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CORRELATION ASSESSMENT OF OBSTRUCTIVE SLEEP APNEA SYNDROME AND COGNITIVE DYSFUNCTION AMONG TYPE-II DIABETES MELLITUS PATIENTS

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Abstract:

Objective: *The objective of this research was to investigate and identify the T2DM patient's cognitive dysfunction along with OSAS (Obstructive Sleep Apnea Syndrome); we also aimed to analyze related features as well.*

Methods: *This research was carried out on a total of 115 T2DM patients at Mayo Hospital, Lahore (October 2017 to June 2018). The patients were divided into two groups of OSAS (83) and non-OSAS (32) groups. Physical assessment of every patient was carried out. We also evaluated the levels of Nocturnal lowest saturation of pulse oxygen (LSPO2), Apnea-hypopnea index (AHI) and simple mental state examination scale (MMSE).*

Results: *In the patients diagnosed with diabetes, OSAS have reduced glycated haemoglobin, thrombocytocrit, platelet count, lowest mean arterial oxygen and MMSE score than non-OSAS patients; glycemic control and cognitive dysfunction state of patients have an association with diabetes duration. Glycemic control is poor along with prolonged diabetes duration which makes the cognitive dysfunction even obvious.*

Conclusion: *T2DM patients with prolonged diabetic duration along with OSAS; the glycemic control tends to be poor which easily causes cognitive dysfunction. Meanwhile, blood system coagulation in the patients of OSAS along with diabetes bear some impact.*

Keywords: *T2DM, Diabetes, Lowest Saturation of Pulse Oxygen (LSPO2), Apnea-Hypopnea Index (AHI) and Simple Mental State Examination Scale (MMSE) and Platelet.*

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INTRODUCTION:

Communities and families face serious issues of cognitive dysfunction of the ageing elders. Various factors are involved in cognitive dysfunction such as frontotemporal dementia, Alzheimer's disease, corticobasal degeneration etc. Meanwhile due to vascular risks which include stroke, hypertension, diabetes, etc. have universally higher incidence which is of grave concern as diabetes-induced cognitive dysfunction has been confirmed by various studies [1 – 4].

Back in 2010, a European author studied patients older than seventy years and followed them for a period of three years for cognitive function assessment. After excluding various effects of education level, age, temporal lobe atrophy and white matter lesions; he mentioned diabetes as a sole risk factor which may lead to cognitive dysfunction [1]. Hypoxia and Hyperglycemia are prime changes in diabetes which are correlated with numerous other complications, these also altered the kinetics, which is diabetes and its associated complications relevant mechanism [5]. Further literary evidence demonstrates 75% T2DM obese patients have OSAS and major OSAS induced pathological change is chronic tissue hypoxia [6]. Therefore, it is clear that tissue hypoxia is an attached pathological change from hyperglycemia among diabetes patients; whereas, hypoxia is an important cognitive dysfunction risk. Thus, cognitive dysfunction has a close relation with chronic tissue hypoxia and hyperglycemia. The objective of this research was to investigate and identify the T2DM patient's cognitive dysfunction along with OSAS (Obstructive Sleep Apnea Syndrome); we also aimed to analyze related features as well.

METHODS:

This research was carried out on a total of 115 T2DM patients at Mayo Hospital, Lahore (October 2017 to June 2018). The patients were divided into two groups of OSAS (83) and non-OSAS (32) groups. Physical assessment of every patient was carried out. We also evaluated the levels of Nocturnal lowest saturation of pulse oxygen (LSPO2), Apnea-hypopnea index (AHI) and simple mental state examination scale (MMSE). A total of 115 patients underwent Polysomnography including 51 males and 64 females. The mean age of male and female patients was respectively (71.60 ± 1.01) and (68.75 ± 1.20). We made two groups (C-I and C-II) from the control group. C-I and C-II presented respective AHI as (< 5 times/hour) and (≥ 5 times/hour) by monitoring polysomnography. We recorded information about age, gender, glycated haemoglobin, fasting blood glucose, vascular plaque, diabetic duration, LSPO2 and platelet. Monitoring of the patients through polysomnography started right from the first night. On the very first day at the hospital, the sleep was not affected by using coffee, tea, sedatives and other factors. The monitoring of AHI, average concentration of oxygen and the lowest concentration of oxygen were monitored for a period of more than six hours. American Sleep Disorder guidelines were used to determine AHI from mild to severe disorder category. Qualified doctors and designated person monitored the breath of the patients for respiration. After monitoring breath clinical assessment was also carried out for blood glucose, 2-hour postprandial blood glucose, glycated haemoglobin, platelet distribution width, platelet count, thrombocytocrit, mean platelet volume, red blood cell distribution width and hematocrit. SPSS software was used for outcomes analysis (P-Value < 0.05).

