



## SCREENING AND HAEMATOLOGICAL PARAMETER OF SOME TRIBALS OF AMRAVATI AND YAVATMAL DISTRICT, FOR SICKLE CELL ANAEMIA

Sandeep M. Chede<sup>1</sup>, Varsha S. Zade<sup>1</sup>, Akanksha R Mahajan<sup>1</sup>, Dinesh Dabhadkar<sup>1</sup> & Deboleena Majumdar<sup>2</sup>

<sup>1</sup>Department of Zoology Government Vidarbha Institute of Science and Humanities, Amravati.

Sant Gadge Baba Amravati University, Amravati. 444601 Maharashtra. India

<sup>2</sup>Anthropological survey of India, Nagpur Central Regional Centre, Nagpur. 440006 Maharashtra. India

### Abstract

Sickle cell disease (SCD) is a major gene disorder among the tribal population of central India. Hence the objective of the present study was to determine prevalence and frequency of sickle cell gene in some selected tribal castes of the Amravati and Yavatmal district. A total of 436 tribal individuals from Amravati and 115 tribal individuals from Yavatmal district were screened for SCD from villages. Blood samples showing positive solubility test were later subjected to capillary electrophoresis and Complete blood count. The allele frequency of sickle cell gene from Amravati District was found to be highest in the Bhil tribal group (0.4934) followed by Gawlan (0.4071), Korku (0.3294) and Nihal (0.2898) and was lowest in the Gowari (0.2871). While the sickle cell allele frequency was found to be 0.4243 in Gond, 0.350 in Kolam and 0.5750 in Pardhans, 0.50 in both Govaris and Madgis and only 0.20 in Banjaras of Yavatmal District.

**Key words:** Sickle cell disease (SCD), Allele Frequency, Tribal group, Amravati, Yavatmal.

### 1. Introduction

Sickle Cell Disease (SCD) is caused by an abnormal type of haemoglobin called haemoglobin S. Haemoglobin S changes the red blood cells. SCD is an autosomal recessive genetically transmitted hemoglobinopathy and passed down through families. Sickle cell disorder is caused by a point mutation at sixth position in  $\beta$  globin chain, valine substituting glutamic acid, due to which in deoxygenated state the red blood cells become fragile and shaped like crescents or sickles (Ingram, 1956). The abnormal cells deliver less oxygen to the body's tissue. They can also easily get stuck in small blood vessels and break into pieces. This can interrupt healthy blood flow and cut down even more on the amount of oxygen flowing to body tissues. Since 1952 the data for SCD has been available when Lehman H. and Catbrush first recorded the Sickle cell traits in Southern India (Lehman & Catbrush, 1952).

The sickle  $\beta$  globin gene is spread widely throughout Africa, the Middle East, Mediterranean countries, and India and has been carried, by population movement, to the Caribbean, North America, and Northern Europe. The frequency of sickle cell carriers is up to 1 in 4 in West Africans and 1 in 10 in Afro-Caribbeans (Department of Health, 1993). It is now firmly reported that these genes harbor amongst different caste groups but with very high prevalence amongst scheduled caste, scheduled tribes and other backward communities (Bhatia & Rao, 1987 and Sharma, 1983). Shukla and Solanki (1985) were the first to report the disease in Vidarbha region of Maharashtra with prevalence from 9.4% to 22.2% in non-tribal population. Prevalence of 5.5% SCD population from few villages of Wardha District has been reported by Ankushe (1993). In addition to this, most of the studies carried out in India and elsewhere have reported comparison of SCD patients with normal population with respect to various anatomical, haematological, and anthropometric parameters of normal and SCD populations. The present study is a small attempt to find out the sickle cell allele frequency and prevalence of SCD along with the haematological parameters in some selected tribal population of forest regions of Amravati district and Yavatmal district.

### 2. Material and Methods

Screening of SCD was conducted in 7 tribal villages from Melghat forest region of Amravati district and 8 tribal villages from Yavatmal district. A total of 436 blood samples from individuals belonging to 5 different tribal castes of Amravati district and 115 blood samples from Yavatmal district were collected by either door to door screening or organizing screening camps in co-ordination with the officials from Primary Health Centres and with written consent of tribals. Few drops of blood were collected by bold finger prick for performing the solubility test for preliminary diagnosis of SCD (Huntsman *et al.*, 1970). Blood samples of positive solubility test were later subjected to capillary electrophoresis (Jenkins & Ratnaik, 2003 and Gulbis *et al.*, 2003). Complete blood count of blood samples were performed on Beckman Coulter Counter (Bourner, Dhaliwal & Sumner, 2005 and Fernandez *et al.*, 2001) in the laboratory of Anthropological survey of India, Nagpur Central Regional Centre, Nagpur to confirm SCD. Allele frequency was calculated using Hardy Weinberg Principle.

