Autistic traits underlying social anxiety, obsessive-compulsive, and panic disorders

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Abstract

Objective: significant autistic traits (AT) may constitute the neurodevelopmental vulnerability array of multiple mental disorders. This view could explain the common comorbidities observed in autism spectrum disorder (ASD). The aim of the present study is to investigate the presence of AT in adults with social anxiety disorder (SAD), obsessive-compulsive disorder (OCD), and panic disorder (PD).

Methods: 40 subjects with ASD, 40 with SAD, 40 with OCD, 40 with PD and 50 HC were assessed with the Adult Autism Subthreshold Spectrum (AdAS Spectrum) questionnaire. Statistical analyses included Kruskal-Wallis test, Chi-square test, and multinomial logistic regression.

Results: A significant descending trend in the total AdAS score and in most domains was observed from the ASD to the SAD and OCD groups, followed by the PD group, reaching minimal scores in HC. SAD group scored higher in the Non-Verbal Communication domain, and OCD groups in the Inflexibility and adherence to routine domain. Elevated total AdAS Spectrum scores were predictive of ASD, SAD, OCD, and PD diagnoses compared to the HC group.

Discussion: results would support the hypothesis of a neurodevelopmental basis, identifiable in the autism spectrum, underlying SAD and OCD. The predictive role of autistic traits toward these disorders suggests the importance of investigating a potential hidden autism spectrum.

Conclusion: AT are distributed along a descending gradient from ASD to HC groups, passing through SAD and OCD. An elevated AdAS total score was predictive of a diagnosis of ASD, SAD, OCD, and eventually PD.

Keywords: autism spectrum disorder, autistic traits, social anxiety disorder, obsessive-compulsive disorder, panic disorder.

1. Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterized by persistent difficulties in social communication and interaction, as well as restrictive and repetitive patterns of behavior and interests [1]. Comorbidity in ASD represents a very frequent occurrence. Besides the usual association with intellectual developmental and language disorders, 70% of ASD patients have other mental disorders [2]. From a dimensional perspective, autistic traits are distributed along a continuum in the general population, with varying expressiveness and evidence based on their quantity and quality, as well as interactions with specific environmental factors [3]. In the universe of subclinical submerged manifestations, significant autistic traits could represent the neurodevelopmental matrix of vulnerability underlying multiple possible psychopathological conditions [4-6]. Indeed, beyond a mere concept of comorbidity, in recent decades, a growing body of literature has supported the hypothesis that underlying different mental disorders may be traced to a common basis of neurodevelopmental impairment. This perspective has led to the development of the “neurodevelopmental continuum” theory. Derived from the complex combination of genetic and environmental factors, an alteration in neurodevelopment - of varying levels according to a gradient of severity in which intellectual disability and ASD would correspond to the greatest degree of impairment - would stand at the origin of multiple psychopathological conditions [7]. A growing body of evidence, especially from preclinical studies, is shedding light on how neurodevelopmental pathology does not exclusively involve disorders included by the DSM within the neurodevelopmental disorders chapter, but also numerous other mental disorders such as schizophrenia, bipolar disorder, obsessive-compulsive disorder and anxiety disorders, as well as

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neurodegenerative conditions such as Parkinson's disease and Huntington's disease [8-10] (Tanaka et al., 2022; Poletti et al., 2023; Kloiber et al., 2020).

Anxiety disorders are among the most common comorbidities in children and adolescents with ASD [11]. In the most extensive study on this subject, it was found that 29% of 1507 ASD adults also had a diagnosed anxiety disorder [12]. Moreover, some genetic variants have been identified that affect both ASD and anxiety [13]. Family-based studies have also shown that there is a tendency for ASD to occur in the children of parents with anxiety disorders [14,15], and anxiety can be more common in family members of individuals with ASD or autistic traits [16,17].

Social Anxiety Disorder (SAD), also known as social phobia, is characterized by an excessive fear of experiencing embarrassment, humiliation, or rejection in situations where there is a possibility of being negatively evaluated by others during public performances or social interaction [18]. Recent research suggests that SAD is relatively common among youth with ASD who do not have cognitive impairments [11]. While there haven't been large-scale epidemiological studies exploring the coexistence of ASD and SAD, findings from community and population samples suggest that a significant proportion of youth with ASD, ranging from 10.7% [19] to 29.2% [20], experience impairing social anxiety. Indeed, the DSM-5 acknowledges social anxiety as a common feature of ASD, especially in children with high-functioning autism [2]. However, there's been limited research into the presence of SAD in adults with ASD. In a study by Joshi et al. [21], which compared clinically referred, age- and gender-matched adults with and without ASD, it was found that the ASD group had notably higher rates of lifetime (56%) and current (40%) SAD comorbidity compared to the non-ASD group (19% and 16%, respectively). This pattern has also been replicated in other studies involving adults [22]. Some studies have examined social anxiety symptoms continuously rather than diagnostically and have likewise discovered elevated levels of social anxiety symptoms in adults with ASD compared to those without ASD [23]. There's also evidence of a significant positive relationship between social anxiety symptoms and ASD symptoms in non-clinical college samples [24]. Research

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also indicates that co-occurring social anxiety adds further challenges beyond the core social deficits experienced by people with ASD. These challenges include increased loneliness [25] and tendencies towards aggression [26]. In a recent study, 50 % of a sample of adults with ASD not seeking treatment for anxiety disorders and without cognitive impairment, met the diagnostic criteria for SAD [27].

