanalytical and CBT assessment. Individuals were assessed for diabetes-specific cognitions and behaviours, such as fear of insulin, hypoglycaemia, and complications; extent of adjustment to the diagnosis of type 1 diabetes; and self-harm as potential additional psychological factors for insulin omission.[5] The psychoanalytical assessment involved determining the unconscious processes that initiate and maintain the behaviours that were impeding optimal management of type 1 diabetes.[6] The assessment aimed to elucidate psychological processes, such as the patient's unconscious phantasies, object relationships, defence mechanisms, unconscious beliefs about the origin of their diabetes, and motivation to change.

The second phase consisted of integrating any of the following: individual psychoanalytical psychotherapy or diabetes-focused CBT for 12 months, antidepressant medication, diabetes care following standard clinical guidelines, family therapy, consultation-liaison, admission to an inpatient eating disorders unit, monitoring of psychiatric risk and medical risk, and regular multidisciplinary team reviews (appendix pp 7–8). The third phase consisted of discharge planning to the spoke or local diabetes team.

The main outcomes were changes in HbA_{1c}, BMI, annual rate of hospital admission for diabetic ketoacidosis, and rates of severe hypoglycaemia from baseline to 12 months follow-up. These main outcomes were also assessed in a subgroup of patients at 24 months. Our secondary

outcomes were to assess whether the T1DE risk score and patient-reported outcomes improved. More information on outcomes is in the appendix (p 9).

The composite T1DE score was generated as the sum of: glycaemia (1 point for each 1% increment of HbA_{1c} >7% and 0·5 points for each 1% increment of HbA_{1c} >10%), low bodyweight (0·5 points for each 1 kg/m^2 decrease of BMI <18 kg/m^2 and 1·0 points for each 1 kg/m^2 decrease of BMI <15 kg/m^2), 1 point for each hospital admission for diabetic ketoacidosis, and 1 point per severe hypoglycaemic episode in the preceding 12 months, with the lowest score set at 0 and no upper limit.

78 individuals were referred to the service, of whom 71 (92%) were offered a first assessment and 66 (93%) of 71 attended the first assessment (appendix p 13). 63 (95%) of 66 were female and three (5%) were male, there was broad representation by ethnicity, age, and duration of type 1 diabetes (appendix p 14). Almost all attendees had HbA_{1c} concentrations of 86 mmol/mol or higher at baseline (table), nearly half had recurrent diabetic ketoacidosis admissions, eight (17%) had at least one recurrent severe hypoglycaemic episode, and seven (12%) had a BMI of less than 15 kg/m^2 (appendix p 14). Among 40 individuals with data at baseline, the total number of diabetic ketoacidosis admissions was 113 (with a maximum number of episodes per individual of 22), and among the 33 patients with data at baseline, the total number of severe hypoglycaemic episodes was 47 (with a maximum episodes per individual of 40; appendix p 16). Over 12 months of follow-up, the mean change in HbA_{1c} was –10·1 mmol/mol (95% CI –16·9 to –3·3; table). The total number of hospital admissions for diabetic ketoacidosis decreased by 86%, from 113 in the 12 months before baseline to 16 episodes during the 12 months follow-up (appendix p 16), and the number admitted for diabetic ketoacidosis at least once in the past 12 months reduced from 18 (45%) of 40 with available data to 12 (30%; table). The total number of severe hypoglycaemia episodes reduced from 47 episodes at baseline to nine episodes at 12 months (appendix p 16). The mean BMI increased slightly (table). The mean composite T1DE risk score reduced by over 40% and the number of patients with HbA_{1c} concentrations of more than 86 mmol/mol doubled from 38 (81%) of 47 to 27 (58%) of 47 at 12 months (table).

	n	Baseline measure	12-month follow-up measure	Change in measure (95% CI)	p value*
HbA_{1c}					
Mean, %	47	11·4 (2·8)	10·5 (2·7)	–0·92 (–1·54 to –0·31)	0·0047
Mean, mmol/mol	47	101·4 (30·4)	91·4 (29·2)	–10·1 (–16·9 to –3·3)	0·0047
Patients with HbA_{1c} ≥10% (≥86 mmol/mol)	47	38 (81%)	27 (58%)	–23% (–41 to –5)	0·025
BMI kg/m^2	40	22·7 (5·4)	23·7 (5·2)	0·98 (0·09 to 1·88)	0·028
Patients who had at least one hospital admission for diabetic ketoacidosis in past 12 months	40	18 (45%)	12 (30%)	–15%†	0·016
Patients who had at least one severe hypoglycaemic episode in past 12 months	33	6 (18%)	4 (12%)	–6%†	0·41
T1DE risk score	45	7·0 (7·0)	3·8 (2·9)	–3·2 (–5·3 to –1·1)	0·0027
Patient Reported Outcomes					
PHQ-9 score	11	14·4 (6·2)	12·3 (6·4)	2·1 (–2·0 to 6·2)	0·28
GAD-7 score	9	11·8 (6·1)	7·7 (6·0)	4·1 (0·62 to 7·6)	0·026
DEPS-R					
Mean score	11	39·3 (11·1)	32·5 (17)	6·8 (–1·5 to 15·2)	0·099
Patients scoring ≥20	11	10 (91%)	8 (73%)	–18% (–50 to –13)	..
DDS-17 score	10	2·9 (0·5)	3·0 (1·3)	–0·1 (–0·86 to 0·58)	0·67

Data are mean (SD) or n (%) unless otherwise indicated. Data are only included for patients who had data at both baseline and follow-up. DEPS-R=Disordered Eating in Diabetes. DDS-17=Diabetes Distress Scale-17. GAD-7=Generalized Anxiety Disorder Assessment-7. PHQ-9=Patient Health Questionnaire. T1DE=type 1 diabetes and disordered eating. *Paired Student's t tests and χ^2 tests. †Absolute change in proportion.

