

# Prospective evaluation of liver stiffness in obese children: the role of shear wave elastography

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## INTRODUCTION

The increased incidence of childhood obesity and related non-alcoholic fatty liver disease (NAFLD) has determined the need to identify a non-invasive technique to diagnose and monitor NAFLD. Two-dimensional shear wave elastography (2D-SWE) has emerged as a reliable, non-invasive, tool to evaluate liver tissue elasticity in clinical practice, but its application in childhood obesity has not yet been widely evaluated.

## AIMS

- 1) To longitudinally evaluate 2D-SWE changes in relation to weight loss, metabolic profile and body composition modifications.
- 2) To investigate the correlation between 2D-SWE variation and clinical and biochemical indices of cardio-metabolic risk in obese children.

## METHODS

Thirty-three children underwent anthropometric (height, weight, BMI, waist circumference) and bioimpedentiometric (fat mass; free fat mass; total body water) evaluations, fasting biochemical assessments (glucose, insulin, total cholesterol, HDL, LDL, triglycerides, transaminases, oral glucose tolerance test), ultrasound and SWE evaluations (Mindray Resona 7 US machine equipped with a broadband SC5-1U convex probe), at baseline (V0) and after a 12-months follow-up (V12).

Homeostasis model assessment of insulin resistance (HOMA-IR) and  $\beta$ -cell function (HOMA- $\beta$ ), Matsuda-index, triglycerides/HDL-ratio, total cholesterol/HDL-ratio, Areas Under the Curves for glucose (AUCg) and insulin (AUCi) and their ratio were assessed. Standardized diet and exercise programs have been prescribed to all patients.

Variation of all parameters was evaluated in intra- and inter-group comparison analysis in children who had not lost weight (Group A) and those who had lost weight (Group B) at V12.

## RESULTS

A significant reduction of mean 2D-SWE value was demonstrated both in the entire cohort ( $p=0.002$ ) and in Group B children ( $p=0.004$ ). Intra-group comparison analysis between V0 and V12 documented a significant decrease of BMI, BMI SDS and a significant improvement of metabolic profile, documented by the reduction of insulin, glucose, HOMA-IR, HbA1c, triglycerides, triglycerides/HDL-ratio, transaminases, and by an increase of Matsuda-index and HDL, in children of Group B but not in those of Group A. Inter-group comparison analysis of clinical and biochemical parameters, showed significant differences for BMI, BMI SDS, insulin, HOMA-IR, HOMA-B, triglycerides, triglycerides/HDL-ratio, total cholesterol/HDL-ratio, transaminases between Group A and Group B children after 12-months follow-up.

Tab. 1 – Intra-group analysis between Group A and Group B children at V0 and V12.

	V0	V12	p
BMI	29.0 ± 3.5	28.6 ± 4.1	0.231
BMI SDS	2.9 ± 0.6	2.9 ± 0.8	0.681
WC	87.8 ± 8.9	89.9 ± 13.1	0.334
WHtR	0.6 ± 0.1	0.6 ± 0.1	0.900
Fasting Glucose (mg/dl)	94.9 ± 7.4	95.2 ± 15.9	0.303
Fasting Insulin ( $\mu$ U/ml)	22.0 ± 14.0	20.7 ± 12.1	0.207
Glucose OGTT-120'	127.6 ± 24.6	116.4 ± 20.8	0.012
Insulin OGTT-120'	143.8 ± 116.9	97.4 ± 67.1	0.025
HOMA-IR	5.3 ± 3.7	5.0 ± 3.4	0.266
HOMA- $\beta$	294.5 ± 287.6	253.9 ± 166.4	0.950
HbA1c	5.7 ± 0.4	5.3 ± 0.3	0.000
Matsuda index	2.47 ± 1.44	2.85 ± 1.6	0.022
AUCi / AUCg	0.96 ± 0.63	0.89 ± 0.57	0.201
TC (mg/dl)	164.1 ± 29.7	173.8 ± 24.1	0.006
HDL (mg/dl)	44.5 ± 10.0	50.1 ± 10.3	0.000
LDL (mg/dl)	95.2 ± 24.0	98.7 ± 25.6	0.274
TG (mg/dl)	87.8 ± 38.9	78.5 ± 28.9	0.092
TG/HDL	2.1 ± 1.1	1.7 ± 0.8	0.005
TC/HDL	3.9 ± 1.2	3.6 ± 0.9	0.086
AIP	0.3 ± 0.2	0.2 ± 0.2	0.009
GPT (U/L)	23.0 ± 13.6	21.5 ± 10.6	0.307
GOT (U/L)	32.5 ± 45.6	19.7 ± 5.4	0.003
GGT (U/L)	15.5 ± 6.2	13.9 ± 6.5	0.520
Uric Acid (mg/dl)	5.2 ± 1.2	5.2 ± 1.3	0.324
FM	28.3 ± 9.6	28.8 ± 10.6	0.487
FFM	39.5 ± 9.7	41.8 ± 10.7	0.002
TBW	28.8 ± 7.5	30.1 ± 8.1	0.004
2D-SWE (kPa)	10.5 ± 2.1	8.9 ± 2.3	0.002

Baseline (V0); 12-month follow-up (V12); Two-dimensional shear wave elastography (2D-SWE) .