According to the DSM-5-TR [1] , obsessive-compulsive disorder is characterized by the presence of obsessions (repeated and involuntary thoughts, impulses, or images causing marked anxiety) and/or compulsions (repetitive mental or behavioral acts aimed at reducing anxiety). Individuals with ASD are two times more likely to be diagnosed with OCD compared to the general population [28]. Among children, studies suggest prevalence rates of 12.5% to 17.4%, [11], higher than those with anxiety disorders or no psychiatric diagnosis [29]. Parents often report higher obsessive-compulsive symptoms in children with ASD compared to children with other disorders [25]. Conversely, children and adolescents with OCD are at an increased risk (four time more likely then the general population) of being diagnosed with ASD [14], with 4% to 8% of them receiving an ASD diagnosis [30]. Family history also suggests a shared genetic basis between OCD and ASD. Diagnosing OCD in individuals with ASD can be challenging due to communication difficulties and reduced insight in those with ASD. Additionally, the presentation of compulsions in OCD can overlap with restricted and repetitive behaviors seen in ASD. However, key distinctions can be made [31]: in OCD, compulsions are ego-dystonic (disturbing and maladaptive), while in ASD, repetitive behaviors are ego-syntonic (pleasurable). OCD individuals often avoid thinking about their obsessions, whereas ASD individuals may not find their intrusive thoughts distressing. Studies show variations in the types and severity of obsessions and compulsions between ASD-OCD combined groups, OCD-only, and ASD-only groups [32-36]. Some findings suggest that ASD-OCD combined groups may have more severe repetitive behaviors and interests but less severe obsessions compared to OCD-only groups. However, there is variability in these findings, and more research is needed to fully understand the distinctions between

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these conditions in both children and adults [31].

The aim of the present study is to investigate the presence of autistic traits in adults with social anxiety disorder (SAD), obsessive-compulsive disorder (OCD), and panic disorder (PD). It also aims to evaluate which autism-specific symptom dimensions are most prevalent in the different disorders, and to explore the relationship between autistic traits and these psychopatological conditions.

2. Methods

2.1 Participants

This study, coordinated by the Department of Clinical and Experimental Medicine of Azienda Ospedaliera Universitaria Pisana (AOUP, Pisa, Italy), included a sample of 210 subjects divided into five groups, all assessed according to DSM-5 diagnostic criteria. Exclusion criteria were age below 18 years, language or intellectual disability that impaired the ability to complete assessments, mental disability, poor cooperation skills, and ongoing psychotic symptoms. More specifically, the five groups were identified as follows: 40 patients with a level 1 autism spectrum disorder (ASD), 40 patients with social anxiety disorder (SAD), 40 patients with obsessive-compulsive disorder (OCD), 40 patients with panic disorder (PD), and 50 healthy controls (HC) belonging to health care and paramedical personnel.

In addition, in the course of analysis, the total sample was divided into three groups based on the presence and severity of autistic traits, according to the total score obtained on the AdAS Spectrum questionnaire: 1) "Autism Spectrum Disorder, ASD" group containing subjects with total AdAS score greater than 70, delineating an overt autism spectrum disorder; 2) "Autistic Traits, AT" group

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containing subjects with total AdAS score greater than 43 and less than 70, indicative of the presence of significant subthreshold autistic traits; 3) "Non Autistic Traits, Non-AT" group containing subjects with total AdAS score less than 43, thus lacking significant autistic traits. For an explanation of the AdAS scores used as cut-offs, refer to the specific section on the questionnaire.

All subjects had to be between 18 and 70 years old in order to be recruited. The Structured Clinical Interview for DSM-5, Research Version (SCID-5-RV) [37] was used to confirm the diagnosis of ASD, SAD, OCD, and PD, as well as the absence of mental disorders among HC subjects. Furthermore, all subjects were assessed with the Adult Autism Subthreshold Spectrum (AdAS Spectrum) questionnaire. A written informed consent was subjected to all eligible participants after receiving an accurate description of the study, with the opportunity to ask questions. The study was led in accordance with the declaration of Helsinki, and approved by the Institutional Ethics Committee of Azienda Ospedaliero Universitaria Pisana.

2.2 Psychometric scales

2.2.1 Structured Clinical Interview for the Disorders of DSM-5 (SCID-5 RV)

The most comprehensive version of the SCID-5**,** a semi-structured clinical interview, is the most widely used instrument in clinical and research settings in order to formulate psychiatric diagnoses according to DSM-5 criteria [37].

2.2.2 AdAS Spectrum

The AdAS Spectrum is a questionnaire developed by Dell'Osso et al. [38], and devised to assess not only full-blown ASD, but also the broader spectrum of subthreshold autism, in subjects with normal intelligence and without language impairment across the lifetime. It allows evaluating a wide area of

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clinical and non-clinical traits, typical and atypical manifestations, including some gender-specific features. The instrument is composed by dichotomous questions, grouped in seven domains: Childhood/adolescence, Verbal communication, Non-Verbal communication, Empathy, Inflexibility and adherence to routine, Restricted interests and rumination, Hyper-hypo reactivity to sensory input. In the validation study [38] the AdAS Spectrum questionnaire demonstrated an excellent reliability and a strong convergent validity with other scales employed in this field, such as the Autism-Spectrum Quotient Test [39] and the Ritvo Autism and Asperger Diagnostic Scale 14-item version [40]. A score equal to or greater than 70 would indicate the presence of ASD (Autism Spectrum Disorder), while a score between 69 and 43 would indicate significant autistic traits (AT) or subthreshold autism spectrum [41].

2.3 Statistical analysis

All statistical analyses were conducted using the Statistical Package for Social Science, version 25.0 (SPSS Inc.). We used an independent-samples Kruskal-Wallis test in order to compare the AdAS total and domain scores obtained each diagnostic group. A p value <.05 was considered statistically significant. In the pairwise comparisons of AdAS scores among diagnostic groups, significance values have been adjusted by the Bonferroni correction for multiple tests. Chi-square tests was used in order to compare sex composition among groups. Furthermore, in order to identify the best predictors of the presence of a probable ASD, SAD and OCD diagnosis, a multinomial logistic regression analysis was performed.

