The red cell distribution width (RDW): Value and role in preterm, IUGR (intrauterine growth restricted), full-term infants

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Objective: To measure the red cell distribution width (RDW) ranges at birth and to evaluate potential association with typical neonatal diseases: patent of the ductus arteriousus (PDA), bronchopulmonary dysplasia (BPD), and late-onset sepsis (LOS) mortality.

Methods: Forty-six full-term, 41 preterm, and 35 intrauterine growth restricted (IUGR) infants participated in this retrospective, observational study. RDW was measured before 3 days of life (T0) in all infants, and at first month of life (T1) in preterm/IURG patients.

Results: RDW% mean (standard deviation) at T0 was: 15.65 (1.18) in full-term newborns; 17.7 (2.06) in preterm; 17.45 (1.81) in IUGR. A negative correlation ($r = -0.51$; $P < 0.001$) between RDW and gestational age was found. RDW at T1 was: 17.25 (2.19) in the preterm group; 17.37 (2.56) in IUGR group. Fourteen preterm infants reported: 12 PDA, 5 LOS, 4 BPD, and 3 died; 10 IUGR infants had: 4 PDA, 6 LOS, 3 BPD, and 1 died.

RDW of IUGR infants suffering from those pathologies was not statistically different compared with unaffected infants, while preterm newborns with pathologies reported higher RDW: PDA vs. PDA absent: $P = 0.008$ at T0; $P < 0.002$ at T1. BPD vs. BPD absent: $P < 0.005$ at T1. LOS vs. LOS absent: $P < 0.005$ at T0.

RDW in preterm/IUGR population was associated with early mortality, T0: dead 21.2 (2.7) vs. alive 16.7 (1.7), $P < 0.0001$.

Conclusion: RDW and gestational age at birth were negatively correlated. High RDW resulted to be an indication of risk for critical newborns. This parameter can be inexpensively and routinely verified and further studies are required to confirm its prognostic role in neonatal pathologies.

Keywords: RDW, Newborn infants, Neonatal pathologies, Mortality

Introduction

The red cell distribution width (RDW) is a measure of heterogeneity in the size of circulating erythrocytes reflecting variability in cell sizes. Results are expressed as a percentage of the coefficient of variation of the red blood cell volume distribution and the adult reference range typically spans between 11 and 14%.[1–3] This parameter is automatically given by modern analyzers, together with the complete blood count (CBC), using a very low amount of blood and in a short period of time.[4] Therefore, this technology is suitable for critical neonates and even for very low birth weight infants (VLBW). RDW has been mainly used in the differential diagnosis of anemia, but has been recently demonstrated to have a role in adult patients as a prognostic marker in different pathologies, mainly for cardiovascular diseases.[2,3,5–8] It is known that neonatal normal laboratory values differ significantly from those of children and adults and our interest is focused on infant RDW. Lately, reference ranges are

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available for most of the neonatal CBC parameters, but there is only a recent article reporting RDW normal value in infants of different gestational ages.[9–12]

We aimed to measure RDW ranges at birth in full-term healthy infants, preterm, and intrauterine growth restricted infants (IUGR), and to evaluate if any prognostic association exists with RDW and the early outcome of critical neonates within the first month of life.

Methods

Patients

The study, after the approval of the Bioethics Committee of our Institute and after informed consent was obtained from both the parents, took place in our Neonatal and Neonatal Intensive Care Unit (NICU), we evaluated RDW in 46 healthy full-term infants, 41 preterm infants, and 35 IUGR infants. Healthy full-term newborns were defined by gestational age ≥37 weeks as elsewhere described, appropriate birth weight, and born after physiological pregnancy;[13] preterm infants were defined as infants born before the end of 36 weeks of gestation and IUGR infants were defined by a birth weight below the 10th percentile due to a deflection of the growth curve during pregnancy with consequent failure of the fetus to reach the growth potentiality.[14] Main exclusion criteria were congenital diseases, malformations, genetic syndromes, blood transfusion, and anemia during the study period.

