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A Study of Stroke Index and Cardiac Index in Severe Anemia

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KEYWORDS

Chronic anemia, left ventricle, stroke index, cardiac index, echocardiography.

A B S T R A C T

Anemia is one of the common cause of morbidity, a factor limiting physical activity, responsible for a poor quality of life, and a predictor of unfavourable outcomes. It affects an estimated 50% of the population. Clinical studies suggest that correction of anemia causes improvement of LV function, improves exercise capacity and decrease adverse outcomes in concomitant heart disease. Hence the present study was undertaken to evaluate stoke index and cardiac index in severe anemia by echocardiography. This study was conducted in Al-Ameen Medical College and Government district Hospital, Bijapur. 31 anemic patients (aged 18-40 yrs) with Hb ≤ 7 gm% and equal no. of age and gender matched normal subjects were selected. All anemic patients were subjected for hemoglobin estimation and M mode 2D Echocardiography. Echocardiographic parameters SV, CO & SI, CI were studied. Statistically significant variations were found in parameters SV, SI, CO, CI in anemic patients compared to controls and correlated negatively with HB levels in anemic patients. The findings of increased SV, SI, CO CI in anemic patients compared to controls may be as a consequence of hyperdynamic circulatory state leading to vascular and cardiac changes. Changes are mainly due to increased preload, decreased afterload. The increased preload condition found in anemic patients may be attributed to Frank Starling mechanism that operates in chronic anemia.

Introduction

Anemia is one of the common cause of morbidity, a factor limiting physical activity, responsible for a poor quality of life, and a predictor of unfavorable outcomes. It affects an estimated 50% of the population. Anemia has a negative

impact on physical work capacity in different age groups as measured by changes in maximum oxygen uptake and other metabolic parameters. Most of the cardiac dysfunction is due to abnormalities of left heart (Kapoor et al., 1999).

Studies conducted to assess LV function by non invasive techniques show LV dysfunction in chronic severe anemia. The noninvasive technique of ultrasonic echocardiography has been developed to provide quantitative information about ventricular performance by derivation of ventricular volumes. Clinical studies suggest that correction of anemia causes improvement of LV function, improves exercise capacity and decrease adverse outcomes in concomitant heart disease. Hence the present study was undertaken to assess the status of left ventricular function in severe anemia patients for detection of cardiac functional impairment, by evaluating stoke index and cardiac index by echocardiography.

Materials and Methods

The present study was conducted in the department of physiology Al-Ameen Medical College, Bijapur. Thirty one (31) patients (20 females, 11 males) with severe anemia (Hb ≤ 7gm%), between 18yrs to 40 yrs age visiting Al-Ameen medical college hospital, Bijapur and District Hospital Bijapur were selected. Equal number of age & gender matched individuals were controls.

Patients presenting with history of acute anemia and other causes of hyperdymanic circulatory state were excluded from the study. All anemic patients underwent history taking and a thorough clinical examination. Physical parameters viz Height, Weight were recorded.

Body surface area (BSA) in Sq. mts (m^2) was calculated by Duboi's nomogram. Their hemoglobin levels were estimated. Transthoracic Echocardiography was done using PHILIPS Envisor C (model no MCMD02AA) diagnostic Ultrasound System using 3.5 MHz transducer, probe (PA 4- 2) by physician experienced in Echocardiography. Measurements were made according to the recommendations of the American Society of Echocardiography (ASE) at end diastole and end systole (Stritzke et al., 2007).

Echocardiographic parameters studied were: Stroke Volume (SV). Cardiac output (CO) was calculated as CO = HR x SV. Stroke index and Cardiac index were calculated.

The stroke volume (SV) was indexed to body surface area.
SI = Stroke volume / Body surface area
The cardiac output (CO) was then indexed to body surface area.
CI= Cardiac output / Body surface area

Student's unpaired't' test was performed (using Graph pad Prism 5 statistical software) to analyze the echocardiographic changes between the control group and anemic patients. Correlation analysis was applied to correlate hemoglobin levels and other parameters. A 'p' value <0.05 was considered statistically significant.

Result and Discussion

Thirty one patients with severe anemia having hemoglobin concentration less than 7 gm/dl, between 18 and 40 yrs of age and equal no. of age and gender matched normal subjects were selected for this study. Student's unpaired't' test was used

to analyze the variation in the parameters in anemic patients and controls. Correlation analysis was done in patients to correlate Hb with other parameters.

