International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

A Study of Hemoglobinopathies in Children with Microcytic Hypochromic Anemia in Southern Part of Assam

[1]Dr. Alomita Paul Choudhury, [2]Dr. Aniruddha Debnath, [3]Dr. R. N. Chaubey
PGT , Department of Pathology, SilcharMedical College and Hospital, Assam
Assistant Professor, Department of Pathology, SilcharMedical College and Hospital, Assam
Professor, Department of Pathology, SilcharMedical College and Hospital, Assam
Corresponding Author: Dr.Alomita Paul Choudhury, Department of Pathology, Silchar Medical College and Hospital.

Date of Submission: 05-04-2025 Date of Acceptance: 15-04-2025

ABSTRACT:
Background: India is one of the nations most affected by thalassemia and hemoglobinopathies where roughly 27 million babies are born each year with pathological hemoglobinopathies.[1]The objective of the study is to find the prevalence of hemoglobinopathies in the southern part of assam among the pediatric patients using HPLC method along with clinico-hematological correlation.
Material and Method: The study was conducted in the Department of Pathology, Silchar Medical College and Hospital, Assam,India from 01-06-2021 to 31-05-2022 using HPLC method in patients who presented with pallor in Pediatric OPD.
Results: A total number of 120 cases were assessed during the study period, of which 69(57.50%) were detected withabnormalhemoglobins.17 cases (24.63%) were HbE trait, 8 cases (11.59%) were HbE disease, 14 cases (20.28%) were Beta thalassemia trait, 9 cases (13.04%) were Beta thalassemia major, 6 cases (8.69%) were HbE-beta thalassemia double heterozygous, 10 cases (14.49%) were HbS trait, 4 cases (5.79%) were HbS disease and one case (1.44%) of sickle cell-beta thalassemia double heterozygous was detected.
Conclusion: .Northeastern region of India is a rich reservoir of hemoglobinopathies and thalassemias. Iron stores are high in thalassemia patients so caution should be taken before advising Iron therapy to an anemic patients. Cation exchange HPLC is an excellent diagnostic tool for detection and quantification of several normal and abnormal hemoglobinopathies with rapid , reproducible and precise results although considerable expertise is required to interpret the data produced.

I. INTRODUCTION:-

A tetramer of two pairs of globin chains makes up haemoglobin. The haemoglobin gene clustersβ and α, which are found on short arm of chromosomes 11 and 16 respectively, are responsible for producing haemoglobin.[2]Diseases are referred to as α or β thalassemia, respectively, when mutations eliminate or decrease the expression of globin genes.Abnormal haemoglobins or Hb variations are produced when mutations in the globin genes modify their structural makeup.[3]Haemoglobin A (between 95% and 98%), Hb A2 (between 2% and 3.5%), and Hb F (up to 2%) are examples of normal haemoglobin fractions. Hb F in the foetus is produced as main haemoglobin during gestation.[4] The abnormal hemoglobins so far detected in India include Hb D, E, H, J, K, L, M, Q, S, Lepore, Norfolk, Koya Dora, Chandigarh and the hereditary persistence of HbF.[5] The sickle cell haemoglobin (S), hemoglobin-E, and hemoglobin-D are the most commonly discovered aberrant hemoglobins in India. [6]Microcytic hypochromic anaemia is most common in our population.[7]Iron deficiency and haemoglobin abnormalities are the primary causes of anaemia. Thalassaemia and hemoglobinopathies can be screened and diagnosed using RBC indices like total RBC count, MCV, MCH, and RDW. Final diagnosis should be made by High Performance Liquid Chromatography or electrophoresis of haemoglobin.[8]High Performance Liquid Chromatography of haemoglobin is a powerful, excellent diagnostic tool for direct identification of different haemoglobin variants with a high degree of precision in the quantification of major and minor, normal and abnormal, haemoglobin fractions.[9]It is generally known that the North Eastern area of India has one of the highest rates of HbE gene occurrence anywhere in the globe. [10]Iron therapy is hazardous in hemoglobinopathies due to the risk of iron toxicity in the tissue, so evaluations of iron status are necessary before iron therapy.

II. MATERIALS AND METHODS:-

The cross sectional study was carried out at Silchar medical college and hospital during June

