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Factitious Hypoglycemia in Children and Adolescents with Diabetes

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Abstract

Background—Factitious hypoglycemia is a condition of self-induced hypoglycemia due to surreptitious administration of insulin or oral hypoglycemic agents. In adults, it is an uncommon, but well known clinical entity observed in individuals with and without diabetes.

Objectives—To report a case of factitious hypoglycemia highlighting diagnostic pitfalls, to identify common characteristics of children and adolescents with factitious hypoglycemia, and to examine whether information on long-term outcome exists.

Methods—We present a case of an adolescent with type 1 diabetes who had self-induced hypoglycemia of several years' duration; and we conducted a systematic literature review on factitious hypoglycemia in pediatric patients with diabetes.

Results—We identified a total of 83 articles of which 14 met the inclusion criteria (describing 39 cases). All but one individual had type 1 diabetes and the majority was female (63%). Average age was 13.5 ± 2.0 years with the youngest patient presenting at age 9.5 years. Blood glucose control was poor (HbA1c $12.1 \pm 4.0\%$). In 35%, psychiatric disorders were mentioned as contributing factors. Only three reports provided follow-up beyond six months.

Conclusions—Factitious hypoglycemia typically occurs in adolescents with type 1 diabetes who use insulin to induce hypoglycemia. Awareness of this differential diagnosis and knowledge of potentially misleading laboratory results may facilitate earlier recognition and intervention. Little information exists on effective treatments and long-term outcome.

Five Key Words

Factitious; Surreptitious; Münchausen; Pediatric Diabetes; Hypoglycemia

Introduction

Factitious disorders, also known as Münchausen syndrome and malingering are characterized by intentionally and surreptitiously inducing symptoms to portray physical and mental illness (1–3). In cases of Münchausen syndrome by proxy, a close person or relative of the patient (e.g. the mother) induces the illness, which represents a form of child abuse. A representative case has been described by Schade et al., which ultimately resulted in the child's death (4). Recently 'Münchausen by Internet' has been coined and describes affected individuals pretending to have an illness in a virtual environment, such as an online support group (5).

Attempts have been made to differentiate between factitious disorders and malingering, based on the premise that individuals with factitious disorders suffer from a mental illness and typically have no or few obvious signs of reward or benefit from producing the illness. In contrast, malingering may be observed in patients who have a definite secondary gain associated with their symptoms. However, these delineations are ultimately artificial and ineffective. We chose the term factitious hypoglycemia in this report because it best describes the condition as 'self-made' and 'engineered' irrespective of an underlying psychiatric disorder and the potential for secondary gains.

There are several warning signs described in adults that raise suspicion of a factitious disorder (1, 3, 6). These include difficult to explain symptoms which at times dramatically worsen or shift, change in symptoms following treatment, involvement of multiple medical providers and recurrent hospitalizations, and having a health care related occupation. Factitious hypoglycemia has been described in otherwise healthy adults and in patients with diabetes who are prescribed medications that can induce hypoglycemia even when taken appropriately (7–9).

We aimed to 1) describe a case of factitious hypoglycemia in an adolescent female with type 1 diabetes focusing on the pitfalls of diagnosis, 2) systematically review the literature on factitious hypoglycemia in children and adolescents with diabetes, 3) determine whether patients share common characteristics and 4) examine whether information on long-term outcome exists.

Case Report

A 15-year-old, multiracial, female patient with type 1 diabetes presented for evaluation of recurrent episodes of unexplained, severe hypoglycemia. Upon admission to the National Institutes of Health (NIH), she and her parent gave written informed assent and consent to enrollment in our natural history study of autoimmune diabetes and its complications (NCT 00896610). She had required three hospitalizations for hypoglycemia, the earliest occurring at age 8 years. At that time, she had developed transient right-sided weakness, numbness, and difficulty reading. A head CT and MRI were normal, but an EEG was notable for focal slowing on the left side upon hypoventilation, consistent with hypoglycemia. The hypoglycemia episodes typically lasted between one to three days (maximally two weeks), and occurred every three to six months. Triggers for hypoglycemia were strenuous activity

and excitement, but most episodes occurred with a 12 to 24-hour delay. She also tended to become hypoglycemic a week after menstruation. Endocrinologists at two other institutions had done extensive evaluations to rule out endocrine and gastrointestinal etiologies, but no pathology was found. During the last hospitalization, she had required three days of intravenous glucose infusions to maintain blood glucose concentrations above 60 mg/dL despite oral intake. Laboratory results showed plasma insulin concentrations of 483.0 μ U/mL on admission with undetectable C-peptide. The patient denied excessive insulin dosing, instead reported appropriately discontinuing insulin administration once she noticed the onset of hypoglycemia. Upon discharge, the working diagnosis was hypoglycemia due to an autoimmune mechanism, potentially related to insulin binding antibodies.