Table: **Change in biomedical and patient-reported outcome variables from baseline to 12 months (unadjusted; N=66)**

Table

Change in biomedical and patient-reported outcome variables from baseline to 12 months (unadjusted; N=66)

Data are mean (SD) or n (%) unless otherwise indicated. Data are only included for patients who had data at both baseline and follow-up. DEPS-R=Disordered Eating in Diabetes. DDS-17=Diabetes Distress Scale-17. GAD-7=Generalized Anxiety Disorder Assessment-7. PHQ-9=Patient Health Questionnaire. T1DE=type 1 diabetes and disordered eating.

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Paired Student's *t* tests and χ^2 tests.

†

Absolute change in proportion.

There were fewer patients who had complete baseline and follow up data for each patient reported outcome measure (table). There were eight admissions to eating disorder units among seven (11%) of 66 patients and seven (11%) patients had an assessment for statutory involuntary detention, of whom four (6%) were formally detained (appendix pp 10–11, 16).

Our cohort had several strengths, including diversity in terms of socioeconomic status, ethnicity, age, and duration of type 1 diabetes (appendix p 14); the standardisation of clinical pathways and interventions by use of manuals and standard operating procedures; maintenance of fidelity by regular external supervision; delivering intensive treatments across multiple health systems across a large city, including during the COVID-19 pandemic; and reaching 80% follow-up for biomedical outcomes, enabling the derivation of a new composite risk score that accounts for multiple diabetes outcomes. We generated a set of diagnostic criteria for severe type 1 diabetes and disordered eating that was derived from widely accepted clinical constructs of severe risk in diabetes and psychiatry.

Our study also had several limitations. We did not have a control group; however, a randomised controlled trial was not appropriate in the context of urgently addressing an unmet need in a population with high risk of mortality. In our literature review (appendix pp 3–4) we found no definitive randomised controlled trials on this topic. Also, we could not adjust for confounding or for multiple testing because our sample was small. Finally, referral bias was potentially present towards patients with recurrent admissions for diabetic ketoacidosis admissions compared with patients who administer the minimal amount of insulin required to avoid diabetic ketoacidosis or who do not attend their diabetes clinic appointments, or both.

A potential alternative explanation for our observations that the intervention improved biomedical outcomes is that, during the COVID-19 pandemic, rates of hospital admissions for diabetic ketoacidosis reduced and the proportion with a HbA_{1c} concentration of 58 mmol/mol or lower increased in the general population with type 1 diabetes in England.[7,8] However, the rate of eating disorders increased during this time;[9] therefore, the circumstances brought about by the COVID-19 pandemic are unlikely to have contributed to the reduction in rates of hospital admissions for diabetic ketoacidosis in patients with psychiatric comorbidities.

There are only a few examples of integrated models for diabetes care. The landmark randomised controlled trial Team Care collaborative model[4] was developed in primary care for people with depression, type 2 diabetes, and cardiovascular disease and its theoretical framework was adapted for this study. In a small, non-randomised, feasibility study, mentalisation-based therapy, a treatment for personality disorder, led to a reduction in diabetic ketoacidosis episodes in people with type 1 diabetes.[10]

The patients in this service were complex with multiple morbidities, and although we did not measure this formally, around half had had adverse childhood experiences. There are several mechanisms of action of the integrated intervention that helped achieve the improvements in biomedical outcomes. We considered peer-to-peer learning as one potential mechanism as diabetes and mental health care professionals shared their respective knowledge and expertise, which allowed the patient to be considered as a whole person by one team. A second key mechanism involved the multi-disciplinary team attending monthly clinical supervision led by an external psychiatrist trained in systemic therapy. This process facilitated the identification of the internal aspects of the patient that they put into or onto the team members (projection), provoking a reaction such as needing to rescue the patient, or adversely reject (discharge) the patient who fails to respond to treatment. These crucial unconscious communications from the patient can become enacted within the team, causing significant splits, or disagreements on management of the case, and in some cases a breakdown in the treatment of the patient. If these can be identified, it enables the team to recognise and process them, creating cohesion amongst team members, and a far deeper understanding of the patients' internal situation. The incomplete data collection for the psychological measures in our study reflected the difficulties of collecting patient-reported outcome measures when working with patients with severe mental illness and the need to capture these data with minimum questionnaire burden.

Further population-based observational research is needed to estimate the prevalence of type 1 diabetes and disordered eating by severity, and by its subtypes, to validate these diagnostic criteria for type 1 diabetes and disordered eating. The high rates of assessment for involuntary detention could suggest impairment in decision making and mental capacity in people with type 1 diabetes and disordered eating who have chronic hyperglycaemia, ketoacidosis, or hypoglycaemia.

Competing Interests

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