

Prevalence of adiposity-based chronic disease and its association with anthropometric and clinical indices: a cross-sectional study

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Abstract

The present study aimed to determine the prevalence of adiposity-based chronic disease (ABCD) and its association with anthropometric indices in the Mexican population. A cross-sectional study was conducted in 514 adults seen at a clinical research unit. The American Association of Clinical Endocrinology/AACE/ACE criteria were used to diagnose ABCD by first identifying subjects with BMI ≥ 25 kg/m² and those with BMI of 23–24.9 kg/m² and waist circumference ≥ 80 cm in women or ≥ 90 cm in men. The presence of metabolic and clinical complications associated with adiposity, such as factors related to metabolic syndrome, prediabetes, type 2 diabetes, dyslipidaemia and arterial hypertension, were subsequently evaluated. Anthropometric indices related to cardiometabolic risk factors were then determined. The results showed the prevalence of ABCD was 87.4% in total, 91.5% in men and 86% in women. The prevalence of ABCD stage 0 was 2.4%, stage 1 was 33.7% and stage 2 was 51.3%. The prevalence of obesity according to BMI was 57.6%. The waist/hip circumference index (prevalence ratio (PR) = 7.57; 95% CI 1.52, 37.5) and the conicity index (PR = 3.46; 95% CI 1.34, 8.93) were better predictors of ABCD, while appendicular skeletal mass % and skeletal muscle mass % decreased the risk of developing ABCD (PR = 0.93; 95% CI 0.90, 0.96; and PR = 0.95; 95% CI 0.93, 0.98). In conclusion, the prevalence of ABCD in our study was 87.4%. This prevalence increased with age. It is important to emphasise that one out of two subjects had severe obesity-related complications (ABCD stage 2).

Key words: Adiposity: Obesity: Anthropometry: CVD: Epidemiology

Obesity is a global health problem associated with an increased risk of coronary artery disease, stroke, cancer and premature mortality⁽¹⁾. It is known that the prevalence of obesity varies between developed and developing countries according to socio-cultural and political factors, including socio-economic, behavioural and environmental factors. According to the 2018 National Health and Nutrition Survey (ENSANUT), the prevalence of

adults with obesity in Mexico is 36.1%, which makes it the country with the second highest obesity rates, behind the USA⁽²⁾. This high prevalence of obesity in Mexico underlines the importance of describing prevalence rates in different ethnocultural populations, while recognising the importance of social determinants and cross-cultural factors⁽³⁾. Currently, obesity and overweight are diagnosed using anthropometric measurements, among

Abbreviations: ABCD, adiposity-based chronic disease; ASM, appendicular skeletal muscle mass; PR, prevalence ratio; SMM, skeletal muscle mass.

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which BMI (kg/m²) is the most commonly used. The historical use of BMI encompasses a series of advantages; it is simple for health-care professionals to calculate, and it has a good association with adiposity and the presence of obesity-related comorbidities, as documented in several epidemiological studies^(4,5). However, BMI has several limitations that result in its underperformance as a single indicator of obesity and predictor of health. These limitations include the misclassification of individuals at risk for obesity-related comorbidities due to high interindividual variability (age, sex and ethnicity), the insufficient conceptualisation of the pathophysiology of adiposity and the contribution of BMI to the social stigmatisation of obesity^(4,6). Thus, in 2014, the American Association of Clinical Endocrinology (AACE), in conjunction with the American College of Endocrinology (ACE) (AACE/ACE), coined the new diagnostic term *adiposity-based chronic disease* (ABCD) as an alternative to obesity⁽⁷⁾. ABCD involves the assessment of obesity based on three dimensions: aetiology, degree of adiposity and health risks. Furthermore, ABCD entails the persistence of maladaptive or pathophysiological processes related to alterations in adipocyte distribution, quantity and/or function, leading to disease stages with specific symptoms and complications⁽⁶⁾. Unlike BMI, the use of this model enables the identification and stratification of heterogeneous populations for more individualised management. Additionally, ABCD allows for early preventive action in subjects with lower BMI and one or more metabolic complications associated with adiposity^(6,7). In comparison to obesity, the definition of ABCD focuses not only on abnormal amounts of adiposity but also on the abnormal distribution and function of adipose tissue⁽⁸⁾. Therefore, this study aimed to determine the prevalence of ABCD, assess its association with anthropometric indicators and compare BMI calculations with the prevalence of obesity in volunteer participants from a tertiary hospital.

Experimental methods

Study design and participants

A cross-sectional study was conducted on participants being treated at the Department of Nutrition Physiology of the Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán (INCMNSZ) in Mexico from January 2015 to September 2019^(9,10). Mestizo Mexican subjects aged 18–60 years, of both sexes, and from different areas of the country were included. Participants diagnosed with previous chronic diseases, such as hypertension, diabetes, dyslipidaemia, thyroid disease and liver or kidney disease, women who were pregnant or breastfeeding, and subjects with any substance abuse were excluded from the study. This study was conducted under the guidelines of the Declaration of Helsinki, and the INCMNSZ Ethics committee approved all procedures involving human subjects (reference numbers 2373 and 1456). All participants were informed about the scope and procedures of the study, and written informed consent was obtained prior to any procedure.