RDW measurement

Neonates had blood collected for measurement of routine CBC and RDW was automatically calculated using the Cell-Dyn Sapphire (Abbott Diagnostic, Santa Clara, CA, USA), a multi-parameter automated hematology analyzer with MAPSS (multi-angle polarized scatter separation) technology plus 3 color fluorescence.[4] The results are expressed as percentage mean (standard deviation (SD)) of the measurement of size variability of the red blood cells. The timing of the samples was during one of the first 3 days of life (T0) and during the fourth week of life for the preterm and IUGR infants only (T1). Full-term newborns were not withdrawn for ethical reason, moreover, they were already discharged from the neonatal wards.

Patent of the ductus arteriousus, bronchopulmonary dysplasia, and sepsis diagnosis

A hemodynamically significant patent of the ductus arteriousus (PDA), was defined as failure of post-natal closure requiring pharmacological or surgical treatment for the closing, as reported elsewhere.[15] Bronchopulmonary dysplasia (BPD) was defined as oxygen dependence for at least 28 post-natal days and confirmed at 36 weeks of post-menstrual age, as reported by literature.[16] Late-onset sepsis (LOS) diagnosis was made according to the International Pediatric Sepsis Consensus Conference guidelines.[17] Blood cultures were processed by Bactec microbial detection system (Becton-Dickinson, Sparks, MD, USA).

Statistical analysis

The Shapiro–Wilk test was used for normal distribution of quantitative variables. When these were normally distributed, results were expressed as mean values and SD, otherwise median values and interquartile range were used (IQR; 25–75th percentile). Parametric or non-parametric tests were used to compare quantitative variables (*t*-test for independent samples/Mann–Whitney test, *t*-test for dependent samples/Wilcoxon test, analysis of variance (ANOVA)/Kruskall–Wallis test, and ANOVA for repeated measures/Friedman test). Correlations were evaluated by means Pearson's coefficient. Chi-squared statistics or Fisher's exact test were applied to compare qualitative variables; $P < 0.05$ was considered statistically significant. All tests were two-sided. Data analysis was performed using STATA statistical package (version 12; Stata Corporation, College Station, 2011, Texas, USA).

Results

Descriptive data of the studied groups are reported in Table 1.

RDW% mean (SD) at T0, in the healthy full-term newborns was 15.65 (1.18); in the preterm group it was 17.7 (2.06) and in the IUGR group it was 17.45 (1.81). The preterm and IUGR groups showed a statistical difference in comparison with healthy full-term infants ($P < 0.001$, both the groups). No statistical difference was reported between the preterm and IUGR infants. A negative correlation was found between RDW at T0 and gestational age at birth (for all the 122 infants: $r = -0.51$; $P < 0.001$), Fig. 1.

RDW% mean (SD) at T1, in the preterm group was 17.25 (2.19) and in the IUGR group was 17.37 (2.56), without a statistical difference (Table 1), whereas RDW values were similar when comparing pattern inside group (preterm T0/T1: $P = 0.418$; IUGR T0/T1: $P = 0.399$).

In the preterm group, 14 infants (34%) were suffering from typical neonatal pathologies: 12 PDA, 4 BPD, 5 LOS, and 3 infants died; while in the IUGR group, 10 infants (28%) reported: 4 PDA, 3 BPD, 6 LOS, and 1 infant died.

In the IUGR population RDW, at T0 and T1, in infants suffering from PDA, BDP, and LOS was not

