

Case Studies

MOLECULAR AND HAEMATOLOGICAL CHARACTERISATION OF HAEMOGLOBIN G-GEORGIA: A CASE SERIES FROM TERENGGANU, MALAYSIA

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ABSTRACT

Haemoglobin G-Georgia (*HBA1*:c.287C>T) is a rare and benign alpha globin chain variant with amino acid substitution of Proline to Leucine in the alpha gene. Hb G-Georgia was initially documented in an African American male in 1970, its occurrence in Malaysia was first characterised in 2015 in a patient of Malay ethnicity. This is a cross-sectional retrospective laboratory-based study describing the haematological parameters, clinical phenotypes, and genotypic profiles of 12 individuals with Hb G-Georgia from the state of Terengganu in Malaysia from the year 2017 to 2022 based on results from the full blood count, haemoglobin analysis, and molecular analysis. Twelve cases of Hb G-Georgia in the heterozygous state, compound heterozygous state of Hb G-Georgia with deletional alpha thalassaemia and in co-inheritance of Hb G-Georgia with heterozygous Hb E (*HBB*:c.79G>A) were reported. Hb G-Georgia is predominantly linked to mutations in the *HBA2* gene. However, all documented cases within the Malaysian cohort occurred on the *HBA1* gene, explaining the measured Hb G-Georgia proportions of 8.9% to 10.6%. All cases identified in Terengganu were clinically asymptomatic, consistent with the benign nature of Hb G-Georgia. Comprehensive molecular characterization and mapping of local genetic variants offer critical insights into the population-specific spectrum of haemoglobin disorders, ultimately enhancing carrier detection, risk assessment, and genetic counselling.

KEYWORDS: Haemoglobinopathy, Alpha Variant, Hb G-Georgia

INTRODUCTION

Thalassaemia is an autosomal recessive disorder characterised by reduced or absent synthesis of one or more globin chains. In contrast, haemoglobinopathies or haemoglobin (Hb) variants are inherited disorders resulting from structural alterations in the globin chains. These structural alterations can be due to point mutations, small deletions or insertions of nucleotide(s). Point mutations of a single nucleotide within exons can lead to benign to clinically significant changes in the form of same-sense, missense, nonsense, or new-sense mutations. To date, 1472 Hb variants have been identified, most of which arise from point mutations in the alpha or beta-globin genes (1).

It is estimated that approximately 6.8% of the Malaysian population are carriers of either the α - or β -thalassaemia trait (2). Alpha thalassaemia is the most prevalent inherited disorder in Malaysia. Alpha variants are not as common as alpha thalassaemia deletions, such as $-\alpha^{3.7}$, $-\alpha^{4.2}$ and $--^{SEA}$ among Malaysian multiethnic and multiracial populations. The common pathogenic alpha variants in the Malaysian population are Hb Constant Spring and Hb Adana. Hb G-Georgia (*HBA1:c.287C>T*) (IthaiD: 3718) is a rare and benign alpha globin chain variant with amino acid substitution of Proline to Leucine in the alpha gene involving either *HBA1* or *HBA2*.

This variant was first reported in an African American male in 1970. Hb G-Georgia was first discovered in Malaysia in an individual from the Malay ethnicity in 2015. A review of existing literature suggests that this is the first case series of Hb G-Georgia in Malaysia. This study investigated the haematological parameters, clinical phenotypes, and genotypic profiles of individuals identified with Hb G-Georgia in Terengganu.

In Malaysia, a variety of rare alpha-globin chain variants have been identified and reported at the Institute for Medical Research (IMR), ranging from benign to pathogenic mutations. The benign rare alpha chain variants, such as Hb Singapore and Hb Arya highlights the diversity of Hb variants in our region (3,4). Meanwhile, the identification of rare significant pathogenic alpha variants such as Poly A (*AATAAA>AATA--*) and Hb Taiping emphasizes the importance of accurate detection of these mutations for genetic counselling, carrier detection and risk assessment (5,6).