Anthropometric measurements and body composition

Anthropometric measurements were taken, including body weight, height, waist circumference, hip circumference and wrist

circumference. Body weight and body composition, including fat-free mass (FFM), skeletal muscle mass (SMM), fat mass, body water and visceral fat area, were determined by multifrequency bioelectrical impedance analysis with Inbody 720 (Biospace Co.). Participants stood on the platform scale holding the device's handles with both hands to provide contact with eight tetrapolar electrodes (two in each foot and two in each hand) for analysis at 1, 5, 50, 250, 500 and 1000 kHz. Body composition was estimated using the manufacturer's equations⁽¹¹⁾. The appendicular skeletal muscle mass (ASM) was obtained with the sum of the lean tissue in both arms and legs, and the skeletal muscle mass index was calculated with the formula ASM (kg)/height(m)²⁽¹²⁾. Height was measured in centimetres using a BSM 370 stadiometer (Biospace Co. Ltd) with an approximation of 1 mm. Waist, hip and wrist circumferences were determined with a flexible measuring tape (SECA, Model 201) with an accuracy of 0.1 cm. All measurements were performed in duplicate by trained personnel according to the method described by Lohman. The anthropometric measurement protocol required subjects to be fasting and wearing light clothing⁽¹³⁾. Anthropometric indices were calculated with the following formulas: BMI = weight (kg)/height(m)²; waist/hip index = waist circumference (cm)/hip circumference (cm); waist/height index = waist circumference (cm)/height (cm); conicity index = waist circumference (m)/(0.109 × (weight (kg)/height(m)^{0.5})⁽¹⁴⁾; body adiposity index = (hip circumference (cm)/height (m)^{1.5}) – 18⁽¹⁵⁾; body shape index (ABSI) = waist circumference (m)/(BMI^{2/3} × height (m)^{1/2})⁽¹⁶⁾; body roundness index = 364.2 – 365.5 × (1 – ((0.5 × waist circumference (m)/π)²/(0.5 × height (m))²))^{0.5}⁽⁴⁾.

Biochemical measurements

A blood sample was taken after a fasting period of 10–12 h. Then, the blood samples were centrifuged at 3000 rpm for 10 min to obtain serum and plasma. Glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol and TAG levels were determined by the enzymatic colorimetric method using the COBAS Integra analyser, Roche Diagnostics. Insulin concentration was determined using a sandwich ELISA kit with dual monoclonal antibodies (80-INSHU-E01). The Homeostasis Model Assessment of Insulin Resistance Index (HOMA-IR index) was determined using the following equation: fasting serum glucose (mmol/l) × fasting plasma insulin (mIU/ml)/22.5⁽¹⁷⁾.

Clinical parameters and physical activity

A trained nutritionist recorded the demographic data and collected the clinical history of the participants. Blood pressure was taken on the right arm with a digital sphygmomanometer (Omron, HEM-781INT) while the participants were seated and had their arm uncovered. Four measurements were taken at 3-minute intervals; the first measurement was discarded, and the last three measurements were averaged to determine the systolic and diastolic blood pressure. Physical activity level was assessed with the long-form International Physical Activity Questionnaire⁽¹⁸⁾. Participants were categorised into high, moderate or low physical activity groups, according to International Physical Activity Questionnaire guidelines⁽¹⁹⁾.



Categorisation of variables

Participants were categorised into the following groups based on BMI, according to the WHO classification of nutritional status: normal weight (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), obesity I (30–34.9 kg/m²), obesity II (35–39.9 kg/m²) and obesity III (≥ 40 kg/m²)⁽²⁰⁾. Insulin resistance was defined as a HOMA-IR index ≥ 2.5 ⁽¹⁷⁾. Metabolic syndrome was defined as the presence of three or more of the following criteria: central obesity (waist circumference ≥ 80 cm in women and ≥ 90 cm in men), fasting glucose ≥ 100 mg/dl, triacyl glyceride concentration ≥ 150 mg/dl, HDL-cholesterol concentration < 50 mg/dl in women and < 40 mg/dl in men and systolic and/or diastolic blood pressure $\geq 130/85$ ⁽²¹⁾. Prediabetes was defined as fasting glucose ≥ 100 mg/dl and < 126 mg/dl, while type 2 diabetes was defined as fasting glucose ≥ 126 mg/dl⁽²²⁾. Hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic ≥ 90 mmHg⁽²³⁾. Dyslipidaemia was defined as a TAG concentration ≥ 150 mg/dl, total cholesterol ≥ 200 mg/dl and HDL-cholesterol < 40 mg/dl in men or < 50 mg/dl in women⁽²⁴⁾.

Determination of adiposity-based chronic disease in the study population

Participants with ABCD were identified according to the AACE/ACE criteria in three steps⁽⁷⁾:

Step 1. Anthropometric component: participants with BMI ≥ 25 kg/m² and those with BMI 23–24.9 kg/m² with a waist circumference ≥ 80 in women or ≥ 90 in men were selected.

Step 2. Clinical component: the presence of metabolic and clinical complications associated with adiposity, such as factors related to metabolic syndrome, prediabetes, type 2 diabetes, dyslipidaemia and arterial hypertension, were assessed.

Step 3. ABCD classification: the presence of ABCD was classified as follows: stage 0: no adiposity-associated complications are found; stage 1: the presence of one or more complications that are mild to moderate and/or that can be treated with moderate weight loss and stage 2: the presence of at least one severe adiposity-related complication, according to cardiometabolic disease staging system applicable to obesity (online Supplementary Table S1)^(7,25,26).