2021 to May 2022. Total 120 cases were assessed. Children less than 12 years age attending the pediatrics outpatient department of silchar medical college who presented with pallor and the patients who were diagnosed to have microcytic hypochromic anemia on peripheral blood smear and complete blood count were included in the study.. Patients above 12 years age and anemic patients having cause other than microcytic hypochromic anemia and confirmed cases of iron deficiency anemia were excluded from the study.. A detailed history, complete general and systemic examination of the patients were performed after fulfillment of inclusion and exclusion criteria. All patients were duly informed about the study and informed and written consent was obtained from all the cases and/or attendants. Samples were collected in K3EDTA vials for CBC, PBF and HPLC and clotted vials for Iron Studies. Complete blood count was done on Sysmex XN 550 automated hematology analyser which dispenses a 26 parameter report. Peripheral blood film using Leishman stain and Iron Profile study was done in Beckman coulter fully automated analyser to rule out iron deficiency anemia and to find out suspected case of hemoglobinopathies. . Liquid chromatography is a well-established technique for the separation of substances. High performance liquid chromatography (HPLC) is a suitable method for the analysis of a wide range of application areas. The suspected case samples were run in HPLC machine to detect and quantify the burden of hemoglobinopathy and assess their clinico-hematological pattern. HPLC was done in all cases by fully automated BIORAD D10 machine. This machine can differentiate 24 different hemoglobin variants.

III. RESULTS:-

During the one year study ,120 patients were assessed for abnormal hemoglobins and a total of 69 cases were found to have abnormal hemoglobins.

Table 1: Table showing percentage of abnormal hemoglobins

Total cases	Abnormal hemoglobins	Percentage
120	69	57.50%

Table 2: Table showing percentage of different hemoglobinopathy cases

Abnormal hemoglobins	**Total cases**	**Percentage**
HbE trait	17	24.63%
HbE dis	8	11.59%
Thal trait	14	20.28%
Thal maj	9	13.04%
HbEthal	6	8.69%
HbS trait	10	14.49%
HbS dis	4	5.79%
Sickle/β thal	1	1.44%

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

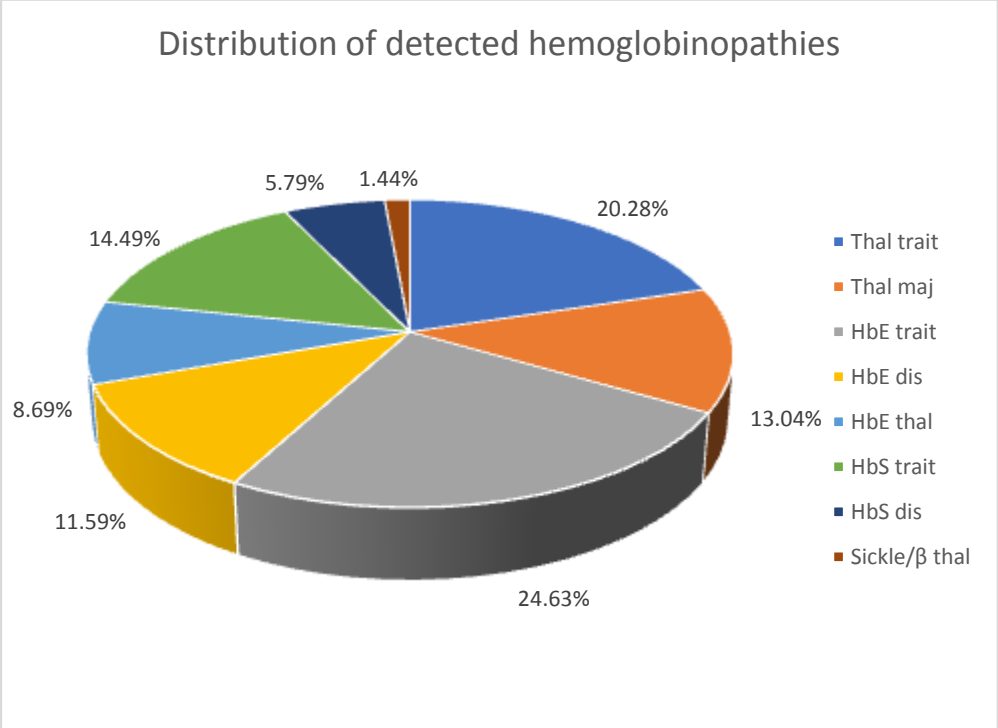


Figure 1: Pie chart showing distribution of detected hemoglobinopathies

Caste	HbE trait	HbE dis	Thal trait	Thal maj	HbEthal	HbS trait	HbS dis	Sickle/β thal
ASSAMESE	1	0	1	0	0	0	0	0
BENGALI HINDU	3	0	5	2	0	0	0	0
BENGALI MUSLIM	5	3	5	2	1	0	0	0
BORO	0	0	1	0	0	0	0	0
TEA TRIBE	6	5	2	5	4	10	4	1
CHRISTIAN	0	0	0	0	0	0	0	0
KHASI	1	0	0	0	0	0	0	0
DIMASA	1	0	0	0	1	0	0	0

