

Original research article

Study of electrocardiographic changes in patients with severe anemia admitted to tertiary care hospital

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Abstract

The clinical presentation depends upon the rapidity of the onset of anaemia, its severity, age of the patient and capacity of the cardiovascular system to adjust to it. Anaemia affects various organs including the heart. It is one of the commonest causes of hyperdynamic state of heart at rest. This leads to increased oxygen demand to myocardium and subsequently supply-demand mismatch resulting in myocardial ischaemia or infarction. Patients with severe anemia with haemoglobin less than 7gm% will be included after applying the exclusion criteria. Detailed history will be taken for all patients, which included history of breathlessness, palpitations, chest pain, swelling of legs. The clinical examination included resting pulse, blood pressure, nail changes, jugular venous pressure, pedal oedema, heart sounds, murmur heard, liver and spleen size and features of congestive heart failure. In ECG, we noticed a prolongation of P wave duration which was seen in a total of 26(26%) patients out of 100, of which 15(23.1%) were females and 11(31.4%) were males. Prolonged P wave duration was predominantly seen in males compared to females.

Keyword: Electrocardiographic changes, severe anemia, p wave

Introduction

Anemia is major public health problem and remains one of the most prevalent and enfeebling morbidities suffered by individuals in the developing world and is a critical co morbid factor which contributes to the excess mortality in these regions. Anaemia is the most common disease and even more so in a tropical country like India ^[1].

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 ^[2].

The clinical presentation depends upon the rapidity of the onset of anaemia, its severity, age of the patient and capacity of the cardiovascular system to adjust to it. Anaemia affects various organs including the heart. It is one of the commonest causes of hyperdynamic state of heart at rest. This leads to increased oxygen demand to myocardium and subsequently supply-demand mismatch resulting in myocardial ischaemia or infarction. The cardiac disturbances persist as long as anaemia is severe and quite strikingly these changes can be rapidly reverted by partial correction of anaemia ^[3].

Clinical manifestations of anaemia referable to cardiovascular system may closely simulate symptoms and signs of organic heart disease. On the other hand, severe anaemia may precipitate or intensify heart

failure or coronary insufficiency in a pre-existing cardiac or coronary disease. Therefore, it is necessary to diagnose and correct anaemia to evaluate the extent to which anaemia is partially or entirely responsible for the symptoms and signs.

With reduction in haemoglobin content of 25% or less, the blood volume is increased; there is generalised vascular dilatation in muscles and skin, a rise in venous blood pressure and a high cardiac output with tachycardia. Congestive heart failure, angina pectoris and ECG changes in anaemia are correlated to the severity of anaemia and increase in cardiac output ^[4].

Anemia has been convincingly shown to be a powerful predictor of rehospitalisation rates and survival in chronic heart failure. Hemodynamic changes accompanying severe anemia include increased preload, reduced peripheral vascular resistance and increased cardiac output. These adaptive responses may ultimately lead to a detrimental increase in the left ventricular mass. Anemia has shown to be associated with reversible elevations in plasma catecholamine's and alpha 2 receptors density among patients with renal failure. Finally, chronically increased myocardial work and adrenergic stimulation caused by decreased oxygen carrying capacity may ultimately lead to progression of heart failure and may accelerate adverse ventricular remodeling ^[5].

The LV internal dimensions in systole and diastole end systolic and end diastolic volume index, and other performance indices are deranged in all anemic patients. Clinical studies suggest that correction of anemia causes regression of left ventricular hypertrophy may improve exercise capacity and decrease adverse outcomes in patients with congestive heart failure ^[6].

Methodology

Patients with severe anemia with haemoglobin less than 7 gm% will be included after applying the exclusion criteria. Detailed history will be taken for all patients, which included history of breathlessness, palpitations, chest pain, swelling of legs. The clinical examination included resting pulse, blood pressure, nail changes, jugular venous pressure, pedal oedema, heart sounds, murmur heard, liver and spleen size and features of congestive heart failure. All patients will have a complete haematological examination and peripheral smear examination. Electrocardiogram will be recorded for all patients in the resting state. Routine posterior-anterior chest roentgenogram will be taken in all cases. All echo will be done using 3.7, 5MHz and 2.0, 2.5MHz probe. The various echo parameters will be analysed.

Inclusion criteria

Patients with Hb <7gm.

Age group between 15 to 40 years.

Exclusion criteria

Age <15yrs and >40yrs.

Pregnancy Patients with known ischemic heart disease, hypertensive heart disease, cardiomyopathy, congenital heart disease.

Patients with valvular heart disease.

