Research Article

Comparative study of cell counter-based parameters in different hemoglobinopathies from north Maharashtra region

Mohammad Ismail Ansari[1], Nisar Patel[2]

[1]Department of Zoology, Haji Saeed Ahmed Sardar Arts and Science College, Deopur, Dhule, Maharashtra, India.
[2]B.F. Patel Arts, Commerce and Science College, Pimpalgaon Kale, Buldhana, Maharashtra, India.
Correspondence to: Mohammad Ismail Ansari, E-mail: ismailansari249@gmail.com

Received February 1, 2016. Accepted February 13, 2016.

Abstract

Background: Thalassemias among hemoglobinopathies are major public health problem in India and bear financial burden on health-care systems.

Objective: To identify carriers in the families of index cases and to differentiate types of hemoglobinopathies on the basis of red cell parameters.

Materials and Methods: The study was carried out on 1,702 family members and close relatives of 242 clinically proved β-thalassemia major patients with different caste and communities. For initial diagnosis of carriers, all the samples were subjected to osmotic fragility test and red cell indices by automatic cell counter. If any abnormality were detected, further confirmatory tests were carried out by cellulose acetate electrophoresis and HPLC for estimation of HbA_2. For comparative analysis of red cell indices between β-thalassemia and other hemoglobinopathies, samples of different hemoglobinopathies were collected and complete blood count was carried out.

Result: A total of 629 β-thalassemia carriers were identified among 1,702 family member tested. Hematological data revealed that, except red cell distribution width, all red cell indices were statistically significant different between β-thalassemia major and minor. Microcytosis and hypochromasia were the common features from the blood samples of β-thalassemia minor. Red cell indices from HbE disease, HbE/β-thalassemia, and HbD Punjab showed marked microcytosis. Moderate degree of microcytosis and hypochromia were seen in all cases of α-thalassemia, sickle/β-thalassemia, and HbE trait. Analysis of hemoglobin from different hemoglobinopathies revealed inconsistency in the presence of percent HbA, HbF, and HbA_2. Percent HbE (HbE%) in HbE diseased, HbE/β-thalassemia, and HbE trait patients were reported in decreasing order.

Conclusion: It has been concluded that the hematological parameters such as red blood cell count, mean corpuscular volume, and mean corpuscular hemoglobin were found to be suitable for screening of large population in resource poor areas.

KEY WORDS: β-Thalassemia major, β-thalassemia minor, hypochromia, microcytosis, red cell indices

Introduction

Thalassemias are the most common inherited autosomal recessive disorders of hemoglobin synthesis reported all over the world. Its prevalence is quite high in Southeast Asian countries and bear serious public health problem.[1] Approximately 80% of the annual births of babies with these conditions occur in low- or middle-income countries owing to lack of resources for their control and management.

Access this article online	
Website: http://www.ijmsph.com	Quick Response Code:
DOI: 10.5455/ijmsph.2016.01022016373	

In India, the prevalence of β-thalassemia and other hemoglobinopathies is quite variable, seen in all states and creating economic and health burden.[2] However, the data on the prevalence of β-thalassemias and other hemoglobinopathies in different communities and ethnic groups of India are scarce.

The early detection of asymptomatic carriers of thalassemia (heterozygotes) makes it possible to provide genetic counseling, which may lead to reduce the incidence of

homozygous condition and its fatal outcome. Therefore, in preventive and control programs, rapid, accurate, and inexpensive screening protocols to identify carriers of thalassemias and its variants, especially in population and families at risk for Hb disorders, are essential.

Automated cell counters are widely used in routine practice and are easily available at every diagnostic center; so, screening can be done without additional costs. The most consistent finding in carriers of β-thalassemia is the combination of a relatively high or normal red blood cell (RBC) count with low mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) along with normal Hb and hematocrit (Hct) values.[3,4] In fact, the MCV alone can identify a high number of thalassemia carriers in both adults and children.[5]

Materials and Methods

The study was undertaken to determine the prevalence of β-thalassemia traits and its associated hemoglobin variants in Dhule and Nandurbar districts by cell counter-based parameters. Ethical approval was obtained by institutional ethics committee. During the extended family screening, a total of 1,702 family members and close relatives of 242 β-thalassemia major patients (receiving regular blood transfusion at Government Civil Hospital, Dhule) with different caste and communities were enrolled in the study.

