ED/5940 /70 August 22, 1970

Dear Dr. Nagaratnam,

I am sorry that it has taken some time for us to prepare a revised manuscript of our paper. As suggested by you we have rearranged the contents to conform to the title " Hereditary Elliptocytosis Associated with Beta Thalassaemia and a Variant of Rh(D) ". I am glad that you agree to the inclusion of my colleague Dr. Urdevia as one of the authors.

We had to recast the paper and for brevity rearrange the tables and have prepared a family tree on the lines mentioned above. I shall be sending you by separate post two copies of the revised manuscript. Some new references are added including a few on Hereditary Elliptocytosis and beta thalassaemia. You may kindly add the references you have to complete the same. Please feel free to make any corrections or alterations if found necessary.

We feel this paper may be send to either of the two Journals mentioned below.

(1) Human Heredity.
Dr. M. HAGUE, (Editor)
University of Human Genetics,
TAGENSVEJ 14,
COPENHAGEN N.
(DENMARK).

(2) Acta Haematologica.
Prof. Dr. H. LUDIN (Editor-in-Chief),
Medical Clinic,
BURGERSPITAL,
CH-4000 BASEL,
(SWITZERLAND)

LG
22-VIII-70

Kindly acknowledge receipt of the manuscript and do hope that you have no serious objections to the changes made in the write-up.

With kind regards,

Yours sincerely,

P.K. Sukumaran.
Scientific Officer.

DR. N.NAGARATNAM M.D.,M.R.C.P.,
Physician,
Govt. Hospital,
GAMAHA W.P., (CEYLON).

Govt. Hospital.
Kegalle.
Ceylon.
20th Jan. 1968.

My dear Mr. Sukumaran.

Thank you for your letter dated 6th Jan. 1968, and the reports. I hope you would have received the reprints of the article "Thalassaemia in Ceylon" by now, as I sent them to you about a month ago by sea mail.

Regarding the manuscript 'Megaloblastic anaemia complicating Thalassaemia' I have sent it to the Postgraduate Medical Journal after some alterations.

The ~~blood~~ patient ~~sample of~~ L.S. whose blood sample I sent on the last occasion, on subsequent checking did not show any inclusion bodies. This boy was admitted with anaemia, fever and has a very large liver and spleen. Peripheral blood picture did not show any abnormality other than a moderate number of target cells. But x-ray of the bones revealed changes consistent with leukaemia. Subsequently, marrow examination showed that he in fact had leukaemia (lymphatic type). His blood picture still is normal.

I have used the results of the ~~Case~~ haemoglobin analysis of Case 2 in my manuscript "Megaloblastic anaemia complicating Thalassaemia" in another

~~Then~~ article of mine – but I have ~~ackt~~ acknowledged Dr. Sanghvi's and the Institutes help in the matter. I hope he will not mind this, and grant me permission to ~~do~~ so. The article is entitled "Megaloblastic anaemia in Ceylonese Infants" and will appear in the Journal of Trop. Med & Hyg.

I would also require his permission to use 2 or 3 other reports in another article. Should I write to him direct?

Thank you for the trouble you have taken, & wishing you all the very best for the New Year.

Yours truly

N. Nagaratnam.

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BY AIR MAIL

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விமானக் கடிதம்
Aerogramme

ශ්‍රී ලංකා
50 CEYLON 50

P. K. Sukumaran, Esqr.
Scientific Officer.
Cancer Research Institute.
Tata Memorial Centre.
Parel, BOMBAY 12.
INDIA.

දෙවනුව මෙතැනින් නමන්න. பின்பு இங்கே மடியுங்கள். Second fold here.

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அனுப்புபவரின் பெயரும் விலாசமும்
Sender's name and address Dr. N. Nagaratnam
Govt. Hospital
Kegalle, Ceylon.

ගුවන් ලියුමක කිසිවක් බහා නොතිබිය යුතුය. එසේ තිබුනහොත් ඊට වැඩිපුර අයකරනු හෝ සාමාන්‍ය තැපෑලෙන් යවනු හෝ ඇත.

இந்த விமானக் கடிதத்தினுள் வேறு ஏதாவது வைத்தனுப்பப்படுமாயின் மேலதிக கட்டணம் அறவிடப்படும் அல்லது சாதாரண தபால் மூலம் அனுப்பப்படும்.

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ED/690 /68 January 24th, 1968.

Dear Dr. Nagaratnam:

This has reference to your letter dated January 20th, 1968.

Thank you very much for sending me the reprints of the article "Thalassemia in Ceylon" which I received last week. I have noted that you have sent the paper "Megaloblastic Anaemia Complicating Thalassemia" to Post-graduate Medical Journal after corrections.

It is interesting to hear that your patient "L.S." whose blood I examined have turned out to be Lymphatic Leukaemia.

I discussed with Dr. Sanghvi about using the results sent by us on your cases with due acknowledgements. He is of opinion that you may use them after intimating us in advance and that in acknowledging, you may mention my name instead of Dr. Sanghvi's name. Even in the paper you expect to come out in Journal of Tropical Medicine and Hygiene, he would like my name substituted for his, if this could be done at proof stage. Thus he feels that with my name and the official address with the name of Institution would be sufficient for acknowledgement.

If you need any further help from this end, please feel free to write to me.

With kind regards and wishing you very best for the New Year.

Yours sincerely,

P. K. Sukumaran.
Scientific Officer.

24.1.68

Dr. N. Nagaratnam,
Government Hospital,
Kegalli, Ceylon.

cBp/

HVG/ 171 /68 January 8th, 1968

Dear Dr. Nagratnam:

Thank you for your letter dated December 18th, 1967, and the revised manuscript of the paper "Megaloblastic anaemia complicating Thalassemia".

The blood samples from the three families (K.S., L.S., and S.D.) were received but as usual in poor condition due to delay in transit. The reports are sent herewith. No abnormal haemoglobins were detected in any of them. In view of the fact that you found few inclusion bodies, samples from three propositii were subjected to starch-gel electrophoresis. No evidence of Hb-H or Bart's were found in them.

Regarding the above mentioned manuscript, you may, depending on the comments from the American Journal of Disease of Children, send it to Postgraduate Medical Journal or to some local Journal in Ceylon or in India. My comments on the paper as such are the same as I mentioned in my letter dated March 22nd, 1967. Further evidence by laboratory tests and family studies ~~it~~ is advisable for confirmation of Thalassemia in them besides the presence of raised foetal haemoglobin detected. Finding of Hb-A+E could easily be confirmed by family investigation. These investigations would further strengthen our hands. You may please note the change in the name of the institution. It is now Cancer Research Institute, Tata Memorial Centre.

With kind regards,

Yours sincerely,

P. K. Sukumaran.
Scientific Officer.

8.1.68

Dr. N. Nagaratnam,
Government Hospital,
Kegallt,
CEYLON

Encl: reports

cBp/

BLOOD REPORTS

(1) Case No: H-835 Date: January 8th, 1968

Name: Kusuma (KS-3) Sex: Female

Referred by: Dr. N. Nagaratnam Age: 4½ years

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 2.2 %

Electrophoresis: No abnormal haemoglobin detected.

(2) Case No: H-835/1 Sex: Female

Name: Mother (KS-4) Age:

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 5.75 %

Electrophoresis: No abnormal haemoglobin detected.

(3) Case No: H-836 Sex: Male

Name: Lionel Silva (LS-1) Age: 1 year

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 2.95 %

Electrophoresis: No abnormal haemoglobin detected.

contd.....2

BLOOD REPORTS

contd....

(4) Case No: H/836/1 Date: January 8th, 1968

Name: Mother (LS-2) Sex: Female

Referred by: Dr. N. Nagratanam Age:

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 5.0 %

Electrophoresis: No abnormal haemoglobin detected.

(5) Case No: H-837 Sex:=Female

Name: Somawathi (SD-1) Age: 8½ years

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 4.5 %

Electrophoresis: No abnormal haemoglobin detected.

cont....3

BLOOD REPORTS

Case No: H-837/1 Date: January 8th, 1968

Name: Mother (SD-2) Sex: Female

Referred by: Dr. N. Nagaratnam Age:

REPORT:

Sickling Test:

Alkali-resistant Haemoglobin: 1.34 %

Electrophoresis: No abnormal haemoglobin detected.

L.D.Sanghvi,M.Sc.,Ph.D.(Columbia)
Head, Epidemiology Division.

NOTE: As the samples were received in poor condition the possibility of raised foetal haemoglobin due to deterioration is to be ruled out.

NOTE:

This report is for your information and for benefit of the patient. A prior permission has to be obtained in case a part or whole of this report is to be used for citation or publication.

PKS/cBp:

ED/2580 /68

March 27th, 1968

28 MAR 1968

Dear Dr. Nagaratnam:

Thank you for sending blood samples for investigation.

As usual all except one sample (Devika) was received in lysed condition. The raised alkali-resistant haemoglobin found in the lysed samples could be due to plasma proteins interfering in the test. Devika shows normal alkali-resistant haemoglobin and no abnormal haemoglobin on electrophoresis; but shows reduced haemoglobin A_2. In the absence of inclusion bodies, iron deficiency should be ruled out.

I have the manuscript of your paper and shall write to you soon about it.

With kind regards,

Yours sincerely,

P. K. Sukumaran.
Scientific Officer.

27.III.68

Dr. N. Nagaratnam,
Government Hospital,
Kagalle, Ceylon

Encl: Reports

Govt. Hospital
Kegalle.
24-4-68.

Dear Mr. Sukumaran.

Thank you very much for your letter, together with the reports.

I wonder whether you could send me the Editors' addresses of the following Journals. (1) Scandinavian Journal of Haematology (2) Blut. (3) Annales Paediatrici.

Thanking you, and with kind regards.

Yours truly
Ratnatunga.

BY AIR MAIL

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விமானக் கடிதம்
Aerogramme

P. K. Sukumaran Esqr.
Indian Cancer Research
Tata Memorial
Parel, Bombay 12,
India.

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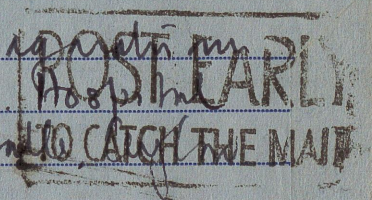
அனுப்புபவரின் பெயரும் விலாசமும்
Sender's name and address

Dr. W. Nagarajan
Govt. Hospital
Vizag



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மாயின் மேலதிக கட்டணம் அறவிடப்படும் அல்லது சாதாரண தபால்
மூலம் அனுப்பப்படும்.

An Aerogramme should not contain any enclosure ; if it does it will be
surcharged or sent by ordinary mail.

