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Autism Spectrum symptoms in a population of extremely undernourished patients with Anorexia Nervosa: a pilot study

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ABSTRACT

Objectives: Symptoms of Autism Spectrum Disorder (ASD) are overrepresented among individuals with Anorexia Nervosa (AN). We aimed to study the prevalence of ASD symptoms in extremely undernourished inpatients with AN and focus on potential cognitive and nutritional correlates.

Methods: We recruited prospectively all extremely undernourished patients with AN admitted to the Nutritional Care Unit of Paul Brousse Hospital over four months. ASD traits and AN psychopathology were assessed by administering the Autism Quotient (AQ) and the Eating Disorder Inventory-2 (EDI-2). Neuropsychological and bio-nutritional data were also collected.

Results: Among 33 participants aged 25.5 ± 9.3 (91 % females, BMI: 12.1 ± 1.4), 12 % scored above the clinical cut-off on the AQ. Independently of age and BMI, EDI-2 scores were significantly correlated with AQ-total scores ($r = 0.62$; $p < 0.01$), higher levels of cognitive rigidity ($r = 0.61$; $p < 0.001$) and sensory sensitivity alterations ($r = 0.69$; $p < 0.01$). In the cross-sectional model, the AQ-communication domain, and the GSQ-hyposensitivity scale were significant predictors ($p < 0.5$) of the variance in EDI-2 scores. Negative beliefs and cognitive confidence were associated with lower levels of Transthyretin ($p < 0.02$).

Conclusions: ASD traits presented high prevalence in extremely undernourished patients with AN. Despite the impact that malnutrition can have on cognitive functioning, most of these traits appear to be independent of BMI but associated with the severity of eating psychopathology; only Transthyretin was associated with dysfunctional metacognitions. These preliminary results suggest that ASD symptoms might co-occur in severe AN and require specific therapeutic interventions.

1. Introduction & Aims

Anorexia nervosa (AN; APA, 2013) is a severe eating disorder (ED; APA, 2013) characterized by very low body weight, restricted caloric intake, an intense fear of gaining weight, and an altered perception of one's body weight or shape (APA, 2013). Still, without standard treatment, AN is much more common in women than in men and generally occurs in adolescence (Berends et al., 2018). AN has the highest mortality rate among EDs and the condition can persist for more than 20 years (Arcelus et al., 2011; Eddy et al., 2016). With the advent of the COVID-19 pandemic, there has been an increase in the monthly incidence of AN cases by more than 60 % (from 24.5 to 40.6 cases) and the number of hospitalizations for the most severe forms of AN has almost tripled (from 7.5 to 20 cases in average) (Goldberg et al., 2022). Both somatic and psychiatric symptoms denote the severity of AN and are affected by the patient's condition of undernutrition (APA, 2013). Extreme undernutrition can result in negative effects not only on the somatic and metabolic levels but also on the cognitive level of the patient. Already in 1945, the "Minnesota Starvation Experiment," showed how a condition of extreme undernutrition in association with

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micronutrient deficiencies can lead to somatic complications and impact cognitive functioning, implying neuro-immune alterations in the hippocampal and prefrontal areas of the cerebral cortex (Keys et al., 1950; Butler et al., 2021). Phenotypically, such cognitive difficulties would appear to be similar to those observed in patients with autism spectrum disorder (ASD; APA, 2013). Although AN and ASD refer to distinct and well-defined diagnostic categories, in 1983 based on Gillberg's clinical observations, people began to hypothesize the existence of a genetic vulnerability common to the two disorders (Gillberg, 1983). Over the past fifteen years, research has documented a high presence of comorbid autistic symptoms in people with AN (Huke et al., 2013; Westwood and Tchanturia, 2017) and similarities between the two conditions in terms of neuropsychological profiles and social-emotional difficulties (Westwood et al., 2017). These common features appear to be related to difficulties in emotion recognition (Kerr-Gaffney et al., 2020b), empathic skills (Kerr-Gaffney et al., 2020a), and emotional introspection, but also attentional skills and the ability to change perspective (Courty et al., 2013). These areas of symptomatic overlap between AN and ASD suggest the need to adjust the symptomatic assessment of the eating disorder with an adequate assessment of cognitive functioning, as any difficulty could lead to an exacerbation of the clinical picture and thus be relevant for treatment implications (Tchanturia et al., 2017). In trying to define a clinical profile of the AN-ASD condition, the presence of comorbid autistic traits in AN would appear to be associated with a body mass index (BMI) of less than 16 kg/m^2 and the absence of purging behaviors (Fornaro et al., 2020; Boltri and Sapuppo, 2021). From a metacognitive perspective, erroneous metacognitive beliefs and negative appraisals concerning food and weight appear to contribute to greater cognitive rigidity and may promote the maintenance of the ED (Sapuppo et al., 2018). Determining the impact of metacognitive beliefs on eating behavior in AN, could be useful in assessing the role of metacognition in psychopathology and impaired cognitive functioning. From a biological perspective, research has also found similar alterations in inflammatory and gut microbial diversity in both disorders (Gabriel et al., 2019; Hanachi et al., 2018; X. Yap et al., 2021). In this context, a clinical assessment of autistic symptoms and their association with biological and nutritional variables would promote future evaluation of the association between the presence of possible AN-ASD comorbidity in gut dysbiosis phenomena. Furthermore, sensory sensitivity alterations, frequently linked to the presence of autistic symptoms have been observed in some patients with AN and could be involved in the exacerbation of food restrictive behaviors (Kinnaird et al., 2017, 2020). An assessment of the sensory profile of patients with AN could therefore clarify the link between AN and ASD and at the same time guide the development of strategies to prevent a state of altered sensory sensitivity from affecting treatment efficacy.

