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Factors associated with refeeding hypophosphatemia in adolescents and young adults hospitalized with anorexia nervosa

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Abstract

Background: Refeeding hypophosphatemia (RH) in individuals with anorexia nervosa (AN) is a potentially fatal complication of nutrition restoration; yet, little is known about risk. This retrospective cohort study examined factors found in hospitalized youth with AN that may contribute to RH.

Methods: We reviewed medical records of 300 individuals diagnosed with AN admitted between the years of 2010 and 2016. Logistic regression examined factors associated with RH. Multivariate regression examined factors associated with phosphorus nadir.

Results: For 300 participants, the mean (SD) age was 15.5 (2.5) years, 88.3% were White, and 88.3% were female. Participants lost an average of 11.3 (9.7) kg of body weight and were 82% (12.1) of median body mass index (BMI). Age ($P = .022$), nasogastric (NG) tube feeding ($P = .054$), weight gain ($P = .003$), potassium level ($P = .001$), and magnesium level ($P = .024$) were contributors to RH. Odds of RH were 13.7 times higher for each unit reduction in magnesium, 9.2 times higher for each unit reduction in potassium, three times higher in those who received NG feeding, 1.5 times higher for each kg of weight gain, and 1.2 times higher for each year of age.

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AUTHOR CONTRIBUTIONS

Meredith Kells contributed to the conception, design of research, acquisition and analysis of the data, and drafting of the manuscript. Andrea K. Garber, Barbara E. Wolfe, Matt Gregas, and Susan Kelly-Weeder all contributed equally to the interpretation of the data and critically revised the manuscript. All authors read and approved the final manuscript and agree to be fully accountable for ensuring the integrity and accuracy of the work.

CONFLICT OF INTEREST

None declared.

Regarding phosphorus nadir, serum magnesium level ($P < .001$) and admission BMI ($P = .002$) contributed significantly.

Conclusion: The results indicate that age, NG feeding, weight gain, electrolyte abnormalities, and BMI on admission are potential indicators of the development of RH in youth. This study identifies clinical risk factors associated with RH and may guide further investigation.

Keywords

anorexia nervosa; enteral nutrition; magnesium; phosphorus; potassium; refeeding syndrome

INTRODUCTION

Anorexia nervosa (AN) is characterized by intense fear of weight gain, behavior that inhibits weight gain, body image disturbance, undue influence of self-worth on body weight and shape, and energy restriction resulting in weight loss or significantly low body weight.[1] Medical hospitalization is recommended in cases of vital sign instability, sustained fasting (food refusal), electrolyte imbalance, precipitous weight loss, and signs of organ damage.[2–5] During medical hospitalization, the reintroduction of nutrition is a key component of care. Nutrition restoration is traditionally approached cautiously, starting with low calories and advancing slowly to minimize the risk of refeeding syndrome, which can be fatal. Refeeding hypophosphatemia (RH), a hallmark of impending refeeding syndrome, is the most common complication associated with nutrition restoration and is more common among low-weight individuals with AN.[6] The incidence of RH in adolescents and young adults has been reported to be as high as 38%, with an average incidence of 14%.[7] Hypophosphatemia can contribute to the development of seizures,[8] inhibited cardiac function,[9] respiratory insufficiency,[10–12] rhabdomyolysis, hemolysis, altered mental status, coma, and death.[13]

The development of RH is multifactorial. During a starvation state, the individual becomes depleted of total body phosphorous levels. With the reintroduction of nutrition, the little available extracellular phosphorous is driven intracellularly by a surge in insulin production for anabolism.[14,15] The reintroduction of nutrition also results in an increased demand for phosphorous as a part of glycolysis.[14–17] Together, this results in low serum phosphorus levels.

