

Original Research Article


Seasonal variations in hematological and hemodynamic parameters

Kiran Singh¹, Navneet Kumar Kaushik^{2*}

¹Professor, Department of Physiology, Pt. B.D. Sharma, PGIMS, Rohtak, Haryana, India

²Demonstrator, Department of Physiology, Pt. B.D. Sharma, PGIMS, Rohtak, Haryana, India

*Corresponding author email: navneetkk24@gmail.com

| | | |
|---|--|--------------------------------------|
|  | International Archives of Integrated Medicine, Vol. 3, Issue 9, September, 2016. Copy right © 2016, IAIM, All Rights Reserved. Available online at http://iaimjournal.com/ | |
| | ISSN: 2394-0026 (P) | ISSN: 2394-0034 (O) |
| | Received on: 08-08-2016 | Accepted on: 21-08-2016 |
| | Source of support: Nil | Conflict of interest: None declared. |
| How to cite this article: Singh K, Kaushik NK. Seasonal variations in hematological and hemodynamic parameters. IAIM, 2016; 3(9): 54-60. | | |

Abstract

Background: Seasonal fluctuation in incidence of invasive pneumococcal disease, pulmonary embolism, deep vein thrombosis and coronary heart disease etc has been reported since long time. So, the present study was conducted to evaluate the seasonal variation in hematological and hemodynamic parameters, heart rate and blood pressure in the month of November, February and May.

Materials and methods: 15 male subjects in the age group of 18.5 ± 1 year were matched on haematological parameters, heart rate and blood pressure in three different seasons and the results were compared.

Results: Packed cell volume in winter (February), Erythrocyte Sedimentation Rate in summer (May) were increased ($P < 0.001$) as compared to basal level in November month with non significant variation in Hemoglobin concentration and Red Blood Cell count. Total Leukocyte count significantly increased ($p < 0.001$) in winter, while on differential and absolute leukocyte count Eosinophil, Basophil, Monocyte, Lymphocyte (%) were elevated ($p < 0.001$) and Neutrophil (%) decreased ($p < 0.001$) in summer. Total Protein concentration, serum Albumin level was higher in winter. Heart rate shows insignificant seasonal changes but blood pressure, both systolic and diastolic was lower ($p < 0.001$) in summer and rate pressure product (RPP) was higher ($p < 0.05$) in winter.

Conclusion: So, these findings indicate that variations in different parameters do occur to adapt to the environmental conditions which may be responsible for susceptibility to different diseases e.g. asthma and ischemic heart diseases in different seasons.

Key words

Blood pressure, Hematological parameters, Heart rate, Seasonal variation, Total protein concentration.

Introduction

In a tropical country like India there is wide variation in environmental temperature during summer (up to 40°C) and winter months (up to 0°C) [1, 2]. Variation in thromboembolic diseases have been noted in different seasons [3]. The epidemiology of invasive disease due to streptococcus pneumoniae exhibits a seasonal fluctuation with a peak incidence during winter months [4]. Significant changes in lipid levels occur seasonally and this variation does not appear to be associated with changes in diet, activity or gender [5-7]. There is growing body of evidence that acute myocardial infarction (AMI) and coronary heart disease (CHD) tend to exhibit a seasonal pattern with peak incidence in winter months [7]. However, there is paucity of data showing seasonal fluctuations in hematological parameters. The liquid connective tissue i.e., blood is having varied compositional heterogeneity and functional diversity. So it was felt necessary to investigate the seasonal changes in hematological parameters which may be responsible for higher incidence of different diseases in different seasons.

Therefore, the present study was planned to note the variations in hematological parameters, total and differential cell count, Hemoglobin (Hb) concentration, erythrocyte sedimentation rate (ESR), total serum protein and albumin levels, heart rate (HR) and blood pressure (BP) and rate pressure product (RPP) in different seasons in same subjects.

Materials and methods

The present study was carried out in male medical students (n=15) of first professional (MBBS) with the mean age 18.5 ± 1.0 years, average height 175.4 ± 2.9 cm, average weight 68.6 ± 2.8 kg, body surface area (BSA) 1.83 ± 0.05 m² after taking written consent.

Parameters were recorded in November (representing comfortable temperature, so control month), February (representing cold temperature), and May (representing hot

temperature). Study was performed on the same subjects, same gender and on the same time of the day.