Statistical analysis

The sample size was calculated based on a binomial distribution, which determined that a sample of at least 371 individuals should be selected to calculate an estimated proportion of 62.8% according to previous studies, with a CI width equal to twice the accepted error (10%) with a confidence level of 95%⁽²⁷⁾. Continuous variables are expressed as the median (25th–75th percentile). Qualitative variables are expressed as frequencies (%). The Kruskal–Wallis analysis was used to assess the differences between demographic, anthropometric, body composition and clinical and biochemical variables between the ABCD categories. The Mann–Whitney *U* test was used to compare anthropometric, biochemical and clinical parameters of the population between men and women. The χ^2 test for trend was used to evaluate the difference in proportions in the qualitative variables. Risk factors were determined with the anthropometric variables associated with the presence of ABCD using a

Poisson regression model adjusted for age, sex and physical activity. The results obtained were considered significant, with a *P* value of < 0.05 . The data were analysed with SPSS for Windows (version 24, SPSS Inc.) and Stata Statistical Software (Release 14: StataCorp LP). Figures were made with GraphPad Prism version 7.

Results

Characteristics of the participants

A total of 514 participants were included, of which 74.7% were women. The median (25th–75th percentile) age was 37 years (27–47), which was higher in women (39 years (27–49)) than in men (34 years (27–44)) (*P* value = 0.05). Men had higher weight, waist circumference, waist:hip ratio, body adiposity index, ABSI, SMM%, FFM%, ASM%, visceral fat area, systolic and diastolic blood pressure, total cholesterol, LDL-cholesterol and TAG but lower fat mass % and HDL-cholesterol than women. A total of 59.3% were engaged in a low level of physical activity; no differences were observed in the level of physical activity between men and women. A total of 16.3% had arterial hypertension, which was higher in men than in women (28.5 *v.* 12.1%, respectively, *P* value < 0.001) (Table 1).

Prevalence of adiposity-based chronic disease and obesity by BMI

The prevalence of ABCD in the population was 87.4%. In men, the prevalence of this condition was 91.5%, while in women, it was 86%. The prevalence of stage 0 was 2.4%, that of stage 1 was 33.7% and that of stage 2 was 51.3%. Men showed a higher prevalence of ABCD stage 2 compared with women, 56.8% *v.* 49.5%, respectively (Fig. 1(a)) (online Supplementary Table S2). Participants with ABCD stage 2 had a higher median age [41 years (32–49 as the 25th–75th percentile)] relative to ABCD stage 0 subjects [28.5 years (24, 35–7)] (*P* value < 0.001) in this population. Regarding the status of obesity observed in the different stages of ABCD, at stage 0, all subjects were overweight, while in those with ABCD stage 1, 3.5% had a normal BMI, 49.1% were overweight and 47.4% were obese. In subjects with ABCD stage 2, 0.8% had normal BMI, 17.7% were overweight and 81.5% were obese (Fig. 1(b)). In contrast, 57.6% of the studied population had a BMI indicating obesity, and it was more prevalent in men than in women (67.6 *v.* 54.4%, respectively, *P* value = 0.008).

Anthropometric, biochemical and clinical variables according to ABCD classification

The anthropometric variables weight, BMI, waist circumference, waist/hip ratio, body adiposity index, ABSI and body roundness index were significantly higher in subjects with ABCD stage 2 (*P* < 0.001). SMM, lean muscle mass, FFM, ASM, body water and osseous tissue % were significantly lower (*P* < 0.001) in subjects with ABCD stage 2, whereas visceral fat area and % fat mass were higher in ABCD stage 2 compared with the other stages (*P* < 0.001). Glucose, LDL-cholesterol, TAG, insulin and HOMA-IR concentrations were significantly higher in subjects



