Biochemical and anthropometric correlates of bio-electrical impedance parameters in severely malnourished children: A cross-sectional study

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List of abbreviations

Bio-electrical impedance - BI

Bio-electrical impedance vector analysis - BIVA

Body composition - BC

Phase angle - PA

Reactance - Xc

Resistance - R

Severe acute malnutrition - SAM

Introduction

Bio-electrical impedance (BI) techniques are non-invasive, safe and easy to use compared with other methods for body composition (BC) assessment (1). The conventional approach assumes that height-adjusted impedance is proportional to total body water, which can be used to calculate lean and fat mass. However, the clinical validity of this approach is compromised in individuals characterised by abnormal fluid status and/or body disproportion. For instance, in children with severe acute malnutrition (SAM), oedema greatly reduces the accuracy of BI estimates (2). To circumvent the challenges in sick individuals, the use of raw BI values alone or in combination (vector analysis or BIVA), has been suggested (3).

In adult patients with chronic cardiac failure phase angle (PA) was associated with lower haemoglobin, poor cardiac function and renal failure; more importantly, those with PA <4.4 degree were at higher risk of death (4). Values of reactance (Xc) and PA are presumed to indicate "cellular health" (5), which in itself is poorly defined. Similarly, patients with oedema show increments in resistance (R) in association with loss of oedema (6). Based on a review of the literature regarding the clinical relevance and applicability of raw BI values, PA was identified as a prognostic marker while BIVA has been recommended as a screening tool to identify patients with impaired functional status (7).

The clinical usefulness of the innovate approaches that can potentially replace the assumption-dependent and error-prone equations is well documented, though data is still scare in clinical nutrition in childhood (8). However, evidence is lacking regarding the biological correlates of BI parameters in sick individuals. Consequently, it is difficult to interpret cross-sectional or serial BI data, and this hinders utilization of the innovative methods. In this study, we examined the relationship between BI parameters and markers of electrolyte homeostasis, liver function and inflammation, and anthropometry in children hospitalized with SAM. These parameters are commonly used, combined with clinical examination, to assess disease progress, treatment outcome or identify patients at high risk of death early.

Methods

Study setting and subjects

The study was conducted in the Nutrition Rehabilitation Unit (NRU) of Jimma University Specialized Hospital, Ethiopia, from November 20010 to September 2011. The authors have previously published data from this site (2). Eligible children were those 6-60 months old admitted with SAM, defined as MUAC <11·0 cm or weight-for-height (WFH) <70% of NCHS growth reference median and/or nutritional oedema. Children with life-threatening illness like shock or sever respiratory distress or who were readmitted with SAM were excluded.

Data collection

Children were weighed with minimal clothing to the nearest 10 g using a pediatric scale (Tanita BD 815 MA, Tokyo, Japan). For children less than 2 years or those not able to stand, length was measured to the nearest 0·1 cm using a length board (SECA 416, Hamburg, Germany). In children older than 2 years of age, 0·5 cm was subtracted if they were not able to stand. In older children, height was measured to the nearest 0·1 cm using a free-standing stadiometer (SECA 214, Hamburg, Germany). MUAC was measured to the nearest 0·1 cm using a strip (SECA 2012, Hamburg, Germany). Pitting oedema was checked by gentle pressure with the thumb on the feet for 3-5 seconds.

BI parameters i.e. impedance in Ohm, R in Ohm, Xc in Ohm and PAin degrees were measured at 50 kHz using a Quadscan 4000 analyser (Bodystat, UK) as described previously (9). It emitted 200 Micro Amps root mean square of alternating current. In brief, self-adhesive disposable electrodes were attached at the right hand and foot, injecting leads were connected to the electrodes just behind the finger and toe and the measuring leads were then connected to the electrodes on the right wrist and right ankle. Measurements were taken in triplicate 5 minutes apart, with children supine and limbs abducted.

Healthy children of 6-60 months old (WFH and height-for-age within ± 2SD of WHO growth standard) were recruited among vaccination attendees and children in day-care centre. The BI parameters in this group were measured with both equipment and protocol as for the SAM children. Two research nurses collected the data.

