LIBRARY OF CHROMATOGRAMS

H100 Hemoglobin Analyzer (Thalassemia Mode)

Version: 1.0

Contents

1. Introduction

Hemoglobinopathies--encompassing thalassemia syndromes and structural hemoglobin (Hb) variants--are the most prevalent monogenic disorders on Earth, affecting an estimated 7% of the global population. Accurate laboratory identification is essential for patient management, genetic counseling, and prenatal screening.

This Chromatogram Library is compiled specifically for users of the Lifotronic H100 Hemoglobin Analyzer operating in β-Thalassemia Mode (hereinafter "H100"). The library serves as an reference for:

(1) Calibrator and quality-control chromatogram patterns;
(2) Normal whole-blood chromatogram acceptance criteria;
(3) Chromatographic profiles of common seen major Hb variants, with structured interpretation descriptions for each case;

Each variant entry is supplemented with evidence-based clinical background citing peer-reviewed literature published from 2020 to 2025.

1.1 Normal Hemoglobin Composition

In healthy adults three hemoglobin species are present: HbA ($\alpha_2\beta_2$) >95%; HbA_2 ($\alpha_2\delta_2$) 2.5–3.5%; HbF ($\alpha_2\gamma_2$) ≤1% after 6 months of age. On the H100 in β-Thalassemia Mode, peaks elute left-to-right as: HbA1a → HbA1b → HbF → $LA1c^+$ → HbA1c → P3 → P4 → HbA0 → HbA2.

1.2 Acceptance Criteria

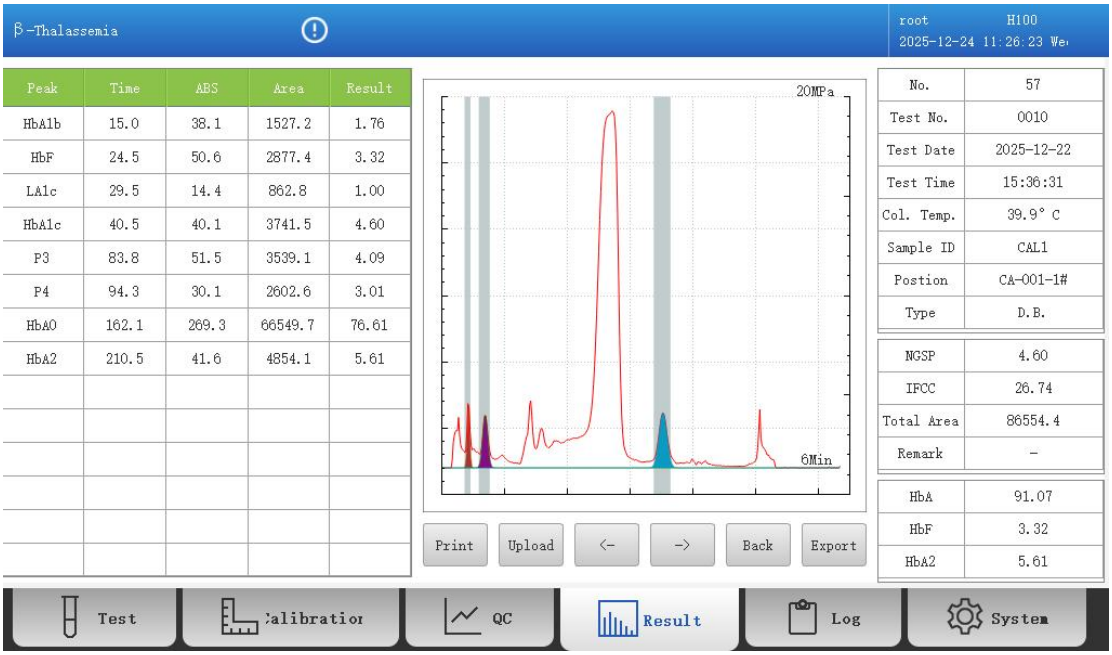
Criterion	Acceptable Range / Condition
HbA1c retention time	35.0 – 50.0 s
HbA0 retention time	209.0 – 213.0 s
Peak morphology	Symmetric; no shoulders, bifurcations, or tailing
E5 alert flag	Absent
P3/P4 peak area	≤ 10% of total
New or unidentified peaks	None

2. Normal Reference Chromatograms

2.1 Calibrations

Calibrators are highly purified, specifically standardized materials used to establish the baseline measurement curve of the instrument. They ensure that the retention times and peak area calculations perfectly align with international NGSP and IFCC standards.

2.1.1 Calibrator Level 1



Peak	Time	ABS	Area	Result
HbA1b	15.0	38.1	1527.2	1.76
HbF	24.5	50.6	2877.4	3.32
LA1c	29.5	14.4	862.8	1.00
HbA1c	40.5	40.1	3741.5	4.60
P3	83.8	51.5	3539.1	4.09
P4	94.3	30.1	2602.6	3.01
HbA0	162.1	269.3	66549.7	76.61
HbA2	210.5	41.6	4854.1	5.61

No.	57
Test No.	0010
Test Date	2025-12-22
Test Time	15:36:31
Col. Temp.	39.9° C
Sample ID	CAL1
Postion	CA-001-1#
Type	D.B.

NGSP	4.60
IFCC	26.74
Total Area	86554.4
Remark	-

HbA	91.07
HbF	3.32
HbA2	5.61

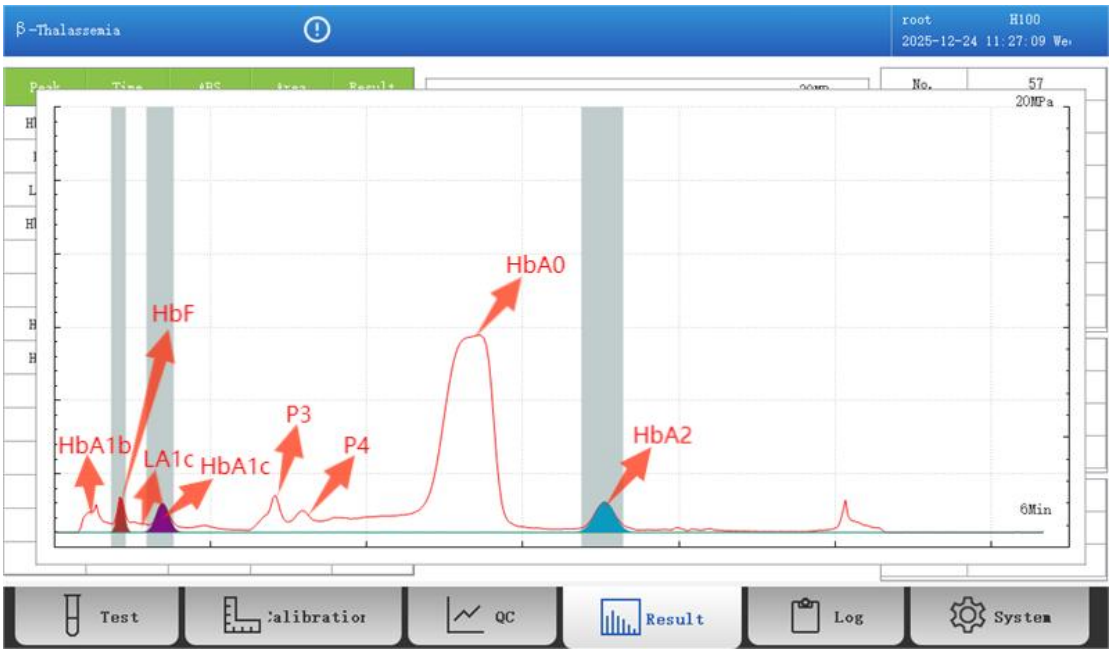
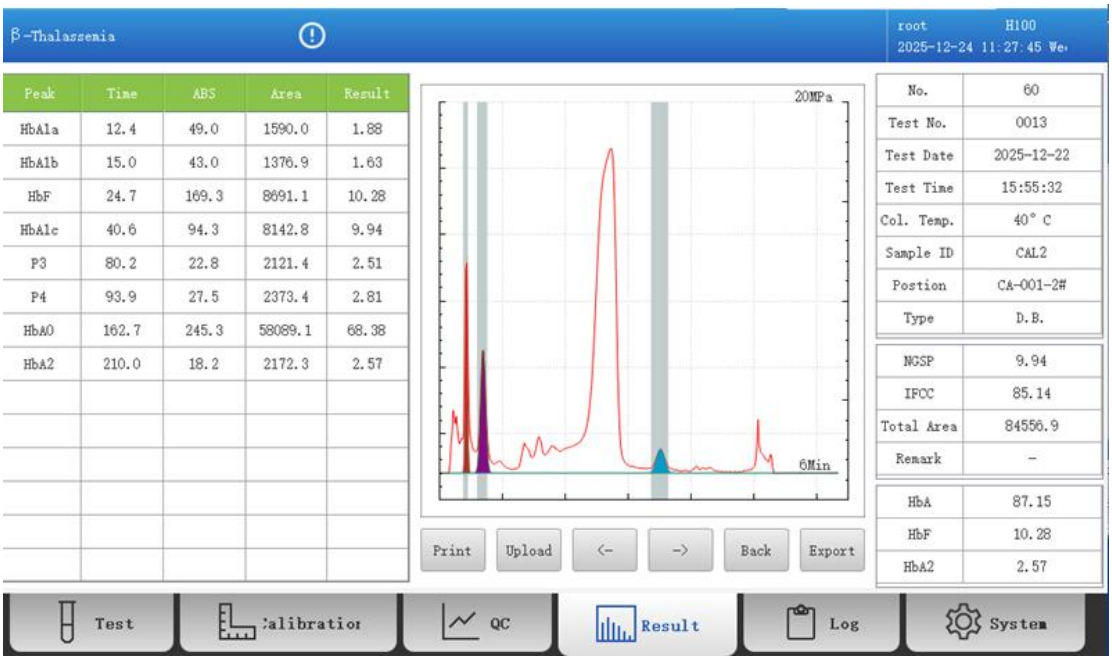


Figure 2.1.1 H100 (β-Thalassemia Mode) — Calibrator Level 1

2.1.2 Calibrator Level 2



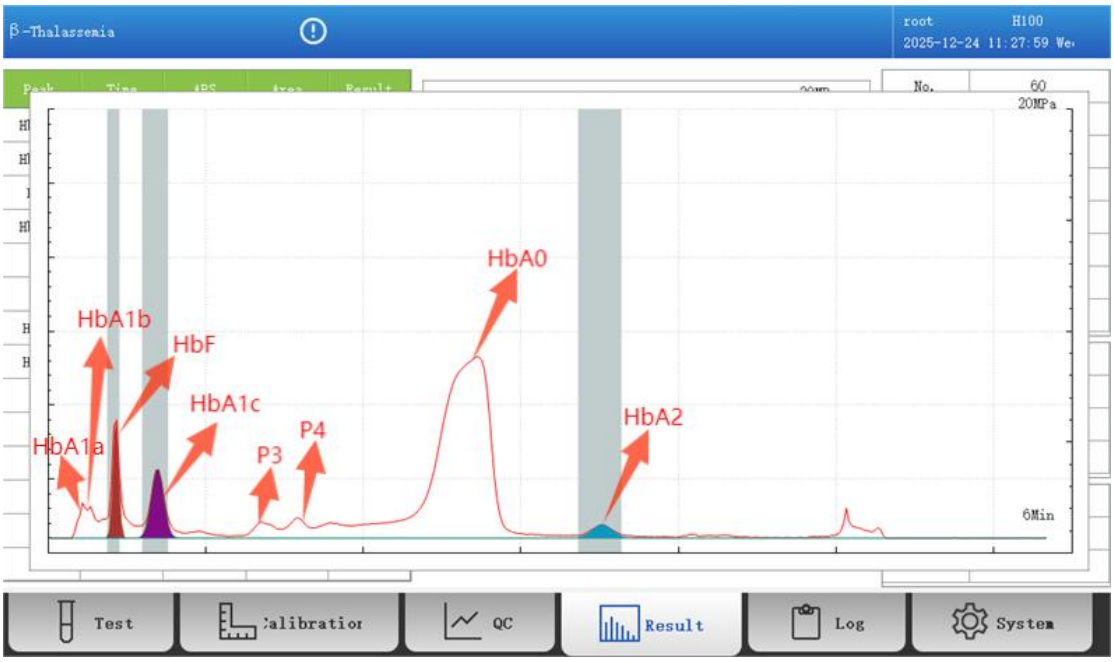
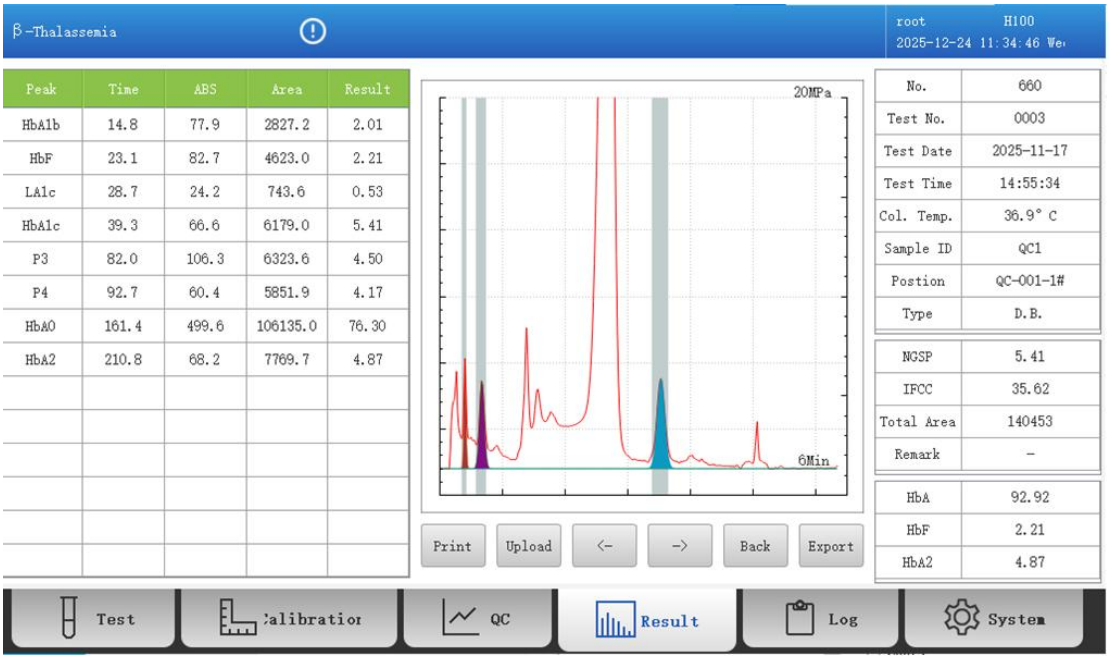


Figure 2.1.2 H100 (β-Thalassemia Mode) — Calibrator Level 2

2.2 Quality Control

Quality control materials are run daily to monitor the ongoing analytical performance, precision, and reliability of the H100 analyzer, ensuring that shifts in column temperature or buffer pH have not negatively impacted peak separation.

2.2.1 QC Level 1



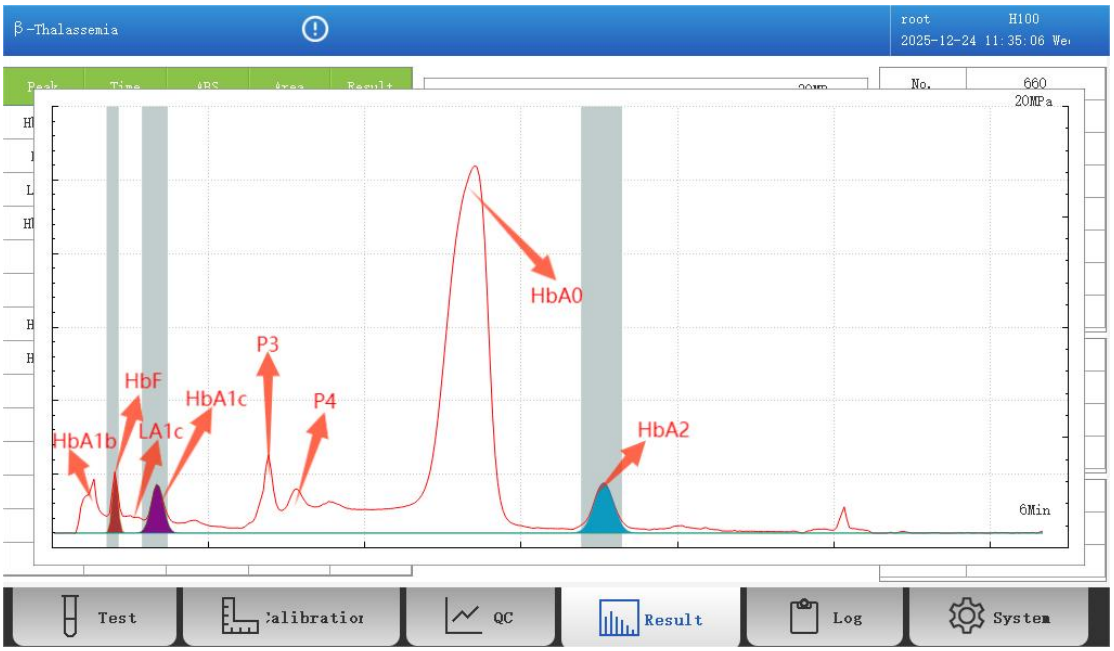
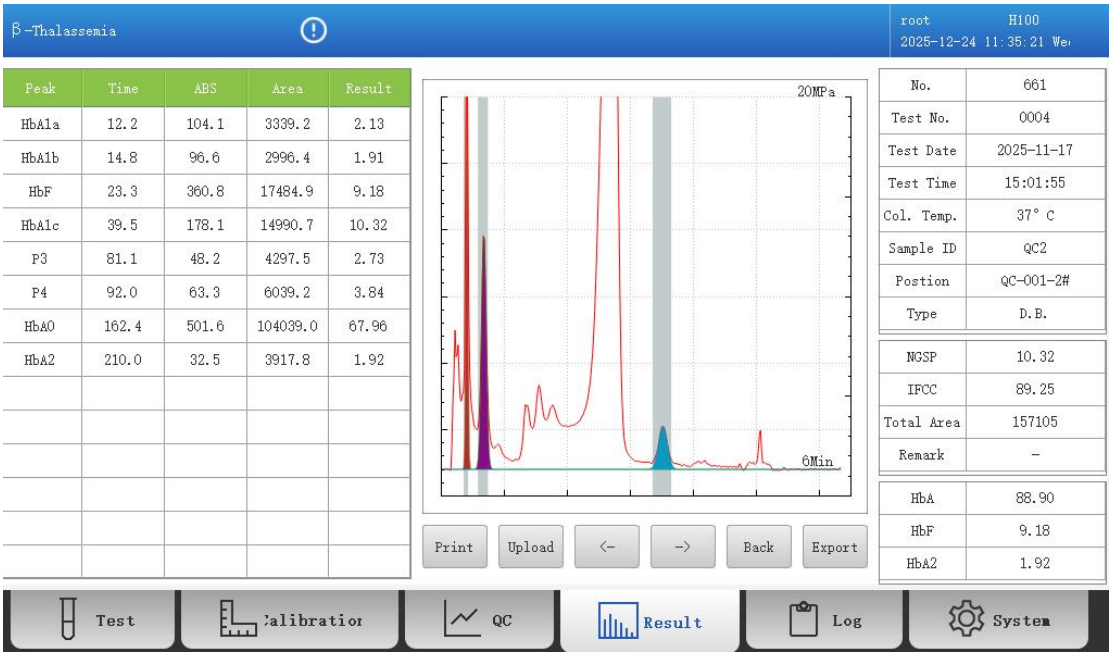


