ORIGINAL ARTICLE

Delayed Sleep Phase Syndrome: A Forerunner of Psychiatric Distress

Surriya Jabeen

ABSTRACT

Objective: To determine the association of extrinsic delayed sleep phase syndrome (DSPS) and psychiatric distress.
Study Design: Cross-sectional, analytical study.
Place and Duration of Study: Dow University of Health Sciences, Karachi, from July 2009 to September 2010.
Methodology: Sales personnel employed in different shopping malls in Karachi operating from 2 pm afternoon to 12.00 mid-night were inducted. The instrument used to define DSPS included difficulty in falling sleep timely at night and early rising in the morning. The proven DSPS subjects were studied by the Aga Khan University Anxiety Depression Scale (AKUADS) to explore the association of extrinsic (motivational) DSPS with anxiety and depression syndrome along with a survey questionnaire having twelve questions based on the criteria of ICSD (International Classification of Sleep Disorder) on DSPS, to study different characteristics of these subjects and its relationship with psychiatric illness.
Results: Eight hundred and eleven subjects were inducted, majority were male (n = 757, 93.3%). Three hundred and forty-five (42.5%) subjects scored > 19 with mean value of 41.4 ± 15.90. Result supported an association between DSPS and psychiatric distress.
Conclusion: Extrinsic DSPS prevention necessitates attention because of its positive relationship with psychiatric distress.

Key Words: *Extrinsic DSPS. Circadian rhythm. Psychiatric illness. Psychiatric distress.*

INTRODUCTION

Delayed Sleep Phase Syndrome (DSPS) is a sleep-wake schedule disorder where sleep onset and wake-up time are both considerably delayed and the person is not able to phase advance their sleep cycle although it is common but little reported reason of insomnia.[1] It is a frequently encountered state in sleep laboratories, however, its occurrence in general population is still not studied well and the affected person usually take-up to tranquilizers.[2,3] Exact cause of DSPS is not known and it is uncertain whether this is a manifestation of intrinsic pathology or a socially reinforced sleep-wake design.[4] Although the disorder is recognized as intrinsic and extrinsic, however, actually there are combinations of endogenous and exogenous factors leading to the development of each.[5]

There are two important determinants, the timing of human sleep wake cycle and sleep structure. Supra-chiasmatic nucleus (SCN) in the anterior hypothalamus is the principal oscillator that maintain the circadian of sleep wakefulness and a sleep homeostat placed exterior to SCN which include environmental stimuli and host of processes.[6-8] Sleep-wake system depends on the endogenous circadian rhythm of a number of biological parameters including temperature, hormones and metabolic parameters.[9] The temporal cue that normally limits the length of the endogenous circadian period to 24 hours is the surrounding light / dark cycle (day and night). It is this state of equilibrium among circadian pacemaker located in the SCN, circadian photoreception as well as feedback from the sleep-wake cycle on to these mechanisms thought to play an important function in the regulation of sleep-wake cycle and their disorders. In the sleep homeostasis, SCN where the rhythm of sleep and wakefulness is control by one or more internal biological clocks, might have reduced sensitivity to environmental cues most notably to surrounding light dark cycle in DSPS.[10] With the result, there is a misalignment in the circadian timing system. The total sleep time, sleep efficiency, spindle density are not altered neither there is any change in REM or in the slow wave activity.[11]

Since most of the subjects having DSPS are overlooked as insomniacs, therefore, role of environment in this pathology necessitates attention. There is emerging evidence that DSPS is often a predecessor of psychiatric illnesses namely depression.[12]

The aim of the study was to examine the impact of life style pertaining to sleeping habits with reference of DSPD on mental performance.

METHODOLOGY

This study was conducted at Dow University of Health Sciences, Karachi, from July 2009 to September 2010. A random sample of 811 subjects included sales personnel from various shopping Malls in Tariq Road

Department of Community Medicine, Dow Medical College, Dow University of Health Sciences, Karachi.