In the patients, venous blood was collected into dry tube and EDTA tube for separation of serum and hemoglobin determination, respectively. Serum was separated within an hour and kept at -80°C for later blood chemistry analysis. Serum phosphate (P), Ca, Mg, Na, K and Cl were

measured (Abbott Diagnostics, ref no 2P32-11 and 2P32-50) at International Clinical Laboratory (ISO 15189, Testing Laboratory No. M0221). Architect C4000 system (Abbott Diagnostics, USA) was used to measure P (Abbott Diagnostics, ref no 7D71-20&7D70-30), Mg (Randox Laboratories, UK, ref no MG531), Ca (Abbott Diagnostics, Ref no 3L79-31), albumin (Abbott Diagnostics, ref no 7D53-20) and bilirubin (Abbott Diagnostics, ref no 6L45-20 and 6L45-40) in serum. An automated Humastar 80 analyser (Human Diagnostics, Wiesbaden, Germany) was used to measure α_1-acid glucoprotein (AGP) (code Q0326, DAKO Denmark A/S, Glostrup, Denmark) and alkaline phosphatase (Human diagnostics ref no 12117) in serum. Hemoglobin was measured from whole blood samples collected in EDTA tubes using HemoCue® (Hb 201+, Ängelholm, Sweden).

Reference ranges for the age group of children in the study provided by the laboratories were: P (1.1–2.0 mmol/L), Ca (2.2-2.7 mmol/L), Mg (0.70-0.95 mmol/L), albumin (38-54 g/L), Na (138-145 mmol/L) , K (3.4-4.7 mmol/L), Cl (98-113 mmol/) and alkaline phosphatase (<400 U/L). The reference value for AGP (0.5-1.2 g/L) was not age-specific.

Written informed consent was obtained from caretakers or parents. Research Ethical Review Committee of Jimma University approved the study. All measurements were done after commencement of standard management and within 24 hours of admission.

Statistics and data handling

Data were double-entered into EpiData version 3·1 (EpiData Association, Odense, Denmark) and analyzed with Stata/IC 12·1 (StataCorp, Texas, USA). WHO growth standard based anthropometric z-scores were calculated using Stata. R and Xc were indexed to height by division (R/H and Xc/H respectively). Continuous data were presented as mean ± standard deviation; categorical data were presented as n (%). Two-sample t-tests and chi-square test were used to compare SAM and healthy children. Correlation between continuous independent and dependent variables was assessed using Pearson's correlation coefficient.

The dependent variables were R/H and Xc/H, and unadjusted PA. Results of Z and R were similar and hence only results for R were shown. The following covariates were included in the final model: anthropometric indices, age, sex, MUAC, and serum AGP, Na, K, P, Ca, Mg, Cl, bilirubin, albumin, alkaline phosphatase and haemoglobin. To test if the relationship of serum albumin and BI varies with oedema, 2-way

interaction term (albumin## oedema) was included in the model. Variance inflation factors to check for multi-collinearity between independent variables. Final models were established using forward selection after comparing models by likelihood ratio test.

Results

Of 55 children with SAM, the mean±SD age was 36±24 month, and 60% were males and 72.7% had nutritional oedema (**Table 1**), and the 80 healthy reference children had mean±SD age of 28±15 month and 47.5 % were males. The reference children were younger than the SAM children. Oedematous children were older ($p=0.04$) and heavier ($p<0.001$) than the non-oedematous children. Moreover, the oedematous children were less stunted than the non-oedematous children ($p=0.01$).

The children with SAM had lower Xc and PA ($p<0.001$) than the healthy children, whereas R was comparable between the two groups (Table 2). In children with SAM, presence of oedema was associated with lower R and Xc ($p<0.01$) but not with PA.

As is shown in Table 3**,** oedematous children had lower serum albumin, K and alkaline phosphatase than non-oedematous children ($p<0.02$). However, serum phosphorus, calcium, magnesium, sodium, chloride, haemoglobin and AGP did not differ by oedema. R was negatively correlated with age, oedema, MUAC, HAZ, WAZ and WHZ while Xc was negatively correlated with age, oedema, HAZ and WAZ (Table 4). PA was positively correlated only with MUAC ($p<0.05$).