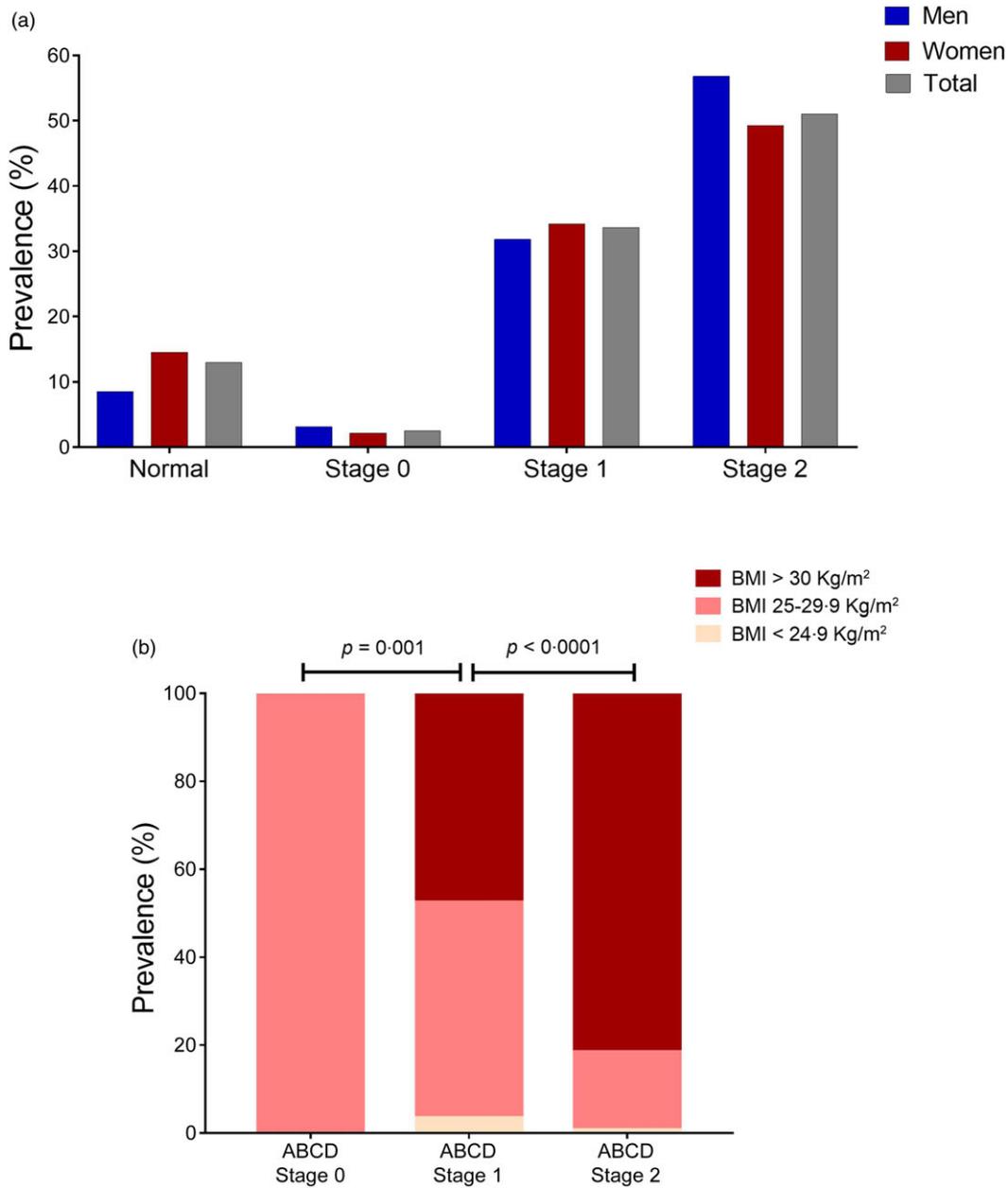


Fig. 1. (a) Prevalence of adiposity-based chronic disease stages by sex. (b) Prevalence of adiposity-based chronic disease stages by BMI.

syndrome, 4.7% had prediabetes and 34.4% had dyslipidaemia (Tables 2 and 3).

Anthropometric risk factors associated with adiposity-based chronic disease

In Poisson regression analysis adjusted for age, sex and physical activity, the waist circumference/hip circumference (WC/HC) index (prevalence ratio (PR) = 7.57; 95% CI 1.52, 37.5, P value = 0.013) and the Conicity Index (PR = 3.46; 95% CI 1.34, 8.93, P value = 0.01) were better predictors of ABCD than weight, BMI, hip circumference, wrist circumference, fat mass %, SMI kg/m², visceral fat area, body roundness index and body

adiposity index. Meanwhile, SMM% (PR = 0.95; 95% CI 0.93, 0.98, P value = 0.001), FFM% (PR = 0.96; 95% CI 0.95, 0.98, P value < 0.001) and ASM% (PR = 0.93; 95% CI 0.90, 0.96, P value < 0.001) were independent protective factors of ABCD (Table S3).

Discussion

The ABCD model aims not only to identify patients with excess adiposity through anthropometric measurements but also to integrate clinical variables that allow the detection of obesity-related complications. This allows for stratification according

Table 2. Anthropometric biochemical and clinical characteristics of the population according to the stage of adiposity-based chronic disease (ABCD)

