Our study was the first to evaluate the WC in a pediatric population with CAH. WC is considered an independent predictor of cardiovascular risk in adults and children. It is an important indicator of insulin resistance, dyslipidemia, and hypertension [19,28,29]. In our study, 48% of the patients presented increased WC, which positively correlated with SBP. Moreover, a significant negative correlation was observed with the serum levels of adiponectin, considered a protective factor for cardiovascular disease [30-32]. Studies with higher numbers of patients would be required to assess whether an increased WC is an independent risk factor for cardiovascular disease in children with CAH.

Only 1 study evaluated the serum levels of adiponectin in children with CAH [16]. The authors found a negative correlation with z-BMI, age, and pubertal stage, whereas no difference was observed in the levels of adiponectin between the different types of glucocorticoids used for the treatment. In our study, no correlation was observed between the levels of adiponectin and these variables. However, a difference between the median levels of adiponectin and the type of glucocorticoids used for the treatment was detected, being higher in the group receiving hydrocortisone, followed by the group treated with prednisone/prednisolone, and lower in patients receiving dexamethasone. Several studies show that glucocorticoids decrease the levels of adiponectin. Degawa-Yamauchi and collaborators [32] reported a diminished release of adiponectin in adipocytes after infusion with dexamethasone. Fallo and collaborators [33] already observed that lower levels of adiponectin were detected in patients with Cushing's syndrome. Dexamethasone is a potent glucocorticoid with a longer half-life than hydrocortisone, which may account for the lower levels of adiponectin in patients receiving dexamethasone and for the higher levels of adiponectin in patients treated with hydrocortisone. However, further studies would be required to assess the real interference of the type of glucocorticoids used for the treatment in the metabolism of adiponectin in patients with CAH.

In this study, the high prevalence of obesity, overweight, hypertension, and dyslipidemia that were observed would justify the early assessment of risk factors for cardiovascular disease in pediatric patients with CAH. WC positively correlated with systolic BP and negatively with adiponectin. The serum levels of HDL already negatively correlated with androstenedione.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests that could be perceived as prejudicing the impartiality of the study.

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REFERENCES

1. Marumudi E, Khadgawat R, Surana V, Shabir I, Joseph A, et al. (2013) Diagnosis and management of classical congenital adrenal hyperplasia. Steroids 78: 741-746.
2. Wedell A (1998) Molecular genetics of congenital adrenal hyperplasia (21-hydroxylase deficiency): Implications for diagnosis, prognosis and treatment. Acta Paediatric 87: 159-164.
3. Joint Lwpes/Espe Cah Working Group (2002) Consensus statement on 21-hydroxylase deficiency from The Lawson Wilkins Pediatric Endocrine Society and The European Society for Pediatric Endocrinology. J Clin Endocrinol Metab 87: 4048-4053.
4. Speiser PW, Azziz R, Baskin LS, Ghizzoni L, Hensle TW, et al. (2010) Endocrine Society, "Congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency: An Endocrine Society clinical practice guideline". J Clin Endocrinol Metab 95: 4133-4160.
5. Kim MS, Ryabets-Lienhard A, Geffner ME (2012) Management of congenital adrenal hyperplasia in childhood. Curr Opin Endocrinol Diabetes Obes 19: 483-488.
6. Reisch N, Arlt W, Krone N (2011) Health Problems in Congenital Adrenal Hyperplasia due to 21-Hydroxylase Deficiency. Horm Res Pediatr 76: 73-85.
7. Speiser PW, White PC (2003) Congenital adrenal hyperplasia. N Engl J Med 349: 776-788.
8. Bachelot A, Plu-Bureau G, Thibaud E, Laborde K, Pinto G, et al. (2007) Long-term outcome of patients with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Horm Res 67: 268-276.
9. Nebesio TD, Eugster EA (2006) Observation of hypertension in children with 21- hydroxylase deficiency: a preliminary report. Endocrine 30: 279-282.
10. Hoepgffner W, Hermann A, Willgerodt H, Keller E (2006) Blood pressure in patients with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. J Pediatr Endocrinol Metab 19: 705-711.
11. Völkl TM, Simm D, Beier C, Dorr HG (2006) Obesity among children and adolescents with classic congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Pediatrics 117: 98-105.
12. Subbarayan A, Dattani MT, Peters CJ, Hindmarsh PC (2014) Cardiovascular risk factors in children and adolescents with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Clin Endocrinol 80: 471-477.
13. Charmandari E, Chrousos GP (2006) Metabolic syndrome manifestations in classic congenital adrenal

hyperplasia. Do they predispose to atherosclerotic cardiovascular disease and secondary polycystic ovary syndrome? Ann N Y Acad Sci 1083: 37-53.