Table 1 Descriptive data

	Preterm infants *n* = 41	IUGR infants *n* = 35	Healthy full-term infants *n* = 46	*P* value		
				Preterm vs. IUGR	Healthy full-term vs. IUGR	Healthy full-term vs. preterm
Maternal age; years	32.02 (6.09)	33.57 (5.18)	31.59 (4.85)	0.644	0.310	0.989
Gestational age; weeks	30.56 (3.37)	33.03 (3.29)	39.3 (1.17)	<0.001	<0.001	<0.001
Hospital stay; days, median (IQR)	34 (23–54)	30 (14–53)	3 (3–4)	0.020	<0.001	<0.001
Weight at birth; g	1413.44 (563.34)	1357.43 (439.04)	3245.65 (567.24)	0.998	<0.001	<0.001
Weight at month of age; g	1998.61 (708.35)	1933.91 (631.86)	4095.22 (555.6)	0.998	<0.001	<0.001
Sex; *n* of male (%)	24 (58.4)	16 (45.7)	23 (50)	0.264	0.803	0.425
Apgar 1 minute	5.85 (2.63)	7.37 (2.04)	9.89 (0.31)	0.002	<0.001	<0.001
Apgar 5 minute	5.2 (3.94)	8.97 (1.54)	10 (0)	<0.001	0.185	<0.001
RBC $\times 10^6$/μl at T0	4.24 (0.58)	4.49 (0.86)	4.73 (0.62)	0.341	0.352	=0.003
Hgb g/dl at T0	15.96 (2.18)	17.39 (3.15)	16.19 (2.52)	0.062	0.132	0.998
RDW% at T0	17.7 (2.06)	17.45 (1.81)	15.65 (1.18)	0.998	<0.001	<0.001
RBC $\times 10^6$/μl at T1	3.5 (0.61)	3.72 (0.92)	n/a	0.468	n/a	n/a
Hgb g/dl at T1	11.68 (2.21)	12.49 (3)	n/a	0.293	n/a	n/a
RDW% at T1	17.25 (2.19)	17.37 (2.56)	n/a	0.285	n/a	n/a
PDA, *n* (%)	12 (29.3)	4 (11.4)	n/a	0.057	n/a	n/a
BPD, *n* (%)	4 (9.8)	3 (8.6)	n/a	0.859	n/a	n/a
LOS, *n* (%)	5 (12.2)	6 (17.1)	n/a	0.541	n/a	n/a
Exitus, *n* (%)	3 (7.3)	1 (2.9)	n/a	0.385	n/a	n/a

Data are expressed as means and SD, if not otherwise specified.
n/a, not attributable.

statistically different compared with RDW of unaffected infants (Table 2), while in the preterm population, infants suffering from PDA, BDP, and LOS reported statistically higher RDW values: PDA vs. PDA absent: $P = 0.008$ at T0 and $P < 0.002$ at T1. BPD vs. BPD absent: $P < 0.005$ at T1. LOS vs. LOS absent: $P < 0.003$ at T0 (Table 2).

Four infants in the whole group (preterm and IUGR) died during the first 2 weeks of the study and RDW was strongly associated with mortality at T0 (dead infants vs. alive: 21.2 (2.7) vs. 16.7 (1.7), $P < 0.0001$).

Discussion

Reference intervals of laboratory values during the neonatal period are different from those of adult and children and they change considerably with prenatal condition, in particular prematurity is a significant variable. Thus, specific ranges are important for neonatal monitoring and diagnosis, on the other hand it is difficult to obtain samples from neonates because of ethical implications and for the paucity of available biological samples. Measures of RDW do not require extra samples and this parameter is routinely given with CBC, since CBC itself is frequently required for clinical management.

Attention for RDW in neonatal population dates back to late 80 seconds. Literature about red cells ontogeny described an increase of RDW% mean (SD) from the fetal period to early post-natal life: in

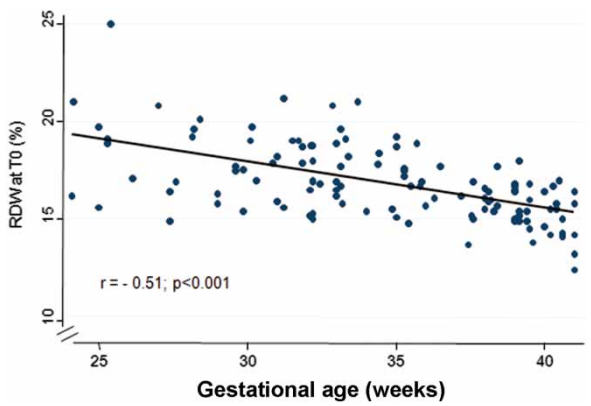

Figure 1 Correlation between RDW values and gestational age at birth in all the studied infants.