Since the diagnosis of type 1 diabetes at age 17 months, the patient had received insulin therapy. Initially she administered insulin via multiple daily injections and as of age seven years, she used continuous subcutaneous insulin infusion (CSII or insulin pump). Most recently, she also used a continuous glucose monitoring device. Her blood glucose control had been suboptimal for at least six years with hemoglobin A1c (HbA1c) results in the 9 to 11% range (desirable < 7%). Her total daily insulin dose was about 0.3 units/kg/day prior to this hospitalization. These low insulin doses were explained by fear of hypoglycemia. The patient's only other medication was an oral contraceptive for irregular periods. She had not undergone any surgeries and her medical history was unremarkable except for type 1 diabetes. At the time of presentation, she was a sophomore in high school with excellent academic performance. She also participated in several athletic activities.

On physical exam, the patient appeared well and had a BMI of 26.9 kg/m². Laboratory results confirmed an elevated HbA1c (10.5%; normal 4.0–6.0%), positive GAD antibodies (48 nmol/L; normal 0–0.02 nmol/L) and a low insulin antibody titer (0.75 nmol/L; normal 0–0.02 nmol/L). The proposed etiology of insulin-binding antibodies causing unpredictable insulin action was excluded because of the low insulin antibody titer and the inconsistent clinical presentation.

During an overnight hospitalization, we confirmed that she tolerated expected doses of insulin (based on weight and pubertal status). Results showed normal blood glucose concentrations during continuous infusion of glulisine (Apidra™) at 1 unit/hour. During exercise on a treadmill, the patient's blood glucose did not drop disproportionately. Thus, abnormally high insulin sensitivity or markedly high insulin-independent glucose uptake were excluded. Other differential diagnoses of hypoglycemia were also addressed; e.g. adrenal insufficiency was ruled out. The patient was dismissed from the hospital and instructed to return when an episode of hypoglycemia occurred to evaluate appropriate counter-regulatory hormone responses. The topic of self-induced hypoglycemia was discussed, but vehemently rejected.

After a hiatus of several months without episodes of severe hypoglycemia, the patient returned due to a new episode. Blood glucose concentrations at home were between 20–40 mg/dL despite insulin pump suspension. The insulin pump dosing history was manually reviewed and reflected the patient's report. Her blood glucose had risen to 94 mg/dL with undetectable insulin and C-peptide concentrations. Anti-insulin antibodies were 0.48

nmol/L, cortisol 13.6 mcg/dL, and growth hormone 17.9 ng/mL. The patient returned five days later after another episode. Insulin and C-peptide were again undetectable. Glucagon was 13 pg/mL, and a drug screen for hypoglycemic agents was negative.

The department of laboratory medicine was contacted to obtain information about the insulin immunoassay used at our institution. The samples had been assayed with a Roche Cobas 6000 analyzer (normal fasting range 2.6–24.9 mIU/mL [19–178 pmol/L]; inter-assay CV 2.5–6.0%; intra-assay CV 1.0–1.2%). It was realized that this assay, while extremely sensitive to endogenous insulin, had highly variable cross reactivity with several insulin analogues. In particular, this assay had no cross-reactivity with insulin glulisine (10–14). Thus, stored plasma samples were sent out to be analyzed with an Abbott Architect immunoassay. This time, the insulin concentration was reported as 26 μ U/mL. Considering that the Abbott Architect has approximately 10% cross reactivity with glulisine, the circulating insulin concentration was estimated in the 200–300 μ U/mL range.

In the weeks to follow, the patient visited one of her other endocrinologists. Upon review of her insulin pump's memory, it was found that she had been priming her insulin pump with large amounts of insulin that corresponded with the dates of her most recent hypoglycemic episodes. Thus, the episodes had been induced by infusing instead of discarding insulin to fill the catheter connecting the insulin pump with the insertion site. Up to 327 units of insulin had been recorded to be used for "priming". When presented with this information, the patient suggested that these doses had been administered unintentionally, but later admitted that she had self-induced hypoglycemia via a variety of strategies (i.e., priming or self-injecting insulin) without her caregiver's knowledge for approximately seven years, including the first episode which occurred at 8 years of age.