Table 3: Hemoglobinopathies in different Ethnic community

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

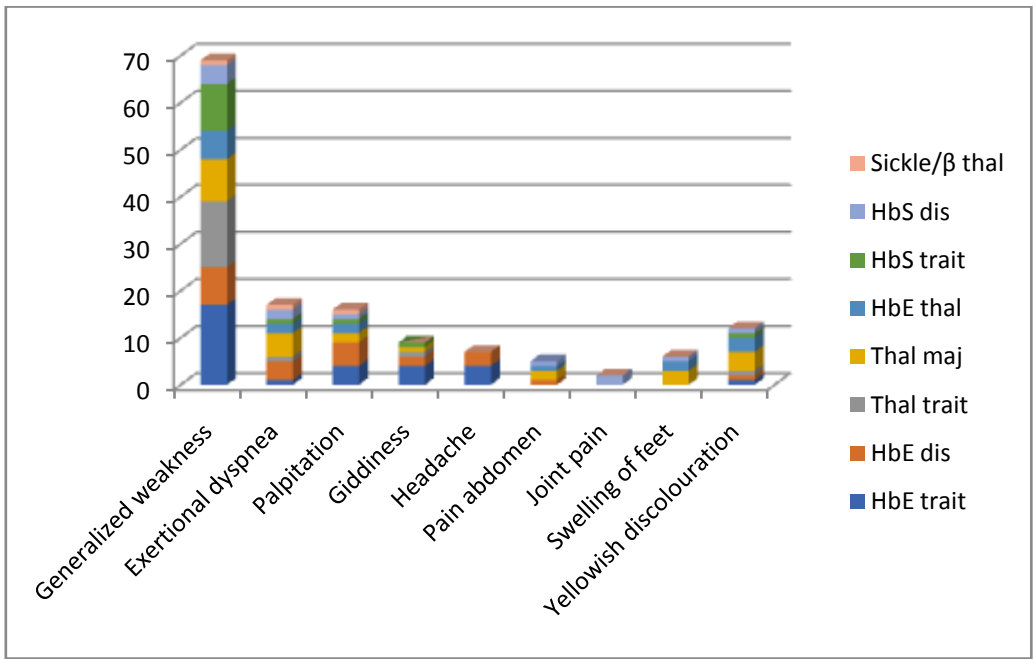


Figure 2: Histogram showing various symptoms in hemoglobinopathy patients

Signs	HbE trait	HbE dis	Thal trait	Thal maj	HbEthal	HbS trait	HbS dis	Sickle/β thal
Pallor	6	5	3	8	6	2	4	1
Icterus	1	1	1	2	2	0	1	0
Oedema	0	0	0	2	1	0	0	0
Splenomegaly	0	2	0	6	5	0	2	1
Hepatomegaly	0	0	0	3	4	0	0	0

Table 4: Various signs in the hemoglobinopathy patients

Table 5: RBC morphology in different hemoglobinopathies

Hemoglobinopathies	Microcytosis	Anisicytosis	Poikilocytosis	Hypochromia	Target cells	Nucleated RBC	Sickle cells
HbE trait	17	5	3	13	17	1	0

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

HbE dis	8	4	2	8	8	2	0
Thal trait	14	6	2	14	14	0	0
Thal maj	9	9	9	9	9	9	0
HbEthal	6	6	6	6	6	6	0
HbS trait	8	5	4	8	7	1	6
HbS dis	3	4	4	4	4	1	4
Sickle/β thal	1	1	1	1	1	1	1
Total	66	40	31	63	66	21	11

TABLE 6: HEMOGLOBIN FRACTIONS IN VARIOUS HEMOGLOBINOPATHIES

Table 6(a): HbE trait:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	60.14	30.19	1.3	1.88	2.49	1.64	1.23	0.69	0	0.88
Range	54.6-69.7	24.8-34.2	0.9-1.6	0-4.3	1.1-4.6	0-3.1	0-1.9	0-1.8	0	0-3.2
SD	3.82	2.26	0.64	1.28	0.99	1	0.55	0.6	0	1.15

Table 6 (b): HbE disease:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	5.81	81.2	5.45	1.76	2.61	1.27	1.08	0.86	0	0.98
Range	4.1-7.3	76.6-93.4	2.6-7.1	0-3.4	1.3-5.1	0-2.1	0.6-1.7	0.2-1.6	0	0-4.4
SD	1.06	5.35	1.67	1.24	1.3	0.83	0.43	0.48	0	1.77

Table 6 (c): Thalassemia trait:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	85.21	6.4	2.36	1.25	1.8	0.89	0.72	0.31	0	1.17
Range	80.3-87.7	4.4-7.5	0.9-3.5	0-6.1	0-4.9	0-1.6	0-1.8	0-1.2	0	0-2.9
SD	2.2	0.86	0.54	1.65	1.06	0.61	0.57	0.41	0	0.9

Table 6 (d): Thalassemia major:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	2.36	4.36	0	0.5	2.2	78.28	11.11	0.85	0	0.31
Range	1.6-3.1	3.4-4.9	0	0-2.2	1.2-4.2	74.4-81.3	8.4-13.9	0-1.6	0	0-1.9
SD	0.53	0.52	0	0.81	1.08	2.01	1.63	0.63	0	0.67