Dr. N. NAGARATNAM
M. D. (Ceylon) M.R.C.P. (GLASG)

Kegalle
8. 2. 1968.

Dear Mr. Srikumaran,

Thank you very much for the permission to use your findings in my papers. Unfortunately the proof of my paper - to be published in the Trop. J. of Med & Hygiene, arrived a few days before your letter, and I was unable to make the alterations as suggested by Dr. Singhvi. I have acknowledged his help in the matter.

I enclose a draft of a paper. There are several papers on this topic, and there appears nothing new in this paper, but I thought it worthwhile as there are one or two points of peculiar to this part of the world.

We may send it up to some Journal editor here or in India.

Could you go through this, and make your suggestions.

With regards

Yours truly

[illegible]

I have not looked up very many references. They may be readily accessible to you.

Sincerely

Dr. [illegible]

BONE CHANGES IN CONGENITAL HAEMOLYTIC ANAEMIAS ? IN CEYLON

Clinical and radiological changes in the bones are not uncommonly seen in association with certain disorders of the blood. They tend to occur in conditions where there is either a compensatory hyperplasia of the erythropoietic tissue due to an increased red cell destruction or where there is uncontrolled and widespread proliferation of the leucopoietic cells infiltrating the bone marrow. The former are represented by the hereditary or congenital haemolytic anaemias where the hyperplasia may result in radiological changes in the bones, The latter by the leukaemias and the non-leukaemia malignant diseases of the bone marrow. Radiological bone changes are only rarely seen in other blood diseases, though this have been described in association with iron defiency anaemia (Rajasuriya et al, 1963, Lie-Injo Luan Eng, 1958), pernicious anaemia and erythroblastosis foetalis (Griffiths,).

This paper is an analysis of the bone changes that occur in the congenital haemolytic anaemias as they are seen in Ceylon.

Material

Of the 18 patients under study, 11 were thalassaemia 1 thalassaemia-Hb.H disease, 4 thalassaemia-Hb.E disease and 4 congenital non-spherocytic haemolytic anaemia.

Findings

The congenital haemolytic anaemias in Ceylon comprise the thalassaemia syndromes, the haemoglobinopathies and the non-spherocytic haemolytic anaemias,

I. Thalassaemias

Table I below summarises some of the clinical and haematological data together with the bone changes in the 11 cases, None of the cases showed any changes in the skull.

Three had changes in the small bones of the hands and one of them in the tubular bones as well. One other had rarefaction of the bones of the hands and forearms.

Table I near here

All three that showed bone changes in the X'rays had alkali resistant haemoglobin to the value of 70%. Two of them are now dead. Two had palpable spleens and their Hb. levels were between 2.6 gms% and 3.3 gms% in the three cases. The youngest was 2 months old and the oldest 5 years.

II. Haemoglobinopathies

The abnormal haemoglobins that has so far been described in Ceylon are Hb.H (Nagaratnam and Sukumaran,1967) and Hb.E (de Silva et al ,1957, Nagaratnam et al ,1958).

Table II summarises the five cases. The only cases of Hb.H-thalassaemia did not show any radiological evidence of bone changes. All four cases of Hb.E -thalassaemia had bone changes,3 showed the characteristic changes in the bones of the hands (Fig.1),two in the tubular bones and one showed rarefaction of the bones of the hands, None had bones changes in the skull.

Table II and Fig. 1 near here

III. Congenital Non-spherocytic Haemolytic anaemias

Of the 4 cases of atypical congenital haemolytic anaemias which had been described in detail by one of us (Nagaratnam et al ,1968) only one showed the characteristic changes in the skull,hands and tubular bones (Fig. 2 and Fig 3). Clinically there was considerable bossing of the frontal and parietal bones of the skull.

Figures 2 and 3 near here

X-rays done at the end of 3 years showed none of the changes

seen earlier. Table III below summarises the clinical and haematological data in relation to the bone changes.

Table III near here

Discussion

Caffey (1957) has described in detail the radiological bone changes in thalassaemia. The first site of detectable changes are in the small bones of the hands. There is widening of the medullary cavities with thinning of the cortex and decreased density of the medulla. The trabecular pattern in contrast appears prominent. Owing to the broadening the small tubular bones assume a rectangular rather than a linear shape. In the membrane bones of the skull there is broadening of the diploe with separation of the tables resulting in thickening of the vault of the skull especially of the frontal and parietal bones. The outer table is thinned and the ~~vault~~ bony trabeculae traversing the widened space give it an appearance of 'hair on end' or 'brush' appearance. The expansion of the marrow in the malar bones give rise to a mongoloid appearance. Growth as a whole is often retarded.

These changes usually develop early in life and as the child grows older they tend to regress in the extremities but may persist in the skull and pelvis. According to Lie -Injo Luan Eng (1958) in ~~case~~ thalassaemia major occurring in Indonesia the bone changes are more pronounced in other parts of the body than in the skull. Our findings are very similar to hers. Besides the characteristic changes seen in these three cases in this series one showed rarefaction of bone. Rarefaction of bone is seen in sickle-cell anaemia( ). and in Hb.E-thalassaemia (Nagaratnam et al,1958). Spontaneous fractures have been known to occur in the bones occasionally (Ray et al,1956) but none of our cases had any spontaneous fracture.

Hankins (1949) Am. J. Roent Reynolds (1962)

According to Astaldi et al (1951) bone changes are conspicuous aspecially in the less severe and

Some Radiological aspects of the S haemoglobinopathies as seen in Ibadan by W. Peter Cockshott (1965) in Abnormal Haemoglobins in Africa, Blackwell, Oxford

milder forms of thalassaemia major. In our series, however all 3 that showed bone changes appear to have been affected severely as the clinical, haematological and the level of alkali resistant haemoglobin indicate,. In thalassaemia major more often than not there are large amounts of Hb.F though lower percentages have been observed. However, according to Fessas (1959) and others the proportion of Hb.F cannot be correlated with the clinical severity of the disease.

Bone changes are also seen in the haemoglobinpaties but are usually less marked. The single cases of Hb H-thalassaemia in our ~~ca~~series did not show any bone changes, but all the cases of Hb.E-thalassaemia showed changes in the bones of the extremities ~~and~~ but not in the skull. Changes in the bones are similar to those that occur in thalassaemia.

References to
work gothis
from S.E Asia.

Changes in hereditary spherocytosis are rare still more so in the congenital non-spherocytic anaemias. They have also been described by others (Kaplan and Zuelzer, 1950, Nelson,1954).

Summary

18 cases of congenital haemolytic anaemias were studied in relation to the bone changes. Of the 11 cases of thalassaemia only 3 showed bone changes and these occurred only in the peripheral bones. The 4 cases of Hb.E - thalassaemia also showed similar changes and distribution. ~~The~~ One case of non-spherocytic haemolytic anaemia showed well marked changes in the skull, hands and tubular bones.

Thalassaemia

Case	Age Yrs	Hb	ADT	Hb gms%	Retic %	Blood picture	Liver	spleen	X'rays Sk.	X'rays Hds.	X'rays Tub.B
1	5/12	nil	36.4	4.2	6	N++++ T++	3f	3f	0	0	0
2	8/12	nil	73.5	3.3	6	N++++ T +	2f	4f	0	+	0
3	5	nil	6.2	11.0	3	T +	0	2f	0	0	0
4	9/12	nil	17.5	3.3	4	T±	2f	0	0	0	0
5	10/12	nil	14.0	3.7	8	N+++ T±	3f	2f	0	+*	0
6	8/12	nil	70.0	3.2	-	N 0 T 0	0	0	0	+	0
7	11/12	nil	68,8	6.0	6	N ± T ±	0	0	0	0	0
8	2/12	nil	40.0	3.4	-	-	0	0?	0	0	0
9	4/12	nil	72.0	2.6	-	N+++ T++	3f	2f	0	+	+
10	3	nil	30.0	3.1	-	N + T+	1f	splect -omy	0	0	0
11	5/12	nik	10.0	7.7	1	N± T++	0	?	0	0	0

Table.I summarises the haematological, clinical and radiological bone changes in patients with thalassaemia

Haemoglobimopathies

Case	Age Yrs	Hb	ADT	Hb gms%	Retic %	Blood picture	Liver	Spleen	Sk	X'rays Hds.	X'rays Tub.B
Hb.H- thalassaemia											
1	5/12	H	-	2.2	-	N+++ T ++	1f	0	0	0	0
Hb.E thalassaemia											
1	6½	EF	3.6	2.1	0.3	N++ T±	4f	5f	0	++	+
2	13	EF	nil	2.1	17.2	0	3f	5f	0	rare-faction	0
3	10	E F	5	6.6	2.8	N± T+++	3f	6f	0	++	+
4	4	EAF	4	6.9	4.0	N+ T+++	1½f	0	0	++	0

Table.II summarises the haematological, clinical and rad iological bone changes in patients with haemoglobinopathies

Congenital Non-shperocytic haemolytic anaemias

Case	Age	Hb	ADT	Hb gms%	Retic %	Blood picture	Liver	spleen	Sk.	X'rays Hds.	Tubs
1	1	nil	0	6	4	mac.	0	0	++	++	++
2	2	nil	sl. raised	4.8	3	N+ TO	0	0	0	0	0
3	18	nil	0	2.8	4.5	N O T O	2f	3f	0	0	0
4	30	A_2 sl. raised	0	3.3	5	NO TO	2F	4f	0	0	0

Table.3 summarises the haematological, clinical and radiological bone changes in patients with congenital non-spherocytic haemolytic anaemia

Government Hospital.
Gampaha, W.P.
28.3.69.

GAMPAHA, W.P.

Dear Mr. Sukumaran,

I hope you received the slides that I sent you some time ago. I wonder what you think of them. I have been screening all the children with anaemia recently for foetal Hb. with the acid elution technique, but all of them were negative.

Do you think it would be worth while to recall these children + their families and repeat the A.E.T. Their alkali denaturation test for Hb. F had always been normal.

Regarding the paper, "Regal blastic Anaemia complicating ~~pregnancy~~ Thalassaemia". I have written several times to the Editor, Acta. Haemat. Japonicum, but failed to get any reply, though he acknowledged receipt of the manuscript in July 1968.

I hope that Dr. L. D. Sanghvi and you will grant me permission to use some of the results of the haemoglobin analysis, in a paper I intend reading, to the Ceylon College of Physicians. I shall acknowledge your help in this connection.

With kind regards.

