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Case Report Open Access

Coinherited Hemoglobin Lansing-Ramathibodi/Southeast Asian Deletional α^0-thalassemia and Hemoglobin E Causing Falsely Low Oxygen Saturation on Pulse Oximetry

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ABSTRACT

Hemoglobin (Hb) variants resulting from mutations in globin genes and leading to qualitative abnormalities of globin proteins can result in a spectrum of clinical presentations. Herein we describe a case of a 10-year-old boy with an Hb variant, Hb Lansing-Ramathibodi (*HBA1*: c.264C>G, codon 87 His>Gln), who was incidentally found to have low pulse oximetry oxygen saturation readings (low SpO_2). Laboratory investigations revealed mild microcytic anemia. Arterial blood gas showed normal partial pressure of oxygen (PaO_2) and arterial oxygen saturation (SpO_2) values, indicating an oxygen saturation gap. Subsequent Hb analysis identified an abnormal peak, prompting further molecular studies which confirmed the coinheritance of Hb Lansing-Ramathibodi, Southeast Asian deletional α^0-thalassemia and the Hb E trait. The patient's mother, carrying the Hb Lansing-Ramathibodi and Hb E traits without Southeast Asian deletional α^0-thalassemia, was asymptomatic. This case underscores the importance of identifying and characterizing rare Hb variants presenting with an oxygen saturation gap for informed clinical management and genetic counseling.

KEYWORDS alpha-thalassemia, coinheritance, hemoglobin Lansing-Ramathibodi, low SpO_2, oxygen saturation

INTRODUCTION

Hemoglobin (Hb), a tetrameric molecule composed of hemes and globin proteins, plays an important role in oxygen transportation from the lungs to tissues. Hb A, the predominant Hb in both children and adults, is composed of two α-globin and two β-globin chains. Hb variants arising from mutations in globin genes can lead to qualitative abnormalities in globin proteins, and can result in a spectrum of clinical presentations, including alterations in oxygen affinity. These variants may affect oxygen affinity through mechanisms such as R and T configuration, 2,3-BPG affinity, globin contact zone, heme pocket and length of globin chain (1, 2). These Hb variants, categorized into high and low oxygen affinity types, exhibit distinctive clinical features.

High oxygen affinity Hbs typically present as erythrocytosis due to elevated erythropoietin levels secondary to reduced tissue oxygen delivery, potentially leading to hyperviscosity, thrombotic events, and hemolysis (3-8). Conversely, low oxygen affinity Hbs are characterized by central cyanosis, low oxygen saturation and anemia (9-13). Additionally, several Hb variants associated with falsely low oxygen saturation measured using a

pulse oximeter (SpO_2) without actual hypoxemia have been reported (13).

Monitoring oxygen saturation is crucial in clinical diagnosis and patient management. Oxygen saturation can be assessed using a pulse oximeter which provide SpO_2 values, or through arterial blood gas measurements yielding partial pressure of oxygen (PaO_2) and arterial oxygen saturation (SaO_2) values. Certain Hb variants can result in an oxygen saturation gap, characterized by low SpO_2 measurements but normal SaO_2 and PaO_2 levels. Pulse oximetry relies on the principle of differential light absorption by oxygenated and deoxygenated Hbs to determine blood oxygen saturation levels. It employs two specific wavelengths of light, typically red (640-660 nm) and infrared (880-940 nm), which are absorbed differently by these Hb forms (14). The SpO_2 results may inaccurately appear lower due to changes in the light absorbance characteristics of the Hb variant (13).

In Thailand, Hb Lansing-Ramathibodi (*HBA1*:c.264C>G, codon 87 His>Gln) has been reported in association with falsely low SpO_2 values in two families (15, 16). The diagnostic process for Hb Lansing-Ramathibodi poses challenges due to its rarity (15-17). This report presents a case of an individual harboring a compound heterozygosity for Hb Lansing-Ramathibodi, α^0-thalassemia and Hb E (*HBB*:c.79G>A, codon 26 Glu>Lys), which provides insight into the clinical characteristics of this uncommon Hb variant.