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

Table 6 (e): HbEβ thalassemia:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	3.96	60.35	2.53	1.36	1.88	23.96	7.28	1.16	0	0.25
Range	1.5- 11.4	52.4-85.9	0- 13.1	0- 2.3	0-3	0- 33.6	3.2- 9.8	0- 1.6	0	0- 1.5
SD	3.68	12.75	5.24	1.08	1.08	12.08	2.25	0.58	0	0.61

Table 6 (f): HbS trait:

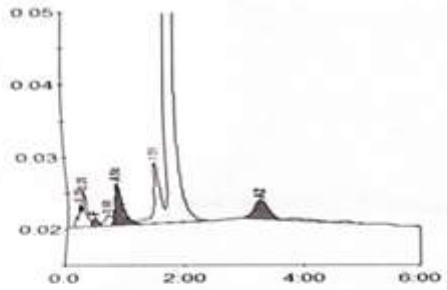
	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	58.16	1.6	1.04	1.43	2.49	1.31	1.31	0.62	31.61	1.02
Range	51.2- 64.2	0- 3.4	0- 1.6	0-6.3	1.6- 4.1	0- 2.7	0- 2.2	0- 1.7	27.8- 38.2	0- 2.9
SD	3.48	0.8	0.67	1.89	0.7	0.81	0.65	0.68	2.75	1.07

Table 6 (g): HbS disease:

	HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
Mean	4.8	2.85	10.07	0	1	0.22	1.22	0.17	79.15	0.22
Range	4.4- 5.2	2.3-3.4	8.2- 12.2	0	0- 1.6	0- 0.9	0- 2.5	0- 0.7	78.2- 80.9	0- 0.9
SD	0.33	0.53	1.65	0	0.69	0.45	1.05	0.35	1.19	0.45

Table 6 (h): Sickle cell/β thalassemia:

HbA(%)	HbA2(%)	HbF(%)	HbA1c(%)	P3(%)	LA1c/C(%)	HbA1a(%)	HbA1b(%)	HbS(%)	Unknown(%)
6.3	8.2	19.5	0	0	0	3.6	0	64.3	0

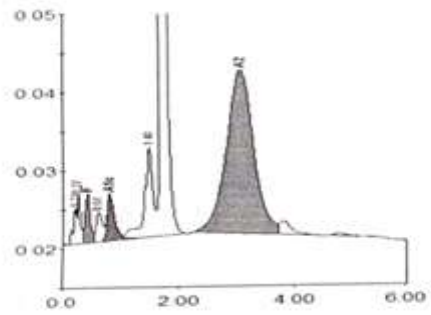


Peak	R.time	Height	Area	Area %
A1a	0.20	3308	12519	0.7
A1b	0.28	5172	21621	1.2
F	0.45	1213	8542	< 0.8 *
LA1c/CHb-1	0.68	1511	11326	0.6
A1c	0.84	5788	55635	4.6
P3	1.51	8465	69999	3.8
A0	1.75	357980	1609909	87.7
A2	3.26	2668	46416	2.6
Total Area:	1835966			

Concentration:	%	mmol/mol
F	< 0.8 *	---
A1c	4.6	27
A2	2.6	---

Figure 3: Normal

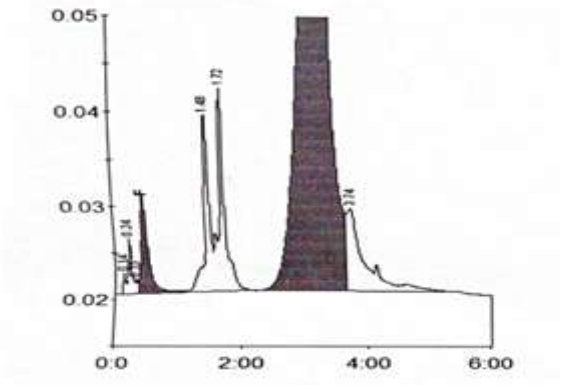
International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018



Peak	R.time	Height	Area	Area %
A1a	0.23	4515	23187	0.9
A1b	0.27	6323	19446	0.8
F	0.42	6315	36595	1.3
LA1c/CHb-1	0.61	3783	28872	1.1
A1c	0.81	5924	53228	4.3
P3	1.48	11468	118399	4.6
A0	1.70	339663	1586729	62.0
A2	3.04	20957	694600	30.5 *
Total Area:	2561055			