Since refeeding syndrome is potentially deadly, risk stratification to target individuals for whom intervention is warranted is the key first step in treatment,[14] and it is critical to ensure safety and determine nutrition restoration. However, the prediction of who is at greatest risk of RH remains poorly understood. Initial serum phosphorous levels are often within normal limits[7] and admission serum concentrations of phosphorous may not accurately predict total body phosphorous depletion that may lead to RH.[17] Therefore, admission laboratory surveillance for phosphorus is insufficient for identifying potential cases of RH during medical hospitalization. Additionally, although RH is most common within the first 6–7 days, nadir may not occur until up to day 24 in 20% of the population.[18]

Numerous studies have associated a lower percent of median body mass index (mBMI) on admission with a higher likelihood of developing RH[7]; however, contributing individual

factors and clinical interventions remain unknown. Large cohort studies examining predictors of RH in adolescents and young adults remain outstanding. Additionally, it is unknown what, if any, contribution the level of psychiatric functioning contributes to the development of RH in youth. There are no consensus guidelines to provide clinicians with an evidence-based approach to the prevention of refeeding syndrome or RH or what interval is most appropriate for electrolyte monitoring.[7,14,19,20] The literature reports several common approaches of treatment, including electrolyte replacement when low or deficient, prophylaxis, and treatment of declining trends[14,19,20]; and, currently, there is wide variability in the approaches to nutrition restoration as well as phosphorous supplementation during the refeeding phase.[20]

Therefore, improved identification of those at greatest risk for the development of RH is imperative for improving clinical care in this population. The purpose of this retrospective cohort study is to identify individual factors and clinical interventions that contribute to the development of RH for adolescents and young adults admitted to a general medical unit with AN.

METHODS

The study included all individuals aged 10–24 years admitted to a large, tertiary care facility in the northeastern United States for treatment of AN over a 7-year time period from 2010 to 2016, using only the most recent admission if an individual had multiple hospitalizations. Reasons for admission included medical instability (eg, bradycardia, low weight, or precipitous weight loss, etc) or failure to meet outpatient goals (eg, weight gain). Inclusion criteria included the diagnosis of AN, either binge/purge or restricting subtypes, or atypical AN. Exclusion criteria included comorbid diagnosis of bulimia nervosa (BN), binge eating disorder (BED), serious mental illness including schizophrenia or schizoaffective disorder, medical or psychiatric hospitalization within the last 14 days, and a feeding method other than oral or nasogastric (NG; eg, nasojejunal or gastric because of low rates of these methods making meaningful analysis challenging). All psychiatric diagnoses (AN, BN, BED, schizophrenia or other schizoaffective disorder, or anxiety disorder) were identified per psychiatry consult liaison team assessment upon admission. During this sampling timeframe, the *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition) (*DSM-5*)[1] was released with revised diagnostic criteria for AN; therefore, diagnosis was made using either the *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition, Text Revision) (*DSM-IV-TR*) or *DSM-5*, accordingly. Research questions were examined using a retrospective cohort design with existing patient medical record data.

All individuals were placed on the institutional clinical practice guideline for restrictive eating disorders, including specific instructions for vital sign and laboratory monitoring, activity, and nutrition rehabilitation; this is briefly described as follows. After meeting with a dietitian to discuss prior intake and habits, parents were required to make meal selections for individuals <18 years of age. Weighing protocol included postvoiding daily measurement at 7 AM with the patient wearing only a hospital gown. Patients stood on the scale with their back to the results screen (blind measurement). Weight gain expectation was at least 0.2 kg per day and liquid nutrition supplement shakes were given for failure to meet weight gain

goals. Patients were offered a tray of solid food at mealtimes three times per day as well as snacks prescribed by the dietitian to complete orally. The range of calorie prescription per day ranged from 1000 to 3000 kcal and increased daily, most commonly by 250 kcal, but this was determined by the dietitian and medical team based on the clinical course. Patients were seated on a bed or chair in their room for meals and remained on bed rest for at least 1-h after the meal. One trained clinician (registered nurse, clinical assistant, or dietitian) was present for mealtimes, in which individuals had 30 min to complete all components of the meal. Failure to complete the meal in the allotted time resulted in a 20-min allowance to complete the oral liquid nutrition supplement shake as a meal replacement under supervision. If an individual was still unable to complete liquid supplementation orally, an NG tube was placed at the bedside and remaining supplementation was given enterally. The NG tube was removed and the patient was offered an oral meal tray or snack at the next scheduled time; no patient was fed via continuous NG feeding in this cohort.