Heart rate and blood pressure of the subjects were recorded by palpation of radial artery and sphygmomanometer using auscultation method in supine posture from right upper limb, respectively. RPP was calculated as systolic BP X HR /100 and value thus obtained was expressed as mmHg. Beats/min [8]. Various blood samples (about 8-10 ml) were drawn without stasis around 10 a.m. from each subject and put in pre-heparinized container and were analyzed for hematocrit parameters. The counting of total red blood cells (RBC), total white blood cells (WBC), Hb estimation, measurement of packed cell volume (PCV) were done as described by Dacie and Lewis [9]. Erythrocyte sedimentation rate (ESR) was estimated by Westergren's method. The differential leukocyte count (DLC) and absolute leukocyte count were carried out by staining the blood film with Leishman's stain and washed with buffered distilled water of pH 6.8. Total plasma protein concentration (TP) and serum albumin levels were analyzed in Biochemistry Department of the institute.

Statistical analysis

The data was analyzed statistically using paired student t-test and p-values were obtained. The statistical analysis was carried out using SPSS PC software version 13.0.

P value > 0.05 was considered as not significant.

P value < 0.05 was considered as significant.

P value < 0.01 was considered as highly significant.

P value < 0.001 was considered as very highly significant.

Results

Hemoglobin (Hb) concentration and RBC count did not show variations in the months of November, February and May. Packed cell volume (PCV) and total leukocyte count (TLC) were increased (P < 0.001) in winter (month of

February) compared to basal level in November month, while Erythrocyte sedimentation rate (ESR) was increased ($P < 0.001$) in summer (month of May) (**Table - 1** and **Table - 2**).

On differential leukocyte count (DLC) and absolute count Neutrophil (N%), Eosinophil (E%), Basophil (B%), Monocyte (M%) and Lymphocyte (L%) count were raised

during summer in comparison to control month (**Table - 2**). Total plasma protein concentration and serum albumin were more during winter compared to November month (**Table - 3**). There was no change in HR in summer or winter seasons, while blood pressure, both systolic and diastolic was reduced significantly ($P < 0.001$) in summer versus November month. RPP was increased significantly ($p < 0.001$) (**Table - 3**).

Table - 1: Seasonal variation in hematological parameters (Mean \pm SD).

| Month and Date | Hb concentration (g%) | PCV (%) | ESR (mm in 1 st hour) | RBC count (million/ cu mm) |
|------------------|-----------------------|-------------------------------------|---------------------------------------|----------------------------|
| 27.11.98 | 12.5 \pm 1.8 | 49.75 \pm 8.4 | 3.6 \pm 1.4 | 4.13 \pm 0.92 |
| 5.2.99 (Winter) | 12.9 \pm 1.89 | 52.9 \pm 5.0*** | 5 \pm 2.06 | 4.37 \pm 0.55 |
| 10.5.99 (Summer) | 12.8 \pm 1.02 | 48.75 \pm 5.82 | 13.33 \pm 9.68*** | 4.63 \pm 0.57 |

*** - very highly significant

Hb – Haemoglobin, PCV – Packed cell volume, ESR – Erythrocyte sedimentation rate

Table - 2: Seasonal variation in hematological parameters (Mean \pm SD).

| Month and Date | TLC (/cu mm of blood) | DLC (%) | | | | |
|------------------|-----------------------|---------------------|------------------|-------------------|-------------------|--------------------|
| | | N | E | B | M | L |
| 27.11.98 | 8,455.55 \pm 2547.3 | 49.9 \pm 8.03 | 9.2 \pm 0.03 | 9.9 \pm 6.42 | 5.0 \pm 0.1 | 30.85 \pm 12.32 |
| 5.2.99 (Winter) | 7,220 \pm 1913.0*** | 37.2 \pm 18.78*** | 8.75 \pm 2.94 | 5 \pm 3.55*** | 9 \pm 2.8*** | 39.75 \pm 6.1*** |
| 10.5.99 (Summer) | 7,755.5 \pm 1257.0* | 34.4 \pm 10.6*** | 10.25 \pm 6.72 | 13.3 \pm 4.7*** | 18.3 \pm 6.3*** | 40 \pm 17.3*** |

*highly significant, ***very highly significant

TLC – Total Leucocyte count, DLC – Differential Leucocyte count

N – Neutrophil, E – Eosinophil, B – Basophil, M – Monocyte, L – Lymphocyte

Table - 3: Seasonal variation in total protein concentration, serum albumin level and hemodynamic parameters (Mean \pm SD).