Variables	ABCD						Normal <i>n</i> 64		<i>P</i>
	Stage 0 <i>n</i> 12		Stage 1 <i>n</i> 171		Stage 2 <i>n</i> 260		Median, Frequency	Percentile, %	
	Median, Frequency	Percentile, %	Median, Frequency	Percentile, %	Median, Frequency	Percentile, %			
Age, years	28.5	24.0, 35.7	39.0	27.0, 49.0	41.0	32.0, 49.0	25.0	23.0, 28.0	< 0.001
Sex									
Male	4	33.3	41	24	73	28.1	11	17.2	0.28
Female	8	66.7	130	76	187	71.9	53	82.8	
Anthropometry and body composition									
Weight, kg	66.2	59.6, 70.0	76.3	67.4, 90.0	86.3	76.8, 99.9	55.8	52.4, 62.4	< 0.001
BMI, kg/m ²	26.1	25.3, 26.8	29.5	27.4, 33.5	34.3	30.8, 38.3	22.5	20.7, 23.7	< 0.001
Waist, cm	78.8	77.6, 83.3	91.0	84.6, 101	103	94.0, 113.8	72.7	69.2, 77.0	< 0.001
Hip, cm	100	97.2, 104	107.7	101, 113.8	111	105, 120.4	95.2	92.0, 97.9	< 0.001
Waist/hip ratio	0.80	0.74, 0.82	0.84	0.80, 0.89	0.88	0.83, 0.93	0.76	0.73, 0.80	< 0.001
Waist/height ratio	0.50	0.48, 0.51	0.57	0.53, 0.62	0.64	0.58, 0.71	0.45	0.43, 0.48	< 0.001
Wrist circumference, cm	14.8	14.2, 15.2	16.0	15.3, 17.0	16.6	16.0, 17.8	14.8	14.2, 15.3	< 0.001
Conicity index	1.13	1.11, 1.15	1.20	1.17, 1.25	1.24	1.19, 1.30	1.12	1.08, 1.15	< 0.001
Body adiposity index	32.4	28.4, 35.3	35.3	31.6, 39.2	37.9	33.4, 43.5	28.9	27.0, 31.6	< 0.001
A body shape index	0.071	0.070, 0.073	0.075	0.072, 0.077	0.076	0.073, 0.079	0.073	0.070, 0.074	< 0.001
Body roundness index	3.53	3.13, 3.70	4.73	3.99, 5.68	5.82	4.90, 7.34	2.56	2.15, 3.06	< 0.001
Body fat mass, %	36.5	24.7, 41.8	42.0	36.9, 46.0	45.1	39.7, 49.7	30.7	26.1, 35.2	< 0.001
Skeletal muscle mass, %	34.8	30.7, 42.6	31.9	29.5, 35.3	30.3	27.6, 33.7	37.6	34.9, 40.2	< 0.001
Skeletal muscle index, kg/m ²	9.28	8.21, 10.9	9.57	8.90, 10.7	10.4	9.69, 11.4	8.22	7.83, 8.77	< 0.001
Lean mass, %	59.6	54.6, 70.9	54.5	50.8, 59.4	51.7	47.2, 56.8	65.1	60.8, 69.6	< 0.001
Fat free mass, %	63.5	58.2, 75.3	58.0	54.0, 63.1	54.8	50.2, 60.3	69.3	64.7, 73.8	< 0.001
Appendicular skeletal mass, kg	16.1	14.3, 21.4	17.2	15.4, 21.6	18.9	16.9, 22.9	15.2	13.5, 17.3	< 0.001
Appendicular skeletal mass, %	25.3	22.6, 30.6	23.1	21.5, 25.5	22.2	20.7, 24.4	26.8	25.5, 29.3	< 0.001
Appendicular skeletal mass, kg/m ²	10.5	9.74, 11.1	9.24	8.66, 9.87	8.77	8.19, 9.38	10.6	10.3, 11.2	< 0.001
Total body water, %	46.3	42.7, 54.9	42.3	39.4, 46.0	40.1	36.8, 44.1	50.5	47.3, 54.0	< 0.001
Minerals, %	4.60	4.26, 5.14	4.13	3.80, 4.47	3.81	3.43, 4.19	4.99	4.76, 5.18	< 0.001
Osseous, %	3.83	3.50, 4.22	3.43	3.16, 3.71	3.16	2.81, 3.47	4.14	3.96, 4.32	< 0.001
Visceral fat area, cm ²	79.5	70.0, 93.0	120.5	101.2, 143	141.5	122.3, 167	69.5	57.8, 79.4	< 0.001

Continuous variables are expressed as medians (25th–75th percentile). Qualitative variables are presented as frequency (%). Statistical analysis was performed using the Kruskal–Wallis test. Statistical analysis of qualitative variables was performed with the χ^2 test. *P* value < 0.05 was considered as statistical significance.

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Table 3. Biochemical and clinical characteristics of the population according to the stage of adiposity-based chronic disease (ABCD)

Variables	ABCD						Normal <i>n</i> 64		<i>P</i>
	Stage 0 <i>n</i> 12		Stage 1 <i>n</i> 171		Stage 2 <i>n</i> 260		Median, Frequency	Percentile, %	
	Median, Frequency	Percentile, %	Median, Frequency	Percentile, %	Median, Frequency	Percentile, %			
Clinical parameters and biochemists									
Systolic blood pressure, mmHg	101.5	96.2, 107.2	108	100, 115	112	104, 120.8	98.5	91.0, 104	< 0.001
Diastolic blood pressure, mmHg	73.5	64, 77	75.0	69.3, 80.6	79.3	73.0 86.0	66.3	62.0, 73.0	< 0.001
Physical activity									
Low	7	70	103	64	133	55.2	37	60.7	0.07
Moderate	–	–	8	5.0	27	11.2	1	1.6	
High	3	10	50	31.1	81	33.6	23	37.7	
Glucose, mg/dl	83	78.2, 87.0	85	79.0, 91.0	100	91.0, 108	83	78.2, 88.7	< 0.001
Total cholesterol, mg/dl	175	161, 210	184	163, 221	186.8	163.2, 214	171.5	155, 201	0.094
HDL-cholesterol, mg/dl	60.5	58.2, 64.2	47	41.0, 55.0	36.0	30.0, 42.0	56.5	45.2, 67.2	< 0.001
LDL-cholesterol, mg/dl	104.1	88.5, 116	116.6	97.2, 140.2	117	94.6, 140.2	99.5	78.4, 118	0.001
TAG, mg/dl	103	75.2, 125.7	119	96.0, 143.7	187.7	151, 248.1	77.0	58.2, 106	< 0.001
Insulin, U/ml	4.39	2.76, 8.68	9.19	5.38, 14.0	14.7	9.75, 21.8	5.23	3.86, 14.9	< 0.001
HOMAR-IR	0.96	0.54, 1.75	1.99	1.00, 3.16	3.59	2.19, 5.65	1.01	0.74, 3.20	< 0.001
Comorbidities									
Insulin resistance									
Yes	1	11.1	32	36.0	110	67.5	10	37.0	< 0.001
No	8	88.9	57	64.0	53	32.5	17	63.0	
Metabolic syndrome									
Yes	–	–	–	–	223	85.7	1	1.6	< 0.001
No	12	100	171	100	37	14.3	63	98.4	
Carbohydrate metabolism disorders									
Normal	12	100	171	100	124	47.7	61	95.3	< 0.001
Prediabetes	–	–	–	–	124	47.7	3	4.7	
Type 2 diabetes	–	–	–	–	12	4.6	–	–	
Dyslipidaemia									
Yes	–	–	103	60.2	240	92.3	22	34.4	< 0.001
No	12	100	68	39.8	20	7.7	42	65.6	
Hypertension									
Yes	–	–	9	5.3	74	28.5	–	–	< 0.001
No	12	100	162	94.7	186	71.5	64	100	