MATERIALS AND METHODS

A cross-sectional retrospective laboratory-based study was conducted describing the haematological parameters, clinical phenotypes, and genotypic profiles of 12 individuals with Hb G-Georgia from the state of Terengganu in Malaysia from 2017 to 2022. Data were obtained from the Laboratory Information System of Hospital Sultanah Nur Zahirah (HSNZ), Terengganu, and IMR, Selangor. The presumptive diagnosis of Hb variant was based on full blood count (FBC) parameters and Haemoglobin Analysis (Hb Analysis) results derived at HSNZ. The definitive diagnosis of this variant was derived from molecular analysis performed in IMR.

Haematological analysis by FBC and Hb analysis

The FBC was analysed utilising the Sysmex XN-Series™ Automated Hematology Analyzers (Sysmex, Japan). A full blood picture (FBP) was performed as a supplementary test for Hb analysis. Hb analysis included High-Performance Liquid Chromatography (HPLC) using the Bio-Rad Variant II system (Bio-Rad Laboratories, USA) and Capillary Electrophoresis (CE) using the Sebia Capillary 2 Flex Piercing system (Sebia, France). Agarose gel electrophoresis at alkaline pH was performed using the Sebia HYDRAGEL HEMOGLOBIN (E) assay (Sebia, France).

Molecular analysis

In IMR, DNA was extracted from the peripheral blood leukocytes using the QIA Symphony SP system (Qiagen, GmbH, Germany), and its concentration was subsequently quantified with a NanoDrop 1000 spectrophotometer (Thermo Fisher Scientific Inc., Wilmington, DE, USA). PCR-based amplification of the *HBA1* and *HBA2* genes was conducted using specific primers. Sanger cycle sequencing was performed using the BigDye® Terminator v3.1 Cycle Sequencing Kit. The resulting sequences were generated on an ABI 3730XL DNA Analyzer (Applied Biosystems, Foster City, CA, USA) and subsequently compared with normal reference sequences using CLC Main Workbench (CLC Bio, Aarhus, Denmark), which facilitated sequence assembly and alignment. Multiplex Gap-PCR for detecting common α -globin gene deletions was also performed following the method described in this paper (7). This assay was used to screen for the $-\alpha^{3.7}$, $-\alpha^{4.2}$, $--SEA$, $--FIL$, $--MED$, $-(\alpha)^{20.5}$, and $--THAI$ deletions. For one case, confirmation of heterozygous Hb E was performed using the Multiplex Amplification Refractory Mutation System (ARMS) PCR method as described in this paper (8).

Statistics

The results were analysed using SPSS version 25 (IBM Corp., Armonk, NY, USA). Because of the small sample size ($n = 12$ with subgroup sizes down to $n = 3$ and $n = 1$), formal normality testing was not performed due to its limited statistical power and poor specificity in small sample sizes, which can result in the inappropriate selection of parametric summary methods. Consequently, continuous variables (Hb, RBC, MCV, MCH, and Hb subtype percentages) are therefore presented descriptively as median (range), while categorical variables (age group, sex, and genotype) are expressed as frequencies and percentages throughout the Results section and Table 1.

RESULTS

Twelve cases of Hb G-Georgia(*HBA1:c.287C>T*) in the heterozygous state, compound heterozygous state of Hb G-Georgia with deletional alpha thalassaemia, and in co-inheritance of Hb G-Georgia with heterozygous Hb E (*HBB:c.79G>A*) were reported. All individuals were of Malay ethnicity. Eleven individuals were 16 years old, and one was 24 years old at the time of diagnosis. Six are female and another six are male. Most individuals were detected during the National Thalassaemia Screening Programme. These individuals were clinically healthy and asymptomatic.

Eight individuals were in the heterozygous state of Hb G-Georgia where the Hb G-Georgia levels ranged from 8.9-10.6% with CE method. The median (range) value of Hb G-Georgia was 10.2 (8.9-10.6) % in these heterozygous Hb G-Georgia individuals. Meanwhile, individuals with compound heterozygous Hb G-Georgia and ($-\alpha^{3.7}$) deletion, the Hb G-Georgia levels ranged from 17.3 to 18.5 % with a median value of 18.0%. Interestingly, the Hb G-Georgia level was only 3% in an individual with co-inheritance of heterozygous Hb G-Georgia with heterozygous Hb E (*HBB:c.79G>A*) and the Hb E level was 22.7%. The median (range) for RBC parameters (Hb, RBC, MCH and MCV) for all cases are described in the Table 1. As this is a descriptive case series with a small sample size, no inferential comparison was performed.