3. Results

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Sample composition

The sample included 99 (47.1%) males and 111 females (52.9%) at baseline. The mean age was 40.31±11.961 years. The five diagnostic groups did not significantly differ from each other in terms of age (p = 0,72; F = 0.526) and sex (p = 0.83, X^2 = 1.499). The ASD group included 40 subjects (19.0% of the total sample) with a mean age of 38.00 years (±12,02) and consisted of 20 (50.0%) males and 20 (50.0%) females. The SAD group included 40 subjects (19.0 % of the total sample) with a mean age of 41.00 (±12.69) years and consisted of 21 (52.5%) males and 19 (47.5%) females. The OCD group included 40 subjects (19.0 % of the total sample) with a mean age of 40.95 (±10.81) years and consisted of 18 (45.0%) males and 22 (55.0%) females. The PD group included 40 subjects (19.0 % of the total sample) with a mean age of 41.48 (±11.33) years and consisted of 16 (40.0%) males and 24 (60.0%) females. The HC group included 50 subjects (23.8 % of the total sample) with a mean age of 40.18 (±12.86) years and consisted of 24 (48.0%) males and 26 (52.0%) females. **(Table 1)**

Comparisons between AdAS total and domain scores among the five diagnostic categories

Regarding the AdAS total score, the highest scores were observed in the ASD group and the lowest scores in the HC group. The score obtained by the ASD subjects was significantly higher than the score obtained by the SAD and OCD groups (p < .01), both of which also had significantly higher scores than the PD group (p < .01), in whom the total AdAS score was significantly lower than in the HC group (p < .01). A statistically significant difference was not observed between the scores obtained by the SAD and the OCD groups.

Concerning the *Childhood/Adolescence* domain, the highest score was obtained by the ASD group, significantly higher then all other groups (p < .01). SAD group scores did not significantly differ from

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the OCD subjects, while both groups had significantly higher scores ($p < .01$) than those in the PD group, which, in turn, had significantly higher scores ($p < .01$) than the healthy controls.

In the *Verbal Communication* domain, the highest scores were obtained in the ASD group, significantly higher ($p < .01$) than in the SAD and OCD groups. In the latter two groups, which were not significantly distinguishable from each other, significantly higher scores ($p < .01$) were observed than in the PD group, which, in turn, had significantly higher scores ($p < .01$) than the HC group.

In the *Non-Verbal Communication* domain, the highest score was obtained by the ASD group, significantly higher than all other diagnostic groups ($p < .01$), except for the SAD group, which did not differ significantly. In the latter group, scores were significantly higher ($p < .01$) than the OCD patients, who did not significantly differ from the PD subjects. As in the other domains, healthy controls reported significantly lower scores than all other groups.

In the *Empathy* domain, a significant decreasing gradient ($p < .01$) was observed from the ASD group to the SAD patients, then to the OCD and PD groups, and finally to the healthy controls. There was no significant difference found between the scores of the PD and OCD groups.

In the *Inflexibility and Adherence to routine* domain, the highest scores were obtained by the ASD and OCD groups, not significantly distinguishable from each other, but significantly higher ($p < .01$) than the PD and SAD groups, in which was not observed a significant difference. As in the other domains, healthy controls reported significantly lower scores than all groups.

In the *Restricted Interests and rumination* domain, the ASD group reported significantly higher scores ($p < .01$) than SAD and OCD groups, not significantly distinguishable from each other. Scores obtained by the OCD groups were significantly higher ($p < .01$) than those of the PD groups, while SAD group did non significantly differ from the PD group. As in the other domains, healthy controls reported significantly higher scores than all groups.

In the *Hyper-Hypo reactivity to sensory Input* domain, a decreasing gradient ($p < .01$) was observed from the ASD group to the SAD patients, then to the PD and OCD groups, and finally to the healthy

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controls. There was no significant difference between the scores of SAD and PD groups, PD and OCD groups, OCD and HC groups.

All comparisons between AdAS total and domain scores among the five diagnostic groups described above, are reported in **Table 2 and Figure 1.**

Figure 2 report a graphical representation of the AdAS total and domain mean scores, normalized with values ranging from 0 to 100, obtained in each diagnostic group

Distribution of autistic traits within diagnostic categories

Based on the presence of autistic symptoms, determined from the total AdAS Spectrum score, the total sample was divided into three subgroups:

1. participants with ASD (ASD group) composed by 54 subjects (22.0%),
2. participants with subtreshold autistic traits (AT group) composed by 72 subjects (29.4%),
3. participants without autistic traits (non-AT group) composed of 119 subjects (48.6%) **(Figure 3)**.

The relationships among the ASD, AT and non-AT groups and the five diagnostic groups were tested using the Chi-square test.

Table 3 shows the size of each AdAS group for each diagnostic category, the percentage of subjects belonging to each diagnosis within the AdAS group (% within AdAS group), and the percentage of subjects belonging to each AdAS group within the diagnostic group (% within diagnosis).

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The ASD, AT and non-AT groups were found to be distributed across all diagnostic groups, and in particular **(Figure 4)**:

1. The ASD group was distributed as follows: 75.0% in the ASD group and 25.0% in the AT group.
2. The SAD group was distributed as follows: 55.0% in the AT group, 32.5% in the non-AT group, and 12.5% in the ASD group.
3. The OCD group was distributed as follows: 55.0% in the AT group, 27.5% in the non-AT group, and 17.5% in the ASD group.
4. The PD group was distributed as follows: 77.5% in the non-AT group, 22.5% in the AT group, and 0.0% in the ASD group.
5. The HC group was distributed 100% in the non-AT group.

So, considering the composition of each of the three AdAS groups, it was possible to observe that **(Figure 5)**:

1. The ASD group consisted of 71.4% of subjects with ASD, 11.9% with SAD, 16.7% with OCD, and no subjects from the PD and HC groups. There was no significant difference between SAD and OCD groups, SAD and PD groups and PD and HC groups.
2. The AT group consisted of 55.0% with SAD, 55.0% with OCD, 25.0% with ASD, and 22.5% with PD. There were no healthy controls in this group as well. There was no significant difference between SAD, OCD and ASD groups and between ASD and PD groups.
3. The non-AT group consisted of 47.6% healthy controls, 29.5% with PD, 12.4% with SAD, 10.5% with OCD, and no subjects from the ASD group. There were no healthy controls in this group as well. There was no significant difference between SAD and OCD group.

Furthermore, a multinomial regression analysis was performed using the diagnostic groups as the dependent variables and the total AdAS score as the independent variable. Among the diagnostic

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groups, HC was considered as the reference category. The analysis showed, in agreement with the previous results, that high total AdAS Spectrum score are predictive of a diagnosis of of ASD (OR = 1.551, p<.01), SAD (OR = 1.409, p<.01), OCD (OR = 1.408, p<.01) and PD (OR = 1.301, p<.01) with respect to the HC group. **(Table 4)**.