All the samples were collected during the period of study in EDTA Vacutainers and were immediately analyzed for complete blood count (CBC) on hematology analyzer (Coulter AcT 5diff; Beckman Coulter) and osmotic fragility for initial screening.[6] For osmotic fragility of RBC, different saline concentrations 0.32%, 0.34%, and 0.36% were used.[7] The blood specimens in which abnormalities were found (MCV < 80 fL and MCH < 27 pg; and/or osmotic resistance in hypotonic solutions) were processed for further series of studies. These include hemoglobin electrophoresis by cellulose acetate at pH 8.6 and automatic HPLC analyzer (Bio-Rad Variant-II β-thalassemia short program) for HbA_2 estimation.[8] Beta-thalassemia trait was diagnosed when the percentage of HbA_2 was 3.5% or higher.[9]

Statistical analysis was carried out by Statistical Package for Social Sciences (SPSS, Inc., Chicago, USA). Descriptive statistics and mean, standard deviation, and range were used to describe hematological characteristics of each thalassemia genotype. Statistical comparison with Student's two-sample *t* test was performed to determine the mean difference of hematological parameters between β-thalassemia major and β-thalassemia minor patients. ANOVA was used to compare means of hematological parameters between different hemoglobinopathies. Correlation coefficient was used to determine any relationship with in the hematological indices, and several relationships were identified. The *P* value < 0.05 was considered as statistically significant. Box plots (median and interquartile range) for each parameter were constructed.

Results

This study was conducted on 242 clinically proved β-thalassemia major patients and their family members. The ages of thalassemia major patients were between 6 months and 22 years, 136 being male and 106 female subjects. Some of their family members were already tested for their carrier state. The cellulose acetate electrophoresis and HPLC studies revealed that there were 629 β-thalassemia carriers among 1,702 family member tested.

The red cell measurements including RBC, white blood cell (WBC), hemoglobin, MCV, MCH, mean corpuscular hemoglobin concentration (MCHC), Hct, and red cell distribution width (RDW) were grouped according to the genotype.

For the cases of β-thalassemia major patients, the overall mean RBC count was 3.5 ± 0.5 10^{12}/L, Hb, 7.9 ± 1.1 g/dL; MCV, 71.5 ± 8.7 fL; and MCH, 22.9 ± 2.6 pg. For the cases of β-thalassemia minor patients, the overall mean RBC count was 4.7 ± 0.6 10^{12}/L; Hb, 9.9 ± 1.0 g/dL; MCV, 69.2 ± 6.8 fL; and MCH, 21.0 ± 2.2 pg. For the cases of β-thalassemia major, the overall mean level of HbA_2 and HbF were 5.2 ± 1.3% and 69.5 ± 13.3%, respectively, while for the cases of β-thalassemia minor, these values were 6.2 ± 1.5% and 2.2 ± 0.9%, respectively. There were significant differences between all red cell indices of β-thalassemia major and minor cases ($P < 0.000$), except RDW, which showed no significant difference.

Sex-wise hematological data of β-thalassemia major and β-thalassemia minor patients are illustrated in Table 1. The MCHC, RBC count, MCV, and RDW showed significant difference between male and female β-thalassemia major patients. In β-thalassemia minor cases, RBC count, MCV, MCH, and RDW showed significant different, while hemoglobin concentration between male and female subjects showed no significant difference.