The main objective of this study was to assess the prevalence of autistic symptoms in patients with AN and extreme undernutrition admitted to the Clinical Nutrition unit of Paul Brousse University Hospital in Paris, France. The secondary objectives of the study were the following: (i) explore the associations between ASD symptoms, the severity of psychopathology, and biological-nutritional variables derived from standard biological analyses; (ii) assess the cognitive and metacognitive functioning of patients with AN and with severe undernutrition; and (iii) determine the sensory profile of these patients, in relation to the severity of the psychopathological profile.

To our knowledge, this pilot study was the first attempt to assess the prevalence of ASD symptoms in a sample of AN patients with severe undernutrition (BMI $\leq$16 kg/m^2) in relation to their nutritional profile.

2. Methods

2.1. Study design

This is a cross-sectional observational monocentric pilot study conducted between November 2021 and March 2022.

2.2. Participants

Participants were recruited from patients hospitalized for AN and severe undernutrition in the Clinical Nutrition Unit of Paul Brousse University Hospital in Villejuif, France. This unit is considered, in France, a regional and national "referral center" by the administrative authorities for the management and treatment of extremely severe undernutrition and its life-threatening medical complications associated with EDs. The unit is partner of both the regional and national ED professional network associating all French ED professionals in a collaborative network. The multidisciplinary approach of the unit involves diverse professionals (nurses, dieticians, physician nutritionists, psychiatrists and psychologists) in the clinical evaluation of patients as well as different therapeutic strategies (vitamin and mineral supplementation, enteral nutrition, dietary re-education and psychological support).

Inclusion criteria for the study were as follows: in-patients being hospitalized for AN, as defined by the DSM-5, admitted with severe undernutrition, defined by a BMI <15 kg/m^2 or a sudden and rapid weight loss, and aged over 18. Patients with no diagnosis of AN, minors, those with an intellectual disability, and those with severe somatic or psychiatric comorbidities were not included. All patients admitted for a brief metabolic-nutritional evaluation were also excluded. Of the 34 patients who were asked to participate in the study, only one declined. The flowchart of inclusions according to the above-mentioned criteria is reported in Fig. 1.

2.3. Procedure

Patients who met the inclusion criteria were asked to give their consent to participate in the study by signing an information note. This document, along with the questionnaires used and the methods of data collection and storage, was approved by the ethics committee of Paris-Saclay University, *Comité d'Ethique de la Recherche* (CER) POLETHIS. The reference for this approval is CER-Paris-Saclay-2022-031.

2.4. Data collection

For each patient, the following parameters were collected: (i) family and personal psychiatric history; (ii) information on environment and perinatality; and (iii) clinical and bio-nutritional data. Patients were asked to respond to self-administered questionnaires for clinical evaluations.

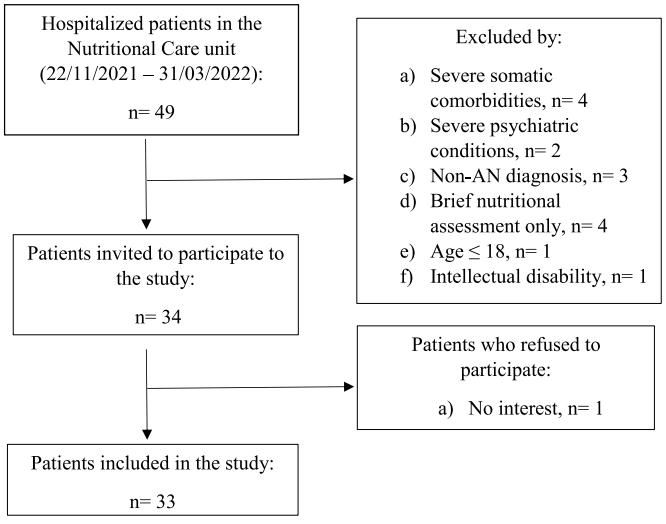

Fig. 1. Flow chart: inclusions of study participants (November 2021–March 2022).

The following clinical data were collected: age, gender, weight, height (BMI calculation), type of AN (AN-R or AN-BP type) according to the DSM-5 definition, associated pathological behaviors (physical hyperactivity, purging behaviors), and psychotropic treatment. The nutritional status variables were collected through standard biological assessment procedures and they included albumin, transthyretin, citrulline, selenium, zinc, copper, thiamin, folates vitamin B12, vitamin D, thyroid-stimulating hormone (TSH) and thyroid hormones T3 and T4. The clinical-biological data were collected at admission to the unit.