Prevalence of adiposity-based chronic disease

Continuous variables are expressed as medians (25th–75th percentile). Qualitative variables are presented as frequency (%). Statistical analysis was performed using the Kruskal–Wallis test. Statistical analysis of qualitative variables was performed with the χ^2 test. *P* value < 0.05 was considered as statistical significance.

to the presence or absence of such complications with the purpose of early identification of the population at risk.

The application of this new definition detected an ABCD prevalence of 87.4% in the Mexican adult population. In contrast, only 57.6% of the subjects included in this study had obesity as assessed by BMI. Although lifestyle modification interventions, such as the adoption of a healthy diet and physical activity, have been the primary treatment for people living with obesity, looking at this type of intervention from a 'complication-centred' framework undoubtedly offers prevention from the point of view of decreasing the risk of disease by preventing an obesogenic environment at the population level. In addition, the current prevalence of overweight and obesity in children between 5 and 11 years in Mexico is 35.6%. This prevalence could be underestimated if we use the ABCD framework to diagnose obesity, as it emphasises the importance of early interventions to prevent the risk of metabolic complications associated with obesity at an earlier age^(2,28).

These findings are of major importance, as a good part of the population with metabolic anomalies recruited for this study did not show a BMI associated with obesity (≥ 30 kg/m²). Consequently, these individuals would have gone undetected as subjects without risk, leading to the advancement of a detrimental metabolic environment. These results make apparent a major drawback of the use of BMI as the sole indicator of obesity. Whereas this index may be a practical tool, it does not consider body composition. Therefore, a BMI greater than 30 kg/m² does not necessarily mean that obesity is present, as it could be biased by a higher abundance of muscle mass or in body water.

Our results showed that 2.4% of patients were overweight or obese but free of cardiometabolic disease risk factors (Stage 0), the so-called 'metabolically healthy obese'. Other countries have shown a higher prevalence of this metabolic condition, e.g. the National Health and Nutrition Examination Survey (NHANES) reported a prevalence of overweight or obese adults free of cardiometabolic diseases of 15% in the USA⁽²⁹⁾. Furthermore, there was a much higher prevalence of stages 1 and 2 of ABCD in our population, with stage 2 being the most prevalent at 51.3%. This high percentage indicates that one out of two subjects in our study had severe adiposity-related complications.

Abnormal adiposity reflected by weight gain and BMI is not always associated with an increased risk of CVD because excessive fat mass is neither an essential nor a sufficient factor for heart disease. However, the abnormal distribution of adiposity, such as that located in ectopic sites, including skeletal muscle, liver and intestines, is associated with an increased risk of CVD and non-alcoholic fatty liver due to increased free fatty acids, inflammatory markers and insulin resistance. This could explain the strong association of waist/hip circumference index with the risk of developing ABCD observed in our study. Adiposity function is another key aspect to evaluate in the ABCD framework because adipokine imbalance associated with abnormal adiposity function is believed to be a key event in promoting both systemic metabolic dysfunction and CVD⁽³⁰⁾.

Although other indicators, such as waist circumference, could indirectly estimate visceral fat deposition, the use of the waist circumference indicator is more difficult in obese subjects since there are complications in specifically recognising the

anatomical position of the waist in this population. Although the measurement of waist circumference in people with overweight, and possibly also in those with stage 1 ABCD, allows a better estimation of metabolic risk than BMI alone, in the higher stages of obesity, waist circumference and metabolic risk are usually also elevated; thus, it is advisable to measure and document body composition as well^(31,32).

Two indicators were associated with ABCD in a protective manner, the SMM% and the ASM%, which are related to the amount of skeletal muscle tissue. This metabolically active organ can interact with other organs through secretory proteins⁽³³⁾. Some of the mechanisms by which muscle acts as a protective effect against metabolic diseases could involve the secretion of myokines such as IL-6, myostatin and irisin by muscle contraction, which could positively affect adipogenesis by promoting PPAR-activated receptor (PPAR- α)-dependent fat browning⁽³⁴⁾. Likewise, IL-6 secretion could regulate glucose production in the liver, lipolysis in adipose tissue and insulin secretion and can intramuscularly promote glucose uptake and fat oxidation through phosphoinositide 3-kinase (PI3K) activated by AMP-activated protein kinase (AMPK), thus improving insulin sensitivity⁽³⁵⁾. Irisin is involved in increasing energy expenditure through PPAR- α -dependent downstream signalling and improves insulin sensitivity⁽³⁴⁾. Therefore, through these mechanisms, the presence of skeletal muscle mass and secretion of its myokines results in beneficial effects on glucose uptake and lipid metabolism, having an important impact on the reduction of metabolic diseases⁽³⁶⁾.