In β-thalassemia major patients, the following indices were positively correlated: RBC and WBC ($r = 0.581$; $P < 0.000$); RBC and Hb ($r = 0.686$; $P < 0.000$); RBC and Hct ($r = 0.702$; $P < 0.000$); Hb and Hct ($r = 0.568$; $P < 0.000$); Hb and MCHC ($r = 0.316$; $P < 0.000$); Hct and MCV ($r = 0.415$; $P < 0.000$); MCV and MCH ($r = 0.385$; $P < 0.000$); and MCH and MCHC ($r = 0.499$; $P < 0.000$). However, a negative correlation was found between RBC and MCV ($r = –0.349$; $P < 0.000$); RBC and MCH ($r = –0.548$; $P < 0.000$); RBC and MCHC ($r = –0.124$; $P > 0.054$); Hb and MCV ($r = –0.107$; $P > 0.096$); Hct and MCH ($r = –0.257$; $P < 0.000$); Hct and MCHC ($r = –0.589$; $P < 0.000$); MCV and MCHC ($r = –0.598$; $P < 0.000$); MCH and RDW ($r = –0.070$; $P > 0.278$) and MCHC and RDW ($r = –0.126$; $P > 0.049$).

Table 2 shows the hematological data of different hemoglobinopathies. The level of hemoglobin was lower in two cases of HbE/β-thalassemia and HbD Punjab, moderate in α-thalassemia, sickle/β-thalassemia, and HbE disease, and normal in HbE traits. Double heterozygous sickle/β-thalassemia showed almost normal red cell indices and

Table 1: Sex-wise hematological data of β-thalassemia major and β-thalassemia minor patients

Parameters	β-thalassemia major (*n* = 242)			β-thalassemia minor (*n* = 629)		
	Male (*n* = 136)	Female (*n* = 106)	*P*	Male (*n* = 322)	Female (*n* = 307)	*P*
RBC, 10^{12}/L	3.6 ± 0.6	3.4 ± 0.5	<0.012*	4.8 ± 0.6	4.7 ± 0.5	<0.019*
WBC, 10^{9}/L	8.7 ± 2.5	8.6 ± 2.0	>0.06	8.2 ± 1.8	8.2 ± 2.2	>0.792
Hb, g/dL	8.0 ± 1.1	7.8 ± 0.9	<0.041*	9.9 ± 0.9	9.9 ± 0.7	>0.737
Hct, %	26.3 ± 4.6	22.9 ± 2.3	<0.000*	32.6 ± 3.2	32.8 ± 2.0	>0.448
MCV, fL	74.0 ± 8.8	68.1 ± 7.5	<0.000*	68.4 ± 6.5	70.1 ± 8.3	<0.000*
MCH, pg	22.7 ± 2.7	23.0 ± 2.5	>0.363	20.8 ± 2.1	21.3 ± 2.8	<0.006*
MCHC, g/dL	31.0 ± 4.6	33.9 ± 2.8	<0.000*	30.6 ± 3.0	30.5 ± 1.5	>0.630
RDW, %	17.1 ± 1.9	16.5 ± 1.6	<0.019*	15.9 ± 1.9	17.9 ± 1.0	<0.000*
HbA2, %	5.1 ± 1.3	5.3 ± 1.3	>0.257	6.2 ± 1.5	6.2 ± 1.0	>0.956
HbF, %	69.1 ± 14.4	70.0 ± 11.7	>0.610	2.3 ± 1.0	2.1 ± 0.7	<0.038*
HbA, %	25.5 ± 14.6	24.4 ± 12.0	>0.531	87.6 ± 1.5	87.6 ± 2.8	>0.649