Hb analysis by CE demonstrated a peak for Hb G-Georgia in zone 7, whereas no additional peak was observed because Hb G-Georgia co-elutes with Hb A when the HPLC method utilised. The Hb analysis results are shown in Figure 1. Haemoglobin electrophoresis on agarose gel electrophoresis at alkaline pH demonstrated that the band of Hb G-Georgia migrated cathodal to the Hb S control band which is shown in Figure 2. Sanger sequencing of the *HBA1* gene identified Hb G-Georgia (*HBA1:c.287C>T*), as illustrated in Figure 3.

Table 1. Haematological parameters and haemoglobin (Hb) subtype profiles of Hb G-Georgia

Case	Age	Gender	Hb (g/dL)	RBC ($\times 10^{12}/L$)	MCV (fL)	MCH (pg)	CE Hb A (%)	CE Hb A2 (%)	CE Hb G-Georgia (%)	HPLC Hb A (%)	HPLC Hb A2 (%)	HPLC Hb F (%)
Heterozygous state of the Hb G-Georgia (<i>HBA1:c.287C>T</i>) mutation												
S1	16	F	12.7	5.23	80.1	24.3	86.9	2.5	10.6	Not done	Not done	Not done
S2	16	M	14.3	5.43	80.7	26.3	87.3	2.6	10.1	Not done	Not done	Not done
S3	16	M	13.5	5.22	74.9	25.9	87.2	2.6	10.2	Not done	Not done	Not done
S4	16	M	13.7	5.10	75.1	26.9	86.7	2.7	10.6	87.6	3.4	0.4
S5	16	F	13.8	5.16	80.6	26.7	87.5	2.3	10.2	87.3	2.7	0.3
S6	16	F	13.9	5.40	78.3	25.7	87.8	2.4	9.8	87.2	3.0	0.2
S7	16	F	12.8	4.77	79.2	26.8	87.1	2.7	10.2	86.4	3.1	0.5
S8	24	M	14.2	5.58	76.5	25.4	88.2	2.9	8.9	86.6	3.2	0.5
N=8	-	-	13.8 (12.7- 14.3)	5.2 (4.8-5.6)	78.8 (74.9- 80.7)	26.1 (24.3- 26.9)	87.2 (86.7- 88.2)	2.6 (2.3- 2.9)	10.2 (8.9-10.6)	87.2 (86.4- 87.6) N=5/8	3.1 (2.7-3.4) N=5/8	0.4 (0.2-0.5) N=5/8
Compound heterozygous state of the ($-\alpha^{3.7}$) deletion and Hb G-Georgia (<i>HBA1:c.287C>T</i>) mutation												
S9	16	F	12.2	4.65	77.8	26.2	79.6	2.4	18	Not done	Not done	Not done
S10	16	M	14.8	6.05	69.1	24.5	79.4	2.1	18.5	87.1	2.7	0.2
S11	16	M	15.7	6.14	77.0	25.6	80.1	2.3	17.3	84.6	2.9	0.3
N=3	-	-	14.8 (12.2- 15.7)	6.0 (4.7-6.1)	77.0 (69.1- 77.8)	25.6 (24.5- 26.2)	79.6(7 9.4- 80.1)	2.3 (79.4- 80.1)	18.0 (17.3-18.5)	84.6 and 87.1 N=2/3	2.7 and 2.9 N=2/3	0.2 and 0.3 N=2/3
Heterozygous state of the Hb G-Georgia (<i>HBA1:c.287C>T</i>) mutation. Co-inheritance with heterozygous Hb E (<i>HBB:c.79G>A</i>)												
S12	16	F	9.8	5.65	60.7	17.3	71.3	3.0	3.0	Not done	Not done	Not done
N=1									Hb E: 22.7			