Yours truly

A. Nagaratnam

PKS to see me
[illegible]
31.iii.69

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விமானக் கடிதம்
Aerogramme

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GAMPAHA
MR.
69

P. K. Sukumaran, Esqr.
Cancer Research Institute,
Tata Memorial Centre,
Parel, Bombay 12,
India.

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BOMBAY
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යවන්නාගේ නම සහ ලිපිනය
அனுப்புபவரின் பெயரும் விலாசமும்
Sender's name and address Dr. N. [illegible]ratnam
Physician.
Govt. Hospital, GAMPAHA, W.P.
CEYLON

ගුවන් ලියුමක කිසිවක් බහා නොතිබිය යුතුය. එසේ තිබුනහොත් ඊට වැඩිපුර අයකරනු හෝ සාමාන්‍ය තැපැලෙන් යවනු හෝ ඇත.

இந்த விமானக் கடிதத்தினுள் வேறு ஏதாவது வைத்தனுப்பப்படுமாயின் மேலதிக கட்டணம் அறவிடப்படும் அல்லது சாதாரண தபால் மூலம் அனுப்பப்படும்.

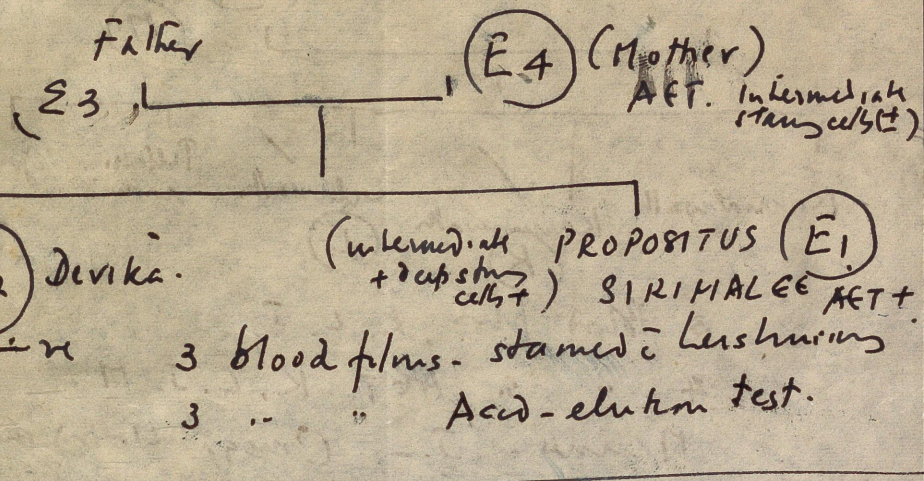
An Aerogramme should not contain any enclosure; if it does it will b surcharged or sent by ordinary mail.

To open cu

Dr. N. NAGARATNAM
M. D: (CEY) M. R. C. P. (GLASG)

GAMPAHA. W.P.
CEYLON
18.2.1969.

(1) EDIRISINGHE FAMILY.



3 blood films. stained ☐ Leishmania
3 " " Acid-elution test.

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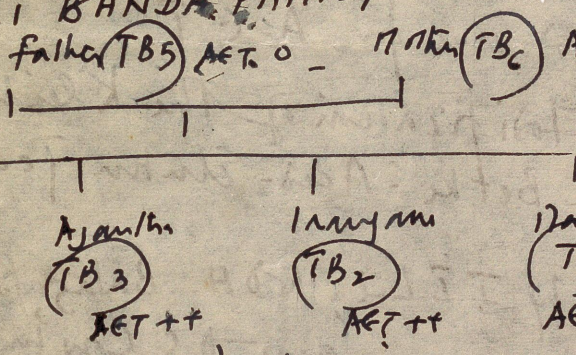
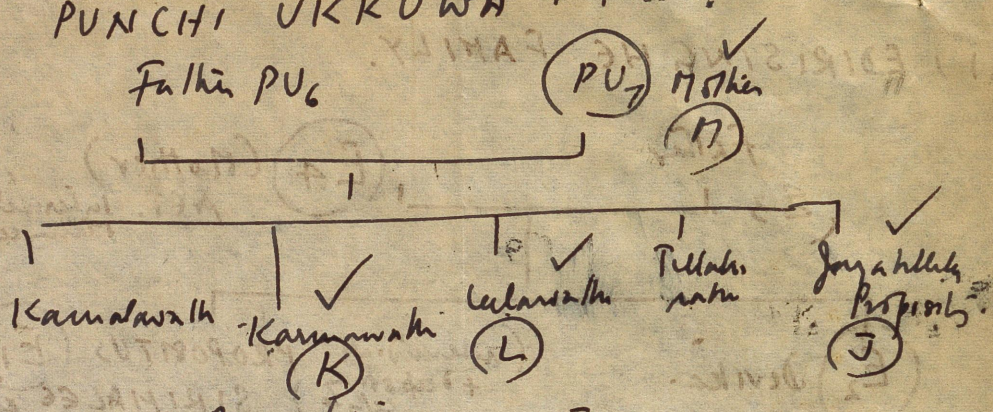
(2) TIKIRI BHANDA FAMILY



6 : Blood films - Leishmania.
6 " " A-E-T.
1 Marrow shows ~~J~~ Ajantha.
(amastigotes + macrophages with normoblastic erythropoiesis)

PUNCHI UKKUWA FAMILY



3 Blood fluids, K. L. J.

4 " " AET K. L. J. M.

Marrow: J.- (Omega-globulin changes)

Method used for A-E-T.

Modification of The Kleihauer-Betke-Acid-Elution Technique.

by J. T. Lee, NDH. Balagas, Berta Lingo and D. C. Cowling.

The Med. J. of Aust. Vol. 1 page 43.

1963.

DR. N. NAGARATNAM
M. D. (Cey) M. R. C. P. (Glasg.)
PHYSICIAN.

Government Hospital,
Gampaha, W.P. Ceylon
18-2-69.

Dear Mr. Subramaniam,

Thank you very much for the reports. I had indeed been extremely unlucky to have missed you at Hyderabad and again in Bombay. I should have written to you earlier. I do hope that we may be able to meet some time soon.

I am sending you details of 3 families. Re: Sirimalee Edirisinghe - necessitated several transfusions, + did not respond to iron initially. She is apparently well now. I saw her last on 23-12-68. at my old station. Acid-elution test shows - an even distribution of HbF in the red cells. I wonder whether this is compatible with the varying values of HbF as determined by the alkali denaturation method. The mother too, to some extent showed this abnormality.

Could these be cases of hereditary persistance of foetal Hb. As far as the Sinhalese goes - in the Greek type the mean value of HbF is 14·5%, in this has 21 - 22% & higher among the Negroes. Even in the other 2 families 2-3 members showed

phenols abnormalities in the blood films with acid elution test.

I shall send you these slides (I have only one set) for you to go through them. If worthwhile if possible could you have them photographed. I find it difficult to get them done here.

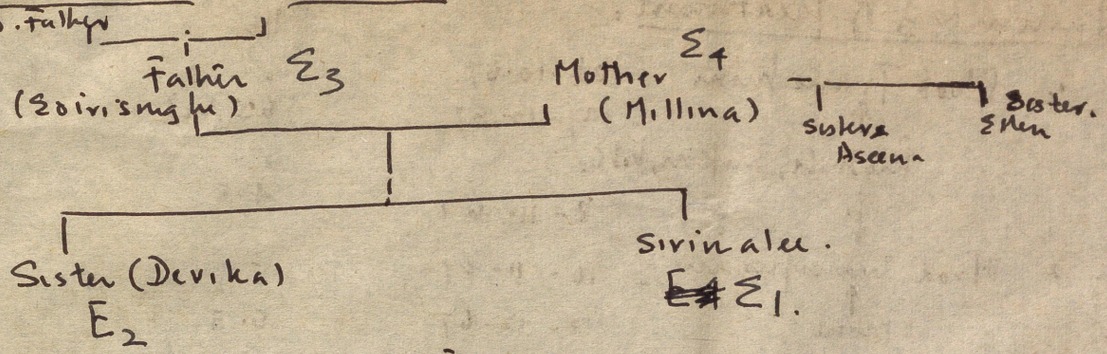
By the way you have addressed the letters wrongly. The name of the place is GAMPAHA and not GAMPALIA.

With kind regards & best wishes.

Yours truly
Jayasekara

I have returned the proof of the manuscript. - 'Some Changes in haemolytic Anaemias' to the publishers & I certainly will let you have the reprints when I receive them.

EDIRISINGHE FAMILY (PROBAND - SIRIMALEE - female).



Sirimalee: E.

Seen on 2.3.68 when she was 1 year & 10 months old. fatigue, tiredness, & pallor. Investigated earlier at the neighbouring hospital & has been transfused on 4 occasions. On exam^{n}: extremely pale. Liver 0. Spleen just palpable.

Investigations: at the time of admission.

Hb: 2.8 gm% (29%) PCV 9%, RBC. 1.48 mill/c.mm MCV 60.6 - MCHC. - 31
Retic 0.5%. WBC. 12,600/c.mm. Infra-cystic cytic microcytic. mil. Sickling -ve.
Serum fe: 70 ugm%. Blood picture: marked hypochromia, microcytosis
fortarget cells. Osfragility. before & after incubation 24 hrs -
showed increased resistance & increased span. Coombs - direct
- indirect - ve, Electrophoresis: done at the Cancer Research
Institute - no abnormal Hb; alkali resistant Hb. 7.6% (
Specimen lysed). x-rays of bones: N.A.D.

Treatment: on 4.3.68. Blood transfusion. - Hb - 8.4 gm%. (58%) Retic 2%.
on 5.3.68.

Re-admitted on 4.5.68. Hb. 2.8 gm%, PCV 13%, MCV 93. Retic 0.5%.
Marrow Biopsy: Normoblastic hyperplasia, defective haemoglobinisation.
consistent with iron deficiency anaemia. Leucocytes & platelets normal.
Perls reaction: no siderotic granules in 'ringed' siderblasts.
Transfused on 8.5.68. On discharge Hb. 9% (7.8 gm%)
Given pyridoxine 50 mgm t.d.s.
On 27-5-68 Hb 37% (5.2 gm%) Retic 0.8%. Pyridoxine
continued.
On 4-6-68 Hb 31% (4.3 gm%. Retic 0.2% and on 13-6-68
Hb was 27% (4.0 gm%.) ~~Admitted on 14~~. and transfused
on 15-6-68.

