

Correlation Between Haemoglobin Level and Electrocardiographic (ECG) Findings in Anaemia: A Cross-Sectional Study

SHASHIKALA GV¹, SHASHIDHAR PK², ANITA HERUR³, SUREKHARANI CHINAGUDI⁴, SHAILAJA S PATIL⁵, ROOPA B ANKAD⁶, SUKANYA V BADAMI⁷

ABSTRACT

Background: Anaemia affects the body by decreased oxygen (O₂) carrying capacity of the blood. There is growing evidence that anaemia contributes to cardiac disease and death. It causes O₂ supply – demand myocardial mismatch causing myocardial ischemia. There is diversity of opinion available in literature on reports of electrocardiographic (ECG) changes in anaemia.

Aim: To study the ECG changes in anemic population and to correlate ECG changes seen with increasing severity of anaemia.

Materials and Methods: In hundred anemic adults, haemoglobin level and resting ECG were recorded. They were grouped according to haemoglobin level. ECG findings and varying severity of haemoglobin (Hb) level of each group were correlated using Pearson's co-relation co-efficient and association was calculated using Chi-square test.

Results: ECG changes in patients with Hb level of 0-5gm% showed ST segment depression in 50-75%, T wave changes in 29-50% and Left Ventricular Hypertrophy (LVH) in 25-30% of patients. Less percentage of patients with 5-7gm% Hb showed such changes, and patients with 7-8gm% Hb, showed no changes.

As the Hb level decreased there was more percentage of patients having tachycardia and ECG changes. There was a strong negative correlation between Hb level and tachycardia and ECG changes.

Conclusion: Diagnosing anaemia in critical care can be supported by ECG changes like ST depression, T wave changes, with/without associated QRS abnormalities to avoid misdiagnosis and also as dramatic clinical and ECG recovery can be achieved with anaemia correction.

Keywords: Anaemia, Electrocardiographic changes, ST depression, Cardiac changes

INTRODUCTION

Anaemia is the most common disease and even more so in a tropical country like India. Although, the prevalence of anaemia is estimated at 9 per cent in countries with high development, in countries with low development the prevalence is 43 per cent [1]. It affects the various organs including the heart. It is one of the most common causes of hyper dynamic state of heart at rest. It affects the heart by impairing the O₂ supply of myocardium [2], thus supply – demand myocardial mismatch causing myocardial ischemia or infarction [3]. A number of mechanisms are available to compensate for the decrease in O₂ transport associated with anaemia. They include an increase in Cardiac Output (CO) and decrease in circulation time [4]. These cardiac disturbances persist as long as the anaemia is severe [2] and quite strikingly these changes can be rapidly reversed by partial correction of anaemia in almost every instance [1]. In severe long standing anaemia, cardiac dilatation and hypertrophy are naturally expected due to hyperdynamic state [5].

To substantiate this, ECG studies for cardiac disturbances have been made less frequently. There is a great diversity of opinion available in literature, on reports of ECG changes in anaemia [6,7]. Early reports have described a decrease in QRS amplitude, T wave flattening and minor degrees of atrioventricular (AV) conduction disturbances [8], but these have not been observed in more recent studies. Later studies have reported frequent non-specific ST-T wave changes [9]. It is not certain, however that these changes are more common in anemic patients [1]. The abnormalities may be proportional to the severity of anaemia [10], or show no correlation to Hb level [11]. Hence in the present study we intended to study the electrocardiographic changes in anemic population and to correlate ECG changes seen with increasing severity of anaemia, with respect to every 1gm% decrease in hemoglobin (Hb) level from 8 gm% onwards.

MATERIALS AND METHODS

This study was conducted on selected hundred (male and female) anemic individuals of age group 18- 30 years, during September 2013 to December 2013. Patients attending medicine Out-Patient Department (OPD) diagnosed as anaemia, irrespective of etiology, in whom, there was Hb < 8gm% were randomly included in the study. Patients with history of smoking, alcoholism, diabetes, hypertension, clinically evident disturbances of the cardiovascular system, or of any extra-cardiac affection that may produce ECG changes, were excluded from the study. To avoid any possible effect of age on ECG, we chose the population only between 18-30 years. Ethical clearance was obtained from the institution. Informed consent was also obtained from the patients selected for the study. Hb level was estimated by semi-automated analyser which provided idea about severity of anaemia. All the patients were grouped according to Hb level. Then in the selected hundred anemic individuals, resting ECG was taken using electrocardiograph machine, in all the twelve leads and analysed for the specific anemic changes.

