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2.4: VASCULAR AND CARDIAC STRUCTURE AND FUNCTION IN ADULT SURVIVORS OF SEVERE ACUTE MALNUTRITION

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Table Changes in predicted* and measured arterial stiffness at 3-month follow up							
		All subjects	p	Δ DBP < -7mmHg	p	Δ DBP > -7mmHg	p
Δ DBP	mmHg	-6 $\pm$ 9	0.002	-12 $\pm$ 6	< 0.001	1 $\pm$ 5	0.40
n		24		14		10	
Δ meds	ddd	1 $\pm$ 1	<0.001	2 $\pm$ 1	<0.001	0 $\pm$ 1	0.18
Δ SBP	mmHg	-14 $\pm$ 17	0.001	-24 $\pm$ 10	<0.001	1 $\pm$ 14	0.83
Δ PP	mmHg	-7 $\pm$ 12	0.007	-12 $\pm$ 9	<0.001	0 $\pm$ 13	0.92
Δ cPWV _{f-f}	m/s	-0.4 $\pm$ 1.6	0.29	-1.0 $\pm$ 1.5	0.026	0.5 $\pm$ 1.4	0.25
Δ cPWV _{DC}	m/s	-0.6 $\pm$ 1.1	0.021	-1.0 $\pm$ 0.8	<0.001	0.1 $\pm$ 1.2	0.80
Δ cPWV _{DC} *	m/s	-0.6 $\pm$ 1.1	0.013	-1.2 $\pm$ 0.9	<0.001	0.1 $\pm$ 0.8	0.58

Mean $\pm$ SD. Δ meds denotes antihypertensive medication change, in daily defined dose (ddd). DBP threshold is twice the intrameasurement standard deviation of 3.5 mmHg for DBP.

2.4

VASCULAR AND CARDIAC STRUCTURE AND FUNCTION IN ADULT SURVIVORS OF SEVERE ACUTE MALNUTRITION

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Severe acute malnutrition (SAM) has oedematous or non-oedematous phenotypes and their metabolic effects may persist into adulthood. We hypothesised that cardiovascular structure and function would differ between phenotypes as young adults and between survivors and persons never exposed to SAM.

Methods: We recruited 54 non-oedematous and 62 oedematous adult survivors of SAM, and 45 age/sex/BMI-matched community controls. Standardised measures of anthropometry, blood pressure (BP), 2D-echocardiography, carotid and femoral artery ultrasound, brachial, radial and carotid tonometry were performed. Cardiac output (CO), stroke volume (SV), left ventricular mass (LVM) index and outflow tract (LVOT) diameter, carotid and femoral intima-media thickness (IMT), carotid-femoral pulse wave velocity (PWV) and augmentation index were derived. Visceral fat mass was assessed by CT scan. **Results:** Mean (SD) age was 28.8 (7.8) years; 55% were male. Visceral fat mass and BP did not differ significantly between groups. There were no significant differences in cardiovascular measures between oedematous and non-oedematous survivors. After adjusting for age, sex, height and weight, measurements in controls exceeded survivors' expressed in mean (SE) standardized units, as follows: PWV 0.35 (0.14, $p=0.02$), CO 0.53 (0.16, $p=0.001$), LVOT diameter 0.71 (0.16, $p<0.001$), SV 0.44 (0.16, $p=0.009$), and femoral IMT 0.76 (0.19, $p<0.001$).

Conclusions: Adult survivors of SAM had less arterial stiffness and decreased CO (due mainly to a smaller SV) compared to controls. The nutritional insult in early childhood may have effects on organ growth and therefore cardiac function. However, at this young age there was no impact on vascular structure and function.

2.5

THE ADDITIVE VALUE OF MEASURING SUBCLINICAL ATHEROSCLEROSIS IS GENDER SPECIFIC

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Aim: Cardiovascular disease (CV) risk-stratification could be improved by adding measures of (subclinical) atherosclerosis to current risk scores, especially in intermediate-risk individuals. Our aim was to prospectively evaluate the additive value of non-invasive measurements of atherosclerosis (NIMA) for CV risk-stratification on top of traditional CV risk factors (tCVRF) in a middle-aged population-based cohort.