Following the diagnosis of factitious hypoglycemia, the patient's care was consolidated (she had been followed by eight physicians and used two different insulin pumps). It was decided that she would receive care from one primary care provider and one pediatric endocrinologist, and use one insulin pump. Individual and family psychotherapy was recommended. Six months later, no further self-induced hypoglycemia occurred. The family decided to only pursue individual psychotherapy. The patient continued to do well academically, but was diagnosed with depression. Hearing loss, which had previously been described as mild, started to interfere with daily life, and hearing aids were recommended.

Methods

We performed a systematic review of the English literature through October 2017. Searches were performed using Google Scholar, PubMed, Web of Science and Embase. Keywords included: factitious hypoglycemia, insulin administration, exogenous, surreptitious, self-induced, diabetes, Münchausen, adolescent, pediatric, IDDM, type 1 diabetes, intentional and overdose. Publications in languages other than English were not included in the present case series, but were referenced in the discussion if an English abstract was available (e.g., references (15, 16). To be considered for this study, reports had to (1) include subjects less than 18 years of age, (2) directly relate to individuals with diabetes (type 1 diabetes, type 2 diabetes, DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy, and deafness) or

Wolfram syndrome, mitochondrial, MODY (maturity onset diabetes of the young) or other), and (3) include intentional, self-induced hypoglycemia. Cases of factitious hypoglycemia in patients without diabetes (e.g., (17, 18) and cases of attempted or completed suicide associated with intentional overdosing (insulin or oral hypoglycemic agents) without a preceding history of factitious hypoglycemia were not included (e.g., (19–21). From qualifying reports the following information was extracted: demographic characteristics, type and duration of diabetes, overall glycemic control, prescribed diabetes treatment, history of diabetic ketoacidosis (DKA), and method of induction of factitious hypoglycemia, therapeutic intervention and long term follow-up.

Results

We identified a total of 83 articles of which 14 met the inclusion criteria (Table 1). These reports described 39 cases. We analyzed patient characteristics including our own patient presented in this report. Of 40 cases, the majority was female (63%). Average age was 13.5 ± 2.0 years (range 9.5–18.0) and average diabetes duration was almost six years. All but one report, which described a 10.8 year old boy with mitochondrial diabetes (case #36, reference (13), included youths with type 1 diabetes. Recurrent diabetic ketoacidosis was mentioned in seven cases and overall poor glycemic control was documented ($HbA1c\ 12.1 \pm 4.0\ %$). However, these results were obtained with various assays, not all reports included normal ranges, and results were only available in 17 cases. While the exact insulin doses were not provided in all reports, low insulin doses (less than 0.5 U/kg/day) were mentioned in twelve cases, and complete cessation of insulin administration in two cases. Thus, the reported insulin doses were not consistent with hypoglycemia but rather with hyperglycemia.

Most case reports (83%) contained reasons for inducing hypoglycemia. Psychiatric or psychological conditions (e.g., depression (n=6), suicidality (n=2), oppositional disorder (2), schizoid and borderline personality (n=2), unspecified psychosis (n=1), and anxiety (n=1)) were among the most frequently cited. In six cases, the wish for more food, specifically sweets, was described. Other explanations included school avoidance, attention seeking, disease denial, wish to reduce parental anxiety about diabetes related complications, cover-up for omitted insulin injections, induction of excitement, and self-punishment. In all reports with a documented method of hypoglycemia induction, insulin was used. In most cases (70%) counseling or psychiatric treatment were mentioned as intervention after the diagnosis of factitious hypoglycemia had been made (with no information on intervention or follow-up in nine cases). Two cases specifically mentioned suicide attempts in the follow-up period and one described switching from factitious hypoglycemia to a new symptom (self-strangulation for excitement).