Earlier investigations at local hospital. * after transfusion
1st admission 24.10.67 - 27.10.67. Hb. 6.6 gm%. on 8.11.67. Hb 4.8 gm%.
Electrophoresis. no abnormal Hb, Serum bilirubin 0.5 gm%.
I° - c ferrolate, Inferon, Vit C. Runs entire stay in hospital.
Transfused on 26.10.67 & on 10.11.67

2nd admission: (2-12-67 - 25-12-67)
Hb. 5.3 gm%. 3/12, 2.3 gm% 13/12 8.2 gm% 17/12. Serum bilirubin 39.5 gm%.
Alkali resistant Hb. 3/12. 1.6%. 8/12 1.1%
Transfusion 14.12.67 & 16th

3rd admission: - Transfusion on 10.1.68. (2)

Summary of Treatment:

	Treatment	Date	Hb
	1. Blood Transfusion	26.10.67.	-
	↑	27.10.67	6.5 gm%.
	Ferrodate, Imferon, Vit C.		
At	↓	8.11.67	4.8
neighbouring	2 Blood Transfusion	10.11.67	-
hospital.	↑ Iron ↓	2.12.67	5.3
	3. Blood Transfusion	14.12.67	
		17.12.67	8.6
	4. Blood Transfusion	10.1.68	
		2.3.68	2.8.
	5. Blood Transfusion	4.3.68.	
Kegalle.	↑ Iron ↓	5.3.68	8.4.
	6. Blood Transfusion	8.5.68.	
	↑	10.5.68	7.8
	Pyridoxine 50mg tds	27.5.68	5.2
		4.6.68	4.3
	↓	13.6.68	4.0
	7. Blood Transfusion	15.6.68	13.0
		27.6.68	9.4
	↑	14.7.68	7.5
		28.7.68	9.8
		11.8.68	8.9.
	Ferrodate	2.9.68	9.4
		30.9.68	8.8
		8.10.68	~~6~~ 8.9
		7.11.68	~~52~~ 7.7
	↓	21.11.68	7.7
Last seen on	--->	13.12.68	9.2.

She was last seen by me on 13-12-68 when she was apparently well. Hb. 62% (9.2 gm%).

Os. fragility: 0.3% - 0.45%

Acid-Elution: ~~evenly~~ unevenly distributed HbF in blood film. (done on 21.XI.68).

Family Studies:

One sister. (Devika).

Pallor, Liver 0. Spleen 0.

Investigations done on 27-10-67.

Hb. 2.8 gm%. (20%.) PCV 12. RBC. 1.75 mill. - Serum Fe. 60 μg%.

Blood picture marked hypochromia & target cells.

Electrophoresis: No abnormal Hgb. ADT. 1.03%. HbA_2 ↓.

No intra-erythrocytic inclusions. Sickling -ve.

Ad. on 4-5-68. Hb 5.1 gm%., PCV 25, MCV 84, MCHC 20.4.

Retic 0.5. Marrow. normoblastic hyperplasia, defective haemoglobinisation consistent c̄ iron deficiency. No 'ringed sideroblasts'. Treated c̄ Stannable Iron. No 'ringed sideroblasts'. Treated c̄ fersolate. till 10-5-68 when Hb. was 37%. (5.2 gm%.)

Given pyridoxine. 150 mg daily & on 27-5-68 Hb. 56%. (7.5 gm%.).

On 4.6.68.	Hb.	56%	(7.8 gm%.)	Pyridoxine.
27-6-68	Hb.	67%	(10.0 gm%.)	
× 14-7.68	Hb.	58%	(7.9 gm%.)	
28-7.68	Hb	70%	(10.4 gm%.)	No treatment
11.8.68	Hb	80%	(11.9 gm%.)	
15.X.68	Hb	80%	(11.9 gm%.)	
7-XI.68	Hb	79%	(11.8 gm%.)	
21.XI.68	Hb	79%	(11.8 gm%.	

Last seen on 13.12.68. Hb. 76% (11.3 gm%.).

Acid-elution technique: Negative. ("ghost cells" only.)

Mother. (Millma).

Pallor. Liver 0, Spleen 0

Hb. 7.0 gm%. (50%.) PCV 27. RBC. 3.820 - Retic 1%.

Hypochromia & target cells. No inclusions, Sickling - ve.

No abnormal Hb. ADT 1.2%. T̄ c̄ fersolate.

Serum Fe: 56 μg%. On 29.5.68 Hb 63%. (9.2) - on 4.6.68 64%. (9.1 gm%.

on 13-6-68 73%. (10.9 gm%.) All the time on Iron (oral).

When last seen on 13-12-68. Hb 72%. (10.7 gm%.)

AET: intermediate staining cells - evenly distributed.

Father: 37 yrs. Clinically NAD. Hb: 10.5 gm%. Retic 0.5%. PCV 42%.

Blood picture: normal.

Serum Fe: 82 μg%. Electrophoresis - NAD.

AET. Negative.

Mother sisters: Aseema. Hb 87% – Blood picture: mild hypochromia
Ellen. Hb 67% (10.0 gm). " shows hypochromia.

Your Reports: re Hb. analyses

28th March, 1968.

Sumala H-888. ADT: 7.6%
Electro: no abnormal Hb.
Sample lysed.

Sumitra H-888/1. ADT. 6.8%
Electr. no abnormal Hb
Sample lysed.

Mithra. H-888/2. ADT 12.3%
Electro: No abnormal Hb.

Devika H-888/3 ADT 1.03%
No abnormal Hb.
Hb A_2 low.

Jan 15th 1969

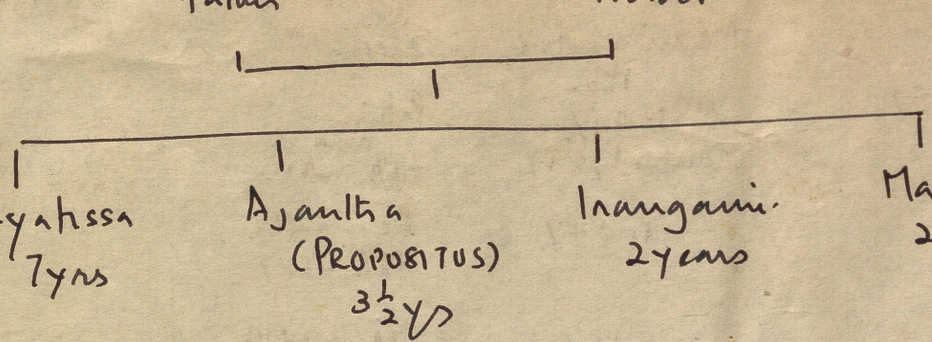
Sumala (Σ_1) H-771. ADT 1.25%.
No abnormal Hb.
Hb A_2 = 2.53%.

Devika (Σ_2) H-771/1 ADT 1.53
(sample partially lysed). No abnormal Hb.
Hb A_2 - 1.53%

Fathi (Σ_3) H-773/2 ADT 0.75%.
No abnormal Hb
Hb A_2 2.37%.

Mathi Σ_4. H-771/3 ADT 1.91%
No abnormal Hb.
Hb A_2 - 2.86%.

TIKIRI BANDA FAMILY.



Ajantha. Male aged 3½ yrs. admitted on 24-11-68.
- Pica, general weakness. On exam: Pallor. Liver 0.
Spleen 0.
Hb. 24% (3.6 gm%) PCV 18% RBC, 2.200h MCV 81 cyn MCHC 20%.
Retic 15% Blood picture: anisocytosis, poikilocytosis, hypochromia macrocytosis. polychromasia Target cells + Normoblasts.
Transfused on. 2.12.68. Os. fragility 0.35 - 0.5% (3% haemolysis)
Hb 55% (8.1 gm%)
Marrow: hypercellular, normoblastic erythropoiesis. macro-normoblastic.
on 1.12.68. No abnormality in white cells

Acid-Elution Test: evenly distributed HbF.
To Mum & Fe: Hb. 69% (10.3 gm%) PCV 38% RBC. 4.47 MCV 80.8 MCHC 27%. Retic 0.5%
Family Studies: on 14.12.69. AET.

Mother Father: Hb. 79% (11.8 gm%) + (few dark stained cells).
Os. frag: 0.3% - 1.0% haemolysis. Blood picture normal

Father Mother. Hb 93% (13.9 gm%) - ve (only ghost cells seen).
Os. frag. at 0.3% Sal. 100% haemolysis
blood picture: normal

Jayatissa Hb. 63% (10.3 gm%) - ve
7 yrs Os. frag at 0.3% Saline. 100% haemolysis
blood picture normal

Mallika Hb. 76% (11.3 gm%). +++
2 months Os frag. at 0.3% N Saline 100% haemolysis evenly distributed.
blood picture hypochromia target cells ++

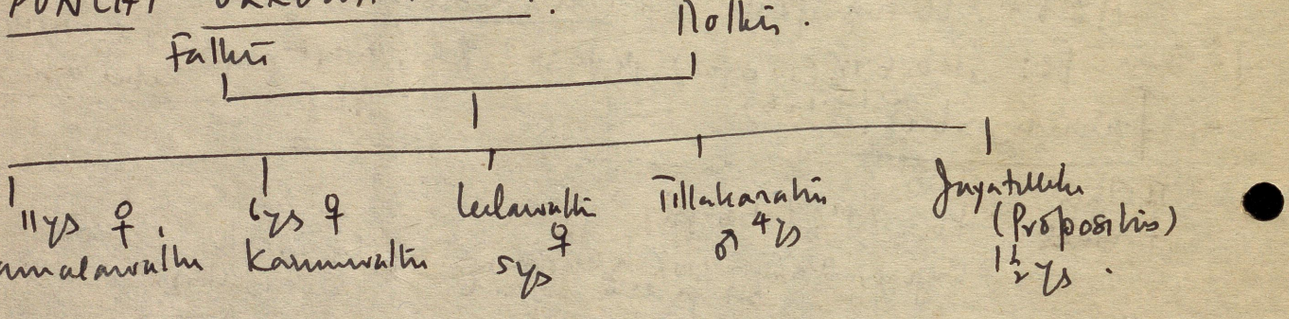
Inangani. Hb. 86% (12.8 gm%) ++
2 yrs. PCV 43%, RBC. 5.70 mill evenly distributed.
MCV 74. MCHC 29.7
Retic 2.0%
Os. frag: 0.3 - 0.45% blood picture hypochromia

Case H-772, -1/2-/3-/4-/5 of 15-1-69.

Mm reports on Hb Analysis etc.

	Alkali resistant Hb.	Electro.
Falkin (TB_5)	1.34% (sample lysed)	Pallim mosaic. HbA_2 not done
Nolkin (TB_6)	1.44%	No abnormal Hb. $HbA_2 = 1.8\%$
Jayatissa. TB_4.	1.45%	No abnormal Hb. $HbA_2 = 2.03\%$
Ajantha (Propositus) TB_3	2.55 (sample partially lysed)	HbA_2 2.21%
Inangani. TB_2.	2.52%	A_2 2.52%
Mallika TB_1 (Age 2 months)	30.5% sample lysed.	HbA_2 not done.