Figure 2.2.1 H100 (β-Thalassemia Mode) — QC Level 1

2.2.2 QC Level 2



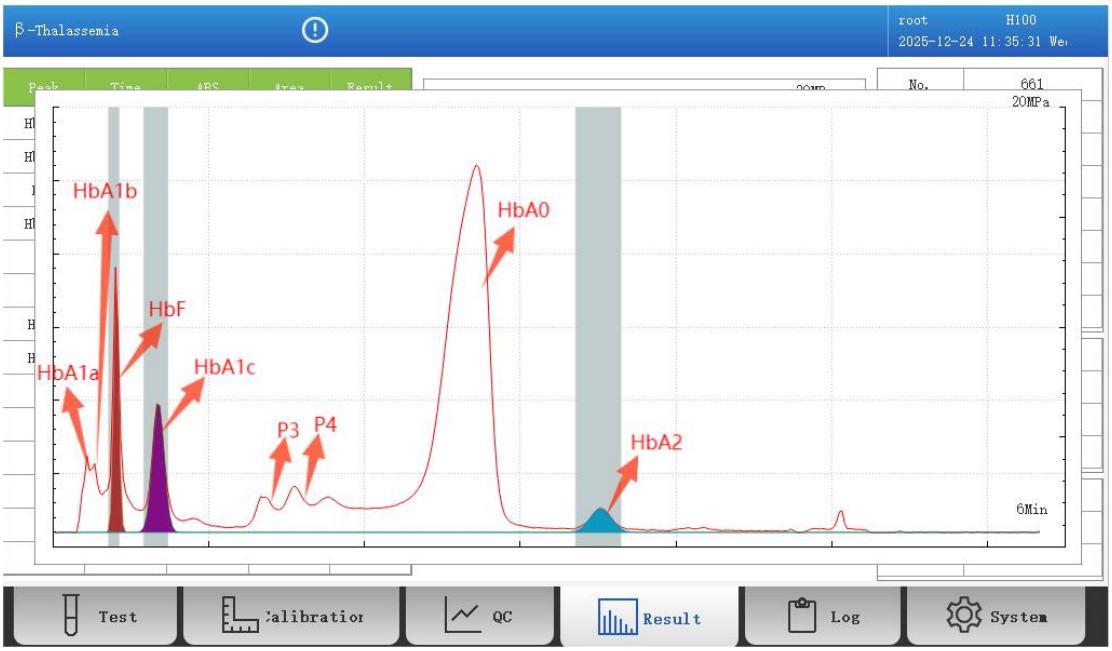


Figure 2.2.2 H100 (β-Thalassemia Mode) — QC Level 2

2.3 Clinical Whole-Blood Specimens

2.3.1 Normal Whole-Blood Specimen

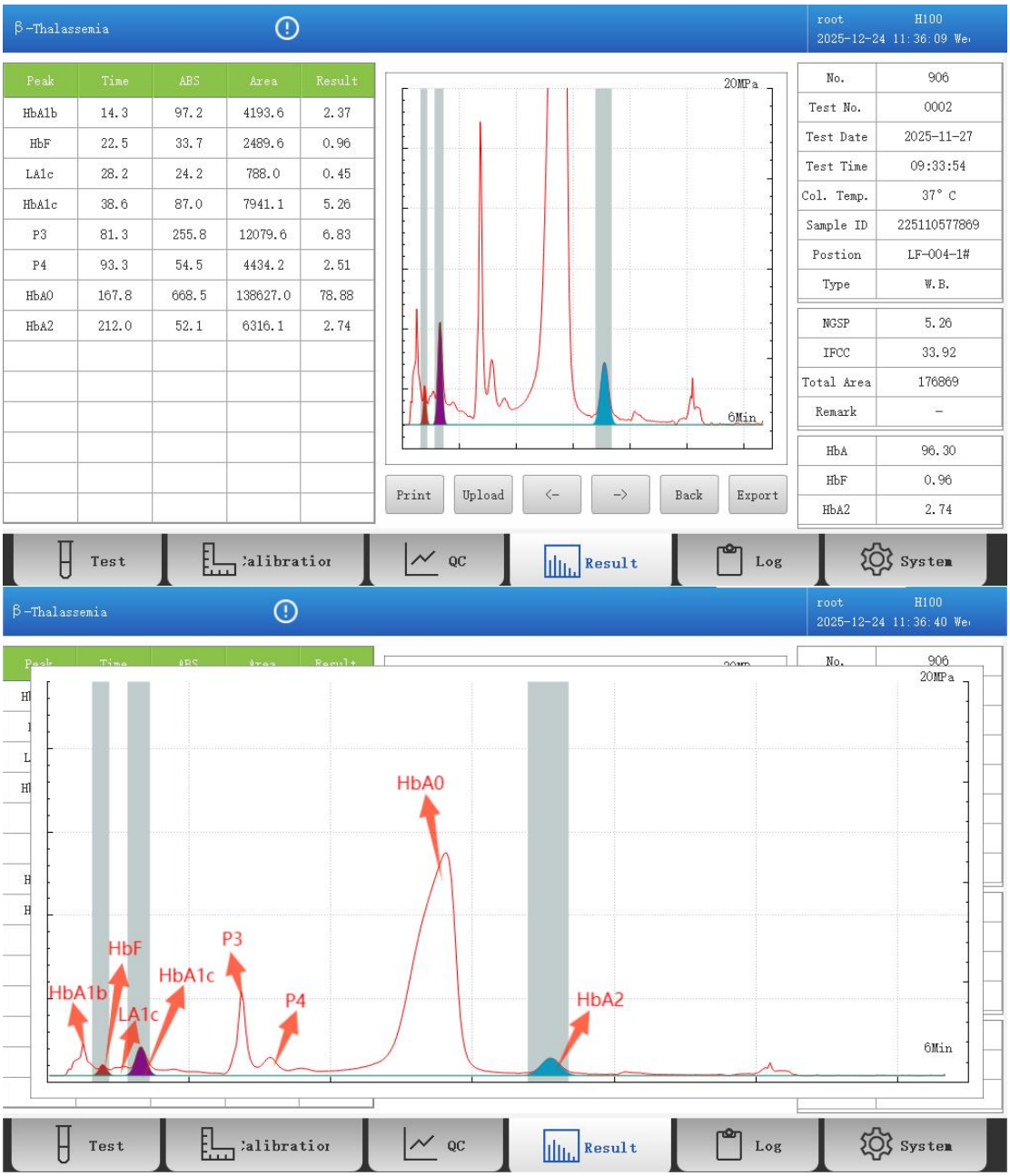


Figure 2.3.1 H100 (β-Thalassemia Mode) — Normal whole-blood value chromatogram

2.3.2 β-Thalassemia Carrier (Elevated HbA_2)

Carriers present with isolated elevation of HbA_2 (typically 3.5–7.0%). The chromatogram morphology is otherwise normal. No E5 flag; no additional peaks.

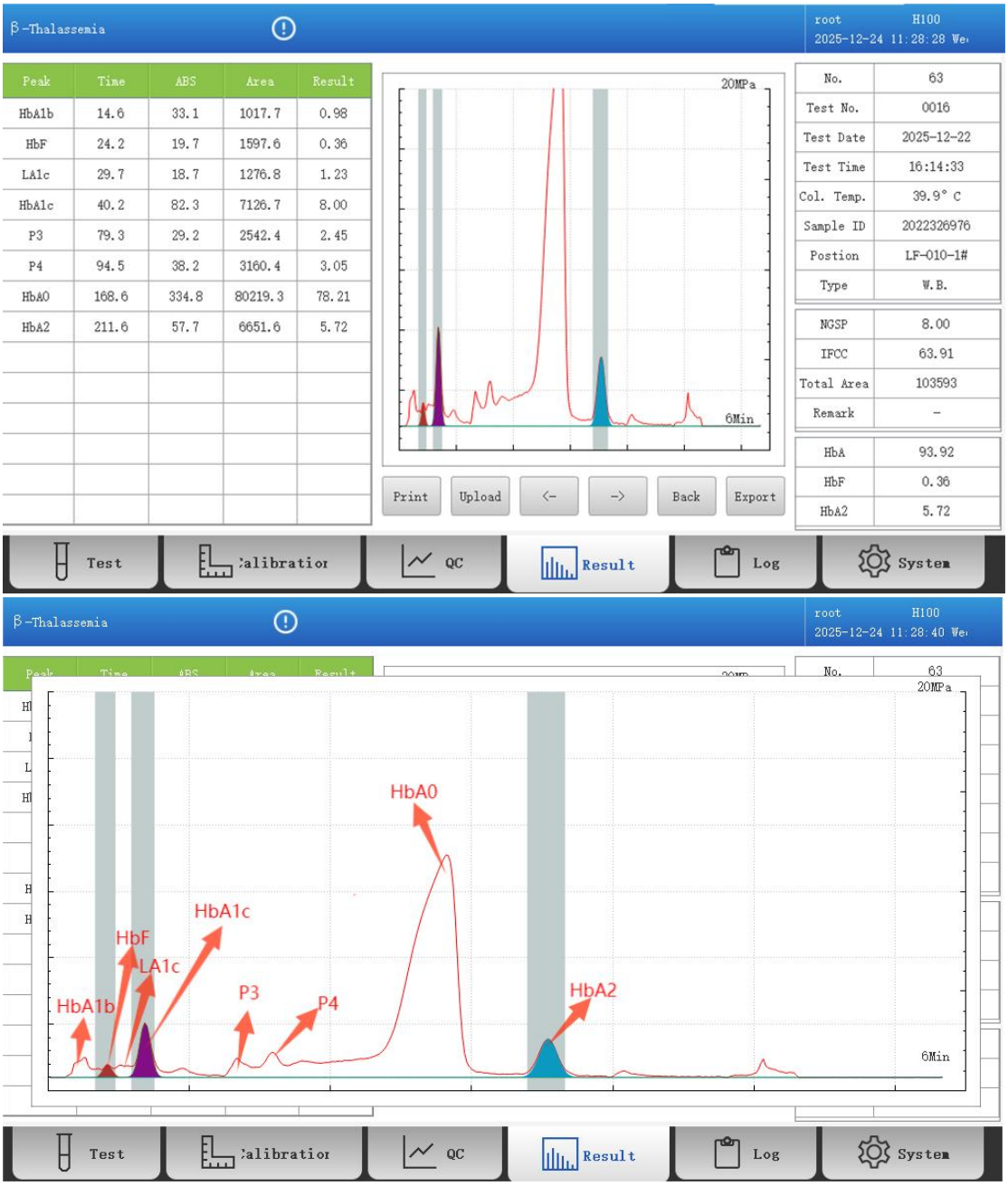


Figure 2.3.2 H100 (β-Thalassemia Mode) — β-Thalassemia carrier with elevated HbA_2

3. Hemoglobin Variant Library

This section presents the chromatographic profiles of nine clinically significant Hb variants encountered in the H100 operating population. Table 3.1 provides a quick-reference overview.

Variant	Chain	Mutation	H100 RT (s)	H100 Window	Typical %
Hb E	β	c.79G>A (Glu26Lys)	240–242	Hb E window	25–74%
Hb D-Punjab	β	c.364G>C (Glu121Gln)	252–254	Hb D window	43–75%
Hb S	β	c.20A>T (Glu6Val)	264–266	Hb S window	43–48%
Hb C	β	c.19G>A (Glu6Lys)	299–303	Hb C window	48–99%
Hb Q-Thailand	α_1	c.223G>C (Asp74His)	~288–292	Hb C window ⚠	20–25%
Hb G-Honolulu	α	c.91G>C (Glu30Gln)	~242–246	Hb E window ⚠	25–30%
Hb G-Coushatta	β	c.68A>C (Glu22Ala)	~228–233	Hb E window ⚠	43–48%
Hb G-Taipei	β	c.68A>G (Glu22Gly)	~238–242	Hb E window ⚠	40–44%
Hb Ottawa	α	c.46G>C (Gly16Arg)	~268–272	Hb S window ⚠	22–27%

⚠ = co-elutes in a different named recognition window; confirmation by CZE or molecular analysis is mandatory.

Profile 1: Hemoglobin E (Hb E)

Parameter	Value
HGVS Notation	HBB: c.79G>A
Amino Acid Change	β26 Glu→Lys (Glutamic acid→Lysine)
Globin Chain	β-chain
H100 Recognition Window	Hb E window
H100 Retention Time	~240 – 242 s
Heterozygote Hb E %	~25 – 30%
Homozygote Hb E %	~70 – 80% (no HbA0 or HbA1c)
Geographic Prevalence	Southeast Asia; China (Yunnan, Guangdong, Guangxi, Sichuan, Hunan)

Clinical and Molecular Background

Hb E results from a G→A transversion at codon 26 of HBB. The mutation simultaneously changes glutamic acid to lysine and activates a cryptic splice site, reducing β-globin mRNA production. Hb E is therefore both a structural variant and a mild form of β-thalassemia. It is the second most common structural Hb variant worldwide after Hb S.

Heterozygotes (HbAE) are clinically silent. Homozygotes (HbEE) have mild microcytic hypochromic anemia with target cells but are not transfusion-dependent. Compound heterozygosity for Hb E and β-thalassemia (HbE/β-thal) produces a phenotype ranging from thalassemia intermedia to thalassemia major, depending on the co-inherited β-thalassemia allele.

Laksap et al. (Clin Chem Lab Med, 2024) confirmed the importance of precise Hb E quantitation using both HPLC and CZE for differential diagnosis in Southeast Asian screening programs. A 2024 clinico-hematological survey in Northeast India identified significant population burden from HbE disease in both heterozygous and homozygous states.

3.1.1 IFCC Reference Material — Hb E Homozygote (Lot 2021-0250)

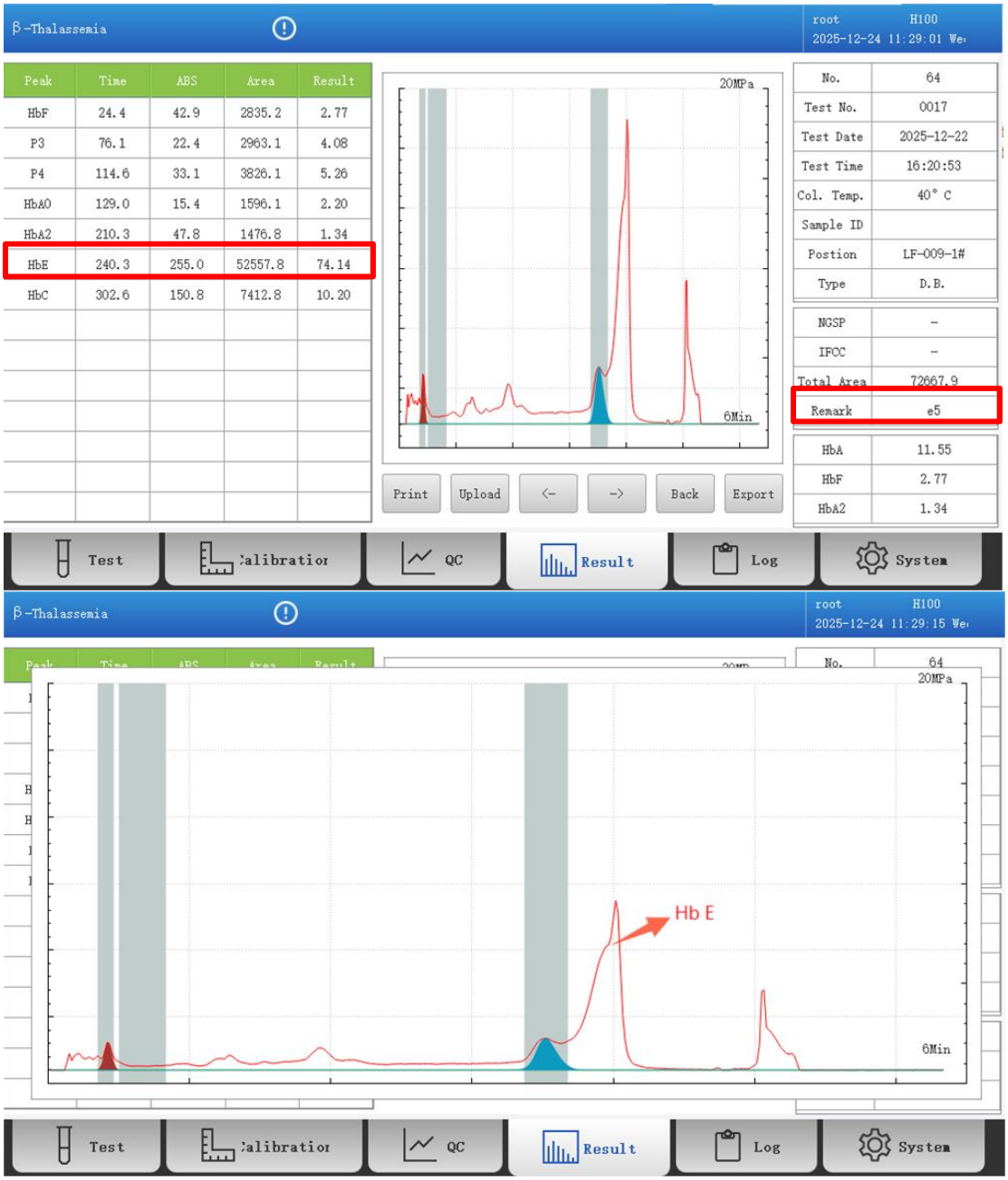
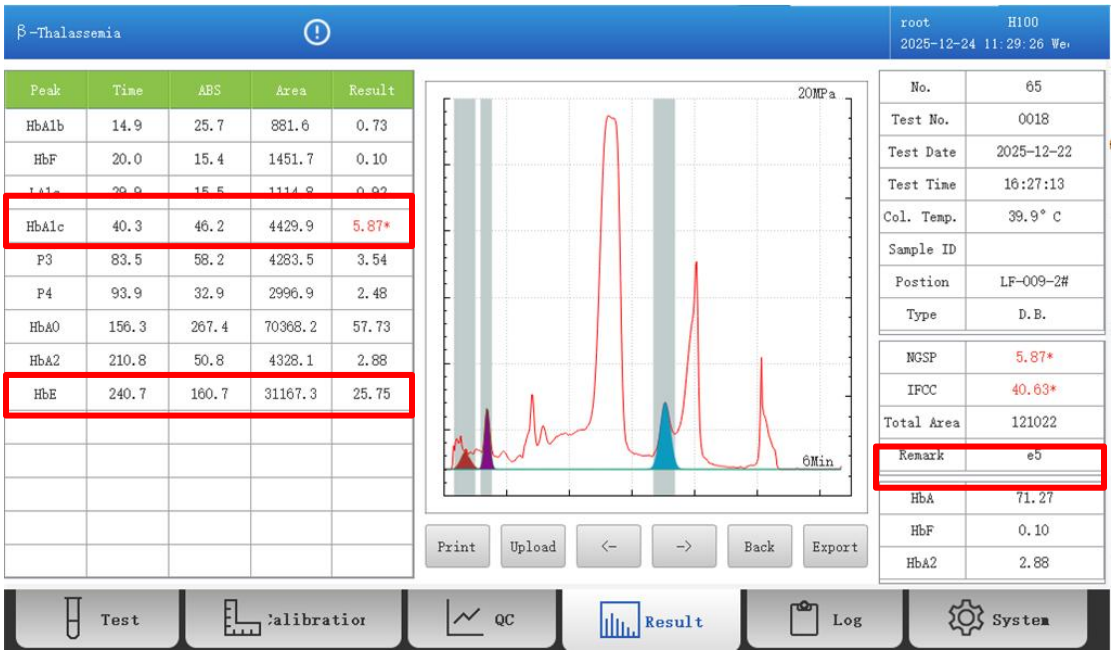


Figure 3.1.1 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2021-0250: Hb E homozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	Not detected (no β^A chain present; Hb E replaces β^A entirely)
HbA0 Peak	Absent — no normal β-chain produced in the homozygous state
Hb E RT	**240.3 s**

Hb E Area	**74.14% (dominant peak)**
Peak Morphology	Single sharp peak eluting after HbA2; characteristic ‘lone tower’ pattern with no preceding HbA0.
E5 Flag	Present — instrument correctly reports possible variant interference
Interpretation	● Chromatogram consistent with Hb E homozygote. ● No HbA0 and no HbA1c detected, as expected when both β-globin alleles carry the Glu26Lys mutation.
⚠ CAUTION	● In Hb E homozygotes, HbA1c cannot be measured by HPLC. ● Fructosamine, glycated albumin, or continuous glucose monitoring should be used for diabetes surveillance in affected patients.

3.1.2 IFCC Reference Material — Hb E Heterozygote (Lot 2020-3033)



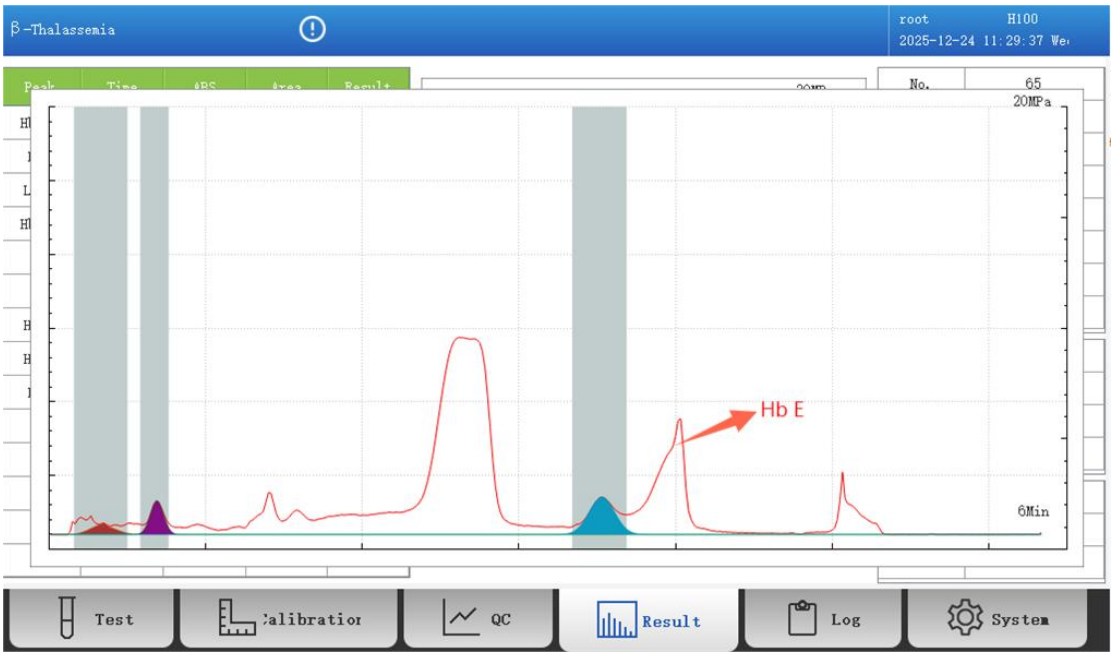


Figure 3.1.2 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2020-3033: Hb E heterozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.87%** (reportable; within normal accuracy)
Hb E Peak RT	**240.7 s**
Hb E Area	**25.75%**
HbA0 Peak	Present — normal β^A chain coexists with β^E chain
Peak Morphology	Sharp, discrete peak appearing immediately after the HbA2 peak; HbA1c and HbA0 peaks retain normal morphology
E5 Flag	Present
Instrument ID	Hb E (correct identification)
Interpretation	• Chromatogram consistent with Hb E heterozygote (HbAE). • HbA1c is accurately reported. • The Hb E peak area (~26%) is typical of the thalassemic reduction in β^E chain output.

