ASSOCIATION OF RED BLOOD DISTRIBUTION WIDTH (RDW) WITH CAROTID ATHEROSCLEROSIS & DETERIORATION OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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OBJECTIVES

Red cell distribution width (RDW), a measure of erythrocyte size variability is routinely reported as part of complete blood count analysis & has traditionally played a role in the differential diagnosis of anemia. Recently it has been shown that higher RDW (anisocytosis) could be a novel predictor for cardiovascular events & progression of CKD. Therefore, we aimed to examine whether RDW was associated with carotid atherosclerosis & progression of CKD in patients with Diabetes Mellitus Type 2 (DMT2).

METHODS

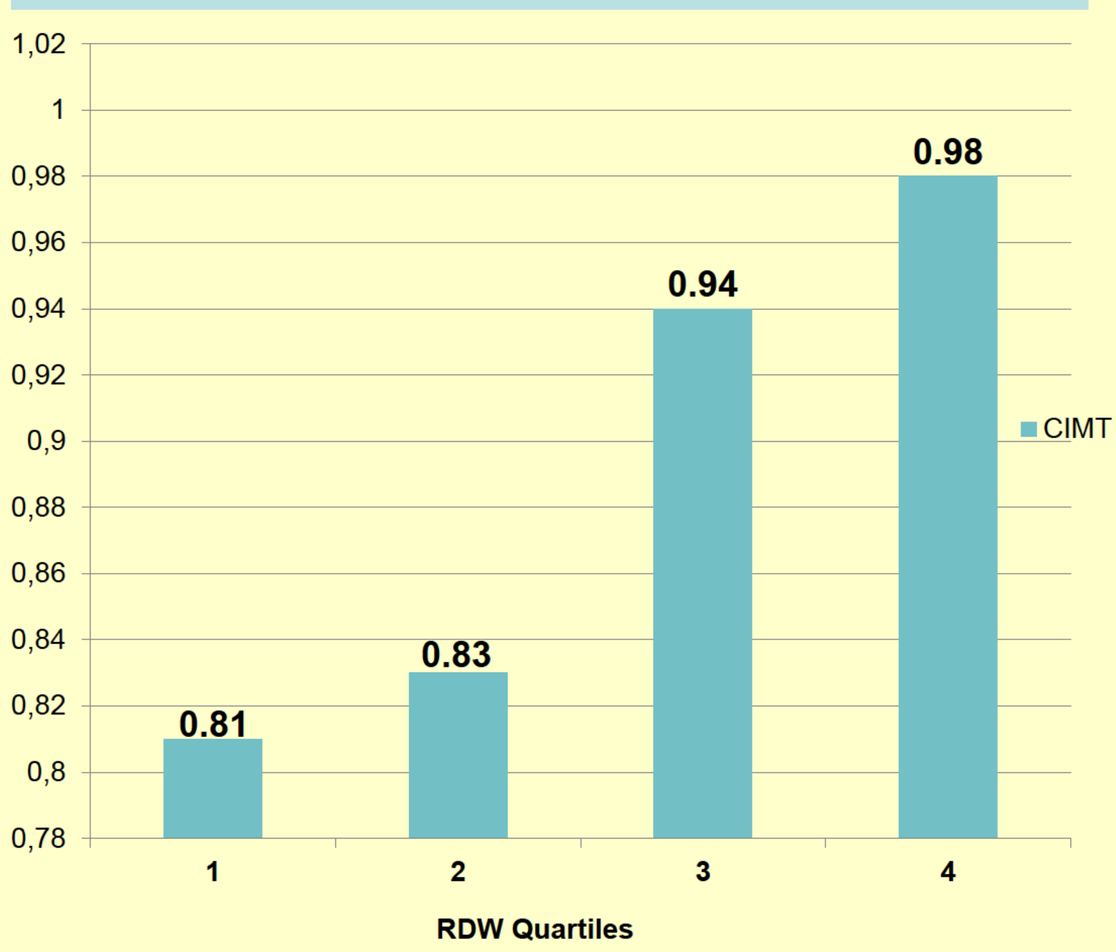
In this study we included 141 patients with DMT2 (76 male and 65 female), with mean age 68.2±8.9 years & 15.2±7.8 years mean duration of DMT2. RDW, proteinuria, albuminuria & estimated glomerular filtration rate (eGFR) were assessed. The sample population consisted of two groups:103 DMT2 patients with diabetic nephropathy, distributed in all five stages of CKD and 38 DMT2 patients as controls, having diabetes for more than10 years, persistent normoalbuminuria, eGFR above 60 mL/min and absence of diabetic retinopathy. All patients underwent Doppler ultrasound of the common carotid artery & Carotid Intima Media Thickness (CIMT) was determined. Stages of CKD were estimated using eGFR which was calculated via CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) formula.

