# Red Cell Distribution Width as a Biomarker for Heart Failure: Still Not Ready for Prime-Time

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**Disclosure:** The authors have no conflicts of interest to declare.
**Citation:** *Cardiac Failure Review* 2019;5(3):180–1. **DOI:** https://doi.org/10.15420/cfr.2019.16.1
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Dear Editor,

We would like to thank García-Escobar and Ingelmo for their letter regarding our article on the use of biomarkers in routine clinical care for heart failure.[1,2] It was with interest that we read their comments on the use of red cell volume distribution width (RDW) as another biomarker for use in the management of patients with heart failure. As mentioned in their letter, RDW is a measure of heterogeneity of red blood cell (RBC) volume or anisocytosis and not a measure of RBC size or volume itself.[3] RDW is a number generated by automated blood count machines. Increased variations in RBC volume – that is, a high RDW, is seen in anaemic states (such as iron deficiency anaemia, sickle cell anaemia, thalassaemia and megaloblastic anaemia), in patients using chemotherapeutic agents, in cardiovascular disease, thyroid disease and myelodysplastic syndromes.[3]

There has been a considerable amount of research into the use of RDW to assess prognosis in various conditions, given that this variable is readily available. García-Escobar and Ingelmo have extensively reviewed this possibility in their letter. They have also analysed the various studies on RDW and heart failure. We agree that data support the use of RDW as a marker of prognosis in patients with heart failure, other cardiovascular diseases and stroke.[4–6] There are various mechanisms that may be involved that could explain how RDW predicts mortality. RDW is a surrogate for iron deficiency and other forms anaemia[7] that are known to be associated with a poorer prognosis in heart failure.[8] In addition to this, there are correlations between high RDW and inflammation, ineffective erythropoiesis, undernutrition and impaired renal function in patients with heart failure.[9] RDW is also a marker of hypoxaemia.[10] Patients who are sicker on presentation due to systemic inflammation, chronic anaemia, hypoxia and impaired renal function might therefore have a higher RDW due to the various processes coexisting alongside their heart failure. RDW is thus not a specific test for heart failure but is a non-specific surrogate for the various pathogenetic mechanisms that occur in heart failure or indeed any other chronic disease.[11–13] RDW therefore cannot be used for the diagnosis of heart failure and as yet there are no studies demonstrating its role in this capacity.

Similarly, there are limited data that can be used to assess the changes that occur in RDW as a result of treatment for heart failure[14] and pulmonary embolism.[15] The studies to date are small and the one on heart failure is retrospective. The lifespan of RBCs is 100–120 days and it would probably take this long for the RDW to show any significant change. Indeed, after the successful treatment of iron deficiency anaemia with iron supplements, studies have shown that it takes up to 3 months for the RDW to come down to reference levels.[16] Extrapolating this to patients with heart failure, a raised RDW might indicate a process that was prevalent up to 3 months prior to the patient's presentation, rather than reflecting an acute issue.

In conclusion, we would like to state that RDW is a marker of a chronic disease process and is not specific to heart failure. It appears to predict prognosis in patients with heart failure. Currently, there are no trials showing what specific cut-off values would reflect worse prognosis, we only have studies showing correlations of higher values (even within normal reference ranges) with worse prognosis. We cannot recommend what course of action a treating clinician should take when presented with a RDW value for a patient with heart failure as there are insufficient data. Therefore, although a great research tool, we cannot at present recommend the use of RDW in routine clinical practice. ■

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