3.1.3 Clinical Specimen — Hb E Homozygote (Case 180)

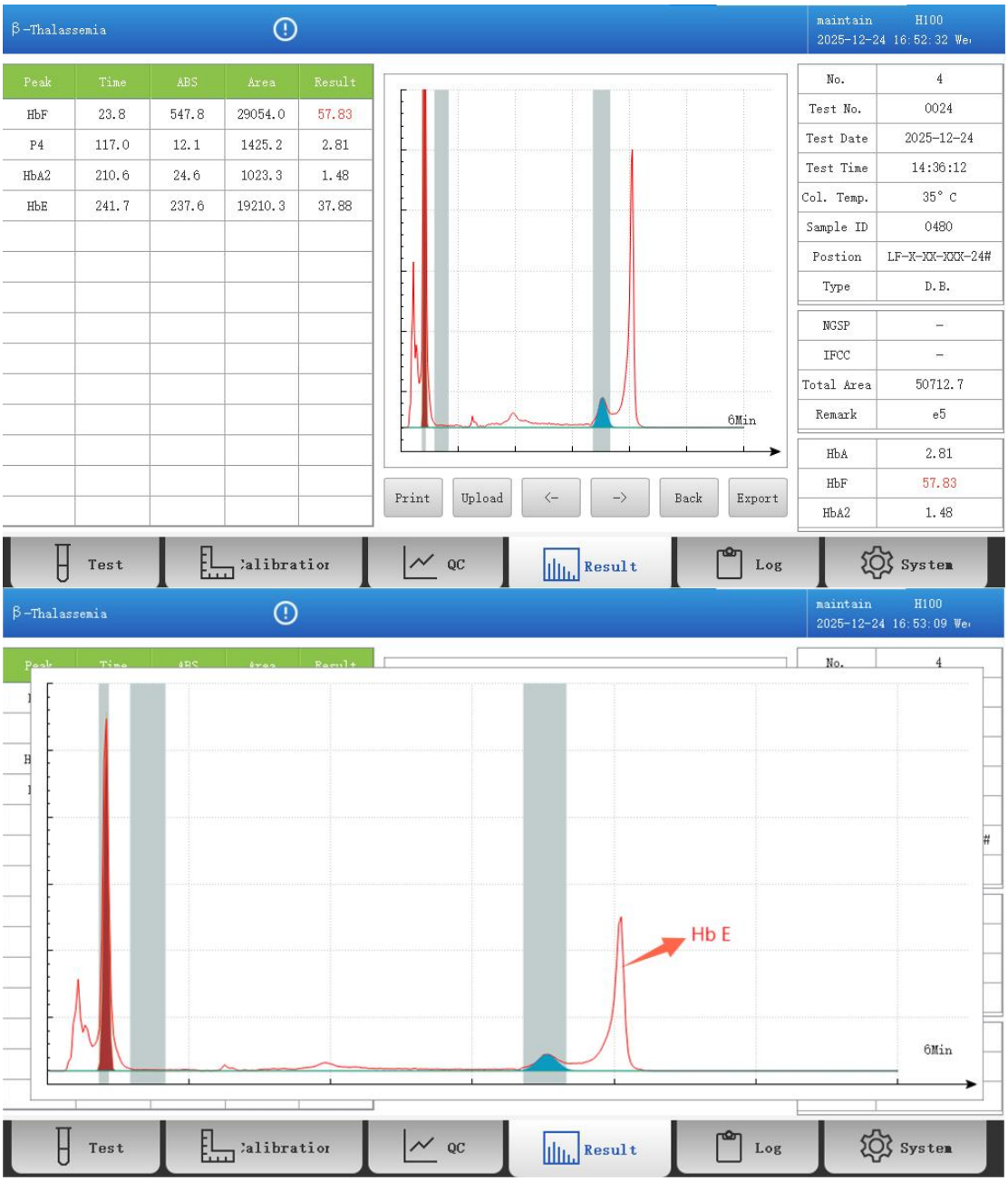


Figure 3.1.3.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case 180)

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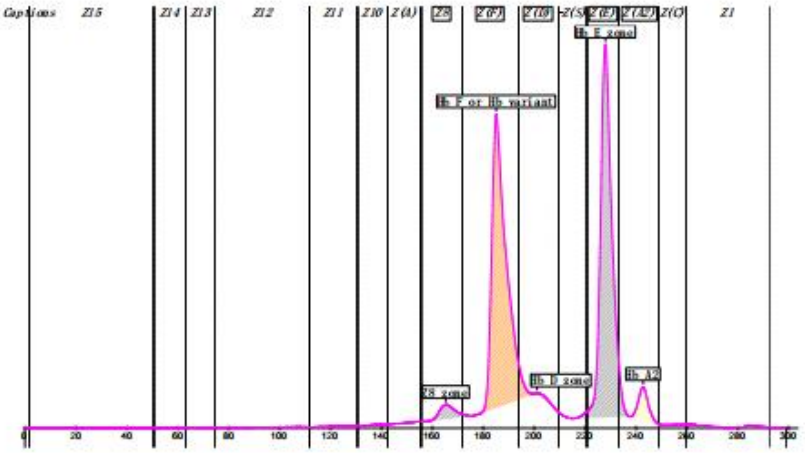
Sample # : 25 Date : 2024/10/11 ID : SAMPLE-1

Depart. : Birth. :



Haemoglobin Electrophoresis

Name	%	Normal Values %
Z8 zone	2.4	
Hb F or Hb variant	47.0	
Hb D zone	0.3	
Hb E zone	46.8	
Hb A2	3.5	

Figure 3.1.3.2 Hemoglobin Analysis Electropherogram of Special Sample No. 180 (Sebia Capillary Electrophoresis Method)

CHROMATOGRAM INTERPRETATION	
HbA1c Result	Not detected
HbA0 Peak	Absent
HbF	Markedly elevated (compensatory)
Hb E RT	**241.7 s**
Hb E Area	**37.88%** (lower than IFCC reference material due to elevated HbF)
E5 Flag	Present
Sebia CZE	**Hb E zone: 46.8%** (confirms variant identity)

Interpretation	• **Absent HbA0 and HbA1c with elevated HbF and a dominant post-HbA2 peak** are pathognomonic of Hb E homozygosity. • The lower Hb E percentage vs. IFCC reference material reflects elevated HbF diluting the total area.
✓ CLINICAL TIP	The elevated HbF observed in this case is a clinically favourable modifier, potentially mitigating anaemia severity by supplementing oxygen delivery.

3.1.4 Clinical Specimen — Hb E Heterozygote (Case 320)

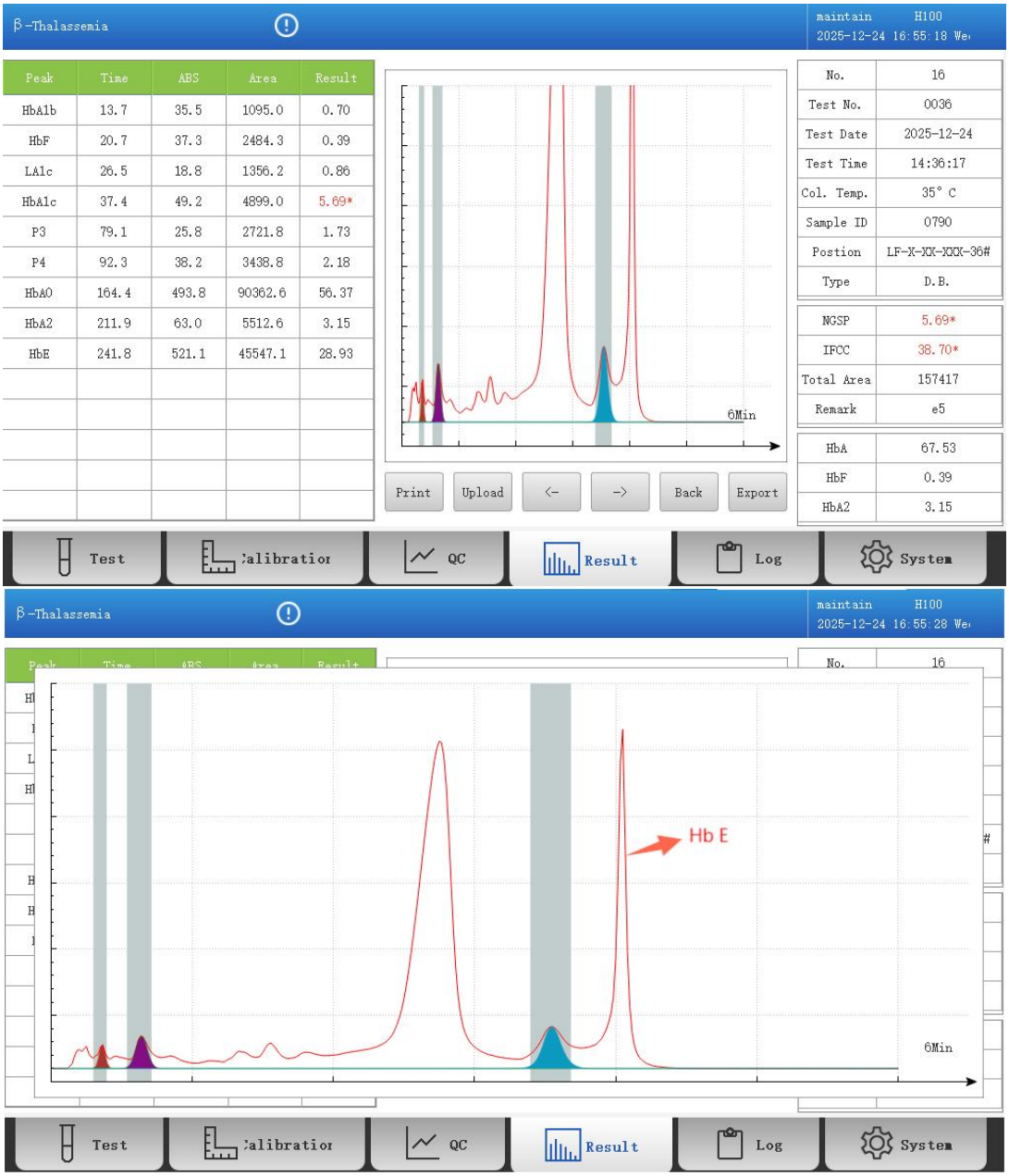


Figure 3.1.4.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case 320): Hb E heterozygote

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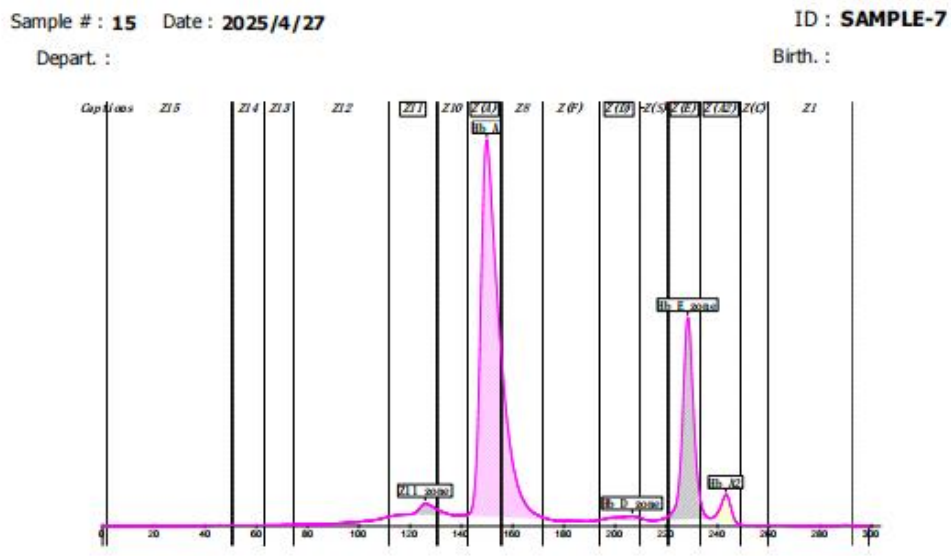


Name	%	Normal Values %
Z11 zone	1.7	
Hb A	71.5	
Hb D zone	1.2	
Hb E zone	22.8	
Hb A2	2.8	

Figure 3.1.4.2 Hemoglobin Analysis Electropherogram of Special Sample No. 320 (Sebia Capillary Electrophoresis).

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.09%** (reportable)
Hb E RT	**241.8 s**
Hb E Area	**28.93%**
Peak Morphology	• Sharp, narrow peak immediately following HbA2; • HbA0 visible and normal
E5 Flag	Present
Sebia CZE	Hb E zone: 22.8% (confirms Hb E heterozygote)
Interpretation	• Pattern consistent with Hb E heterozygosity. • HbA1c accurately reported.

	• No clinical intervention required; genetic counseling advised for reproductive planning.

Profile 2: Hemoglobin D-Punjab (Hb D-Los Angeles)

Parameter	Value
HGVS Notation	HBB: c.364G>C
Amino Acid Change	β121 Glu→Gln (Glutamic acid→Glutamine)
Globin Chain	β-chain
H100 Recognition Window	Hb D window
H100 Retention Time	~252 – 254 s
Heterozygote Hb D %	~43 – 48%
Homozygote Hb D %	~70 – 80%
Geographic Prevalence	Northwest India, Xinjiang/Silk Road corridor, northern Africa, northern Europe; scattered throughout China

Clinical and Molecular Background

Hb D-Punjab is caused by a G→C transversion at codon 121 of HBB, replacing the negatively charged glutamic acid with the neutral glutamine. Unlike Hb S, this substitution does not cause polymerization or sickling under deoxygenation. Heterozygotes and homozygotes are generally asymptomatic.

Clinical significance increases markedly when Hb D-Punjab is co-inherited with Hb S (producing HbSD disease with intermediate sickling) or with β-thalassemia (producing symptomatic microcytic anemia). A 2025 cross-sectional study from Sindh, Pakistan (Effendi et al., PLOS ONE) identified Hb D as the predominant structural variant in 4,783 HPLC chromatograms, representing 56.6% of all Hb variants detected.

A 2024 case report (Dholariya et al., Indian J Clin Biochem) highlighted that Hb D-Punjab trait may cause falsely elevated HbA1c on certain HPLC platforms due to co-elution artifacts, reinforcing the importance of platform-specific performance validation.

3.2.1 IFCC Reference Material — Hb D Homozygote (Lot 2021-0643)

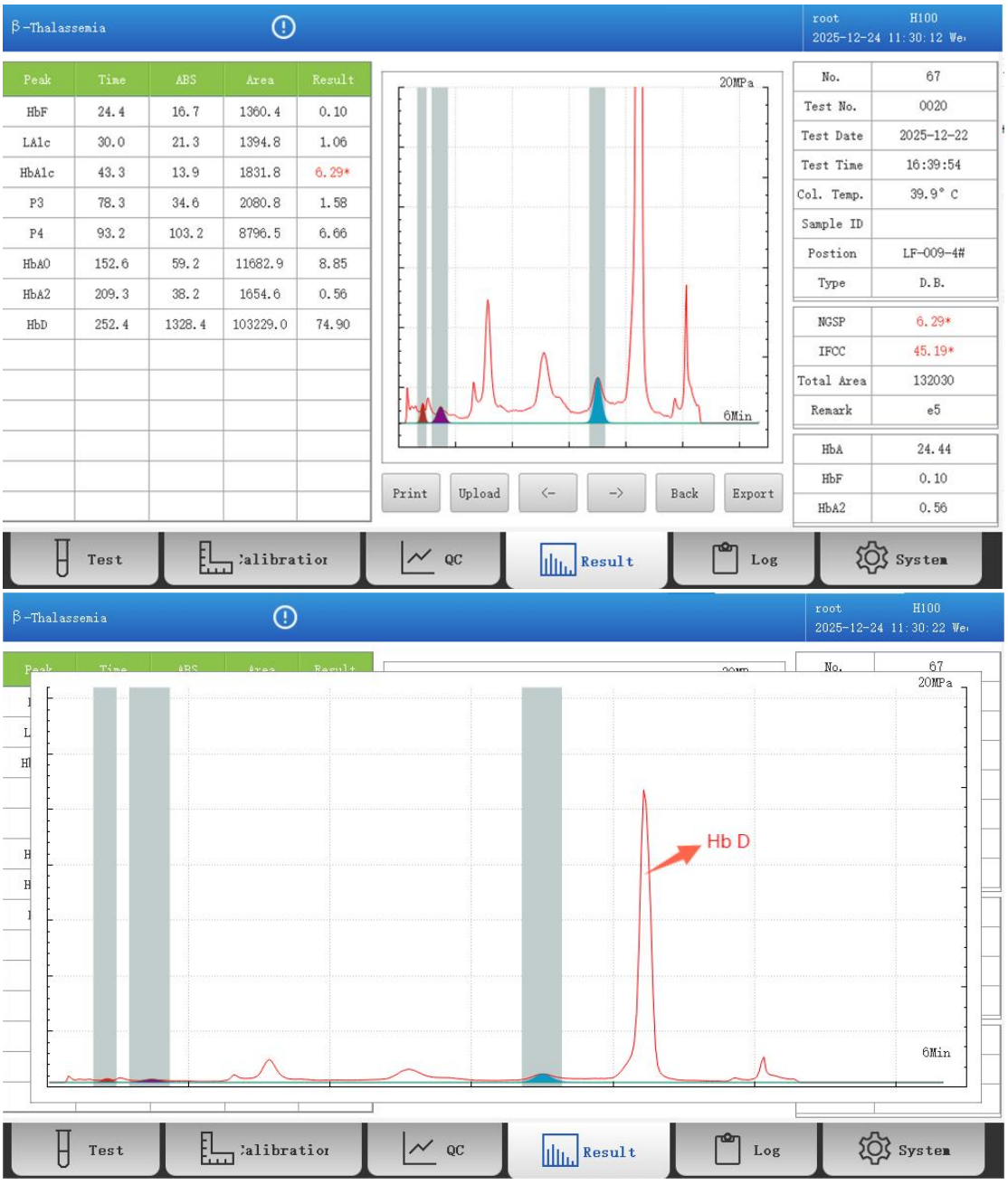
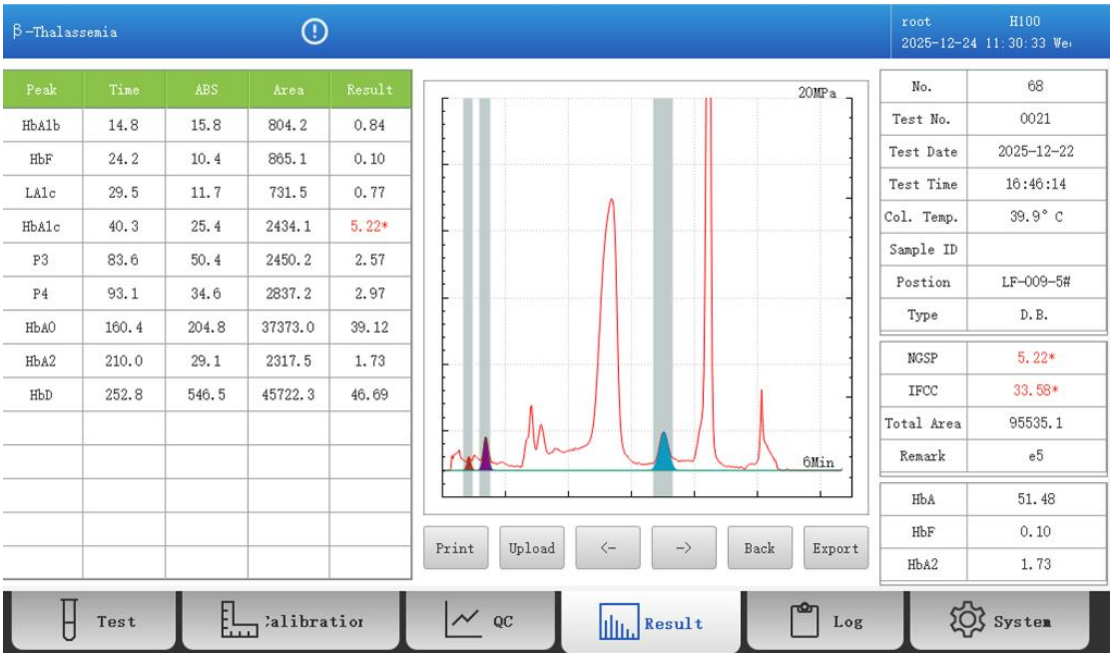


Figure 3.2.1 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2021-0643: Hb D homozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**6.29%** (reportable)
Hb D RT	**252.4 s**
Hb D Area	**74.90%** (dominant peak after HbA2)
Peak Morphology	Tall, sharp, narrow peak immediately post-HbA2; taller than the HbA0 peak region
E5 Flag	Present
Instrument ID	Hb D (correct identification)
Interpretation	● Chromatogram consistent with Hb D homozygote. ● The Hb D peak is the dominant post-HbA2 feature. ● HbA1c is reportable.