| Month and Date | Total Protein Concentration (g%) | Serum Albumin Level (g%) | HR (Beats/ min) | BP (mm/ Hg) | | RPP (mm Hg. bpm) |
|------------------|----------------------------------|--------------------------|-------------------|------------------|-----------------|------------------|
| | | | | Systolic | Diastolic | |
| 27.11.98 | 6.2 \pm 0.25 | 3.05 \pm 0.26 | 72.0 \pm 6.02 | 120 \pm 9.8 | 72 \pm 7.9 | 85.4 \pm 3.5 |
| 5.2.99 (Winter) | 7.16 \pm 0.16*** | 3.9 \pm 0.12* | 75.75 \pm 10.03 | 122 \pm 12.5 | 76 \pm 9.4 | 91.35 \pm 4.8* |
| 10.5.99 (Summer) | 6.47 \pm 0.20 | 2.98 \pm 0.93 | 76.25 \pm 9.28 | 111 \pm 8.3*** | 60 \pm 7.6*** | 83.58 \pm 2.73 |

*highly significant, ***very highly significant

HR – Heart rate, BP – Blood pressure, RPP – Rate pressure product

Discussion

Utilizing the same test subjects and one gender during whole of the study period helped to ensure a homogenous test group for seasonal comparisons. Circannual variability of thromboembolic diseases and pulmonary embolism has been reported in literature [3]. It is stated that seasonal changes in serum lipid and cholesterol levels with peak level in winter and trough in summer may play important role in winter excess of cardiovascular deaths [6, 10]. There is no variation in Hb concentration and RBC count in summer or winter in the present study as seasonal changes in erythropoietin level is not reported while increase in RBC count in cold temperature is demonstrated by Kneating, et al. [6, 11]. Similarly it is documented that Hemoglobin I_c (Hb I_c) is 0.4% lower in summer than in winter [6]. However we found PCV was increased in winter season. An increase in hematocrit following different types of stress has been reported [12]. It is also suggested that lower water intake during wet season would lead to hemoconcentration. In another study highest Hb, PCV and RBC value were obtained in winter than summer. During summer high ambient temperature (HAT) increases body temperature, respiration, respiratory water loss, and O₂ consumption in birds. Increased O₂ consumption raises pO₂ in blood, which reduces erythropoiesis and thus RBC count. While in winter increased O₂ demand increases BMR, increases food intake which results in increased erythropoiesis. Raised ESR in summer in this study can be explained by decreased viscosity with raised temperature [13].

Excess leukocyte count in winter may be stress (cold) related, and thus raised glucocorticoids may be responsible for increased count in winter, which may be correlated with higher incidence of infections in winter months [4, 14-16]. Although exact cause of such variation is not known but it may be due to alteration in environmental factors and host airway mucosa, variation in light dark exposure, less clearance of encapsulated organisms by respiratory mucosa. All these factors may be responsible for peak incidence of

pneumococcal disease and infections in winter month [4].

On Differential (%) and absolute leukocyte count Eosinophil, Basophil, Monocyte and Lymphocyte shows an increased pattern in summer while Neutrophils show down fall in those months. It has been clinically observed that asthma and allergic disorders are common in summer that may attribute to increased count of these cells in this duration [17]. Total protein concentration, serum albumin level show increment in winter month in present study. It is known that platelets, RBC count and blood viscosity increase with cold temperature [11]. Fibrinogen concentration also shows seasonal variation as its concentration is higher (23%) in colder part of the year [18]. Reason for raised protein concentration is not understood or it may be related to lower water intake with decrease temperature and this could be the one reason of increased blood viscosity and relatively increased hypercoagulability of blood and may explain higher incidence of thromboembolic phenomena and coronary heart disease in winter months [11].

HR in present study does not appear to be affected by seasons. In contrast, increased HR is demonstrated by Kumar in winter [1]. They explained it by raised level of catecholamines in winter [19]. Arterial blood pressure both systolic and diastolic is found to be reduced in summer in current study. It is in collaboration with other scientists [1, 2].