*statistically significant

Table 2: Hematological features of different hemoglobinopathies

Parameters	Sickle/ β-thalassemia (*n* = 24)	HbE disease (*n* = 5)	HbE/ β-thalassemia (*n* = 2)	HbE trait (*n* = 5)	HbD Punjab (*n* = 6)	α-thalassemia trait (*n* = 13)	*P* (for overall difference)
RBC, 10^{12}/L	4.7 ± 0.5	4.9 ± 0.3	4.0, 4.5	4.6 ± 0.6	4.0 ± 0.2	4.2 ± 0.4	<0.003*
WBC, 10^{9}/L	7.7 ± 1.4	7.5 ± 0.9	5.5, 6.1	7.5 ± 0.8	6.0 ± 0.4	7.3 ± 0.8	<0.022*
Hb, g/dL	10.1 ± 0.9	10.3 ± 0.4	8.3, 8.9	12.3 ± 1.1	9.3 ± 0.8	10.4 ± 1.1	<0.000*
Hct, %	37.6 ± 2.2	35.0 ± 1.8	27.9, 31.5	38.4 ± 3.9	28.1 ± 2.1	31.4 ± 2.3	<0.000*
MCV, fL	81.1 ± 8.5	70.5 ± 4.9	68.9, 70.5	84.2 ± 8.7	69.1 ± 3.2	75.2 ± 7.8	<0.001*
MCH, pg	22.1 ± 2.2	21.5 ± 1.0	21.5, 19.8	27.2 ± 3.6	23.3 ± 2.3	24.9 ± 1.3	<0.000*
MCHC, g/dL	27.0 ± 2.2	29.4 ± 1.6	29.7, 28.3	32.1 ± 1.7	33.2 ± 3.3	33.6 ± 4.0	<0.000*
RDW, %	15.5 ± 1.2	16.3 ± 1.0	22.5, 26.4	15.4 ± 0.9	16.5 ± 0.8	15.4 ± 1.3	<0.000*
HbA2, %	6.4 ± 1.0	0.0	0.0	0.3 ± 0.3	1.8 ± 0.4	2.5 ± 0.3	<0.000*
HbF, %	9.3 ± 4.0	6.1 ± 0.7	8.2, 9.6	1.8 ± 0.4	0.8 ± 0.3	0.7 ± 0.2	<0.000*
HbA, %	25.2 ± 7.9	6.0 ± 2.7	40.2, 38.9	68.7 ± 1.7	66.8 ± 2.4	89.3 ± 3.1	<0.000*
HbS, %	58.0 ± 7.4	—	—	—	—	—	—
HbE, %	—	87.7 ± 3.3	48.8, 51.3	28.9 ± 1.4	—	—	<0.000*
HbD, %	—	—	—	—	29.9 ± 2.4	—	—

*statistically significant

is asymptomatic. Moderate degree of microcytosis and hypochromia reported in all cases of α-thalassemia, sickle/β-thalassemia, HbE disease, and trait. However, microcytosis was more marked in HbD Punjab and HbE/β-thalassemia when compared with other hemoglobinopathies.

A box plot of each hematological parameter was constructed and depicted in Figure 1a–d. As compared with other hemoglobinopathies, viz., β-thalassemia minor, sickle/β-thalassemia, HbE disease, HbE/β-thalassemia, HbE traits, HbD Punjab, and α-thalassemia traits (groups 2–8, respectively), β-thalassemia major (group 1) showed significant reduction in RBC [Figure 1a] and Hb [Figure 1b]. The reverse increasing trend was found with MCH [Figure 1d; groups 2–5]. In contrast, homozygous HbE and HbE/β-thalassemia [Figure 1c; groups 4 and 5] showed no significant difference in MCV from β-thalassemia major (group 1), moreover, HbD Punjab [Figure 1e; group 7] showed no difference in MCH from β-thalassemia major (group 1). On the other hand, insignificant difference was found between RDW of β-thalassemia major, β-thalassemia minor, HbE disease, and HbD Punjab. Analysis of hemoglobin revealed inconsistency in the presence of percent HbF among all hemoglobinopathies [Figure 1e]. Decreasing trend of percent HbE (HbE%) was reported between all three HbE cases: HbE disease, HbE/β-thalassemia, and HbE trait [Figure 2].