14. Mooij CF, Kroese JM, Grinten HRC, Tack CJ, Hermus RMM (2010) Unfavorable trends in cardiovascular and metabolic risk in pediatric and adult patients with congenital adrenal hyperplasia? Clin Endocrinol 73: 137-146.
15. Strohmayer EA, Krakoff LR (2011) Glucocorticoids and cardiovascular risk factors. Endocrinol Metab Clin North Am 40: 409-417.
16. Volkl MKT, Simm D, Korner A, Kiess W, Kratzsch J, et al. (2009) Adiponectin levels are high in children with classic congenital adrenal hyperplasia (CAH) due to 21-hydroxylase deficiency. Acta Pædiatrica, 98: 885-891.
17. World Health Organization. The WHO Child Growth Standards. Available online at: http://www.who.in
18. National Health and Nutrition Examination Survey (2004) Anthropometry Procedures Manual. Available online at: https://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf
19. Fernandez JR, Redden DT, Pietrobelli A, Allison DB (2004) Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. J Pediatr 145: 439-444.
20. Brazilian Society of Cariology (2005) Guidelines for the prevention of atherosclerosis in childhood and adolescence of the Brazilian Society of Cardiology. Braz Arch Cardiol 85: 1-36.
21. Stewart MP, Krone PN (2011) The adrenal cortex. In: S. Melmed, K. S.Polonsky, P. R. Larsen, H. M. Kromemberg (Org). Williams Textbook of Endocrinology. 12th edn, Philadelphia: Elsevier, pp: 479-534.
22. Madeira R, Carvalho CNM, Gazolla FM, Matos AH, Borges MA, et al. (2008) Cut-off point for Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index established from Receiver Operating Characteristic (ROC) curve in the detection of metabolic syndrome in overweight pre-pubertal children. Arq Bras Endocrinol Metabol 52: 1466-1473.
23. Finkielstain GP, Kim MS, Sinaii N, Nishitani M, Ryzin CV, et al. (2012) Clinical characteristics of a cohort of 244 patients with congenital adrenal hyperplasia. J Clin Endocrinol Metab 97: 4429-4438.
24. Oliviera LMB, Faria JAD, Silva DN, Lago R, Toralles MBP (2013) Elevated levels of leptin and LDL-cholesterol in patients with well controlled congenital adrenal hyperplasia. Arq Bras Endocrinol Metabol, 57: 354-359.
25. Zimmermann A, Grigorescu-Sido P, AlKhzouz C, Patberg K, Bucerzan S, et al. (2010) Alteration in lipid and carbohydrate metabolism in patients with classic congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Horm Res Paediatr 74: 41-49.
26. Rahmouni K, Correia MLG, Haynes WG, Mark AL (2005) Obesity-associated hypertension: New insights into mechanisms. Hypertension 45: 9-14.
27. Damiani D, Kuba VM, Cominato L, Damiani D, Dichtchekenian V, et al. (2011) Metabolic Syndrome in children and adolescents: Doubts about terminology, but not about cardiometabolic risks. Arq Bras Endocrinol Metabol 55: 576-582.
28. Maffeis C, Pietrobelli A, Grezzani A, Provera S, Tato L (2001) Waist circumference and cardiovascular risk factors in prepubertal children. Obes Res 9: 179-187.
29. Prins B (2002) Adipose tissue as an endocrine organ. Best Pract Res Clin Endocrinol Metab 16: 639-651.
30. Costa JV, Duarte JS (2006) Adipose tissue and adipokines. Portuguese Medical Acta 19: 251-256.
31. Madeira AR, Carvalho CNM, Gazolla FM, Pinto LW, Borges MA, et al. (2009) The impact of obesity on metabolic syndrome components and adipokine levels in prepubertal children. J Pediatr 85: 261-268.
32. Degawa-Yamauchi M, Moss KA, Bovenkerk JE, Shankar SS, Morrison CL, et al. (2005) Regulation of adiponectin expression in human adipocytes: Effects of adiposity, glucocorticoids, and tumor necrosis factor alpha. Obes Res 13: 662-669.
33. Fallo F, Scarda A, Sonino N, Paoletta A, Boscaro M, et al. (2004) Effect of glucocorticoids on adiponectin: A study in healthy subjects and in Cushing's syndrome. Euro J Endocrinol 150: 339-344.