



Corrigendum: The Spectrum of SPTA1-Associated Hereditary Spherocytosis

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OPEN ACCESS

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Specialty section:

This article was submitted to
Red Blood Cell Physiology,
a section of the journal
Frontiers in Physiology

Received: 03 September 2019

Accepted: 04 October 2019

Published: 18 October 2019

Citation:

Chonat S, Risinger M, Sakthivel H, Niss O, Rothman JA, Hsieh L, Chou ST, Kwiatkowski JL, Khandros E, Gorman MF, Wells DT, Maghathe T, Dagaonkar N, Seu KG, Zhang K, Zhang W and Kalfa TA (2019) Corrigendum: The Spectrum of SPTA1-Associated Hereditary Spherocytosis. *Front. Physiol.* 10:1331. doi: 10.3389/fphys.2019.01331

Keywords: SPTA1, α -spectrin, α -LEPRA, hereditary spherocytosis, next generation sequencing, hemolytic anemia, hydrops fetalis

A Corrigendum on

The Spectrum of SPTA1-Associated Hereditary Spherocytosis

by Chonat, S., Risinger, M., Sakthivel, H., Niss, O., Rothman, J. A., Hsieh, L., et al. (2019). *Front. Physiol.* 10:815. doi: 10.3389/fphys.2019.00815

In the original article, there was a mistake in **Table 1** as published. The SPTA1 mutation of Allele 2 in Patient 1, is stated as "c.4294T>A (p.L1432*)." The correct mutation should read "c.4295del (p.L1432*)." The corrected **Table 1** appears below.

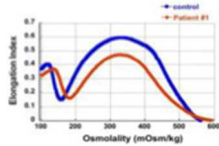
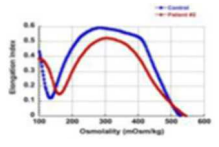
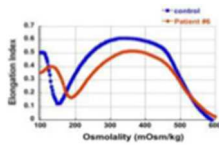
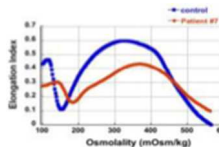
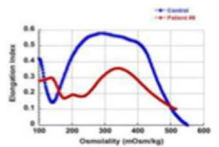
The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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Bogardus, H., Schulz, V. P., Maksimova, Y., Miller, B. A., Li, P., Forget, B. G., et al. (2014). Severe nondominant hereditary spherocytosis due to uniparental isodisomy at the SPTA1 locus. *Haematologica* 99, e168–e170. doi: 10.3324/haematol.2014.110312

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TABLE 1 | Genetic mutations and associated phenotype in HS due to SPTA1 mutations.

Phenotype	Patient	Allele 1	Allele 2	Age at time of report and comments	Ektacytometry	α -spectrin in RBC ghosts (% of control)
GROUP I (patients 1–4) Severe, recessive HS (transfusion-dependent, responding to splenectomy)	1	c.4339-99C > T	c.4295del (p.L1432*)	11 year-old, chronic transfusion requirement with partial response to partial splenectomy, resolved after total splenectomy		54%
	2	c.4339-99C > T	c.5102A > T (p.L1701*)	7 year-old, chronic transfusion requirement, improved with partial splenectomy		64%
	3	c.4339-99C > T	c.3267A > T (p.Y1089*)	11 year-old, not splenectomized due to family preference, continues to require frequent transfusions	Not evaluable in a transfused sample	
	4	Mutation not identified	Gross deletion of SPTA1	3.5 year-old, RT-PCR demonstrated significantly decreased α -spectrin expression; hemoglobin has normalized after recent splenectomy	Not evaluable in a transfused sample	
GROUP II (patients 5–8) Severe to moderately severe, recessive HS	5	c.4339-99C > T	c.1120C > T (p.R374*)	4 year-old, chronic transfusion requirement for first three years with improved pattern since.	Sample not provided after age 3, when transfusion-independent	
	6	c.4339-99C > T	c.1351-1G > T	7 year-old, occasional transfusion requirement, resolved after splenectomy at 5 years of age		59%
	7	c.4339-99C>T	c.2671C > T (p.R891*)	4 year-old, has not been transfused so far, Hgb 7.1–8.9 g/dL, ARC 420–572 x 10 ³ / μ l.		61%
	8	c.4339-99C > T	c.3257delT	8 year-old, transfused once as neonate, Hgb 10.6–11.8 g/dL, ARC 354–535 x 10 ³ / μ l; now Hgb 15–16 g/dL with normal ARC after splenectomy at 6 years of age (splenectomy performed because of chronic abdominal pain due to co-morbidities)		Not performed.
GROUP III (patients 9–11) Life-threatening anemia in utero leading to fatal <i>hydrops fetalis</i> if untreated (transfusion-dependent, not responding to splenectomy)	9	c.4206delG (fs)	c.4180delT (fs) in haplotype with c.6631C > T (p.R2211C)	Died at birth. Post-mortem diagnosis from parental studies and DNA extracted from liver tissue saved in paraffin block	N/A	
	10	c.6788+11C > T	c.6788+11C > T	11 year-old, born prematurely at EGA of 33 weeks with <i>hydrops fetalis</i> , remained transfusion-dependent even after splenectomy; now doing well after matched sibling transplant	Not evaluable in a transfused sample (required chronic transfusions up until bone marrow transplant)	26% (performed in CD71+ cells)
	11	c.6154del (p.Ala2052fs)	c.6154del (p.Ala2052fs)	2 year-old, severe in-utero anemia requiring five <i>in-utero</i> transfusions. Born with severe neonatal hyperbilirubinemia requiring exchange transfusion. Remains transfusion-dependent	Not evaluable in a transfused sample	

Of note, all the SPTA1 variants reported here except c.4339-99C > T (α^{LEPRA}) and c.2671C > T; p.R891* (Bogardus et al., 2014) have not been previously described.