4. Discussion

The present study has yielded intriguing results that suggest the presence of an underlying common thread of autistic traits across different diagnostic categories, along a continuum that underscores the interrelatedness of various conditions, particularly Social Anxiety Disorder and Obsessive-Compulsive Disorder, within the broader framework of ASD.

First of all, analyzing AdAS total score, we observed a common trend in the distribution of autistic traits within the total sample, ranging from the highest expression in patients with ASD, as expected, to minimal scores in healthy control subjects. Between these two extremes, we find the diagnoses of SAD and OCD, just below the ASD group and above individuals with PD, who, in turn, score higher than healthy controls. This finding, on the one hand, lends support to previous studies suggesting that high rates of comorbidity between ASD and other diagnoses imply that current diagnostic categories share more similarities than differences [42, 43], aligning with the substantial body of evidence regarding the potential role of the autistic spectrum in the development of other mental disorders [4,44,5]. Furthermore, considering that the diagnoses of SAD and OCD, as observed in this study, predominantly fall within the group characterized by subtreshold significant autistic traits, it is essential to emphasize the importance of the already mentioned autistic spectrum model [44], a transnosographic concept that encourages a reevaluation of all mental disorders from a broader perspective, potentially guiding us towards a neuro-psychogenetic approach that transcends the existing categorical and descriptive framework in contemporary psychopathology. Regarding the

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finding of high autistic traits within the SAD group, we already know that a large proportion of individuals with ASD also fulfill the criteria for SAD [24,20] and that elevated autistic traits are prevalent in individuals diagnosed with SAD [46]. It is increasingly acknowledged that both SAD and ASD can be conceived as the extreme ends of continuous phenotypes, meaning that there are no clear-cut borderlines between having no impairments, elevated sub-clinical traits, and a full-blown diagnosis, and even among the latter group, there are relevant individual differences in symptom strength [3,47]. Beyond a mere issue of comorbidity, especially considering that the diagnoses within the present sample were mutually exclusive, it's important to highlight that a growing body of research supports the notion of social anxiety disorder as a chronic neurodevelopmental condition [48,49]. Indeed, as mentioned in the introduction, studies involving animal models and neurobiological investigations in humans have identified specific brain circuits associated with early-life anxiety, including the amygdala, hippocampus, and orbitofrontal cortex [48]. Individual differences in SAD symptoms in subjects with ASD may be related to atypical functioning and structure of the amygdala, a subcortical area linked to fear processing, association learning and social cognition, particularly orienting to human faces [50]. In this regard, South et al. [51] found a positive correlation between autonomic fear conditioning and social anxiety in a group with ASD, but a negative correlation with autistic traits. Taken together, this suggests that a dimensional approach is important for understanding the biological mechanisms underlying ASD. The analysis of specific AdAS domains has allowed us to identify a particular profile of autistic expression in the SAD group, where the most intense autistic traits would be situated in the domain *Non-Verbal Communication*. This finding is in line with previous studies, according to which the symptomatic overlap between the conditions is mainly found in areas of social interaction and social skills, whereas restricted and repetitive behaviors and atypical social cognition may be unique to ASD [24, 52]. The observation of high scores in the *Non-Verbal Communication* domain, aligns with previous researches affirming that both SAD and the ASD have been linked to a atypicalities in social attention. In SAD, avoidance

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of social stimuli such as faces with direct gaze may conduce to reduced chances to reappraise or habituate to the perceived threat. Avoidance is therefore believed to be a maintaining factor in the SAD symptomatology [53]. In ASD, studies have shown atypical social attention to be a precursor of clinical symptoms [23,54]. Nonverbal social signal processing (NVSC), an essential skill for interpersonal functioning and particularly relevant to patterns of social anxiety [28]. The main nonverbal signal examined in the context of social anxiety in numerous studies has been facial emotional expression [55], a feature essential for social development and functioning and emotional regulation [56], as well as associated with the functioning of certain altered brain areas in SAD [57]. These studies suggest that individuals with high social anxiety exhibit increased processing of all facial expressions in some tasks, difficulty disengaging threatening and smiling expressions (as opposed to neutral expressions) in other tasks, and increased vigilance or reactivity to angry expressions in selected [28]. Regarding the finding of high autistic traits within the OCD group, despite substantial evidence on premorbid (i.e., neurodevelopmental antecedents) and prodromal features preceding the clinical onset [58], the pathophysiology of OCD in youth is poorly investigated as compared to the great attention to the cross-sectional study of OCD functioning. A recent review proposed a developmental pathogenic cascade from early childhood alterations of corollary discharge signals, phenotypically expressed in childhood through motor coordination disturbances, to distal psychotic symptoms in adolescence or young adulthood, through intermediate phenomena represented by an altered sense of agency and anomalous subjective experiences of the basic self [9]. Regarding neurodevelopmental antecedents, different studies reported that subjects with OCD present an increased prevalence of prenatal and perinatal hazards [59-62], indicating early environmental adversities that may produce alterations in neurodevelopment. Furthermore, neuroimaging showed signs of an altered neurodevelopment or alterations in pediatric OCD, hypogyrification in adult OCD patients, functional and structural alterations in children and adolescents OCD patients [63-65]. Moreover, another study reported the involvement genetic