### 3. Results and Discussion

In the present work individuals of 5 tribal castes from Amravati i.e. Bhil, Gawlan, Gowari, Korku and Nihal were found to be suffering from SCD in the study area. The allele frequency of sickle cell gene was found to be highest in the Bhil tribal group (0.4934) followed by Gawlan (0.4071), Korku (0.3294) and Nihal (0.2898) and was lowest in the

Gowari (0.2871) (Table 1). Similarly, highest allele frequency of Sickle cell gene was found in Pardhans (0.5750) of Yavatmal district, followed by Gowaris and Madgis (0.50), Gonds (0.4243) and lowest in Kolams (0.350) (Table 2). The prevalence of sickle cell gene has been reported in many parts of India including central India, where the prevalence in the different communities ranges from 9.4% to 22.2 %5 (Shukla and Solanki, 1985). According to studies carried out by Kate (2001), 10% of total population of the state of Maharashtra belongs to tribal population groups. Kate also reported SCD prevalence of 10% among the korku tribes of Amaravati District and a very high prevalence amongst the tribal population groups of Nandurbar and Gadchiroli districts of the state.

Table1: Data showing the values of Allele Frequencies of five tribal communities from Amravati District.

Population (n)	Allele Frequency	
	A	S
Korku (n=212)	0.6706	0.3294
Bhil (n=53)	0.5066	0.4934
Gawlan (n=54)	0.5929	0.4071
Gowari (n=54)	0.7129	0.2871
Nihal (n=63)	0.7102	0.2898

Table2: Data showing the values of Allele Frequencies of six tribal communities from Yavatmal District.

Population (n)	Allele Frequency	
	A	S
Gond (n=33)	0.5757	0.4243
Kolam (n=10)	0.650	0.350
Pardhan (n=60)	0.425	0.575
Gowari (n=3)	0.50	0.50
Madgi (n=4)	0.50	0.50
Banjara (n=5)	0.80	0.20

The complete blood count (CBC) of the tribals from both Districts Amravati and Yavatmal was computed and it was found that the tribals belonging to Amravati District had slightly lesser red blood cell (RBC) count ( $3.63 \pm 0.36/\mu\text{l}$ ) than the tribal of Yavatmal District ( $3.851 \pm 0.09/\mu\text{l}$ ) (Table 3). Similarly, the overall mean haemoglobin concentration (Hgb) for cases (SS) from Amravati District was ( $8.22 \pm 0.77\text{g/dl}$ ) while the overall mean haemoglobin concentration (Hgb) was slightly higher for cases (SS) from Yavatmal District ( $9.27 \pm 0.36\text{g/dl}$ ) (Table 4). The values of mean cell volume (MCV) of diseased cases (SS) were found to be higher than the control group (AA) from both the Districts. Whereas, mean cell haemoglobin (MCH) and Corpuscular Haemoglobin Concentration (MCHC) were found to be lower in cases (SS) as compared to control. On the basis of significance level WBC, RBC, Hgb, Hct, MCV, PLT, MCHC, MO, LY, LY#, GR# and RDW were found to be highly significant except MCH, GR, MO# and MPV in SS individuals of Amaravati district. Whereas, in SS of Yavatmal District WBC, RBC, Hgb, Hct, MCV, PLT, MCH, MCHC, MO, LY, LY#, GR, GR# and RDW were found to be highly significant except MO# and MPV. Homozygous sickle cell disease patients have lower values of red cell parameters, but higher values of white cell and platelets counts compared to haemoglobin phenotype AA controls (Akinsegun *et al.*, 2012). The rate of chronic haemolysis associated with sickle cell anaemia patients could account for these lower values. There is also a blunted response to erythropoietin secretion in sickle cell anaemia; the rate of increase is not proportional to the degree of anaemia (Sherwood *et al.*, 1987). This may be due to right—shifted haemoglobin dissociation curve seen in sickle cell disease (Morris *et al.*, 1991). Similarly lower values were obtained by Omoti in Benin City, Nigeria (Omoti *et al.*, 2005) amongst homozygous sickle cell disease patients in steady state.

Table3: Data showing the values of Haematological parameters of the Sickle cell Positive Tribal Population as Compared with the Normal individuals from Amravati District.