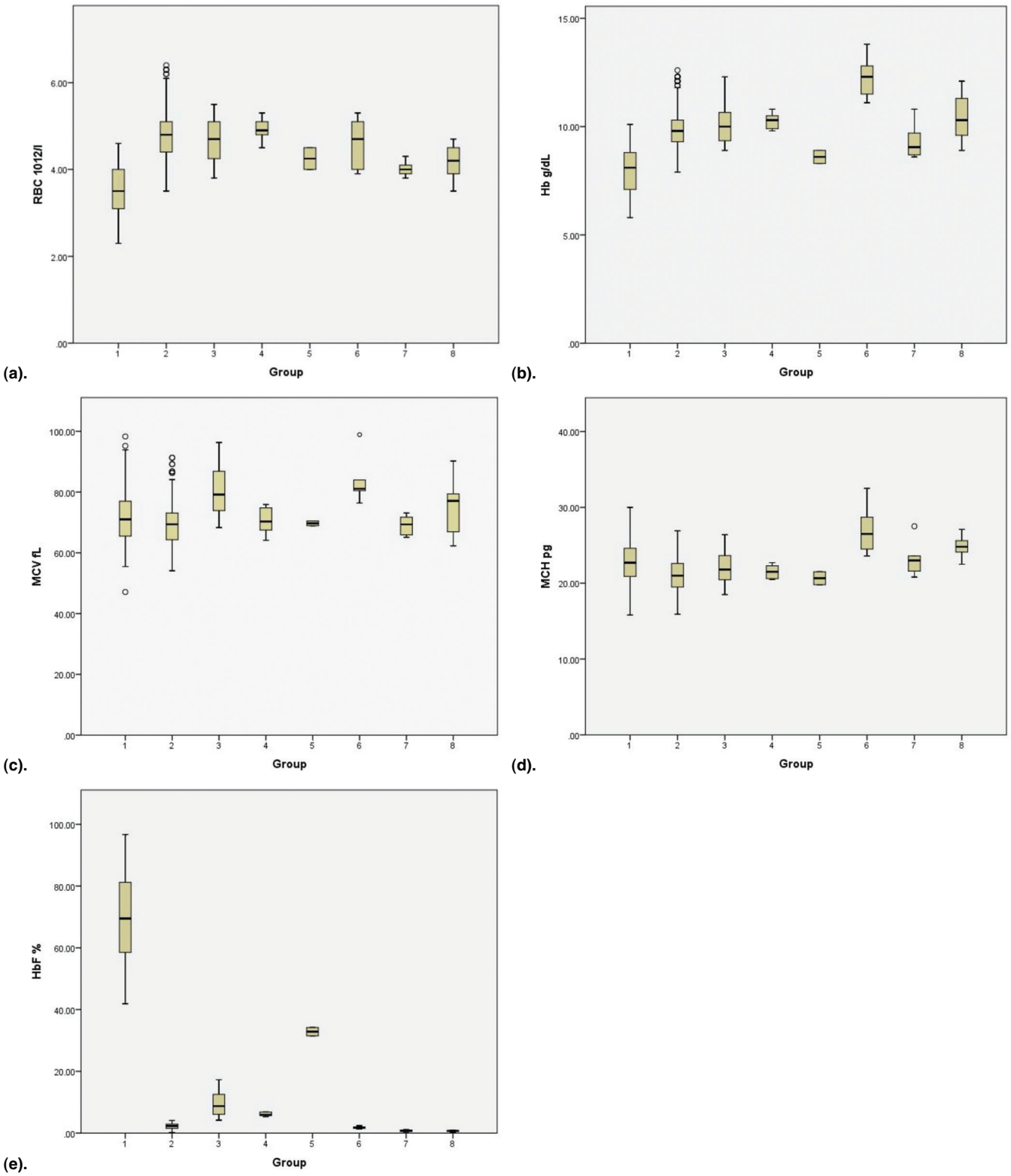

Figure 1: Comparison of (a) RBCs, (b) Hb, (c) MCV, (d) MCH, and (e) percent HbF between various hemoglobinopathies: Groups: 1, β-thalassemia major; 2, β-thalassemia minor; 3, sickle/β-thalassemia; 4, HbE disease; 5, HbE/β-thalassemia; 6, HbE trait; 7, HbD Punjab; and 8, α-thalassemia trait.