Correspondence: Dr. Surriya Jabeen, B-100, Block W, Iqbal Town, North Nazimabad, Karachi.
E-mail: surriyaa.jabeen@duhs.edu.pk

Received: March 06, 2012; Accepted: August 06, 2013.

and Clifton, Karachi. Using open epi, sample of 811 subjects was calculated by using 95% confidence interval, a maximum error of +1.5%, aged between 20 to 50 years, test power of 80% and prevalence of 5.0%. Participants were distinctively included from shopping centres functioning from 2 p.m. afternoon to 12.00 mid-night. Diagnosis of DSPS was based on the inability to sleep before 1.00 p.m. and intense difficulty in rising for at least one year. Tool exercised to identify this sleep disorder included Insomnia Management 1 possible diagnosis and management technique from the Department of Human Service, Government of South Australia (by Dr. Leon Lock, Repatriation General Hospital of sleep disorders). This study was approved by Ethical Review Committee of Dow University of Health Sciences, Karachi and a written informed consent was obtained from all the participants. Respondents with a history of head trauma, obesity, drug abuse, excessive caffeine intake or having active psychotic symptoms were excluded.

AKUADS (Aga Khan University Anxiety and Depression Scale) derived from HADS (Hospital Anxiety Depression Scale) was employed to address the association of motivational DSPS and the psychiatric illness, where a score of 19 and above signifies a case. Demographic data such as age, gender, occupation and education was obtained. Reliance was placed on questionnaire data rather than polysomnography which is not required in routine assessment of DSPS.

All data analysis was carried out on Statistical Package for Social Sciences (SPSS) version 16.0 for windows. Quantitative variables were presented by their mean ± SD value, however, the qualitative variables were presented by frequency and percentages. Chi square test was employed to determine the association of depression anxiety with categorical variables. Comparison of age between depression and non-depression group was done by Student's t-test. The results were considered significant at $p < 0.05$. 95% Confidence interval was calculated for incidence of anxiety depression among DSPS proven group.

Table I: Demographic features of study population.

Variable	n (%)	Mean±SD	p-value	95% CI
Subject scoring (>19) anxiety and depression	345 (42.5%)	41.40±15.90	--	39.1- 45.9
Gender				
Male	757 (93.3%)	--	--	--
Female	54 (6.7%)	--	--	--
Age (years)				
Overall subjects	811 (100%)	26.90±6.35	--	26.5-27.3
20-25	432 (53.3%)	--	--	--
< 30	590 (72.7%)	--	--	--
> 30	221 (27.3%)	--	--	--
With depression and anxiety	345 (42.5%)	27.33±6.65	0.093	26.03-27.14
Without depression and anxiety	466 (57.5%)	26.58±6.12		26.63-28.04

RESULTS

Out of 811 DSPS proven subjects, 345 (42.5%) were found positive for depression anxiety when assessed through AKUADS. The mean age was 26.90 ± 6.35 years and 590 (72.7%) of the subjects who were below 30 years of age. Majority of the subjects afflicted by depression were between 20 – 25 years of age (n = 432, 53.3%). The group not affected by anxiety/depression (n = 466) had a mean age of 26.59 ± 6.11 years and that affected by depression anxiety (n = 345) aged 27.33 ± 6.65 years (p = 0.093, Table I). There were 757 (93.3%) males and 54 (6.7%) females. Out of the 757 males, 302 (87.5%)

Table II: Characteristics of DSPS subjects with and without depression.