3.2.2 IFCC Reference Material — Hb D Heterozygote (Lot 2023-0136)



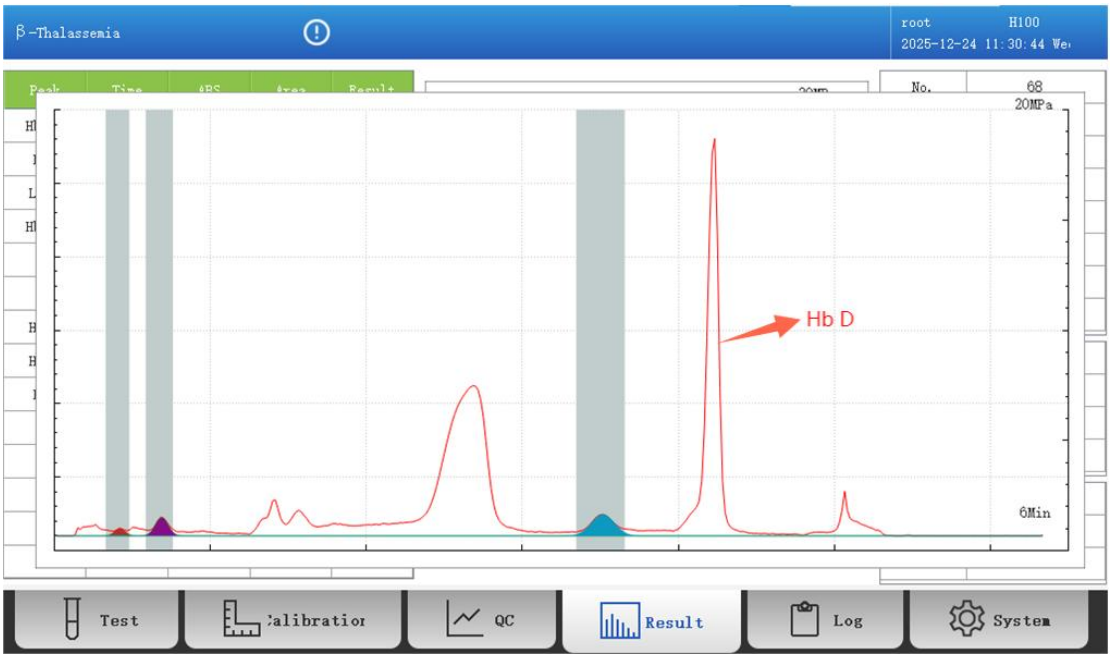


Figure 5.3 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2023-0136: Hb D heterozygote.

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.22%** (reportable)
Hb D RT	**252.8 s**
Hb D Area	**46.69%**
Peak Morphology	● Sharp discrete peak post-HbA2; ● comparable in height to HbA0; ● HbA1c and HbA0 peaks morphologically normal.
E5 Flag	Present
Instrument ID	Hb D (correct identification)
Interpretation	● Chromatogram consistent with Hb D heterozygote (HbAD). ● HbA1c accurately reported. ● No clinical consequence; genetic counseling recommended.

3.2.3 Clinical Specimen — Hb D-Punjab (Case 264)

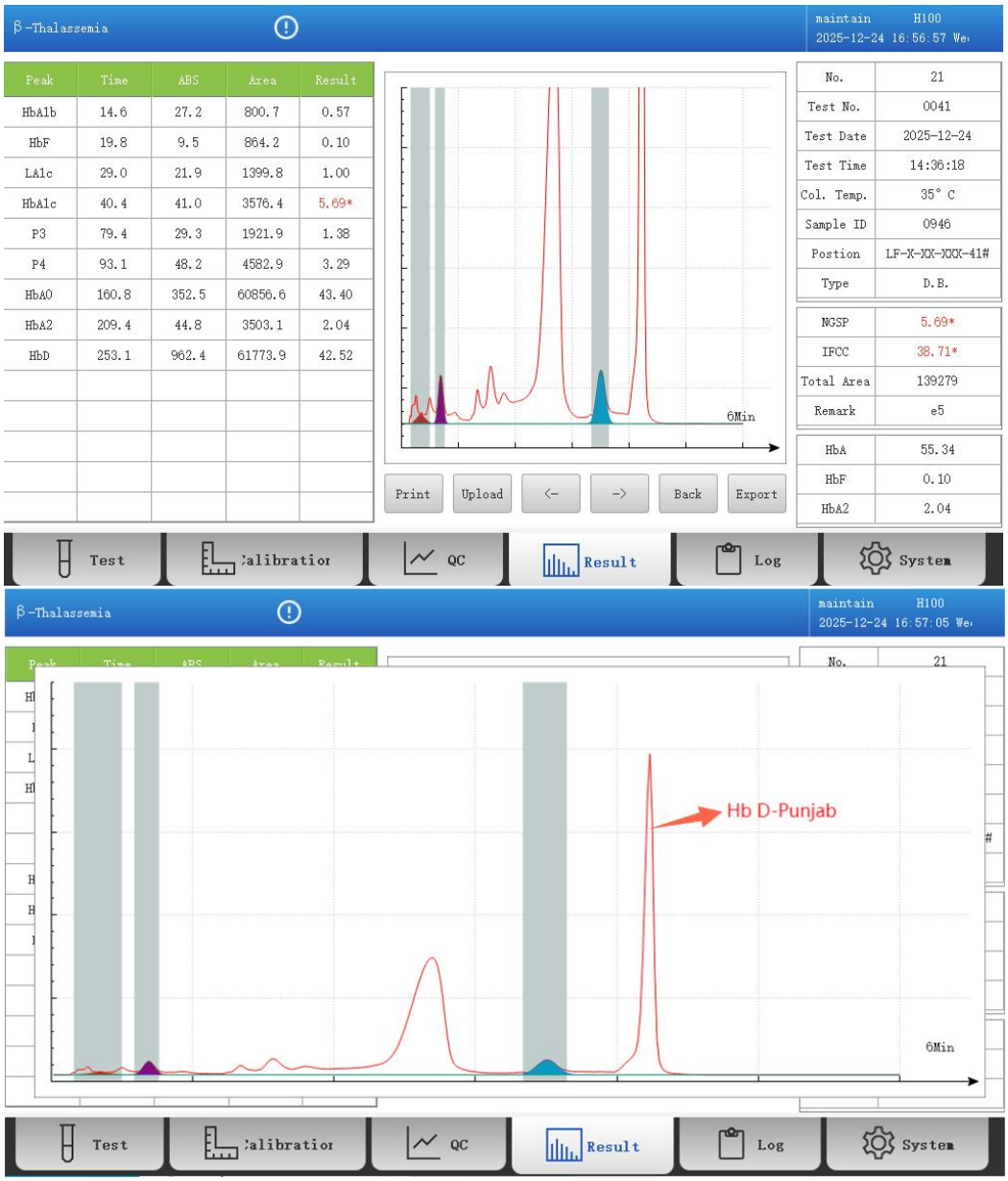


Figure 3.2.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case 264): Hb D-Punjab

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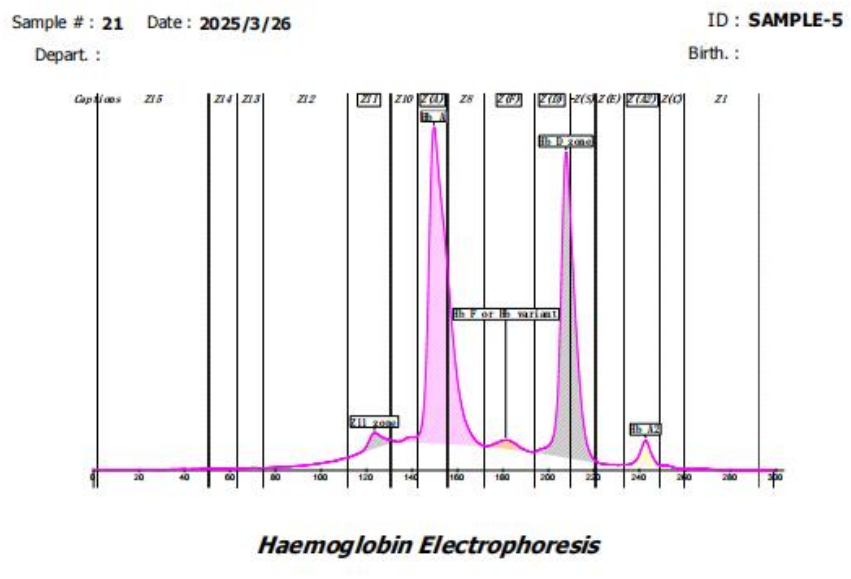

Haemoglobin Electrophoresis

Name	%	Normal Values %
Z11 zone	2.0	
Hb A	55.1	
Hb F or Hb variant	1.5	
Hb D zone	39.0	
Hb A2	2.4	

Figure 3.2.3.2 Hemoglobin Analysis Electropherogram of Special Sample No. 264 (Sebia Capillary Electrophoresis Method)

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.09%** (reportable)
Hb D RT	**253.1 s**
Hb D Area	**42.52%**
Peak Morphology	● Sharp peak post-HbA2; slightly lower than HbA0; ● HbA1c peak morphologically intact.
E5 Flag	Present
Confirmatory	● Sanger sequencing: HBB c.364G>C (Hb D-Punjab). ● Sebia CZE: Hb D zone 39.0%
Interpretation	● Pattern consistent with Hb D-Punjab heterozygosity. ● HbA1c reportable. ● Correlation between H100 (42.5%) and Sebia CZE (39.0%) is within expected inter-method variation.

3.2.4 Clinical Specimen — Hb D (Case 366)

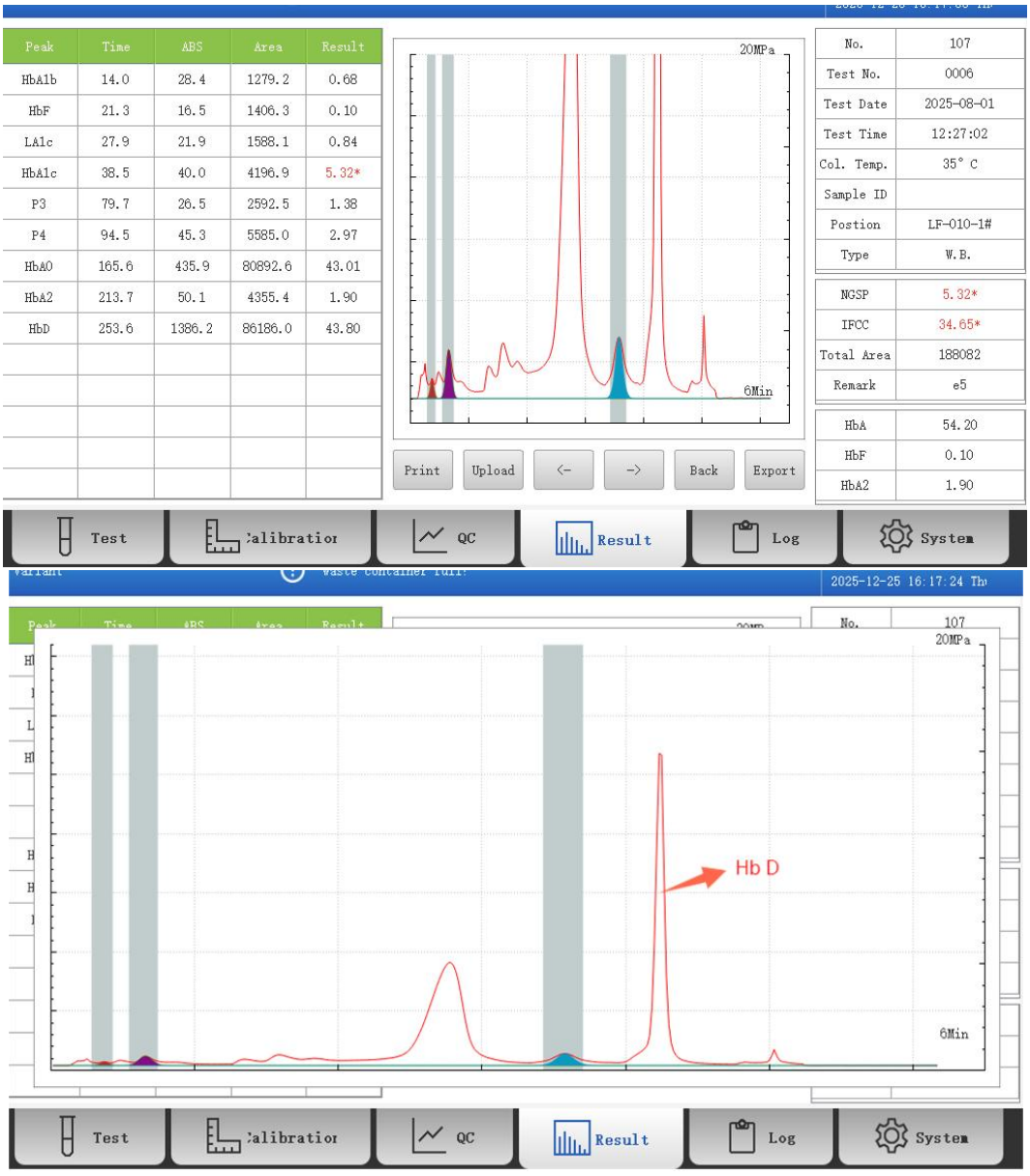

Figure 3.2.4.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case 366): Hb D heterozygote

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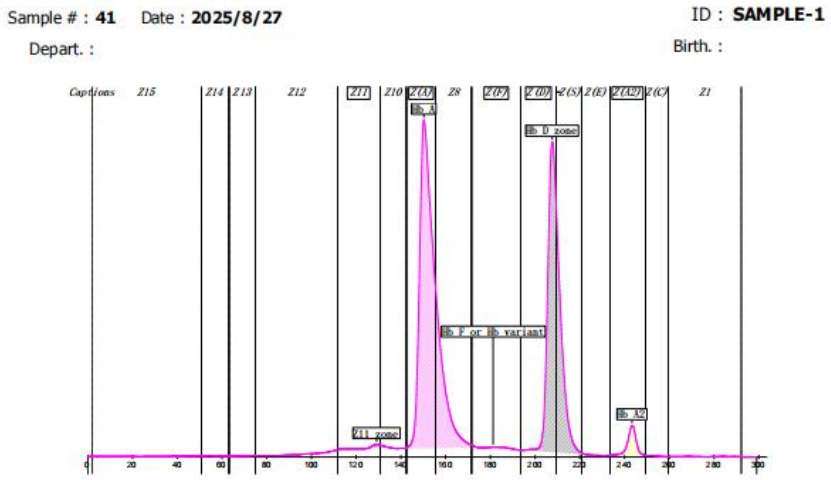


Haemoglobin Electrophoresis

Name	%	Normal Values %
Z11 zone	0.4	
Hb A	56.4	
Hb F or Hb variant	0.3	
Hb D zone	40.2	
Hb A2	2.7	

Figure 3.2.4.2 Hemoglobin Analysis Electropherogram of Special Sample No. 366 (Sebia Capillary Electrophoresis Method)

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.32% (reportable)**
Hb D RT	**253.6 s**
Hb D Area	**43.80%**
Peak Morphology	Discrete sharp peak post-HbA2; taller than HbA0; HbA1c unaffected
E5 Flag	Present
Confirmatory	Sebia CZE: Hb D zone 40.2%
Interpretation	● Chromatogram consistent with Hb D heterozygote. ● The Hb D peak height exceeding HbA0 is a consistent feature distinguishing the heterozygote from β-thalassemia trait.

Profile 3: Hemoglobin S (Hb S)

Parameter	Value
HGVS Notation	HBB: c.20A>T
Amino Acid Change	β6 Glu→Val (Glutamic acid→Valine)
Globin Chain	β-chain
H100 Recognition Window	Hb S window
H100 Retention Time	~264 – 266 s
Trait Hb S %	~43 – 48%
E5 Flag	Present
Geographic Prevalence	Sub-Saharan Africa (carrier rate up to 30%); Mediterranean, Middle East, India, diaspora populations

Clinical and Molecular Background

Hb S results from an A→T transversion at codon 6 of HBB, substituting valine for glutamic acid. Under deoxygenation, the hydrophobic valine residue drives polymerization of HbS tetramers into rigid fibers, distorting red cells into the sickle shape. Vaso-occlusion, hemolysis, and multi-organ ischemia follow.

Sickle cell trait (HbAS) is clinically benign in the vast majority of individuals. Sickle cell anemia (HbSS) is characterized by chronic hemolytic anemia, painful vaso-occlusive crises, acute chest syndrome, stroke, and progressive organ damage. HbSC disease is intermediate in severity.

In Chinese laboratories, Hb S is primarily encountered in patients of African or Middle Eastern background. The key diagnostic pitfall on the H100 is that Hb Ottawa (an α-chain variant of East Asian origin) co-elutes in the Hb S recognition window. Clinical context and CZE are essential before reporting Hb S in Asian patients.

3.3.1 IFCC Reference Material — Hb S Heterozygote, Example 1 (Lot 2022-0581)

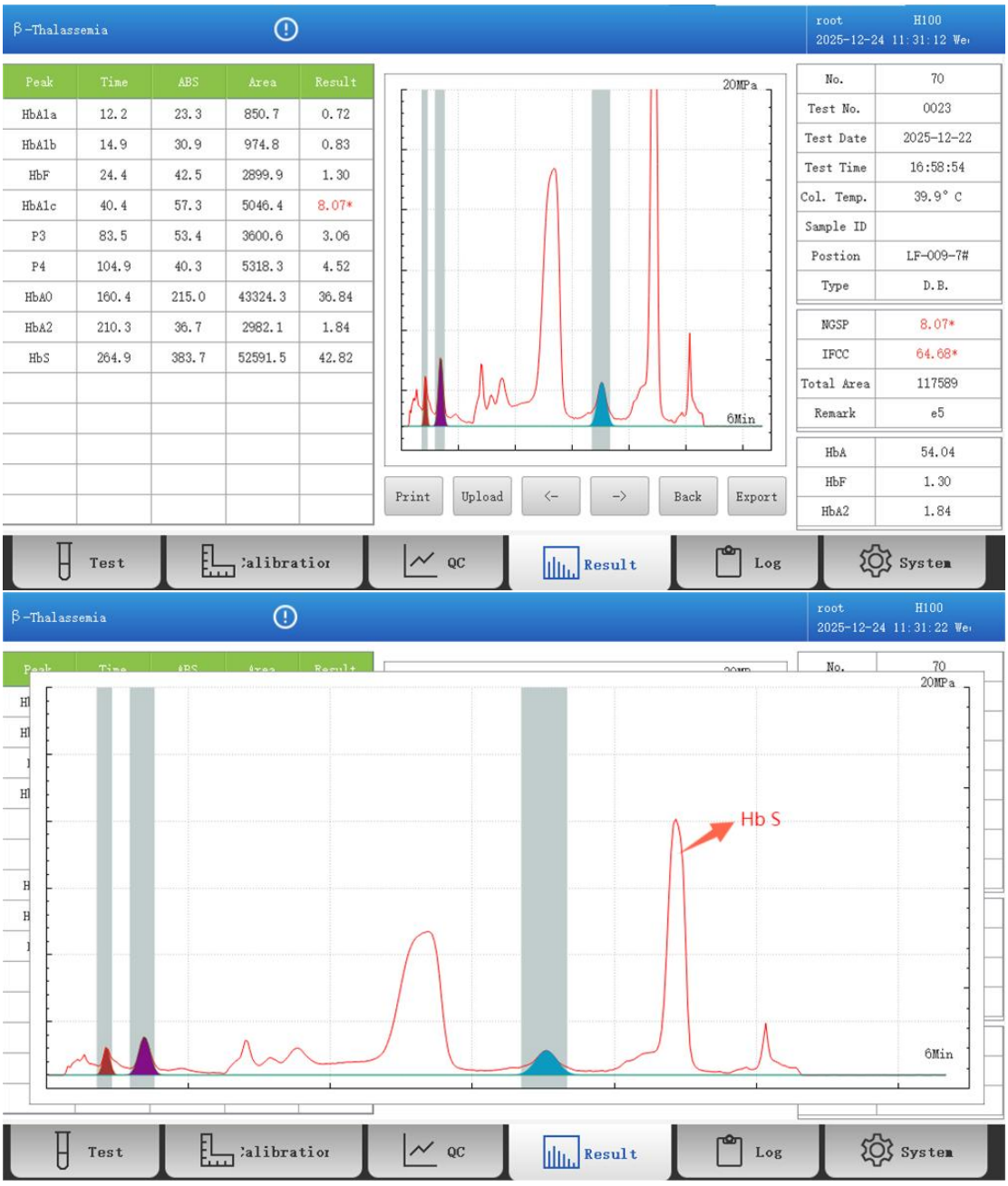
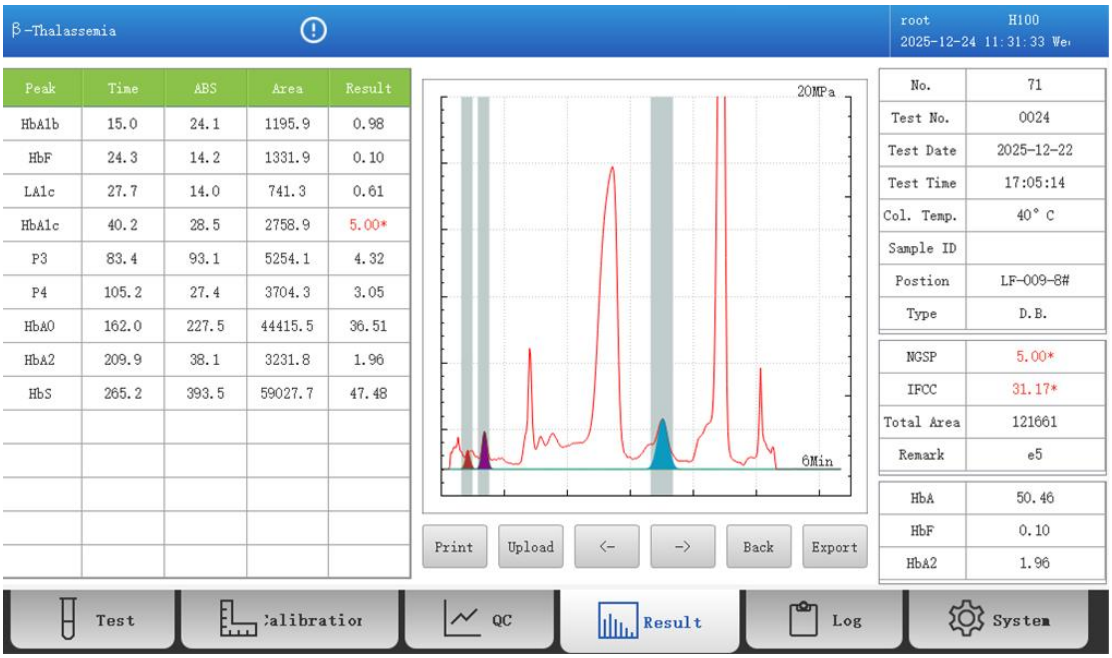