Abbreviations: M, male; F, female; RBC, red blood cells; Hb, haemoglobin; MCV, mean cell volume; MCH, mean cell haemoglobin; CE, capillary electrophoresis; HPLC, high performance liquid chromatography

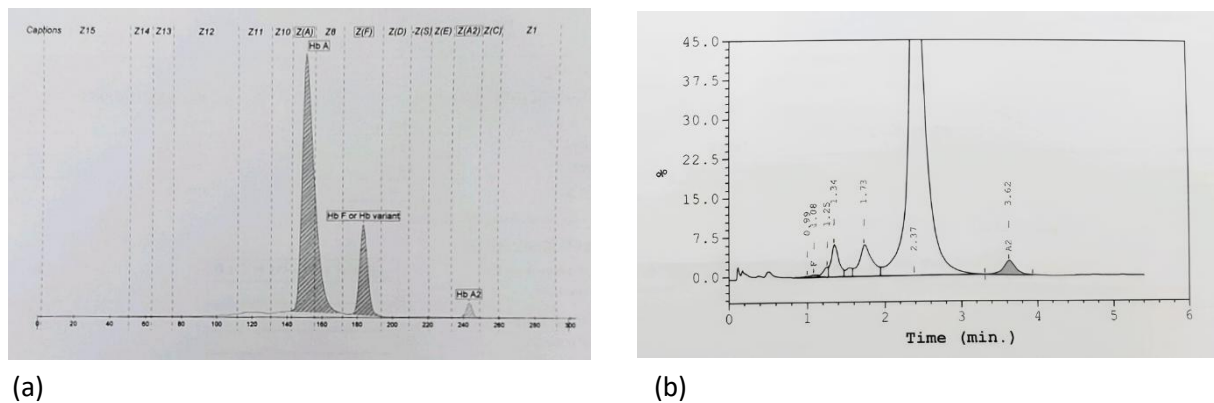


Figure 1. Figure (a) represents CE analysis. The peak of Hb G-Georgia (10.6%) was observed at zone 7. Figure (b) shows HPLC analysis. The total area is in between 1 to 3 million. The Hb F is in between 0.98-1.20 min. The Ao peak showed an Hb A level of 87.1%, and the peak A2 showed an Hb A2 level of 2.7%. No abnormal peaks were observed. Hb G-Georgia co-elutes with Hb A (Ao peak).

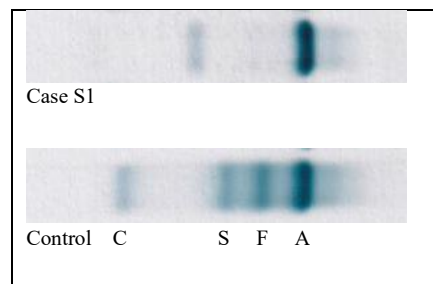


Figure 2. Haemoglobin electrophoresis on agarose gel electrophoresis at alkaline pH demonstrated that the band of Hb G-Georgia migrated cathodal to the Hb S control band. The agarose gel electrophoresis results of Case S1 compared to the control bands labelled as C, S, F and A.

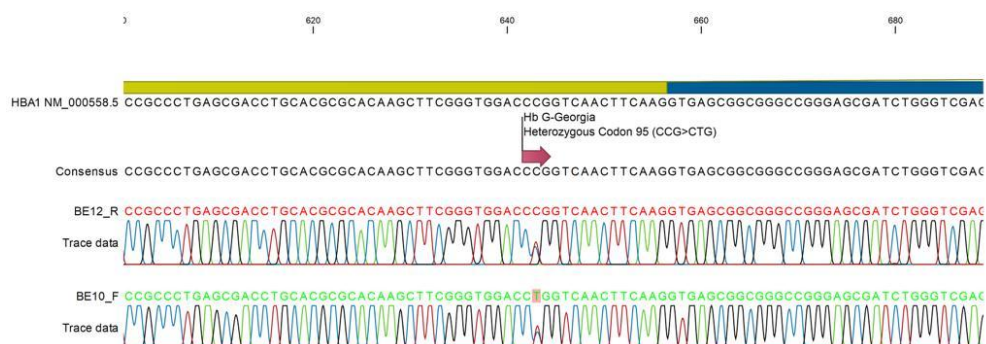


Figure 3. Sanger sequencing of *HBA1* gene in Case S1 identified Hb G-Georgia (*HBA1*:c.287C>T) in the heterozygous state.