Concentration:	%	mmol/mol
F	1.3	---
A1c	4.3	23
A2	30.5 *	---

Figure 4: HbE trait

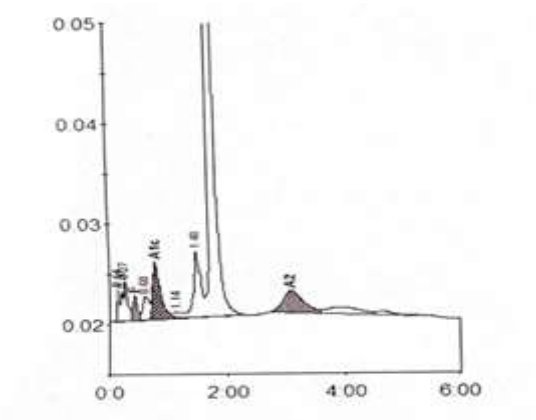


Peak	R.time	Height	Area	Area %
Unknown	0.14	2283	4720	0.2
A1a	0.24	6232	23783	1.0
A1b	0.33	1577	4743	0.2
F	0.47	10175	72398	3.4
P3	1.48	18963	115161	5.1
A0	1.72	21732	143108	6.3
A2	3.17	59208	1785286	93.4 *
Unknown	3.74	8867	123467	5.4
Total Area:	2272665			

Concentration:	%	mmol/mol
F	3.4	---
A2	93.4 *	---

Figure 5: HbE disease

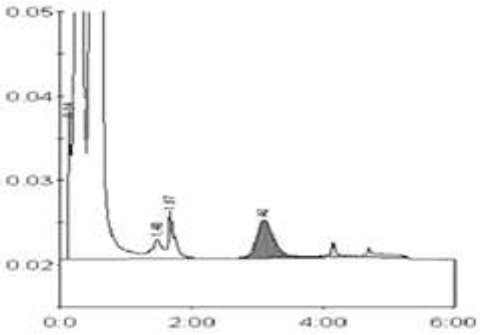
International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018



Peak	R.time	Height	Area	Area %
Unknown	0.14	3370	6449	0.6
A1a	0.20	3056	12169	1.0
A1b	0.27	3983	14241	1.2
F	0.43	2449	13553	0.9
LA1c/CHb-1	0.60	2326	18140	1.6
A1c	0.78	5612	50511	6.1
Unknown	1.14	623	6738	0.6
P3	1.48	6577	56669	4.9
A0	1.74	224992	936255	80.3
A2	3.08	2173	51674	6.1
Total Area:	1166399			

Concentration:	%	mmol/mol
F	0.9	---
A1c	6.1	44
A2	6.1	---

Figure 6: Thalassemia trait

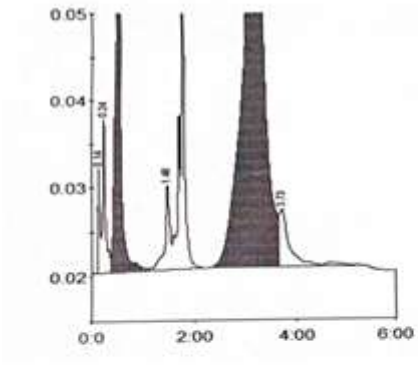


Peak	R.time	Height	Area	Area %
Unknown	0.14	17738	32035	1.5
A1b	0.29	73283	549704	25.3
LA1c/CHb-1	0.58	177176	1448953	66.8
P3	1.48	2242	24979	1.2
A0	1.67	5642	35275	1.6
A2	3.09	4465	79029	4.2
Total Area:	2169976			

Concentration:	
% A2	4.2

Figure 7: Thalassemia major

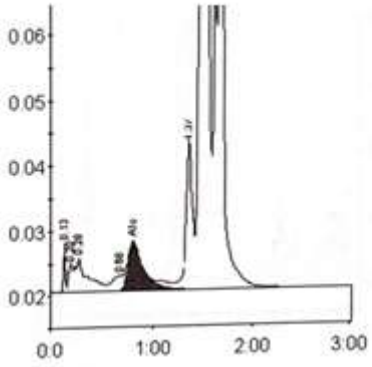
International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018



Peak	R.time	Height	Area	Area %
Unknown	0.14	12448	24202	1.1
A1a	0.24	17497	66951	3.2
F	0.51	35945	264394	13.1
P3	1.48	9523	64193	3.0
A0	1.78	43920	240294	11.4
A2	3.22	47816	1363615	85.9 *
Unknown	3.73	6477	86346	4.1
Total Area:	2109995			

Concentration:	%	mmol/mol
F	13.1	---
A2	85.9 *	---

Figure 8: HbE -β thalassemia (double heterozygous)

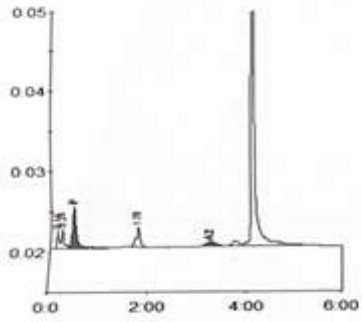


Peak	R.time	Height	Area	Area %
Unknown	0.13	7678	9422	0.4
A1a	0.20	4591	13413	0.5
A1b	0.28	5343	43577	1.7
LA1c/CHb-1	0.68	2329	17693	0.7
A1c	0.81	7404	77290	6.3
P3	1.37	23435	104787	4.1
A0	1.48	357969	1407810	55.7
S-Window	1.67	489999	853066	33.8
Total Area:	2527057			