Figure 3.3.1 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2022-0581: Hb S heterozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**8.07% (reportable)**
Hb S RT	**264.9 s**
Hb S Area	**42.82%**
Peak Morphology	● Sharp, distinct peak post-HbA2; ● Clearly separated from HbA0 with visible valley between; ● HbA1c peak intact.
E5 Flag	Present
Interpretation	● Chromatogram consistent with Hb S heterozygosity (sickle cell trait). ● The post-HbA2 peak at ~265 s occupies approximately half the total area, typical of the heterozygous state.
⚠ CAUTION	● Hb Ottawa also elutes in the Hb S window. ● When the patient is of East or Southeast Asian ancestry, molecular confirmation is required before reporting 'Hb S trait'.

3.3.2 IFCC Reference Material — Hb S Heterozygote, Example 2 (Lot 2024-0597)



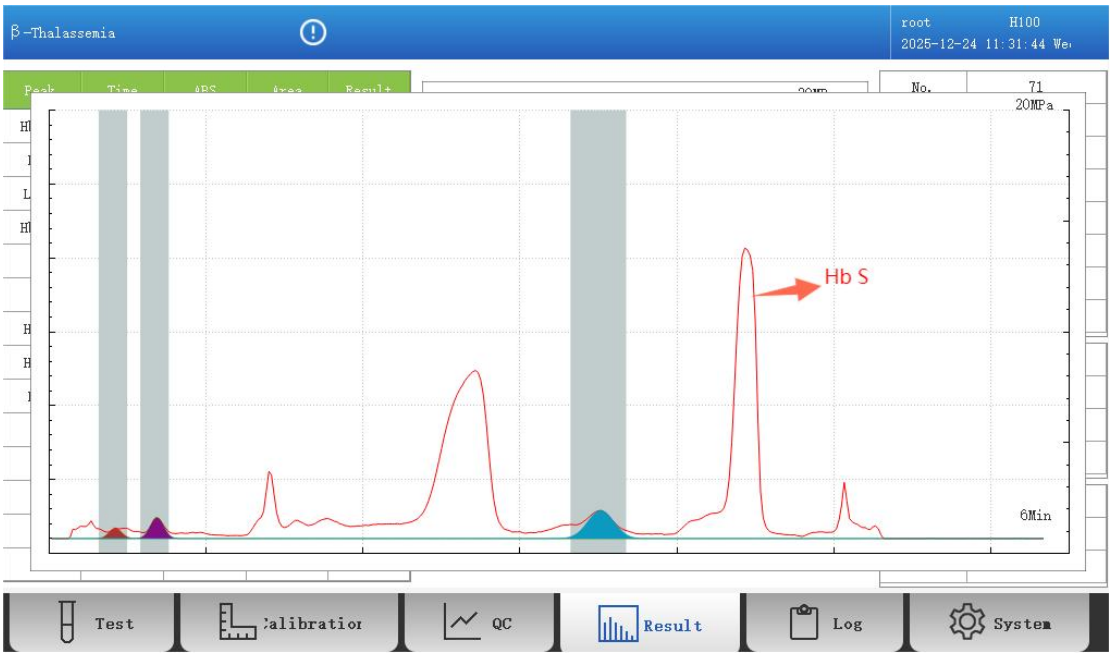


Figure 3.3.2 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2024-0597: Hb S heterozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.00%** (reportable)
Hb S RT	**265.2 s**
Hb S Area	**47.48%** (taller than HbA0 in this specimen)
Peak Morphology	Tall, sharp post-HbA2 peak; HbA0 clearly visible; HbA1c normal.
E5 Flag	Present
Instrument ID	Hb S (correct identification)
Interpretation	Second IFCC example confirming H100 reproducibly identifies Hb S trait across different reference material lots and glycaemia levels.

3.3.3 Clinical Specimen — Hb SC Compound Heterozygote (Case 359)

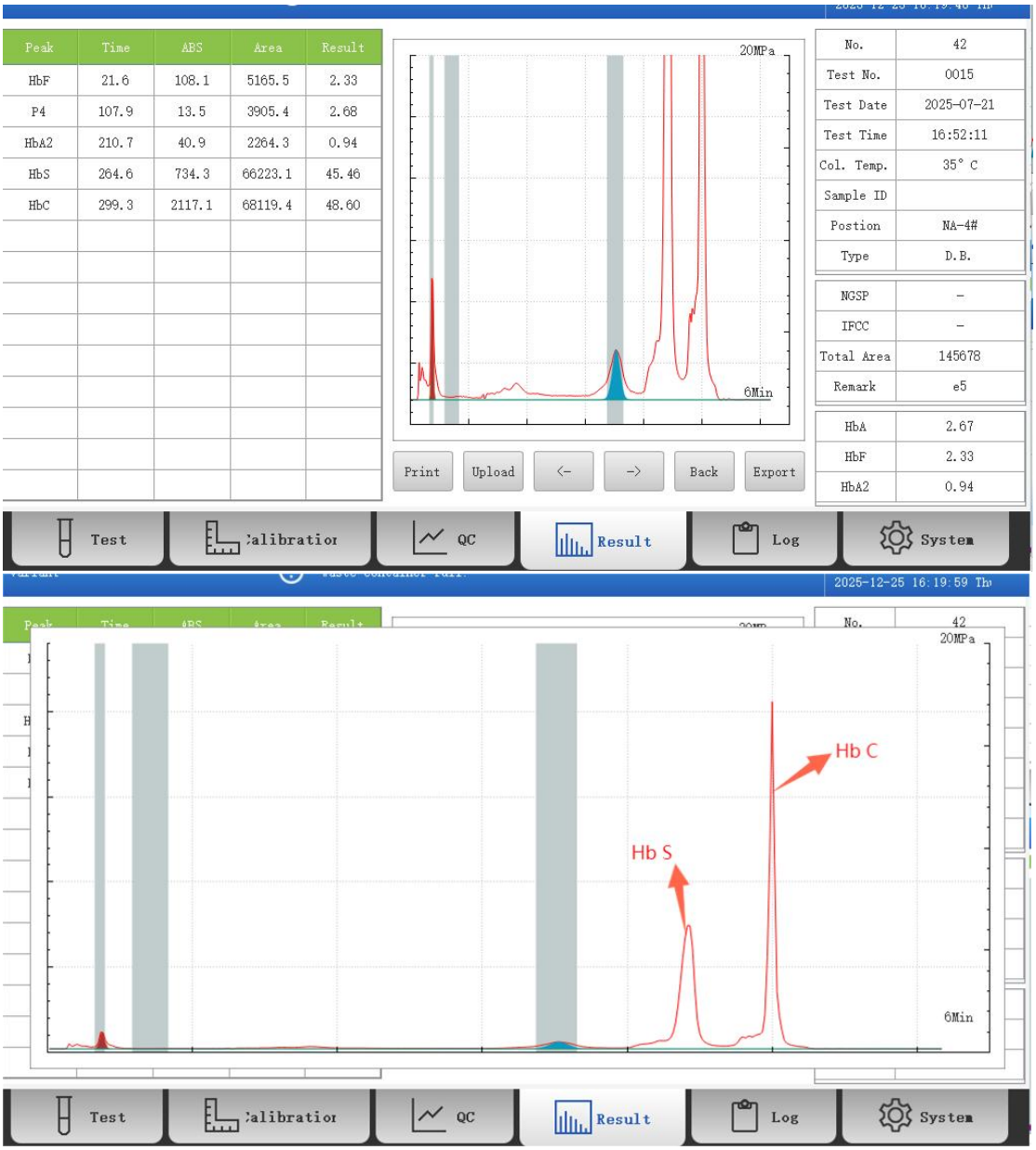
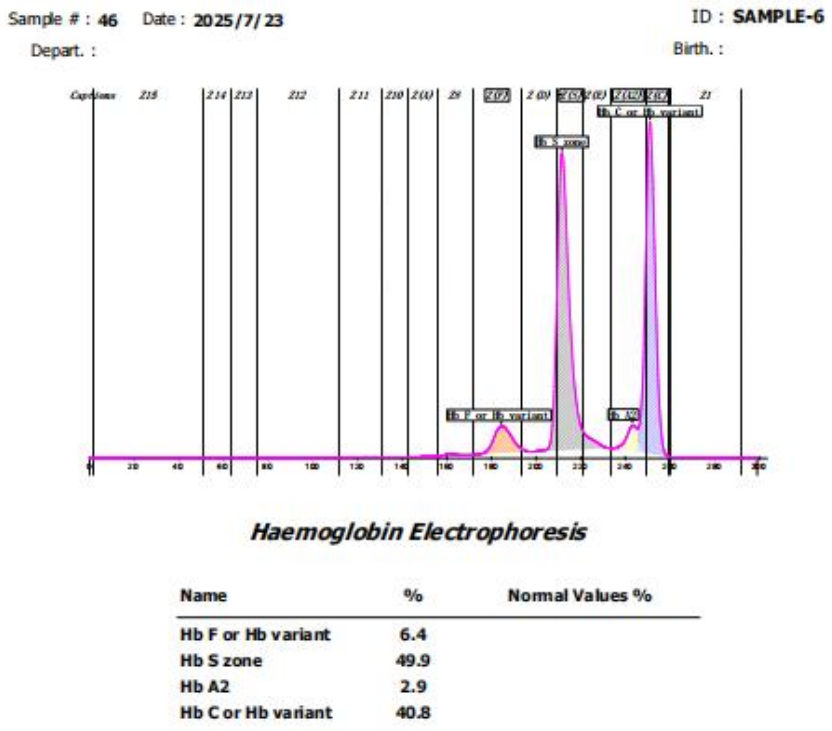


Figure 3.3.3.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case 359): Hb SC compound heterozygote

sebia



Name	%	Normal Values %
Hb F or Hb variant	6.4	
Hb S zone	49.9	
Hb A2	2.9	
Hb C or Hb variant	40.8	

Figure 3.3.3.2 Hemoglobin Analysis Electropherogram of Special Sample No. 359 (Sebia Capillary Electrophoresis)

CHROMATOGRAM INTERPRETATION	
HbA1c Result	No value
HbA0 Peak	Absent
Hb S RT	**264.6 s**
Hb S Area	**45.46%**
Hb C RT	**299.3 s**
Hb C Area	**48.00%**
Peak Morphology	• Two sharp sequential peaks post-HbA0 region: earlier peak = Hb S; later peak = Hb C. • No HbA0 or HbA1c peaks visible
E5 Flag	Present — both Hb S and Hb C flagged
Sebia CZE	Hb S zone 49.9%, Hb C zone 40.8% (confirms compound heterozygosity)

Interpretation	● The simultaneous appearance of two post-HbA2 peaks with absent HbA0 and HbA1c is characteristic of Hb SC disease. ● HbA1c cannot be measured; alternative glycaemic monitoring is required.
✓ CLINICAL TIP	● Hb SC disease requires clinical follow-up. ● Although milder than HbSS, patients can experience vaso-occlusive crises, proliferative retinopathy, and osteonecrosis.

Profile 4: Hemoglobin C (Hb C)

Parameter	Value
HGVS Notation	HBB: c.19G>A
Amino Acid Change	β6 Glu→Lys (Glutamic acid→Lysine)
Globin Chain	β-chain
H100 Recognition Window	Hb C window (~299 – 303 s)
H100 Retention Time	~299 – 303 s
Heterozygote Hb C %	~45 – 50%
Homozygote Hb C %	~95 – 100%
E5 Flag	Present
Geographic Prevalence	West and Central Africa; African diaspora in Caribbean, Americas, and Europe

Clinical and Molecular Background

Hb C results from a G→A transition at codon 6 of HBB, substituting the positively-charged lysine for glutamic acid. Unlike Hb S (same position, different substitution), Hb C does not polymerize; instead, intracellular crystallization in homozygous erythrocytes shortens red cell survival.

Hb C trait is benign. Hb C disease (HbCC) causes mild chronic hemolytic anemia with splenomegaly; transfusion is rarely required. HbSC disease (see Section 6.4.3) is the clinically important compound heterozygous state.

Xu et al. (Am J Clin Pathol, 2022) confirmed that on the H100-equivalent D-100 platform, Hb C is identified as a discrete, late-eluting peak at ~300 s, well resolved from HbA2, with negligible HbA1c interference in the heterozygote (relative bias −0.8‰ observed in our IFCC reference material testing).

3.4.1 IFCC Reference Material — Hb C Homozygote (Lot 2021-0195)

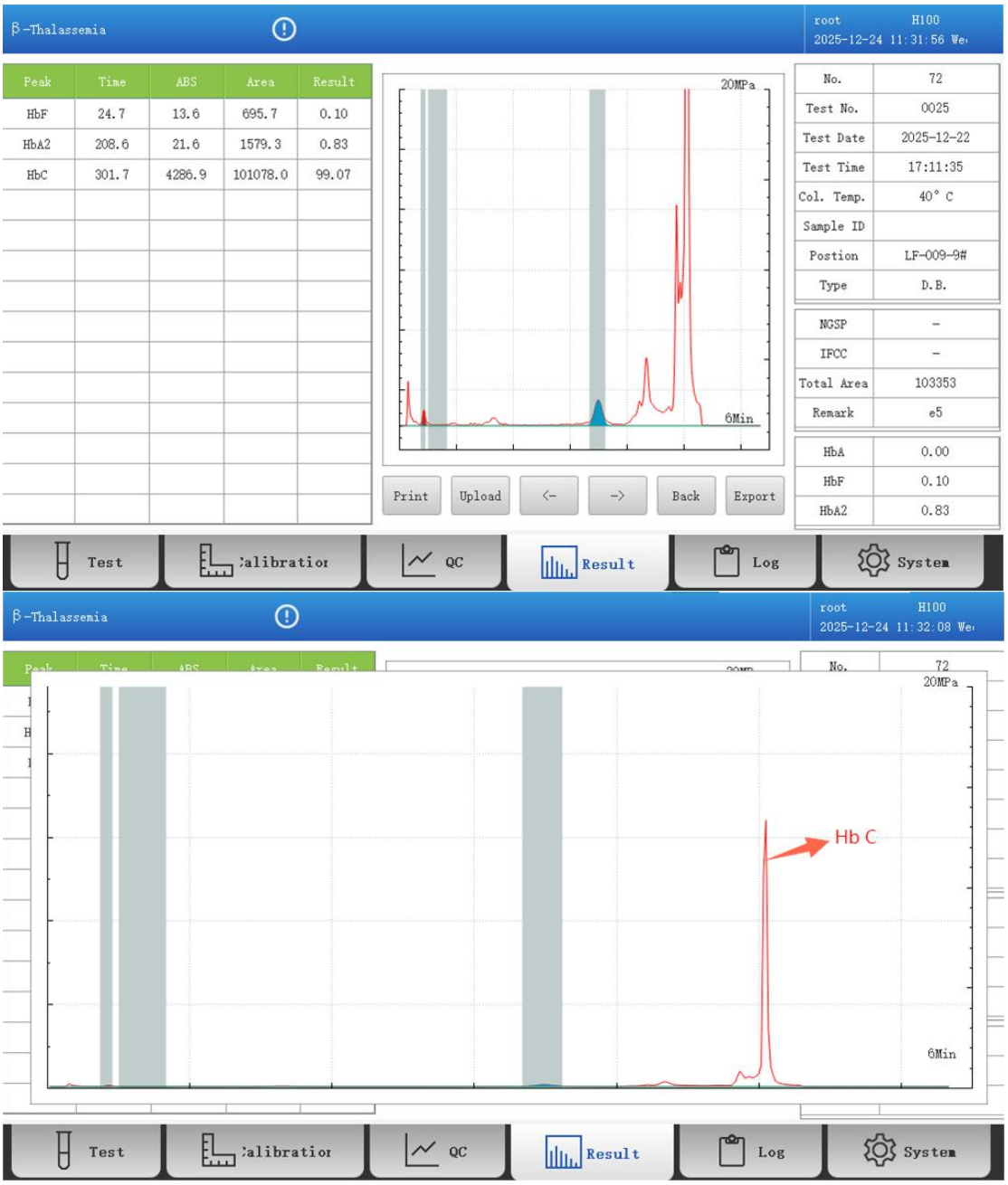
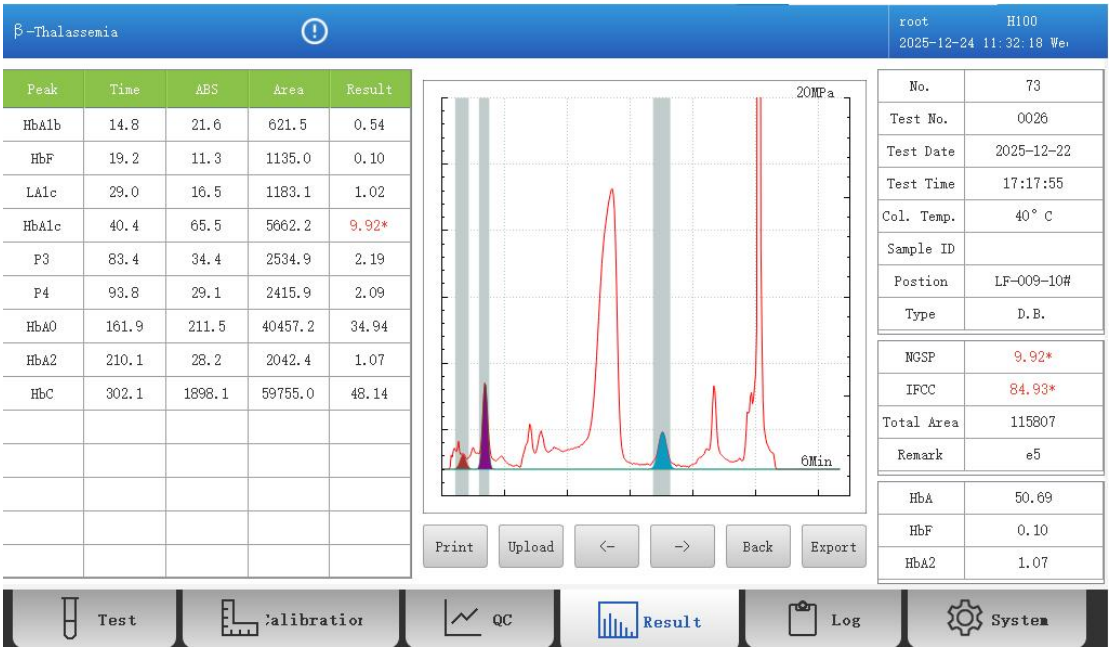


Figure 3.4.1 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2021-0195: Hb C homozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	No A1c value; Hb CC contains only Hb C and HbA_2.
Hb C RT	**301.7 s**
Hb C Area	**99.07%** (sole dominant peak)
Peak Morphology	● A single tall sharp peak far to the right of the HbA2 position; ● virtually the only chromatographic feature present.
E5 Flag	Present
Interpretation	● The 'single-peak' chromatogram with complete absence of HbA1c and HbA0 is pathognomonic of Hb C homozygosity. ● Note the very late elution (>300 s) compared with Hb E (~241 s) or Hb D (~253 s).
⚠ CAUTION	● HbA1c cannot be measured in Hb CC patients. ● Glycated albumin or fructosamine testing is required for glycaemic monitoring.