STATISTICAL ANALYSIS

ECG findings and varying severity of Hb level of each group were correlated using Pearson's co-relation co-efficient and association was calculated using Chi-square test (SPSS version 11).

RESULTS

Hundred patients were grouped based on their Hb level as Group 1 to Group 7, each group with the range of 1gm% Hb, beginning from 0gm% Hb to 8gm% Hb. There was no significant correlation between clinical history with varying severity of anaemia, also between peripheral smear picture and Hb level. There was significant correlation between Hb level and ECG changes. The ECG findings are tabulated in [Table/Fig-1].

We found that, 72% of patients were distributed in Group 5,6 and 7 (Hb 5-8gm%) and the remaining 38% were distributed in group 1,2,3 and 4 (Hb level 0-5gm%). As the Hb level decreased there were more percentage of patients having tachycardia, and the Chi-square association showed high significance. As the Hb level decreased there was more percentage of patients having ECG changes, although the Chi-square association was not significant. In Group 7 (Hb level 7-8gm%) no ECG changes were seen. In Group 5 and 6 (Hb level 5-7gm%) showed less percentage of patients with ECG changes. In Group 1, 2, 3 and 4 (Hb level 1-5gm%) showed increasing percentage of patients with ECG changes – 50-75% having ST depression, 29-50% T wave changes and 25-30% LVH. The ECG pattern with only ST depression was seen as in [Table/Fig-2], and with both ST depression and T wave inversion was seen as in [Table/Fig-3].

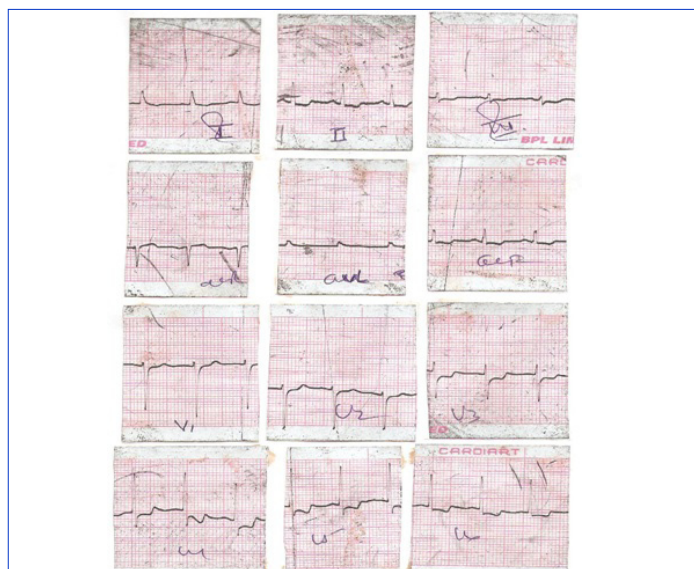
The correlation between the level of Hb and number of patients with tachycardia and ECG findings is shown in [Table/Fig-4].

There is strong negative correlation between Hb level and tachycardia and ECG changes, indicating that as the Hb level decreases, there is an increase in occurrence of tachycardia and ECG changes.

Group	N	ECG changes							
		Tachycardia		LVH		STD		Tw	
		n	%	n	%	N	%	n	%
Group 1	4	3	75	1	25	3	75	2	50
Group 2	4	4	100	1	25	3	75	1	25
Group 3	7	7	100	2	29	5	71	2	29
Group 4	13	10	76	1	0.07	7	54	1	0.07
Group 5	14	7	50	1	0.07	2	0.14	1	0.07
Group 6	24	13	54	1	0.04	4	0.17	2	0.08
Group 7	34	1	0.03	0	0	0	0	0	0
Chi-square test		x ² = 46.40		x ² = 2.16					
p-value		p=0.0001(HS)		p=0.99(NS)					

[Table/Fig-1]: ECG changes in different hemoglobin groups

N = Number of patients, n= Number of patients in sub group, LVH = Left ventricular hypertrophy, STD = ST depression, Tw = T wave changes, HS = Highly significant, NS = Non significant