ICSD criteria*	Without depression (n = 466)	With depression (n = 345)	p-value
Time of sleep at night			
01:00 AM	106 (22.7%)	61 (17.7%)	0.038
02:00 AM	168 (36.1%)	108 (31.3%)	
03:00 AM	108 (23.2%)	105 (30.4%)	
04:00 AM	84 (18.0%)	71 (20.6%)	
Sleep latency			
< 30 minutes	221 (47.4%)	114 (33.0%)	< 0.001
> 30 minutes	245 (52.6%)	231 (67.0%)	
Quality of sleep			
Refreshing	372 (79.8%)	229 (66.4%)	< 0.001
Nonrefreshing	94 (20.2%)	116 (33.6%)	
Time of arousal			
07:00 AM	27 (5.8%)	30 (8.7%)	0.098
08:00 AM	31 (6.7%)	38 (11.0%)	
09:00 AM	90 (19.3%)	59 (17.1%)	
10:00 AM	136 (29.2%)	94 (27.2%)	
11:00 AM	107 (23.0%)	65 (18.8%)	
12:00 PM	47 (10.1%)	31 (9.0%)	
After 12:00 PM	28 (6.0%)	28 (8.1%)	
Tiredness on early awakening			
Yes	306 (65.7%)	280 (81.2%)	< 0.001
No	160 (34.3%)	65 (18.8%)	
Craving to sleep extra on weekends			
No craving	235 (50.4%)	159 (46.1%)	0.102
2 hours	121 (26.05)	99 (28.7%)	
4 hours	76 (16.3%)	66 (19.1%)	
> 6 hours	30 (6.4%)	13 (3.8%)	
> 8 hours	4 (0.9%)	8 (2.3%)	
Disturb daily routine			
Yes	145 (31.1%)	191 (55.4%)	< 0.001
No	321 (68.9%)	154 (44.6%)	
Family history of DSPS			
Yes	204 (43.8%)	140 (40.6%)	0.362
No	262 (56.2%)	205 (59.4%)	
Duration of DSPS			
1 year	44 (9.4%)	29 (8.4%)	0.788
2 years	56 (12.0%)	40 (11.6%)	
3 years	56 (12.0%)	47 (13.6%)	
4 years	61 (13.1%)	46 (13.3%)	
5 years	74 (15.9%)	52 (15.1%)	
6 years	70 (15.0%)	64 (18.6%)	
> 6 years	105 (22.5%)	67 (19.4%)	

were positive for depression symptoms. Amongst females, out of 54, 43 (12.5%) females were demonstrating symptoms of depression. The gender showed higher major proportion of depression anxiety among the males when tested by chi-square test of proportion ($p < 0.0001$). Chi-square showed a significant association of sleep latency and psychiatric distress, where sleep latency was more than 30 minutes ($n = 231$, 67.0% out of 345, $p < 0.001$), quality of sleep of these subjects had a positive relationship with depression. Tiredness on early arousal was also found to be statistically significant for depression ($p < 0.001$). DSPS subjects who acknowledged disturbed daily routine were positive for depression ($p < 0.001$). However, time to bed ($p = 0.038$), time of arousal ($p = 0.098$), craving for extra sleep on weekends ($p = 0.102$), family history of DSPS ($p = 0.362$) and duration of the DSPS ($p = 0.788$) were found to have insignificant difference between those with and without depression (Table II).

DISCUSSION

The findings in this study were in accordance with some of the earlier studies on this subject showing a positive relationship of DSPS and depression.[13] Respondents in the current study were influenced mainly by exogenous factors which in this situation was their job demand that had made subjects vulnerable to DSPS, as the respondents have acknowledged their work responsible for disturbing the sleep schedule, therefore, variables job duration was compared in relation to troubled emotions duration and time period was recorded accordingly in this study. In an earlier study, it was determined that the extrinsic type DSPS was more frequently seen than the intrinsic type.[14]

The respondents in the current study were aged between 20 – 50 years and the majority of population showing a higher frequency for anxiety depression were falling under the age of 30 years. This was consistent with another study voicing that DSPS commences in the adolescence to some variation in circadian systems that may be triggered by long vacation, hormonal changes, peer reinforced behaviour pattern such as staying up late and sleeping in.[15] In one study, 11.5% of university students manifested symptoms of DSPS which was about twice as great as the general population.[16] Furthermore, the frequency of troubled sleep pattern were higher than in preceding study. The same research also supported that the students lifestyle has a key influence in the development of DSPS and included recommendations that might help universities to manage sleep difficulties amongst students.[16] In current study, the sleep latency was more than 30 minutes in the subjects who were positive for depression as compared to the other group with a sleep latency of less than 30 minutes. One of the findings observed in the current study was that DSPS subjects who were not fresh after their major sleep was over, were showing significant relationship with a depression. Walters and Pilcher noticed that many college students, deliberately rob of sleep during week days, try to balance on the weekend by sleeping long hours; such unbalanced sleep patterns deteriorate this condition linked with DSPS.[17]

