

Asperger Syndrome, Alexithymia and Perception of Sleep

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Key Words

Asperger syndrome · Alexithymia · Sleep

Abstract

Two research traditions, the one on Asperger syndrome (AS) and the other on alexithymia, have produced similar findings independently of each other indicating a possible association between these two phenomena. Both conditions are also associated with impaired initiation and continuity of sleep. Twenty AS adults were compared with 10 healthy controls using the Toronto Alexithymia Scale and the Basic Nordic Sleep Questionnaire. AS subjects were significantly more alexithymic and reported lower sleep quality as compared with controls. AS and alexithymia are associated although the mediating factors are unknown. It is possible that alexithymic traits predispose to anxiety, which in turn lowers the sleep quality in AS adults. Alternatively, low sleep quality might be due to AS itself.

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Introduction

In 1944, an Austrian pediatrician, Hans Asperger, described a group of boys who, in spite of normal intelligence, displayed a seriously disturbed reciprocal social communication, circumscribed special interests and excessive adherence to routines, a condition which he named as autistic psychopathy in childhood [1]. Autism, Asperger syndrome (AS) and pervasive developmental disorder not otherwise specified are commonly referred to as 'autistic spectrum disorders' (ASD) [2]. As AS has become a separate entity quite recently in DSM-IV (1994), there is scarcity of reports on AS and one must rely on studies with autism to further psychophysiological hypotheses [3].

Sifneos [4] coined the term alexithymia already 30 years ago to mean a multifaceted personality construct with difficulty in identifying and describing feelings, difficulty in distinguishing feelings from bodily sensations of emotional arousal, a constricted fantasy life and an externally oriented cognitive style [5]. The etiology of alexithymia is still unclear.

In studies about AS and alexithymia, some converging features are found. The connections between limbic and prefrontal cortex have been reported to play a role in

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alexithymia [6] and in AS [7]. The other target area is the right hemisphere of the brain, the function of which is proposed to be impaired both in alexithymia [8] and in AS [9].

Using Cloninger's model of personality structure and the questionnaire measuring the temperament dimensions, the Temperament and Character Inventory, it has been shown that low self-directedness, low reward dependence, and to some extent harm avoidance are predictors of alexithymia [10]. On the other hand, adults with AS have scored high on harm avoidance and lower on self-directedness [11]. Furthermore, an association between poor social skills and alexithymia has been reported in young adults [12]. The core feature of AS, qualitative impairment in social interaction [13], leads to diminished social skills as well [14].

There is some evidence that alexithymia is associated with poor sleep quality, especially impaired initiation of sleep [15, 16], and with conditions known to impair sleep quality, e.g. depression, posttraumatic stress disorders and chronic pain [17]. The restricted imaginative process typical of alexithymic persons is illustrated by limited dream recall [18].

More evidence has accumulated concerning children with ASD who display profound difficulties in initiation and continuity of sleep [19]. Problems occur at a high frequency and they are more severe than those found in normally developing children and in children with other developmental disabilities.

There is a paucity of studies concerning sleep of adults with ASD. AS subjects often have an impaired function of the prefrontal cortex [20], which is the part of the brain most vulnerable to sleep deprivation [21]. Therefore, it is possible that these individuals are especially sensitive to deleterious effects of low sleep quality on daytime performance.

We did not find studies on the relationship of alexithymia and AS. We hypothesized that AS subjects are more alexithymic than persons without AS. The other aim was to characterize the subjective experience of sleep of AS adults with respect to alexithymia.

Methods

The diagnosis of AS was a careful lifetime best-estimate evaluation using multiple sources of information as follows: AS subjects were recruited from the Helsinki Asperger Center, a unit to which patients with a tentative diagnosis of AS are referred from all parts of Finland. Prior to inclusion in the present study, participants were interviewed in detail by a clinician familiar with ASD focusing on life

history elements essential for the diagnosis of AS. The participants' mother or other significant relative or family member(s) were interviewed on developmental issues relevant for the diagnosis. All available previous medical records were reviewed including data from well baby clinics and school health especially concerning developmental milestones.

The Autism Spectrum Screening Questionnaire (ASSQ) is a continuous measure rating scale validated for screening children with ASD [22]. In the present study, the mothers or other persons in close contact with the subject in childhood filled in the ASSQ concerning the subject's childhood behavior. Each positive score had to be documented with several examples. The ASSQ was used mainly to exclude the possibility of autistic symptoms in control subjects. In 1 AS subject, there was no competent relative to be interviewed and he filled in the ASSQ by himself.