Table No. 1 shows the mean ± SEM of age, Ht, Wt, BSA in anemic patients and controls. The mean weight of anemic patients was found to be less than that of controls, with a statistically significant difference; ($t = 4.277$, $p < 0.0001$). The mean value of BSA anemic patients was less as compared to that of controls. There was a statistically significant difference; ($t = 3.451$, $p < 0.01$).

Table No. 2 Shows the mean ± SEM of Hb, PR in anemic patients and controls. The mean value of pulse rate of anemic patients was higher as compared to that of controls with a statistically significant difference; ($t = 7.01$, $p < 0.0001$).

The mean ± SEM of hemoglobin in anemic patients was 4.958 ± 0.22 gm/dl; in controls 13.46 ± 0.13 gm/dl.

Table No.3 shows the mean ± SEM of SV, CO, SI, CI in anemic patients and controls.
In anemic patients the mean of stroke volume was higher as compared to that of controls. There was a difference of 14.23 ml between the means. This difference was found to be statistically highly significant; ($t=4.22$, $p < 0.0001$) (Fig.1)

The mean of stroke index in anemic patients was higher when compared to that of controls. This difference was statistically highly significant; ($t=5.79$, $p < 0.0001$) (Fig.2)

In anemic patients the mean of CO was higher as compared to that of controls with a statistically significant difference ($t=5.55$, $p < 0.0001$) (Fig.3). The mean of cardiac index in anemic patients was found to be much higher with a difference of 2.09 $L/min/m^2$ when compared with that of controls. This difference was found to be statistically highly significant; ($t = 7.21$, $p < 0.0001$) (Fig.4).

Table No.4 shows the correlation analysis of Hb with SV, CO, SI, CI in anemic patients. Anemic patients had a statistically significant negative correlation between stroke volume and hemoglobin levels; ($r = -0.571$, $p < 0.001$). CO correlated negatively with hemoglobin levels which was statistically significant ($r = -0.566$, $p = 0.001$). SI and CI also negatively correlated hemoglobin levels, with a statistical significance; ($r = -0.669$, $p < 0.0001$) & ($r = -0.641$, $p < 0.001$) respectively.

In the present study the means of Wt, BSA of anemic patients were found to be lower than that of controls. This difference is probably due to poor nutritional status of anemic patients. Pulse rate was significantly faster in anemic patients when compared with controls (Bahl et al, 1992). Singh and Singh (1994) conducted a study and observed that there was an elevation of heart rate in anemics. Our finding of an elevated pulse rate in anemic patients suggest the possibility of increased activity of sympathetic part of autonomic nervous system due to reduced oxygen delivery to the tissues (Aessopos et al., 2004).

The SI of anemic patients was 87.1 ± 38 ml/m^2 and of controls 49.8 ± 20.8 ml/m^2 in a study wherein 31 patients with chronic severe anemia of more than 3 months' duration and no underlying heart disease were studied by means of M-mode, 2-dimensional, and Doppler echocardiography; an equal number of

Table.1 Shows the mean ± SEM of Age, Ht, Wt, BSA in anemic patients and controls

PARAMETER	ANEMIC PATIENTS (n = 31) MEAN±SEM	CONTROLS (n = 31) MEAN±SEM	p value
AGE (yrs)	31.8 ± 1.33	30.3 ± 1.04	p > 0.05
Ht (cms)	155.1 ± 1.61	157.03 ± 0.83	p > 0.05
Wt (kgs)	45.94 ± 1.04	52.9 ± 1.25	p < 0.0001***
BSA (m^2)	1.014 ± 0.02	1.084 ± 0.01	p < 0.01**

Table.2 Shows the mean ± SEM of Hb, PR in anemic patients and controls

PARAMETER	ANEMIC PATIENTS (n = 31) MEAN±SEM	CONTROLS (n = 31) MEAN±SEM	p value
Hb (gm/dl)	4.958 ± 0.22	13.46 ± 0.13	p < 0.0001***
PR (beats/min)	88.06 ± 1.08	78.83 ± 0.74	p < 0.0001***

Table.3 Shows the mean ± SEM of SV, CO, SI, CI in anemic patients and controls

PARAMETER	ANEMIC PATIENTS (n = 31) MEAN±SEM	CONTROLS (n = 31) MEAN±SEM	p value
SV (ml)	65.25 ± 2.82	50.93 ± 1.88	p < 0.0001***
CO (Lts/ min)	5.8 ± 0.276	4.01 ± 0.16	p < 0.0001***
SI (ml/m^2)	63.98 ± 2.31	47.11 ± 1.77	p < 0.0001***
CI ($L/min/m^2$)	5.72 ± 0.25	3.63 ± 0.14	p < 0.0001***