Patients with Chronic kidney disease.

Patients with pulmonary disease.

Patients with other hyperdynamic circulatory states like hyperthyroidism, Beriberi, AV fistula, fever.

If patients had congestive cardiac failure due to anemia, they were included in the study two weeks later after failure was controlled.

Results

Table 1: ECG changes according to Gender

ECG	Gender		Total
	Female(65)	Male(35)	
Sinus Tachycardia	65(100%)	35(100%)	100(100%)
LVH	23(35.3%)	16(45.7%)	39(39%)

A total of 26 patients had p wave prolongation in ECG, out of which 15(23.1%) were females and 11(31.4%) were males, there was no significant p value when we compared p wave duration

prolongation with gender of the patients.

Table 2: Prevalence P wave duration prolongation with respect to Gender

P wave prolongation	Gender		Total
	Female	Male	
No	50(76.9%)	24(68.6%)	74(74%)
Yes	15(23.1%)	11(31.4%)	26(26%)
Total	65(100%)	35(100%)	100(100%)

P=0.364; Not significant, Chi-Square Test

Out of 39 patients with LVH in ECG, pressure overload was seen in 15 patients and volume overload was seen in 24 patients. Volume overload (61.5%) was predominant compared to pressure overload (38.4%). LVH pressure overload was seen in total of 15 patients of which 8(12.3%) were females and 7(20%) were males. LVH pressure overload was slightly more in males compared to females. There was no significant p value when we correlated LVH pressure overload with gender of patients.

Table 3: Prevalence LVH PO pattern according to Gender

LVH PO	Gender		Total
	Female	Male	
NO	57(87.7%)	28(80%)	85(85%)
YES	8(12.3%)	7(20%)	15(15%)
Total	65(100%)	35(100%)	100(100%)

P=0.304; Not significant, Chi-Square Test

Presence of ECG LVH PO pattern was correlated with systolic and diastolic dysfunction. Out of 15 patients with LVH PO 7 patients had systolic dysfunction and 4 patients had diastolic dysfunction, remaining 4 patients neither had systolic dysfunction nor diastolic dysfunction. LVH PO was not significantly associated with systolic and diastolic dysfunction although diastolic dysfunction (28.6%) was slightly more compared to systolic dysfunction (21.2%) in patients with LVH PO.

Table 4: Correlation of LVH PO pattern with Systolic Dysfunction

LVH PO	Systolic Dysfunction		Total
	No	Yes	
No	59(88.1%)	26(78.8%)	85(85%)
Yes	8(11.9%)	7(21.2%)	15(15%)
Total	67(100%)	33(100%)	100(100%)

P=0.222, Not Significant, Chi-Square Test

Table 5: Correlation of LVH PO pattern with Diastolic Dysfunction

LVH PO	Diastolic Dysfunction		Total
	No	Yes	
No	75(87.2%)	10(71.4%)	85(85%)
Yes	11(12.8%)	4(28.6%)	15(15%)
Total	86(100%)	14(100%)	100(100%)

P=0.125, Not Significant, Chi-Square Test.

Presence of ECG LVH volume overload pattern was seen in 24 patients out of which 15 were females and 9 were males. LVH VO was equally seen in both male and female patients, LVH VO was not significantly associated with gender of the patients with p value of 0.768.

Table 6: Prevalence of LVH VO pattern with Gender

LVH VO	Gender		Total
	Female	Male	
NO	50(76.9%)	26(74.3%)	76(76%)
YES	15(23.1%)	9(25.7%)	24(24%)
Total	65(100%)	35(100%)	100(100%)

P=0.768; Not significant, Chi-Square Test.

LVH VO is not significantly associated with Gender with P value of 0.768. Presence of ECG LVH volume overload pattern was correlated with systolic and diastolic dysfunction. Out of 24 patients with LVH volume overload 13 patients had systolic dysfunction and 7 patients had diastolic dysfunction. LVH volume overload was significantly associated with both systolic and diastolic dysfunction p value of 0.011 and 0.014 for both systolic and diastolic dysfunction respectively.

Table 7: Correlation of LVH VO pattern with Systolic Dysfunction

LVH VO	Systolic Dysfunction		Total(n=100)	P Value
	No(n=67)	Yes(n=33)		
No	56(83.6%)	20(60.6%)	76(76%)	0.011*
Yes	11(16.4%)	13(39.4%)	24(24%)	0.011*

Chi-Square Test/Fisher Exact Test

Presence of LVH VO is significantly associated with systolic dysfunction with P value of 0.011.