Concentration:	%	mmol/mol
A1c	6.3	46

Figure 9 : sickle cell trait

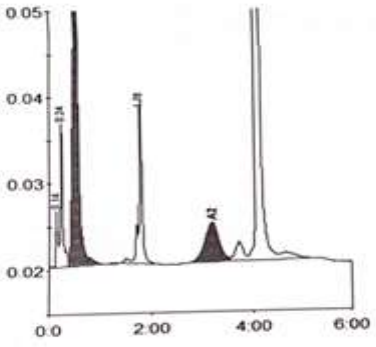
International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018



Peak	R.time	Height	Area	Area %
Unknown	0.14	2358	5738	2.0
A1a	0.24	2515	7018	2.5
F	0.47	5148	26435	10.2
A0	1.78	2418	13701	4.9
A2	3.23	462	6726	2.5
S-Window	4.14	54550	221709	78.8
Total Area:	281328 *			

Concentration:	%	mmol/mol
F	10.2	---
A2	2.5	---

Figure 10 : sickle cell disease



Peak	R.time	Height	Area	Area %
Unknown	0.14	6833	16279	1.1
A1a	0.24	16431	51209	3.6
F	0.52	38064	266456	19.5 *
A0	1.78	19100	90810	6.3
A2	3.19	4575	88963	8.2
S-Window	4.13	199070	924445	64.3
Total Area:	1438161			

Concentration:	%	mmol/mol
F	19.5 *	---
A2	8.2	---

Figure11: Sickle cell- beta thalassemia (double heterozygous)

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

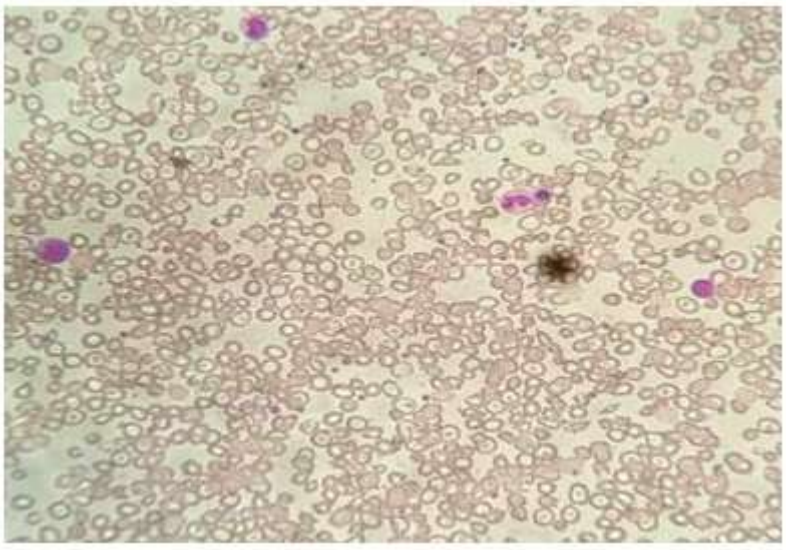


Figure 12: Peripheral blood smear of thalassemia major case showinganisopoikilocytosis,microcytosis, hypochromia, target cells and nucleated RBC.

Figure 13: Buffer 1 , Buffer 2 and Wash buffer used in Biorad D10 HPLC machine

International Journal Dental and Medical Sciences Research
Volume 7, Issue 2, Mar - Apr 2025 pp 252-265 www.ijdmsrjournal.com ISSN: 2582-6018

IV. DISCUSSION:-

In the present study, out of 120 suspected cases abnormal hemoglobins were detected in 69 cases, out of which HbE (HbAE=17, HbEE=8) was detected in 25 cases. 14 cases of thalassemia minor, 9 cases of thalassemia major, 6 cases of HbEβ thalassemia and 17 cases of HbS were found where 14 cases with 10 cases of HbS traits and 4 cases of HbS disease. Also there was 1 case of sickle cell/β thalassemia.