Tab. 2 – Intra-group analysis between Group A and Group B children at V0 and V12.

	Group A (n=18)		p	Group B (n=15)		p
	V0	V12		V0	V12	
BMI	29.23 ± 3.47	30.46 ± 3.60	0.071	28.65 ± 3.60	26.39 ± 3.69	0.001
BMI SDS	2.87 ± 0.32	3.27 ± 0.61	0.001	2.93 ± 0.82	2.46 ± 0.73	0.001
WHtR	0.60 ± 0.06	0.64 ± 0.05	0.362	0.57 ± 0.04	0.56 ± 0.08	0.400
Fasting Glucose (mg/dl)	95 ± 7.25	98.11 ± 0.07	0.777	94.86 ± 7.8	91.6 ± 7.91	0.123
Fasting Insulin ( $\mu$ U/ml)	23.3 ± 17.4	26.1 ± 13.5	0.557	20.4 ± 8.58	14.14 ± 5.39	0.013
Glucose OGTT-120'	129.5 ± 30.4	121.3 ± 2.51	0.151	125.4 ± 15.6	110.4 ± 17.4	0.041
Insulin OGTT-120'	136.1 ± 108.9	120.6 ± 75.2	0.528	153.1 ± 129.1	69.6 ± 43.5	0.011
HOMA-IR	5.5 ± 4.6	6.5 ± 3.9	0.459	4.9 ± 2.35	3.18 ± 1.14	0.013
HOMA- $\beta$	351.1 ± 380.5	305.4 ± 96.9	0.679	226.6 ± 64.25	192.02 ± 93.07	0.191
HbA1c (%)	5.7 ± 0.37	5.4 ± 0.25	0.001	5.5 ± 0.4	5.2 ± 0.3	0.003
Matsuda index	2.35 ± 1.48	2.4 ± 1.67	0.500	2.6 ± 1.4	3.3 ± 1.38	0.009
AUCi / AUCg	0.98 ± 0.65	1.07 ± 0.69	0.879	0.92 ± 0.61	0.66 ± 0.24	0.1
TC (mg/dl)	171 ± 24.8	178.8 ± 21.1	0.162	155.8 ± 33.5	167.8 ± 26.6	0.014
HDL (mg/dl)	42.7 ± 9.8	47.4 ± 9.5	0.004	46.6 ± 10	53.3 ± 10.6	0.021
LDL (mg/dl)	99.5 ± 23.15	106.5 ± 1.38	0.098	90 ± 24.8	89.2 ± 27.6	0.826
TG (mg/dl)	83.4 ± 33.3	86.7 ± 31.5	0.705	93 ± 45.5	68.5 ± 22.4	0.008
TG/HDL	2.07 ± 1.03	1.9 ± 0.97	0.287	2.1 ± 1.2	1.3 ± 0.5	0.005
TC/HDL	4.2 ± 1.34	3.9 ± 0.9	0.163	3.4 ± 0.7	3.3 ± 0.8	0.307
AIP	0.27 ± 0.19	0.24 ± 0.19	0.368	0.26 ± 0.24	0.09 ± 0.14	0.011
GPT (U/L)	26.27 ± 17.07	26.8 ± 11.18	0.679	19 ± 6.01	15 ± 4.8	0.009
GOT (U/L)	43.1 ± 60.25	21.3 ± 5.06	0.061	19.8 ± 5.6	17.6 ± 5.19	0.003
GGT (U/L)	15.6 ± 7.4	16.2 ± 7.5	0.112	15.4 ± 4.5	10.8 ± 3.2	0.016
Uric Acid (mg/dl)	5.5 ± 1.18	5.8 ± 1.2	0.410	4.8 ± 1.2	4.47 ± 0.9	0.031
2D-SWE (kPa)	10.8 ± 2.2	9.0 ± 2.18	0.068	10.05 ± 2.01	8.65 ± 2.55	0.004

Baseline (V0); 12-month follow-up (V12); Two-dimensional shear wave elastography (2D-SWE) .

## CONCLUSIONS

These results suggested an association between weight loss, metabolic profile improvement and 2D-SWE value reduction. SWE could play a significant role in the non-invasive assessment of NAFLD in children and adolescents with obesity.