Laboratory values for phosphorous were derived from comprehensive metabolic panel blood samples taken by a trained phlebotomist or nurse, tested, and reported by the hospital laboratory. Standard practice included phosphorus prophylaxis with 250 mg twice per day of an oral phosphorous supplement as well as a multivitamin. The phosphorus supplementation dose was increased at clinician discretion for down-trending serum levels on daily laboratory evaluation, or if the level fell below the accepted definition of a serum phosphorus level of <3.0 mg/dl.[7,15]

The level of psychiatric functioning was obtained on admission, and patients were screened using either the Global Assessment of Functioning (GAF) or the Children's Global Assessment Scale (CGAS). This score and documentation was performed by the psychiatry consult liaison team. Two hundred thirty individuals were scored using CGAS score, 59 with GAF score, and 11 with neither score. As the tools are scored on the same scale (0–100 points, with a higher score reflecting a greater level of functioning) and measure the same variable, they are reported here together.

All variables were obtained from the individual patient medical record via automated data mining system as well as manual extraction by study staff. These variables included weight, height, BMI, heart rate, blood pressure, feeding route, caloric supplementation (from nursing documentation), age, sex (from the patient profile), percent median body weight as determined by 50th percentile BMI for height and age, caloric prescription (from dietitian documentation), laboratory values (from results), psychiatric diagnosis, and GAF or CGAS score (from psychiatry documentation).

The relationships between independent variables and the dependent variables were examined using chi-square analyses and one-way analyses of variance. Logistic regression examined factors associated with RH using the definition of a serum phosphorus level of < 3.0 mg/dl,[7,15] multivariate regression examined factors associated with serum phosphorus nadir. Multiple regression was repeated two additional times, excluding those diagnosed with atypical AN (n = 6) and again excluding outliers (cases with studentized residuals >2.5; n = 5). To determine if there were statistically significant differences between those who

received NG feeding and those who did not, chi-square analysis, *t*-tests, and logistic regression were performed. The institutional review board approved this study.

RESULTS

Three hundred inpatient charts were included in the analysis. The mean age at the time of admission was 15.5 years (SD = 2.5 years; range, 10.1–22.7 years), with a majority in the range of 10–19 years (288 of 300 patients, 96%). The cohort was primarily female (265 of 300 patients, 88.3%) and White/Caucasian (265 of 300 patients, 88.3%). See Table 1 for additional summary of patient characteristics.

The mean length of stay was 7.4 days (SD = 5.9 days). A large portion of the sample was diagnosed with the AN restricting subtype (255 of 300 patients, 85%), with smaller groups representing the AN binge/purge subtype (35 of 300 patinets, 11.7%) and atypical AN (6 of 300 patients, 2.0%); 1.3% (4 of 300) were not subtyped in clinical documentation. The mean baseline weight on admission was 42.8 kg (SD = 9.6 kg). The mean admission BMI was 16.3 (SD = 2.6) and the average percent mBMI was 82% (SD = 12.1%). Prior to the onset of the disorder, mean weight was 54.2 kg (SD = 15.3 kg), and individuals reported an average of 11.3 kg (SD = 9.7 kg) weight loss before admission. During hospitalization, the average weight gain was 1.8 kg (SD = 1.5 kg). Initial caloric prescription varied, with a mean of 1714 kcal per day (SD = 324.2 kcal per day).

The mean CGAS or GAF score on admission was 39.1 (SD = 11.6; range, 5–75). With regard to comorbid psychiatric diagnoses, >40% (125 of 300 patinets, 41.7%) of individuals were diagnosed with an anxiety disorder. Of those, 38% (114 of 300 patients) were classified as generalized anxiety disorder, 0.8% (24 of 300 patients) panic disorder, 0.8% (24 of 300 patients) posttraumatic stress disorder, 1.6% (5 of 300 patients) social anxiety disorder, 54% (162 of 300 patients) “Other” (eg, unspecified anxiety disorder, adjustment disorder with mixed anxiety, or anxiety disorder not otherwise specified), and 4% (12 of 300 patients) missing. See Table 2 for a further summary of clinical characteristics.