Table 2 Mean (SD) of RDW at T0 and T1 in the preterm and IUGR infants

T0	Preterm infants *n* = 41	*P*	IUGR infants *n* = 35	*P*
PDA	19.0 (2.2)		18.2 (2.4)	
PDA absent	17.1 (1.7)	$P = 0.008$	17.3 (1.7)	$P = 0.3996$
BPD	18.8 (0.9)		17.2 (1.9)	
BPD absent	17.6 (2.1)	$P = 0.277$	17.5 (1.8)	$P = 0.8349$
LOS	20.5 (3.2)		16.9 (0.8)	
LOS absent	17.4 (1.7)	$P = 0.003$	17.5 (1.9)	$P = 0.4687$
T1				
PDA	19.1 (2.1)		16.8 (1.3)	
PDA absent	16.6 (1.8 1)	$P = 0.002$	17.4 (2.7)	$P = 0.6921$
BPD	20.1 (2.0)		16.8 (1.3)	
BPD absent	16.9 (2.0)	$P = 0.005$	17.4 (2.7)	$P = 0.6921$
LOS	17.9 (1.2)		16.3 (1.0)	
LOS absent	17.2 (2.3)	$P = 0.620$	17.7 (2.8)	$P = 0.2487$

n/a, not attributable.

fetuses at 18–24 weeks of gestation it was 17.9 (1.2) and 20.5 (1.8) at term birth.[12] While it is known that during gestation red cells of fetuses are less uniform than those of children or adults, the largest RDW of the full-term infants was less expected. The authors therefore speculated that at birth erythropoiesis is not in a steady-state condition and produces smaller erythrocytes than fetal ones. The above results are partially different with respect to newest literature, possibly due to the small sample size (19 fetuses and 14 newborns) together with the availability in the past years of less sophisticated technology. Other authors further evaluated VLBW infant's red cells count and RDW in relation of gestational age, birth weight, sex, race and concluded that RDW values are affected by gestational age and race together with hemoglobin and hematocrit parameters.[18] A recent study, dealing with a large number of infants of different gestational age, confirmed that gestational period affects RDW. RDW% mean (SD) values ranged from 17.86 (2.23) at 32–34 weeks of gestational age to 16.65 (1.81) at 37–42 weeks.[11] These results are in agreement with ours, because we found an important negative correlation between RDW and gestational age of the neonatal population ($r = -0.51$; $P < 0.001$, Fig. 1). Considering the three groups individually, the highest RDW was found in the preterm newborns, followed by IUGR infants, and therein healthy full-term infants. RDW of all the neonates resulted however to be higher than RDW of children and adults comparing our results with literature.[2] Therefore, we may speculate that an instable erythropoiesis and/or stress condition is reflected in the higher RDW values of neonates at birth, in particular in the critical newborns. In these infants, RDW is still high at 1 month of age. Because at the moment we have a too short follow-up, we have no information about a possible RDW normalization with infant's age or path to recovery and to our knowledge there are no clinical investigations about neonatal RDW role, a part from the anemia diagnosis. [2,3] We are aware about a study in children undergoing surgery for cardiac hearth failure demonstrating that RDW is a prognostic preoperative predictor: the risk of post-operative death was five times higher with a RDW of 16% or more.[19] This predictor resulted especially important in non-anemic patients, for whom higher RDW clearly reflected underlying inflammatory stress.[2,19] We consequently excluded anemic patients from our study to evaluate a potential correlation between RDW and neonatal pathologies, like PDA, BPD, LOS, and neonatal mortality.

Hemodynamically significant PDA is an important complication in preterm infants, with a reported incidence of 28% for gestational age of <32 weeks and up to 60–70% for gestational age of <29 weeks.[15,20] In our study, PDA was always associated with higher RDW and in the preterm infants this increase was statistically significant, both at T0 and T1, when medical or surgical intervention was required. Among factors affecting the etiopathology and maintenance of PDA, pro-inflammatory molecules are involved.[21] An hypothesis could be that high RDW levels are correlated to a general inflammatory state of the critical preterm infants, that can affect erythropoiesis for a long period.[12] BPD is a chronic lung disease of prematurely born infants with a respiratory distress syndrome requiring mechanical ventilation.[16] Preterm newborns, who developed BPD, presented a statistically higher RDW at 1 month of life. This pathology is confirmed as oxygen dependence at 36 weeks of neonatal post-menstrual age and predisposing factors include prolonged exposure to high airway pressures and high inspired oxygen concentrations, altered lung angiogenesis, presence of infection, and/or inflammation. Therefore, we may try to formulate the hypothesis that possible prognostic RDW values for BPD are those around 1 month of post-natal life, when predisposing factors for BPD start to have an influence in the development of the pathology. On the other hand, for the preterm infants, who suffered from LOS, RDW was significantly higher at T0. In fact, all the affected babies had the infection within the first 3 weeks of life (data not reported). Morbidity and mortality due to LOS is still high in NICU, especially among VLBW infants, and an accurate stratification of infants for the risk of LOS is important, thus altered RDW may be included among predisposing factors.[22,23] We also found an important association with higher RDW and neonatal mortality, in preterm and IUGR groups. Because of the sample size, we cannot establish a prognostic role of RDW in neonatal pathologies and mortality, but we can suggest considering this parameter as a further indication of risk, that can easily monitored by a routine CBC control.