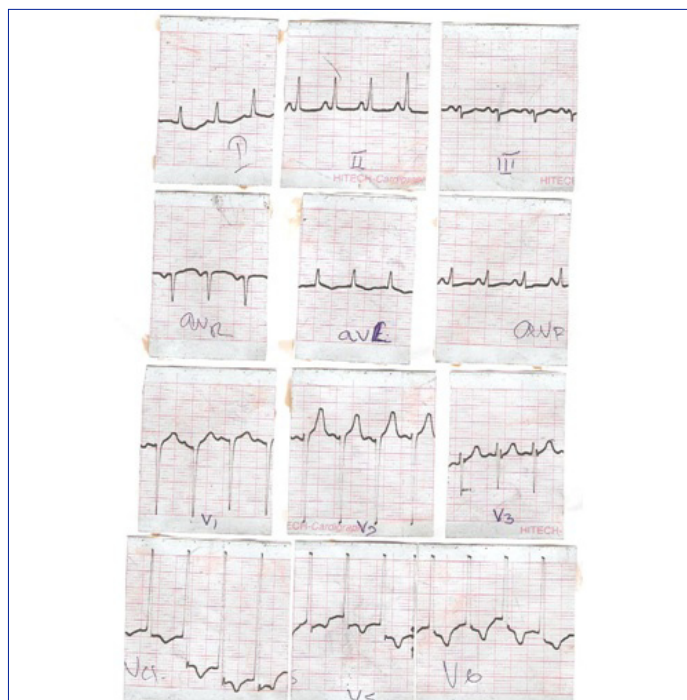


[Table/Fig-2]: ECG changes in a case showing tachycardia with ST depression in lead I, II, III, aVF, V3-6

DISCUSSION

In the present study on 100 patients, the Hb level showed strong negative correlation with the ECG changes.

Tachycardia, seen in the present study seems to be a clinical evidence of physiological adjustments in circulation due to anaemia, as a compensatory increase in Cardiac output (CO), in order to maintain



[Table/Fig-3]: ECG changes in a case showing tachycardia with ST depression in Lead I, aVL, V₄-V₆ and T wave inversion in V₄-V₆

Parameters	Haemoglobin level	
	r	p
Tachycardia	-0.646	0.0001
ECG changes	-0.572	0.0001

[Table/Fig-4]: Correlation between Haemoglobin level with ECG changes and tachycardia

adequate O₂ supply. Increase in CO can be achieved by increase in blood volume, preload, Heart Rate (HR) and stroke volume, along with a decrease in after load [12]. Literature shows that, though tachycardia contributes to higher CO than those with normal HR, no direct correlation between CO and HR could be established. However, it has been shown that, stroke volume is more closely related to elevated CO than tachycardia [13]. In a study, where in autonomic function were assessed in anemic patients, showed that they had low basal parasympathetic outflow to increase the HR as compensatory mechanism [14]. Hence we can say that tachycardia seen in anemic patients could be due to low basal parasympathetic outflow, to increase CO but doesn't contribute much to the needed CO, unlike stroke volume, which was not included in our study, since we concentrated only on the ECG changes in anemic patients.

One of the ECG changes noted in our study was LVH, indicating cardiac enlargement. This was pointed out a century ago [15]. Cardiac enlargement without other etiologies has been observed more frequently in patients with anaemia, particularly in patients with low Hb [15]. In anaemia there is combination of increased HR and stroke volume, to increase CO, which in turn improves O₂ delivery. To accommodate this greater output, there is an increase in LV chamber size, both systolic and diastolic [16,17]. The factor for cardiac enlargement could be both dilatation as well as hypertrophy. It seems that anaemia of shorter duration results in cardiac dilatation, whereas that of longer duration results in hypertrophy [15]. It was assumed that cardiac enlargement and hypertrophy in anemic patients was due to increased work of heart, but now it has been attributed to insufficient O₂ supply to the myocardium [15]. It is interesting to note that cardiomegaly reportedly returns to normal within few weeks of resolution of anaemia [18].

The other changes seen in our study were ST segment depression and T wave flattening and inversion. Earlier studies have reported decreased QRS amplitude, T-wave flattening and minor degrees

of atrioventricular conduction disturbances [8]. A study has also noted accentuation of T wave, appearances of Q wave in lead III, diminution and enlargement of QRS complex [11]. But, later many studies have reported changes in ECG like ST segment depression, flat or inverted T waves, but without corresponding changes in QRS complex [9,15,19,20].