Patients who participated in the study answered five self-administered questionnaires in paper version. Completion of the questionnaires was self-administered; the patient was left alone in his or her room for the entire duration of completion (between 40 and 60 min in total). The questionnaires used were: Eating Disorder Inventory 2nd Edition (EDI-2; Garner, 1991), Autism spectrum Quotient (AQ; Baron--Cohen et al., 2001), Detail and Flexibility Questionnaire (D-Flex; Roberts et al., 2011), Metacognitive Questionnaire 30 (MCQ-30; Cartwright-Hatton and Wells, 1997), Glasgow Sensory Questionnaire (GSQ; Robertson and Simmons, 2013).

2.5. Psychometric instruments

Eating Disorder Inventory 2nd Edition (EDI-2; Garner, 1991): it is a self-administered instrument that assesses cognitive and behavioral characteristics commonly associated with AN and BN in 91 items. Responses are formulated on a 6-point Likert scale, constituting 11 factors: the drive for thinness (DT), bulimia (B), body dissatisfaction (BD), perfectionism (P), ineffectiveness (IN), interpersonal distrust (ID), maturity fears (MF), interoceptive awareness (IA), social insecurity (SI), asceticism (ASC), and impulse regulation (I). Higher scores correspond to greater severity of disordered eating behaviors. Subscales are used to examine the characteristics of these behaviors. Chronbach's α: 0.88.

Autism spectrum Quotient (A; Baron-Cohen S et al., 2001) is a self-administered questionnaire assessing ASD symptomatology in 50 items, constituting five domains. The screening cut-off is set at 26 items and the clinical cut-off at 32. The five domains include social skills, set-shifting, attention to detail, communication, and imagination. The AQ is used as first screening procedure to detect the presence of ASD. For the purpose of this study, both the screening and clinical cut-offs were used to indicate the presence of elevated symptoms associated with ASD rather than to diagnose ASD. Chronbach's α: 0.84.

Detail and Flexibility Questionnaire (D-Flex; Roberts et al., 2011): it is a 24-item self-administered questionnaire that assesses two aspects of neurocognitive functioning: cognitive rigidity and attention to detail. Higher scores represent greater cognitive rigidity/attention to detail. Chronbach's α: 0.95.

Metacognitive Questionnaire 30 (MCQ-30; Cartwright-Hatton and Wells, 1997): it is a self-administered questionnaire assessing metacognition based on responses to 30 items. Generates scores for the following five subscales: positive beliefs related to worry (POS); the need for control (NC); (lack of) cognitive confidence (CC); negative beliefs (NEG); cognitive self-consciousness (CSC). Higher scores indicate high levels of unhelpful metacognitions. Chronbach's α: 0.89.

2.6. Data analyses

Statistical analyses were conducted using R 3.6.0 statistical software for Windows. Descriptive statistics for demographic, clinical, and bio-nutritional variables were calculated for the entire sample, and quantitative data were expressed as Mean (M) and Standard Deviation (SD). Descriptive and comparative analyses of the total scores and subscales of the following questionnaires were performed: EDI-2, AQ, D-Flex, MCQ-30, and GSQ. Shapiro's test was performed to assess the normality of the data distribution. Not all continuous variables followed a normal distribution, so a logarithmic transformation of these variables was performed to meet the normality assumption required by the parametric procedure and to calculate Pearson's correlations. Multiple regressions were also conducted controlling for age and BMI to study significant correlations between the EDI-2 scores and the various subscales of the other psychometric questionnaires. Two-tailed significance of the results was based on p values ≤ 0.05. Finally, a cross-sectional model was conducted by multiple regression with a step-wise method to identify the possible predictors of the variance of EDI-2 score.

3. Results

3.1. Clinical and biological sample characteristics

Of the 34 patients who met the inclusion criteria and were proposed to participate in the study, only one declined (see flow chart of inclusions). The clinical-biological and demographic characteristics of the

Table 1
Clinical, psychometric and biological variables.