Table 8: Correlation of LVH VO pattern with in Diastolic Dysfunction

LVH VO	Diastolic Dysfunction		Total(n=100)	P Value
	No(n=86)	Yes(n=14)		
No	69(80.2%)	7(50%)	76(76%)	0.014*
Yes	17(19.8%)	7(50%)	24(24%)	0.014*

Chi-Square Test/Fisher Exact Test

Discussion

In ECG, we noticed a prolongation of P wave duration which was seen in a total of 26(26%) patients out of 100, of which 15(23.1%) were females and 11(31.4%) were males. Prolonged P wave duration was predominantly seen in males compared to females.

Simsek *et al.* in his study showed that iron deficiency anemia may be associated with prolonged p wave duration and impaired diastolic left ventricular filling. Our study showed prolonged p wave duration was associated with systolic dysfunction. His study found contradicting result when compared to our study because the HB was 7.5gm which is high when compared to 5.36gm in our study^[7].

In a study by Nikitha Hegde *et al.* showed various ECG abnormalities in iron deficiency anemia. Sinus tachycardia and LV hypertrophy are common. T wave abnormalities have also been noted. Low voltage on the ECG has been reported by many investigators^[8]. However, ST and T wave abnormalities is not specific to anemia it is subjected to variability like rate related changes, bundle branch block, pericarditis, it weakens the strength of association.

ECG, LVH with volume overload and pressure overload pattern was correlated with systolic and diastolic dysfunction. Out of 14 patients who had diastolic dysfunction alone, 10 patients (71.4%) had no LVH pressure overload on ECG, whereas 4 patients (28.6%) had LVH pressure overload on ECG. When compared to 33 patients who had systolic dysfunction patient, 7 patients (21.2%) had LVH pressure overload on ECG whereas 26 (78.8%) had no LVH pressure overload.

Out of 39 patients with LVH in ECG 24(61.5%) patients had LVH volume overload on ECG and 15(38.4%) patients had pressure overload on ECG. LVH volume overload was predominant than LVH pressure overload. Till date no study was conducted on ECG LVH volume overload and pressure overload with systolic and diastolic dysfunction.

Sir Bu *et al.* in his study showed that multiple studies have examined LA function, but the main inconvenience for the quantitative assessment is the requirement of invasive pressure-volume diagrams that do not allow routine clinical use from non-invasive techniques a variety of indices were examined, but the approaches are complex and the accuracy of these parameters is limited. The 2D echocardiographic LA volumes have been proposed as indicators of atrial size, but some potential sources of error related to the geometric assumptions remain to be corrected^[9, 10].

Conclusion

Severe anemia maybe associated with LVH on ECG with both pressure and volume overload patterns. Volume overload is more common.

References

1. Bruno de Benoist *et al.*, World Health Organization worldwide prevalence of anemia. Geneva: World Health Organization, 2008, 1993-2005.
2. 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; Available at: <http://www.unicef.org/india/10>.
3. Johansen D, Y Trehus K, Baxter GF. Exogenous hydrogen sulphide (H₂S) protects against regional myocardial ischemia-reperfusion injury. *Basic research in cardiology*. 2006;101:53-60.
4. Fowler Homes, John C. Holmes. Dextran exchange in anaemia and reduction in blood viscosity in the heart-lung preparation. *American Heart Journal*. 1964;68(2):204-13.
5. P. Greer. Clinical effects of anaemia (ch.22) In: Wintrobe's Clinical Haematology. 13th ed. Wolters Kluwer UK, 2014, 589p.
6. Tendon O P, Katiyar B C. Ballistocardiographic study in severe anaemia. *Circulation*. 1961;23:195-199.
7. Simsek H, Gunes Y, Demir C, Sahin M, Gurucuoglu HA, Tuncer M. The effects of iron deficiency anemia on p wave duration and dispersion Clinics effects of iron on. 2010 Aug;65(1):1067-1071.
8. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, *et al.* Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogram*. 2005 Dec;18(12):1440-41doi:10.1016J.echo. 10.005. PMID:16376782
9. Mohammed A-Biltagi, Osama Tolba, Mohammed Elshanshory, Ibrahim Badraia, Sahar M. Hazaa. Atrial function and glutathione in children with iron deficiency anemin: Tanta-Egypt-2012. *International Blood Research & Reviews*. 2013 Aug;1(2):72-86
10. Naito Y, Tsujino T. Matsumoto M, Skoda T. Olyanagi M. Masuyama T. Adaptive response of the heart to long-term anemia Induced by iron deficiency. *Am J Physiol Heart Circ. Physiol*. 2009 Mar;296o:H85-93.