The Wechsler Adult Intelligence Scale-Revised [23] was assessed by a psychologist familiar with ASD in all participants except in 1 control and in 1 patient (both having a university degree) who refused.

All these data were critically reviewed in corpore by 3 authors (P.T., T.N. and L.v.W.). All 20 participants fulfilled the ICD-10 and DSM-IV criteria of AS in childhood. In adulthood, 2 subjects did not meet the full diagnostic criteria of AS anymore, but because their problems were essentially similar to those of the participants with the full diagnosis, they were included in the study group. Exclusion criteria of the DSM-IV were followed.

Twenty AS subjects were matched for age, gender, intelligence, and body mass index (BMI) with 10 controls (table 1). The control group consisted of 10 volunteers from the hospital staff without anamnesis of neuropsychiatric disorders, with no psychiatric diagnoses and no subjective complaints about sleep quality.

Participants were medication free (with a minimum interval of 2 weeks for hypnotics, 3 months for antidepressant medicines and 1 year for neuroleptics) except for one 44-year-old female who had been using thyroxine substitution since childhood.

Possible confounding intercurrent somatic disease was ruled out by means of clinical examination and by a blood test screening for electrolytes, liver and kidney functions, thyroid hormones, and calcium metabolism, which all gave normal results. Brain MRI (1.5 T) was obtained from 17 AS subjects and from 9 controls with no signs of focal abnormality in visual inspection by a neuroradiologist.

The 20-item Toronto Alexithymia Scale (TAS-20) was used as the measure of alexithymia, since of the different methods for measuring alexithymia, the TAS-20 is the most widely used and presumably the most carefully validated one. Its internal consistency, test-retest reliability, and convergent, discriminant, and concurrent validity have been demonstrated to be good [24]. The psychometric properties of the Finnish version of the TAS-20 have been shown to be satisfactory [25]. The items are rated on a 5-point scale ranging from 'strongly disagree' to 'strongly agree'. The TAS-20 consists of 3 subscales or 'factors' (TAS factors 1–3), which reflect the 3 main facets of the alexithymia concept: TAS factor 1 assesses difficulties in identifying feelings (e.g. 'I have feelings that I can't quite identify'), TAS factor 2 concerns the difficulty in describing feelings (e.g. 'It is difficult for me to find the right words for my feelings') and TAS factor 3 reflects concrete externally oriented thinking or a preoccupation with the details of external events (e.g. 'I prefer talking to people about their daily activities rather than their feelings'). The TAS-20 total score as well as the TAS factors correlate negatively with different measures of psychological mindedness and awareness of own affects [5, 17].

According to the recommendation by the developers of the scale, the following cutoff point of alexithymia was used: TAS-20 total scores >60 are defined as alexithymic cases [5]. The participants were only afterwards informed about the purpose of the TAS-20 (Finnish version) in order to avoid leading statements.

The Structured Clinical Interview of Diseases (SCID) for DSM-III-R [26] was performed for comorbid axis I diagnoses. Because AS subjects are prone to affective disorders [27, 28], their mood was further explored with the Beck Depression Inventory (BDI) [29] to quantitate current depressive symptoms potentially affecting results. None of the subjects in the AS group had schizophrenia or another chronic psychotic illness according to previous medical records or the SCID.

The Basic Nordic Sleep Questionnaire (BNSQ) assesses the frequency of various subjective sleep complaints during the past 3 months [30]. It is a standardized questionnaire developed by the Scandinavian Sleep Research Society containing altogether 21 items and 14 of them with a 5-point quantitative scale (score range 14–70), the higher score indicating lower sleep quality. The remaining items concern subjective evaluation of various sleep parameters, e.g. time in bed before falling asleep.

Statistics

Comparison between AS subjects and controls was made with Student's *t* test and in cases when the parameters had a nonnormal distribution, the Mann-Whitney rank sum test was applied. Analyses were performed with commercial computer software (SigmaStat for Windows, version 2.03, SPSS Inc.) and the general linear model [31] procedure with SAS software, version 8, was applied to adjust TAS scores in respect of the BDI score. Fisher's exact test was used in calculating dichotomized differences between AS subjects and controls.

Ethics

Informed consent was obtained from the patients and controls and the study was accepted by the ethical committee of the Helsinki University Hospital. The principles of the declaration of Helsinki were adequately followed.