N = 33	Mean (SD), N (%)	Min-Max
Age (years)	25.5 (9.3)	–
Gender (F/M)	30/3 (91 % female)	–
AN type	AN-R = 26 (78.8 %); AN-BP = 7 (21.2 %)	–
Weight (kg)	32.7 (5.6)	–
BMI (kg/m^2)	12.1 (1.4)	–
Duration of illness (years)	8.1 (10.2)	–
Number of hospitalizations (lifetime)	3.6 (4.8)	–
Psychotropic treatment	13 (4.3 %)	–
EDI-2 total score	80.0 (37.1)	15–165
AQ- total score	21.2 (8.6)	6–46
NAS (AQ score ≤25)	82 % (n = 26)	–
SCAS (AQ score ≥26)	6 % (n = 2)	–
HAS (AQ score ≥32)	12 % (n = 4)	–
AQ *social skills*	3.6 (2.7)	0–10
AQ *attention switching*	4.8 (2.6)	1 10
AQ *local details*	5.4 (2.7)	1–10
AQ *communication*	3.5 (2.9)	0–10
AQ *imagination*	3.8 (1.8)	1–10
DFlex total scores	89.3 (24.3)	42–132
D-Flex-R	48.9 (11.4)	26–67
D-Flex-D	40.4 (14.0)	15–68
MCQ-30 total	67.4 (20.0)	35–117
POS	12.7 (4.7)	6–24
NEG	14.7 (4.7)	7–22
CC	10.4 (5.0)	6–24
NC	12.8 (5.0)	6–23
CSC	16.8 (4.6)	7–24
GSQ hyposensitivity	26.4 (10.7)	7–48
GSQ hypersensitivity	31.2 (14.7)	4–66
Albumin (g/L)	39.9 (5.0)	29–50
Transthyretin (g/L)	0.23 (0.6)	0.1–0.4
Citrulline (μmol/L)	28.3 (9.3)	12–47
TSH (mUI/L)	2.0 (1.5)	0.3–7.5
T3 (pmol/L)	3.3 (0.8)	1.9–5.7
T4 (pmol/L)	13.1 (3.1)	1.6–18.4
Selenium (μmol/L)	1.4 (0.2)	0.7–1.8
Zinc (μmol/L)	16.8 (6.7)	8.6–37.6
Copper (μmol/L)	14.5 (4.0)	8.0–23.7
Thiamine (nmol/L)	162.3 (86.1)	45.0–456.0
Vitamin B12 (ng/L)	865.2 (589.6)	200.0–2448.0
Folates (ng/L)	12.9 (5.4)	3.2–20.0
Vitamin D (ng/L)	35.5 (19.1)	10–81

AN anorexia nervosa; AN-BP anorexia nervosa binge-purging; AQ autism quotient; AN-R anorexia nervosa restrictive type; BMI body mass index; CC cognitive confidence; CSC cognitive self-consciousness; D-Flex-R detail and flexibility questionnaire rigidity subscale; D-Flex-D detail and flexibility questionnaire attention to details subscale; EDI-2 eating disorder inventory 2nd edition; GSQ glasgow sensory questionnaire; HAS high autism spectrum symptoms; MCQ-30 metacognitive questionnaire 30 item; NAS non autism spectrum symptoms; NC need of control; NEG negative beliefs; POS positive beliefs; SCAS sub-clinical autism spectrum symptoms; SD standard deviation; TSH thyroid stimulating hormone; T3 triiodothyronine; T4 thyroxine.

sample are summarized in Table 1.

Among 33 patients (91 % females) with a mean age of 25.5 ± 9.3 and a mean BMI of 12.1 ± 1.4, 78.8 % (N = 26) had AN restrictive type (AN-R), while 21.2 % (N = 7) had the binge-purging type (AN-BP). The mean illness duration was 8.1 ± 10.2 years and at data collection 4.3 % (N = 13) of participants were under psychotropic treatment. The sample had a mean number of lifetime hospitalizations of 3.6 ± 4.8. Despite low body weight (32.7 ± 5.6) and severe undernutrition, mean deficiencies in micronutrients were not significant. 75.7 % of the sample (N = 25) had family psychiatric antecedents (first-degree relatives). Regarding eating disorders, 32 % (N = 8) had antecedents of AN and 8 % (N = 2) of BN; none reported antecedents of Binge Eating Disorder (BED; APA, 2013). The most frequent family psychiatric antecedents in the sample included anxiety disorders (20 %), major depression (44 %), sleep disorders (44 %), and suicide attempts (40 %); only 8 % (N = 2) of the sample had family psychiatric antecedents of ASD. Relative to personal psychiatric antecedents, 30 % of the sample (N = 10) reported suffering from anxiety disorders, 27.3 % (N = 9) from obsessive-compulsive disorder, 24.2 % (N = 8) from depression, 27.3 % (N = 9) from sleep disorders, 12.1 % (N = 4) from self-harm, and 9 % (N = 3) reported attempting suicide; only one participant reported having an overt diagnosis of ASD. Data about family and personal psychiatric antecedents, environment and perinatality are reported in Table S1 in annexes.

3.2. Psychometric characteristics of the sample

With regard to the psychometric characteristics of the sample, the mean total eating psychopathology score, as measured by EDI-2, was 80.0 ± 37.1; particularly high mean scores were found in the sub-scales measuring the drive for thinness (DT), difficulties in interoceptive awareness (IA), ineffectiveness (IN), social insecurity (SI) and body dissatisfaction (BD). For mean scores of the different questionnaires and their sub-scales in detail, refer to Table S2 in annexes.

In terms of autistic symptomatology, 18 % (N = 6) scored above the screening-cut off (≥26) on the AQ; and among them, 12 % (N = 4) scored above the clinical cut-off (≥32) for ASD. Particularly high scores were observed in the attention-switching and local details domain (see Table 1). Mean scores on cognitive rigidity and attention to details at the DFlex were high (89.3 ± 24.3), particularly, participants exhibited low cognitive flexibility (see Table 1). At the metacognitive level, the highest mean scores were found in the sub-scales NEG (negative beliefs) and CSC (cognitive self-consciousness), while concerning sensory sensitivity, measured using the GSQ questionnaire, the sample exhibits sensory abnormalities, in terms of hyper- and hyposensitivity, especially at visual and gustatory levels (see Table S2 in annexes).

