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higher in obese group in comparison with controls (p =0,005, p=0,026 and p=0,018, respectively). A subgroup of obese children could present LV remodeling characterized by increased volume and wall thickness. These findings are superposable to Binnetoğlu et al´s [25]. In healthy children, the mean LGS value is -20,5% [26]. In our study, obese children compared with controls had lower 2D STE-derived LV LGS (-15,90 ± 3,84% vs 19,44 ± 5,75%, p=0,001). LGS in our study was not affected by sex or age. Similar observations of decreased longitudinal and circumferential strain in children with obesity were concluded by Kulkarni et al [27]. These findings are extensible to radial strain and early diastolic strain rate values [28]. Segmental analysis of the LV could provide in the future subtle markers for the emergence of future obesity-related cardiac disease [29]. Current guidelines recommend pulmonary hypertension screening in patients with severe obstructive sleep apnea. Prevalence of pulmonary hypertension in pediatric patients with obstructive sleep apnea is low and none of the patients with pulmonary hypertension had severe obstructive sleep apnea in a recent study over 163 patients [30]. According to this in our study we found significative higher pulmonary pressure in obese children, but no one had pulmonary hypertension (mPAP >25 mmHg) [31]. Normal TAPSE values including z-scores based on patient age have been established in pediatric patients [16]. The TAPSE/PASP ratio has recently been reported as an independent prognostic parameter in heart failure. When stratified by terciles, patients in the low tercile showed significantly compromised hemodynamic, functional and echocardiographic status [18]. This index may be a step forward a more efficient RV function evaluation. In our sample TAPSE/PAPS was significantly negative correlated with triglycerides (r -0,503, p=0,002) and total cholesterol levels (r -0,419, p=0,006). Diastolic function of obese participants was assessed by pulsed-wave Doppler (PWD) and TDI. We didn´t find a statistically significant difference that could indicate impaired LV filling like Gandhi et al [32]. However we found relation between HOMA-IR and triglycerides levels, correlated negatively with diastolic function of left ventricle estimated by E/A ratio with PWD. In previous studies LV diastolic disfunction in hypercholesterolemic children compared with controls is also described [33]. There wasn´t positive correlation between HOMA-IR, cholesterol, triglycerides and remodeling of left ventricle [34,35]. One reason could be that we didn´t estimate the LV mass index. And now the question is, can we take an active attitude with these patients that have already remodeling and some index of RV and LV function altered? Gores et al [36] describe an improvement in RV function assessed by TAPSE in a group of overweight patients participating in vigorous 3-month football training. We think next interventions could follow this line and select between the obese patients those that could beneficiate from personalized training and enter in a program of cardiac rehabilitation.

Limitations

The statistical power of the analysis is limited by the small sample size. We didn´t find relation between HOMA-IR, total cholesterol and triglycerides levels with LGS. Perhaps the view selection, defining end-systole or tracing the myocardium was not the most adequate, in obese patients there is an added difficulty due to bad transthoracic window. There were only very few patients with dyslipidemia and correlation coefficient was borderline. The correlations between echocardiographic and laboratory parameters need to be confirmed in upcoming and larger studies to know future health care implications.

Conclusion

Remodeling of LV and sub clinic systolic dysfunction could be relatively common in asymptomatic patients with obesity and easily detected by functional echocardiography. It is necessary to perform an standardized cardiovascular evaluation in obese children for early identification of LV changes and subclinical dysfunction especially in those with insulin resistance and dyslipidemia.

Conflict of interest disclosure:

Nothing to disclose.

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Patient and Public Involvement:

Patients were not involved in the recruitment to and conduct of the study.

Author contributions:

Authors 1 and 2 conceived the study; authors 2 and 3 enrolled the patients and supervised the data collection; authors 1 and 4 are the responsible in performing the echocardiographs; authors 1 and 2 drafted the manuscript; all authors contributed substantially to its revision.

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