Results

AS subjects did not differ from controls in respect of age, gender, BMI and intelligence (table 1). They had a significantly higher ASSQ score reflecting anamnesis of ASD and an elevated BDI score as compared with controls (table 1).

AS subjects were more alexithymic than controls (table 2). The difference was significant in case of the TAS-20 total score, and TAS factors 1 and 2. The AS subjects also had a higher score in case of TAS factor 3, but the difference did not reach statistical significance. When the TAS score was dichotomized, it was found that 40% of the AS subjects (43% of men and 33% of women) and none of the controls were alexithymic ($p = 0.03$). Because the mean BDI score differed between AS subjects and con-

Table 1. Clinical variables of 20 AS subjects and 10 controls

	AS subjects	Controls	p
Females	6/20 (30%)	3/10 (30%)	
Education college or more	16/20 (80%)	8/10 (80%)	
Age	27.2 ± 7.3	26.5 ± 8.1	n.s. ^a
BMI	22.6 ± 3.7	23.4 ± 3.1	n.s. ^a
IQ total	111.6 ± 11.9	111.2 ± 10.4	n.s. ^a
IQ verbal	110.5 ± 12.7	111.4 ± 11.4	n.s. ^a
IQ performance	111.0 ± 11.8	109.4 ± 9.3	n.s. ^a
BDI	8.50 ± 6.3	0.60 ± 1.1	<0.001 ^b
ASSQ	23.95 ± 12.9	1.30 ± 2.2	<0.001 ^b

Values are given as means ± SD.

^a Student's *t* test.

^b Mann-Whitney rank sum test.

trols, a multivariate analysis (general linear model) was created to control for the effects of the BDI score on inter-group differences in the TAS total score and the TAS factors. The differences in the TAS total score and TAS factors 1 and 2 between AS subjects and controls remained significant.

In the AS group, no gender difference was found in respect of age, BDI, TAS total score and the TAS factors.

On the basis of SCID-I and BDI, the psychiatric status in the 20 AS subjects was as follows: 7 subjects had no other current axis I diagnoses besides AS, and 13 subjects had comorbid anxiety disorder alone ($n = 7$), or in combination with mood disorder ($n = 6$). Anxiety disorders included social phobia ($n = 8$), generalized anxiety disorder ($n = 2$), obsessive-compulsive disorder ($n = 3$), and other anxiety disorders ($n = 5$). Of those 6 persons who currently had a mood disorder, 2 displayed a moderate depressive episode and the others either a mild depressive episode or dysthymia. None of the participants had a diagnosis of alcohol misuse or alcohol dependency. The controls had no current axis I diagnoses assessed with the SCID-I.

There was no difference in the TAS total score or in the TAS factors between the AS subjects with and those without psychiatric axis I comorbidity.

The scores of the BNSQ items are shown in table 3 for AS subjects and controls. The AS subjects had, compared with the controls, more commonly difficulties in falling asleep (item 1), longer subjective sleep latency during working days (item 2a) but not in their free time (item 2b), a general feeling of low sleep quality (item 6), more often

Table 2. The means and standard deviations of different alexithymia scores measured by the TAS-20 in 20 subjects with AS and in 10 controls

TAS variable	AS subjects	Controls	p
TAS total score	54.2 ± 12.4	34.5 ± 5.1	<0.001 ^b
TASF1: difficulties in identifying feelings	19.7 ± 5.8	10.3 ± 3.3	<0.001 ^a
TASF2: difficulty in describing feelings	15.6 ± 5.1	8.7 ± 1.9	0.001 ^b
TASF3: externally oriented thinking	18.9 ± 5.3	15.5 ± 3.8	n.s. ^a

TASF1 = TAS factor 1.
^a Student's t test (two-tailed).
^b Mann-Whitney rank sum test.