3.3. Analyses results

12 % of patients (N = 4) had high autistic symptoms (HAS group) and 6 % (N = 2) had sub-threshold clinical symptoms (SCAS group), while 82 % (N = 23) of the sample had no autistic symptoms (NAS group) (see Table 1). Significant Pearson's correlations were found between EDI-2 scores and AQ scores (r = 0.62; p < 0.01). EDI-2 total scores were also significantly associated with DFlex cognitive rigidity scores (r = 0.61; p < 0.001) and with the presence of sensory abnormalities, measured on the GSQ (r = 0.69; p < 0.0001). Neither age, BMI, nor illness duration was significantly associated with total EDI-2 and AQ scores. No biological-nutritional variables were found to be significantly associated with AQ and EDI-2 total and subscales scores. We found a statistically significant negative correlation between serum level of Transthyretin (TTR) and NEG (negative beliefs) subscale scores (r = − 0.43; p = 0.01) and CC (lack of cognitive confidence) subscale scores (r = − 0.41; p = 0.02) on the MCQ-30 questionnaire. Main correlations are summarized in Table 2.

Multivariate linear regressions between the EDI-2 total score and other psychometric measures showed statistically significant associations between EDI-2 and AQ-attention-switching (β = 0.50; p = 0.01), AQ-communication (β = 0.50; p = 0.02), D-Flex-cognitive rigidity (β = 0.61; p = 0.0002), NEG (β = 0.60; p = 0.0005), sensory hypersensitivity (β = 0.55; p = 0.001) and sensory hyposensitivity (β = 0.71; p < 0.0001)

Table 3
Multivariate analyses and cross-sectional regression model.

	M(SD)	β	*t*	*p* value[a]		
DV: EDI-2 total score				MLR	Step-wise Model	Adj. R^2
AQ Attention-Switching	4.8 (2.6)	0.50	2.80	0.01*	0.3	–
AQ Communication	3.5 (2.9)	0.50	2.50	0.02*	**0.03***	0.66
Dflex, cognitive rigidity scale	48.9 (11.4)	0.61	4.20	0.0002**	0.8	–
GSQ hypersensitivity score	31.2 (14.7)	0.55	3.54	0.001**	0.4	
GSQ hyposensitivity score	26.4 (10.7)	0.71	6.13	0.0001**	**0.02***	0.55
MCQ Negative Beliefs	14.7 (4.7)	0.60	3.93	0.0005**	0.4	–

*p < 0.05; **p < 0.01.
DV dependent variable; MLR multiple linear regression.
[a] Adjusted for age and BMI.

Table 2
Pearson's correlations (*r* coefficient).

	Age	BMI	EDI-2 tot	AQ tot	DFlex-R	GSQ-tot	NEG	CC	TTR
Age									
BMI	-0.11								
EDI-2 tot	-0.17	-0.005							
AQ tot	0.12	0.2	0.62***						
DFlex -R	-0.12	0.02	0.61***	0.63***					
GSQ tot	-0.14	-0.01	0.69***	0.66***	0.63***				
NEG	-0.15	-0.02	0.60***	0.34*	0.56***	0.57***			
CC	0.04	0.01	0.45**	0.48**	0.63***	0.42**	0.54***		
TTR	0.08	- 0.01	- 0.16	-0.13	- 0.33	- 0.28	- 0.43*	- 0.41*	

*p < 0.05; **p < 0.01; ***p < 0.001.
D-Flex R cognitive rigidity; TTR transthyretin.

on the GSQ. These multivariate analyses are summarized in Table 3.

Subsequently, a cross-sectional model was constructed to investigate the possible and best predictors of variance in EDI-2 scores. These analyses were conducted step-wise (including each variable successively). Besides the variables resulting significant after multivariate analyses, also age and BMI were included in the model. 65 % of the variance (corresponding to the adjusted R^2) in the EDI-2 scores was explained by the cross-sectional model ($F(2.32) = 31.6$; $p < 0.001$), where only communication difficulties (AQ-communication) and sensory hyposensitivity (GSQ-hyposensitivity) were statistically significant ($p < 0.05$). Multivariate analyses and cross-sectional models are summarized in Table 3.

In the cross-sectional model, AQ-communication domain and GSQ-hyposensitivity were the best predictors of variance of EDI-2 scores and strongly associated with them. Correlation graphs between EDI-2 total scores, AQ-communication and GSQ-hyposensitivity are reported in Figs. 2 and 3.