3.4.2 IFCC Reference Material — Hb C Heterozygote (Lot 2023-0088)



Peak	Time	ABS	Area	Result
HbA1b	14.8	21.6	621.5	0.54
HbF	19.2	11.3	1135.0	0.10
LA1c	29.0	16.5	1183.1	1.02
HbA1c	40.4	65.5	5662.2	9.92*
P3	83.4	34.4	2534.9	2.19
P4	93.8	29.1	2415.9	2.09
HbA0	161.9	211.5	40457.2	34.94
HbA2	210.1	28.2	2042.4	1.07
HbC	302.1	1898.1	59755.0	48.14

No.	73
Test No.	0026
Test Date	2025-12-22
Test Time	17:17:55
Col. Temp.	40° C
Sample ID	
Postion	LF-009-10#
Type	D.B.

NGSP	9.92*
IFCC	84.93*
Total Area	115807
Remark	e5

HbA	50.69
HbF	0.10
HbA2	1.07

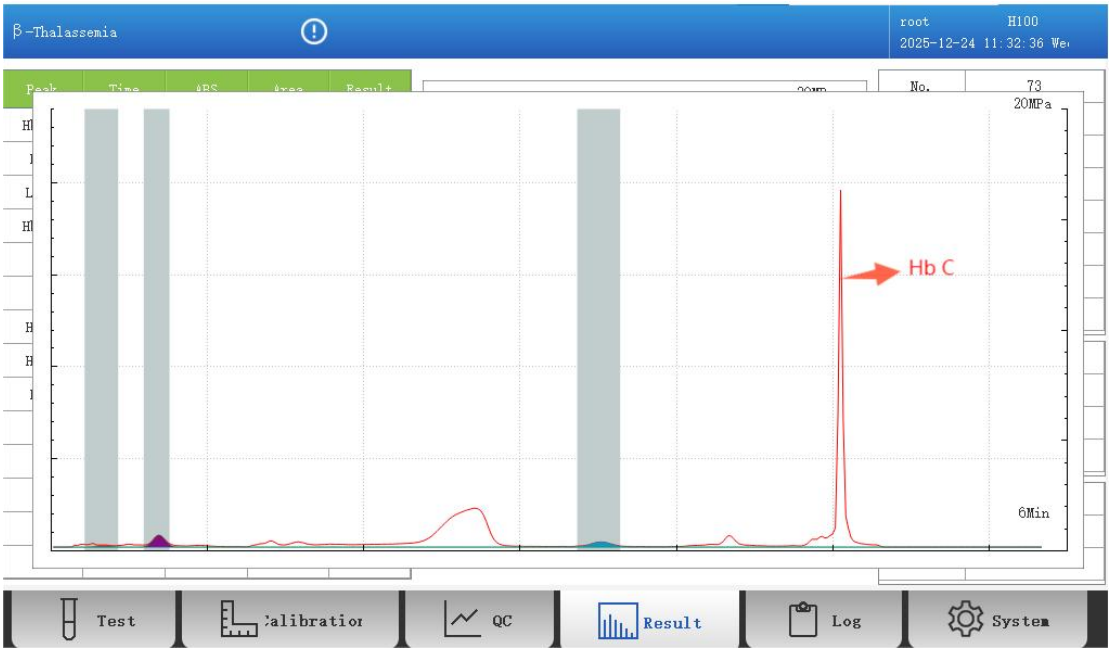


Figure 3.4.2 H100 (β-Thalassemia Mode) — IFCC Reference Material Lot 2023-0088: Hb C heterozygote

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**9.92%** (reportable; relative bias vs. target value = −0.8‰-- clinically insignificant)
Hb C RT	**302.1 s**
Hb C Area	**48.14%**
Peak Morphology	Sharp discrete peak eluting ~90 s after HbA2; HbA0 and HbA1c peaks morphologically normal.
E5 Flag	Present
Interpretation	● Chromatogram confirms Hb C heterozygosity. ● HbA1c is accurately reportable. ● The Hb C peak is well separated from all other major peaks, demonstrating excellent H100 resolution at this late retention time.
✓ CLINICAL TIP	● Hb C heterozygosity does NOT significantly interfere with HbA1c measurement on the H100. ● This is a clinically important advantage over platforms where Hb C co-elutes with HbA2.

3.4.3 Clinical Specimen — Hb SC Compound Heterozygote (Case 359) — Hb C Component

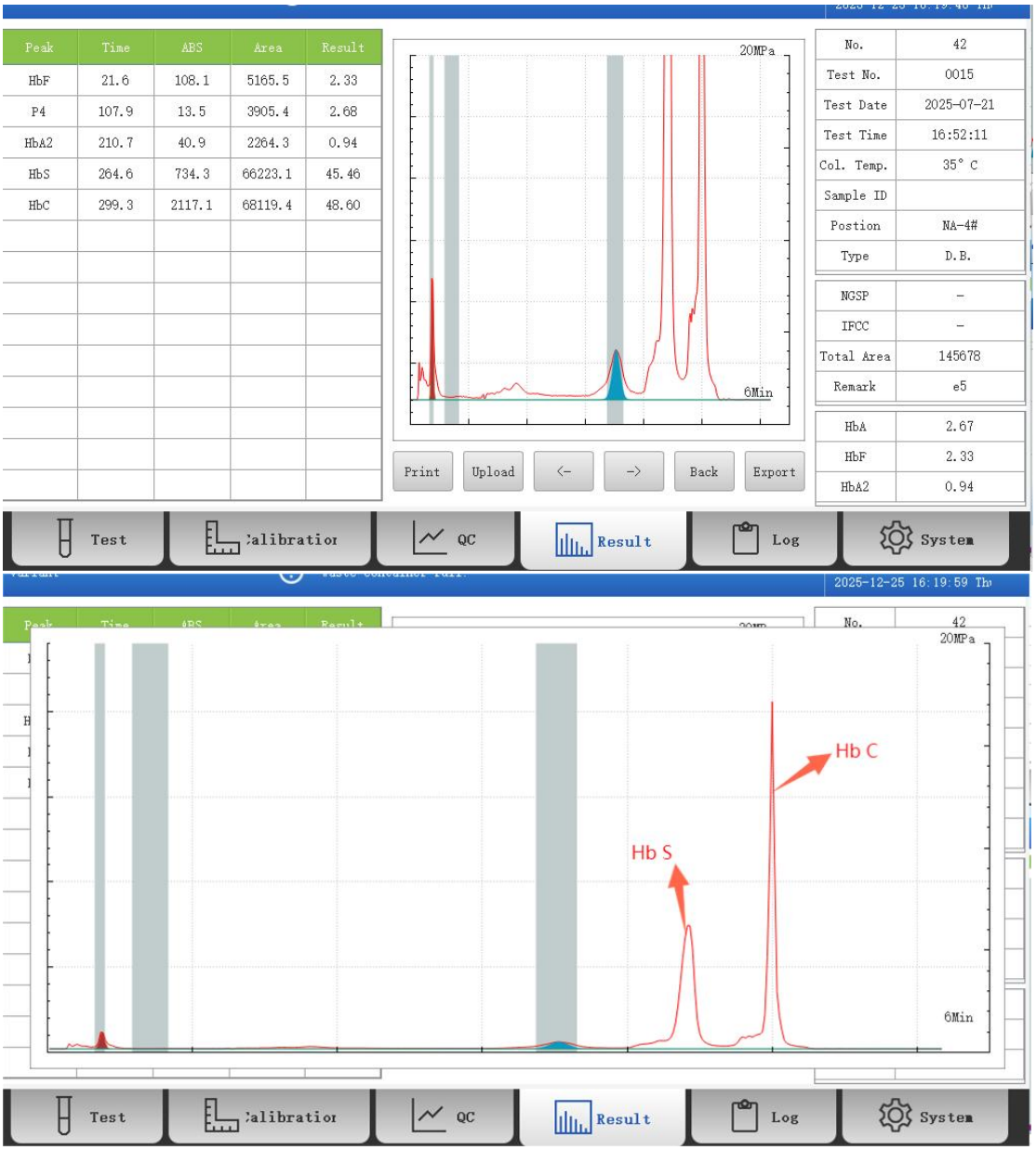


Figure 3.4.3.1 H100 (β-Thalassemia Mode) — See Figure 6.4 for full chromatogram of Case 359. The Hb C peak (RT ≈ 299.3 s, area 48.0%) is clearly resolved from the Hb S peak (RT ≈ 264.6 s).

sebia

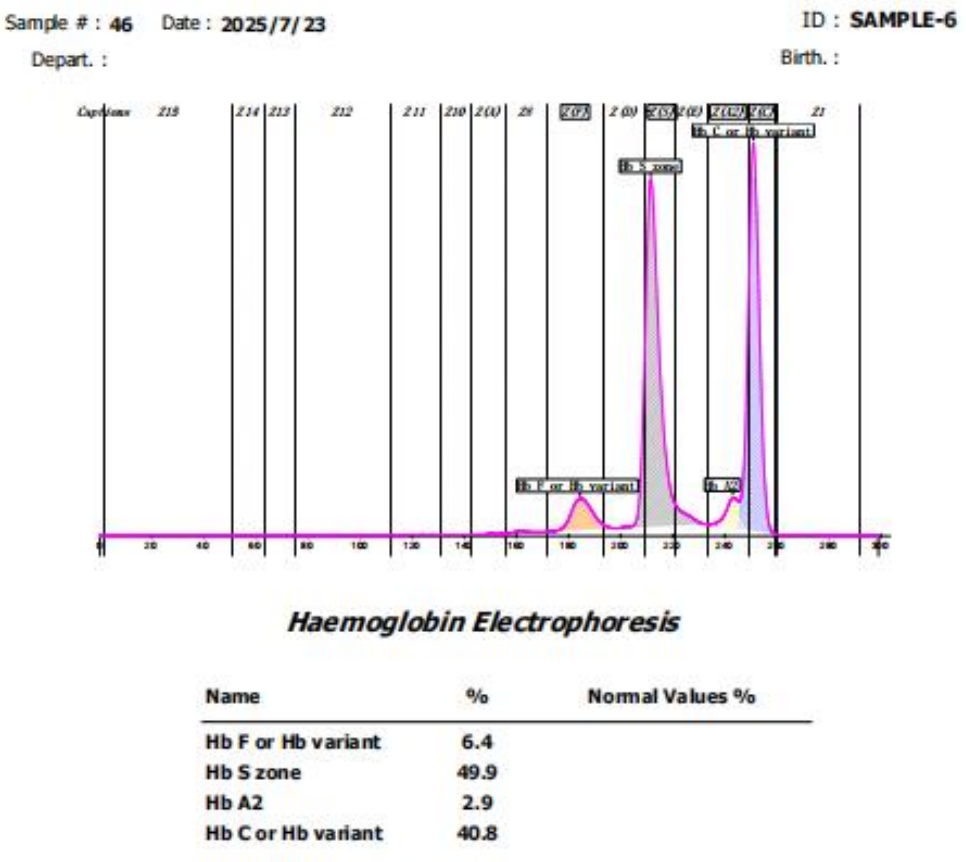


Figure 3.4.3.2 Hemoglobin Analysis Electropherogram of Special Sample No. 359 (Sebia Capillary Electrophoresis)

CHROMATOGRAM INTERPRETATION	
Hb C RT	**299.3 s**
Hb C Area	**48.00%**
Hb S RT	**264.6s**
Hb S Area	**45.46%** (co-existing variant; see Section 6)
HbA1c / HbA0	Both absent in this compound heterozygote
Interpretation	The late-eluting Hb C peak (>299 s) is clearly distinguishable from the earlier Hb S peak (~265 s), allowing simultaneous, unambiguous identification of both variants in a single H100 run.

Profile 5 Hb Q-Thailand

Parameter	Value
HGVS Notation	HBA1: c.223G>C
Amino Acid Change	α74 Asp→His (Aspartic acid→Histidine)
Globin Chain	α-chain (HBA1 gene)
H100 Display Label	Hb C ⚠ (co-elutes in Hb C window — NOT true Hb C)
H100 Retention Time	~288 – 292 s
Heterozygote %	~20 – 25%
E5 Flag	Present
Geographic Prevalence	Southern China (Fujian, Guangdong, Guangxi), Thailand, Japan; commonly linked in cis with the $-\alpha^{4.2}$ deletion

Clinical and Molecular Background

Hb Q-Thailand is characterized by an aspartate-to-histidine substitution at position 74 of the α1-globin chain. It is characteristically linked in cis to the leftward single α-gene deletion ($--\alpha^{4.2}$), making this allele an α^{+}-thalassemia determinant in addition to a structural variant. Unlinked heterozygotes are exceedingly rare; long-read SMRT sequencing was required to confirm the first cases in southern China (Hemoglobin, 2023).

When the Hb Q-Thailand allele (with linked $--\alpha^{4.2}$) is co-inherited with α^{0}-thalassemia (--SEA), the result is Hb Q-H disease, presenting as moderate-to-severe anemia resembling Hb H disease. A 2024 survey in southern Thailand (Tepakhan et al., Sci Rep) identified Hb Q-Thailand at 0.6% prevalence among 13,391 participants.

On the H100, Hb Q-Thailand elutes at ~288–292 s within the Hb C recognition window. The instrument displays “Hb C”-- a misleading label that has no clinical basis, since Hb Q-Thailand is an α-chain variant with entirely different inheritance implications. Molecular confirmation is mandatory.

3.5.1 Clinical Specimen — Hb Q-Thailand (Case RT17)

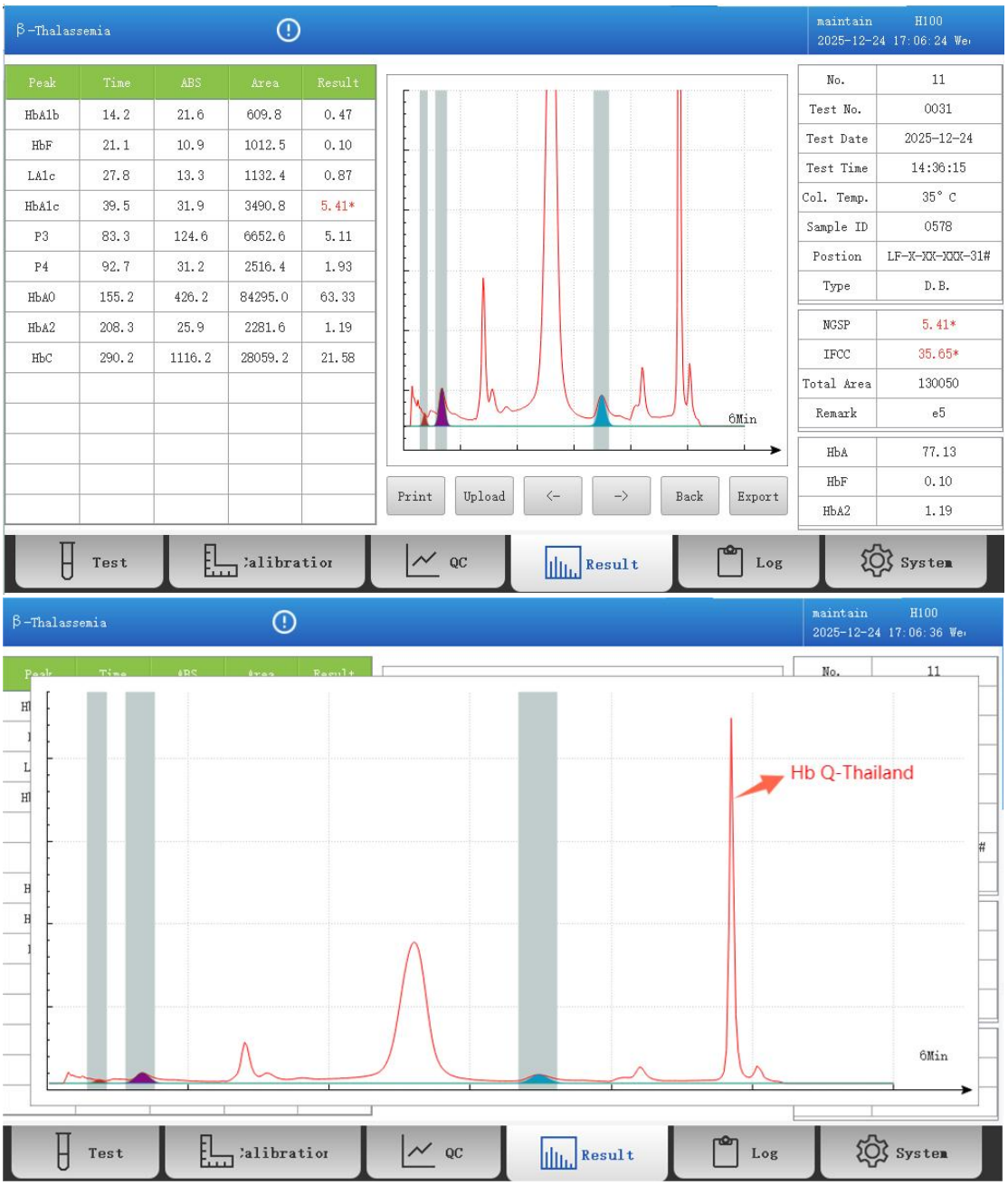


Figure 3.5.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case RT17): Hb Q-Thailand confirmed by Sanger sequencing (Peking University Hospital).

sebia

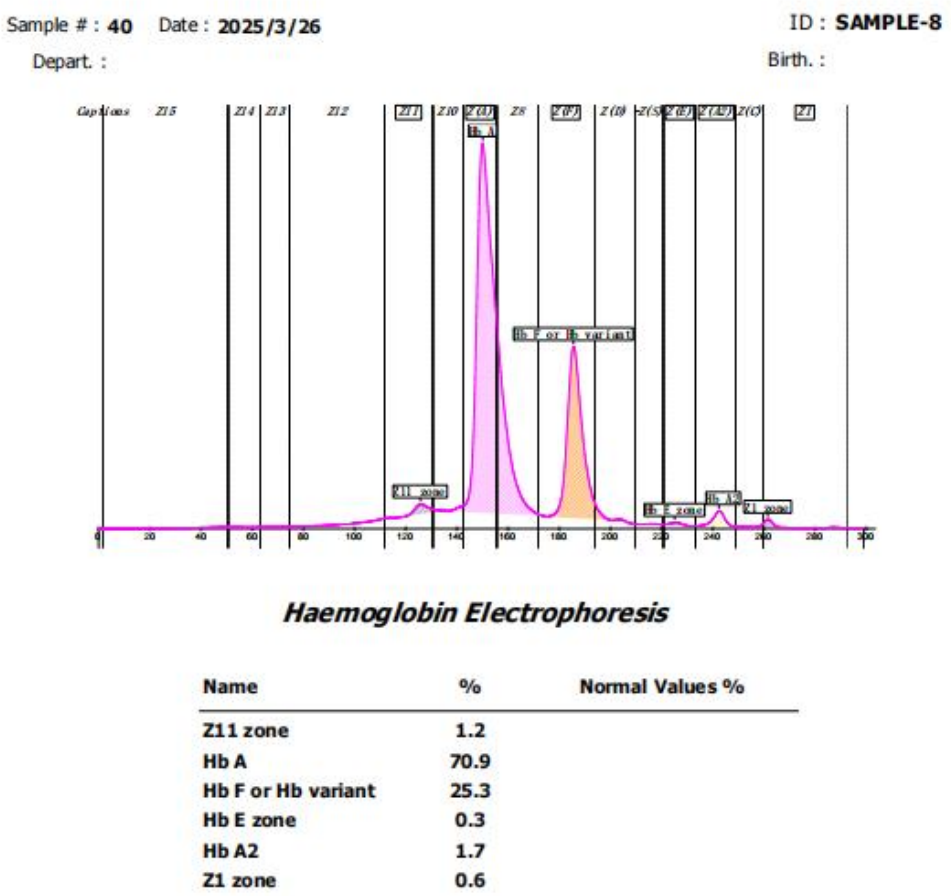


Figure 3.5.1.2 RT No. 17 Special Sample: Hemoglobin Analysis Electropherogram via Sebia Capillary Electrophoresis.