Discussion

Our 15-year old patient with type 1 diabetes had multiple characteristic features of factitious hypoglycemia: female gender, adolescent age, long-standing type 1 diabetes, low reported insulin doses, poor blood glucose control, involvement of many health care providers, use of two different insulin pumps, and an atypical history of hypoglycemia (e.g., occurring one day after, not during or immediately following strenuous exercise). The primary gains from

self-induced hypoglycemia were removal from stressful situations (e.g., exams at school or athletic competitions), receiving extensive attention from her family and health care providers, and being able to eat large amounts of carbohydrate containing foods. She had first learned about self-induced hypoglycemia when attending a summer camp for children with type 1 diabetes (similar to reports in references (15, 22)) at the age of eight years. The diagnosis was delayed because of misleading laboratory results (no or low plasma insulin concentrations due to the use of a highly specific insulin assay which did not cross-react with insulin analogues, also described in references (10–14), and due to only reviewing the insulin pump's memory of total daily insulin doses, and not downloading the insulin pump's "priming history" (see references (23, 24)). Our interventions consisted primarily in consolidation of care and individual psychotherapy. The diagnosis of depression and hearing loss were made, and self-induced hypoglycemia seized during a follow-up period of 12 months. It is unclear whether the patient developed other coping mechanisms which explain this change in behavior.

Definitions and Nomenclature

The terms to describe factitious disorders are confusing since 'factitious' (which derives from the Latin verb *facere* and means *to do* or *to make*) has been used in the context of willfully induced illnesses and haphazardly experienced conditions. An example of the latter is factitious Graves' disease due to biotin immunoassay interference described in an asymptomatic patient with abnormal laboratory results (25). This patient did not pretend to have an illness, but merely took a medication which caused laboratory results mimicking hyperthyroidism. In the present review, we define *factitious* as *intentionally fabricated*, which is equivalent to Münchausen syndrome. The original description states that "like the famous Baron von Münchausen, the persons affected have always travelled widely; and their stories, like those attributed to him, are both dramatic and untruthful" (1). In The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), factitious disorders fall under the section of *Somatic Symptom and Related Disorders*. The DSM- 5 categorizes two types: self-imposed and imposed on another.

Prevalence

In adults, the 1-year prevalence of factitious disorders has been estimated at 1.3% based on a survey of physicians in private practice and hospital consultants (26), however, it may be markedly higher in specialty clinics for patients with diagnostically challenging problems. Compared to other subspecialties, endocrinology is a field in which factitious disorders are disproportionality often reported (3). In addition to factitious hypoglycemia, presentations include Cushing's syndrome (27), thyrotoxicosis, and endocrine neoplasias. Pediatric factitious disorders have been estimated to occur at a similar prevalence compared to adults (0.7 to 1.8%) but the prevalence of factitious hypoglycemia in children and adolescents with diabetes is completely unknown (28, 29). We identified only 40 cases in this review (including our own), but the frequency is likely under-reported since most pediatric endocrinologists can recall one or more cases in their practice. A structured interview of 241 Austrian children and adolescents with type 1 diabetes revealed that 55 (23%) admitted to intentional insulin overdosing. However, the study was conducted to assess the prevalence of under- and over-dosing, and not to examine self-induced, surreptitious hypoglycemia.

Diagnostic Challenges

It is worth noting that in many factitious disorders, symptoms are often inconsistent and difficult to explain. However, factitious hypoglycemia in patients with diabetes mimics a side effect of the prescribed treatment. Thus, diagnosing this disorder is even more difficult. The typical laboratory paradigm to differentiate exogenous insulin induced factitious hypoglycemia (low C-peptide, high insulin) from an insulinoma (high C-peptide, high insulin) or other causes (low C-peptide, low insulin) (30) may not be easily applicable. Today's highly specific insulin assays do not (or to a lower degree) detect recombinant insulin analogues (10–14). An additional diagnostic challenge is the preconceived notion that children do not intentionally harm themselves. However, it is known that the ultimate self-harm, suicide, occurs in children and adolescents (31). Another factor may be the preference of the involved physicians, especially at academic centers, to rather pursue an intellectually challenging diagnosis than to face the unsettling prospect of factitious hypoglycemia. This is particularly the case, when the treating physician and health care professionals have a long-standing and caring relationship with the patient. Thus, the correct and timely diagnosis requires a high level of suspicion, a very careful review of the administered insulin doses comparing expected insulin use with actual use, down-load of insulin pump information or review of insulin/blood glucose logs, and a blood sample obtained during hypoglycemia to simultaneously determine plasma glucose, C-peptide and insulin (measured with an assay that picks up exogenous insulin).