Table.4 Shows the correlation analysis of Hb with SV, CO, SI, CI in anemic patients

SL. no	PARAMETER	ANEMIC PATIENTS (N=31)		
		r	p	S/NS
1	Hb vs SV	-0.571	<0.001	S***
2	Hb vs CO	-0.566	0.001	S***
3	Hb vs SI	-0.669	<0.0001	S***
4	Hb vs CI	-0.641	0.0001	S***

Figure.1 Mean of SV (ml) of anemic patients and controls

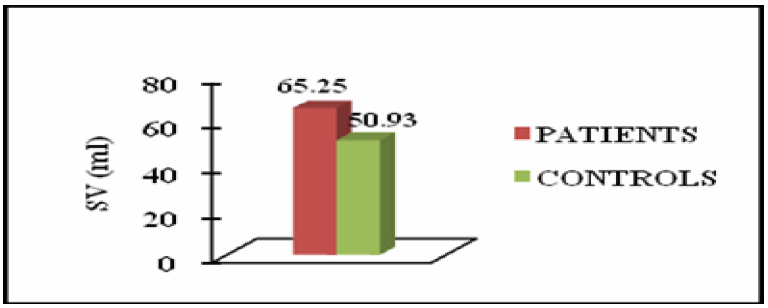


Figure.2 Mean of SI (ml/m^2) of anemic patients and controls

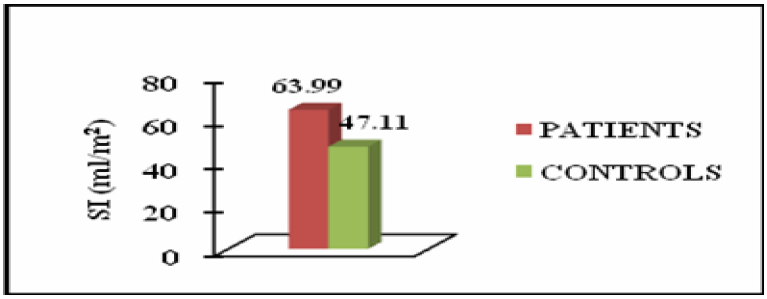


Figure.3 Mean of CO (L/min) of anemic patients and controls

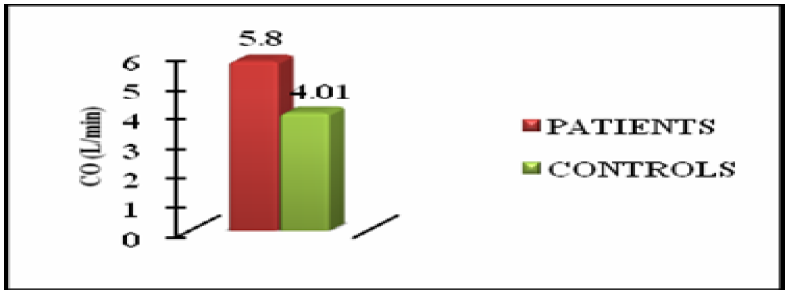
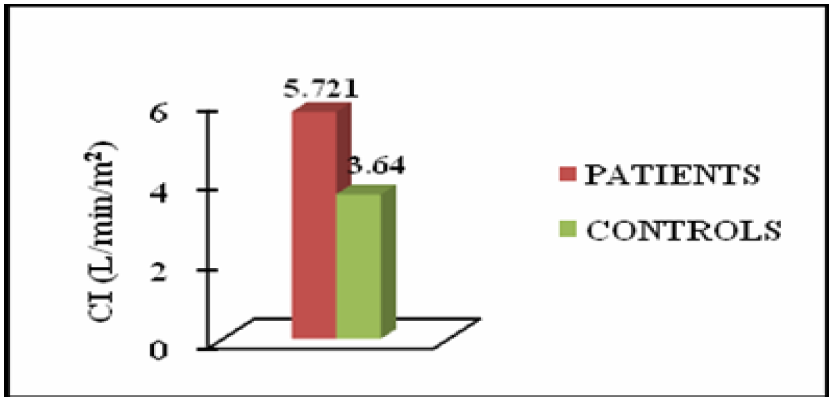


Figure.4 Mean of CI (L/min/m^2) of anemic patients and controls



normal control subjects were also studied. Anemic patients were found to have significantly higher stroke index as compared to controls (Bahl et al, 1992).