RESULTS:

In the patients diagnosed with diabetes, OSAS have reduced glycated haemoglobin, thrombocytocrit, platelet count, lowest mean arterial oxygen and MMSE score than in-OSAS patients; glycemic control and cognitive dysfunction state of patients have an association with diabetes duration. Glycemic control is poor along with prolonged diabetes duration which makes the cognitive dysfunction even obvious.

Detailed outcomes analysis of T-Value, P-Value and General Information is given in the tabular data underneath:

Table – I: General Information (Mean ± SD)

Group		OSAS (83)	nOSAS (32)
Age	Mean	70.4	69.3
	±SD	10.9	12.4
MMSE	Mean	23.4	24.9
	±SD	2.9	3.5
Blood Glucose	Mean	6.2	5.9
	±SD	2.4	1.5
HBA1C%	Mean	7.4	6.8
	±SD	1.1	0.8
Duration	Mean	5.0	3.4
	±SD	5.8	6.7
Plaque	Mean	8.9	9.4
	±SD	18.7	17.3
LASPO2	Mean	81.9	84.4
	±SD	4.7	8.8

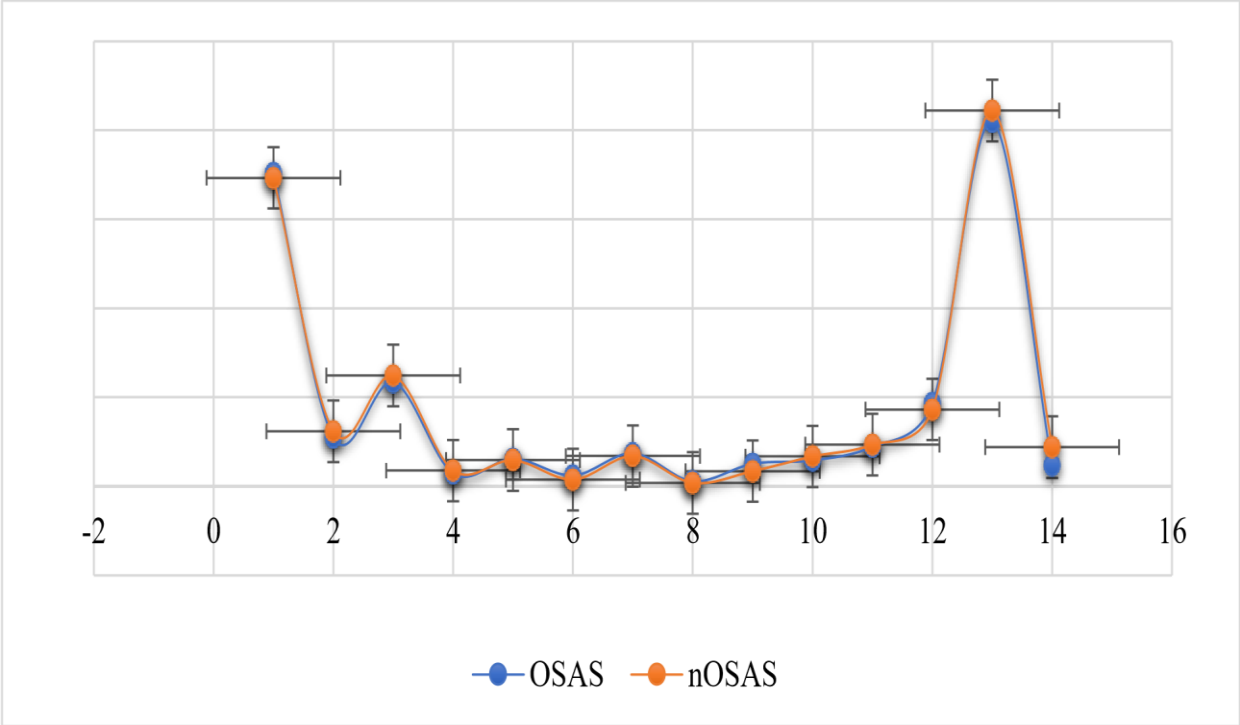

Table – II: General Information (Mean ± SD)

Group		OSAS (83)	nOSAS (32)
PLT	Mean	190.5	212.5
	±SD	53.2	47.0
PLT Width	Mean	13.6	13.4
	±SD	2.5	2.3
PLT Volume	Mean	10.9	10.8
	±SD	1.2	1.0
Thrombocytocrit	Mean	0.2	0.2
	±SD	0.1	0.0
Hematocrit	Mean	37.9	38.8
	±SD	3.5	2.6
RBC Width	Mean	42.8	42.6
	±SD	2.8	2.9
RBC Width	Mean	13.2	13.0
	±SD	0.7	0.7

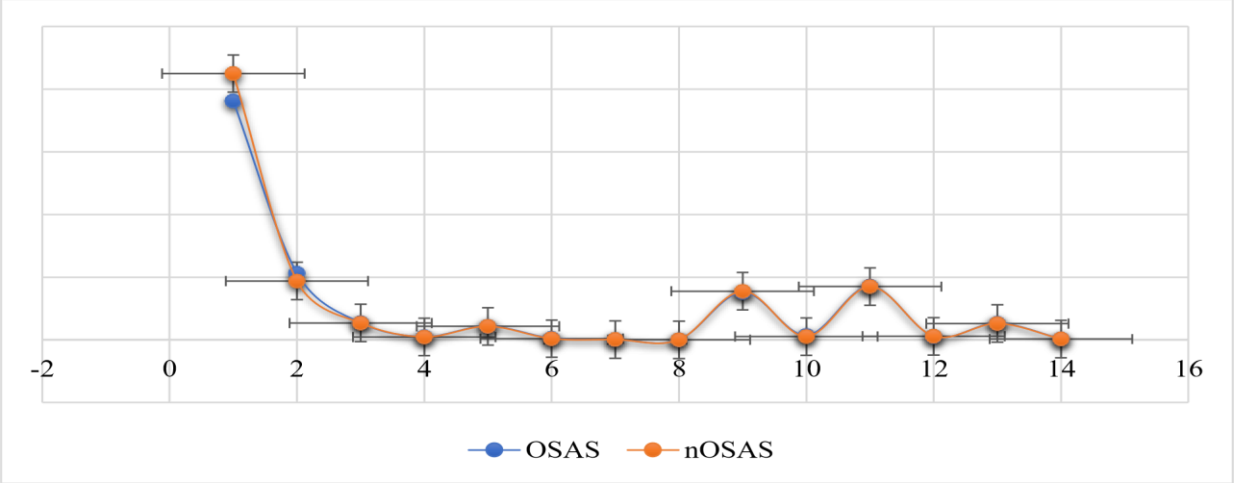

Table – III: T-Value Versus P-Value

Value	T-Value	P-Value
MMSE	2.4	0.02
HBA1C	3.15	0.02
PLT	2.05	0.04
Thrombocytocrit	2.17	0.03
LSPO2	2.02	0.04
PLT Width	4.39	0
PLT Volume	4.89	0
HCT	5.31	0
Erythrocyte Width	2.09	0.04
Erythrocyte Width	2.28	0.02

DISCUSSION:

OSAS refers to repeated upper airway obstruction episodes during sleep with reduced saturation of blood oxygen; diagnosis needs a criterion of excessive sleepiness or daytime sleepiness and sleep apnea for 5 times or more than five times in an hour as observed through polysomnography. There are three clinical types of this syndrome known as a central, obstructive or mixed type. Most repeated type is the obstructive type. The T2DM cognitive impairment with OSAS may have an association with daytime sleepiness that is because of nighttime sleep structural disorder or nocturnal hypoxemia. The severity of cognitive impairment is different as sleep-disordered breathing severity is different among patients. OSAS is greatly due to the increased level of sugar in the blood. Diabetes can possibly accelerate cognitive dysfunction which can also develop into senile dementia. Diabetes-associated cognitive dysfunction refers to another prolonged complication if diabetes progression [8].