CASE REPORT

A 10-year-old Thai boy was referred to our hospital due to low SpO_2 values detected by pulse oximetry. Approximately one month prior to the admission, he had been hospitalized for food poisoning, during which an SpO_2 of 74% was recorded. Arterial blood gas while in room air showed pH 7.515, $PaCO_2$ 29.7 mmHg, PaO_2 111.5 mmHg, HCO_3 23.8 mmol/L, and SaO_2 98.5%. Administration of supplemental oxygen via a heated humidified high flow nasal cannula (HHHFNC) with FiO_2 of 0.6 resulted in an increase in SpO_2 to 84-85%. Arterial blood gas while on HHHFNC showed PaO_2 221.7 mmHg and SaO_2 99.7%. Notably, the patient remained asymptomatic, with no complaints of dyspnea or chest pain, and had been in overall good health.

The patient was born at term, with a gestational age of 40 weeks, and had been delivered by Cesarean section due to fetal distress. The Apgar score was 9 at 1 and 5 minutes. At birth, his SpO_2 in room air was 82%, with a slight improvement to 89-90% following the administration of supplemental oxygen. At that time, the patient was diagnosed with mild persistent pulmonary hypertension of the newborn (PPHN).

He had no history of other medical conditions. He was the only child of the family. Both parents were healthy. Antenatal thalassemia screening indicated that the parents were not at risk of having a fetus with severe thalassemia. Specifically, the mother was identified as a carrier of Hb E, while the father carried Southeast Asian deletional α^0-thalassemia. There was no familial history of low oxygen saturation or hematologic disorders. The SpO_2 levels in room air for both parents were 98-99%.

At our hospital, physical examination revealed normal vital signs, with SpO_2 ranging from 75% to 77% in room air. He weighed 41.1 kg (75^{th} percentile) and measured 143 cm in height (25^{th}–50^{th} percentile), showing no cyanosis, pallor, or jaundice. Additionally, the examination revealed normal cardiac and respiratory profiles, absence of hepatosplenomegaly, and no digital clubbing.

Results of laboratory investigations of the patient and his mother are shown in Table 1. Hb analysis was done by high-pressure liquid column chromatography (HPLC) using the Variant II HPLC system (Bio-Rad Laboratories, Hercules, CA, USA). An abnormal Hb peak was detected during the analysis, as illustrated in Figure 1. PCR for Southeast Asian deletional α^0-thalassemia was performed using a method with modifications from a previous study (18). β-thalassemia mutations were searched for using the PCR-high resolution melting analysis method as previously described (19). Subsequent Sanger DNA sequencing of α-globin genes *HBA2* and *HBA1* (Figure 2) identified heterozygous mutations causing Hb Lansing-Ramathibodi (*HBA1*:c.264C>G, codon 87 His>Gln) in both the patient and the mother. The patient was found to have Hb Lansing-Ramathibodi, Southeast Asian deletional α^0-thalassemia and Hb E, while his mother had Hb Lansing-Ramathibodi and Hb E trait without Southeast Asian deletional α^0-thalassemia.

Table 1. Results of laboratory investigations of the patient and his mother

	Patient	Mother
Hb (g/dL)	10.6	13.6
Hct (%)	34.0	41.4
RBC count (x10^6/mm^3)	5.94	5.63
MCV (fL)	57.2	73.5
MCH (pg)	17.8	24.2
MCHC (g/dL)	31.2	32.9
Reticulocyte (%)	1.14	1.68
WBC (/mm^3)	6,720	8,210
Platelet (/mm^3)	292,000	350,000
Hb analysis (by HPLC)	Hb A 62.0%, Hb A_2/E 15.9%, Hb F 1.2%, abnormal peak at retention time 1.92 min 17.5%	Hb A 63.2%, Hb A_2/E 27.9%, Hb F 1.2%
α-globin genes (*HBA2* and *HBA1*) analysis	Heterozygous Hb Lansing-Ramathibodi Heterozygous Southeast Asian deletional α^0-thalassemia	Heterozygous Hb Lansing-Ramathibodi
β-globin gene (*HBB*) analysis	Heterozygous Hb E	Heterozygous Hb E

Hb, hemoglobin; Hct, hematocrit; HPLC, high-pressure liquid column chromatography; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell

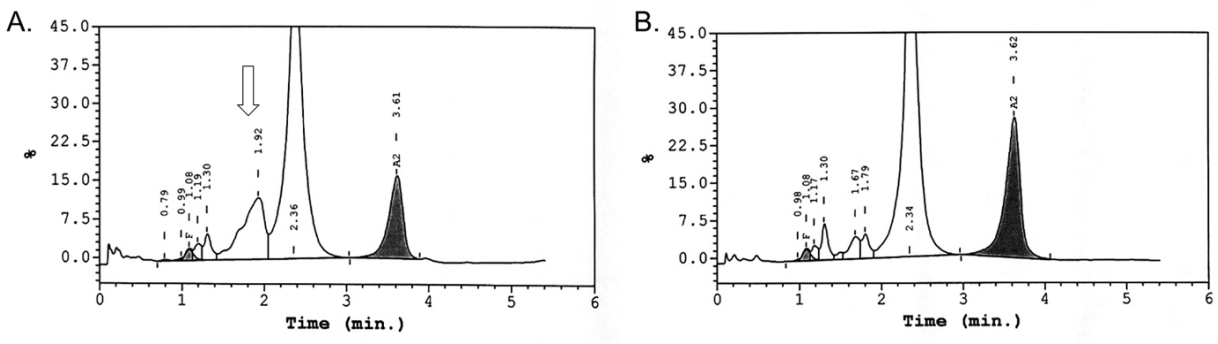

Figure 1. Hemoglobin analysis results using the high-pressure liquid column chromatography (HPLC) method from (A) the patient and (B) his mother. An abnormal hemoglobin peak at a retention time of 1.92 min (arrow) was detected in the patient.

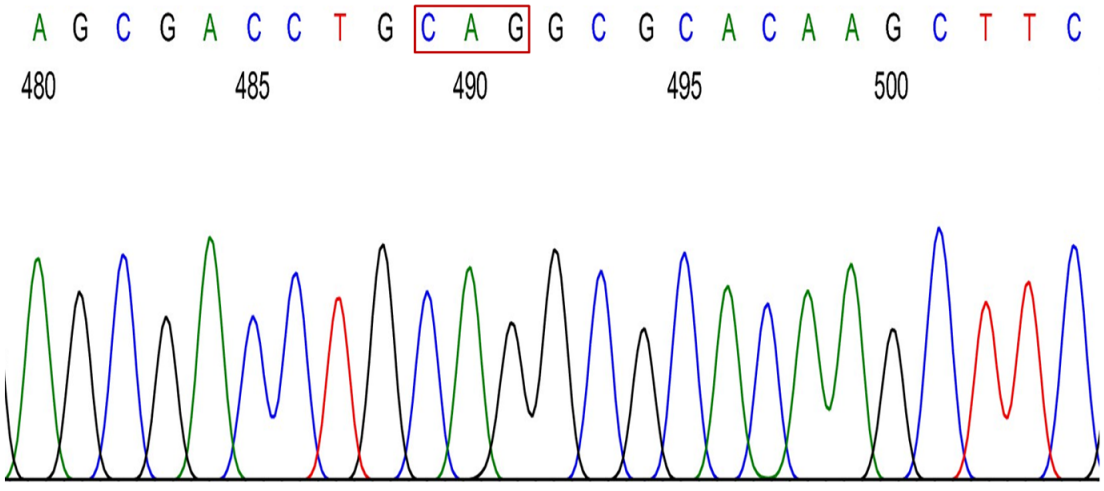

Figure 2. Sanger DNA sequencing of the *HBA1* gene from the patient identified a missense mutation causing hemoglobin Lansing-Ramathibodi (*HBA1*:c.264C>G). The mutation was co-inherited with Southeast Asian deletional α^0-thalassemia.

Other laboratory investigations revealed normal results, with a methemoglobin level of 1.2% (<2%), G6PD level of 18.6 U/gHb (normal range for males >4.0 U/gHb), serum iron level of 82 μg/dL (33-193 μg/dL) and total iron binding capacity of 251 μg/dL (228-428 μg/dL).

DISCUSSION

This case report illustrates the clinical presentation of low SpO_2 measurements with normal PaO_2 and SaO_2 and mild microcytic anemia in a patient with coinheritance of Hb Lansing-Ramathibodi, Southeast Asian deletional α^0-thalassemia and the Hb E trait. The impact of the heterozygous mutation causing Hb Lansing-Ramathibodi without co-inherited α-thalassemia appeared to be minimal as evidenced by the asymptomatic status of the mother who harbored only the heterozygous Hb Lansing-Ramathibodi mutation. Hb analysis of the mother did not reveal an Hb Lansing-Ramathibodi peak. Additionally, the coinheritance of Hb E also appeared to exert minimal effect.