In the present study such alterations in electrical conduction was seen in more per centage of patients with <5gm%, in patients with 5-7gm% very less percent of them showed such changes and in patients with Hb 7gm% there were no changes. Similar observations were made in a study of anemic patients with Hb level of 4-5gm% or less [15]. Yet another study has reported such findings in young patients, where Hb values were between 2.7gm% and 3.5gm% [15].

This could be because the ECG changes are not seen when the heart is in compensated state. But at 7gm%, there is a transition from a high-output (compensated) cardiac state to a state of LV dysfunction (decompensated). As the Hb level drops further, so does the LV function [21]. To substantiate this, few studies have shown elevated brain natriuretic peptide (BNP) levels in patients with clinical evidence of LV decomposition [22,23].

Though the changes in ECG are seen in more percentage of patients as the Hb level decreases, there was no association found in the present study. Similarly, no close parallelism was found between the degree of anaemia and that of cardiac disturbances in a study [11]. Experiments in animals have proved that, anaemia may produce ischemic disturbances of the myocardium. But ECG changes seen are not due to necrosis of heart muscle, but purely due to metabolic disturbances in myocardium resulting from O₂ deficiency, caused by diminution of O₂ – carrying power of the blood [11]. This could be the reason for not finding typical ischemic pattern in ECG of anemic patients. Unlike in ischemic pattern, the ECG changes due to anaemia, especially T- wave changes reverted back to normal within a week of correction of anaemia [19].

The difficulty of diagnosing anaemia just with signs and symptoms is known by various differential diagnosis, like other high output states (thyrotoxicosis, aortic regurgitation), coronary artery disease, heart failure, especially in emergency situations. Also, there are evidences that anaemia contributes to cardiac diseases and death. For example in chronic kidney failure, anaemia is an independent risk factor for development of cardiovascular disease [24]. There is growing evidence that anaemia contributes to cardiac disease and death, independently as well as a co-morbid factor. Thus by picking up/ not missing anaemia by signs and symptoms and supported by ST depression, T wave changes, with/without associated QRS abnormalities (LVH) in ECG as seen in the present study, in any situation is advantageous, that wrong treatment can be avoided in emergency situations [20], dramatic clinical and electrocardiographic recovery can be achieved with anaemia correction [21] and better clinical outcomes can be achieved in conditions of anaemia as a co-morbid factor [25].

We have not considered the etiology of anaemia, duration of anaemia, bone marrow picture, and to confirm the cardiac changes we could not do echocardiography in the present study.

CONCLUSION

Thus, it can be concluded that diagnosing anaemia in critical care can be supported by ECG changes to avoid misdiagnosis and also as dramatic clinical and ECG recovery can be achieved with anaemia correction.