4. Discussions

This pilot study aimed primarily to assess the prevalence of autistic symptoms in severely undernourished patients with AN and, secondly, to investigate the associations between ASD symptoms, the severity of the eating psychopathology, cognitive and metacognitive style, and sensory profile. Although previous studies (Fornaro et al., 2020; Numata et al., 2021; Pruccoli et al., 2021) have already investigated the prevalence of autistic symptoms in AN patients, this study represents the first attempt to assess the presence of ASD symptoms in patients with a diagnosis of severe undernutrition and a BMI <15 kg/m^2, undergoing somatic care in an in-patient clinical nutrition unit, and to investigate their associations with bio-nutritional and cognitive variables. Literature (Boltri and Sapuppo, 2021; Westwood and Tchanturia, 2017) suggests that between 10 and 30 % of AN patients show a relevant ASD symptomatology. Despite the small sample size, the results of this pilot study, appear to be in line with previous research, as 18 % of patients reported ASD symptoms above or below the clinical cut-off and, overall, 12 % had clinically relevant ASD symptoms, scoring above the clinical cut-off for ASD. Although previous studies (Fornaro et al., 2020) had hypothesized that the presence of autistic symptoms might be greater in the most severe cases of AN, in terms of BMI (when the latter is below 16 kg/m^2), the present study suggests that the percentage of autistic traits in this population tends to remain unchanged. It could be hypothesized that the presence of these symptoms does not depend directly on undernutrition and low body weight, but rather on other specific psychopathologic factors since ASD symptoms were found to be significantly correlated with the severity of eating psychopathology, but not specifically with BMI. However, the small range of BMI due to the specific characteristics of the sample prevents the possibility of drawing any conclusions on the role of BMI in the development of autistic symptoms.

Undernutrition can affect cognitive functioning by exacerbating

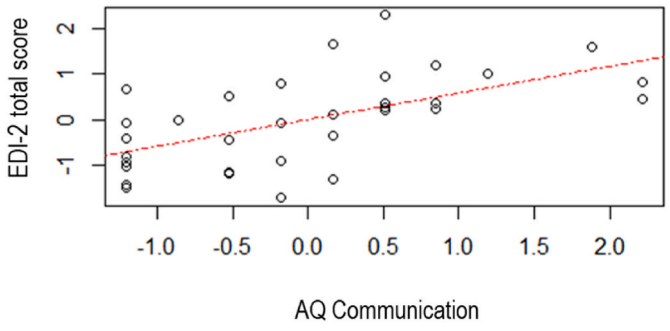

Fig. 2. Pearson's correlations between EDI-2 total scores and AQ-*communication* domain ($r = 0.62$; $p = 0.0001$).

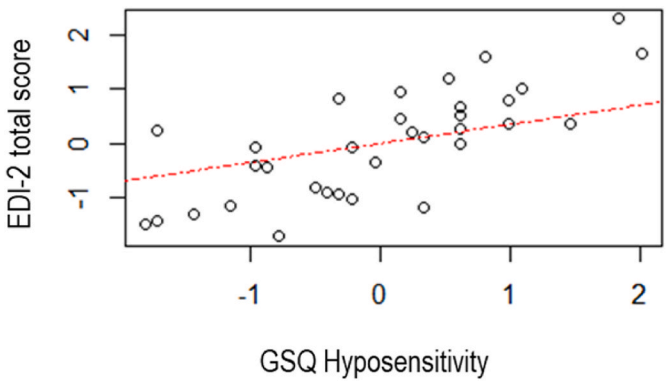

Fig. 3. Pearson's correlations between EDI-2 total scores and GSQ-hyposensibility total scores ($r = 0.75$; $p < 0.0001$).

cognitive rigidity (Butler et al., 2021; Keys et al., 1950), however, our results show that cognitive rigidity was associated with greater severity of the eating psychopathology, without being influenced by particular bio-nutritional deficiencies. This is in line with previous studies showing that weight recovery does not necessarily reflect a recovery in cognitive functioning (Olivo et al., 2019). The correlation between cognitive rigidity and ASD symptoms was also found to be significant, which makes it difficult to discern between patients who have extreme social difficulties and extreme cognitive rigidity and those who have real AN-ASD comorbidity. Furthermore, as in the study led by Westwood et al. (2017), no association was found between the presence of autistic symptoms and a marked focalization on local details. Further studies have assessed the prevalence of EDs in patients with ASD and without intellectual disability, and results seem to reinforce the hypothesis of a link between the two conditions, since these patients presented clinically-relevant eating disorder symptoms (Demartini et al., 2021; Nisticò et al., 2022), regardless of the level of undernutrition.

However, the step-wise regression model shows that the only significant predictors of variance in the severity of eating psychopathology (EDI-2 scores measures) were alterations in the communication domain and a marked sensory hyposensitivity, both factors commonly related to ASD conditions (Baum et al., 2015; Sapey-Triomphe et al., 2018).