Among serum biochemical markers presented in Tables 5, only serum albumin correlated with both R and Xc. Haemoglobin and phosphate levels correlated only with Xc. Of note, PA correlated with none of these markers.

Table 6 provides the independent predictors of bio-electrical impedance among children with SAM. After adjustment for all the covariates oedematous children had lower R (B = -486, 95%CI-888, -85), Xc (B = -29.3, 95%CI -41.1, -17.6) and PA (B= -0.86, 95%CI -1.3, -0.42) compared with non-oedematous children with SAM. Additionally, R decreased with WHZ (B = -125.6, 95%CI -184.2, -67.0) but increased with albumin level (Coeff. = 16, 95%CI 7, 25). Despite the mean serum albumin in oedematous children being only half that of non-oedematous SAM children, no interaction was found between albumin and oedema for all dependent variables (data not shown). Xc was higher with increasing calcium (B = 18.5, 95%CI 3.6, 33.5) and Chloride (B = 1.1, 95%CI 0.2, 1.9). PA increased with MUAC (B = 0.26, 95%CI 0.13, 0.38).

Discussion

This study provides new data on the association of BI parameters with anthropometric and biochemical markers of nutritional status. All three BI parameters (R, Xc and PA) were divergent between SAM children and healthy children. Among SAM children nutritional oedema was an independent factor for the divergence in R and Xc. We found that R decreased with WHZ and PA increased with MUAC. In contrast, Xc did not relate with physical parameters but increased with Ca and Cl levels. All the three BI parameters were lower in the presence of oedema. Of note, serum albumin did not influence the relationship between R and oedema.

Unlike conventional biochemical markers of nutritional status, BI is a blend of three highly interrelated components (R, Xc and PA). Though the physical basis of variability in BI parameters requires further theoretical development, particularly in disease states, the components are presumed to be influenced by, and hence reflect variability in, differing biological aspects.

In this study R/H correlated with oedema, WHZ and serum albumin level. Because fat tissue behaves as a non-conductor, electrical current traverses the body against the resistance by lean tissue (9). In lean tissue, resistance is inversely related to its fraction of hydration and the concentration of electrolytes in the fluid (10). Thus, difference in lean tissue mass (healthy>non-oedematous SAM) and lean mass hydration (oedematous SAM>healthy>non-oedematous) can explain the indirect relationship of R with both oedema and WHZ (11,12). There are no published data on the composition of nutritional oedema. But the fact that albumin level had direct correlation with R may reflect the contribution of albumin to plasma colloid and osmotic pressure (13).

Interestingly, no correlation was found between R and both serum Na and Cl; the two main determinants of serum osmolality. Moreover, serum albumin did not influence the relationship of oedema with R. These finding demonstrate the complexity of the mechanism of nutritional oedema (14,15).

Based on physio-electrical principles, Xc is considered to be influenced by integrity of the cell, in particular the membrane. In this study the healthy children had the highest Xc and oedematous SAM children the lowest. This pattern may demonstrate that cellular integrity is perturbed in SAM, but is also worse in oedematous SAM. Among children with critical illness, Xc/H was better than R/H in predicting evolution for septic shock and organ dysfunction (16). Of note, there is evidence relating cell membrane abnormality and leakage with nutritional oedema (17). The

stronger relationship of Xc with biochemical parameters than with anthropometric parameters substantiates that Xc reflects physiological more than physical abnormalities (18). In Bangladeshi children with SAM, case fatality was three fold higher in children with hypocalcaemia (19). In severe diseases, chloride is considered to play a role beyond plasma tonicity (20); its serum level in critically sick patients is correlated with survival (21). Albumin has strong inverse relationship with severity of illness, but we did not find any correlation between albumin level and Xc (22).