Table7 : Comparison showing occurrence of HbE in different series

Series	HbE Trait	HbE Disease	HbE/β thalassemia
Shrivastav et al. [11]	0.15%	0.14%	0.06%
Bhavani et al. [12]	1.31%	0.7%	0.2%
Teli et al. [13]	1.16%	0.33%	0.33%
Chandrashekar et al. [14]	23.2%	18.9%	4.6%
Philip et al.[15]	0.85%	0.46%	0.06%
Goswami et al. [16]	34.4%	25.3%	15.1%
Baruah et al.[17]	25.48%	21.02%	1.26%
Present study	24.63%	11.59%	8.69%

Table8 : Comparison showing percentage of other hemoglobinopathies

Series	Thal Minor	Thal Major	SCT	SCD
Shrivastav et al. [11]	11.55%	4.02%	2.95%	1.17%
Bhavani et al. [12]	22.19%	0.6%	0.4%	0%
Teli et al. [13]	8.64%	1.25%	16.53%	4.9%
Chandrashekar et al. [14]	37.9%	2.3%	5.3%	1.4%
Philip et al.[15]	10.49%	0.55%	1.24%	0.11%
Goswami et al. [16]	17.8%	1.5%	1.1%	0.1%
Baruah et al.[17]	3.48%	0.36%	2.10%	2.26%
Present study	20.28%	13.04%	14.49%	5.79%

Among the HbE variants like the HbE trait, HbE disease and HbE β thalassemia, 15 cases was found in tea tribe followed by 12 cases of Bengalis. Thalassemia minor was found more common among the Bengali Muslims and thalassemia minor among the Tea tribes. The Sickle cell Hemoglobinopathies were found exclusively amongst the Tea Garden labourers. Hazarika D et al has also found HbS gene mainly restricted to the tea garden community. In their study they also found HbE trait, HbE β thalassemia, thalassemia major and sickle cell/β thalassemia among the tea garden community. [18]

Generalised Weakness, dyspnea on exertion and palpitations were the commonest presenting symptoms in the present study which is obvious due to the fact that the RBCs of the patients with hemoglobinopathy are unable to carry oxygen as the normal hemoglobin. In a study done by Pathak et al. the common clinical symptoms were generalized weakness present in 50% cases of HbE Trait, 87.5% cases of HbE Disease, 62.5% cases of Thalassemia Minor, 91.66% cases of HbE βThalassemia, 71.43% cases of sickle cell trait and 100% patients of sickle cell disease, thalassemia major and sickle cell/β thalassemia.[19]

Pallor was present in 50.7% cases of Hemoglobinopathies. Icterus was present in 11.59% cases comprising maximum number of HbEβ thalassemia cases and thalassemia major cases. Splenomegaly was found in 23.19% cases, comprising maximum number of cases of thalassemia major and HbE/β thalassemia. Hepatomegaly was found in 10.14% cases comprising maximum number of cases of thalassemia major and HbE/β thalassemia.Karthika et al. reported pallor in 92.15% cases, icterus in 9.80% cases, ascites in 1.96% cases, splenomegaly in 9.80% cases and hepatomegaly in 11.76% cases. [20]Fucharoen S and Weatherall DJ has reported pallor in 39.7% cases, fever in 19.1%, jaundice in 4.2%, edema in 2.4%, abdominal pain and mass in 5.6%. splenomegaly in 97.6% cases and hepatomegaly in 92.6% cases respectively in HbE/β thalassemia cases. [21]

Hematological parameters showed varying degrees of anemia in the different cases. The HbEβ Thalassemia, thalassemia major and sickle cell/β thalassemia cases had severe anemia. Most of the

cases showed reduced MCV, reduced hematocrit and MCH. The WBC count was found to be raised in most cases but it may be an incidental finding or due to inflammation present. The platelet counts were found to be normal in most of the cases.The red cell morphology was mostly microcytic and target cells were seen in all the cases. Anisopoikilocytosis and hypochromia were marked mainly in HbEβ thalassemia, thalassemia major, sickle cell anemia and sickle cell/β thalassemia cases. These cases also showed increase of nucleated red cells. Sickle cell shaped cells were seen in cases of Sickle cell Trait, Sickle cell disease and Sickle cell/β thalassemia.

Iron profile studies showed high iron load in HbEβ thalassemia, thalassemia major, sickle cell disease and sickle cell/β thalassemia cases. The cases with HbE trait, HbE disease, thalassemia minor, HbS trait and HbD trait had a normal iron profile.The iron profiles of the patients with HbE Trait. Both heterozygotes and homozygotes for HbE; the most common form of β-thalassemia, are asymptomatic and minimally anemic, and they have microcytic and hypochromic red blood cells. Since this patients are either asymptomatic or have mild degree of anemia, the requirement for blood transfusion is also very low, further decreasing the chances of Iron overload.

V. CONCLUSION:-

Northeastern region of india is a rich reservoir of hemoglobinopathies and thalassemias. The importance of detailed complete blood count and peripheral blood smear (PBS) examination cannot be understated in the diagnosis of various hemoglobin disorders. Iron stores are high in thalassemia patients so caution should be taken before advising Iron therapy to an anemic patients. Cation exchange HPLC is an excellent diagnostic tool for detection and quantification of several normal and abnormal hemoglobinopathies.The simplicity of sample preparation, accurate quantification of Hb concentration, rapid , reproducible and precise results combined with complete automation, makes HPLC an ideal methodology for the routine diagnosis of Hb disorders. Although considerable expertise is required to interpret the data produced. Many hemoglobins may have same retention times as normal hemoglobins or other variants and co-inheritance of different traits can further confuse the issue. Definite identification usually requires DNA analysis or amino acid sequencing.