Reasons for seasonal changes in BP are complex, involving both long term regulating factors and acute responses to environmental temperature. Reduction in BP is in part because of decreased peripheral resistance in response to warm environmental temperature in summer and vice versa [1, 2, 20]. Similar observations were reported by Rose and Brennan in mild hypertension [21, 22]. In contrast, some workers reported rise in BP in winter [1, 2]. They explain it by increased sympathetic reflex triggered by skin cooling and raised level of catecholamines resulting in vasoconstriction [19, 23]. In present

study, although BP is not found to be elevated in winter (though found to be insignificantly higher than basal level), but RPP is raised in winter suggestive of increased sympathetic activity [8]. Total protein concentration and serum albumin level are also found to be more in present study in winter. Thus increased viscosity may be the contributing factor for raising BP. It is reported that elevated BP is associated with increased WBC count (6700 cells/cu.mm) in patients of coronary syndrome [24-28]. In this study although subjects are young normotensives, leucocytes are within normal limits in winter but are significantly higher ($p < 0.001$) than base line WBC count.

Recent evidences suggest that neutrophils are leucocytes, most strongly associated with coronary risk [25, 27, 29]. It is interesting to note that on differential and absolute count of WBCs, neutrophils are found to be more in winter in this study. They are thought to be associated with increased incidence of ischemic heart disease [25, 30]. Finally stress induces not only sympathetic nervous system but also vascular reactivity to catecholamines through glucocorticoids, thus increases leukocyte count [16, 25]. Increased leukocyte count may cause spasm, microvascular injuries, hypercoagulable state and endothelial dysfunction, helping in raising the BP [24, 25, 29, 32]. It is also suggested that cold induced dehydration leads to decreased tissue perfusion caused by insufficient blood volume due to inappropriate secretion of antidiuretic hormone (ADH) and angiotensin II. RPP is regarded as an index of myocardial O_2 consumption and work done and indicates load on the heart [8]. Thus raised BP along with increased RPP may contribute to more incidence of ischemic heart disease in winter [33].

Conclusion

Present study gives some positive indications of disturbances in hematological and hemodynamic parameters in different seasons. Our study puts forward that antihypertensive and anticoagulant therapy must be used carefully in winter months

(dose has to be increased) to avoid any ill effects of seasons. And further studies are needed to explore the basic mechanisms behind these variations.

References

1. Kumar H. Blood pressure has a tendency to be higher during winter season. *Ind Heart J.*, 1995; 295: 631.
2. Sharma BK, Sagar S, Sood GK, Verma S, Kalra OP. Seasonal variations of arterial blood pressure in normotensive and essential hypertensives. *Ind Heart J.*, 1990; 42(1): 66- 73.
3. Stein PD, Kayali F, Olson RE. Analysis of occurrence of venous thrombo-embolic disease in the four seasons. *Am J Cardiol.*, 2004; 93: 511-3.
4. Talbot TR, Poehling KA, Hartert TV, Arbogast PG, Halasa NB, et al. Seasonality of invasive pneumococcal disease: Temporal relation to documented influenza and respiratory syncytial viral circulation. *Am J Medicine*, 2005; 118(3): 283-91.
5. Tung P, Wiviott SD, Cannon CP, Murphy SA, McCabe CH, et al. Seasonal variation in lipids in patients following acute coronary syndrome on fixed doses of pravastatin (40 mg) or Atorvastatin (80) mg from (From the Pravastatin or Atorvastatin evaluation and infection therapy-Thrombolysis in myocardial infarction [prove IT-TIMI22] study. *Am J Cardiol.*, 2009; 103: 1056-60.
6. Donahoo WT, Eckel RH. Physiological mechanisms for the seasonal changes in serum cholesterol level. *Arch Intern Med.*, 2004; 164: 2506.
7. Senthil velour M, Purushothaman P, Krishna R, Prakash ES. Gender differences in vasoconstrictor reserve. *Ind J Physiol Pharmacol.*, 2006; 50(3): 316-8.
8. Gabel FL, Nordstrom LA, Nelson RR, Jorgenson CR, Wang Y. The rate