PUNCHI UKKUWA FAMILY.



Jayatilleke - Aged 1½ yrs admitted on 14-11-68.
Anaemic. Hb. 17% (2.5 gm%)
Blood picture: moderate anisocytosis - poikilocytosis - hypochromia, nucleated red cells.
Marrow: Megaloblastoid change.
No stainable Fe.
Intra-cytoplasmic inclusions - ve.
Acid Elution Test. Large number of reticulocytes + intermediate cells evenly distributed.
ADT & Electrophoresis. Not done
Transfused.

Leelawathi . age 5 yrs
seen on 28-6-68.
Hb. 23% (3.4 gm%) PCV. 12 mm. MCHC. 28.5%
Transfused. No investigations done at that time.

Seen in Nov. 1968.
Pallor +.
Abnormal Hb. nil.
(done here) - ADT -ve.

On 23. XI. 68.
Hb. 24% (3.6 gm%)
PCV 16%. RBC. 1.960 —
MCV 81 MCHC 22.5
Retic. Cnt. 5.0%.
Os. frag 0.3-0.45%.

AET. ++.

Karunawathi. 6 yrs
History of anaemia.
Rx in another Hospital in 1964.
Transfused.
On 23. XI. 68. Hb. 21% (3.1 gm%)
PCV. 14%
RBC. 2.060 —
MCV 70 cu — MCHC. 22.5%.
Retic 3.0%.
Abnormal Hb. nil
(done here) ADT -ve.
Os. frag: 0.45 - 0.35%.

Mother:
Blood film: normal.
AET: "intermediate" staining cells
(done here) ADT -ve. No abnormal Hb.

Other members of family not investigated.

BY AIR MAIL

ED/2316 /69 April 3rd, 1969

Dear Dr. Nagratnam:

Thank you very much for your letter dated March 28th, 1969.

I am in receipt of your previous letter of February 18th, 1969, along with the Bone Marrow Smears as well as Blood films including Acid Elution Test preparations. I wanted to write to you after I succeed in getting good photographs of the marrow preparations. There has been some delay on this.

Regarding the Acid Elution preparation you sent me, I feel I should be cautious in my comments. First of all these bloods do not show foetal haemoglobin by alkali denaturation test and yet your slides do show presence of Hb-F. From the samples you sent me, though they were lysed, the red cells I could salvage were subjected to Acid Elution Test and were found to be negative while methanol fixed smear sent along with specimen was positive. This raises a problem whether the results on stored blood smears are that much dependable. In this connection, I shall draw your attention to an article in BLOOD (1963) XXI:553, where the authors say that falsely high reading for fetal cells are seen in stored blood smears. Thus, I feel, you should call your interesting patients for a re-check and use cord blood in various dilutions with adult blood, used as controls. Perhaps, wherever you find Acid Elution Test positive, and alkali denaturation test negative, it may be worthwhile confirming presence of Hb-F by agar-gel electrophoresis at acid pH.

3.IV.69

I discussed your letter with Dr. Sanghvi regarding reading a paper before the Ceylon College of Physicians using some of our results on haemoglobin analysis. We feel that it would be in order, provided you make the necessary acknowledgements (vide our letter No. ED/690/69 of January 24th, 1969). May I again remind you to send me reprints of some of your articles published recently ?

With kind regards,

Yours sincerely,

PKS

P. K. Sukumaran
Scientific Officer

ED/4593/69 July 4th, 1969

Dear Dr. Nagaratnam:

I thank you very much for your letter of 17th June 1969 and enclosed Summary of Case Notes of your interesting case (EDIRISINGHE) and also copies of letters to you from Drs. Heimpel and Kleihauer.

It is indeed very nice of you to keep me posted of the developments in the investigations of this case. From the very beginning, I had a feeling that this was a interesting case and I congratulate you for persuing so far which ordinarily very few Clinicians do.

I am glad to note that Dr. Kleihauer is prepared to investigate the samples for the presence of unstable haemoglobins besides other investigations. You are, perhaps, aware that non-spherocytic haemolytic anaemias where anaemias attributable to unstable and heat-labile Hb are reported. Prof. Lehmann alongwith Prof. Dacie's group has extensively investigated such cases. If you so desire, we can have this investigated besides or after Dr. Kleihauer does his bit in this case. After hearing from you, I shall write to Prof. Lehmann about this case.

I believe you have published some papers in your local journals besides those in foreign journals. Please send me whatever you have at hand and you can send the others when you receive them.

4.VII.69

With kind regards,

Yours sincerely,

P. K. Sukumaran,
Scientific Officer

Dr. N. Nagaratnam,
Government Hospital,
Gampaha, W.P.

DR. N. NAGARATNAM
M. D. (Cey) M. R. C. P. (Glasg.)
PHYSICIAN.

17. 6. 69.

Dear Mr. Sukumaran.

I thank you for your letter dated April 8th 1969.

I enclose a copy of the Case Notes of Sivanathan Edirisinghe, which will interest you. I shall keep you informed about this case.

With regard to the papers that I have published recently, the publishers have not as yet sent me the reprints. I shall send you the reprints as soon as I receive them.

With kind regards

Yours very sincerely,

N. Nagaratnam

SUMMARY OF CASE NOTES

PROPOSITUS - SIRIMALE EDIRISINGHE

Female, aged 1year and 10 months was admitted on 2.3.68 with a history of fatigue and listlessness of several months duration and pallor of two weeks duration. She had been investigated at the neighbouring hospital from 20.10.67 to 23.11.67 and again between 2.12.67 to 25.1.68 during which time she had been transfused on 4 occasions. The results of these investigations are given at the end of the notes.

On examination sha was breathless at rest. She was weel nourished but extremely pale. There wwere no palpable lymph glands. No abnormality was detected in her heart or lungs The liver was palpable but the spleen was palpable 2 finger-breadths below the left costal margin.

Investigations:at the time of admission: Hb 2.8G%(20% PCV 9%, RBC 1,480,000 /c.mm, MCV 60.6 cu,MCHC , Retic 0.5%, WBC 22,600/c.mm, Intra-erythrocytic inclusions (Brilliant Cresyl blue) nil, tests for sickling negative, Serum iron 70mgm%. Blood picture: marked hypochromia and a few target cells. Os.frag. before and after incubation for 24 hours showed os. resistance and increased span. Coombs direct and indirect negative Electrophoresis: no abnormal haemoglobin and ADT1.25%.X8rays of skull and hands revealed no abnormality.

Treatment: on 4.3.68 Blood transfusion (i pint) and sent home on 5.3.68 when her Hb was 8.4 g% (60%) Retic.2%.

Re-admitted on 4.5.68, she was once again pale,Hb 2.8 PCV 13%,MCV 93cu, Ret.0.5%. Marrow biopsy: Normoblastic hyper-plasia with evidence of defective haemoglobinisation,consist-ent with iron deficiency. The white cells and platelets showed no abnormality. Per's reactionshowed no siderotic granules or 'ring' sideroblasts. She was transfused on 8.5.68 and on d sgharged Hb 7.6g% (54%).She was given 50mgms TDS of pyridoxine.

On 8.5.68 Hb 5.2g% (37%) Retic 0.3%. Pyridoxine was co-ntinued and on 4.6.68 her Hb was 4.3 g% (31%), retic 0.2%, and on 13.6.68 27% (4.0g%). She wasadmitted and on 14.6.68 she received another blood transfusion.

Earlier investigations at the neighbouring hospital.
1st admission: 27.10.67 Hb 6.6 G% ,On 8.11.67 4.8G%. No abnormal haemoglobin detected, Coombs negative, Serum bilirubin 0.5mg % treated with Fersolate Imferon Vit,C. Transfused on 26 .10.67. and on 10.11.67.
Re-admitted (2.12.67-25.12.67) Hb 3.1267 5.3G% 13.12. 2.3G% 17.12. 8.2G% Serum iron 39.5 Mgm%, ADT 1.0% and repeat 1.1 %. Transfused on 14.12.67.
3rd. admission: transfused on 10.1.68.

From 27.6.68 30.10.68 she was on fersolate.

The ~~results~~ Haemoglobin levels together with tratment are summrised below.

She was seen again on on 20.5.69 when her Hb was 4.7 g%. She was transfused one week later.

FAMILY STUDIES:

One sister (DEVIKA EDIRISINGHE)

She was pale when first seen in March 1968. The liver nor the spleen were palpable. Hb. 2.8G%,PCV 12%, RBC 1,750,00. Serum iron 60mgm%. Blood picture marked hypochromi a No abnormal haemoglobin was detected on electrophoresis. ADT 1.03%, HbA_2 reduced. No intraerythrocytic inclusions were seen. and tests for sickling was negative. Given Iron (oral) Admitted on 4,5.68 Hb 8.1 g% (37%) pCV 25% ,MCV 84 MCHC 20.4%

retic 0.5%. Marrow normoblastic hyperplasia and defective haemoglobinisation.consistent with iron deficiency. Perl's reaction; no siderotic granulesor ring sideroblasts. She was then given pyridoxine 150 mgmstds and on 27.5.68 Hb 56% (7.5 G%). and this was continued for some time.
10.5.68 Hb 37% (5.2G%) 27.5.68 65% (9.1g%)
4.6.68 56 (7.5G%) 27.6.68 67% (10.0G%)
on 20.5.69 9.4G%.

MOTHER(MILLIE NONA) 35 years.Pale when first seen Hb 7.0G% PCV 27%, RBC 3,820,000 , Retic 1%. Blood picture: marked hypochromia and few target cells. No inclusions with brilliant creysl blue were seen, sickling negative. No abnormal Hb. ADT 1.2%. Treated with oral iron. on 10.5.68 Hb 40 % (5.6) on 27.5.68 65% (9.2) and on 4.6.68 64% (9.1) and on 13.6.68 73% (10.9G%) and all the time she was on oral iron.
On 20.5.69 5.2 g%. She now 5 months pregnant.

FATHER (EDIRISINGHE). 37 YEARS. Clinically no abnormality. Hb. 10.5 g% (75%),PCV 42% Ret.0.5%. Blood picture normal.No abnormal Hb. ADT normal. Serum iron 82Mgm%.
On 20.5.69 10.2 G% Hb.

MOTHER'S SISTERS: (1) Aseena Hb.87% 13.0) Blood picture Mild hypochromia
(2) Ellen Hb 67% (10.0 G%) Blodd picture Shows hypochromia.