It is important to note that although few studies address the prevalence of ABCD, the prevalence data observed in the present study have been higher than those reported in other populations. For example, in Venezuela, a similar trend was reported. Nevertheless, the prevalence of obesity according to the AACE/ACE framework was 63.5% and the prevalence of obesity by BMI (BMI ≥ 30) was 29.3%, which is lower than the prevalence observed in the present study⁽³⁷⁾. The prevalence was 62.8% in the Czech Republic in adults aged 25–64 years⁽²⁷⁾. Therefore, early detection and risk stratification are of the utmost importance as essential characteristics that the ABCD model takes into account and that other models centred on BMI lack. These characteristics allow for a window of opportunity to be observed for early interventions in primary care systems, which can have a favourable impact on the prevention and reduction of complications, and therefore, a significant decrease in morbidity and mortality as well as an increase in the quality of life of patients and families.

Exploring the association of different anthropometric measurements with ABCD might provide health practitioners with additional tools to simplify the assessment of adipose functionality. While ABCD has proven valuable in assessing cardiovascular risk, its application requires several variables that may not be available in a first-contact patient. To tackle this, we assessed the level of association of different anthropometric indicators that may be more readily attainable in a first-contact visit evaluation with ABCD. Here, our results show that the WC/HC index, conicity index, waist circumference and wrist circumference are predictors associated with ABCD and might



better reflect adipose functionality given their ability to indirectly estimate visceral fat deposition⁽³⁸⁾.

The limitations of this study are inherent to the type of design, which, being a cross-sectional study, is influenced by the temporal ambiguity of simultaneously measuring both exposure and the presence of the condition. Additionally, the sample of our study might not be representative of the general population because the participants were referred to a specialised centre; however, it may be useful as a reference for assessing the prevalence in the general population. The analysis of adiposity-based complications for this study was only evaluated with the available data; however, several other complications, such as the presence of reflux, fatty liver disease, polycystic ovary disease in women and sleep apnoea, could not be evaluated. Likewise, another limitation of our study is that we excluded participants with a previous diagnosis of diabetes, arterial hypertension and dyslipidaemia to represent a homogeneous sample.

Conclusions

In summary, in our study population, one out of every two subjects classified according to the ABCD had a severe obesity-related complication. Therefore, early detection and risk stratification are of the utmost importance. Taking into account the essential characteristics of the ABCD model can provide a window of opportunity for timely interventions in primary care systems, which can have a favourable impact on the prevention and reduction of complications. These results may be a starting point for future research aiming to evaluate the impact of different nutritional interventions on the adipose tissue amount, distribution and function rather than focus solely on weight and BMI.

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There are no conflicts of interest.

Supplementary material

For supplementary material/s referred to in this article, please visit <https://doi.org/10.1017/S0007114522002963>

References

1. Hales CM, Carroll MD, Fryar CD, *et al.* (2020) Prevalence of obesity and severe obesity among adults: united States, 2017–2018. *NCHS Data Brief* **360**, 1–8.
2. INEGI (2018) *National Institute of Public Health. National Health and Nutrition Survey*. Mexico: INEGI.
3. Vazquez-Duran M, Jimenez-Corona ME, Moreno-Altamirano L, *et al.* (2020) Social determinants for overweight and obesity in a

