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ORIGINAL ARTICLE

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Effect of premenstrual stress on cardiovascular system and central nervous system

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OBJECTIVE(S): To study the effect of premenstrual stress on cardiovascular and central nervous system.

METHOD(S): Effect of premenstrual stress on cardiovascular system i.e. pulse rate, blood pressure and weight, and on central nervous system i.e. visual and auditory reaction time, was studied on 60 healthy female students between the age of 17 and 20 years during premenstrual and postmenstrual phase.

RESULTS: There was significant increase in pulse rate, blood pressure, weight, auditory reaction time (ART), and visual reaction time (VRT) during premenstrual period as compared to postmenstrual period.

CONCLUSION(S): These changes could be attributed to fluid and salt retention due to ovarian steroids and to exaggerated response to hormonal changes leading to decrease in the processing capability of central nervous system.

Key words : premenstrual stress, pulse rate, blood pressure, weight, auditory reaction time, visual reaction time

Introduction

Premenstrual syndrome is a major clinical entity affecting a large segment of female population [1]. It is the name given to a group of physical and emotional symptoms that some women experience on regular basis in relation to menstruation. The symptoms occur monthly generally within 7 to 14 days prior to menstruation. Symptoms may seem to worsen as menstruation approaches and subside at the onset or after several days of menstruation [2]. Certain behavioral and neurological symptoms occur in women especially during premenstrual phase. Headache, painful enlargement of breast, decreased ability to concentrate, nervous irritability, emotional instability, poor judgement, depression, tension, weight gain, and increased blood pressure have been reported during premenstrual phase, and are associated with salt and water retention.

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Reaction time means time taken by an individual to react to an external stimulus. It provides an indirect index of the processing capability of the central nervous system and also a simple means of determining sensorimotor performances [5]. The aim of the present study was to see, whether premenstrual stress affects sensory motor association and processing capability of central nervous system. We evaluated the significance and corelation of these changes, if any, to hormonal influences during that phase.

Methods

The study was carried out on 60 normal healthy female students 17 to 20 years of age. They were mainly medical students without any hearing or visual disorder. Their detailed menstrual history was noted and premenstrual and postmenstrual phase was calculated. Premenstrual phase was taken as 1 to 7 days prior to the onset of next menstruation and postmenstrual phase as 5^{th} to 10^{th} day of menstrual cycle. Their pulse rate (per minute), blood pressure (mm Hg), weight (kg), and auditory reaction time (ART) and visual reaction time (VRT) (seconds) were measured during both premenstrual and postmenstrual phases.

The instrument used to record reaction time was reaction

time apparatus supplied by Medicaid system RTM – 604. It is an electronic reaction time meter equipped with very sensitive quartz clock which can measure up to 1/10th of msec. Accuracy of the instrument is ± one digit. For auditory reaction time (ART) the stimulus used was a continuous beep of 1 KHz on speaker. For visual reaction time (VRT) soothing yellow, red and green lights incorporated on the instrument were used. Before measuring ART and VRT each subject was made familiar with the apparatus. All the subjects were righthanders and used their right hand to press the switch immediately after receiving visual or auditory stimulus. Three readings for each stimulus were noted by autodisplay after giving three practical trials and the lowest was taken as the reaction time.

The study was conducted in a quiet room and paired t test was applied to before and after readings of each parameter to find the P value.

Results

Table 1 shows the comparison of cardiovascular system parameters during premenstrual and postmenstrual phases. During premenstrual phase there was significant increase in pulse rate (P<0.001), in both systolic and diastolic blood pressure (P<0.001) and in weight (P<0.001).

Table 2 shows the comparison of ART and VRT during premenstrual and postmenstrual phases. Both ART and VRT were significantly increased (P<0.001) during premenstrual phase as compared to those in postmenstrual phase.

Table 1. Comparison of cardiovascular parameters during premenstrual and postmenstrual phases (n=60).

Parameters	Premenstrual Mean ± SD	Postmenstrual Mean ± SD	t	P value	Remarks
Pulse rate (beats/minutes)	82.48 ± 0.93	81.60 ± 0.97	4.65	<0.001	HS
Systolic BP (mm Hg)	107.16 ± 1.09	101.30 ± 1.22	27.53	<0.001	HS
Diastolic BP (mm/Hg)	74.36 ± 0.99	68.10 ± 1.00	34.36	<0.001	HS
Weight (kg)	49.35 ± 1.07	48.71 ± 1.07	3.27	<0.01	S

HS – Highly significant S – Significant

Table 2. Comparison of central nervous system parameters during premenstrual and postmenstrual phases (n=60).