SUMMARY OF TREATMENT OF SIRIMALEE

26.10.67	Blood transfusion	HB (G%)
27.10.67		6.5
8.11.67	Ironoral,Parenteral Vit.C	4.8
10.11.67	Blood transfusion	
3.12.67	Iron preps.	5.3
13.12.67		2.3
14.12.67	Blood transfusion	
17.12.67		8.6
10.1.68	Blood transfusion	
2.3.68		2.8
4.3.68	Blood transfusion	
5.3.68		8.3
4.5.68		2.0
8.5.68	Blood transfusion	
10.5.68		7.8
27.5.68	Pyridoxine	5.2
4.6.68		4.3
13.6.68		4.0
15.6.68	Blood transfusion	13.0
27.6.68	Oral Tron	9.4
14.7.68		7.5
28.7.68		9.8
11.8.68		8.9
2.9.68		9.4
30.9.68		8.8
30.10.68		8.9
7.11.68		7.7
21.11.68		7.7
13.12.68		9.2
20.5.69		4.7

Blood transfusion.

In reply to my letter to Dr. Heimpel, University of ULM
? congenital transferrin deficiency

He replied,

' from the observations stated in your letter it seems to be sure, that this is an hereditary disorder of erythropoetesis with impairment by merely of the synthesis of haemoglobin. A congenital transferrin deficiency as suggested by your letter is highly unlikely because the seum -iron-conc -entration of 60-7- um% is much more than we observed in one case of congenital atranferrerrinaemia. Also stainable iron should be enormously increased in thr reticulum cells of the bone marrow The lack of sideroblasts is against the diagnosis of hereditary siderpchrestic anaemia. I would suggest youu send me unstained peripheral blood smears of each member of the family and bone mar -row smears as far as possible'. 5th. August.1968.

On 28.4.69 I received the following letter from him.

'I am so ry to tell you that most of the slides have been broken at the transport so I can tell you the results of the slides of SE (propositus) and DE (sister). In SE, the cellularity of the marrow seems normal,and all haemopoietic cells are seen. There is slight increase of the erythroid series,E/G ratio being slightly elevated to 1.1 : 1. This about twice our normal values and a nonspecific finding in many anaemias. The distribution of proerythroblasts,erythroblasts and normoblasts is about normal. but maturing of the protoplasm is distinctly delayed compared to the nucleus. There are no specific anomalies,however of the nucleus itself. No ~~iron-in-th-e-marrow~~ karyomeres are seen. In the iron stain, the total amount of iron in the marrow seems to be normal,the iron positive granules in the ~~eetre~~ erythroblasts are distinctly increased but no sideroblasts are seen as in sidero chrestic anaemia. Morphlogy of all other bone marrow cells is non characreistic. Eosinophils are slightly increased.

Findings in DE are essentially normal but some increase of sideroblasts is also seen.

The latent iron binding capacity has been estimated in all the samples with the following results:

D.A.	266 gamma%	(AUNT)
S.E.	158	(PROPOSITUS)
T.E.	348	(FATHER)
D.E.	210	(SISTER)
R.E.	369	(AUNT)
M.E.	279	(MOT ER)

I amnot able to make a distinctive diagnosis on those cases However, I feel I can tell you which of the possible diagnosis can be excluded on t e basis of the material I have examined. This is not congenital atransferrinaemia because LEBK is normal or even elevated in the cases T.E. and R.E. There is no iron defisiency and no sid rochrestic anaemia consecutive to the findings of the iron stain. Congenital dyserythropoietic anaemia (s,reprint) has characteristic anomalies of the nucleus which definitely are not present. It is remarka le that in the propo itus S.E. iron binding capacity is the lowest of a ll samples investigated. This could be cosecutive to transfusions but could also be a symptom of beginning secondary haemochramat osis resulting from ineffective erythropoetesis. From the data you gave me and the material examined,I should think of a h haemoglobin anomaly as first most likely possiblity. We are not working on pa hological haemoglobins ourselves, but Prof. Kleihauer at Munich will surely be glad to look for anomal haemoglobin in your patients.'

To Dr.N.Nagaratnam M.D., M.R.C.P.
Government Hospital,
Gampaha, W.P.Ceylon.

Dear Dr. Nagaratnam,

Thank you very much for sending blood samples of the family Edirisinghe.

The Haemoglobin analysis gave the following results:

	HbA 2 %	Hb F cells	Inclusion bodies
Father	2,0	none	none
Mother	1,7	none	+
Sister	1,9	none	(+)
Propositus	1,5	none	+
Normal values	2,0- 3,0	none	none

Haemoglobin electrophoresis (starch grain and cellulose acetate strip) at different pH did not result in showing abnormal fractions, The activity of G-6-PD was normal (screening test).

Since we found inclusion bodies in the red cells from propositus, motherand sister heat denaturation test was performed in order to exclude an unstable haemoglobin. Only in the propositus' sample a turbidity appeared in the mother's hemolysate.

It is difficult to give a satisfactory judgement from these data.The tather low HbA 2 values would be in agreement with the pronounced anaemia one should expect HbH, at least in the propositus.However, electrophoresis did not reveal a fast moving fraction and inclusions as found in HbH disease in brilliant crysylblue preparations were missing. Decreased HbA 2 values have also been reported in iron deficiency. Inclusion bodies and heat labile haemoglobin are characteristic features of unstable haemoglobins causing haemolytic anaemias. Often the haemological changes are more pronounced after splenectomy (high Heinz body count, dark urine). The results obtained from the propositus and the mother are not in full agreement with this entity providing that it may develop after the spleen is removed. An alternative possibility is glutathione redutase deficiencyin which inclusion bodies are regularly found.

I would be interested in a more detailed history of the patient, ecpecially in the red cell morphology. It is always difficult to judge from only some laboratory data.

Concerning your question about HbF in red cells (letter from May, 30, 1969) the most falsely high readings result from a too high pH (above3,4) in the elution buffer or a alcohol concentration below 80 per cent, These errors can be excluded soon as other blood smears treated under same conditions give normal results.On the other hand alkali denaturation if not performed by an experienced investigator can give wrong results,

Please sen me a sample one of these patients in order to have a control to your findings.

Yours Sincerely,

(Doz. Dr.E.Kleihauer)

Govt. Hospital,
Kegalle.
26.11.68.

Dear Mr. Subramaniam,

It is quite some time I wrote to you. The Journal Radiologica et clinica have accepted our paper, "Some changes in Congenital haemolytic Anaemias". Regarding the other paper, 'Megaloblastic Anaemia complicating Thalassaemia', the Editor of the Acta Haemat. Japonicum wrote to me a few months ago to say that it is under consideration. Only papers from members of their Haematological Society are published. However, he would let me know as to whether our paper would be accepted by the Editorial Board. Since then I have not heard from him.

I am moving out from my present station on 1.1.69. My new address would be, Govt. Hospital, Gampaha.

I have one or two problems to finish before I leave. I have come across 2 families with several members whose peripheral blood smears show marked hypochromia, resembling iron deficiency. I have done the acid elution test on some of them and have found it to be strongly +ve. The cells appear to be evenly distributed. I have not as yet done the estimation of foetal Hb by the alkali denaturation test. I wonder whether you

cells for the HbA_2 on them. Should you be able to do so, I shall send you the blood samples.

Hoping to hear from you soon.

With regards.

Yours truly

N. Nagarathinam

පළමුව මෙතැනින් නමන්න. முதலில் இங்கே மடியுங்கள். First fold here.

BY AIR MAIL

PAR AVION
ගුවන් ලියුම
விமானக் கடிதம்
Aerogramme

ශ්‍රී ලංකා இலங்கை CEYLON 30 30

P. K. Sukumaran, Esqr.
Indian Cancer Research Institute
Tata Memorial Centre,
Parel, Bombay 12
INDIA.

දෙවනුව මෙතැනින් නමන්න. பின்பு இங்கே மடியுங்கள். Second fold here.

යවන්නාගේ නම සහ ලිපිනය
அனுப்புபவரின் பெயரும் விலாசமும்
Sender's name and address

Dr. N. Nagarathinam
Govt. Hospital,
Kegalle,
Ceylon.

BOMBAY-12

ගුවන් ලියුමක කිසිවක් බහා නොතිබිය යුතුය. එසේ තිබුනහොත් ඊට වැඩිපුර අයකරනු හෝ සාමාන්‍ය තැපෑලෙන් යවනු හෝ ඇත.

இந்த விமானக் கடிதத்தினுள் வேறு ஏதாவது வைத்தனுப்பப்படுமாயின் மேலதிக கட்டணம் அறவிடப்படும் அல்லது சாதாரண தபால் மூலம் அனுப்பப்படும்.

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ED/9127/68

December 3rd, 1968

4 DEC 1968

Dear Dr. Nagratnam:

I am very glad to receive your letter dated November 26th, 1968, and am happy to note that the paper on "Bone changes in haemolytic anaemia" has been accepted for publication.

I shall be pleased to investigate the families with hypochromia wherein you found even-distribution of foetal haemoglobin in red cells. I shall do the estimation of Hb-A_2 to establish hereditary persistence of foetal haemoglobin in them. You may do a careful estimation of Hb-F and haematology including osmotic fragility.

I do hope you shall soon get an affirmative reply from Acta Haemat. Japonicum about the paper "Megaloblastic anaemia complicating thalassemia".

Please arrange to send about 5 ml. of clotted blood from each of the family member. I do hope they would reach here without much haemolysis so that I can do the acid elution test here as well. Ideal way to send blood, as you know, is by Air Cargo which I guess is expensive.

With kind regards,

Yours sincerely,

PKS

P. K. Sukumaran,
Scientific Officer

3.XII 68

Dr. N. Nagratnam,
Government Hospital,
K e g a l l e ,
CEYLON

cBp/

ED/265/69. January 15th, 1969

Dear Dr. Nagaratnam:

Thank you for referring your cases in EDIRSINGHE FAMILY and TTRIKI BANDA FAMILY. Some of the samples unfortunately were received in lysed condition after having taken five days in transit.

I am enclosing the reports and are being sent to your new address at GAMPALI as per your letter of 26.11.68. You will find from the reports that none of them show raised foetal haemoglobin. Acid elution test on the samples, though not satisfactory in some cases, as well as the methanol fixed blood smears sent by you, did not show anything exciting. I wonder whether these are cases of hereditary persistance of high foetal haemoglobin ! You will find the haemoglobin A_2 has been estimated in most of the specimens. We have found Hb-A_2 decreased in iron-deficiency anaemia as well. Please enlighten me further on these cases.