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variants related to glutamatergic, dopaminergic, and neurodevelopmental pathways in determining the white matter microstructure of child and adolescent with OCD [66]. Furthermore, in line with our results, previous studies described a sensory over-responsivity in OCD patients [67-69], for example, in terms of enhanced startle reaction or impaired sensory gating, and in multiple sensory domains, such as tactile and acoustic. The analysis of specific AdAS domains has allowed us to identify a particular profile of autistic expression in the OCD group, where the most intense autistic traits would be situated in the domains of *Inflexibility and adherence to routine* and *Restricted Interests and rumination*. In this regard, previous studies showed that OCD was more common in parents of those children with autism that scored high on repetitive behaviour and stereotypies [70]. This connection is also supported by a genetic study linking treatment resistant OCD with Asperger syndrome and autism [71]. Furthermore, hoarding is commonly reported in ASD [72] and frequently seen in clinical practice in adults with ASD. Whereas in a Japanese study only 6 percent of young adults with autism were hoarders according to their caregivers (Kobayashi and Murata, 1998), in another study [29] 10 out of 40 individuals with ASD and normal intelligence had OCD and 12 were hoarders. In the autism literature, it is generally supposed that rituals in Asperger syndrome are ego-syntonic [73] and hence differ in nature from those of bona fide OCD. According to Wing [73] rituals have a positive function for people with autism in many cases, without distressing them. Although this may often be true, attitudes towards rituals vary considerably among persons with ASD [74, 75, 29]. It was also supposed that Asperger syndrome and High-Functioning Autism (HFA) are frequently obscured by an ego-dystonic OCD, and that symptoms of Asperger and HFA in these cases often are somewhat inappropriately referred to as comorbid personality disorders, such as paranoid, schizoid, schizotypal, avoidant or obsessive-compulsive, whose diagnosis may serve as a sensitive indicator, identifying an autistic dimension in OCD patients [76]. According to this view, OCD patients supposedly constitute a continuum, ranging from “almost normal personality” to severely autistic personality [76], proposing a subtype of OCD with autistic traits characterized by high levels of symmetry ordering,

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hoarding, aloof, been bullied, avoidant, obsessional and cluster A personality features, and soft neurological signs [74,76].

According to our results, the group of subjects with panic disorder was characterized by very low scores overall, almost always lower than SAD and OCD, except in the domains where these diagnoses reach the lowest levels (*Inflexibility and adherence to routine* and *Restricted Interests and rumination* for patients with SAD and *Non-Verbal Communication* and *Empathy* for the OCD group) and in the *Hypo/Hyper-reactivity* domain where they overlap with both SAD and OCD. This is interesting considering that sensory hyper-reactivity has been linked to intolerance of uncertainty and anxiety in autistic children [77-79]. Furthermore, when controlling for autism traits, a recent study found sensory hyper-reactivity to be significantly related to separation anxiety [80], a really interesting result considering that a childhood diagnosis of separation anxiety disorder significantly increases the risk of adult-onset panic disorder [81].

Taken together, these findings support the concept of a neurodevelopmental continuum where the boundaries between different disorders become less distinct. Instead, a spectrum of traits emerges, with ASD serving as the overarching framework.

Some limitations should be taken into account: the relatively small sample size, the self-report nature of the questionnaires, which may have led to either overestimation or underestimation of the symptoms by the patients, and lastly, the cross-sectional design of the study, which restricts the exploration of temporal and causal relationships among the variables under investigation.

5. **Conclusion**

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In conclusion, the results of this study illuminate several important insights into the interconnectedness of various mental health conditions and the potential underlying role of autistic traits. These findings point towards a neurodevelopmental continuum that challenges traditional diagnostic boundaries.

Firstly, the shared autistic dimension that underlies both Social Anxiety Disorder (SAD) and Obsessive-Compulsive Disorder (OCD) suggests a common neurodevelopmental origin for these conditions. This not only aligns with existing literature but also emphasizes the need for further research in this direction. It opens the door to exploring these disorders from a different perspective, one that considers the broader framework of ASD.

Secondly, the distinct profile of autistic symptomatology observed in individuals with SAD and OCD provides a valuable perspective for clinicians. Understanding the specific traits associated with these disorders can guide more personalized and effective diagnostic and treatment approaches. Recognizing these unique profiles is essential for providing comprehensive care to individuals with these conditions.

In summary, this study contributes to the growing body of evidence that supports a dimensional and interconnected view of mental disorders. It emphasizes the need for a more comprehensive and nuanced approach to diagnosis and treatment, one that considers the common thread of autistic traits that may run through various conditions. These findings offer new avenues for research and have the potential to lead to more effective strategies for understanding and managing these complex disorders.

To gain a more comprehensive understanding of the connection between autistic traits and SAD, OCD, and PD, it is advisable to conduct further longitudinal studies involving a larger sample and employing hetero-administered instruments.

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Table 1. Frequency and demographic characteristics of diagnostic group

Diagnostic Category	Frequency (percent)	Mean age (SD)*	Sex (%)
ASD	40 (19.0%)	38.00 (12.02)	• Female = 20 (50.0%) • Male= 20 (50.0%)
SAD	40 (19.0%)	41.00 (12.69)	• Female = 19 (47.5%) • Male= 21 (52.2%)
OCD	40 (19.0%)	40.95 (10.81)	• Female = 22 (55.0%) • Male= 18 (45.0%)
PD	40 (19.0%)	41.48 (11.33)	• Female = 24 (60.0%) • Male= 16 (40.0%)
HC	50 (23.8%)	40.18 (12.86)	• Female = 26 (52.0%) • Male= 24 (48.0%)
p	-	0.72 (F = 0.526)	0.83 (X^2 = 1.499)

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Table 2. Comparisons between AdAS total and domain scores among the five diagnostic groups

AdAS domains	ASD mean±sd (Mean rank)	SAD mean±sd (Mean rank)	OCD mean±sd (Mean rank)	PD mean±sd (Mean rank)	HC mean±sd (Mean rank)	p	Post-Hoc
Childhood/adolescence	12.65±4.21 (169.15)	9.32± 4.71 (164.79)	8.55± 5.59 (125.11)	4.22 ±3.21 (80.74)	0.74 ±1.19 (35.27)	<.01	ASD>SAD, OCD,PD,HC; SAD,OCD>PD,HC; PD>HC
Verbal Communication	10.27±3.34 (169.80)	6.70± 3.87 (126.61)	6.92± 4.68 (127.40)	3.80± 3.50 (86.83)	0.50± 0.61 (34.75)	<.01	ASD>SAD,OCD,PD,HC; SAD,OCD>PD,HC; PD>HC
Non-Verbal Communication	13.70±5.48 (163.29)	10.75 ±6.05 (136.09)	8.10 ±6.12 (114.10)	5.97 ±5.49 (94.30)	1.20± 1.30 (36.88)	<.01	ASD>OCD,PD,HC; SAD>PD,HC; OCD,PD>HC
Empathy	7.47± 2.45 (175.30)	4.70± 2.87 (137.33)	3.05± 2.87 (105.34)	1.75± 2.17 (85.86)	0.18 ±0.48 (40.04)	<.01	ASD>SAD,OCD,PD,HC; SAD>OCD,PD,HC; OCD,PD>HC
Inflexibility and adherence to routine	19.60 ±10.09 (155.73)	11.30 ±5.72 (113.96)	16.90 ±8.31 (146.91)	8.40± 5.02 (93.58)	1.60± 1.69 (34.96)	<.01	ASD,OCD>SAD,PD,HC ; SAD,PD>HC
Restricted interests and rumination	11.40±5.03 (165.68)	5.95± 3.57 (114.35)	7.37± 4.19 (128.60)	4.50± 3.90 (92.79)	1.00± 1.45 (41.97)	<.01	ASD>SAD,OCD,PD,HC ; SAD,PD>HC; OCD>PD,HC