- pressure product as an index of oxygen consumption during exercise in patients with angina pectoris. *Circulation*, 1978; 57: 549-56.
9. Dacie JV, Lewis SM. In *Practical Hematology*, 7th edition, London: Churchill Livingstone; 1991, p. 1-175.
 10. Ockene IS, Chiriboga DE, Stanek EJ III, Harmatz MG, Nicolosi R, et al. Seasonal variation in serum cholesterol level: treatment implications and possible mechanism. *Arch Intern Med.*, 2004; 164: 863-70.
 11. Keatinge WR, Coleshaw RK, Cotter F, Mattock M, Murphy M, et al. Increase in platelet and red cell counts, blood viscosity and arterial pressure during mild surface cooling. Factors in mortality from coronary and cerebral thrombosis in winter. *BMJ*, 1984; 289: 1405-8.
 12. Ivanov AP. Changes in certain parameters of blood rheology in simulated weightlessness experiments. *Kosn Biol Aviakosm Med.*, 1983; 17: 25-30.
 13. Jain AK. In *manual of Practical Physiology*. 1st edition, H.P.: Avichal Publishing Company; 2000, p. 54.
 14. Sundaresen G, Suthanthirarajan V, Namasivayam A. Certain immunological parameters in subacute stress. *Ind J Physiol Pharmacol.*, 1990; 34: 1492-3.
 15. Crokard A, Boylan MT, Droogan AG. Methyl prednisolone induced neutrophil leucocytosis- down modulation of neutrophil L selection and mac -1 expression and induction of granulocyte colony stimulating factor. *Int J Clin Lab Res.*, 1998; 28: 110-5.
 16. Ganong WF. Review of medical physiology. The adrenal medulla. 22nd edition, San Francisco: McGraw Hill; 2005, p. 370.
 17. Mc Fadden Jr. ER. Seasonal concentrations of airborne Ags such as pollen can rise high enough to result in epidemics of asthma. In: Kasper, Fauci B, Hauser, Lango, Jameson, editors. *Harrison's principles of internal medicine*, 16th edition, McGraw Hill; 2005, p.1510.
 18. Stout RW, Crawford V. Seasonal variation in fibrinogen concentrations among elderly people. *Lancet*, 1991; 338: 9-13.
 19. Kunes J, Trembay J, Bellavance F, Hamet P. Influence of environmental temperature on the blood pressure of hypertensive patients. *Am J Hypertens.*, 1991; 4: 422-6.
 20. Bonch EK, Harari G, Green MS. Seasonal changes in 24 hour blood pressure and heart rate is greater among smokers than nonsmokers. *Hypertension*, 1997; 30: 436-41.
 21. Rose G. Seasonal variation in blood pressure in man. *Nature*, 1961; 189: 235.
 22. Brennan PJ, Greenberg G, Miall WE, Thompson SG. Seasonal variation in arterial pressure. *BMJ*, 1982; 285: 919-23.
 23. Montagu KA. Seasonal changes of the catecholamine compounds present in rat tissue. *J Biochem.*, 1959; 71: 91-3.
 24. Yen MH, Bhatt DL, Chew DP, Harrington RA, Newby LK, et al. Association between admission white blood cell count and one year mortality in patients with acute coronary syndromes. *Am J Med.*, 2003; 115: 318-21.
 25. Ernst E, Hammerschmidt DE, Bagge U, Matrai A, Dormandy JA. Leucocyte and risk of ischaemic disease. *JAMA*, 1987; 257(17): 2318-24.
 26. Cushman M. Leucocyte count in vascular risk prediction. *Arch Intern Med.*, 2005; 165: 487-8.
 27. Shoenfeld PJ. Leucopenia and low incidence of myocardial infarction. *Lancet*, 1981; 1: 1806.
 28. Gokulakrishnan K, Deepa R, Sampathkumar R, Balasubramanyam M, Mohan V. Association of leucocyte count and hsCRP with metabolic

- abnormalities in subjects with normal glucose tolerance curves. JAPI, 2009; 57: 27-32.
29. Wheeler JG, Mussolini ME, Gillum RF, Danesh J. Association between differential leucocyte count and incident coronary heart disease. Eur Heart J., 2004; 25: 1287-92.
 30. Olivares R, Ducimetiere P, Claude J. Monocyte count: a risk factor for coronary heart disease? Am J Epidemiol., 1993; 137: 49-53.
 31. Margolis KL, Manson JE, Greenland P, Rodabough RJ, Bray PF, et al. Leukocyte count as a predictor of cardiovascular events and mortality in postmenopausal women. Arch Intern Med., 2005; 165: 500-8.
 32. Mukamal KJ, Wellenius GA, Mittlemann MA. Hematological parameters, Atherosclerotic progression and prognosis in patients with previous coronary artery bypass grafting (from the post CABG trial). AM J Cardiol., 2009; 103: 328-32.
 33. Green MS, Harari G, Kristol BE. Excess winter mortality from ischaemic heart disease and stroke during colder and warmer years in Israel: an evaluation and review of role of environmental temperature. Eur J Publ Health, 1994; 4: 3-11.