Generally, serum alanine aminotransferase, bilirubin, alkaline phosphatase and albumin are taken as markers of liver damage or dysfunction (23). In this study, the mean values of albumin and alkaline phosphatase were abnormal and also differed by oedema. Though fatty liver is frequently reported in SAM patients (24), there are mixed result regarding its markers. In malnourished pig models, serum alanine aminotransferase and bilirubin were increased, and liver showed evidence of increased triglyceride content (25). However, serum albumin was increased in the same pigs. In adults with chronic hepatitis C, phase angle was positively correlated with lean body mass while it was negatively correlated with serum high density lipoprotein (26). But only lean body mass was related when adjusted for age and sex, which was similar with our finding of positive correlation between PA and MUAC.

The inflammatory marker serum AGP was found to be equally elevated in the oedematous and non-oedematous SAM children. In this study children were enrolled soon after admission, and hence the elevated inflammatory markers may indicate ongoing inflammatory process which in most SAM children can be presumed to be related to infection. The small sample size, exclusion of severely ill children and absence of systematic assessment for infection were major limitation of this study. Moreover, it would have been invaluable to evaluate the short-term longitudinal relationship between the BI parameters and the other nutritional markers.

This study demonstrated that R and PA correlated strongly with anthropometric parameters irrespective of the biochemical profile whereas Xc had strong relation with physiological markers. Normally serum albumin is used as marker of nutritional status as well as prognostic indicator in severs illnesses. Thus, its positive association with R indicates that in children with SAM (with or without oedema), R can be used to monitor nutritional recovery. It is possible to examine if R cut-off can be used to categorize children with SAM according to their serum albumin level.

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TG and JW were involved in the conception and design of the study. TG, AA, PK and AL contributed to acquisition of data. TG was responsible for write up of the paper and all co-authors reviewed the draft manuscript and accepted the final version.

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Table 1. Anthropometry, age and sex by oedema of children hospitalized with severe acute malnutrition and healthy children without malnutrition

	Healthy reference n = 80	Severe acute malnutrition n =55	p	Severe acute malnutrition		P
				Oedematous n = 40	Non-oedematous n = 15	
Male sex	38 (47.5)[1]	33 (60.0)	0.15	22 (55.0)	11 (73.6)	0.21
Age, mon	28±15	36±24	0.02	35±24	27±18	0.04
Growth, z-score						
Height-for-age	-0.8± 0.9	-3.4± 1.7	<0.001	-3.0±1.6	-4.4±1.5	0.01
Weight-for-age	-0.3±0.9	-3.5±1.4	<0.001	-3.0±1.2	-4.9±0.8	<0.001
Weight-for-height	0.1± 0.9	-2.4± 1.7	<0.001	-1.9±1.5	-3.9±0.7	<0.001
Mid-upper arm circumference, cm	14.9±1.3	11.0±1.6	<0.001	11.6±1.6	9.7±0.8	<0.001

[1]Data in cell are mean ± SD or n (%), Chi-square, unpaired t-test

Table 2. Bio-electrical impedance values by oedema of children hospitalized with severe acute malnutrition and healthy children without malnutrition

	Healthy children	Severe acute malnutrition	p	Severe acute malnutrition		p
				Oedematous	Non-oedematous	
	n=80	n=55		n=40	n=15	
Resistance, Ohm[1]	839±118[2]	825±270	0.66	725±234	1091±155	<0.001
Reactance, Ohm	57±11	33±17	<0.001	29±14	46±16	<0.001
Phase angle, degree	3.8±0.7	2.2±0.7	<0.001	2.1±0.6	2.4±0.8	0.12
Resistance index	1005±196	1042±393	0.48	882±314	1471±224	<0.001
Reactance index	67±8	43±23	<0.001	36±19	63±23	<0.001

[1] Bio-electrical impedance values were at 50 kHz, index = value/height in m, [2] Data in cell are mean ± SD

Table 3. Serum levels of electrolytes, haemoglobin, acute phase protein and markers of liver function by oedema of children hospitalized with severe acute malnutrition