Parameters	Premenstrual Mean ± SD	Postmenstrual Mean ± SD	t	P value	Remarks
Visual reaction time (seconds)	0.56 ± 0.014	0.54 ± 0.016	7.20	<0.001	HS
Auditory reaction time (seconds)	0.84 ± 0.040	0.74 ± 0.029	15.41	<0.001	HS

HS – Highly singificant

Discussion

There was a highly significant increase in pulse rate, and in both systolic and diastolic blood pressure during premenstrual phase as compared to those in postmenstrual phase. This could be explained on the basis of increased fluid and salt retention induced by ovarian steroids and higher sympathetic activity due to premenstrual stress. Changes in the autonomic functions may also be responsible for some of the symptoms produced through endorphins and have been held responsible for behavioral changes [4].

Increased blood pressure due to premenstrual stress is due to increase in peripheral resistance and is mediated by adrenocortical stimulation causing precapillary resistance. This could be due to increase in sympathetic nervous activity or to elevation of circulating catecholamines while other active hormones like renin-angiotensin - aldosterone system also might contribute. Rise in blood pressure due to stress leads to increased epinephrine secrection and this rise in blood pressure is important sympathoadrenal response to physiological stressful experience caused by premenstrual stress [6].

The increase in weight in premenstrual phase is due to

generalized fluid accumulation associated with sodium retention caused by changes in ovarian steroid secretions during latter half of the menstrual cycle. Patients suffering from severe premenstrual fluid accumulation sometimes gain even 4.5 kg in weight. Possible neuroendocrine causes of premenstrual edema are estrogen, progesterone, and renin-angiotensin - aldosterone axis. Estrogen increases the level of plasma renin substrate through enhanced hepatic synthesis, which leads to elevation of plasma angiotensin. This results in an elevated secretion and excretion of aldosterone. It shows that sodium retaining effect of estrogen is independent of its ability to augment aldosterone production. Progesterone is a partial agonist of aldosterone and blocks the effect of aldosterone at renal tubules [1]. During the luteal phase of menstrual cycle, there is no change in level of plasma renin substrate but there is rise in renin concentration leading to an increase in plasma renin activity with an increase in plasma aldosterone level and excretion [1].

The present study also shows the prolongation of both ART and VRT during premenstrual phase as compared to those during postmenstrual phase. Retention of water and sodium due to variation in sex steroid levels during menstrual cycle might influence the process of axonal conduction time and availability of neurotransmitter at synapses in auditory pathways. Changes in either of these two processes cause conduction time to vary during menstrual cycle.

Progesterone acts at the level of plasma membrane of selected cells by a nongenemic mechanism to inhibit the activation of adenylcyclase. Progesterone is metabolized by extraadrenal 21- hydroxylation to deoxycorticosterone, which acts by way of mineralocorticosteroid receptor [7].

Another metabolite formed by reduction of progesterone at C5 ? ?and C3 ? viz., 5 ? pregnan 3 ? - 01 – 20, acts in the brain as an anasthetic / anxiolytic agent by binding to gamma aminobutyric acid ($GABA_A$) receptor. GABA is a inhibitory neurotransmitter, an endogenously produced anxiolytic like compound. These metabolites are formed in women during premenstrual phase when progesterone excretion is high. Apparently the confirmation changes of $GABA_A$ receptor after anxiolytic steroid binding increase the affinity of GABA for this receptor. GABA favors influx of chloride ions into the cells. Increased chloride entry into brain cells serves to hyperpolarize the membrane and thereby inhibits neural transmission. This neural transmission inhibition affects sensorimotor association and processing capability of central nervous system [7].

Our finding that reaction time is significantly prolonged during the premenstrual phase could be attributed to modulation in neurotransmitter involved due to hormonal fluctuations affecting sensorimotor association and processing capability of central nervous system [5].

Conclusion

During premenstrual phase there is a significant increase in pulse rate, blood pressure, weight, auditory reaction time, and visual reaction time.

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