DISCUSSION

Hb G-Georgia is an infrequently reported alpha chain variant. To date, there has been limited literature on this variant, particularly in our population. The occurrence of this variant among Malays was observed in all the cases. Based on the ITHANET portal and HbVar database, this variant has been classified as a benign variant, and this study's cases support this classification.

Since Hb G-Georgia's first report in 1970, this variant has been observed in several multi-ethnic cohorts, with documented cases among individuals of African American, Turkish, Portuguese, and Thai population (9-19). The first case report in 1970 showed the presence of heterozygous Hb G-Georgia in a 12-year-old African American male. This case concluded that Hb G-Georgia constituted 25% of the total haemoglobin and did not affect the carrier (9). The reported proportion of heterozygous Hb G-Georgia in Terengganu spans 8.9%–10.6%. This percentage varies according to whether the mutation resides on the *HBA1* or *HBA2* gene, thereby explaining the notable disparity between the local Hb G-Georgia levels and those reported internationally.

Previous case reports have identified several instances of co-inheritance of Hb G-Georgia and beta variants like Hb S, Hb C and Hb Montreal II. A co-inheritance of Hb G-Georgia with Hb S was detected in a patient with severe hypochromic anaemia, mild hepatomegaly and reticulocytosis but these findings were deemed not representative of the co-inheritance of Hb G-Georgia with Hb S as the patient had severe iron deficiency anaemia due to epistaxis. The author was unable to demonstrate any changes in the sickling effect of Hb S in the presence of Hb G-Georgia (10).

The co-inheritance of Hb G-Georgia with Hb C was totally benign where the Hb A level was 50.8%, Hb C level was 37.3%, and an unidentified Hb level of 9.3% with quantitation from isoelectric focusing (11). Another case of co-inheritance of Hb G-Georgia with the rare unstable beta chain variant Hb Montreal II was reported where the patient presented with haemolytic anaemia following a viral infection. The presence of splenomegaly and the episode of haemolytic anaemia were attributed to Hb Montreal II (12). The only case of Hb G-Georgia in Terengganu in association with beta variant is with Hb E trait (*HBB:c.79G>A*) and this co-inheritance is completely asymptomatic.

An interaction between Hb G-Georgia and deletional alpha thalassaemia was observed in a case from Thailand. In this case, an adult male with mild hypochromic microcytic anaemia portrayed an interaction between α^0 -thalassaemia (SEA deletion) and Hb G-Georgia (*HBA2:c.287C>T*). A relatively high level of Hb G-Georgia (44.9%) was observed. This report concluded that the individual did not manifest Hb H disease, hence proving that Hb G-Georgia is not an alpha thalassaemic allele (13). No cases of Hb G-Georgia co-inherited with alpha-zero thalassaemia have been reported in Terengganu.

Interestingly, the first case of Hb G-Georgia, involving the *HBA1* gene, was reported in Southern Thailand. The level of Hb G-Georgia (*HBA1:c.287C>T*) was notably lower than that of Hb G-Georgia (*HBA2:c.287C>T*), at 10.3–10.6% vs. 17.0% respectively (14). In previous studies, Hb G-Georgia in the heterozygous state, involving *HBA2* gene showed percentage of Hb variant of 17-25% (9,14). Hb G-Georgia typically involved the *HBA2* gene but interestingly all the cases reported in Malaysia involved the *HBA1* gene, hence explaining the value Hb G-Georgia in Terengganu ranging from 8.9 to 10.6%.

