Brief report

Carbohydrate-deficient transferrin parallels disease severity in anorexia nervosa

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Abstract

Carbohydrate-deficient transferrin (CDT) is a widely used biomarker for alcohol abuse; however, recent findings questioned its diagnostic value in catabolic subjects. We have thus investigated possible changes of CDT in patients suffering from eating disorders. Retrospectively, CDT values of patients with eating disorders were identified. Twenty-four non-alcoholic subjects could be found, in which CDT was determined at least once during stationary psychotherapy. Anorexia nervosa patients had pathological CDT concentrations in 57% of cases; conversely, bulimia nervosa patients had normal CDT. Patients with initially elevated CDT tended to be more seriously ill than those without. During therapy, the body mass index of anorexia nervosa patients normalized, paralleled by declining CDT. In anorexia nervosa patients, CDT is unsuitable as a marker of alcohol abuse, but it might serve as a parameter indicating prognosis and disease severity. However, case-control studies with larger samples are warranted.

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1. Introduction

Serum carbohydrate-deficient transferrin (CDT) is a widely used biomarker for alcoholism. High levels of specificity and sensitivity were reported initially, so that CDT was considered to be a reliable parameter reflecting long-term alcohol abuse. (Stibler et al., 1988) However, several follow-up studies and meta-analyses suggested that CDT is not superior to other parameters like MCV and γ-GT as previously assumed. (Salaspuro, 1999; Scouller et al., 2000) We have previously reported that CDT is elevated in catabolic females suffering from psychiatric disorders, limiting its diagnostic value in this subgroup (Reif et al., 2001). In order to specify this finding, it was investigated whether CDT was also elevated in patients suffering from eating disorders (EDs) and
whether psychotherapy had any influence on CDT levels.

2. Methods

Twenty-seven female patients receiving inpatient treatment for ED in our department have been identified retrospectively from the years 1997 to 2002. During cognitive-behavioral psychotherapy, CDT was determined routinely to ensure alcohol abstinence in 31 females and, in those, chart reviews were performed. In four patients, CDT was determined during control admissions only; they were therefore excluded from further analysis (body mass index was always above 17 kg/m²; CDT was slightly elevated in three of the four subjects). From the remaining 27 patients, three were excluded due to concomitant alcohol abuse (all had clearly elevated CDT). The other 24 patients were abstinent or "social" drinkers and were included for further analysis; CDT, body mass index (BMI) and routine laboratory parameters including thyroid hormones were recorded from the charts as well as case histories, smoking status and blood pressure. Nineteen patients suffered from anorexia nervosa (AN; ICD-10 F50.0), one from atypical anorexia (AA; F50.1) and four from bulimia nervosa (BN; F50.2). The mean interval between age of onset and hospitalization was 8 years, and the mean age of the patients was 32 years.

3. Results

All AN patients had a pathological BMI (mean, 13.9 kg/m²; mean weight, 37 kg); in contrast, the BMI of the BN patients was in the normal range (mean, 20.7 kg/m²; mean weight, 60 kg). In 11 of the 19 AN, but none of the BN patients, CDT was clearly elevated; CDT and BMI on admission were negatively correlated, \( r = -0.51 \) (Fig. 1). Neither BMI nor CDT correlated with MCV, \( \gamma \)-GT, uric acid, serum iron, cholesterol, triglycerides, T3 or blood pressure; more seriously ill patients tended to have pathological values. Uric acid, \( \gamma \)-GT, triglycerides and T3 were pathological in AN patients only. Interestingly, in all patients with elevated CDT, total protein was in the normal range; in the eight AN patients with normal CDT, total protein was decreased in six cases. Smoking status did not have any influence on CDT concentrations.

In 12 AN patients, CDT was determined repetitively during inpatient psychotherapy; the mean duration of the follow-up period was 7 months. In this subgroup, BMI increased significantly by 3 kg/m² from 15 to 18 kg/m² (\( P = 0.018 \), two-tailed Student's \( t \)-test). BMI at the starting point and CDT change (in % of control values) were correlated: the lower the starting BMI, the more CDT decreased during the follow-up period (\( r = -0.42 \); Table 1). Patients were then analyzed separately, depending on their initial CDT values. In five patients, CDT was initially elevated (mean, 62% above cut-off). During therapy, CDT decreased in all patients, on average by 28% (in three patients to normal levels). BMI in this subgroup increased significantly (\( P = 0.02 \), Student's \( t \)-test) from 15.5 to 19.1 kg/m²; all

Fig. 1. Correlation between body mass index (BMI) (kg/m²) and CDT values on admission (in % of the upper cut-off value, represented by the dotted line). Both AN and BN patients are shown; BMI and CDT values were negatively correlated as indicated by the solid line (\( r = -0.51 \)), i.e. the lower the BMI on admission, the higher the respective CDT concentrations. In 11 of the 19 AN, but none of the BN (BMI >19 kg/m²) patients, CDT was elevated.
patients had an endpoint BMI above 17.8 kg/m² and were therapy responders. Initial BMI and change in BMI correlated well (r = 0.94, i.e. the lower the weight at the start, the more weight gain was achieved) (Table 1).

4. Discussion

The present study replicated the finding that CDT is elevated in catabolic females (Reif et al., 2001). AN patients had elevated CDT values in 57% of cases; on the other hand, BN patients had normal CDT levels, which might be due to the fact that catabolic metabolism is more frequent in AN. The biochemical mechanism underlying the CDT elevation in catabolic patients remains elusive; interestingly, BMI and CDT correlated quite well, in contrast to the previous study, which might reflect the more homogenous patient sample.

In almost half of the patients, CDT and BMI were determined repetitively during therapy. During cognitive-behavioral therapy, the BMI of AN patients improved; however, this is only a surrogate parameter. CDT levels, when initially elevated, mirrored weight gain in that they decreased and finally normalized. Thus, anabolic metabolism seem to normalize elevated CDT levels attributable to weight loss. Whether the same is true for alcoholic subjects remains to be established. Interestingly, in subjects with initially normal concentrations, the CDT values rose transiently, albeit the reason for this is unclear. In patients with initially elevated CDT levels, an increase in BMI was mirrored by a decrease in CDT. However, initially elevated CDT values tended to predict a more severely ill patient group that responded poorly to therapy.

We are aware of the limitations of our study, namely retrospective study design and small sample size, and thus would like to prompt other investigators to replicate the study. Clearly, CDT has no role in the diagnostic process of eating disorders. However, since CDT is determined routinely to screen for alcoholism, patients with elevated CDT levels will perhaps hastily be misdiagnosed as alcoholics, which might have therapeutic as well as forensic implications. Especially in psychotherapy facilities, abstinence during inpatient treatment is a prerequisite for therapy, and CDT represents a commonly used biomarker to ensure abstinence. Therefore, elevated CDT levels could lead to an unjustified discontinuation of AN patients. Clinicians thus should be aware that the differential diagnosis of CDT elevation involves both weight loss and alcohol abuse.

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References