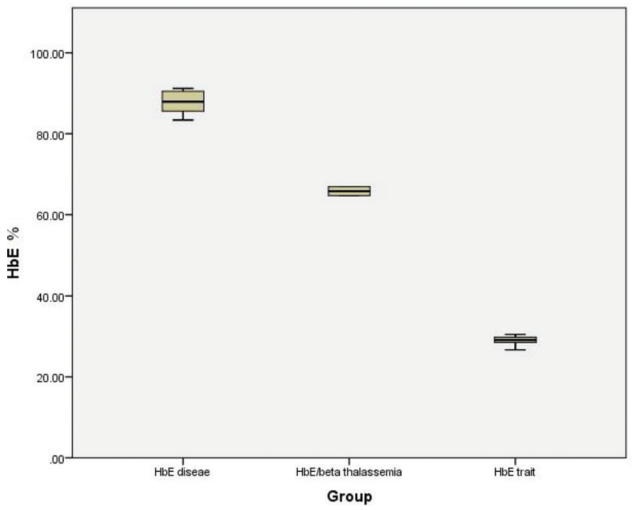

Figure 2: Comparison of percent HbE between HbE disease, HbE/β-thalassemia, and HbE trait.

Discussion

In this study, hematologic features of β-thalassemia major, β-thalassemia minor, α-thalassemia trait, double heterozygous sickle/β-thalassemia, double heterozygous HbE/β-thalassemia, HbE disease, HbE trait, and HbD Punjab were analyzed. In general, screening for all forms of thalassemia and hemoglobinopathies usually depends on a CBC obtained using automated blood cell counter. The clue for thalassemia is MCV < 80 fL and/or MCH < 27 pg, and individuals with these characteristics usually undergo a further investigation by hemoglobin and DNA analysis to identify types of the defect.[9]

Hematological parameters of β-thalassemia major patients and β-thalassemia minor were compared. Individuals with β-thalassemia major showed severe anemia with very low levels of RBC count, Hb, and Hct ($P < 0.000$) and increased levels of MCV, MCH, and MCHC ($P < 0.000$) when compared with patients with β-thalassemia minor. This result is in agreement with the finding of Mehdi and Al Dahmash,[10] and individual with β-thalassemia minor showed mild microcytic and hypochromic anemia as observed by others.[11]

Majority of the β-thalassemia major cases [188 of 242 (77.7%)] showed packed cell volume between 20% and 30%, followed by 31 of 242 (12.8%) who showed values more than 30% and only 23 of 242 (9.5%) with values less than 20%. On the other hand, 491 of 629 (78.1%) cases of β-thalassemia minor showed packed cell volume greater than 30%, while 138 of 629 (21.9%) cases showed less than 30% packed cell volume.

An RBC count of more than 5.0×10^{12}/L was observed in more than 35% cases in the β-thalassemia minor group, 37% cases in the double heterozygous sickle/β-thalassemia group, and two cases in each HbE disease and HbE trait groups. An increased RBC count despite a low hemoglobin concentration in β-thalassemia minor group has been reported, and this finding correlated with the findings of Lec.[12] The β-thalassemia minor group showed significantly lower values for MCV and MCH ($P < 0.000$) compared with β-thalassemia major cases. However, in the β-thalassemia minor, HbE disease, and HbE/β-thalassemia groups, the MCV and MCH values did not show significant differences ($P = 0.898$ and $P = 0.839$, respectively).

In this study, an MCV of less than 80 fL was reported in 96.5% cases of the β-thalassemia minor cases, 54.2% cases of sickle/β-thalassemia, 100% (5/5) cases of HbE disease, 100% (2/2) cases of HbE/β-thalassemia, 40% (2/5) cases of HbE traits, 100% (6/6) cases of HbD Punjab (100%), and 76.9% cases in α-thalassemia trait. An MCH value less than 27 pg was observed in 100% of β-thalassemia minor cases, 92% of α-thalassemia trait, 100% with sickle/β-thalassemia cases, 100% of HbE disease, 100% of HbE/β-thalassemia, 60% of HbE trait, and 83.3% of HbD Punjab. Similar results were reported earlier.[13,14] The cases of β-thalassemia minor showed reduction in MCV and MCH values, and, thus, these parameters found to be important in diagnosis of β-thalassemia carriers.