Reasons for Self-Inducing Hypoglycemia

The most obvious and frequently mentioned reason is the wish to eat more, especially sweets. Depression, a cry for help and attention, and school avoidance are other common factors. One early case report describes a patient's intense enjoyment about her parents' distress and physicians' helplessness (15). This 14 year old girl expressed a wish for revenge since she alone had type 1 diabetes in her family, while all the others were healthy. Some children try to avoid hyperglycemia at all cost causing frequent hypoglycemia in response to their parents' fear of long-term diabetic complications (32). Other adolescents were described who had let their diabetes control slip, had not injected insulin for a while and then overcompensated (32). One 15 year old boy described the rapid onset of hypoglycemia as exciting and associated with euphoria (22). In adults, these exhilarating feelings have also been reported when insulin is administered together with illicit drugs and alcohol (33). Depression appears to be an important contributing factor. In youth with type 1 diabetes, its prevalence has been found as high as one in seven youths, which is at least twice as high as in youths in general (34). Several cases of attempted or completed suicide with insulin overdoses have been reported in adolescents in whom no clear history of preceding factitious hypoglycemia was known (20, 21, 35–37). One can only speculate that previous hypoglycemic episodes were also intentionally self-induced and might have been a warning sign. A recent analysis documented excess all-cause mortality in young individuals (less than 30 years of age) with T1D and found that the most common causes were ketoacidosis, likely hypoglycemia and suicide (38). Though no details about the individual cases are available, it is possible that some of the hypoglycemia episodes were induced intentionally and some of the suicides were committed with insulin overdoses.

Typical Characteristics

Our results show that a high percentage of youths with factitious hypoglycemia suffer from depression and suicidality compared to the overall percentage of children and adolescents (US population) with or without type 1 diabetes (35, 39). Other common features are being in puberty, having long-standing type 1 diabetes with poor blood glucose control and using disproportionately low insulin doses. Difficult family and psychosocial environments were frequently reported spanning across a wide range of socioeconomic levels. Surprisingly, several case reports mentioned that the affected youths performed well in school, as was the case in our patient. We did not identify cases which occurred due to ingestion of oral hypoglycemic agents; instead all youths used insulin to induce hypoglycemia. Despite type 2 diabetes representing a considerable proportion of pediatric diabetes, no cases of factitious hypoglycemia were found in the literature.

Effect on Patient-Parent-Physician Relationship

Patients who surreptitiously inflict self-harm disrupt the intimate, therapeutic pact between themselves and their care takers (parents, physicians, and other health professionals). While providers should maintain their caring attitude and empathy, suspicion and mistrust are introduced. Feelings of betrayal, disappointment, loss of trust affect the relationship before and after the diagnosis are made. Parents have often suffered for years in fear of the next unpredictable severe hypoglycemia, have lost sleep and had to interrupt their work schedules. Thus, resentment on their side may easily emerge. Immense costs can occur due to frequent hospitalizations, lengthy diagnostic evaluations and sometimes invasive procedures. Most physicians spend an inordinate amount of time and energy before they confront a patient with the diagnosis of a factitious disorder.

Follow-up and Long-term Outcome

Follow-up information on patients with diabetes and factitious hypoglycemia is sparse. Case reports describe varying outcomes in adults ranging from resolution of hypoglycemia, to a shift towards other factitious symptoms and suicide (7). Similar to adults, systematic follow-up of children suffering from factitious hypoglycemia does not exist. This is inherent in the diagnosis since patients often avoid continued care provided by physicians and health professionals who made the diagnosis and thus “exposed” them. However, two cases are described in which referral to counseling and treatment by child psychiatry prevented further episodes for 1.5 and 4.5 years, respectively (22, 40). Another factor preventing long-term follow-up is the necessary transition from pediatric to adult endocrine care, during which information on the detailed history of the patient may get lost. The importance of follow-up is also highlighted by the fact that some cases of Münchausen by proxy are inflicted by parents who themselves displayed factitious disorders in adolescence (41). Furthermore, it has been estimated that many cases of factitious disorders occurring in young adults started in adolescence (42).