The presence of autistic symptoms, may thus precede the onset of the eating disorder (Brede et al., 2020; Mandy and Tchanturia, 2015; Solmi et al., 2021) and contribute to a neuropsychological profile characterized by cognitive rigidity (Danner et al., 2012), socio-communicative difficulties, and poor emotion recognition skills (Bentz et al., 2017), and this tends to resist to the process of re-nourishment, micronutrient supplementation, and weight gain. The complex mechanism that may explain the co-occurrence of these two disorders, remains unknown, however, some studies have hypothesized that alexithymia, the difficulty in recognizing and expressing emotions, could possibly mediate the relationship between autistic symptoms and the severity of the eating psychopathology in women with AN (Vuillier et al., 2020). In the general population, social-emotional difficulties, similar to those found in ASD, may be risk factors for the occurrence of EDs (Mansour et al., 2016). Nonetheless, it might be possible that autistic traits in AN are indirectly associated with the ED, through the mediation of anxiety or depressive symptomatology (Calderoni et al., 2015), and these factors must be considered during the assessment process.

Over the past decade, scientific interest continues to grow regarding the role of communication skills in facilitating the onset of EDs (Oldershaw et al., 2011). Indeed, it seems that people with EDs exhibit difficulties in communication compared to healthy subjects (Ambwani et al., 2016; Renwick et al., 2015). In accordance with previous studies (Mason et al., 2021; Solmi et al., 2021), our results show that, regardless of the severity of undernutrition, ASD-like communication difficulties can predict 66 % of the variance in ED severity. In a recent longitudinal

study, Schaumberg and colleagues (2021), found that in female subjects, communication difficulties at age 8 were associated with the presence of ED symptoms at age 14, particularly in terms of body dissatisfaction and food restriction; no such association was found in male peers. Furthermore, since having a psychiatric family history of AN could lead to an increased risk of ASD in offspring, the association between social-communicative difficulties and EDs would seem to have a possible genetic basis (Koch et al., 2015; Lundin Remnélius et al., 2021). A careful screening procedure to identify socio-communicative difficulties, which were already present during childhood, and then exacerbated by the onset of an ED, would be useful to understand and learn more about the patient's global functioning, but also to consider the introduction of specific therapeutic interventions to target these difficulties, which could indirectly promote successful recovery from the ED.

Sensory sensitivity and perception are pivotal in determining the development of higher-order cognitive functions and play a crucial role in linguistic, communicative, and interactional processes (Baum et al., 2015). More than 90 % of patients with ASD complain of sensory abnormalities (Crane et al., 2009; Marco et al., 2011), involving multiple modalities simultaneously (Kern et al., 2007) and characterized by hypersensitivity (Baranek et al., 2006) or sensory hyposensitivity (Baum et al., 2015; Marco et al., 2012). In the present study, we found a significant correlation between ASD symptoms (AQ scores), and the presence of sensory abnormalities (GSQ scores). Results align with previous literature since the presence of sensory and perceptual abnormalities in ASD appears to be associated with greater severity of the disorder (Sapey-Triomphe et al., 2018). In particular, studies report that in ASD a condition of sensory hyposensitivity can be associated with greater difficulties in the social-communicative domain (Foss-Feig et al., 2012; Green et al., 2018). Both these psychopathological features specific to ASD, seem, therefore, to corroborate the severity of AN. In our cross-sectional model, we found that ASD-like communication difficulties could alone explain 65 % of the variance in EDI-2 scores. To date, only a few studies have explored the role of sensory abnormalities in AN and EDs in general, and results can be controversial (Fernández-Aranda et al., 2016; Hartman-Petrycka et al., 2022; Kinnaird et al., 2020). However, sensory abnormalities in AN, such as the presence of reduced olfactory discrimination ability, seem to be partly associated with cognitive rigidity (Karavia et al., 2022), and the presence of comorbid ASD traits (Tonacci et al., 2019). Similarly, reduced taste sensitivity would seem to be associated with the presence of EDs in adult ASD patients without intellectual disability (Nisticò et al., 2022).

The results of the present study show how difficulties in the socio-communicative domain and sensory abnormalities, both common in ASD, can independently of age and body weight, influence the severity of eating psychopathology in patients with AN. This could lead to new hypotheses on the presence of a possible clinical and psychopathological link between the two conditions, AN and ASD.

Research shows that the metacognitive profile in AN is characterized by higher levels of negative beliefs about uncontrollability and danger and lower cognitive confidence (Cooper et al., 2007; Woolrich et al., 2008), which would seem to correlate to the severity of the ED (Georgantopoulos et al., 2020; Sapuppo et al., 2018). Correlation analyses seem, in part, to confirm the existence of an association between psychopathological severity and the metacognitive characteristics, which would, then, appear to be associated with the presence of ASD symptoms. However, in the cross-sectional model, the impact of these metacognitive dysfunctions on the variance of eating psychopathology does not remain significant. This result could be explained by the presence of intervening variables, such as anxious or depressive symptoms, which are frequent in AN and could mediate the relationship between metacognitive characteristics and the eating disorder's symptomatology (Kaye et al., 2004; Keys et al., 1950; Pleplé et al., 2021). Regarding the association between the nutritional status, cognitive and metacognitive variables, the presence of metacognitive dysfunction, such as low cognitive confidence and negative beliefs about uncontrollability and danger, was significantly associated with low plasma levels of Transthyretin, an index of the level of malnutrition and an indicator of response to nutritional therapy (Winkler et al., 1989). This protein would appear to have a potential neuroprotective role and thus be involved in alterations in cognitive functioning, but literature findings are still controversial (Araghi et al., 2021; Vieira and Saraiva, 2014; Durmuş et al., 2021).