The degree of microcytosis and type of thalassemia mutation has shown wide variations in ranges of MCV.[14,15] Carriers of α-gene deletion show mild microcytosis with or without anemia. Owing to this, it is important to diagnose α-thalassemia to ascertain the cause of microcytosis and to avoid repeated expensive analysis and/or prolonged iron therapy. However, it was found that the red cell indices were unable to discriminate α-thalassemia except MCH was a better discrimination index.[16,17]

Hematologic comparison between the β-thalassemia and α-thalassemia traits revealed that the Hb, MCV, MCH, and MCHC were higher and RBC, Hct, and RDW were lower in β-thalassemia traits when compared with α-thalassemia traits. This is in agreement with the findings of Mehdi and Al Dahmash.[10] Similarl to α-thalassemia traits, hematologic parameters of HbE traits showed increasing trends for Hb, Hct, MCV, MCH, and MCHC and decreasing trend for RBC and RDW when compared with β-thalassemia traits. This result is in agreement with the study of Karnpean et al.[18] Almost all the cases of HbE heterozygotes in this study revealed normal MCV and MCH, and this finding correlated with the findings of Chan et al.[19] and Sanchaisuriya et al.[20]

Iron deficiency anemia and anemia associated with other hemoglobinopathies are the most common cause of a low MCV and/or MCH; it seems likely that this finding will point to thalassemia in thalassemic-prone ethnic regions. However, the number of patients with HbE disease, trait, HbE/β-thalassemia, and HbD Punjab are not considerable, and owing to this, the comparative analysis with other hemoglobinopathies will be not so efficient. But, to some extent, this study helps to understand the hematological variation in different hemoglobinopathies.

Of total (927) patients of different hemoglobinopathies, 486 (52.4%) were male and 441 (47.6%) female subjects.

This study reported that β-thalassemia minor was the most common form of hemoglobinopathy (67.9%), followed by β-thalassemia major (26.1%). While the prevalence of β-thalassemia minor in male subjects was 66.3% (322/486), in females, it was 69.6% (307/441). The incidence of β-thalassemia major cases in male subjects were 56.2% (136) and in female subjects 43.8% (106) of total 242 β-thalassemia major patients. The ages of these patients were between 6 months and 22 years and are dependent on blood transfusion.

Conclusion

The automated cell counter-based parameters provide an excellent hematological data and continue to play a crucial role for screening and differentiation of all forms of thalassemias such as α-thalassemia traits, homozygous α-thalassemia, and β-thalassemia major and minor. The key characteristic features for initial diagnosis of β-thalassemia trait were found to be MCV < 80 fL and MCH < 27 pg. It has been concluded that the RBC count, MCV, and MCH are suitable for screening in a large population in resource poor areas and are the best discriminant functions among all red cell indices and continue to provide an essential support to the diagnosis and monitoring of hematological disorders. Automated cell counters with differential counts are now readily available in almost all primary health-care clinics of Maharashtra state, where expensive laboratory equipments for screening and diagnosis of different hemoglobinopathies are not adoptable. This would definitely help physicians to diagnose these hemoglobinopathies without spending much time and cost.