Parameters	Sickle cell patient (SS) (n=30)		Normal (AA) (n=10)	
	Mean±S.E.	Range	Mean±S.E.	Range
W.B.C.	$10.22 \pm 0.87^{***}$	5.8-19.6	$7.97 \pm 0.70$	4.5-10.6
R.B.C.	$3.27 \pm 0.18^{***}$	1.75-5.04	$5.17 \pm 0.26$	4.30-6.8
Hgb	$8.73 \pm 0.48^{***}$	6.2-13.5	$14.04 \pm 0.75$	11.7-18.9
Hct	$26.48 \pm 0.80^{***}$	20-35.1	$36.75 \pm 2.44$	22-50.2
MCV	$84.39 \pm 1.76^{***}$	70-92	$70.7 \pm 3.37$	62-79.4
MCH	$25.95 \pm 1.10^{ns}$	18.8-36.1	$33.20 \pm 4.53$	18.9-78.3
MCHC	$31.075 \pm 0.91^{***}$	25.9-44.1	$33.05 \pm 0.84$	29.3-38
Plt	$332.2 \pm 21.55^{***}$	192-555	$297.91 \pm 25.95$	249-535
LY	$45.23 \pm 2.33^{***}$	28.3-60.6	$28.65 \pm 1.31$	21.9-35
MO	$17.74 \pm 1.41^{***}$	3.2-27.1	$10.50 \pm 2.15$	4.1-27.7
GR	$36.66 \pm 2.55^{ns}$	22.3-64.1	$38.81 \pm 3.75$	24.4-69.3
LY#	$5.47 \pm 0.55^{***}$	2.1-9.7	$3.45 \pm 0.40$	1.7-5.6
MO#	$1.52 \pm 0.26^{ns}$	0.1-4.8	$1.56 \pm 0.41$	0.4-4.5
GR#	$3.69 \pm 0.31^{***}$	1.9-6.1	$4.45 \pm 0.20$	3.5-5.4
RDW	$19.82 \pm 0.82^{***}$	12.7-25.5	$13.54 \pm 0.57$	10.8-17.2
MPV	$8.72 \pm 0.29^{ns}$	7.3-12	$10.81 \pm 1.12$	7.5-21.9

Table 4: Data showing the values of Haematological parameters of the Sick cell Positive Tribal Population as Compared with the Sick cell Gene Carriers and Normal individuals from Yavatmal District.

Parameters	Sickle cell patient (SS) (n=30)		Normal (AA) (n=10)	
	Mean±S.E.	Range	Mean±S.E.	Range
W.B.C.	9.80±0.61***	6-21.2	8.27±0.31	5.3-12.9
R.B.C.	3.80±0.17***	1.25-5.89	5.17±0.10	3.99-5.83
Hgb	9.26±0.31***	4.0-12.4	12.90±0.33	9.4-15.7
Hct	29.42±0.97***	12.9-36.2	39.91±0.92	32.8-49.8
MCV	78.62±2.01***	57.5-99.3	75.80±2.36	57.5-95.4
MCH	24.85±0.71***	17.9-31.6	25.30±0.68	20.8-33.7
MCHC	31.57±0.24***	29.7-33.4	32.35±0.22	30.0-34.4
Plt	322±29.21***	127-795	258.56±11.62	191-464
LY	42.49±1.62***	29-58.3	37.14±1.39	25.2-49.1
MO	7.43±0.94***	1.7-14.8	6.00±0.57	1.7-10.2
GR	49.48±2.19***	32.4-65.4	56.49±1.63	37.7-76.63
LY#	4.33±0.44***	2.4-14	2.97±0.21	2.0-4.6
MO#	0.82±0.14 <sup>ns</sup>	0.1-3.8	0.76±0.18	0.1-1.4
GR#	4.64±0.24***	1.6-6.7	4.79±0.26	3.2-6.1
RDW	18.32±0.74***	9.6-30.7	15.18±0.59	4.1-16.6
MPV	9.84±0.30 <sup>ns</sup>	7.6-12.9	9.54±0.24	7.8-11.9

Values are expressed in Mean±S.E. (Standard error), \*P<0.05, \*\*P<0.01, \*\*\*P< 0.001, ns = non-significant. (WBC-White Blood Cell Count, RBC-Red blood Cell Count, Hb- Haemoglobin Count, HCT- Hematocrit Count, MCV- Mean Corpuscular volume, MCH-Mean Corpuscular Haemoglobin, MCHC-Mean Corpuscular Haemoglobin Concentration, PLT-Platelet Count, LY-Lymphocytes Count, MO- Monocytes Count, GR-Granulocytes Count, LY#- Lymphocyte number, MO#- Monocyte number, GR#- Granulocyte number, RDW Red Blood Cell Distribution Width, MPV-Mean Platelet Volume).