In a systematic review conducted by Verhovsek et al., eleven Hb variants with low SpO_2 and discordantly normal SaO_2 were identified (13). All of these variants are caused by missense mutations, comprising six α-globin Hb variants: Hb Lansing (*HBA2*:c.264C>G, codon 87 His>Gln), Hb Titusville (HBA2 or *HBA1*:c.283G>A, codon 94 Asp>Asn), Hb Bonn (*HBA1*:c.262C>G, codon 87 His>Asp), Hb Delaware (*HBA2*: codon 91 Leu>Val), Hb M-Iwate (HBA2 or *HBA1*:c.262C>T, codon 87 His>Tyr), and a novel Hb (*HBA2* or *HBA1*: codon 62 Val>Ala), along with five β-globin Hb variants: Hb Hammersmith (*HBB*:c.128T>C, codon 42 Phe>Ser), Hb Cheverly (*HBB*:c.137T>C, codon 45 Phe>Ser), Hb Okazaki (*HBB*:c.280T>C, codon 93 Cys>Arg), Hb Regina (*HBB*:c.289C>G, codon 96 Leu>Val), and Hb Köln (*HBB*:c.295G>A, codon 98 Val>Met). Studies examining the absorption spectra of Hb Bonn, Hb Cheverly and Hb Köln have revealed alterations in the light absorbance characteristics of oxyhemoglobin and/or deoxyhemoglobin (13). These changes can potentially contribute to erroneous measurements observed on pulse oximetry. Other reported Hb variants with low SpO_2 and normal SpO_2 are Hb Hirosaki (*HBA2*:c.132C>G, codon 43 Phe>Leu), Hb Santa Ana (*HBB*:c.266T>C, Leu>Pro) and Hb Grifton (*HBA1*:c.263A>C, codon 87 His>Pro) (20-22).

Hb Lansing (*HBA2*:c.264C>G, codon 87 His>Gln) is among the Hb variants linked with unexpectedly low SpO_2 levels (13). Initially observed in a family of Hispanic background by Sarikonda et al. in 2009, subsequent cases have been reported spanning different ethnic backgrounds in individuals of Japanese, Turkish (Hb Lansing A) and Omani descent (23-26). Recently, a case of coinherited Hb Lansing, Hb S and α^+-thalassemia was reported with falsely low SpO_2 levels and hemolytic anemia (27). Hb Lansing-Ramathibodi (*HBA1*:c.264C>G, codon 87 His>Gln) results from the same missense mutation at codon 87, but on the *HBA1* gene. This variant was first identified in four members of a Thai family by Trakulsrichai et al. in 2016 and later reported in a Thai newborn by Prachukthum et al. in 2017 (15, 16). Recently, a case of a 2-year-old boy with Hb Lansing-Ramathibodi who presented with similarly low SpO_2 readings was reported (17).

The clinical findings from the three previously reported families with Hb Lansing-Ramathibodi are summarized in Table 2 (15-17). All cases presented with incidental findings of low SpO_2 levels. Hb levels and mean corpuscular volume (MCV) in cases with heterozygous Hb Lansing-Ramathibodi were normal or borderline low. In our case with the Hb Lansing-Ramathibodi, α^0-thalassemia and Hb E trait, both Hb levels and MCV were low. In terms of Hb analysis, a distinct, measurable abnormal Hb peak at a retention time of 1.7-2.0 minutes, as analyzed by HPLC method, was observed only in cases with co-inherited α-globin gene mutations (Hb Pakse, *HBA2*:c.429A>T, or Southeast Asian deletional α^0-thalassemia). Using the capillary electrophoresis (CE) method, a non-measurable peak was located between Hb A and Hb F. Notably, in the present report, the mother, who had the Hb Lansing-Ramathibodi trait and also harbored the Hb E mutation, exhibited normal SpO_2 levels, whereas two cases from the initial report of the Hb Lansing-Ramathibodi trait presented with SpO_2 levels of 88-90% in room air. The potential impact of co-inherited Hb E on SpO_2 levels warrants further investigation.