With kind regards,

Yours sincerely,

P. K. Sukumaran

16.1.69

Dr. N. Nagaratnam, M.D., M.R.C.P.
Government Hospital,
Gampalia,
Ceylon

Encl: Reports.

BY AIR MAIL/REGISTERED

ED/810 /69 February 5th, 1969

Dear Dr. Nagaratnam:

On my return from Hyderabad on the afternoon of 27th January, I came to know from my colleagues that you were in Bombay on your way back from the XXIV Joint Conference of Physicians at Hyderabad.

You must have known while at our Institute that I was in Hyderabad for the same Conference. How unfortunate that we could not meet ! I gather you met Dr. Sanghvi and some colleagues of mine. I would have been only too glad to meet you and your wife and renew our contact. Although we have in regular correspondance, we have never met. I do hope we will have an apportunity to meet in no distant a future.

I have, today, received the proof (2 copies) of the article "Bone Changes in Congenital Haemolytic Anaemia" which is to appear in Radiologica Clinica et Biologica, for correction and return. This was received on your name but addressed to Tata Memorial Centre. I am sending them to you for necessary correction and return directly to publishers.

The following are some of my suggestions by way of correction:-

1. Please include "CEYLON" after Kegalle in your address and also correct the authors' address at the end of the paper.

2. In the acknowledgements, you please add, after "for permission to publish this paper","and to Dr. L. D. Sanghvi, M.Sc.,Ph.D.(Columbia); Head, Epidemiology Division, Cancer Research Institute, Tata Memorial Centre, Parel, Bombay-12, for his continued encouragement in this work".

3. In table I, column 3, Hb may be mentioned as Abn. Hb., and ADT may be substituted by "Hb-F". The same corrections may be made in other tables as well.

Please send me reprints of some of your recent papers. As for the reprints of the present paper, I shall be happy if you will spare 50 reprints from the free quota allowed by the publisher.

With kind regards,

Yours sincerely,

PKS

P.K. Sukumaran

Encl: Manuscripts & proof.

Govt. Hospital.
Gampaha, W.P
Ceylon
8.2.70

Dear Mr. Subramaniam,

It is quite some time since I heard from you. Did you receive the reprints on "Some changes in Cytochemical Haematology in Anaemias" ~~extracts~~ I sent to you ~~Several~~ few months ago? As I did not receive an acknowledgement & this is also a reply to a letter written to you two months ago, I was wondering whether you had gone abroad.

I trust you are keeping well.

By the way, I shall send you the reprints on "~~Thalassaemia~~ ~~complicat~~ Megaloblastic anaemia" Complicating Thalassaemia which should have appeared in the Ind. J. of Pediatrics in December, as soon as I receive them.

I have a few cases with Hereditary Elliptocytosis & one member has besides elliptocytes a few % of target cells. I would very much like the Hgb. analysis done in these cases. On hearing from you I shall send the blood samples.

I read in the Ind. J. of Med. Research that the Blood Group Reference Centre (I.C.M.R.) Seth. G.S.

Medical College, Parel Bombay 12 would be willing to help in red cell enzyme studies (G6PD, PK etc). Could you possibly get these done on the blood of this particular family. If this is possible please let me know how I should send the blood.

With kindest regards & the very best for the New Year

Yours truly
Nagaratnam

PKS
11/2/70
Dr. J.V. Car
we do anything in this connection?

පළමුව මෙතැනින් නමන්න. முதலில் இங்கே மடிப்புங்கள். First fold here.

BY AIR MAIL
PAR AVION
Aerogramme

CEYLON FOR QUALITY TEAS

P.K. Subramaniam Esqr,
Cancer Research Institute,
Tata Memorial Centre,
Parel, Bombay, 12

CEYLON 50

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Sender's name and address
Dr. K. Nagaratnam
Govt. Hospital
Gampaha, W.P.
Ceylon

An Aerogramme should not contain any enclosure; if it does it will be surcharged or sent by ordinary mail.

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*. It is better to dispatch samples on a Friday or Saturday if sent by post.

Dear Dr. Nagaratnam,

Thank you for your letter dated 8.2.70.

I did receive the reprints you sent me of the paper "Some Changes in Congenital Haemolytic anaemias" which I have acknowledged. The last letter I had from you was dated 17.6.69 enclosing Case notes of EDIRISINGHE family and Copy of letter from you received from Dr. K Leithauer. I replied this letter on 4.7.69 giving my Comments.

I am glad to note that you have an interesting Case with hereditary spherocytosis + target cells. I shall be pleased to do haemoglobin studies on them. We shall be able to do G-6-PD on them as well, but the trouble is about the transport of the samples. Usually your samples, though sent by Air Mail Post, reach me after 5-7 days, perhaps delayed at Madras. This delay may effect the enzyme activity, especially when not refrigerated. Anyway we can try. You may send Clotted sample of the patient and include a sample from a female whom you consider to be normal for G-6-PD activity to be used as Control under identical conditions. I do hope samples may not be lysed. Such a sample we shall be able to do haemoglobin study as well as for G-6-PD.

May I remind you to send me some of your cases?

With Kind regards + all the best for the X-mas.

Yours sincerely,

We can do the G-6-PD on the samples. The only problem will be of transport in refrigerated condition. I am afraid if they arrive by ordinary mail - there may not be any enzyme activity left. However we shall try. Dr. Nagarathnam should also include any one female sample - which we will presume to be normal & have G6PD activity & compare both.

A. Senderia
11/2/70.

ED/ 116 /70 February 13, 1970

14 FEB 1970

Dear Dr. Nagaratnam:

Thank you for your letter dated 8th February, 1970.

I did receive the reprints you sent me of the paper "Bone changes in congenital haemolytic anaemias" which I have acknowledged. The last letter I had from you was dated 17.6.69 enclosing case notes of EDIRISINGHE family and copy of letter you received from Dr.Kleihauer. I replied this letter on 4.7.69 giving my comments.

I am glad to note that you have an interesting case with hereditary spherocytosis and target cells. I shall be pleased to do haemoglobin studies on them. We shall be able to do G-6-PD on them as well, but the trouble is about the transport of the samples. Usually your samples, though sent by Air Mail Post, reach me after 5-7 days, perhaps delayed at Madras. This delay may effect the enzyme activity, especially when not refrigerated. Any way we can try. You may send Clotted Sample of the patient and include a sample from a female whom you consider to be normal for G-6-PD activity, to be used as Control under identical conditions. It is better to despatch samples on a Friday or Saturday, if sent by Post. I do hope samples may not be lysed. On such a sample we shall be able to do haemoglobin study as well as for G-6-PD.

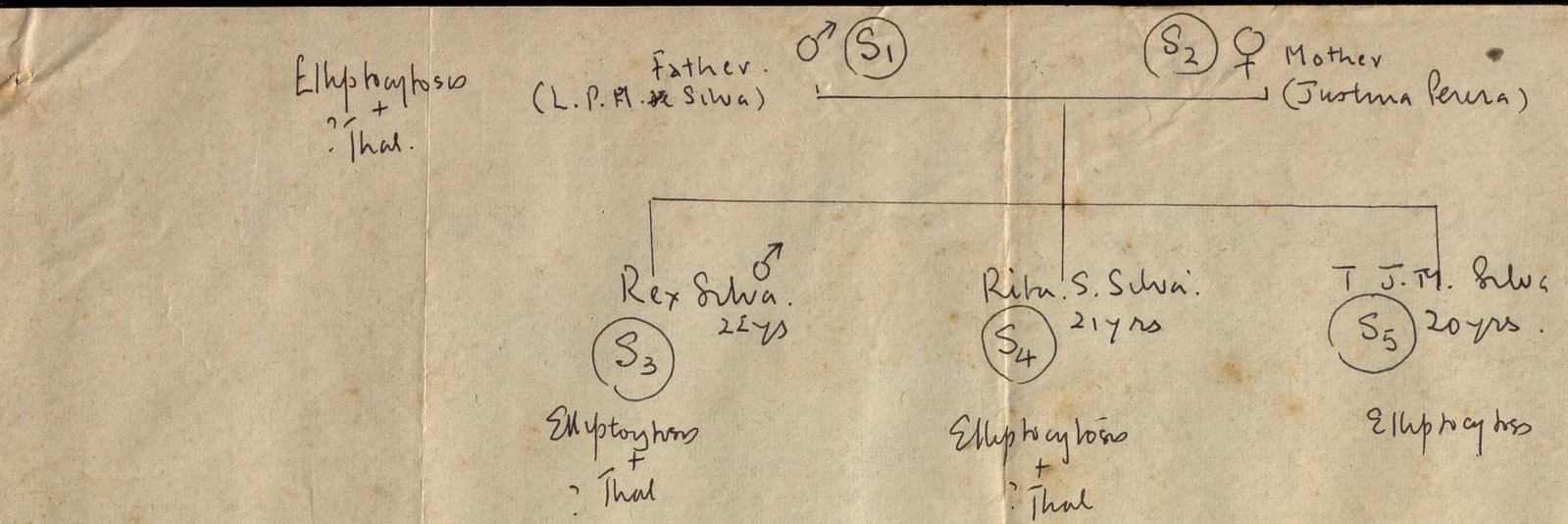
May I remind you to send me some of your reprints ?

With kind regards & all the best for the New Year.

Yours sincerely,

P. K. Sukumaran
Scientific Officer.

Dr.N.Nagaratnam,
Government Hospital,
Gampaha,
Ceylon



	Hb gm%	PCV %	MCHC %	MCV c/μ	Retic %	Blood picture	W Total	B P	C L	& M	D E	C.	Bld. Group ABO Rh.	Intra erythr. cyt. inclus.	Sickling Test	Brunch Test.	Coombs Test D.C.Test.	Osmotic fragility before	Osmotic fragility after	Liver function Tests. Bilirub.	ZnSO4 turb.	Thy. Tol. flocc.	Alk. Phos.	Lep. Alk.	Mal. Pigm.	X'rays
S1	11.1	39	28.5	97.5	1.2	E ++ + A + P + Tg ++	6,400	64	26	2	8		O Rh +ve	-ve	-ve	-ve	-ve	•							nil.	?
S2	13.4	42	-	-	0.2	Normal.								-ve	-ve	-ve	-ve	R	R	0.6	29	6 and 9		2+	nil	normal
S3	10.3	37	30.3	92.5	0.2	E +++ + A ± + P ± + Tg ±	9,400	70	26	-	4		O Rh +ve	-ve	-ve	-ve.	-ve	R	R						WIL	normal
S4	9.4	35	26.9	97.2	0.4	E ++ + A ++ + P + T ++ + Sp ±	6,800	82	39	1	8		O Rh +ve	-ve	-ve	-ve	-ve	N	N	0.6	24	3 and 10.		4+	nil	normal
S5	14.4	46	31.3	93.9	0.2	E ++ + Ov +	6,200	60	34	2	4		B Rh -ve	-ve	-ve	-ve	-ve	N	N	0.4	19	2 and 13		2+	nil.	normal

Blood samples for

(1) & Abnormal Hgb (A2). ADT
(2) Glutathione
(3) Haptoglobinemia. etc.