While an increasing amount of scientific papers demonstrate the association between RDW and negative outcome for adult patients and children, this is the first study indicating an analogous situation in neonatal patients,[2,3,5–8,24–26] even if we are not able to explain in detail the underlining mechanism that makes RDW such a promising marker for critically ill neonates. If we had the possibility to resume absolute RDW measure as the SD in fL, or if we had the mean corpuscolar volume (MCV) and reticulocytes values available, we could have determined if these parameters play a role in the increased RDW. In fact, neonates are known to have physiological reticulocytosis, which translates in higher MCV and because RDW% is expressed relative to MCV, therefore we cannot exclude that this consecutive connection

could possibly influence the RDW% itself. Unfortunately, being a retrospective study, these data are not available. On the other hand, we may say that we compared a homogenous situation because we studied all newborn infants who are characterized, in particular in the early post-natal period, by high reticulocyte count.[27,28] Furthermore, a quality note of our study is that all the samples were analyzed by the same instrument during the whole study period and for all the infants. In fact, we know that considerable differences in RDW may exist between various hematology analyzers.[29] As reported, we are conscious of the limitations of our study nevertheless, this could be a starting point for a wider investigation. Thus, the 'take home message' we would like to share, is that RDW is a simple and inexpensive measure, automatically given by the modern analyzers, with high level of precision, and it could be a future prognostic tool or an additional biomarker for monitoring critically ill newborns, in particular preterm infants. Further studies are required to confirm the role of RDW as predictive marker in neonatal clinical care.