Conclusions

The herein described case has typical characteristics of a factitious disorder, which is especially evident in hindsight. These include difficult to explain timing of hypoglycemia

(i.e., long after exercise), unexplained waxing and waning of severe hypoglycemia episodes, use of two insulin pumps and involvement of multiple medical providers. Even when laboratory results supported the diagnosis of surreptitious insulin administration during her last emergency room visit and hospitalization, another differential diagnosis was given priority (interference by insulin antibodies). This likely occurred due to the long-standing, close and caring patient-physician relationship. Acknowledgement of the factitious nature of the symptoms by both, physicians and parent, would have meant acknowledgement of betrayal. The diagnosis was further delayed during the subsequent work-up because of the use of an insulin assay that did not detect exogenous insulin. Similar cases have previously been reported (10–14). Many inventive strategies have been described for surreptitious insulin administration. Our patient is not the first to have used the priming function of her insulin pump (23, 43). Only through careful review of the “priming” or “cannula fill” history was this technique discovered, while the review of daily insulin dosing does not include the amount used for priming.

Further common features of our patient and others described in the literature include overall poor metabolic control. Additionally, comorbid psychiatric conditions or psychosocial challenges are frequently found. Depression and suicidality stood out as underlying causes of factitious behavior in several cases. A recent systematic review/meta-analysis of anxiety and depressive symptoms in youth with type 1 diabetes substantiated that there is indeed a high prevalence of symptoms of depression in youth with type 1 diabetes (44). Depression and suicidality in children and adolescents are often overlooked, in part because they are not regarded as childhood diseases and because of the helplessness of medical providers regarding adequate treatment. Considering that depression and suicidality appear to frequently co-occur in type 1 diabetes and factitious hypoglycemia, screening and continued monitoring for psychosocial pathology may assist in earlier diagnosis. Moreover, earlier detection and treatment of psychosocial pathology could decrease the likelihood of the development of factitious hypoglycemia and its complications.

Finally, we highlight a lack of knowledge on effective treatments and long-term outcome. Information on treatment and outcome for these patients is of vital importance, as the discovery and diagnosis of factitious hypoglycemia can often be dramatic and distressing. Additionally, because factitious hypoglycemia is a life-threatening behavior, it is imperative to be addressed in a timely manner to prevent catastrophic outcomes such as permanent brain damage or death.

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Table 1

Extraction table for case report analysis.

Case # (Ref.)	Age (years)	Gender	Diabetes Type	HbA1C (%)	Duration of T1DM (years)	Prescribed Treatment (insulin dose)	History of DKA	Method of Factitious Hypoglycemia	Reason for inducing hypoglycemia	Duration of factitious disorder	Long term outcome
Case # 1 (45)	16	F	T1DM	No info	5	Reportedly off insulin × 2 months	No info	Self-injection	No info	9 months	Patient did not admit to self-injection. Insulin restarted
Case # 2 (46)	16	F	T1DM	No info	No info	No info	No info	No info	No info	No info	No info
Case # 3 (47)	15	F	T1DM	18.0	3	No info	Recurrent episodes of DKA	No info	Oppositional disorder	No info	No follow-up reported
Case # 4 (47)	15	M	T1DM	16.6	2	No info	1 Hospitalization for DKA	Self-injection	Disease denial	4 months	Two suicide attempts, continued psychotherapy, continued to have poor metabolic control
Case # 5 (47)	14	F	T1DM	12.2	7	No info	Recurrent episodes of DKA	Self-injection	Depression & schizoid personality trait	No info	Admitted for short term psychiatric treatment; no long-term F/U
Case # 6 (47)	15	F	T1DM	21.5	9	No info	Recurrent episodes of DKA	No info	Depression	No info	No follow-up reported
Case # 7 (47)	12	M	T1DM	17.1	5	No info	Recurrent episodes of DKA	No info	Depression	No info	No follow-up reported
Case # 8 (47)	15	F	T1DM	10.6	14	No info	Recurrent episodes of DKA	No info	Oppositional disorder	No info	No follow-up reported
Case # 9 (40)	11	M	T1DM	No info	3	Injection schedule not specified; Estimated 0.2 U/kg/day	Report states no history of DKA	Self-injections	No info	2 years	No follow-up reported
Case # 10 (40)	13	F	T1DM	No info	No info	MDI	9 hospitalizations for DKA	No info	Depression, school avoidance	2.5 years	Referred to counseling, no further hypoglycemia × 1.5 year
Case # 11 (48)	12	M	T1DM	No info	4	Self-injections	7 episodes of DKA in two years	Self-injections	Suicidal, difficult psychosocial situation	3+ years (still ongoing at time of report)	Transferred to psychiatric services, placed in residential treatment program following another suicide attempt
Case # 12 (22)	15	M	T1DM	“increasing HbA1C levels”	9	Injection schedule not described; Typically 0.9 U/kg/day, but before FH diagnosis 0.26	No info	Self-injections	Borderline personality disorder, sense of excitement & euphoria	No info	Child psychiatry, no recurrence × 4.5 years
Case # 13 (49)	14	F	T1DM	14.3	10	NPH and Regular insulin twice daily (1.1 U/kg/day)	Report states no history of hospitalizations	Self-injection	Anxiety about starting high school; Feelings of rejection from parents	No info	Referred to psychiatric evaluation