Despite this pilot study not allowing us to draw conclusions about the relationship between deficiencies in Transthyretin and the metacognitive profile, it has highlighted that nutritional deficiency, observed in severe AN, could have an impact on the cognitive and metacognitive profile, thus suggesting the importance of a nutritional assessment and a translational approach to treat EDs.

5. Limitations

The present pilot study was the first attempt to assess the prevalence of ASD symptoms in hospitalized patients with AN and severe undernutrition, focusing on cognitive and bio-nutritional correlates. However, this observational, cross-sectional study has several limitations. Firstly, the small sample size can limit both the interpretation and generalizability of results, but also the type of statistical analyses that were conducted. The small number of patients in the HAS and SCAS groups only allowed the performance of correlational analyses, but no between-group analyses could be conducted. These analyses could more clearly help draw assumptions on the link between ASD levels and ED severity. In addition, we only used a self-administered questionnaire, the AQ, to assess autistic symptomatology, whereas specific assessment instruments for neurodevelopmental disorders such as the Autism Diagnostic Interview-Revised (ADI-R; Lord et al., 1994) and the Autism Diagnostic Observation Schedule- 2nd Edition (ADOS-2; Lord et al., 2012) could lead to more clinically relevant results; however, this type of assessment is time-consuming to administer and would have been unsustainable for patients in such somatically and mentally fragile conditions. Due to the small sample size, it was not statistically possible to analyze differences according to the type of AN presented, since most of the sample had restrictive type profiles. Even with the EDI-2 being a self-administered questionnaire, the quality of responses may have been partly biased by the denial of psychopathology, which occurs very often in the most severe cases of AN. In addition, the possible presence of significant psychiatric comorbidities, including anxiety and depressive disorders, might be relevant confounding variables (Pleplé et al., 2021). Despite the low body weight and the severity of undernutrition, no particular micronutrient deficiencies were found, which could explain the lack of significant associations between nutritional status and cognitive and psychopathological variables. Most of the patients admitted to the unit were treated with enteral nutrition from the very beginning of their hospitalization, and very often they had previously been admitted to intensive care units, where they had already received supplementation in micronutrients and vitamins. Because of the great heterogeneity of psychotropic treatments and the low dosage to which patients were subjected, it was not possible to assess the effect of these on symptomatology; therefore, a longitudinal approach would be more appropriate. The results of the study show a significant association between the socio-communicative difficulties, typical of ASD, and the severity of eating psychopathology; however, when looking at the patients' antecedents, some of them have a severe disorder that counts numerous hospitalizations and onset in adolescence or pre-adolescence. Intuitively, such a developmental context and the isolation that the pathology entails do not seem to be favorable prerequisites for the proper development of communication and social skills. In addition, the physical and psychological impairment derived by food restriction behaviors that led the patient to the current state of undernutrition may have altered their sensory sensitivity, especially in terms of taste alteration. For this reason, it would be preferable for future studies to investigate sensory sensitivity through specific tests, obviating the

limitations of self-administered questionnaires, such as the GSQ.

6. Conclusions

The main objective of the present study was to assess the prevalence of ASD symptoms in severely undernourished patients with AN, taking into account cognitive, metacognitive, and bio-nutritional factors that may contribute to the complexity of the clinical picture. Despite the limitations represented by the small sample size and the study design, results indicate that the presence of socio-communicative and sensory difficulties, attributable to ASD traits, could contribute to a more severe AN profile in terms of psychopathology. The presence of these difficulties, regardless of the patient's nutritional status, appears, therefore, to be a relevant issue in AN, needing specific and targeted interventions and integration into the patient's global care. Since AN and EDs involve a dual problematic, somatic and psychological, future studies could adopt integrated and targeted approaches to understand and treat these disorders, researching possible associations between psychiatric comorbidities, specific biomarkers of nutritional status, and neuropsychological vulnerabilities, to be able to contribute to treatment in the direction of personalized care.

CRediT authorship contribution statement

Margherita Boltri: Conceptualization, Investigation, Data curation, Formal analysis, Writing – original draft, Methodology. **Tristan Gabriel-Segard:** Formal analysis, Methodology, Validation, Visualization. **Walter Sapuppo:** Supervision, Validation, Methodology, Writing – review & editing. **Nadja Kayser:** Validation, Supervision, Visualization, Writing – review & editing. **Marika Dicembre:** Data curation, Validation, Resources, Software. **Ani Yeganyan:** Validation, Visualization. **Maeva Duquesnoy:** Conceptualization, Supervision, Validation, Visualization, Writing – review & editing. **Marion Leboyer:** Supervision, Funding acquisition, Validation, Visualization, Writing – review & editing. **Mouna Hanachi:** Conceptualization, Supervision, Methodology, Project administration, Validation, Visualization, Writing – review & editing.

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Declaration of competing interest

The authors have no conflicts of interest to declare. All co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report.

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Appendix A. Supplementary data

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