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References

1 Lappe JM, Horne BD, Shah SH, May HT, Muhlestein JB, Lappe DL, *et al.* Red cell distribution width, C-reactive protein, the complete blood count, and mortality in patients with coronary disease and a normal comparison population. Clin Chim Acta 2011;412(23–24):2094–9
2 Montagnana M, Cervellin G, Meschi T, Lippi G. The role of red blood cell distribution width in cardiovascular and thrombotic disorders. Clin Chem Lab Med. 2012;50(4):635–41.
3 Patel KV, Semba RD, Ferrucci L, Newman AB, Fried LP, Wallace RB, *et al.* Red cell distribution width and mortality in older adults: a meta-analysis. J Gerontol A Biol Sci Med Sci. 2010;65(3):258–65.
4 Costa O, Van Moer G, Jochmans K, Jonckheer J, Damiaens S, De Waele M. Reference values for new red blood cell and platelet parameters on the Abbott Diagnostics Cell-Dyn Sapphire. Clin Chem Lab Med. 2012;50(5):967–9.
5 Felker GM, Allen LA, Pocock SJ, Shaw LK, McMurray JJ, Pfeffer MA, *et al.* Red cell distribution width as a novel prognostic marker in heart failure: data from the CHARM Program and the Duke Databank. J Am Coll Cardiol. 2007;50(1):40–7.
6 Hong N, Oh J, Kang SM, Kim SY, Won H, Youn JC, *et al.* Red blood cell distribution width predicts early mortality in patients with acute dyspnea. Clin Chim Acta 2012;413(11–12):992–7.
7 Oh J, Kang SM, Won H, Hong N, Kim SY, Park S, *et al.* Prognostic value of change in red cell distribution width 1 month after discharge in acute decompensated heart failure patients. Circ J. 2012;76(1):109–16.
8 Patel KV, Ferrucci L, Ershler WB, Longo DL, Guralnik JM. Red blood cell distribution width and the risk of death in middle-aged and older adults. Arch Intern Med. 2009;169(5): 515–23.
9 Christensen RD, Del Vecchio A, Henry E. Expected erythrocyte, platelet and neutrophil values for term and preterm neonates. J Matern Fetal Neonatal Med. 2012;25(Suppl 5) :77–9.
10 Christensen RD, Henry E, Jopling J, Wiedmeier SE. The CBC: reference ranges for neonates. Semin Perinatol. 2009; 33(1):3–11.
11 Tonbul A, Tayman C, Catal F, Kara S, Tatli MM. Red cell distribution width (RDW) in the newborn: normative data. J Clin Lab Anal. 2011;25(6):422–5.
12 Alter BP, Goldberg JD, Berkowitz RL. Red cell size heterogeneity during ontogeny. Am J Pediatr Hematol Oncol. 1988; 10(4):279–82.
13 Myatt L, Eschenbach DA, Lye SJ, Mesiano S, Murtha AP, Williams SM, *et al.* A standardized template for clinical studies in preterm birth. Reprod Sci. 2012;19(5):474–82.
14 Zhang J, Merialdi M, Platt LD, Kramer MS. Defining normal and abnormal fetal growth: promises and challenges. Am J Obstet Gynecol. 2010;202(6):522–8
15 Hamrick SE, Hansmann G. Patent ductus arteriosus of the preterm infant. Pediatrics 2010;125(5):1020–30.
16 Borghesi A, Massa M, Campanelli R, Bollani L, Tzialla C, Figar TA, *et al.* Circulating endothelial progenitor cells in preterm infants with bronchopulmonary dysplasia. Am J Respir Crit Care Med. 2009;180(6):540–6.
17 Goldstein B, Giroir B, Randolph A. International pediatric sepsis consensus conference: definitions for sepsis and organ dysfunction in pediatrics. Pediatr Crit Care Med. 2005;6(1):2–8.
18 Alur P, Devapatla SS, Super DM, Danish E, Stern T, Inagandla R, *et al.* Impact of race and gestational age on red blood cell indices in very low birth weight infants. Pediatrics. 2000;106(2 Pt 1):306–10.
19 Massin MM. Relation between red cell distribution width and clinical outcome after surgery for congenital heart disease in children. Pediatr Cardiol. 2012;33(7):1021–5.
20 van der Lugt NM, Lopriore E, Bokenkamp R, Smits-Wintjens VE, Steggerda SJ, Walther FJ. Repeated courses of ibuprofen are effective in closure of a patent ductus arteriosus. Eur J Pediatr. 2012;171(11):1673–7.
21 Majed BH, Khalil RA. Molecular mechanisms regulating the vascular prostacyclin pathways and their adaptation during pregnancy and in the newborn. Pharmacol Rev. 2012;64(3):540–82.
22 Ng PC, Lam HS. Biomarkers for late-onset neonatal sepsis: cytokines and beyond. Clin Perinatol. 2010;37(3):599–610.
23 Vergnano S, Menson E, Kennea N, Embleton N, Russell AB, Watts T, *et al.* Neonatal infections in England: the NeonIN surveillance network. Arch Dis Child Fetal Neonatal Ed. 2011; 96(1):F9–14.
24 Fujita B, Strodthoff D, Fritzenwanger M, Pfeil A, Ferrari M, Goebel B, *et al.* Altered red blood cell distribution width in overweight adolescents and its association with markers of inflammation. Pediatr Obes. 2013;8(5):385–91.
25 Majercik S, Fox J, Knight S, Horne BD. Red cell distribution width is predictive of mortality in trauma patients. J Trauma Acute Care Surg. 2013;74(4):1021–6.
26 Seretis C, Seretis F, Lagoudianakis E, Gemenetzis G, Salemis NS. Is red cell distribution width a novel biomarker of breast cancer activity? Data from a pilot study. J Clin Med Res. 2013; 5(2):121–6.
27 Piva E, Brugnara C, Chiandetti L, Plebani M. Automated reticulocyte counting: state of the art and clinical applications in the evaluation of erythropoiesis. Clin Chem Lab Med. 2010; 48(10):1369–80.
28 Brugnara C, Platt OS. The neonatal erytrocyte and its disorders. In: Orkin SH, Nathan DG, Ginsburg D, Look AT, Fisher DE, Lux SE, editors. Nathan and Oski's Hematology of Infancy and Childhood. 7th edn. Philadelphia, PA: Saunders by Elselvier; 2008. pp. 21–66.
29 Hoffmann JJ. Red cell distribution width and mortality risk. Clin Chim Acta 2012;413(7–8):824–5.