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Table 2. Comparisons between AdAS total and domain scores among the five diagnostic groups

Hyper-hypo reactivity to sensory input	7.87± 4.89 (170.23)	3.92± 4.45 (122.63)	1.65± 2.11 (85.63)	2.60± 3.90 (112.28)	1.00± 1.96 (50.50)	<.01	ASD>SAD, PCD, PD, HC; SAD>OCD,HC
Total score	82.97±17.86 (182.19)	52.65±17.32 (126.74)	52.55±19.27 (127.25)	31.25±16.20 (83.05)	5.58± 4.58 (27.72)	<.01	ASD>SAD, PCD, PD, HC SAD,OCD>PD,HC; OCD>PD,HC

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Table 3. Composition of the three autistic severity groups ASD, AT and Non-AT

AdAS groups	ASD (n, %)	SAD (n, %)	OCD (n, %)	PD (n, %)	HC (n, %)
ASD					
n*	30**c**	5**a,b**	7**a**	0**a,b**	0**b**
% within diagnosis	75.0%	12.5%	17.5%	0.0%	0.0%
% within AdAS group	71.4%	11.9%	16.7%	0.0%	0.0%
AT					
n*	10**a,b**	22**a**	22**a**	9**b**	0**c**
% within diagnosis	25.0%	55.0%	55.0%	22.5%	0.0%
% within AdAS group	15.9%	34.9%	34.9%	14.3%	0.0%
Non-AT					
n*	0**c**	13**a**	11**a**	31**b**	50**d**
% within diagnosis	0.0%	32.5%	27.5%	77.5%	100%
% within AdAS group	0.0%	12.4%	10.5%	29.5%	47.6%

* For all variables with a different letter, the difference is statistically significant (Pearson Chi-Square = 170.050; p < .01)

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Table 4. Multinomial logistic regression using diagnostic groups as dependent variables (HC as reference category) and AdAS total score as independent variable

Diagnosis	B	OR	p	CI (95%)
ASD	0.439	1.551	<.01	1.373; 1.752
SAD	0.343	1.409	<.01	1.253; 1.584
OCD	0.342	1.408	<.01	1.253; 1.583
PD	0.263	1.301	<.01	1.164; 1.455

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Figure 1. Graphical representation of comparisons between AdAS total and domain scores (percentile means) among diagnostic groups

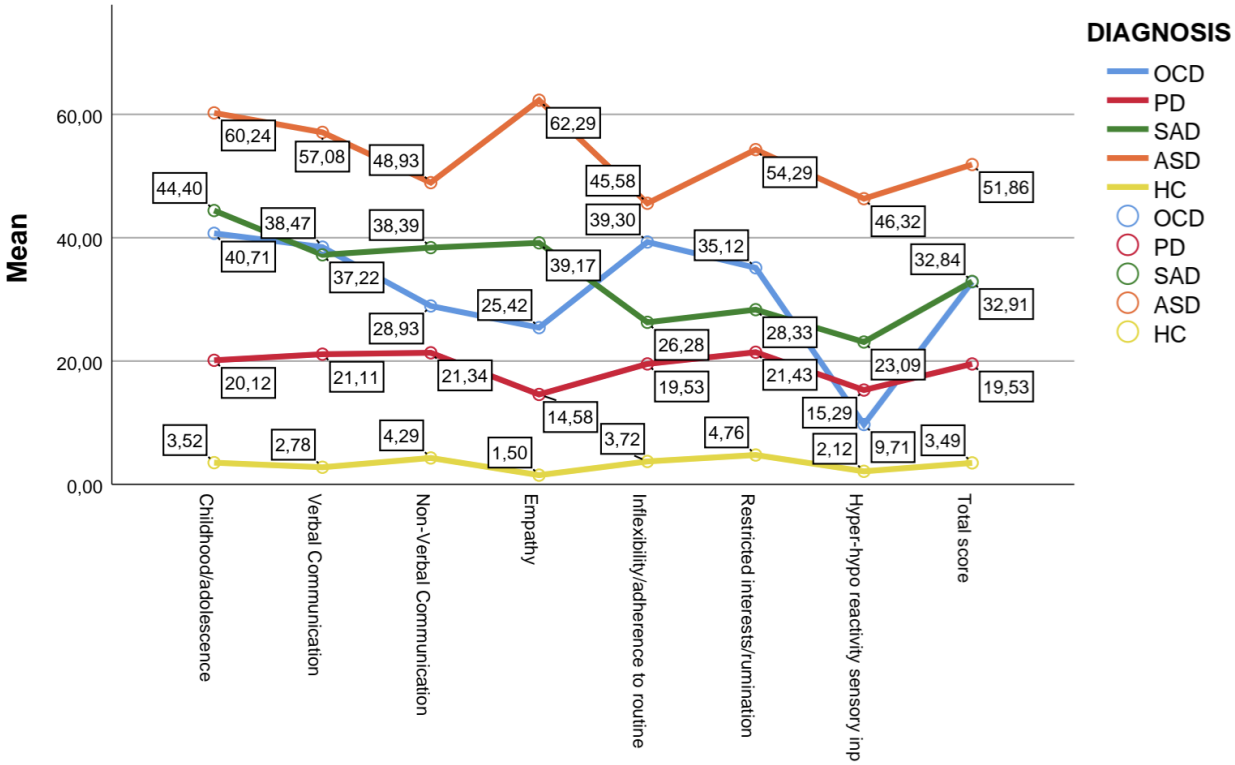

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Figure 2. Graphical representation of AdAS total and domain scores (percentile means) obtained in each diagnostic group.