In this study, it appeared the subjects who could not maintain their daily routine were showing a higher frequency of depressing symptoms. In the last few decades due to change in the lifestyle such as television, playing computers games, mid-night broad casting program, additionally many employees such as student with part time jobs work in places like convenience stores and video rental shops that remain open 24 hours. All of it seems to work as circadian oscillators and increases the tendency of population to be evening type which has resulted in delay onset of sleep and psychiatric distress.[18] Other studies direct that in younger subjects who were otherwise normal it was uncertain if it was a sign of intrinsic pathology or socially drive sleep wake mode that could be easily changed or modified if situation arises, a mismatch between biological sleep clock and the environment cues.[19]

There were studies where the notion was that DSPS was not merely a disorder of sleep timings but a disorder of sleep system itself.[20] According to one of the researches, DSPS could start even in early childhood or infancy and is particularly associated with poor academics. This study which was carried out in between the ages of 8 and 53 years on 14 subjects attending a sleep clinic all of whom reported extreme anguish as a result of troubled morning arousal; the education was severely disturbed in nine subjects in whom the DSPS started in school years. The current study supported this research since present demonstrated early morning arousal in DSPS population was significant for anxiety depression. In the same preceding study, it was illustrated that personality abnormality with high mean scores on Minnesota multiphase personality inventory for paranoia, schizophrenia and depression occurred in two participants with a trait deviance > 2 SD one additional subject had a high depression rating on beck depression inventory.[21]

Despite these findings, the recognition of DSPS has remained complicated at the primary care level when it is misunderstood as primary insomnia. It was estimated that between 5 – 10% of presenting insomniacs have actually circadian rhythm disorder underneath.[22] Some of recent studies have indicated that desynchronized circadian clock in humans was associated not only with sleep disorders but a number of physiological disorders and mood disorders.[23] Consequently DSPS can be disabling and socially isolating state. Following strict ICSD criteria, Japanese research estimated 0.13% prevalence of DSPS with more disciplinary problem and depression in adolescence.[24] View is growing that any disruption of circadian clock can produce physical and mental symptoms and morbidities. Montelone and Maj

outlined the evidence supporting a supposed relation between alteration in endogenous rhythmicity and mood disorder and also focused on strategies for managing disturbed circadian rhythm as a probable treatment choice and other means able to resynchronize the human biological clock system that included, sleep deprivation, light therapy and drugs specifically acting on the endogenous clock system, that have revealed antidepressant properties. The knowledge arising from the wide spectrum of physiological process driven by the circadian timing would be helpful for detailed understanding of the etiopathogenesis of mood disorders and the development of more effective management.[25] In the same analysis, it was noticed that effectively managing DSPS can bring about restitution in the patients mood and make antidepressant more useful and in addition treatment of the depression can make patients more effective following DSPS treatment. Most of the studies conducted demonstrated DSPS to be multifactorial in origin, there were multiple lines of evidence suggesting dysfunctions at the behavioural, physiological, and genetic level.

Further work is desirable because DSPS is often misdiagnosed as primary insomnia. Recent data indicate that psychiatric illness is foremost public health problem, however, little data is available on DSPS and its relationship with psychiatric illnesses. Therefore, more focused efforts are desired to uncover its dimension and behavioural aspect to recognize the mechanism implicated in the physiology of the circadian clock system for a deeper understanding of the etiopathogenesis of mental disorder and the development of more effective therapeutic strategies. The current study will add to the body of literature already available on DSPS and propose future research direction to the research participants.

CONCLUSION

Results supported an association between DSPS and psychiatric distress.