Table 3. BNSQ assessed in 20 AS subjects and in 10 controls concerning sleep during the past 3 months

BNSQ item	AS subjects mean ± SD	Controls mean ± SD	p value
1 (<i>How often</i>) have you had difficulties in falling asleep?	2.85 ± 1.35	1.10 ± 0.32	<0.001 ^a
2a How many minutes as an average do you stay awake in bed before you fall asleep during working days?	40.95 ± 42.98	14.10 ± 9.82	0.04 ^a
2b How many minutes as an average do you stay awake in bed before you fall asleep during free time?	26.90 ± 23.69	11.80 ± 5.81	n.s. ^a
3 <i>How often</i> have you awakened at night?	3.50 ± 1.40	2.70 ± 1.25	n.s. ^b
4 How many times do you usually wake up during one night?	2.25 ± 0.85	1.90 ± 0.88	n.s. ^a
5 <i>How often</i> have you awakened too early in the morning without being able to fall asleep again?	1.95 ± 1.05	1.30 ± 0.48	n.s. ^b
6 How well have you been sleeping?	2.85 ± 1.04	1.40 ± 0.52	<0.001 ^b
7 <i>Have you used some sleeping pills?</i>	1.25 ± 0.91	1.00 ± 0.00	n.s. ^a
8 (<i>How often</i>) do you feel excessively sleepy in the morning after awakening?	3.70 ± 1.52	1.60 ± 0.70	0.002 ^a
9 (<i>How often</i>) do you feel excessively sleepy during daytime?	3.10 ± 1.29	1.60 ± 0.70	0.002 ^b
10 (<i>How often</i>) have you suffered from irresistible tendency to fall asleep while at work?	1.37 ± 0.68	1.10 ± 0.32	n.s. ^a
11 (<i>How often</i>) have you suffered from irresistible tendency to fall asleep during free time?	1.90 ± 0.97	1.10 ± 0.32	0.04 ^a
12 How many hours do you usually sleep per night?	8 h 6 min ± 91 min	7 h 45 min ± 67 min	n.s. ^b
13a At what time do you usually go to bed during working week? ^c	23.25 ± 90 min	23.02 ± 88 min	n.s. ^b
13b At what time do you usually go to bed during free days? ^c	0.21 ± 93 min	0.00 ± 85 min	n.s. ^b
14a At what time do you usually wake up during working week? ^c	7.36 ± 110 min	7.15 ± 53 min	n.s. ^b
14b At what time do you usually wake up during free days? ^c	9.58 ± 124 min	8.39 ± 82 min	ns ^b
15a <i>How often</i> do you have a nap at daytime?	1.85 ± 0.99	1.70 ± 0.67	ns ^a
15b If you have a nap, how long does it usually last for?	79 ± 53 min	61 ± 36 min	n.s. ^b
16 (<i>How often</i>) do you snore while sleeping?	1.61 ± 1.14	1.40 ± 0.70	n.s. ^a
17 How do you snore (additional information from significant others)?	1.50 ± 0.71	1.40 ± 0.70	n.s. ^a
18 <i>Have you had (or have others noticed) breathing pauses at sleep?</i>	1.00 ± 0.00	1.00 ± 0.00	n.s. ^a
19 If you snore at least 1–2 nights per week, how many years have you been snoring? ^d			
20 How many hours would you sleep per night if you had the possibility to sleep as long as you need to?	8 h 59 min ± 77 min	7 h 53 min ± 65 min	0.03 ^b
Total score	30.10 ± 7.40	20.40 ± 4.60	<0.001

Items are slightly abbreviated. Items indicated in italics have a 5-point scale (1 = never or less than once per month, 2 = less than once per week, 3 = on 1–2 days per week, 4 = on 3–5 days per week, 5 = daily or almost daily).

^a Mann-Whitney rank sum test.

^b Student's t test (two-tailed).

^c Military time is given.

^d Only 2 subjects in the AS group and 1 control reported snoring at least 1–2 nights per week.

excessive sleepiness after awakening (item 8) and during daytime (item 9), as well as more frequently an irresistible tendency to fall asleep during their free time (item 11). AS subjects also reported a need of longer sleep compared with controls (item 20).

Discussion

As far as we know, this is the first study concerning alexithymia in ASD.

The prevalence of alexithymia was very high among both male and female subjects with AS, as several popula-

tion studies in Finland have shown that the prevalence of alexithymia is 10–17% among men and between 5 and 10% among women [32–34]. AS men had a higher incidence of alexithymia as compared with AS women in accordance with population studies. Controls did not have alexithymia, partly because of the lack of psychiatric comorbidity. Depression has been reported to increase alexithymic traits [33]. However, the difference in the TAS-20 score and TAS factors 1 and 2 between AS subjects and controls remained significant after adjusting to the BDI score, thus rendering it improbable that depressive symptoms would solely explain intergroup differences.