	Severe acute malnutrition				
	Oedematous		Non-oedematous		
	n	mean ± SD	n	mean ± SD	P[1]
Phosphate , mmol/L	39	0.86± 0.3	13	1.0 ± 0.4	0.18
Calcium, mmol/L	38	1.9 ± 0.3	13	1.9 ± 0.6	0.78
Magnesium, mmol/L	39	0.9 ± 0.2	13	0.9 ± 0.3	0.63
Sodium, mmol/L	40	135.7 ± 6.3	15	134.5 ± 8.0	0.57
Chloride, mmol/L	40	110.9 ± 5.8	15	108.9 ± 7.5	0.29
Potassium, mmol/L	40	3.8 ± 0.8	15	4.8 ± 2.3	0.02
Albumin, g/L	39	13 ± 6.7	12	21 ± 9.0	<0.001
Alkaline phosphatase, U/L	33	347 ± 106	18	531 ± 412	0.02
Bilirubin, mg/dl	33	13.1 ± 10.5	12	16.2 ± 10.5	0.41
Alanine aminotransferase, U/L	33	44 ± 21	18	38 ± 29	0.40
Haemoglobin, gm/dl	36	9.8 ± 2.2	22	10.0 ± 1.7	0.24
α_1-acid glycoprotein, g/L	33	2.9 ± 0.7	15	2.7 ± 1.0	0.72

[1] Unpaired t-test

Table 4.Correlations between anthropometry, age, sex and bio-electrical impedance indices of children hospitalized with severe acute malnutrition

	Resistance[1]	Reactance[1]	Phase angle
Age, mon	-0.45***	-0.42***	-0.18
Sex	0.11	0.02	0.001
Oedematous	-0.67***	-0.52***	0.22
Mid-upper arm circumference, cm	-0.34*	-0.10	0.31*
Height-for-age z score	-0.37*	-0.35**	-0.25
Weight-for-age z score	-0.64***	-0.37**	-0.03
Weight-for-height z score	-0.58***	-0.22	0.19

[1]Bio-electrical impedance values were measured at 50 kHz and resistance and reactance were indexed as value /height in m; Pearson's correlation coefficient *<0.05, **<0.01, ***<0.001

Table 5 Correlations between serum electrolyte level, haemoglobin, markers of liver function and bio-electrical impedance indices of children hospitalized with severe acute malnutrition

	Resistance/H[1]	Reactance/H[1]	Phase angle
Phosphate, mmol/L	0.18	0.33*	0.22
Calcium, mmol/L	0.21	0.23	0.14
Magnesium, mmol/L	0.27	0.26	0.08
Sodium, mmol/L	-0.05	0.17	0.17
Chloride, mmol/L	-0.08	0.20	0.13
Potassium, mmol/L	0.24	0.16	0.11
Albumin, g/L	0.59**	0.41**	0.19
Alkaline phosphatase, U/L	0.27	0.12	0.01
Bilirubin, mg/dl	0.27	0.22	0.05
Alanine aminotransferase, U/L	-0.28	-0.01	0.16
Haemoglobin, gm/dl	0.20	0.30*	0.26
Alpha-1-acid glycoprotein, g/L	-0.13	0.10	0.21

[1] Bio-electrical impedance values were measured at 50 kHz and resistance and reactance were indexed as value /height in m; Pearson's correlation coefficient, *<0.05, **<0.001;

Table 6. Independent correlates of bio-electrical impedance indices in children hospitalized with severe acute malnutrition

		Coefficient (95%CI)	P	R^2
Resistance index[1]	Oedematous	-247 (-442, -54)	0.014	0.66
	Albumin, mg/dl	16 (7, 25)	<0.001	
	Weight-for-height Z score	-88 (-134, -40)	0.001	
Reactance index	Oedematous	-29.3 (-41.1, -17.6)	<0.001	0.38
	Serum calcium	18.5 (3.6, 33.5)	0.016	
	Serum chlorine	1.1 (0.2, 1.9)	0.016	
Phase angle	Oedematous	-0.86 (-1.3, -0.42)	0.001	0.30
	Mid-arm circumference, cm	0.26 (0.13, 0.38)	<0.001	

[1] Bio-electrical impedance values were at 50 kHz, index = value/height in m. Model was adjusted for anthropometric indices, age, sex, mid-arm circumference, and serum α_1-acid glycoprotein, Na^+, K^+, P, Ca^+, Mg^+, Cl^-, bilirubin, albumin, alkaline phosphatase and haemoglobin. No interaction between albumin and oedema.