#### 4. Conclusion

In the present study from Amravati district sickle cell disease was found to be highly prevalent in the tribal population of Bhil, Gawlan, Korku, Nihal and less prevalent in Gowaris. While in Yavatmal district sickle cell disease was found to be highly prevalent in the tribal population of Gond, Kolam, Pardhans, Govaris and Madgis and less prevalent in the Banjaras. Homozygous sickle cell disease patients (SS) have lower values of red cell parameters, but higher values of white blood cell and platelets counts compared to haemoglobin phenotype AA (controls).

#### Acknowledgment

Authors are grateful to Anthropological survey of India, Nagpur Central Regional Centre for providing the lab facilities and their guidance wherever needed.

#### References

- Akinsegun, Akinbami, Adedoyin Dosunmu, Adewumi Adediran, Olajumoke Oshinaike, Phillip Adebola and Olanrewaju Arogundade. (2012). Haematological values in homozygous sickle cell disease in steady state and haemoglobin phenotypes AA controls in Lagos, Nigeria. *BMC Research Notes*, 5, pp. 396.
- Ankushe, RT. (1993). Clinico-epidemiological Study of Sickle Cell Disorder in Rural Population of Wardha District. M.D. Thesis, Nagpur: University of Nagpur.
- Bhatia, H.M and Rao, V.R. (1987). *Genetic Atlas of the Indian Tribes*, Institute of Immunohaematology, (ICMR), Bombay, India.
- Bourner, G. Dhaliwal, J. and Sumner, J. (2005). Performance evaluation of the latest fully automated hematology analyzers in a large, commercial laboratory setting: a 4-way, side-by-side study. *Laboratory Hematology*. 11, pp. 285–297.
- Department of Health. , (1993). Report of a working party of the Standing Medical Advisory Committee on Sickle Cell, Thalassemia, and other Haemoglobinopathies. London: HMSO.
- Fernandez, T., Domack, L.B., Montes, D., Pineiro, R., Landrum, E., and Vital, E. (. 2001). Performance evaluation of the coulter LH 750 hematology analyzer. *Laboratory Hematology*. 7, pp. 217–228.
- Gulbis, B, Fontaine, B. Vertongen, F. Cotton, F (2003). The place of capillary electrophoresis techniques in screening for haemoglobinopathies. *Ann Clin Biochem*. 40, pp. 659-662.
- Huntsman, R.G., Barclay, G.P., Canning, D.M. and Yawson, G.I. (1970). A rapid whole blood solubility test to differentiate the sickle-cell trait from sickle-cell anaemia, *J Clin Pathol*. 23(9), pp. 781- 783.
- Ingram, V.M. (1956). A specific chemical difference between the globins of normal human and sickle- cell anaemia haemoglobin, *Nature* 178, pp. 792- 794.
- Jenkins, M. and Ratnaik, S. (2003). Capillary electrophoresis of haemoglobin. *Clin Chem Lab Med*. (41). pp. 747-754.
- Kate, S.L. (2001). Health problems of tribal population groups from the state of Maharashtra, *Indian J Med Sci*, 55, pp. 99-108.
- Lehman, H. and Catbrush, M. (1952). Sickle cell trait in Southern India. *British medical journal*, pp. 404- 405.
- Morris, J., Dunn, D., Beckford, M., Grandison, Y., Mason, K., Higgs, D.R., De Ceulaer, K., Serjeant, B.E., Serjeant, G.R. (1991). The haematology of homozygous sickle cell disease after 40. *Br J Haematol*, 77, pp 382–385.
- Omoti, C.E. (2005). Haematological values in sickle cell anaemia in steady state and during vaso-occlusive crises in Benin City, Nigeria. *Ann Afr Med*, 4(2), pp 62–67.

- Sharma, A. (1983). Hemoglobinopathies in India. Peoples of India, XV International Congress of genetics, *New Delhi India, Dec*, pp. 12-21.
- Sherwood, J.B., Goldwesser, E., Chilcoat, R., Carmichael, L.D. & Nagel, R.L. (1987). Sick cell anaemia patients have low erythropoietin levels for their degree of anaemia. *Blood*, 67, pp 46–49.
- Shukla R. M. and Solanki B.R. (1985). Sick trait in Central India. *Lancet* 1, pp. 297-298.