In a study by Fernando Florenzano et al. 17 patients with chronic anemia (hematocrit range 17 to 30%) and 17 control subjects, matched by age and sex were studied. They found that in patients with anemia, stroke volume increased by 43% ($p < 0.01$). Riku Hayashi et al. (1999) showed a decrease in stroke index of anemic patients after iron therapy. These patients were studied before and after iron therapy by echocardiography. Stroke index was found to be higher in anemic patients with tachycardia when compared with healthy controls in a study by Panwar et al.(1991). Takahashi et al. (1990), studied the changes in hemodynamic parameters before and after treatment in 14 subjects. These results were compared with those of normal subjects. In anemic patients, LV stroke volume (SV) increased.

Stritzke J et al. (2007) studied 687 women and 648 men, aged 25-74 years, participating in the third population-based MONICA Augsburg study. They observed that the stroke volume correlated inversely with haematocrit levels.

The findings of our study are in accordance with other studies. The increased SV and SI in anemic patients may be attributed towards hyperdynamic state in chronic anemia which causes an increased LV preload and lower afterload due to reduced systemic vascular resistance. The decreased after load may be due to peripheral vasodilatation related to anemia (Hayashi et al., 1999). The increase in SV and SI in our study may be due to the Frank Starling mechanism that operates in the hyper dynamic state of chronic anemia (Kapoor, et al., 1999). As the preload increases the left ventricular filling increases leading to increased LV end-diastolic volume in order to maintain a high stroke work (Metivier et al., 2000). Stroke volume can also be increased in chronic anemia even when heart rate is rapid (Heriburt Schunkert, Hans W. Hense, 2001).

SV and SI were correlated negatively with hemoglobin levels. An increase in SV is an important determinant of high cardiac output in chronic anemia, because it parallels the severity of anemia (Takahashi et al., 1999).

Significantly higher cardiac index was found in anemic patients (7.3 ± 3.0 $L/min/m^2$) when compared with normal subjects (3.8 ± 1.6 $L/min/m^2$) (VK Bahl et al, 1992).[3] In a study it was observed that the cardiac index of thalassemia patients was much higher ($p < 0.0001$) than the controls (Bosi et al. 2003). Schafer GE et al. in 2002 conducted a study in which patients were divided into 2 groups: in patients with severe anemia (Hb 5.7 ± 0.6 g/dl; n = 7), cardiac index was higher (4.8 ± 0.4 $L/min/m^2$ < 0.01) compared with the other group (Hb 9.8 +/- 1.7 g/dl; n = 24; CI 3.9 ± 1.1 $L/min/m^2$). It was found that the increase of cardiac index caused by anemia correlated with increased stroke volume and heart rate and lowered pulmonary and peripheral resistance. Patients with severe anemia showed a tendency to an impaired cardiac index below Hb < 5-6 gm/dl. (Schäfer et al., 2002).

Higher cardiac index was observed in patients with sickle β thalassaemia as compared to controls when they were examined by M mode 2 dimensional echocardiography (I Moyssakis et al. 2005). Cardiac index was found to be higher in anemic patients with tachycardia when compared with healthy controls (RB

Panwar et al.1991). In a study, 30 patients with iron deficiency anemia were studied by pulsed Doppler echocardiography. It was found that the cardiac index decreased in patients with IDA after therapy when compared with before therapy (Riku Hayashi et al. 1999).

Our finding of an increased CI in anemic patients is in agreement with other studies mentioned above. None of the patients in our study suffered from cardiac failure indicating well compensated state. The increased CI in anemic patients is probably due to reduced oxygen supply to the tissues found in chronic anemia that causes hemodynamic mechanisms to operate to compensate for anemia. Increased cardiac output is the main hemodynamic factor mediated by lower afterload, increased preload and positive chronotropic effects.
Decreased afterload is due to vasodilatation and reduced vascular resistance as a consequence of lower blood viscosity, hypoxia induced vasodilatation, and enhanced nitric oxide activity. The increased volume load is a reflection of the Frank–Starling mechanism and an increase in heart rate. In the present study probably the increased heart rate and stroke index resulted in an increased cardiac output. This increased cardiac output has mainly contributed to the high output state of chronic anemia.

Conclusion

The findings of our study of raised SV, CO, SI, CI in anemic patients as compared to controls may be as a consequence of hyperdynamic circulatory state that operates in severe anemia. This leads to vascular and cardiac changes. The cardiac changes are mainly due to increased preload, decreased afterload and positive chronotropic effects. The increased volume load is a reflection of the Frank–Starling mechanism that is operating in hyperdynamic state of chronic anemia. The increased heart rate and stroke index resulted in an increased cardiac output. This increased cardiac output has mainly contributed to the high output state of chronic anemia. Absence of cardiac failure may be due to well compensated state.

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