The Hb G-Georgia percentage in this study is lower compared to the reported cases in other part of the world because this mutation involves the *HBA1* gene. The two allelic $\alpha 2$ genes are usually responsible for 75% of alpha chain production. It is expected that the alpha variant involving the $\alpha 1$ gene gives rise to an alpha Hb variant level of approximately 12.5% of the total haemoglobin. The

percentage may be reduced, as observed in this case series, due to synthesis at a reduced rate or the lesser affinity of this alpha variant chain for the beta chain compared to the normal alpha chain (20).

The percentage of Hb E is markedly reduced compared to the level known among Hb E heterozygotes due to co-inheritance of this alpha Hb variant in this case series. The percentage of Hb G-Georgia was also reduced in this individual. The level of Hb G-Georgia is low, around 3% only in co-inheritance of Hb E trait probably indicating less preference of Hb G-Georgia formation.

In general, β^E chain exhibits a reduced affinity to alpha chain because it is more electropositive than normal beta chain. So, in the presence of β^E and Hb G-Georgia, normal alpha chain preferentially pair with normal beta chain, thereby reducing the Hb E and Hb G-Georgia levels. The percentage of Hb G-Georgia is higher in co-inheritance of alpha plus or alpha zero thalassaemia because in the event of reduced alpha globin chains, the formation of Hb G-Georgia is preferred. For to the same reason, in co-inheritance of Hb G-Georgia with double gene deletion, the percentage of Hb G-Georgia was 44.9% (13).

LIMITATIONS

There are limitations to this study. This study is primarily limited by its small sample size and small subgroup sizes, which constrained the analysis to descriptive reporting. This study was intended to characterise molecular and haematological findings of this rare alpha variant in Terengganu without inferential statistical analysis.

CONCLUSION

Haemoglobin variants are mainly clinically silent and are mostly detected by Hb analysis and molecular genotyping. Hb analysis by CE demonstrated a peak for Hb G-Georgia in zone 7, whereas no additional peak was observed when the HPLC method utilised. Haemoglobin electrophoresis on agarose gel electrophoresis at alkaline pH demonstrated the band of Hb G-Georgia migrated cathodal to the Hb S control band. Comprehensive characterization and mapping of genetic variants provide valuable insights into the molecular spectrum of disorders within a population.

This information is critical for improving genetic counselling, enabling more accurate carrier detection and risk assessment. Furthermore, these findings support the development of population-based targeted diagnostic panels, which may enhance screening efficiency, reduce diagnostic costs, and contribute to better prevention and management strategies.

Conflicts of Interest: The authors declared no conflict of interest.

Author Contributions: Study conceptualisation, design and draft writing: SS;. Analysis, interpretation of results and data collection: SS, UKS, ESZ, NAA, FSAH, SH, ASAG, WAHWH, WZWE; Proofreading and editing of manuscript: ANMN, ENMS, SNZ, EE, YMY, NMY. All authors contributed to the article and approved the submitted version. IPNB; methodology: IPNB, NSMD;

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Ethical Declaration: This study was registered with the National Medical Research Register (NMRR), Malaysia (NMRR ID: NMRR-23-02276-ZSL). Ethical review was not sought for this study, as it involved the secondary analysis of retrospective, fully anonymized diagnostic data from HSNZ and IMR without patient-identifiable information. The study adhered to the principles of the Declaration of Helsinki.