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Case # (Ref.)	Age (years)	Gender	Diabetes Type	HbA1C (%)	Duration of T1DM (years)	Prescribed Treatment (insulin dose)	History of DKA	Method of Factitious Hypoglycemia	Reason for inducing hypoglycemia	Duration of factitious disorder	Long term outcome
Case # 14– 29 (32, 40)	Mean age 12.9 ±0.5	15 F 1 M	T1DM	8.1±0.3	6.2±0.7	9 of 16 pts with < 0.5 U/kg/d	No info	Self-injection; All 16 patients had > 2 hypoglycemic comas within 3 months	Psychosis (n=1). Family discord (n=7). Parental fear of diabetic complications (n=5). Masking of poor blood glucose control (n=4).	No info	All admitted to self-injection between 1–7 years of report; referrals to child psychiatry
Case # 30 (24)	10	M	T1DM	9.7	5	Initiation of pump	No info	Priming	Wanting to eat more sweets	A couple of weeks	Less parental micromanagement
Case # 31 (50)	18	F	T1DM	9.2–10.4	No info	No info	No info	No info	Depression, suicidality	Several months	Started on antidepressants, hospitalized and referred for psychiatric F/U
Case # 32 (23)	13	M	T1DM	No info	12	Insulin pump	No info	Priming	To get out of school, wanted more food	No info	Removed from pump therapy and began supervised injections; no long- term F/U
Case # 33 (23)	12	M	T1DM	No info	2	Insulin pump	No info	Priming	High-functioning autism; wanted more food	No info	No follow up reported
Case # 34 (51)	13	M	T1DM	9.8	7	Insulin pump	No info	Insulin pen to inject into pump catheter	No info	1 year	Resolution of hypoglycemia, but new symptoms (self-strangulation for excitement)
Case # 35 (13)	9.5	F	T1DM	10.7	1.5	Multiple daily injections 0.66 U/kg/day	1 hospitalization for DKA	Self-injection	Wanted to become and remain sick	1 year	No follow up reported
Case # 36 (13)	10.8	M	DIDMOAD	6.8	No info	MDI 1.03 U/kg/day	No info	No info	No info	1 month	Referred to psychiatric clinic
Case # 37 (52)	13	M	T1DM	9.6	6	MDI; No insulin × 10 days prior to FH diagnosis	1 episode of DKA	Self-injections	Depression	3 years	Referred to a psychologist, doing well during 6-month follow-up
Case # 38 (52)	13	M	T1DM	9.4	4	MDI 0.7 U/kg/day	No info	Self-injections	Access to sweets and specific foods	2 months	Counseling for child and parents; doing well
Case # 39 (52)	14	M	T1DM	10.5	4	MDI 1 U/kg/day	No info	Self-injections	Wanting more food and attention	2 months	Counseling for child and parents
Case # 40 Present case report	15	F	T1DM	10.5		Insulin pump (~0.3 U/kg/d)	Hospitalization for DKA only at T1DM diagnosis	Priming	Stress, self-punishment, ability to eat more carbs	7 years	Referred to child psychiatry and family counseling, no hypos × 1 year
Total number of cases, N=40	13.5±2.0	25 F, 15 M	39 of 40 with T1DM	12.1±4.0 (17 reports available)	5.9±3.4 (19 reports available)	0.6 ±0.4 Units/kg/day (only 10 reports available)	7 pts with recurrent DKA (of 12 reports available)	4 reports with priming	See text	1.6±2.0 years (Median 11.0 months)	See text

Patient characteristics of 39 individuals reported in the literature and one additional new case (Case # 40). The order reflects the year of publication.