Eighty-six (28.7%) patient charts reported hypophosphatemia at some point during hospital stay; the mean day of occurrence was 3.9 (SD = 3.4), the mean phosphorus nadir of the cohort overall was 3.2 mg/dl (SD = 0.6 mg/dl), and the mean phosphorus nadir of those who reported hypophosphatemia was 2.5 mg/dl (SD = 0.3 mg/dl). Variables that were associated with hypophosphatemia during admission included age ($P = .001$), receiving NG feeding during hospitalization ($P = .052$), percent mBMI ($P = .019$), weight loss prior to admission ($P = .043$), weight gain during admission ($P < .001$), potassium nadir ($P < .001$), magnesium nadir ($P = .001$), and total liquid supplement in kcal ($P = .032$).

Table 3 shows the likelihood of hypophosphatemia. Magnesium nadir ($P = .024$), potassium nadir ($P = .001$), NG feeding during admission ($p = .054$), weight gained during hospitalization ($P = .003$), and age at admission ($P = .022$) contributed significantly to the likelihood of hypophosphatemia whereas weight loss prior to admission, percent mBMI on admission, and total formula in kcal did not. The model was significant ($x^2 = 59.3$; $P < .001$), and accounted for 21.9% (Cox and Snell R^2) to 31.0% (Nagelkerke R^2) of the

variance in the likelihood of RH. The Hosmer and Lemeshow test was not statistically significant ($P = .445$), indicating the data was a good fit to the model. For each unit reduction in potassium, the odds of RH increased by 9.2 and for each unit reduction in magnesium, the odds of RH were 13.7 higher. Those who received NG feeding during hospitalization were found to have three-times higher odds of RH, every kilogram of weight gain during hospitalization was associated with 1.5-times higher odds of RH, and increasing age was associated with 1.2-times higher likelihood of RH. Results were unchanged when outliers (n = 5) and individuals diagnosed with atypical AN (n = 6) were excluded.

Those who received NG feeding (n = 44) did not differ at baseline assessment by sex, age on admission, BMI, percent mBMI, premorbid weight, or weight loss prior to admission. Additionally, when considering hypophosphatemia, the NG group did not differ on weight gain during admission, potassium nadir, or magnesium nadir. However, the starting energy prescription of those who received NG feeding was significantly higher than those who did not ($P = .023$), and those individuals received more total calories of formula over the course of admission ($P < .001$).

Variables that were significantly associated with phosphorus nadir were bradycardia ($P = .047$), admission BMI ($P = .030$), overnight heart rate minimum ($P = .025$), magnesium nadir ($P = .002$), liquid supplement amount in kcal ($P = .037$), and anxiety disorder diagnosis subtype ($P = .002$). Factors that significantly predicted minimum serum phosphorus level (Table 4) included BMI and magnesium nadir. Magnesium nadir was the strongest contributor ($P < .001$), with every one-unit increase in magnesium resulting in a 1.213 increase in phosphorus, followed by BMI ($P = .002$), with each point adding a .060 increase in phosphorus.

DISCUSSION

Our findings indicate that serum magnesium and potassium, NG feeding, weight gain during admission, and age on admission were predictors of RH and that magnesium nadir and BMI on admission were predictors of phosphorus nadir in adolescents and young adults with AN, despite standardized phosphorus supplementation. This indicates that multiple co-occurring factors may be used to evaluate the likelihood of hypophosphatemia or down-trending serum phosphorus levels during medical hospitalization. The odds of developing RH were highest in those with other electrolyte disturbances, suggesting particular risk in this group. To the authors' knowledge, this is the largest sample specifically examining the predictors of RH in youth, building on prior studies including small cohorts of youth and adults.

