

Listening to the umbilical cord: A call to rethink anemia management through placental evidence

Moses M. Obimbo^{1*}

1. Editor-in-Chief, JOGECA

*Correspondence: editor@jogeca.com

DOI: [10.59692/jf0b3776](https://doi.org/10.59692/jf0b3776)

Copyright © 2025, The authors. Published by JOGECA. This is an open access article under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted reuse, distribution, and reproduction in any medium provided the original author(s) and the source are properly cited.

There is untold story in every placenta, that within lies a biological record of pregnancy including signatures of maternal health, fetal adaptation, and the often-unseen consequences that arise when physiological demands outpace supply. Njoroge and colleagues in this issue present compelling evidence that early pregnancy anemia has potential to leave measurable, lasting imprints on the umbilical cord and I dare say the future human being. These findings challenge us to reconsider both the timing and the approach to managing anemia associated with pregnancy in clinical practice (1).

Gestational anemia affects over half of pregnant women in Kenya and many other countries in Africa (2). WHO classifies anaemia as a severe public health concern when its prevalence exceeds 40% (3). Yet despite decades of iron supplementation programs, antenatal screening programs, and public health campaigns, the needle has barely moved. Perhaps the reason lies not in the interventions themselves, but in their timing.

The study by Njoroge et al. examined umbilical cords from 36 women, comparing those diagnosed with anemia before 20 weeks gestation to those with normal hemoglobin levels (1). They reported significant differences in cord diameter, Wharton's jelly volume, and umbilical vein wall thickness between the two groups. These are not subtle histological findings; they may as well represent fundamental alterations in the very conduit that sustains fetal life.

The umbilical vein obtained from women with anemia showed intima-medial thickening. This is normally a compensatory response to altered blood flow dynamics (1). More concerning is the disruption and reduplication of the internal elastic lamina, particularly pronounced at the fetal end of the cord. These structural alterations closely parallel those observed in systemic vascular disease, such as atherosclerosis and diabetic

vasculopathy where sustained physiological stress drives adaptive remodeling that, over time, compromises vascular integrity and function.

Consider the implications. The umbilical vein delivers oxygen-rich, nutrient-laden blood from the placenta to the developing fetus. When its walls thicken and stiffen, compliance decreases. The vessel's capacity to regulate and accommodate fluctuations in blood flow becomes limited. Coupled with reduced Wharton's jelly volume, which normally cushions and protects umbilical vessels (4-5), and we have an anatomical explanation for why early anemia translates into adverse pregnancy outcomes (6-7).

The finding that these changes are more pronounced toward the fetal end of the cord is particularly noteworthy. The changes may be associated with a higher expression of vascular endothelial growth factor (VEGF) in this region, a compensatory response to hypoxia, (1). The fetus, most dependent on optimal umbilical blood flow, bears the greatest structural burden of maternal anemia.

Perhaps the most clinically relevant insight from this study concerns timing. The umbilical cord achieves its final structural form by the 12th week of gestation (4), the same critical window when anemia appears to exert its most profound effects. Previous research has demonstrated that restoration of hemoglobin levels after 20 weeks does not significantly improve pregnancy outcomes (6). The structural damage, it seems, has already been done. This finding has significant implications for how we conceptualize anemia management. Our current approach that involves screening at the first antenatal visit, which often is well into the second trimester, followed by oral iron supplementation may be intervening too late. By the time many women initiate treatment, the umbilical cord has already developed under some form of hypoxia, with structural adaptations that

are likely already established. This is akin to addressing a deficit whose imprint has already been biologically inscribed.

This study exemplifies an underutilized approach in obstetric medicine: examining placental and umbilical cord pathology to understand, predict, and ultimately prevent adverse outcomes (8-9). The placenta is the only organ that both mother and fetus contribute to building. It is also the only organ routinely discarded after fulfilling its purpose. Within its tissues lies retrospective evidence that could inform prospective care.