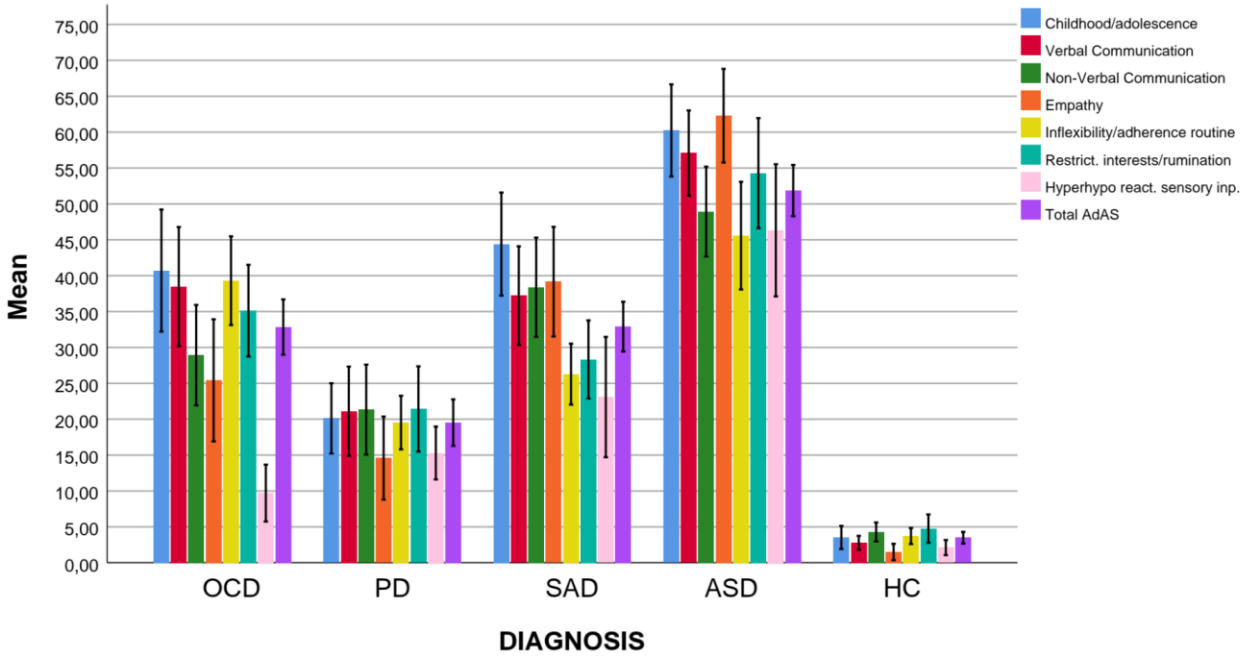

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Figure 3. Graphical representation of AdAS total and domain scores (percentile means) among the three AdAS groups