Methods: Carotid plaques, Intima-Media-Thickness (IMT), Ankle-Brachial-Index at rest (ABI-r) and after exercise (ABI-ex), Pulse-Wave-Velocity (PWV), Augmentation-Index (AIx), Central-Augmented-Pressure (CAP), and Central-Systolic-Pressure (CSP) were measured in 1367 CVD-free participants aged 50-70 years. CV-disease (CVD) was evaluated and validated after a mean

follow-up of 3.8 years. The additive value of NIMA on top of tCVRF was evaluated using R^2 , area-under-the-curve (AUC), and net-reclassification-improvement (NRI)-analyses.

Results: CVD was reported in 39 men and 32 women. Individual NIMA did not increase R^2 and AUC of the baseline-model (including tCVRF) and additionally showed no substantial reclassification, except for plaque-thickness in women (total-NRI=30.2%, $p=0.021$). In intermediate-risk men, baseline-model was improved by CSP (NRI=20.0%), plaque-thickness (NRI=19.2%), plaque-presence (NRI=16.7%), and ABI-r (NRI=13.6%). In intermediate-risk women all individual NIMA improved baseline-model (IMT showed highest NRI (102%). Combined NIMA improved risk-stratification in all women, and even more in intermediate-risk women. In men, combined NIMA showed additive value in intermediate-risk only. The optimal combinations were PWV-AIx-CSP-CAP-IMT in men (total-NRI=14.5% ($p=0.087$), IDI=0.016 ($p=0.148$), clinical-NRI=46.0%), and IMT-plaque-thickness in women (total-NRI=28.0% ($p=0.009$), IDI=0.047 ($p=0.061$), clinical-NRI=169.2%).

Conclusions: In a middle-aged population-based cohort, individual NIMA had additive value on top of tCVRF in intermediate-risk women, and to a lesser extent in intermediate-risk men and could improve CV risk-stratification. Combined NIMA resulted in larger reclassification in both men and women at intermediate-risk, but the optimal combination of NIMA differs between men and women.

2.6

COMPARISON OF HIGH-EFFICIENCY ON-LINE HEMODIAFILTRATION AND HIGH-FLUX HEMODIALYSIS ON VASCULAR FUNCTION AND STRUCTURE IN END-STAGE RENAL DISEASE PATIENTS

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The effects of increasing the convective clearance of uremic toxins using high-efficiency on-line hemodiafiltration (ol-HDF) on vascular function and structure in end-stage renal disease (ESRD) patients remain unknown.

Forty-two ESRD patients were randomized to switch from high-flux hemodialysis (HD) to ol-HDF ($n=22$) or to continue HD ($n=20$) for 4 months in a prospective double-blind study. Brachial artery endothelium-dependent flow-mediated dilatation (FMD), carotid artery structure and stiffness, and cardiovascular coupling, uremic toxins (beta-2 microglobulin, phosphatemia) and circulating markers of inflammation (TNF and inducible NO-synthase blood mRNA) and oxidative stress (reactive oxygen species, total antioxidant status) were assessed at baseline and at follow-up.

FMD increased in patients on ol-HDF (152 ± 27 to 177 ± 34 m, $P=0.002$) but not in patients on HD (165 ± 46 to 161 ± 36 m, $P=0.25$; $P<0.01$ vs. ol-HDF). Peripheral and central blood pressures, augmentation index, carotid artery diameter and intima-media thickness remained stable in both groups. Carotid distensibility was higher ($P=0.04$ vs. HD) and elastic modulus tended to be lower ($P=0.09$ vs. HD) in patients on ol-HDF. Beta-2 microglobulin ($P=0.02$) and phosphatemia ($P=0.01$) decreased in patients on ol-HDF. Moreover, in patients on ol-HDF, there was a trend for a decrease in inducible NO-synthase ($P=0.07$) and plasma nitrite ($P=0.06$), a marker of NO availability, while TNF increased only in patients on HD ($P=0.04$; $P=0.01$ vs. ol-HDF). Oxidative stress markers remained stable in both groups.

High-efficiency ol-HDF improves endothelial function and decreases arterial stiffness in ESRD patients as compared with high-flux HD. This may be notably related to the reduction in uremic toxins and vascular inflammation.