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.41%** (reportable — α-chain variant does not affect HbA1c measurement)
H100 Display	"Hb C" ⚠ (due to co-elution)
Variant RT	**290.2 s** (Note: ~10 s earlier than true Hb C at ~300 s-- a useful differentiating feature)
Variant Area	**21.58%** (notably lower than typical Hb C heterozygote of ~48%; this lower % should raise suspicion)
Peak Morphology	Sharp discrete post-HbA2 peak; elutes ~10 s before the Hb C position.
E5 Flag	Present
Confirmatory	Sanger sequencing: HBA1 c.223G>C (Hb Q-Thailand). Sebia CZE: complex

	multi-zone pattern confirming α-chain variant.
Interpretation	• Retention time ~10 s shorter than true Hb C, combined with a much lower area percentage (~22% vs. expected ~48% for Hb C heterozygote), are key clues that the variant is NOT Hb C. • Hb Q-Thailand must be confirmed molecularly.
⚠ CAUTION	**When "Hb C" is reported on H100 in a patient of Chinese or Thai ancestry with a percentage <35%, suspect Hb Q-Thailand and proceed to confirmatory sequencing before reporting.**

Profile 6 Hb G-Honolulu

Parameter	Value
HGVS Notation	HBA1: c.91G>C or HBA2: c.91G>C
Amino Acid Change	α30 Glu→Gln (Glutamic acid→Glutamine)
Globin Chain	α-chain (HBA1 or HBA2)
H100 Display Label	Hb E ⚠ (co-elutes in Hb E window-- NOT true Hb E)
H100 Retention Time	~242 – 246 s
Heterozygote %	~25 – 30%
E5 Flag	Present
Geographic Prevalence	First described in Singapore; common in southern China (Guangdong, Guangxi)

Clinical and Molecular Background

Hb G-Honolulu is an α-chain structural variant in which glutamic acid at position 30 is replaced by glutamine. The mutation can occur in HBA1 or HBA2. Heterozygotes are asymptomatic with normal blood counts.

Xu et al. (Am J Clin Pathol, 2022) identified Hb G-Honolulu as the sixth most common Hb variant in China, identified in samples submitted for routine HbA1c testing. Importantly, the Tosoh HLC-723 G8 yields no HbA1c result for Hb G-Honolulu due to co-elution; the H100 correctly reports HbA1c in these patients. A 2023 case report documented the first co-inheritance of Hb G-Honolulu (HBA2:c.91G>A) with Hb S in an Italian child, with a benign phenotype.

On the H100, Hb G-Honolulu elutes at RT ~242–246 s, overlapping the Hb E recognition window. The instrument reports 'Hb E', which is clinically incorrect — Hb G-Honolulu is an α-chain variant with entirely different clinical and genetic implications from the β-chain Hb E.

3.6.1 Clinical Specimen — Hb G-Honolulu (Case GH3)

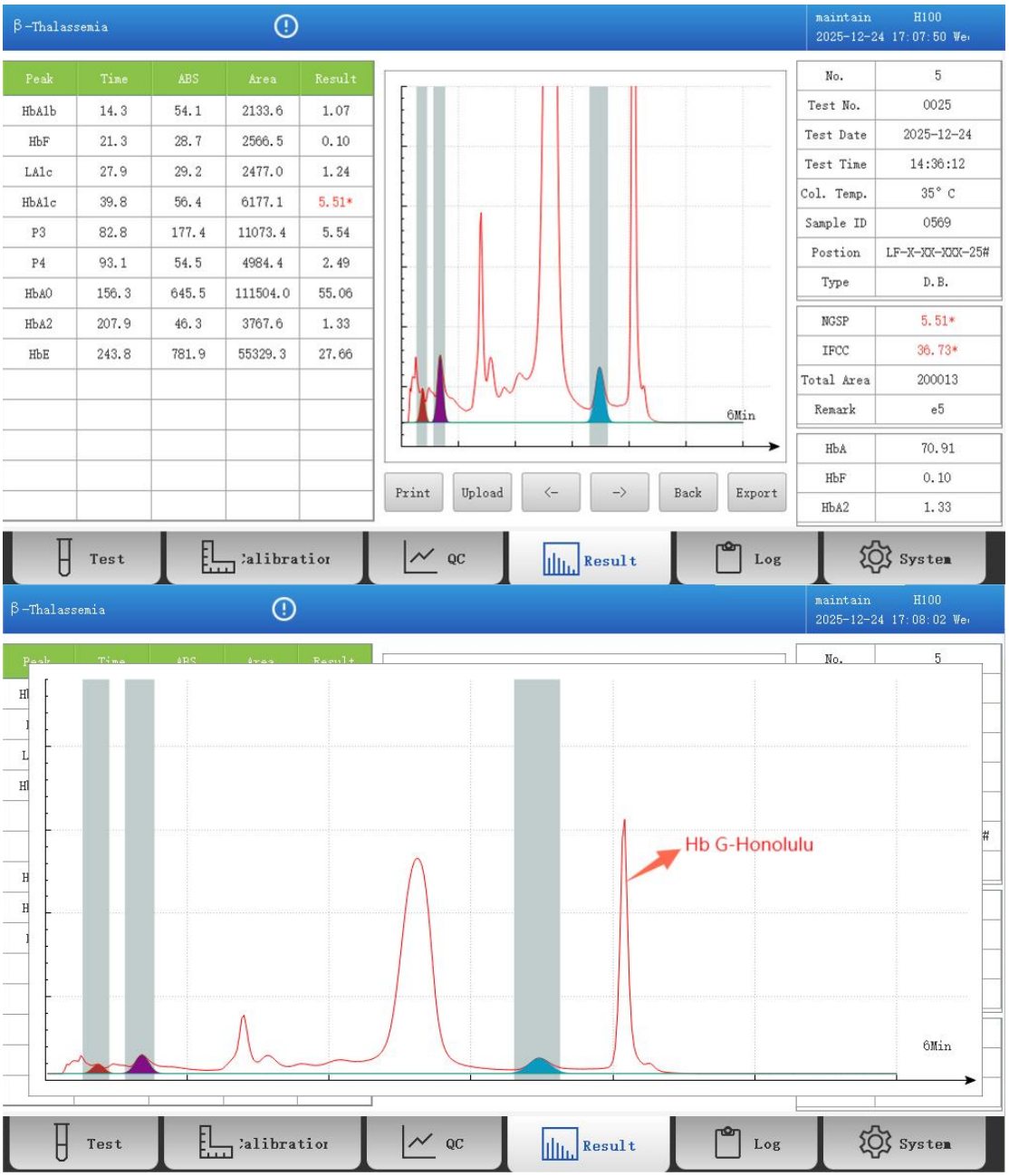
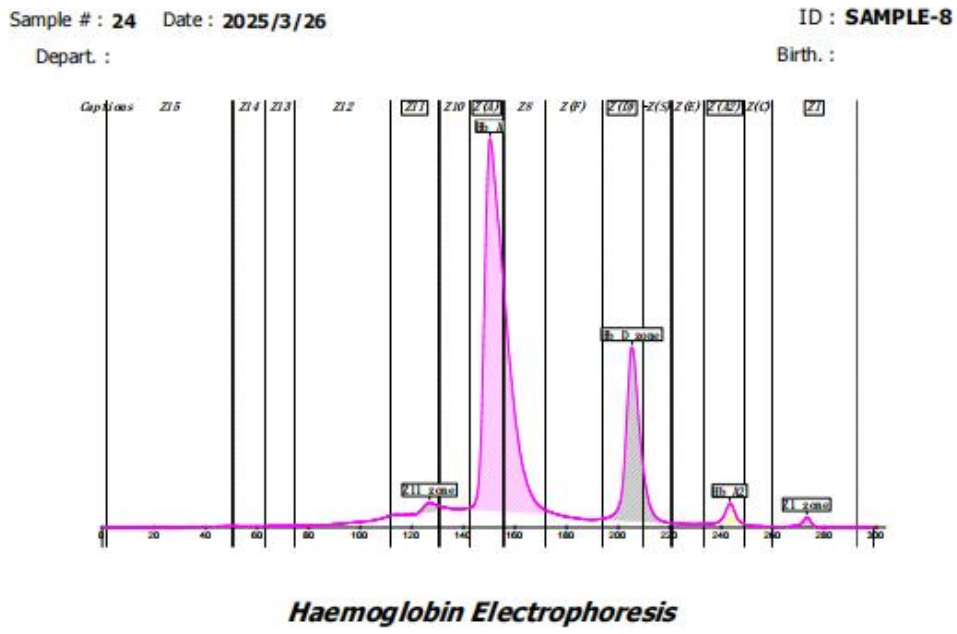


Figure 3.6.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case GH3): Hb G-Honolulu confirmed by Sanger sequencing (Peking University Hospital).

sebia



Name	%	Normal Values %
Z11 zone	1.3	
Hb A	73.3	
Hb D zone	22.4	
Hb A2	2.3	
Z1 zone	0.7	

Figure 3.6.1.2 Hemoglobin Analysis Electropherogram of Special Sample GH3 (Sebia Capillary Electrophoresis Method)

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.51%** (reportable -- α-chain variant; HbA1c unaffected)
Variant RT	**243.8 s** (~2–4 s later than Hb G-Coushatta and within the Hb E window; compare carefully with RT of true Hb E at ~241 s)
Variant Area	**27.66%** (lower than true Hb E heterozygote ~26–30% --overlap makes RT comparison critical)
Peak Morphology	Sharp narrow peak eluting just after HbA2; very similar in appearance to Hb E heterozygote.
E5 Flag	Present
Confirmatory	Sanger sequencing: HBA1/2 c.91G>C (Hb G-Honolulu). Sebia CZE: peaks in zones 1, 11, and Hb D zone — a CZE pattern distinct from Hb

Interpretation	E. ● The Hb E window RT overlap makes H100 discrimination of Hb G-Honolulu from true Hb E impossible on retention time alone. ● The CZE pattern (Hb D zone migration vs. Hb E zone migration for true Hb E) is the key discriminating test before molecular confirmation.
⚠ CAUTION	**CZE is the recommended first-line confirmatory step when 'Hb E' is reported in a patient without typical Southeast Asian risk factors, or when the percentage or RT pattern is atypical.**

Profile 7 Hb G-Coushatta

Parameter	Value
HGVS Notation	HBB: c.68A>C
Amino Acid Change	β22 Glu→Ala (Glutamic acid→Alanine)
Globin Chain	β-chain
H100 Display Label	Hb E ⚠ (elutes in Hb E window at earlier RT)
H100 Retention Time	~228 – 233 s (Note: ~8–12 s EARLIER than Hb E)
Heterozygote %	~43 – 48%
E5 Flag	Present
Geographic Prevalence	China (Silk Road / northern regions), Thailand, Egypt, Japan, Korea, Turkey, North America

Clinical and Molecular Background

Hb G-Coushatta results from an A→C transversion at codon 22 of HBB, substituting the neutral alanine for glutamic acid at an external surface residue. Neither sickling nor significant instability results. Both heterozygotes and the rare homozygotes have at most mild anemia.

Xu et al. (Am J Clin Pathol, 2022) ranked Hb G-Coushatta as the fifth most common Hb variant in China, identified in 32 samples over a 5-year period of HbA1c monitoring. Yang et al. (Scand J Clin Lab Invest, 2023) confirmed that Tosoh HLC-723 G8 produces a significant negative bias for HbA1c in Hb G-Coushatta specimens due to partial co-elution of the variant peak with the HbA1c window; the H100 correctly identifies and separates Hb G-Coushatta.

The key differentiating feature from true Hb E on the H100 is the earlier retention time (~228–233 s vs. ~240–242 s for Hb E) and a higher variant percentage (~44–48% vs. ~25–30% for Hb E heterozygote). These two observations together strongly suggest Hb G-Coushatta over Hb E.

3.7.1 Clinical Specimen — Hb G-Coushatta (Case G1008)

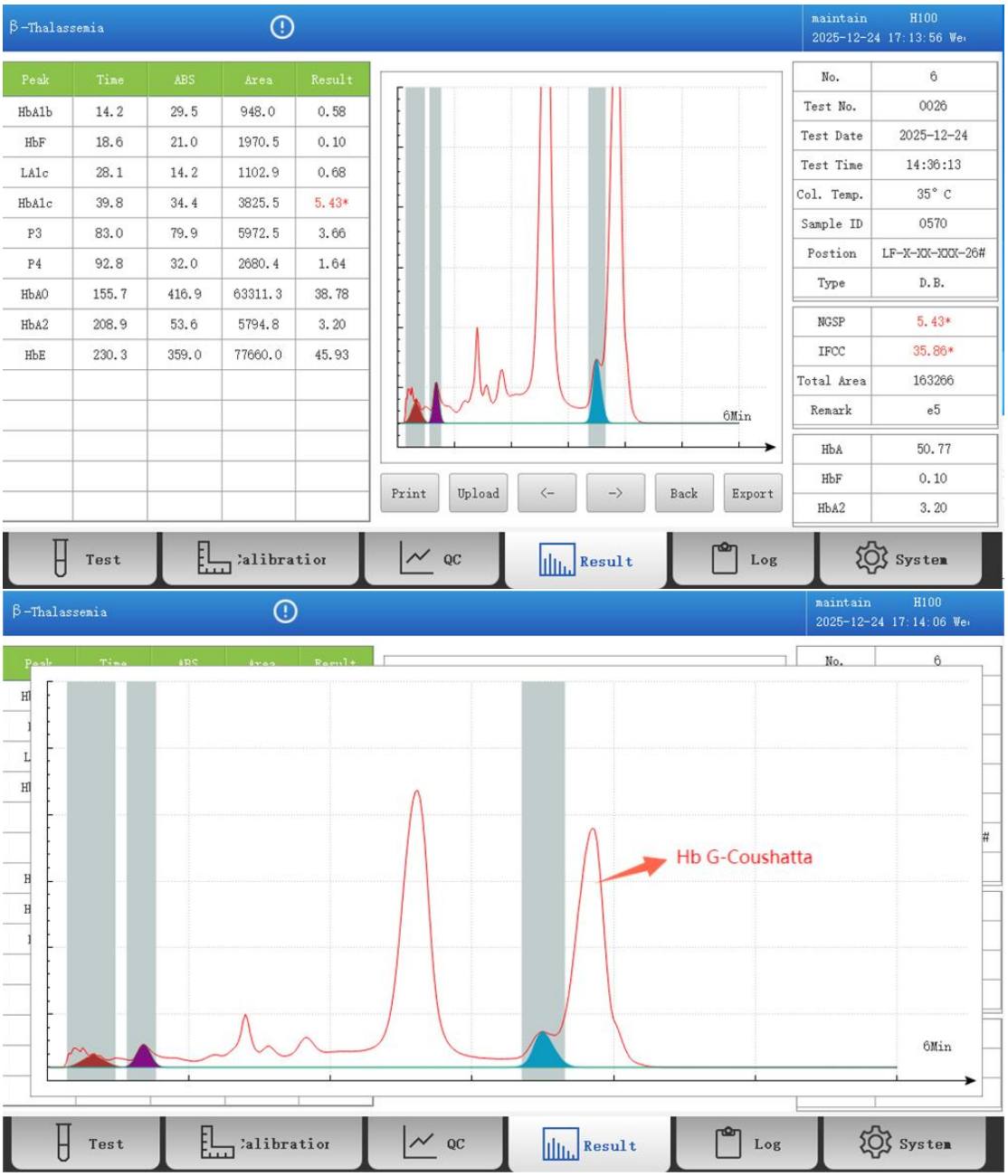
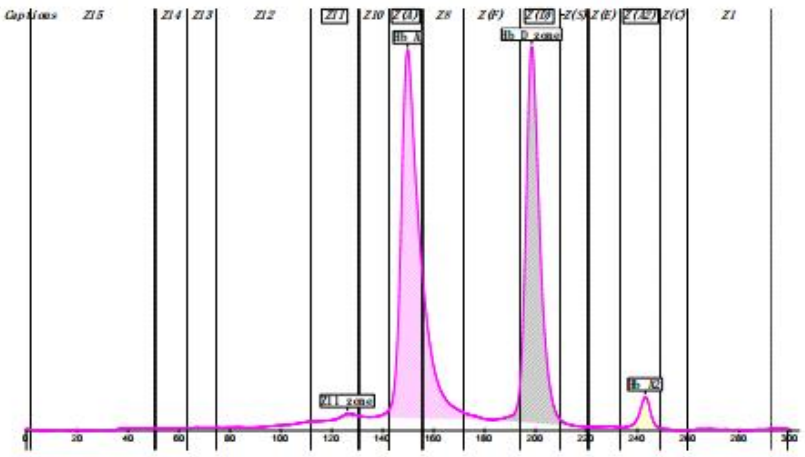


Figure 3.7.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case G1008): Hb G-Coushatta confirmed by Sanger sequencing (Peking University Hospital).

sebia

Sample # : 28 Date : 2025/3/26 ID : SAMPLE-4
Depart. : Birth. :



Haemoglobin Electrophoresis

Name	%	Normal Values %
Z11 zone	0.4	
Hb A	55.4	
Hb D zone	41.5	
Hb A2	2.7	

Figure 3.7.1.2 Hemoglobin Analysis Electropherogram of Special Sample G1008 via Sebia Capillary Electrophoresis

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.43%** (reportable)
H100 Display	'Hb E' ⚠ (not true Hb E)
Variant RT	**230.3 s** ← KEY CLUE: significantly earlier than true Hb E (~241 s); difference of ~11 s
Variant Area	**45.93%** ← KEY CLUE: much higher than expected Hb E heterozygote (~26%); closer to 50%, suggesting β-chain dominance.
Peak Morphology	• Sharp post-HbA2 peak; • elution well before the true Hb E position; • HbA0 and HbA1c peaks intact.
E5 Flag	Present
Confirmatory	Sanger sequencing: HBB c.68A>C (Hb G-Coushatta).

Interpretation	Sebia CZE: zones 11 + Hb D zone (distinct from Hb E zone migration). ● The combination of earlier RT (~230 s) and higher percentage (~46%) compared with expected Hb E heterozygote values should alert the operator to investigate further. ● Sebia CZE and molecular analysis confirmed Hb G-Coushatta.
⚠ CAUTION	**Practical rule: if the 'Hb E' peak RT is <236 s AND the percentage is >38%, suspect Hb G-Coushatta and order confirmatory CZE/sequencing.**

Profile 8 Hb G-Taipei

Parameter	Value
HGVS Notation	HBB: c.68A>G
Amino Acid Change	β22 Glu→Gly (Glutamic acid→Glycine)
Globin Chain	β-chain
H100 Display Label	Hb E ⚠ (co-elutes in Hb E window)
H100 Retention Time	~238 – 242 s (overlaps directly with Hb E)
Heterozygote %	~40 – 44%
E5 Flag	Present
Geographic Prevalence	Predominantly Chinese; both northern and southern China, especially Silk Road region

Clinical and Molecular Background

Hb G-Taipei results from an A→G transition at codon 22 of HBB, replacing glutamic acid with the smallest amino acid, glycine. Both heterozygotes and the rare homozygotes are clinically and hematologically normal.

Hb G-Taipei is one of the most common Hb variants in China. It causes significant HbA1c interference on Tosoh HLC-723 G8 and Arkray HA-8180V (negative bias), but is correctly handled by the H100 which separates it from the HbA1c peak. Ye et al. (Sichuan Da Xue Xue Bao Yi Xue Ban, 2023) reviewed multiple cases of Hb G-Taipei causing diagnostic confusion. Li et al. (Pract Lab Med, 2024) described compound heterozygosity of Hb G-Taipei with Hb Lepore-Boston-Washington discovered incidentally during HbA1c testing.

On the H100, the Hb G-Taipei peak elutes at RT ~238–242 s, almost directly overlapping with true Hb E (~240–242 s), making retention-time discrimination extremely difficult. The higher percentage (~40–44% vs. ~25–30% for Hb E) is the most reliable H100 clue.