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REFERENCES

1. Giardine B, Borg J, Vienonas E, Pavlides M, Wadamori W, Hardison RC, et al. Updates of the HbVar database of human hemoglobin variants and thalassemia mutations. *Nucleic Acids Res.* 2014;42(D1)–9.
2. Ibrahim HM, Modak AI, Ismail P, Mohamed-Kassim FA, Abdullah W, Hassan S, et al. Observational study on the current status of thalassaemia in Malaysia: a report from the Malaysian Thalassaemia Registry. *BMJ Open.* 2020;10(6).
3. Tan AS, Quah TC, Low PS, Chong SS. A rapid and reliable 7-deletion multiplex polymerase chain reaction assay for α -thalassemia. *Blood.* 2001;98(1):250–1.
4. Hassan S, Ahmad R, Zakaria Z, Zulkafli Z, Abdullah W. Detection of β -globin gene mutations among β -thalassaemia carriers and patients in Malaysia: application of multiplex amplification refractory mutation system–polymerase chain reaction. *Malays J Med Sci.* 2013;20(1):13–20.
5. Yasin NM, Ismail A, Mohammad S, Yusoff NM. The haematological and molecular profile of Hb Singapore [HBA2: c.425G>C] unique among the Malays from Kelantan, Malaysia. *Malays J Hum Genet.* 2022;3(2):1–8.
6. Nahanthiran S, Yasin NM, Rosli S, Abdullah W, Zakaria Z. Family study of haemoglobin Arya in a Malaysian family. *Malays J Pathol.* 2024;46(2):315–20.
7. Yasin NM, Ismail A, Yusoff NM, Hassan S, Ahmad R. The clinical and laboratory profiles of a deletional $\alpha 2$ -globin gene polyadenylation signal sequence (AATAAA>AATA--)[HBA2: c.93_94delAA]: the Malaysian experience. *Diagnostics.* 2025;15(10):1284.
8. Yasin NM, Ismail A, Yusoff NM, Ahmad R. ACMG/AMP-based variant classification of a novel HBA2 variant (HBA2: c.297del, Hb Taiping) in compound heterozygosity with Hb Adana (HBA2: c.179G>A) causing non-deletional Hb H disease. *Int J Lab Hematol.* 2026. [Epub ahead of print].
9. Huisman TH, Dozy AM, Adams HR, Wilson JB, Schroeder WA. Hemoglobin G Georgia or $\alpha 295$ Leu (G-2) $\beta 2$. *Biochim Biophys Acta.* 1970;200(3):578–80.
10. Wrightstone RN, Hubbard M, Huisman TH. Hemoglobin S-G α Georgia disease: a case report. *Acta Haematol.* 1974;51(5):315–20.
11. Reynolds S, Kutlar A, Kutlar F, McKie VC, Huisman TH. Hemoglobin C--G-Georgia double heterozygosity: a case report. *Ann Clin Lab Sci.* 1992;22(6):414–6.
12. Medri C, Pastore YD, Robitaille N. Unstable hemoglobin Montreal II uncovered in an adult with unexplained hemolysis exacerbated by a presumed viral infection: a case report. *J Med Case Rep.* 2022;16(1):145.

13. Srivorakun H, Fucharoen G, Sanchaisuriya K, Fucharoen S. Novel interactions of two α -Hb variants with SEA deletion α^0 -thalassemia: hematological and molecular analyses. *Hematology*. 2018;23(3):187–91.
14. Theingi M, Ruengdit C, Punyamung M, Pornprasert S. First case of compound heterozygotes for Hb G-Georgia (HBA2.287C>T)/ α^0 -thalassemia --SEA deletion found in northern Thailand. *Nat Life Sci Commun*. 2025;24(4).
15. Smith LL, Wrightstone RN, Huisman TH. Subunit dissociation of the abnormal hemoglobins G Georgia ($\alpha^{295}\text{Leu}$ (G2) β^2) and Rampa ($\alpha^{295}\text{Ser}$ (G2) β^2). *J Biol Chem*. 1972;247(5):1433–9.
16. North ML, Thillet J, Gandar R, Oberling F. A new case of hemoglobin G Georgia. *Nouv Rev Fr Hematol*. 1975;15(4):460–7.
17. van Houte DP, Kluin-Nelemans HC, van Noord MJ, Nijhuis AW, Bernini LF. Hemoglobin G Georgia in a Turkish family in the Netherlands. *Ned Tijdschr Geneesk*. 1986;130(8):360–3.
18. Felice AE, Webber BB, Huisman TH. α -Thalassemia and the production of different α chain variants in heterozygotes. *Biochem Genet*. 1981;19(5):487–98.
19. Altay Ç. Abnormal hemoglobins in Turkey. *Turk J Haematol*. 2002;19(1):63–74.
20. Bain BJ, Rees DC. *Haemoglobinopathy Diagnosis*. 3rd ed. Hoboken (NJ): Wiley-Blackwell; 2025.