REFERENCES

1. Weitzman ED, Czeisler CA, Colman RM. Delayed sleep phase syndrome. *Arch Gen Psychiatry* 1981; **38**:737-46.
2. Chung K, Cheung M. Sleep-wake pattern and sleep disturbance among Hong Kong Chinese adolescents. *Sleep* 2008; **31**:185-94.
3. Gordon NP, Cleary PD, Parker CE, Czeisler CA. The prevalence and health impact of shift work. *Am J Public Health* 1986; **76**:1225-8.
4. Barion A, Zee CP. A clinical approach to circadian rhythm sleep disorders. *Sleep Med* 2007; **8**:566-77.
5. Morgenthaler TI, Chiong T, Alessi C. Practice parameter for the clinical evaluation and treatment of circadian rhythm sleep disorders. *Sleep* 2007; **30**:1445-59.
6. Dijk DJ, Lockley SW. Functional genomics of sleep and circadian rhythm invited review: integration of human sleep-wake regulation and circadian rhythmicity. *J Appl Physiol* 2002; **92**:852-62.
7. Granada AE, Cambras T, Díez-Noguera A, Herzel H. Circadian desynchronization. *Interface Focus* 2011; **1**:153-66. Epub 2010 Nov 17.
8. Scheer FA, Wright KP, Kronauer RE, Czeisler CA. Plasticity of intrinsic period of human circadian timing system. *PLoS One*. 2007; **2**:e721.
9. Fischer S, Smolink R, Herms M. Melatonin acutely improves the neuroendocrine architecture of sleep in blind individuals. *J Clin Endocrinol Metab* 2003; **88**:5315-20.
10. Okawa M, Uchiyama M. Circadian rhythm sleep disorder: characteristics and entrainment pathology in delayed sleep phase and non-24 sleep-wake syndrome. *Sleep Med Rev* 2007; **11**:485-96 .
11. Kayumov L, Brown G, Jindal R, Buttoo K, Shapiro CM. A randomized, double-blind, placebo-controlled cross over study of the effect of exogenous melatonin on delayed sleep phase syndrome. *Psychosomat Med* 2001; **63**:40-8.
12. Ford ED, Kamerow BD. Epidemiological study of sleep disturbance and psychiatric disorders. *JAMA* 1989; **262**:1479-84.
13. Kripke DF, Rex KM, Ancoli-Israel S, Nievergelt CM, Klimecki W, Kelsoe JR. Delayed sleep phase cases and controls. *J Circadian Rhythms* 2008; **6**:6.
14. Schrader H, Bovim G, Sand T. The prevalence of delayed and advance sleep phase syndrome. *J Sleep Res* 1993; **2**:51-5.
15. Sack RL, Auckley D, Auger RR, Carskadon MA, Wright KP Jr, Vitiello MV, *et al*. Circadian rhythm sleep disorder: part 1 basic principles, shift work and jet lag disorders an American Academy of Sleep Medicine review. *Sleep* 2007; **30**:1460-83.
16. Brown CF, Barlow S, Bublotz JR, Walter C. Prevalence of delayed sleep phase syndrome in university students. *Coll Student J* 2001; **35**:472-6.
17. Pilchler JJ, Walters AS. How sleep deprivation affect psychological variable related to college students cognitive performance. *J Am Coll Health* 1997; **46**:121-6.
18. Nakade M, Takeuchi H, Kurotani M. Effects of meal habits and alcohol /cigarette consumption on morningness-eveningness preference and sleep habits by Japanese female students aged 18-29. *J Phsiol Anthropol* 2009; **2**:83-90.
19. Thorpy MJ, Kormen E, Spileman AJ. Delayed sleep phase syndrome in adolescent. *Healthcare* 1988; **9**:22-7.
20. Campbell SS, Murphy JP. Delayed sleep phase syndrome in temporal isolation. *Sleep* 2007; **30**:1225-8.
21. Alvaraz B, Dahlitz MJ, Vignau J, Parkes JD. The delayed sleep phase syndrome: clinical and investigative finding in 14 subjects. *J Neurol Neurosurg Psychiatry* 1992; **55**:665-70.
22. Sedwick PM. Disorder of the sleep-wake cycle in adults. *Postgrad Med J* 1998; **74**:134-8.
23. Regestein QR , Monk TH. Delayed sleep phase syndrome: a review of its clinical aspects. *Am J Psychiatry* 1995; **152**:602-8.
24. Yazaki M, Shirakawa S, Okawa M, Takashashi K. Demography of sleep disturbance associated with circadian rhythm disorder in Japan. *Psychiatry Clin Neurosci* 1999; **53**:267-8.
25. Montelone P, Maj M. The circadian basis of mood disorders: recent development and treatment implications. *Eur Neuropsychopharmacol* 2008; **18**:701-11.