REFERENCES:-

[1]. World population data sheet. Washington DC: Population reference bureau; 2009.
[2]. Michael R, Baun D, Mellisa J, Frei Jones EP. Vichin sky: Hemoglobinopathies: chapter 462: page 2336-2348: Nelson TextBook of Pediatrics. First south Asia edition. 2016;2.
[3]. P.C.Giordano: Carrier Diagnostics and Prevention of Haemoglobinopathies using Capillary Electrophoresis, Companion handbook for the Physician, the laboratory doctor and Genetic counselor 2007:9-10
[4]. Weatherall DJ, Clegg JB. The thalassaemia syndromes. John Wiley & Sons; 2008 Apr 30.
[5]. Balgir RS. The burden of haemoglobinopathies in India and the challenges ahead. Current Science. 2000 Dec 10:1536-47.
[6]. Manna AK, Dutta SK, Chatterjee A. Relative incidence of different thalassaemias and haemoglobinopathies in South Bengal. Journal of the Indian Medical Association. 2009 Jun 1;107(6):347-9.
[7]. Beyan C, Kaptan K, Ifran A. Predictive value of discrimination indices in differential diagnosis of iron deficiency anemia and beta-thalassemia trait. European journal of haematology. 2007 Jun;78(6):524-6.
[8]. Samavat A, Modell B. Iranian national thalassaemia screening programme. Bmj. 2004 Nov 11;329(7475):1134-7.
[9]. Lekhwani S, Vaswani ND. Haemoglobin Variants Detection by HPLC (High Performance Liquid Chromatography) Method. Paediatric oncall [Serial Online]. 2010 Jan;7.
[10]. Deka R, Reddy AP, Mukherjee BN, Das BM, Banerjee S, Roy M, Dey B, Malhotra KC, Walter H. Hemoglobin E distribution in ten endogamous population groups of Assam, India. Human heredity. 1988;38(5):261-6.
[11]. Shrivastav A, Patel U, Joshi JR, Kaur A, Agnihotri AS. Study of hemoglobinopathies and Hb variants in population of Western India using HPLC: A report of 7,000 cases. Journal of Applied Hematology. 2013 Jul 1;4(3):104.
[12]. Bhavani D, Ranjan S. Studies on The Prevalance of Hemoglobinopathies and Thalassemia Among Microcytic Hypochromic Anemia Cases in

Metropolitan City of Chennai, Tamilnadu, India.

[13]. Teli AB, Deori R, Saikia SP. Haemoglobinopathies and β-thalassaemia among the tribals working in the tea gardens of Assam, India. Journal of Clinical and Diagnostic Research: JCDR. 2016 Dec;10(12):LC19.

[14]. Chandrashekar V, Soni M. Hemoglobin disorders in south India. International Scholarly Research Notices. 2011;2011.

[15]. Philip J, Sarkar RS, Kushwaha N. Microcytic hypochromic anemia: Should high performance liquid chromatography be used routinely for screening anemic and antenatal patients?. Indian journal of pathology and microbiology. 2013 Apr 1;56(2):109.

[16]. Goswami BK, Pramanik R, Chakrabarty S, Pal PP, Banerjee S, Bandyopadhyay A. Spectrum of hemoglobin variants in the population of northern region of West Bengal: An ethnogenetic proposition. Journal of family medicine and primary care. 2014 Jul;3(3):219.

[17]. Baruah MK, Saikia M, Baruah A. Pattern of hemoglobinopathies and thalassemias in upper Assam region of North Eastern India: high performance liquid chromatography studies in 9000 patients. Indian Journal of Pathology and Microbiology. 2014 Apr 1;57(2):236.

[18]. Hazarika D, Nath AK, Biswanath P, Borah A, Patir J. A retrospective study on pattern of thalassemia and other haemoglobinopathies in paediatrics age group using high performance liquid chromatography in Jorhat District of North East India. Journal of Evolution of Medical and Dental Sciences. 2016 Jul 14;5(56):3818-23.

[19]. Pathak MS, Borah MS, Kalita D. Disorders of Hemoglobin variants in Paediatric Patients attending in a tertiary care hospital of Northeast India. Int J Biol Med Research. 2014;5(1):3841-6.

[20]. Karthika M, Devi KG, Rymbui DB, Bhardwaj P, Ao S, Kumar S. Prevalence of Hemoglobinopathies in Manipur. Journal of Dental and Medical Science. 2015;14(8):17-20.

[21]. Fucharoen S, Weatherall DJ. The hemoglobin E thalassemias. Cold Spring Harbor perspectives in medicine. 2012 Aug 1;2(8):a011734.