E = Elliptocytosis
A = anisocytosis
P = poikilocytosis
Tg = Targetcells
Sp = Spherocytes
Ov. = Ovalocytosis.

DR. N. NAGARATNAM
M. D. (Cey) M. R. C. P. (Glasg.)
PHYSICIAN.

Govt. Hospital,
Gampaha, W.P.
Ceylon.
31. 3. 70.

Dear Mr. Inkumarum.

I am sending you 10 blood samples (heparinised & clotted) from the family members with Elliptocytosis The summaries of the investigations done is attached.

There is a suspicion of interaction with the Thal: gene in some of them. It had not been possible to do alkali-denaturation test and also A_2 here. If it is possible could you do any of the studies - haptoglobins many of the enzyme tests.

I do hope the samples will reach you in good state, as it would not be

possible to send you ~~an good~~ any more ~~condition~~ blood for they have refused to give us any more blood.

I shall send by separate post, your share of the reprints – 'Megaloblastic Anaemia complicating Thalassaemia' which was published in the Ind. J. of Paediatrics, together with some reprints of mine.

With kind regards

Yours truly

J. Nagaratnam

Dr. J.V.U

These are the samples Dr. Nagaratnam send just now by post (4.30 PM) for studies including enzyme studies

P.K.S. Kumar
2/4/70

Govt. Hospital,
Gampaha, WP.
25.4.70.

Dear Mr. Sukumaran.

I sent blood samples from a family with Hereditary Elliptocytosis on or about the 28th of March. As I have not heard from you I wonder whether you have received them. I would appreciate if you would let me know the results of the tests done.

I have the micro photographs of the peripheral blood pictures ready. The father's (S_1) and the proposita (S_3) had resistant osmotic fragility curves & these were now marked after incubation.

I have several cases who appear to have iron deficiency anaemia many of them with enlarged spleens & who gave a positive or intermediate reaction to Brewis test for G6PD deficiency. I wonder whether you could do the haptoglobins on them, for any evidence for haemolytic element. All of them are chronically anaemic.

Anxiously awaits an early reply.

With best wishes

Yours truly

M Gunasingam

BY AIR MAIL

PAR AVION
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விமானக் கடிதம்
Aerogramme

P. K. Sukumaran, Esqr.
Scientific Officer,
Cancer Research Institute,
Tata Memorial Centre.
Parel, Bombay-12
India.

පළමුව මෙතැනින් නමන්න. முதலில் இங்கே மடியுங்கள். First fold here.

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යවන්නාගේ නම සහ ලිපිනය
அனுப்புபவரின் பெயரும் விலாசமும்
Sender's name and address

Dr. N. Nagarathinam.
Govt. Hospital,
Gampaha, W.P.
Ceylon

ගුවන් ලියුමක කිසිවක් බහා නොතිබිය යුතුය. එසේ තිබුනහොත් ඊට වැඩිපුර අයකරනු හෝ සාමාන්‍ය තැපෑලෙන් යවනු හෝ ඇත.

இந்த விமானக் கடிதத்தினுள் வேறு ஏதாவது வைத்தனுப்பப்படுமாயின் மேலதிக கட்டணம் அறவிடப்படும் அல்லது சாதாரண தபால் மூலம் அனுப்பப்படும்.

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BY AIR MAIL

ED/3539/70 May 28, 1970.

Dear Dr. Nagaratnam,

This has reference to your letter dated 31.3.70 alongwith Blood samples of your interesting case (Silva family) and the subsequent letter dated 25.4.70.

I am extremely sorry for the delay in sending you the reports. These samples were investigated by three of us and the respective reports are enclosed. These reveal some interesting findings.

S_1, S_3 and S_4 show evidence of thalassemia trait (raised A_2) alongwith elliptocytes while S_5 shows only elliptocytes with normal A_2 .

As for studying haptoglobins on your iron-deficiency cases with splenomegaly, we are interested in Haptoglobin types. If you need any investigations on these lines we shall be glad to do so.

In your letter dated 31.3.70 you said that you shall be sending some reprints. I have so far not received any of them. You please look into the matter.

With kind regards,

Yours sincerely,

P.K. Sukumaran,
Scientific Officer.

Dr. N.Nagaratnam,
Physician,
Government Hospital,
Gampaha, W.P.
CEYLON.

Encl:

/swn

Brewer's test may be repeated with correction for anaemia if not done in earlier tests. We can confirm or make by performing a G-6-D screening test on them.

Known Normal, intermediate and deficient samples may be sent

JK
8/6

Govt. Hospital,
Gampaha. WP.
2.6.70.

Dear Mr. Subramaniam,

I thank you very much for your letter and the results of the tests on the family with hereditary Elliptocytosis & Thalassaemia. As I had no reply from you, I wrote to Dr. L.D. Sanghvi, thinking that you may have gone abroad. By now, this letter may have reached him, & he would have shown it to you. I have just started writing this family up & shall send you a draft within the next few days.

I am very sorry that I forgot to send you the reports I promised, but I have already posted them under separate cover. (sea-mail)

I have a family one of whose members have been diagnosed as Thalassaemia elsewhere & one of them ~~found~~ 14 years has received several blood transfusions. This girl & her brother it appears has reported at Banks of Ceylon. When I saw her she was mildly jaundiced otherwise well. Their peripheral blood pictures are consistent with a diagnosis of Thalassaemia, but this girl has a few microspherocytes & the brother nine. I am afraid. No haemoglobin analyses which had ever been done on them. Both show marked bone changes, hand, forearm bones, ribs, scapulae, vertebrae, pelvis etc. In view of the mild jaundice & the microspherocytes I am in whether they have another abnormality — hereditary Spherocytosis. Both have large spleens. I shall send you their blood samples next week.

As I wrote to you earlier, we have several cases, both young & old who have a Chance (? 1 in 4 persons) anaemia. Who are not out of the woods & who give a intermediate or positive reaction to Bowen's test. Many of them have a sizeable spleen.

I would like to investigate these cases further. Some workers have recorded chronic haemolysis with G6PD deficiency. Please let me know your views on these & the tests that may be useful. (? Estimation of haptoglobin levels etc).

I wonder whether you know anyone who could do B12 estimation ~~&~~ folate levels for me. No one has undertaken tests these in this country.

With kind regards

Yours truly

Nagaratnam

Dr. J.V.U.
for information
Please give your
comments if you
have any.
PKS 8/6

පළමුව මෙතැනින් නමන්න. முதலில் இங்கே மடிப்புங்கள். First fold here.

BY AIR MAIL
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Aerogramme

CEYLON FOR QUALITY TEAS

P. K. Sukumaran, Esqr.
Scientific Officer.
Cancer Research Institute
Tata Memorial Centre
Parel, Bombay 12.
India.

Sender's name and address
Dr. N. Nagaratnam
Govt. Hospital
Gampaha, W.P. Ceylon.

Second fold here.

An Aerogramme should not contain any enclosure; if it does it will be surcharged or sent by ordinary mail.

To open cut here.

ED/4429/68 June 8,1968

Dear Dr.Nagaratnam:

I am sorry that I could not reply your letter earlier wherein you wanted the addresses of Editors of three Journals.

I am afraid that none of these Journals are available in Bombay and hence the delay in my reply. Somehow I managed to get the addresses of two of them. The Editor-in-Chief of Scandinavian Journal of Haematology is

Prof.dr.med.Aage Videbaek
Amtssyghuset,
Hellerup, Kobenhavn. DENMARK.

The address of the Editor of Blut as gathered from the German Consulate is

Prof.dr.W.Stich
I Medical University Clinic
Ziemssenstrasse 1,
Munich, Federal Republic of Germany

I could not get the address of the Editor of Annales Paediatrici.

Regarding the article"Bone Changes in Congenital Haemolytic anaemias", I feel the title may stay as such but a sub-title "Report of Cases in Ceylon" may be added. A recent reference on bone changes in sickle cell anaemia is "Some Radiological Aspects of the S Haemoglobinopathies in Ibadan" by W.Peter Cookshott(1965) in Abnormal Haemoglobins in Africa, Blackwell, Oxford.

With kind regards.

Yours sincerely,
DCS
P.K.Sukumaran

Dr.N.Nagaratnam.
Government Hospital,
Kagalle,CEYLON

ED/4509/70 July 4, 1970

Dear Dr. Nagaratnam,

I am extremely sorry for the long delay in replying your letter and sending my comments on the manuscript you sent me.

My colleague Dr. J.V.Undevia(who did G-6-PD, haptoglobins and Rh typing) and I, went through the paper carefully. It will be seen that the three genetic conditions, instead of two, are exhibited in this interesting family. The third involves a gene, the D^u variant, on the Rh chromosome which makes this case more interesting. Dr. Undevia feels that he can elaborate on this aspect in a small paragraph along with interpretation on Haptoglobin types to be included in this paper provided such a proposal meets with your approval. In that case this paper has to be modified a bit which will include slight change in the title. Perhaps it can read as "Hereditary Elliptocytosis Associated with - β-Thalassemia and a variant of Rh (D)".

Needless to say that, with this change, Dr.Undevia be one of the authors. Please let me know your views in the matter. Meanwhile, to save time we are modifying the manuscripts and the final form shall be sent to you.

With kind regards,

Yours sincerely,

P.K. Sukumaran,
Scientific Officer.

3.VII.70

Dr. N. Nagaratnam, M.D.,M.R.C.P.,
Physician,
Government Hospital,
Gampaha. W.P.
CEYLON.

/snn

ED/6075 /70 August 27, 1970

Dear Dr. Nagaratnam,

In continuation of my letter dated August 22, 1970, I am sending you here with two copies of the manuscript of the paper titled " Hereditary Elliptocytosis Associated with Beta Thalassaemia and a Variant of Rh (D^U)".

I have already indicated our choice of two Journals to any of which this could be sent. I have also given the addresses of the editors in my letter cited above. Please acknowledge receipt of the manuscript.

With kind regards,

Yours sincerely,

P.K. Sukumaran
Scientific Officer.

22.VIII.70

Dr. N. Nagaratnam M.D.,M.R.C.P.,
Physician,
Govt. Hospital,
GAMPAHA W.P., (CEYLON).