References

1. Weatherall DJ, Clegg JB. Inherited haemoglobin disorders: an increasing global health problem. Bull World Health Organ 2001;79:704–12.
2. Sinha S, Black ML, Agarwal S, Colah R, Das R, Ryan K, et al. Profiling β-thalassaemia mutations in India at state and regional levels: implications for genetic education, screening and counselling programmes. Hugo J 2009;3:51–62.
3. Aghai E, Shabbad E, Quitt M, Froom P. Discrimination between iron deficiency and heterozygous beta-thalassemia in children. Am J Clin Pathol 1986;85:710–12.
4. England JM, Fraser PM. Differentiation of iron deficiency from thalassaemia trait by routine blood count. Lancet 1973;1:449–52.
5. Borgna-Piagnatti C, Zonta L, Bongo I, De Stefano P. Red blood cell indices in adults and children with heterozygous beta-thalassemia. Haematologica 1983;68:149–56.
6. Kattamis C, Efremov G, Pootrakul S. Effectiveness of one tube osmotic fragility screening in detecting β-thalassaemia trait. J Med Genet 1981;18:266–70.
7. Silvestroni E, Bianco I. A highly cost effective method of mass screening for thalassaemia. Br Med J 1983;286:1007–9.
8. Steinberg MH, Adams JG. Haemoglobin A2: origin, evolution and aftermath. Blood 1991;78:2165–177.
9. The laboratory diagnosis of haemoglobinopathies. Br J Haematol 1998;101:783–92.
10. Mehdi SR, Al Dahmash BA. A comparative study of hematological parameters of α and β-thalassemias in a high prevalence zone: Saudi Arabia. Indian J Hum Genet 2011;17:207–11.
11. Bhukhanvala DS, Sorathiya SM, Shah AP, Patel AG, Gupte SC. Prevalence and hematological profile of β-thalassemia and sickle cell anemia in four communities in Surat city. Indian J Hum Genet 2012;18:167–71.
12. Lec GR. Microcytosis and the anemias associated with impaired hemoglobin synthesis. In: Greer JP, Foerster J, Lukens JN (Eds.). *Wintrobe's Clinical Hematology*, 9th edn. Philadelphia: Lippincott Williams & Wilkins, 2001.
13. Madan N, Sikka M, Sharma S, Rusia U, Kela K. Red cell indices and discriminant functions in the detection beta-thalassaemia trait in a population with high prevalence of iron deficiency anaemia. Indian J Pathol Microbiol 1999;42:55–61.
14. Rathod DA, Kaur A, Patel V, Patel K, Kabrawala R, Patel V, et al. Usefulness of cell counter-based parameters and formulas in detection of β-thalassemia trait in areas of high prevalence. Am J Clin Pathol 2007;128:585–9.
15. Rahim F. Microcytic hypochromic anemia patients with thalassemia: genotyping approach. Indian J Med Sci 2009;63:101–8.
16. Hall GW, Thein SL, Newland AC, Chisholm M, Traeger-Synodinos J, Kanavakis E, et al. A base substitution T-G in codon 29 of the α2-globin gene causes α-thalassemia. Br J Haematol 2009;85:546–52.
17. Tritipsombut J, Sanchaisuria K, Fucharoen S, Fucharoen G, Siriratmanawong N, Pinmuang-ngam C, et al. Hemoglobin profiles and hematologic feature of thalassemic newborns: application of alpha-thalassemia 1 and hemoglobin E. Arch Pathol Lab Med 2008;132:1739–45.
18. Karnpean R, Pansuwan A, Fucharoen G, Fucharoen S. Evaluation of the URIT-2900 Automated Hematology Analyzer for screening of thalassemia and hemoglobinopathies in Southeast Asian populations. Clin Biochem 2011;44:889–93.
19. Chan LC, Ma SK, Chan AY, Ha SY, Waye JS, Lau YL, et al. Should we screen for globin gene mutations in blood samples with mean corpuscular volume (MCV) greater than 80 fL in areas with a high prevalence of thalassaemia? J Clin Pathol 2001;54:317–20.
20. Sanchaisuriya K, Fucharoen G, Sae-ung N, Jetsrisuparb A, Fucharoen S. Molecular and hematological features of hemoglobin E heterozygotes with different forms of α-thalassemia in Thailand. Ann Hematol 2003;82:612–6.

How to cite this article: Ansari MI, Patel N. Comparative study of cell counter-based parameters in different hemoglobinopathies from north Maharashtra region. Int J Med Sci Public Health 2016;5:1887-1892

Source of Support: Nil, Conflict of Interest: None declared.