Although a mean phosphorus nadir of 3.2 mg/dl in the sample does not represent RH, it is of clinical significance when considering that all participants in this cohort were provided with phosphorus supplementation. The accepted cutoff for a normal range of serum phosphorus is 3.0 mg/dl,[7,15] therefore a nadir on the low end of normal, even while on supplementation, may indicate that, without such intervention, more in the population may have reached the threshold for RH and, thus, is supportive of prophylactic phosphorus supplementation. This also suggests that, even with supplementation, an individual's phosphorus levels bear close clinical monitoring and support frequent laboratory surveillance. Given the gravity

of RH and the potential for serious medical complications, clinicians may make treatment-related decisions, such as supplemental phosphorus dose, energy prescription, or transition to outpatient or psychiatric care, based on down-trending serum phosphorus prior to the event of RH.[20] As such, the identification of what predicts phosphorus nadir is also of great clinical significance, in addition to examining RH.

The results suggest that low levels of serum potassium and magnesium predicted both RH and phosphorus nadir. This finding is consistent with literature that highlights the importance of electrolyte imbalances during nutrition restoration.[13,28,35–37] Despite hypokalemia's importance as being the most frequent electrolyte disturbance in this population,[17] in this study, individuals with hypomagnesemia were at a higher risk of RH than those with hypokalemia, and hypomagnesemia was notably a predictor of both RH and phosphorus nadir. As such, clinicians should consider hypomagnesemia as a part of standard laboratory monitoring. Additionally, the temporal relationship between hypokalemia, hypomagnesemia, and hypophosphatemia were not elucidated within the scope of this work and may yield results that guide clinical management.

Underscoring the clinical importance of NG feeding, it was associated with three-times higher odds of RH in this population. Considering the clinical implications, NG feeding was kept in analysis despite the variable association *P*-value ($P = .052$) and was reported in the model despite the corresponding *P*-value ($P = .054$). There is not a clear consensus in the literature on whether NG feeding itself or a method of NG feeding as continuous or bolus nutrition is predictive of, or protective against RH. Systematic and integrative reviews have found between 1% and 35% incidence of RH among individuals diagnosed with AN and fed via NG.[21,22]

The meal-based protocol utilized in this population determined that NG feeding was only provided in cases of food refusal or failure to complete meals, in contrast with other programs in which medically unstable adolescents with AN continuously fed via NG on admission showed no RH during a 2.5-week hospitalization.[23] Therefore, group differences were compared between those who received and did not receive this intervention. The results presented here suggest that those who received NG feeding did not display a greater degree of starvation, as there were no significant differences in weight on admission, percent median body weight, BMI, or reported weight loss prior to admission compared with those who did not receive NG feeding. Though the potential association with RH when fed via NG warrants further investigation in future studies, this type of feeding may be an optimal method in cases in which patients are unable to feed orally or to promote short-term weight gain,[22] and it has been found to be a safe alternative to oral feeding.[21] Of particular interest for future work would be to investigate the severity of ED-specific symptomatology as a potential moderator of the association between RH and NG feeding, which was outside the scope of this work.

Traditionally, a lower energy diet during hospitalization has been suggested to prevent refeeding syndrome. However, higher energy prescription at admission has been recommended to expedite weight gain and promote shorter duration of hospitalization,[24,27] and it has not been associated with poor outcomes in cohort studies[24–26] or randomized

control trials examining energy intake.[27,28] Starting energy prescription was not significantly associated with either hypophosphatemia or phosphorus nadir in this population of patients who received phosphorus prophylaxis as the standard of care. These findings support higher energy prescription during nutrition rehabilitation, with consideration of frequent laboratory assessments and phosphorus supplementation. Additionally, although kcal from NG was ascertained in this work, energy load related to solid food intake was not within the scope of this study and would be of clinical interest.