The alexithymia construct originally arose in the context of attempts to validate the role of repression in psychosomatic diseases [35]. In more recent studies, attention has been drawn to a pervasive deficit in the capacity to experience and describe emotions [36] instead of the more discrete effect of repression. Moreover, as it is rather difficult to objectively verify the contents of subjective experience, it has been hypothesized that studying the degree of differentiation in the perception of exteroceptive emotional cues nearly approximates the corresponding interoceptive differentiation [36]. Some evidence to this hypothesis has come from the finding of the association between higher alexithymia scores and impaired recognition of facial expressions [37], a feature reported in AS as well [27].

The current view of the neuropsychological deficits in ASD emphasizes a deficit in ‘theory of mind’ (ToM) [38], which describes our ability to understand the mental states – beliefs, desires and intentions – of others, and to appreciate how these differ from our own mental state. The neural basis of the ToM consists of a core, domain-specific component that is centered in the amygdala circuitry and widely distributed co-opted systems, including the right temporoparietal cortex, language-related regions of the left hemisphere, and frontal lobes [38]. Studies on the neural basis of alexithymia have reported deficits in the limbic-prefrontal circuitry [6] and in the right hemisphere [8]. Future studies with larger patient samples and carefully designed neuropsychological test batteries in conjunction with functional brain imaging procedures might shed light on the intriguing question whether the alexithymic features reported in the present study in fact reflect a deficit in ToM in AS adults. The verbal IQ was within normal range in all AS adults (and did not differ from that of the controls without alexithymia), which makes it improbable that the alexithymic features would be an artifact due to low verbal capacity.

AS subjects differed from controls in more frequent difficulties in sleep initiation and excessive sleepiness after awakening and during daytime as well as in poor perceived sleep quality. This finding may have several explanations.

First, AS adults had a high degree of anxiety disorders which might predispose to nonorganic insomnia. For example, in a sleep questionnaire study of patients with generalized social phobia, impaired initiation of sleep with poor subjective sleep quality was reported [39]. In patients with panic disorder and social phobia, alexithymic features related to difficulties in identifying and differentiating emotions and physical sensations were significantly elevated as compared with external-oriented thinking [40], the former ones correlating with measures of anxiety. In a population study, functional insomnia associated with alexithymic features [41]. Thus, it is possible that in AS adults, alexithymia predisposes to anxiety, which in turn produces insomnia.

The slightly elevated BDI score in AS adults might reflect anxiety [29] instead of subclinical depression, which did not, however, explain the differences of TAS-20 and subscores between the AS subjects.

Some degree of pervasive anxiety is an inherent property of AS [27]. In the present study, only anxiety reaching the threshold of a psychiatric anxiety disorder was classified, but the remaining individuals were not anxiety free. In AS children, a profound pervasive difficulty in initiation and continuity of sleep is reported [42]. Together with the fact that in PDD patients classification of anxiety in terms of specific disorders might be difficult [27], one might speculate whether poor sleep quality in AS adults is indirectly due to AS itself as well.

Increased sleepiness during waking might also have organic causes. We have previously reported increased motor activity in AS adults during controlled rest in waking [43], which, if present also in sleep, might predispose to hypodopaminergic sleep disorders like periodic leg movements in sleep.

The accuracy of the sleep questionnaire with respect to other measures of sleep quality, like sleep diary, actigraphy and polysomnography, should also be confirmed.

The strength of the present study was that AS subjects were carefully matched with controls with regard to possible confounding factors. The diagnostic process utilized multiple sources of information both in the AS group and in controls. In many previous studies concerning pervasive developmental disorders, the normality of the control group has been inadequately assessed [7], which we tried to avoid as far as possible. The major limitation of the

present study is the small sample size, especially in controls, who did not represent a population sample.

In a cross-sectional study, the causal relationships are usually impossible to evaluate and the stability of alexithymic features should be evaluated in a follow-up study. In spite of frequent psychiatric comorbidity, the AS adults in the present sample did not use any psychotropic drugs, and they were mostly employed or studying, thus AS in these patients representing the mildest end of ASD. However, for the applicability of our results to AS adults in general, a replication in a larger population is needed.

The alexithymia construct derives from adult psychiatric practice and studies on autism and AS are published mostly by pediatric psychiatrists or neurologists. Both these fields have their own traditions of research and we have not found studies considering the differences and similarities of these theoretical models of mind.

It might be concluded that in clinical practice, ASD should be taken into account when considering differential diagnosis in persons with alexithymia. In adults with AS, special attention should be paid to assessing sleep quality in order to detect a primary sleep disorder and to minimize the effects of already compromised prefrontal functions on daytime performance.

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