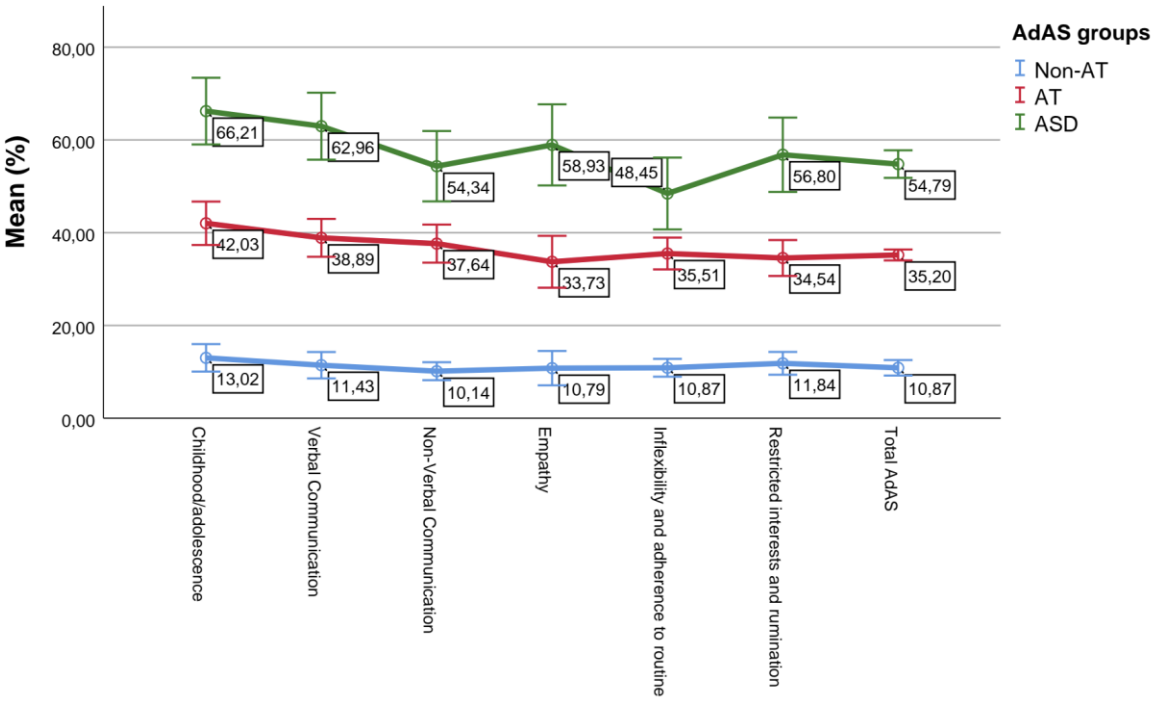

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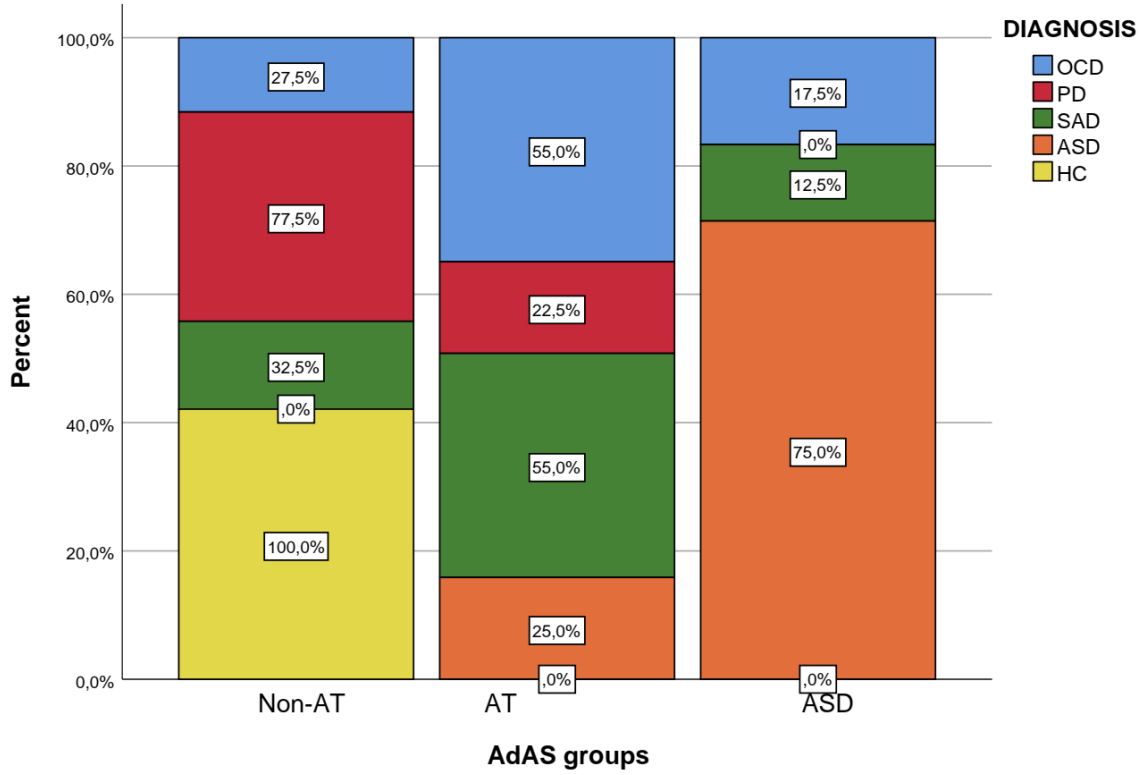
Figure 4. Graphical representation of the diagnostic composition, expressed in



percentage, of each AdAS group.

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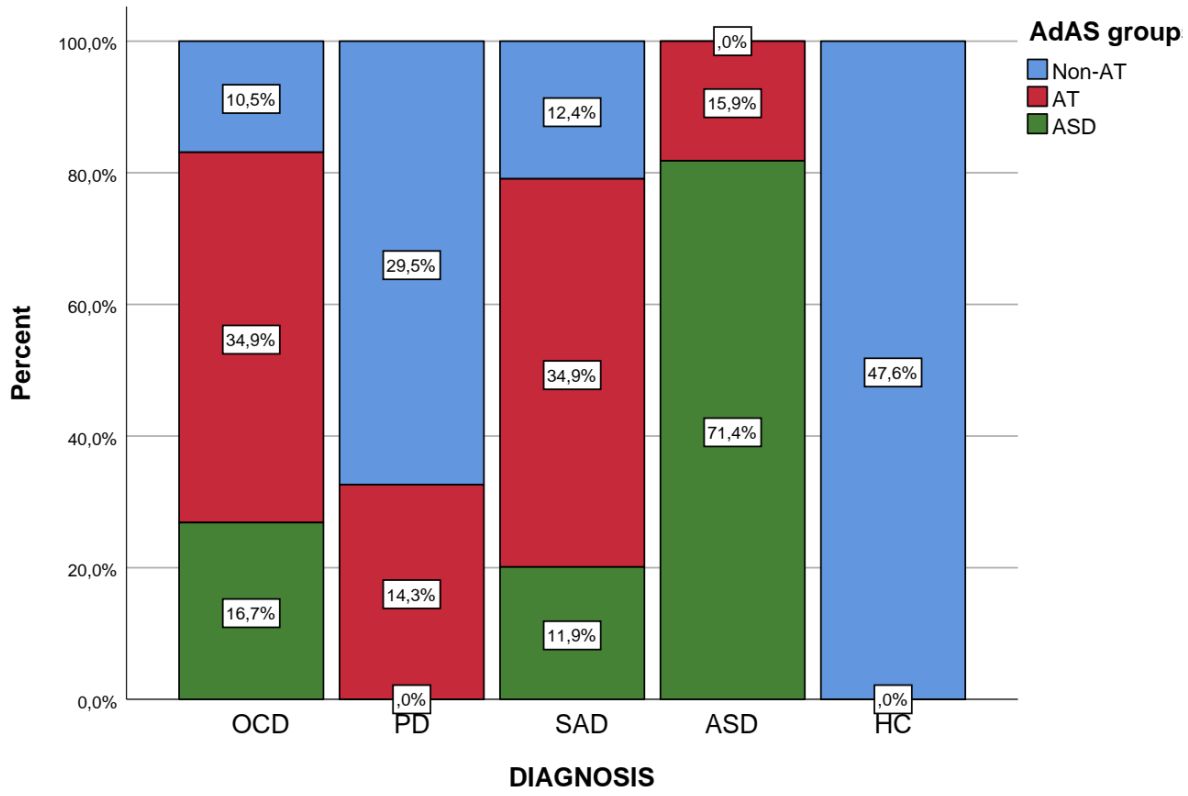
Figure 5. Graphic representation of the percentages of individuals belonging to each AdAS group within the five diagnostic categories.



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