In this cohort, BMI on admission was predictive of phosphorus nadir but not RH, and percent mBMI on admission was associated with RH but was not statistically significant in the model. The fact that absolute BMI was predictive of nadir but not RH may point to clinician intervention with an increased phosphorus dose for those who have down-trending serum phosphorus levels on monitoring. Low BMI on admission has been previously cited in the literature as a predictor of RH in individuals with AN, whereas other reports highlight the importance of percent mBMI as an indicator of malnutrition.[7,18,26,41] The findings presented here indicate the importance of both weight measures, however they do not support percent mBMI as a predictor of RH.

Weight restoration is a primary goal of medical hospitalization for individuals with AN.[2–5] The results here show that every kilogram of weight gained during hospitalization was associated with 1.5-times higher odds of developing hypophosphatemia. This finding is in line with research that suggests that low BMI on admission was predictive of RH,[7] in that those with lower weight on admission have more weight to gain during the nutrition rehabilitation process. This study did not examine the rate of weight gain; however, previous reports have found that the rate of weight gain was not predictive of RH.[28] The reported weight loss prior to admission was not predictive of RH in this population. This is in contrast with recent studies that have suggested weight loss, not absolute weight at the time of admission, was predictive of poor outcomes like vital sign abnormalities and low serum phosphorus.[30]

For every year of age, the likelihood of RH increased in this sample. Previous studies have described poorer outcomes for individuals with older onset or age at initial assessment for AN.[30–32] Considering the peak incidence of AN occurs between 15 and 19 years of age,[32,33] older age at assessment or treatment may be related to longer duration of illness.[34] Identifying the difference between age as a predictor of RH vs longer duration of illness was not ascertained in this study and will be an important consideration in future studies to determine if longstanding nutrition depletion has implications in the risk of RH. Clinicians may consider older age on admission and duration of illness when determining appropriate phosphorus supplementation or the frequency of electrolyte monitoring.

This study is not without limitations. First, retrospective studies are limited by the availability and existing documentation of variables. As such, factors such as white blood cell count[28] were not available for analysis. Intrinsic to the 7-year date range utilized in this study are variations in clinical practice, which influenced both documentation and procedures. Additionally, the classification of AN was redefined with the release of the *DSM-5* in 2013. Subsequently, AN subtype and the identification of AN cases may reflect

these changes in persons admitted prior to 2013 and those admitted after the *DSM-5* publication date. The individuals included in this study may not be representative of all adolescents and young adults diagnosed with AN (eg, an underrepresentation of males and minority populations and no available data related to sexual or gender orientation).The setting was a single site in the northeastern part of the United States, which utilized a site-specific protocol for nutrition rehabilitation. Alternative feeding regimens, phosphorus supplementation, laboratory monitoring, psychiatric care plans, and other aspects of inpatient medical hospitalization were not explored or compared in this current study. Finally, it is notable that prescribed energy may differ from the actual energy consumed. Future prospective work should consider energy counts to more accurately assess actual intake.

The overall results of this study provide information regarding the risk of RH; yet, they do not fully capture the risk in this population. Together, predictors accounted for only 21%–31% of the variance in the likelihood of RH during medical admission in this population. This relatively low percentage of explained variance highlights the need for further investigation. However, the findings highlight that multiple predictors may need to be considered when examining RH risk, and, in particular, for individuals who are older, have received NG feeding, gained more weight during admission, and have low serum magnesium and potassium levels. Clinical scoring tools that consider multiple predictors of an outcome have been useful in a number of medical settings, including the determination of acute appendicitis and cancer risk.[39,40] Future research directed toward further identification of models that can accurately predict RH may inform the development of a clinical scoring tool similar to those found in other populations. Such clinical scoring tools for use in this population may guide clinicians with regard to energy prescription, phosphorus supplementation, electrolyte monitoring, and risk stratification during inpatient care.