This case report underscores the clinical presentation of low SpO_2 measurements with normal PaO_2 and SpO_2 and mild microcytic anemia in a patient with co-inherited Hb Lansing-Ramathibodi, Southeast Asian deletional α^0-thalassemia,

Table 2. Summary of case reports with Hb Lansing-Ramathibodi

	Case number	Age (y)	Gender	Genotype	OxyHb (%) by co-oximetry	SpO_2 (%)	PaO_2 (mmHg)	SaO_2 (%)	Hb (g/dL)	MCV (fL)	Hb analysis
Trakulsrichai et al. 2016 (16)	1	40	Female	$\alpha^{Pakse}\alpha/\alpha\alpha^{L-R}$	69.1	84 in room air 89 on O_2 canula 5 L/min	385 on O_2 mask 10 L/min	100 on O_2 mask 10 L/min	11	81.8	Hb A 80.6%, Hb A_2 2.3%, Hb F 0.6%, abnormal peak at retention time 2.03 min (HPLC) A peak with a shoulder between Hb A and Hb F window (CE)
	2	48	Male	$\alpha\alpha/\alpha\alpha^{L-R}$	69.3	88	-	-	15.5	84.7	Hb A 74.8%, Hb A_2 2%, Hb F 0.8%, abnormal peak at retention time 1.70 min (HPLC) No peak seen by CE
	3	44	Male	$\alpha^{Pakse}\alpha/$	64.1	71	-	-	13.3	77.9	Hb A 76%, Hb A_2 1.7%, Hb F 2.1%
	4	17	Male	$\alpha\alpha^{L-R}$	-	90	-	-	14.5	78.7	-
Prachukthum et al. 2017 (15)	1	Term newborn (GA 38 weeks)	Male	$\alpha\alpha/\alpha\alpha^{L-R}$ $^{--SEA}/\alpha\alpha^{L-R}$	-	84 90 in O_2 hood, 90 on HHHFNC 5 L/min, FiO_2 1.0	389.5 on HHHFNC 5 L/min, FiO_2 1.0, 85.1 in room air	100 on HHHFNC 5 L/min, FiO_2 1.0, 97 in room air	16	-	Hb A 14.7%, Hb F 69.1%, Hb A_2 0.3%, Hb Bart's 15.9% by CE
Quinn and Klouda 2024 (17)	1	2	Male	Hb L-R mutation	-	92 in room air, not improved with O_2 supplement	normal	-	-	-	-
This report	1	10	Male	$^{--SEA}/\alpha\alpha^{L-R}$ β/β^{E}	-	75-77 in room air	221.7 on HHHFNC, FiO_2 0.6, 111.5 in room air	99.7 on HHHFNC, FiO_2 0.6, 98.5 in room air	10.6	57.2	Hb A 62.0%, Hb A_2/E 15.9%, Hb F 1.2%, abnormal peak at retention time 1.92 min 17.5%
	2	38	Female	$\alpha\alpha/\alpha\alpha^{L-R}$ α/α^{E}	-	99 in room air	-	-	13.6	73.5	Hb A 63.2%, Hb A_2/E 27.9%, Hb F 1.2%

CE, capillary electrophoresis; Hb, hemoglobin; HHHFNC, heated humidified high flow nasal cannula; HPLC, high-pressure liquid column chromatography; L-R, Lansing-Ramathibodi; MCV, mean corpuscular volume; PaO_2, partial pressure of oxygen; SaO_2, arterial oxygen saturation; SpO_2, oxygen saturation readings on pulse oximetry

and Hb E trait. The minimal impact of the heterozygous Hb Lansing-Ramathibodi mutation without co-inherited β-thalassemia is evidenced by the asymptomatic status of the mother harboring solely the heterozygous Hb Lansing-Ramathibodi mutation, with no detectable Hb Lansing-Ramathibodi peak in her Hb analysis. This case adds to the understanding of the rare Hb variant and its clinical implications for accurate diagnosis and management.

Research ethics approval

The research protocol complies with the research with exemption category. It has been certified as exempt from ethical review by the Research Ethics Committee of the Faculty of Medicine, Chiang Mai University (No. 0487/2023).

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CONFLICTS OF INTEREST

All authors declare no conflicts of interest.

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