ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Biological Clock: Serum Melatonin in Hypertension.

Hemalatha D*, Shanthi B, and Manjula Devi AJ.

Department of Biochemistry, Sree Balaji Medical College, Chrompet, Chennai, Tamil Nadu, India.

ABSTRACT

To study the significance of serum melatonin level in essential hypertensive patients compared with age and sex matched healthy controls. Case control Study conducted at Sree Balaji Medical College & Hospital. Total number of people recruited in the study was 100. Of which, 50 are essential hypertensive patients and 50 are healthy controls. It was estimated by Melatonin ELISA KIT - Quantitative sandwich enzyme immunoassay technique. Serum melatonin levels was found to be significantly low in hypertensives compared to healthy controls with significant p value (0.001). In our study, significant reduction of melatonin levels is obseved in hypertensives when compared with normotensives.The reduced melatonin level in hypertensives may lead to the risk of developing cardiovascular complication insisting prompt control of blood pressure.

Keywords: biological clock, hypertension, melatonin

Corresponding author

ISSN: 0975-8585

INTRODUCTION

Melatonin is the endocrine product of pineal gland with circadian rhythm produced predominently during night time. Melatonin synchronizes day- night cycle through photo signal received from retina, hence acts as internal clock predictive of solar time - BIOLOGICAL CLOCK. Melatonin represents an important chronobiological regulatory molecule that is released from the pineal gland with peaks during the night-time [1]. New era of melatonin research reveals its diverse functions from Aging to Aggression, Sleep to Stress, Reproduction to Regulation of tissue, Hibernation to Hypertension.

Melatonin receptors are present in the vasculature and mediate vascular constriction and vasodilation through MT1 and MT2 receptors, respectively. Melatonin administration generally induces a decrease in blood pressure [2]. One possible mechanism contributing to the melatonin hypotensive effects is through its sympatholytic properties [3].

Aim and Objectives

- To study the significance of serum melatonin level in essential hypertensive patients
- To compare serum melatonin in hypertensives with with age & sex matched healthy controls

MATERIALS AND METHODS

- Case control Study conducted at Sree Balaji Medical College & Hospital
- Total number of people recruited in the study - 100.
- 50 - Essential hypertensive patients
- 50 - Healthy controls.
- It includes 48 male and 52 female, with age ranging from 30 to 60 yrs.
- Blood is collected in a plain vacutainer for measuring serum melatonin level in early morning, estimated by Melatonin ELISA KIT - Quantitative sandwich enzyme immunoassay technique.

RESULTS

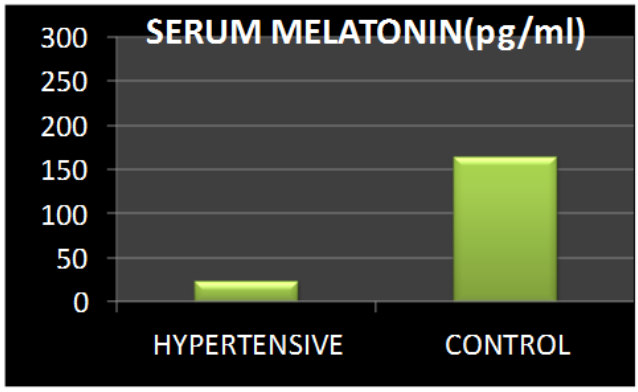
Serum melatonin levels were significantly low in hypertensives compared to healthy controls.

Statistical analysis was done using SPSS software.

Table 1: Depicts the low melatonin level in hypertensives than the control with significant p value (0.001)

SERUM MELATONIN LEVELS (pg /ml)	HYPERTENSIVE PATIENTS	NUMBER	MEAN	S.D	P VALUE
		50	23.5	6.71	0.001
	CONTROLS	50	165.2	130.5	

Bar diagram also indicates the same finding with reduced serum melatonin level in hypertensives compared with healthy controls.