- highly marginalized population from Comitan, Chiapas, Mexico. *Salud Publica Mex* **62**, 477–486.
4. Thomas DM, Bredlau C, Bosy-Westphal A, *et al.* (2013) Relationships between body roundness with body fat and visceral adipose tissue emerging from a new geometrical model. *Obesity* **21**, 2264–2271.
5. Batsis JA, Mackenzie TA, Bartels SJ, *et al.* (2016) Diagnostic accuracy of body mass index to identify obesity in older adults: NHANES 1999–2004. *Int J Obes* **40**, 761–767.
6. Fruhbeck G, Busetto L, Dicker D, *et al.* (2019) The ABCD of obesity: an EASO position statement on a diagnostic term with clinical and scientific implications. *Obes Facts* **12**, 131–136.
7. Garvey WT, Garber AJ, Mechanick JI, *et al.* (2014) American association of clinical endocrinologists and American college of endocrinology consensus conference on obesity: building an evidence base for comprehensive action. *Endocr Pract* **20**, 956–976.
8. Nieto-Martinez R, Gonzalez-Rivas JP & Mechanick JI (2021) Cardiometabolic risk: new chronic care models. *JPEN J Parenter Enteral Nutr* **45**, 85–92.
9. Orozco-Ruiz X, Pichardo-Ontiveros E, Tovar AR, *et al.* (2018) Development and validation of new predictive equation for resting energy expenditure in adults with overweight and obesity. *Clin Nutr* **37**, 2198–2205.
10. Gonzalez-Salazar LE, Pichardo-Ontiveros E, Palacios-Gonzalez B, *et al.* (2021) Effect of the intake of dietary protein on insulin resistance in subjects with obesity: a randomized controlled clinical trial. *Eur J Nutr* **60**, 2435–2447.
11. Sullivan PA, Still CD, Jamieson ST, *et al.* (2019) Evaluation of multi-frequency bioelectrical impedance analysis for the assessment of body composition in individuals with obesity. *Obes Sci Pract* **5**, 141–147.
12. Purcell SA, Mackenzie M, Barbosa-Silva TG, *et al.* (2020) Prevalence of sarcopenic obesity using different definitions and the relationship with strength and physical performance in the Canadian longitudinal study of aging. *Front Physiol* **11**, 583825.
13. Lohman TG & Roche AF (1988) *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books.
14. Valdez R (1991) A simple model-based index of abdominal adiposity. *J Clin Epidemiol* **44**, 955–956.
15. Bergman RN, Stefanovski D, Buchanan TA, *et al.* (2011) A better index of body adiposity. *Obesity* **19**, 1083–1089.
16. Krakauer NY & Krakauer JC (2012) A new body shape index predicts mortality hazard independently of body mass index. *PLoS One* **7**, e39504.
17. Matthews DR, Hosker JP, Rudenski AS, *et al.* (1985) Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* **28**, 412–419.
18. Medina C, Barquera S & Janssen I (2013) Validity and reliability of the International physical activity questionnaire among adults in Mexico. *Rev Panam Salud Publica* **34**, 21–28.
19. Mehta S & Ostrum RF (1998) A calcaneal fracture with extrusion of the posterior facet. *Foot Ankle Int* **19**, 248–251.
20. James PT (2004) Obesity: the worldwide epidemic. *Clin Dermatol* **22**, 276–280.
21. Grundy SM, Cleeman JI, Daniels SR, *et al.* (2005) Diagnosis and management of the metabolic syndrome: an American heart association/National heart, lung, and blood institute scientific statement. *Circulation* **112**, 2735–2752.
22. American Diabetes Association Professional Practice Committee (2022) 2. Classification and diagnosis of diabetes: standards of medical care in diabetes-2022. *Diabetes Care* **45**, S17–S38.

23. Unger T, Borghi C, Charchar F, *et al.* (2020) 2020 International society of hypertension global hypertension practice guidelines. *Hypertension* **75**, 1334–1357.
24. Graham I, Atar D, Borch-Johnsen K, *et al.* (2007) European guidelines on cardiovascular disease prevention in clinical practice: executive summary. *Atherosclerosis* **194**, 1–45.
25. Guo F, Moellering DR & Garvey WT (2014) The progression of cardiometabolic disease: validation of a new cardiometabolic disease staging system applicable to obesity. *Obesity* **22**, 110–118.
26. Mechanick JI, Hurley DL & Garvey WT (2017) Adiposity-based chronic disease as a new diagnostic term: the American Association of Clinical Endocrinologists and American College of Endocrinology Position Statement. *Endocr Pract* **23**, 372–378.
27. Gonzalez-Rivas JP, Mechanick JI, Hernandez JP, *et al.* (2021) Prevalence of adiposity-based chronic disease in middle-aged adults from Czech Republic: the Kardiovize study. *Obes Sci Pract* **7**, 535–544.
28. Khandelwal S (2020) Obesity in midlife: lifestyle and dietary strategies. *Climacteric* **23**, 140–147.
29. Guo F & Garvey WT (2016) Trends in cardiovascular health metrics in obese adults: national health and nutrition examination survey (NHANES), 1988–2014. *J Am Heart Assoc* **5**, e003619.
30. Mechanick JI, Farkouh ME, Newman JD, *et al.* (2020) Cardiometabolic-based chronic disease, adiposity and dysglycemia drivers: JACC state-of-the-art review. *J Am Coll Cardiol* **75**, 525–538.
31. Lean ME, Han TS & Morrison CE (1995) Waist circumference as a measure for indicating need for weight management. *BMJ* **311**, 158–161.
32. Nazare JA, Smith J, Borel AL, *et al.* (2015) Usefulness of measuring both body mass index and waist circumference for the estimation of visceral adiposity and related cardiometabolic risk profile (from the INSPIRE ME IAA study). *Am J Cardiol* **115**, 307–315.
33. Kim G & Kim JH (2020) Impact of skeletal muscle mass on metabolic health. *Endocrinol Metab* **35**, 1–6.
34. Jeremic N, Chaturvedi P & Tyagi SC (2017) Browning of white fat: novel insight into factors, mechanisms, and therapeutics. *J Cell Physiol* **232**, 61–68.
35. Kistner TM, Pedersen BK & Lieberman DE (2022) Interleukin 6 as an energy allocator in muscle tissue. *Nat Metab* **4**, 170–179.
36. Nishikawa H, Fukunishi S, Asai A, *et al.* (2021) Pathophysiology and mechanisms of primary sarcopenia. *Int J Mol Med* **48**, 156.
37. Nieto-Martínez R, González-Rivas J, Ugel E, *et al.* (2018) Application of the AACE/ACE advanced framework for the diagnosis of obesity and cardiometabolic disease staging in a general population from 3 regions of Venezuela: the Vemsols Study Results. *Endocr Pract* **24**, 6–13.
38. Fox CS, Massaro JM, Hoffmann U, *et al.* (2007) Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* **116**, 39–48.