REFERENCES

- [1] Ministry of Health and Family Welfare, Government of India. Guidelines for control of Iron deficiency anaemia. New Delhi: Ministry of Health and Family Welfare, Government of India; 2013. Available at: http://www.unicef.org/india/10_National_Iron_Plus_Initiative_Guidelines_for_Control_of_IDA.pdf. Accessed 18 January 2014.
- [2] Tandon OP, Katiyar BC. Ballistocardiographic study in severe anaemia. *Circulation*. 1961; 23: 195-99.
- [3] Bailey D, Aude YW, Gordon P, Burt D. ST segment elevation myocardial infarction, severe anaemia and non-obstructive coronary disease: case report and brief comment. *Conn Med*. 2003; 67(1):3-5.
- [4] Harvey N. Anaemia. In: Jhons RJ, Harvey AM, McKusick VA, Owens AH, Ross RS editors. *Principles and practice of medicine*, 22nd ed. New Delhi: Prentice-Hall International Inc; 1988. p. 311.
- [5] Friedberg CK. The heart and circulation in anaemia. In: Diseases of the heart. 3rd ed. Philadelphia: W.B.Saunders; 1969. 1678-86.
- [6] Ellis LB, Faulker JM. The heart in anaemia. *New Eng J Med*. 1939; 220:943-5.
- [7] Sanghvi LM, Mishra SN, Banarji K. Electrocardiogram in chronic severe anaemia. *Am Heart J*. 1958; 56: 79-86.
- [8] Porter WB. Heart changes and physiologic adjustments in hookworm anaemia. *Am Heart J*. 1937; 13:550.
- [9] Hunter A. The heart in anaemia. *Quart J Med*. 1946; 15:107.
- [10] Winsor M, Burch L. The ECG and cardiac state in active sickle cell anaemia. *Am Heart J*. 1945; 29:685-90.
- [11] Szekely P. Electrocardiographic findings in anaemia. *Br Heart J*. 1940; 2(1):1-8.
- [12] Pereira AA, Sarnak MJ. Anaemia as a risk factor for cardiovascular disease. *Kidney Int Suppl*. 2003; 87:S32-9.
- [13] Roy SB, Bhatia ML, Mathur VS, Virmani S. Hemodynamic effects of chronic severe anaemia. *Circulation*. 1963;28:346-56
- [14] Lokhotia M, Shah PK, Gupta A, Jain SS, Agarwal M, Dadhich S. Clinical assessment of autonomic functions in anemics. *J Assoc Physicians India*. 1996;44(8): 534-6.
- [15] Wintrobe MM. The cardiovascular system in anaemia: with a note on the particular abnormalities in sickle cell anaemia. *Blood*. 1946;1:121-8.
- [16] GeorgievaZ, GeorgievaM. Compensatory and adaptive changes in microcirculation and left ventricular function of patients with chronic iron deficiency anaemia. *Clin Hemorheol Microcirc*. 1997;17:21-30.
- [17] Hayashi R, Ogawa S, Watanabe Z, Yamamoto M. Cardiovascular function before and after iron therapy by echocardiography in patients with iron deficiency anaemia. *Pediatr Int*. 1999;41:13-7.
- [18] Varat MA, Adolph RJ, Fowler NO. Cardiovascular effects of anaemia. *Am Heart J*. 1972;83:415-26.
- [19] Gonzales-de-cassio A, Sanchez-Medal L, Smyth JF. Electrocardiographic modifications in anaemia. *Am Heart J*. 1964;67:166.
- [20] Sareban MT. ECG changes in anaemia. *Iranian Journal of Paediatric Haematology*. 2010;1(1):104.
- [21] Hegde N, Rich MW, Gayomali C. The cardiomyopathy of iron deficiency. *Tex heart Inst J*. 2006;33(3):340-4.
- [22] Tsujita K, Nikolsky E, Lansky AJ, Dangas G, Fahy M, Brodie B, et al. Impact of anaemia on clinical outcomes of patient with ST segment elevation myocardial infarction in relation to gender and adjunctive anti-thrombotic therapy. *Am J Cardiol*. 2010;105(10):1385-94.
- [23] Wold Knudsen C, Vik-Mo H, Omland T. Blood haemoglobin is an independent predictor of b type natriuretic peptide (BNP). *Clin Sci*. 2005;109:69-74.
- [24] Sarnak MJ, Levey AS, Choolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American heart association council on kidney in cardiovascular disease. High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. *Circulation*. 2005;112:1121-7.
- [25] Lang CC, Macini DM. Non cardiac comorbidities in chronic heart failure. *Heart*. 2007; 93(6): 665-71.

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Physiology, S. Nijalingappa Medical College and HSK Hospital, Bagalkot, India.
2. Assistant Professor, Department of Medicine, S. Nijalingappa Medical College and HSK Hospital, Bagalkot, India.
3. Associate Professor, Department of Physiology, S. Nijalingappa Medical College, Bagalkot, India.
4. Associate Professor, Department of Physiology, S. Nijalingappa Medical College, Bagalkot, India.
5. Assistant Professor, Department of Physiology, S. Nijalingappa Medical College, Bagalkot, India.
6. Assistant Professor, Department of Physiology, S. Nijalingappa Medical College, Bagalkot, India.
7. Assistant Professor, Department of Physiology, S. Nijalingappa Medical College, Bagalkot, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Shashikala GV,
Assistant Professor, Department of Physiology, S.N. Medical College and HSK Hospital, Bagalkot-587102, India.
Phone: 9986697172, E-mail: dr.gvs@rediffmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Feb 16, 2014**
Date of Peer Review: **Feb 22, 2014**
Date of Acceptance: **Mar 13, 2014**
Date of Online Ahead of Print: **Mar 28, 2014**
Date of Publishing: **Apr 15, 2014**