TABLES AND FIGURES

TABLE 1: Anthropometric, clinical, and biochemical characteristics of patients at different stages of Diabetic Nephropathy.

	Stage of Diabetic Nephropathy					P[a]
N=141	**Controls (n=38)**	**1&2 (n=18)**	**3 (=31)**	**4 (n=30)**	**5-HD (n=24)**	
Age (years)	64.4 (8.0)	69.4 (8.5)	68.8 (7.8)	71.1 (9.1)	68.8 (10.4)	**0.02**
Gender (M/F)	16/22	10/8	16/15	22/8	12/12	0.14
SBP (mm Hg)	134.6 (18.0)	142.4 (11.9)	143.9 (15.8)	146.9 (16.1)	136.4 (23.2)	**0.03**
DBP (mm Hg)	76.1 (8.6)	80.7 (7.8)	80.2 (7.8)	81.1 (10.3)	79.1 (11.2)	0.18
Duration of T2DM (years)	11.7 (6.2)	14.2 (7.4)	16.0 (9.3)	18.5 (8.3)	16.5 (6.0)	**0.005**
RDW (%)	13.9 (12.3-20.8)	14.3 (12.5-18.2)	14.7 (13.2-18.9)	14.8 (13.0-21.5)	16.3 (12.0-19.8)	**0.002**
Albumin (g/dl)	4.4 (0.3)	4.3 (0.2)	4.2 (0.4)	3.8 (0.5)	4.1 (0.4)	**<0.0001**
HbA1c (%)	7.4 (0.9)	7.3 (0.9)	7.7 (1.3)	7.9 (1.4)	7.3 (0.9)	0.30
CRP (mg/dl)	0.10 (0.0-0.8)	0.16 (0.0-11.0)	0.20 (0.0-2.6)	0.40 (0.03-14.0)	1.05 (0.0-4.5)	**<0.0001**
UPCR	0.08 (0.01-0.29)	0.18 (0.02-1.1)	0.25 (0.01-3.73)	2.8 (0.03-9.7)	7.0 (5.0-9.0)	**<0.0001**
UACR (mg/g)	13.3 (1.0-29.0)	52.0 (2.4-401.0)	87.0 (3.0-2200.0)	965.0 (2.8-9700.0)	7000.0 (5000.0 - 9000.0)	**<0.0001**
Mean cIMT (mm)	0.76 (0.40-1.50)	0.86 (0.60-1.30)	0.90 (0.60-1.50)	0.80 (0.50-1.50)	1.00 (0.80-1.50)	**<0.0001**

Figure 1. Median CIMT values according to RDW Quartiles



RESULTS

A statistically significant positive correlation was revealed between RDW & hemoglobin (r=411, p<0.0001), while RDW values showed a significant inverse correlation with eGFR (r=-324, p<0.0001). Also RDW was significantly increased with progression of CKD stages (p<0.001, Kruskal-Wallis test: Stage 0-controls: 14.2±1.7, Stage 1+2: 14.6±1.8, Stage 3: 15.0±1.6, Stage 4: 15.4±1.9 & Stage 5: 16.0±2.0)-Table1. Similarly, CIMT showed a significant increase with progression of CKD stages (p=0.002, Kruskal-Wallis test)-Table 1. There was a significant positive correlation with both proteinuria & albuminuria (r=411, p<0.0001 and r=425, p<0.0001 respectively). Furthermore, RDW was significantly correlated with CIMT (r=206, p=0.003) -Figure 1.

CONCLUSIONS

RDW values were significantly increased with the progression of chronic diabetic nephropathy & were strongly correlated with CIMT in this group of patients. This study suggests that RDW could be a new beneficial predictor of carotid atherosclerosis & CKD progression in DMT2 patients. Even in absence of explanatory mechanism for this strong association, it seems justified to monitor RDW in this high risk population as it seems to reflect vascular calcification status and predict development of chronic renal damage.

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