In summary, variations in clinical practice with regard to length of stay, phosphorus supplementation, use of NG feedings, and energy prescription are indicative of a need for more definitive research into what individual or cumulative factors lead to the greatest chance of RH during the nutrition rehabilitation phase of treatment for AN.[20] Results of this study advance the knowledge of the risk of development of RH or changes in serum phosphorus levels in this population. The factors identified provide insight into potential areas for monitoring and individualized care plans for the prevention and management of the outcome. Despite phosphorus supplementation, individuals may be at risk for RH and warrant close clinical monitoring. Clinicians should consider age, use of NG feeding, weight gain during hospitalization, and electrolyte values when evaluating the frequency of phosphorus monitoring, the use of phosphorus supplementation, and energy prescription.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author, Meredith Kells, upon request.

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TABLE 1

Demographic characteristics of sample

	Mean (SD)	Range
Age	15.5 (2.5)	10.1–22.7
	n	**%**
Age categories		
Adolescent[a] (10–19 years)	288	96
Young adult (20–22.7 years)	12	4
Gender		
Female	265	88.3
Male	35	11.7
Race		
Asian	13	4.3
Black/African American	4	1.3
Other	16	5.3
White/Caucasian	241	80.3
Missing	26	8.7
Ethnicity		
Hispanic or Latino	11	3.7
Not Hispanic or Latino	241	80.3
Missing	48	16

[a]The World Health Organization (WHO) definition of adolescence is 10–19 years of age.

TABLE 2

Descriptive characteristics of sample

	n	%
Anorexia nervosa subtype		
Restricting	255	85
Binge/purge	35	11.7
Atypical	6	2.0
Missing	4	1.3
Anxiety disorder diagnosis	126	41.7
Anxiety disorder subtype		
Generalized anxiety disorder	48	38
Panic disorder	1	0.8
Posttraumatic stress disorder	1	0.8
Social anxiety disorder	3	1.6
Other	68	54
Missing	5	4
	Mean (SD)	**Range**
Length of stay	7.4 (5.9)	1–71
Admission		
Baseline weight, kg	42.8 (9.6)	17.4–75.6
BMI, kg/m^2	16.3 (2.6)	10.1–25.5
Percent mBMI	82 (12.1)	52–139.3
Premorbid Weight		
Weight, kg	54.2 (15.3)	23.1–136.1
Weight loss, kg	11.3 (9.7)	−7.3 to 75.5
Weight gain during admission, kg	1.8 (1.5)	−5.3 to 7
Initial calorie prescription, kcal	1714 (324.2)	1000–3000
CGAS or GAF score	39.1 (11.6)	5–75
Phosphorus nadir, mg/dl	3.2 (0.6)	1.5–5.0
Hospital Day # of Hypophosphatemia	3.9 (3.4)	1–23

Abbreviations: BMI, body mass index; CGAS, Children's Global Assessment Scale; GAF, Global Assessment of Functioning; mBMI, median BMI.

TABLE 3

Logistic regression predicting likelihood of hypophosphatemia using all factors associated with the outcome

	B	SE	Wald	df	*P*-value	Odds ratio	95% CI
NG feed during admission	1.078	0.559	3.713	1	.054	2.937	0.982–8.789
Percent mBMI	−0.003	0.015	0.031	1	.861	0.997	0.968–1.028
Weight loss prior to admit	0.011	0.018	0.361	1	.548	1.011	0.975–1.048
Age at admit	0.169	0.074	5.245	1	.022	1.184	1.025–1.368
Weight gain during admit	0.427	0.142	8.985	1	.003	1.533	1.159–2.2026
Potassium nadir	−2.222	0.671	10.958	1	.001	9.259	2.475–34.482
Magnesium nadir	−2.622	1.164	5.080	1	.024	13.7	1.408–142.857
Total formula, kcal	0.000	0.000	0.210	1	.647	1.000	0.999–1.001

Note: Constant 8.968.

Abbreviations: B, unstandardized regression coefficient; mBMI, median body mass index; NG, nasogastric.

TABLE 4

Summary of multiple regression analysis

	B	SE_B	β
Intercept	−.135	.695	
BMI	.060	.019	.271[a]
Magnesium nadir	1.213	.314	.324[a]

Abbreviations: B, unstandardized regression coefficient; β, standardized coefficient; BMI, body mass index; SE_B, standard error of the coefficient.

[a] $P < .05$.