3.8.1 Clinical Specimen — Hb G-Taipei (Case G1006)

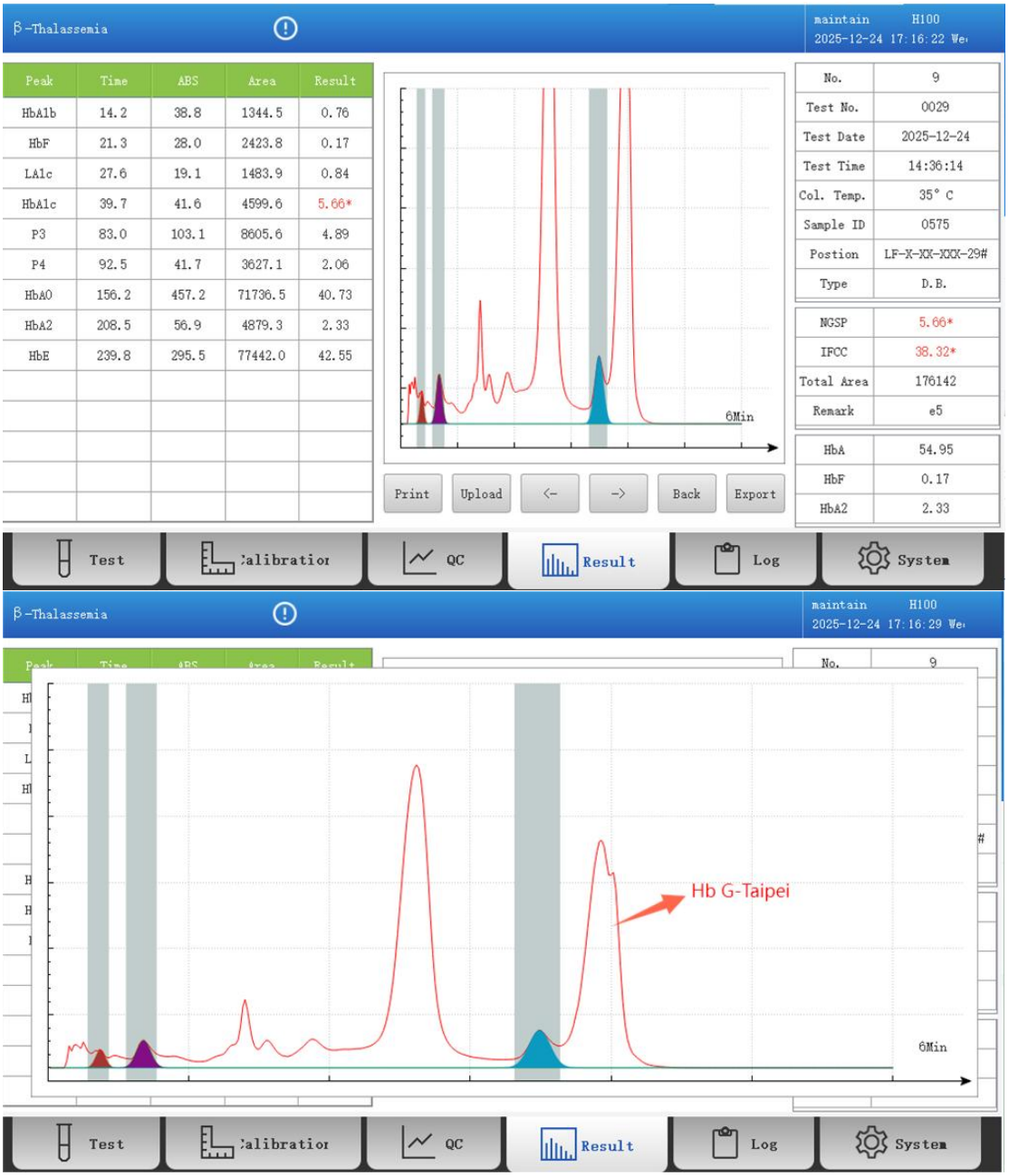


Figure 3.8.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case G1006): Hb G-Taipei confirmed by Sanger sequencing (Peking University Hospital).

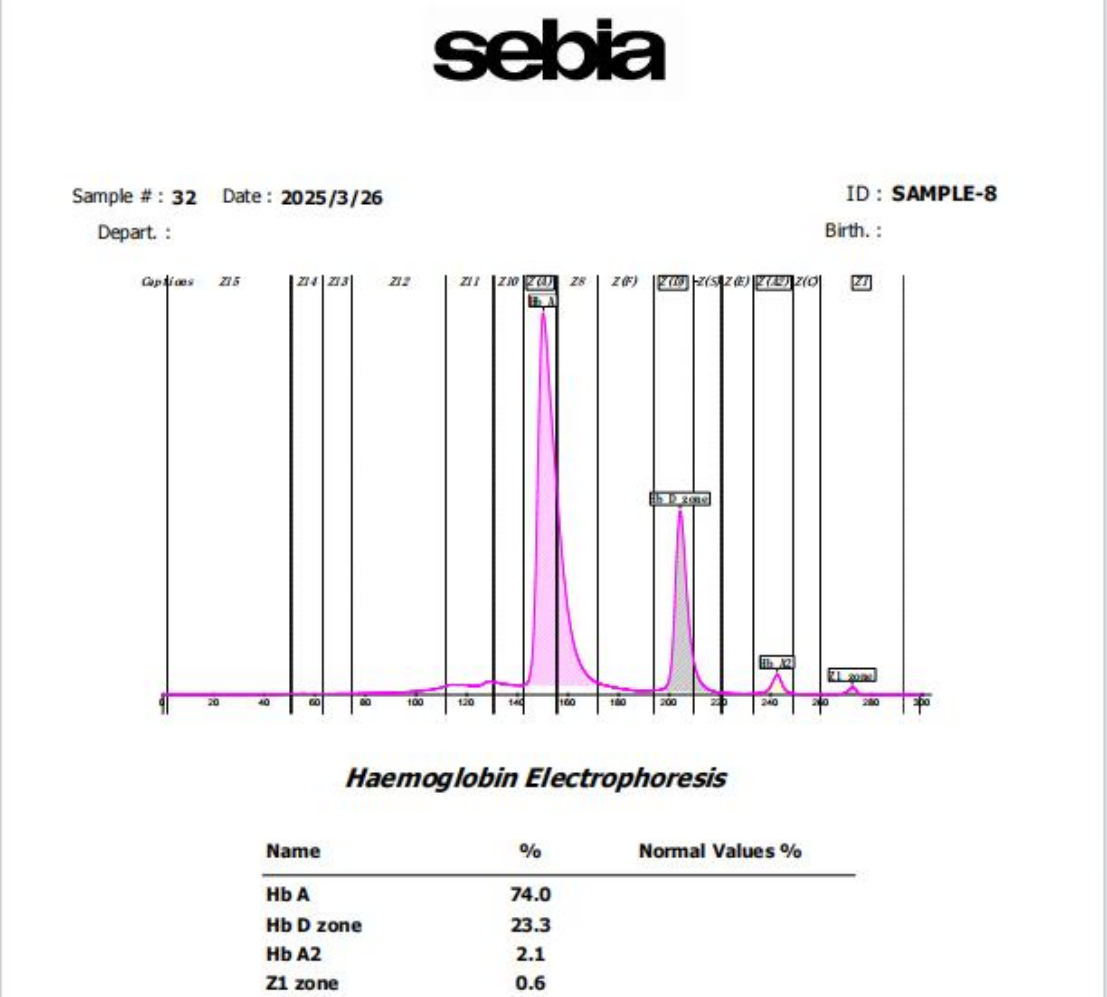


Figure 3.8.1.2 Hemoglobin Analysis Electropherogram of Special Sample G1006 using Sebia Capillary Electrophoresis

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.66%** (reportable)
H100 Display	'Hb E' ⚠ (not true Hb E)
Variant RT	**239.8 s** (virtually identical to Hb E RT; RT alone cannot discriminate)
Variant Area	**42.55%** ← KEY CLUE: substantially higher than typical Hb E heterozygote (~26%); approaching 50% expected for a β-chain dominant variant
Peak Morphology	Broader peak profile compared with the typically sharp Hb E peak; slight right-shoulder may be visible on the expanded view.
E5 Flag	Present
Confirmatory	Sanger sequencing: HBB c.68A>G (Hb G-Taipei). Sebia CZE: peaks in zone 1 and Hb D zone (Hb G-Taipei migrates to the

Interpretation	Hb D zone, not the Hb E zone — the most important CZE discriminator). ● RT is indistinguishable from true Hb E on the H100. ● The elevated percentage (~43%) is the primary H100 clue. ● Sebia CZE migration to the Hb D zone (not Hb E zone) unambiguously differentiates Hb G-Taipei from Hb E and is recommended as the first confirmatory step.
⚠ CAUTION	**Practical rule: any 'Hb E' result >36% on H100 in a patient with northern Chinese ancestry warrants immediate CZE to exclude Hb G-Taipei or Hb G-Coushatta.**

Profile 9 Hb Ottawa

Parameter	Value
HGVS Notation	HBA1 or HBA2: c.46G>C
Amino Acid Change	α15 Gly→Arg (Glycine→Arginine)
Globin Chain	α-chain (HBA1 or HBA2)
H100 Display Label	Hb S ⚠ (co-elutes in Hb S window -- NOT true Hb S)
H100 Retention Time	~268 – 272 s (~5 s later than true Hb S)
Heterozygote %	~22 – 27%
E5 Flag	Present
Geographic Prevalence	Southern China (Guangdong, Hainan), Thailand, Canada, New Zealand; isolated reports in India and France

Clinical and Molecular Background

Hb Ottawa (also called Hb Siam) arises from a G→C transversion at codon 15 (or 16 by older counting) of either HBA1 or HBA2, substituting the large, positively-charged arginine for the small neutral glycine. Heterozygous Hb Ottawa carriers are hematologically normal. When co-inherited with α- or β-thalassemia, mild microcytic indices may appear.

The most clinically alarming scenario is co-inheritance with Hb S. A landmark 2024 French family study (Bobillier et al., eJHaem) described homozygous Hb Ottawa co-existing with Hb S trait producing severe vaso-occlusive crises, significantly worse than isolated sickle cell trait. The putative mechanism involves altered α-chain interactions potentiating HbS polymer formation.

Pullon & Moore (Thalass Rep, 2020) reported the first identification of Hb Ottawa in an Indian patient using CZE, noting variant percentages of 14–33% depending on co-inherited α-globin status -- consistent with our H100 observations (~22–27%).

3.9.1 Clinical Specimen — Hb Ottawa (Case GH14)

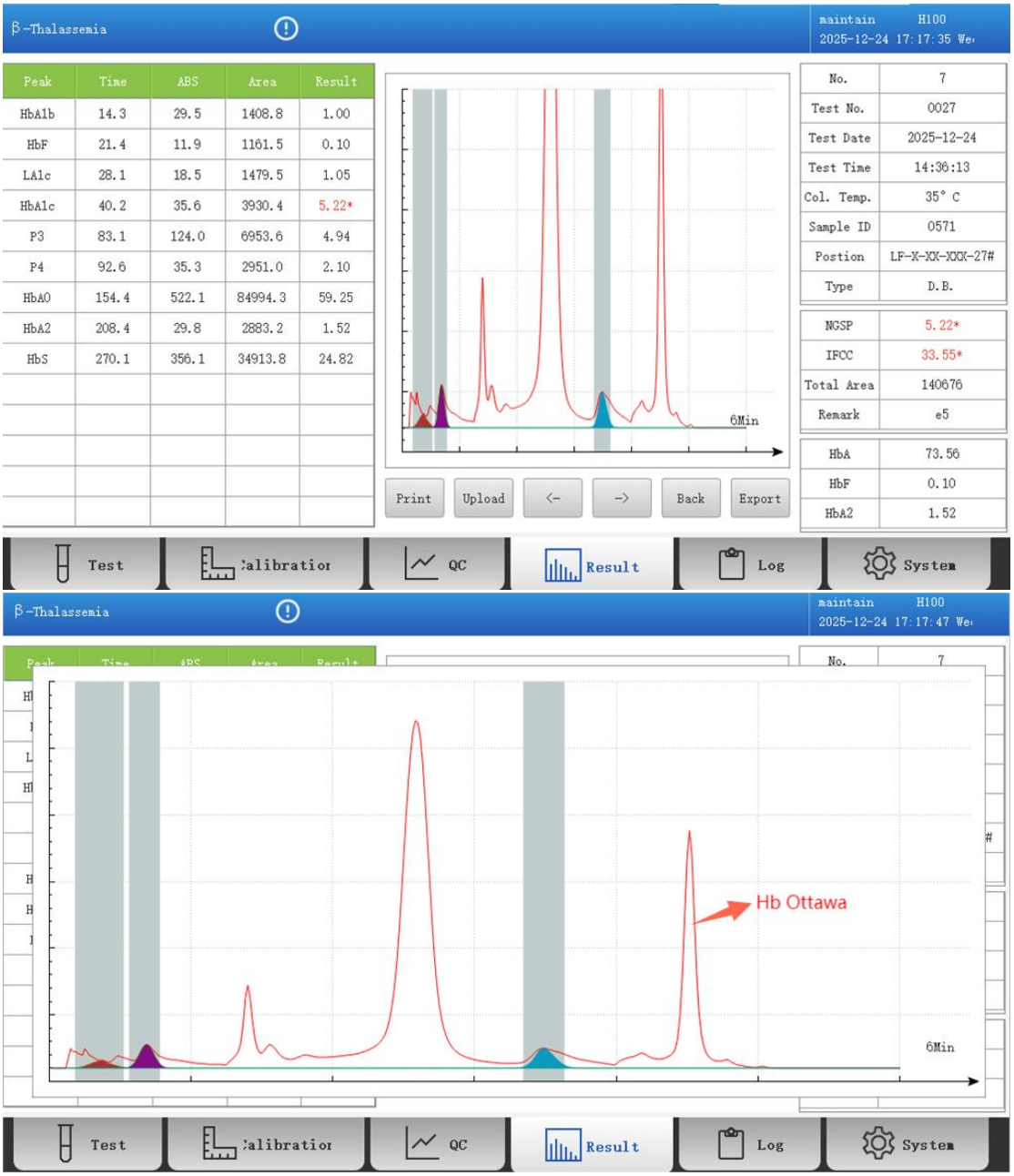
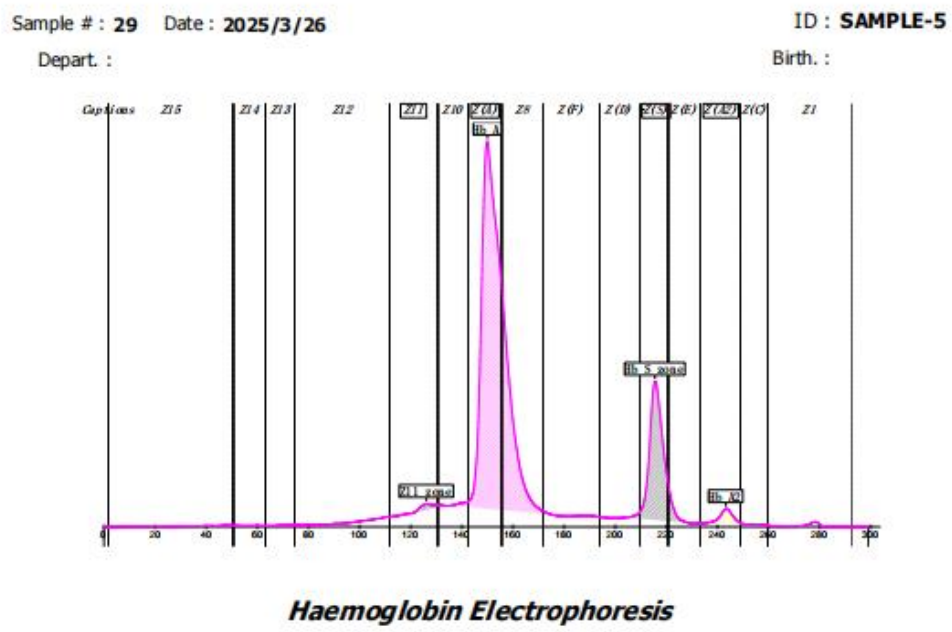


Figure 3.9.1.1 H100 (β-Thalassemia Mode) — Clinical whole-blood specimen (Case GH14): Hb Ottawa confirmed by Sanger sequencing (Peking University Hospital).

sebia



Name	%	Normal Values %
Z11 zone	0.8	
Hb A	78.4	
Hb S zone	18.9	
Hb A2	1.9	

Figure 3.9.1.2 Hemoglobin Analysis Electropherogram of Special Sample GH14 by Sebia Capillary Electrophoresis

CHROMATOGRAM INTERPRETATION	
HbA1c Result	**5.22%** (reportable — α-chain variant; HbA1c unaffected)
H100 Display	'Hb S' ⚠ (not true Hb S)
Variant RT	**270.1 s** ← KEY CLUE: ~5 s later than true Hb S (~265 s); careful RT inspection may reveal this difference
Variant Area	**24.82%** ← KEY CLUE: lower than typical Hb S heterozygote (~44%); approximately half the expected Hb S trait percentage
Peak Morphology	● Sharp discrete post-HbA2 peak; ● later-eluting than typical Hb S; ● HbA0 and HbA1c peaks intact.
E5 Flag	Present

Confirmatory	Sanger sequencing: HBA1/2 c.46G>C (Hb Ottawa / Hb Siam). Sebia CZE: peaks in zones 11 and Hb S zone — cannot be distinguished from true Hb S on CZE alone; molecular sequencing is the definitive test.
Interpretation	Two key H100 clues pointing away from true Hb S: (1) the variant RT is ~5 s later than typical Hb S; (2) the area (~25%) is substantially less than expected for Hb S trait (~44%). In a patient of East/Southeast Asian ancestry, these observations should prompt immediate molecular analysis.
⚠ CAUTION	Hb Ottawa migrates to the Hb S zone on BOTH HPLC and CZE (Sebia) — making molecular sequencing the ONLY definitive confirmatory method for this variant.
⚠ CAUTION	● **Reporting 'Hb S trait' in a patient with Hb Ottawa without molecular confirmation carries a risk of inappropriate sickle cell disease counseling.** ● **Verify all H100 'Hb S' results in East/Southeast Asian patients before final reporting.**

4. Look-Alike Variant Comparison Panels

This section summarizes the discriminating features between variants that co-elute in the same H100 recognition window, enabling operators to select the correct confirmatory strategy.

4.1 Hb E Window Look-Alikes (~228 – 246 s)

Variant	RT (s)	Area %	CZE Zone	Confirmatory Action
Hb E (true)	~240–242	~25–30%	Hb E zone (CE)	Confirm in at-risk population; consider CZE
Hb G-Coushatta	~228–233	~43–48%	Hb D zone (CE)	RT <236 s + area >38% → order CZE + sequencing
Hb G-Taipei	~238–242	~40–44%	Hb D zone (CE)	Area >36% → order CZE (D-zone) + sequencing
Hb G-Honolulu	~242–246	~25–30%	Hb D zone (CE)	Clinically benign α-chain variant; CZE + sequencing

4.2 Hb S Window Look-Alikes (~264 – 272 s)

Variant	RT (s)	Area %	CZE Zone	Confirmatory Action
Hb S (true)	~264–266	~43–48%	Hb S zone (CE)	CZE confirms; appropriate in at-risk populations
Hb Ottawa	~268–272	~22–27%	Hb S zone (CE)	RT ~5 s later + area <30% → molecular sequencing

4.3 Hb C Window Look-Alikes (~288 – 303 s)

Variant	RT (s)	Area %	CZE Zone	Confirmatory Action
Hb C (true)	~299–303	~45–99%	Hb C zone (CE)	CZE confirms; appropriate in African-ancestry patients
Hb Q-Thailand	~288–292	~20–25%	Hb F/Hb E zone	RT ~10 s earlier + area <30% + α-chain → CZE + sequencing

5. Diagnostic Algorithm — E5 Flag Investigation

The following algorithm guides operators from an H100 E5 flag to a final variant identification.

Step 1	Verify instrument calibration and QC status. Compare the suspect chromatogram with same-day calibrator and QC references.
Step 2	Record the variant retention time (RT) and area percentage reported by the H100.
Step 3	Identify the H100 recognition window (Hb E/D/S/C). Consult the Look-Alike Comparison Panels (Section 13) for RT and percentage clues pointing to the true variant.
Step 4	Review CBC parameters: MCV, MCH, Hb concentration, and RBC morphology on peripheral smear (if have). Cross-reference with expected values in each variant's CBC Correlation field.
Step 5	Assess patient ethnicity, geographic origin, and family history. East/Southeast Asian ancestry with 'Hb S' → suspect Hb Ottawa. Northern Chinese with 'Hb E' area >36% → suspect Hb G-Taipei or Hb G-Coushatta.
Step 6	Perform capillary zone electrophoresis (CZE, e.g., Sebia Capillarys). Compare CZE zone migration pattern with Section 13 tables.
Step 7	Order molecular analysis (Sanger sequencing of HBA1, HBA2, HBB; or NGS hemoglobinopathy panel) for definitive identification. Interpret in conjunction with HPLC, CZE, CBC, and family studies.
Step 8	Issue a confirmed variant report. Provide genetic counseling referral if the result has reproductive or clinical implications.

6. Bilingual Glossary

English Term	中文术语	Definition / 释义
Hemoglobin variant	血红蛋白变异体	A structural hemoglobin in which one or more amino acids in a globin chain differ from the normal sequence 球蛋白肽链中一个或多个氨基酸发生改变的血红蛋白
Thalassemia	地中海贫血	Hereditary disorder caused by reduced globin chain synthesis 遗传性珠蛋白合成减少引起的贫血
Heterozygote / Carrier	杂合子 / 携带者	Individual with one normal and one mutant allele 携带一个正常等位基因和一个突变等位基因的个体
Homozygote	纯合子	Individual with two copies of the same mutant allele 携带两个相同突变等位基因的个体
Compound heterozygote	复合杂合子	Individual carrying two different mutant alleles at the same locus 同一基因座携带两种不同突变等位基因
Retention time (RT)	保留时间	Time (seconds) from injection to peak apex in HPLC chromatogram HPLC 色谱图中从进样到峰顶的时间（秒）
E5 flag	E5 警示	H100 instrument alert indicating possible hemoglobin variant interference H100 仪器提示可能存在变异体干扰的警示信息
Capillary zone electrophoresis (CZE)	毛细管区带电泳	Complementary separation method using electric charge differences 利用电荷差异进行分离的辅助检测方法
IFCC Reference Material	IFCC 参考品	Certified reference material from the International Federation of Clinical Chemistry 国际临床化学联合会认证参考品
Vaso-occlusive crisis (VOC)	血管闭塞危象	Painful event caused by sickle red cells blocking blood vessels 镰状红细胞阻塞血管引起的疼痛发作

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DISCLAIMER

This Chromatogram Library is intended as a reference guide for trained laboratory professionals. All HPLC-based variant identification is presumptive; final diagnosis requires clinical correlation and confirmatory molecular analysis. This document does not constitute medical advice.