OSAS has also another close link with neurological diseases causing sleep apnea. It also causes cognitive dysfunction, daytime sleepiness and various neurological diseases. Another possible link of OSAS is with cerebrovascular diseases and Alzheimer's disease (AD). Studies establish that after an acute stroke about (32% – 7%) patients face an onset of sleep-disordered breathing and among these OSAS cases are (69% – 95%). The patients diagnosed with moderate to severe Parkinson's diseases also face breathing obstructions while sleeping and awake. OSAS cognition function damage may face impairment of alertness, memory, attention, judgment, concentration, reasoning, abstraction, execution, intelligence and psychomotor. Cerebral cortical thinning in the cerebral cingulate gyrus, hippocampus in patients with OSAS9 and frontal lobe causes memory, study and attention poor than controls. Nocturnal intermittent hypoxia can result in degenerative hippocampus in mice variations which causes mice spatial learning disorder, while nocturnal sleep apnea has an association with behavioural and cognitive defects [10]. Few reports also point out that after OSAS, patients receive continuous positive airway pressure (CPAP), vigilance/ attention of OSAS patients can possibly increase and memory & executive function of fifty percent patients can also be enhanced [11]. Bedard reported that CPAP management can normalize the nighttime sleeping pattern of the patients with improved alertness during the daytime. They can also plan and execute well along with all other improved cognitive functions [12]. According to Naegele, in OSAS patients there was a shift of cognitive functioning before and ager four to six months of CPAP management except for short-term memory span and related cognitive functions associated to frontal lobe (attention, planning, learning and organizational skills).

Few authors report that fifty percent of the cerebrovascular disease patients have OSAS that can pose multisystem damage such as nocturnal hypoxemia, hypertension and hypercapnia [14]. Small vessel lesion due to OSAS may result into long-term hypoperfusion status of vital cerebral cortex parts which are involved in brain tissues and cognitive function; it is also sensitive to hypoxia and ischemia, particularly limbic temporal lobe and hippocampus. After the occurrence of necrosis of limbic temporal lobe and hippocampus, associated released white matter will have mild cognitive impairment and ischemic demyelination. Dementia patients to some extent face sleep disorders like OSAS patients.

We reported 72% of cases of OSAS out of which 63% cases of severe OSAS. Few research studies also report a higher incidence of OSAS among T2DM cases. According to Kosseifi, 70% of diabetic cases were OSAS as diagnosed through polysomnography [15]. Another Japanese author also reported 77.5% occurrence of OSAS among diabetes patients [16]. Every research outcome portrays that OSAS is high among diabetes patients which needs more attention [17]. Few authors also put emphasis on the increased care of diabetes patients as diabetic autonomic neuropathy can result in autonomic respiratory neuromuscular dysfunction [18]. Few consider nocturnal hypoxemias as a reason behind cognitive impairment among OSAS patients; whereas, the degree of hypoxia has a link with cognitive impairment [19]. Various factors cause upper airway obstruction while sleeping among OSAS patients which may also cause hypoxia-reperfusion and hypoxemia injury [20]. Hypoxemia can activate coagulation of blood and it can also damage endothelial cells [21]. Furthermore, it causes thrombosis and fibrin deposition which also affect cognitive function.

It is important for OSAS diabetic (T2DM) patients to start blood glucose management at the earliest. If the condition allows than early management of CPAP is also suggested to reduce the chances of cognitive dysfunction [22].

CONCLUSION:

T2DM patients with prolonged diabetic duration along with OSAS; the glycemic control tends to be poor which easily causes cognitive dysfunction.

Meanwhile, blood system coagulation in the patients of OSAS along with diabetes bear some impact.

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