ISSN: 0975-8585

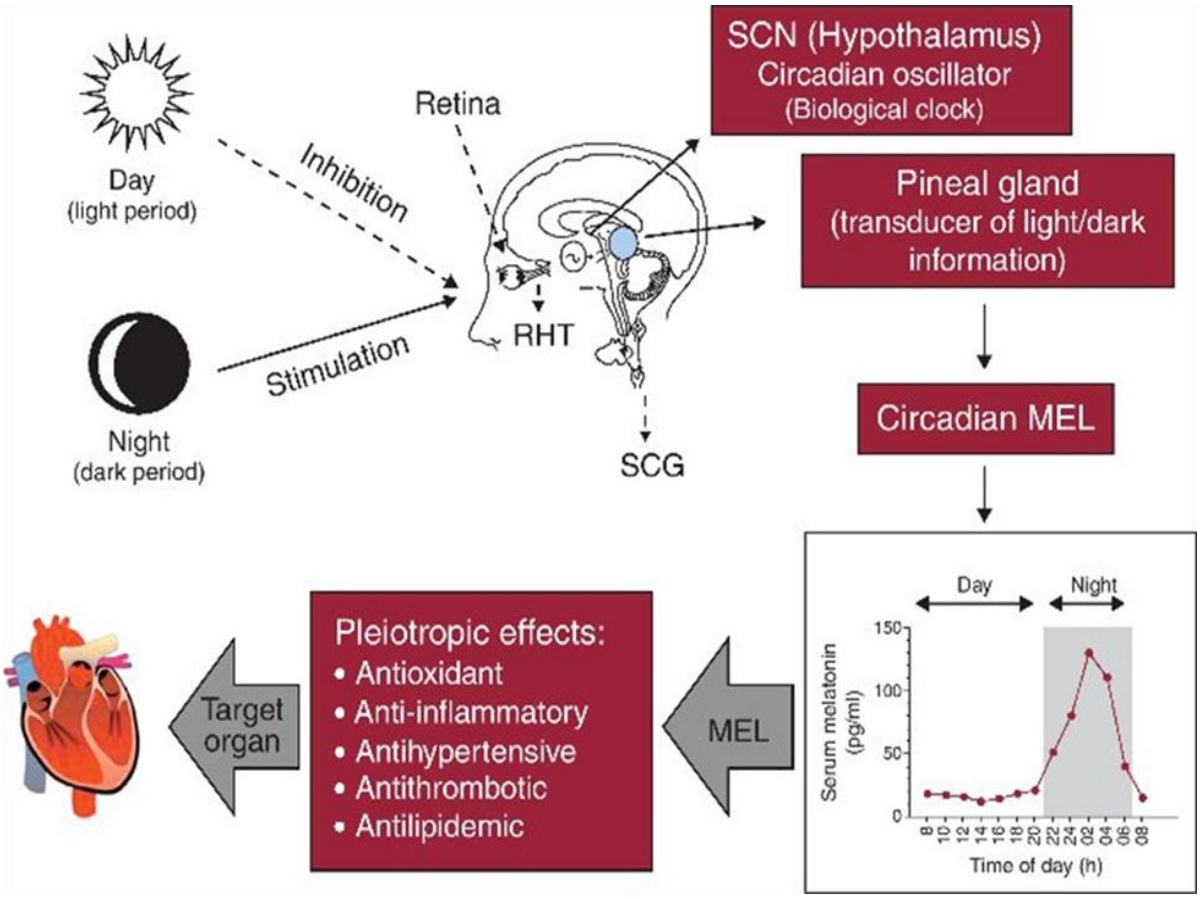
DISCUSSION

Melatonin synthesis from pineal gland is controlled by suprachiasmatic nucleus (SCN) - **"endogenous circadian pacemaker"** located in hypothalamus of brain . The mechanisms by which melatonin is antagonizing Ang II actions in cardiovascular and metabolic diseases are comprising its antihypertensive, antioxidant, and anti-inflammatory functions [4]. Melatonin has direct free radical scavenging and indirect antioxidant activity. Through these marked antioxidant properties, melatonin has cardioprotective effects, in particular in myocardial damage after ischemia-reperfusion [5].

Renin-Angiotensin system(RAS) modulate melatonin secretion .Melatonin antagonizes Angiotensin II action along with Increased nitric oxide release & Reduced sympathetic output together contribute to its anti-hypertensive effect. Both angiotensin and melatonin are synthesized in the brain. Angiotensin produced locally in central nervous system in nuclei involved in cardiovascular and fluid-electrolyte homeostasis interacts with other systems, such as sympathetic, vasopressinergic ones [6, 7]. Moreover, there is a local pineal RAS that modulates the synthesis of melatonin, which represents the main hormonal output of the pineal gland [8, 9].

In STZ-induced diabetic animals, melatonin was shown to decrease serum lipid oxidation [10] and protein glycosylation [11], as well as regulate the activity of antioxidant enzymes, improving the protection against the oxidative damage caused by diabetes [12–14].

Genetic manipulation of clock genes in transgenic mouse models has uncovered new functions of internal clocks in pathogenesis of cardiovascular diseases [15]. Recent evidence is suggesting that a disruption of central or peripheral clocks may contribute to the progression of cardiovascular diseases [16].



ISSN: 0975-8585

CONCLUSION

- In our study, significant reduction of melatonin levels is observed in hypertensives when compared with normotensives.
- The reduced melatonin level in hypertensives may lead to the risk of developing cardiovascular complication insisting prompt control of blood pressure.
- Our study is a small attempt to find a meaningful association of melatonin role in hypertension. The result is surely encouraging to undertake large scale studies to establish definitive role of melatonin.

REFERENCES

[1] Stehle JH, Saade A, Rawashdeh O, et al. J Pineal Res 2011; 51: 17–43
[2] Slominski RM, et al. Mol Cell Endocrinol 2012;351(2): 152–166.
[3] Tengattini S, Reiter RJ, Tan DX, Terron MP, Rodella LF and Rezzani R. J Pineal Res 2008;44(1): 16–25.
[4] Dominguez-Rodriguez A. Exp Opin Investigat Drugs 2012;21(11): 1593–1596.
[5] Dominguez-Rodriguez A, Abreu-Gonzalez P and Avanzas P. Front Biosci 2011;17(7): 2433–2441.
[6] M Bader, et al. J Mol Med 2001;79(2): 76–102.
[7] LA Campos, AS Couto, R Iliescu et al. Regulatory Peptides 2004;119(3): 177–182.
[8] O Baltatu, H Nishimura, S Hoffmann et al. Brain Res 1997;752(1-2): 269–278.
[9] Baltatu O, Lippoldt A, Hansson A, Ganten D and Bader M. Mol Brain Res 1998;54(2): 237–242.
[10] Armagan A, Uz E, Yilmaz HR, Soyupek S, Oksay T and Ozcelik N. Asian J Androl 2006;8(5): 595–600.
[11] Montilla P L, et al. J Pineal Res 1998;25(2): 94–100.
[12] Guven A, et al. Acta Histochemica 2006;108(2): 85–93.
[13] Winiarska K, Fraczyk T, Malinska D, Drozak J and Bryla J. J Pineal Res 2006;40(2): 168–176.
[14] Kanter M, Uysal H, Karaca T and Sagmanligil HO. Arch Toxicol 2006;80(6): 362–369.
[15] Takeda N and Maemura K. Hypertension Res 2010;33(7): 645–651.
[16] Takeda N and Maemura K. J Cardiol 2011;57(3): 249–256.