More immediately, placental findings can validate ultrasound-based screening tools. The structural changes Njoroge and colleagues identified histologically may have sonographic correlates detectable in real-time (1). If umbilical cord diameter and blood flow characteristics on Doppler assessment can predict which anemic women are at highest risk for adverse outcomes, we gain a powerful tool for risk stratification and targeted intervention especially with the advent of the AI guided point of care ultrasound.

My take from these findings is that anemia prevention must begin preconception. Women of reproductive age should receive nutritional counseling, screening, and treatment for anemia as

part of routine primary care (10). Second, when pregnancy is confirmed, early and proactive management of anemia is essential within the first 20 weeks (1,4). Third, we must fully recognize the placenta for what it is: a dynamic, information-rich organ and a powerful diagnostic resource. Rigorous, systematic placental examination correlating with clinical data and outcomes can generate the evidence needed to develop predictive models and guide targeted interventions. (8-9).

Njoroge et al. acknowledge key limitations, including a small sample size, lack of red cell indices to define anemia subtypes, and a retrospective design (1). These gaps highlight the need for prospective, larger-scale studies integrating early detailed full hemogram profiling, serial ultrasound imaging, and standardized placental examination to establish causality. Importantly, future research should also incorporate the vaginal microbiome, recognizing both the anaemia and vaginal microbiome as interconnected systems that can shape development of placenta and influence maternal-fetal health.

This study gives voice to that silent testimony of early signatures shaping the future outcomes. It is now our responsibility to listen - and to act, before the damage is inscribed in tissue.

References

1. Njoroge F, Olabu BO, Kigera JWM, Pulei AN. Umbilical cord structural changes in Kenyan women with anemia diagnosed within the first 20 weeks of pregnancy: A retrospective cohort study. *J Obstet Gynaecol East Cent Afr.* 2025;37(3):116-124. doi:10.59692/jogeca.v37i3.503
2. World Health Organization. Anaemia [Internet]. Geneva: WHO; 2023 [cited 2026 Mar 16]. Available from: <https://www.who.int/news-room/fact-sheets/detail/anaemia>
3. Tirote LL, Areba AS, Tamrat H, Habte A, Abame DE. Determinants of severity levels of anemia among pregnant women in Sub-Saharan Africa: multilevel analysis. *Front Glob Womens Health.* 2024;5:1367426. doi:10.3389/fgwh.2024.1367426
4. Heil JR, Bordoni B. Embryology, Umbilical Cord. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2026 Mar 16]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557490/>
5. Debebe SK, Cahill LS, Kingdom JC, Whitehead CL, Chandran AR, Parks WT, et al. Wharton's jelly area and its association with placental morphometry and pathology. *Placenta.* 2020;94:34-38. doi:10.1016/j.placenta.2020.03.008
6. Chen Y, Zhong T, Song X, Zhang S, Sun M, Liu X, et al. Maternal anaemia during early pregnancy and the risk of neonatal outcomes: a prospective cohort study in Central China. *BMJ Paediatr Open.* 2024;8(1):e001931. doi:10.1136/bmjpo-2023-001931
7. Figueiredo ACMG, Gomes-Filho IS, Silva RB, Pereira BLS, Mata FAFD, Lyrio AO, et al. Maternal anemia and low birth weight: A systematic review and meta-analysis. *Nutrients.* 2018;10(5):601. doi:10.3390/nu10050601
8. Khong TY, Mooney EE, Ariel I, Balmus NCM, Boyd TK, Brundler MA, et al. Sampling and definitions of placental lesions: Amsterdam Placental Workshop Group Consensus Statement. *Arch Pathol Lab Med.* 2016;140(7):698-713. doi:10.5858/arpa.2015-0225-CC
9. Redline RW. Four major patterns of placental injury: a stepwise guide for understanding and implementing the 2016 Amsterdam consensus. *Mod Pathol.* 2021;34(6):1074-1092. doi:10.1038/s41379-021-00747-4
10. Correa-Agudelo E, Kim HY, Musuka GN, Mukandavire Z, Miller FD, Tanser F, et al. The epidemiological landscape of anemia in women of reproductive age in sub-Saharan Africa. *Sci Rep.* 2021;11:11955. doi:10.1038/s41598-021-91198-z