Original Research Article

Lactic dehydrogenase enzyme and acid-base status in severe preeclampsia with obesity. Case and control study.

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ABSTRACT

Aims: Compare and correlate blood concentration of the LDH enzyme and arterial blood gas parameters of pregnant patients with severe preeclampsia (SP) with and without obesity.
Study design: Case-control study.
Place and Duration of Study: Intensive Care Unit of High Specialty Medical Unit, Gynecology and Obstetrics Hospital No. 3. National Medical Center “La Raza”, Mexican Institute of Social Security, Mexico City, in the year 2022.
Methodology: Case-control study carried out in 73 pregnant patients with SP. Case group: 34 patients with pregestational Body Mass Index (BMI) with obesity (BMI >30) who remained obese during pregnancy. Control group: 39 women matched by gestational age with normal pregestational BMI (BMI <25) who remained with BMI normal during pregnancy. Blood concentration of total LDH enzyme and arterial blood gas parameters upon admission to the Intensive Care Unit (ICU) were compared. The LDH correlations vs arterial blood gas parameters were calculated. Statistical analysis: descriptive statistics, paired Student's t test and Pearson correlation coefficient (r) were used with the SPSS™ version 24 program. The *P* value <0.05 was significant.
Results: No differences were found in the blood concentration of total LDH enzyme (case group 669.54±71.79 vs control group 474.75±30.33 U/L, *P*= .21). The arterial blood gas parameters showed data of compensated metabolic acidosis in both groups, but without significant differences (pH *P*= .42, lactate *P*= .060, bicarbonate *P*= .89, std bicarbonate *P*= .74, ecf base deficit *P*= .93, base deficit *P*= .73). The correlations between blood concentration of total LDH enzyme and arterial blood gas parameters were not significant.
Conclusion: Blood concentration of total LDH enzyme was similar. Both groups showed compensated metabolic acidosis without differences of gasometric parameters. No significant correlations were found.

Keywords: Lactic dehydrogenase enzyme; Arterial blood gas; Severe preeclampsia; Maternal obesity; High risk pregnancy; Intensive care in obstetrics.

1. INTRODUCTION

Preeclampsia is the hypertensive state that most frequently complicates human pregnancy [1]. In our country, it is one of the main causes of maternal death due to serious complications that, for the most part, can be prevented if early diagnosis and timely management are carried out which includes termination of pregnancy [2]. The clinical horizon of the maternal stage of preeclampsia is located around week 20 of gestation. Unfortunately, most patients are identified late, when arterial hypertension and signs of organic and systemic endothelial deterioration appear. Mild alterations can progress to complications that endanger the life of the mother and fetus [3].

It has been documented that lactic dehydrogenase (LDH) enzyme is a useful tool to identify hepatocellular damage as an individual finding of severe preeclampsia (SP) or as part of the HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count) [4]. Specifically, blood concentration of the LDH enzyme ≥ 600 U/L has been directly related to an increase in maternal and perinatal mortality [5]. Under conditions of a compromised aerobic environment or low availability of glucose, the LDH enzyme is responsible for greater production of Lactate, which functions as an alternative energy substrate, but which ultimately deteriorates the vitality of cells, tissues and organs if the main cause is not corrected [6].

Arterial blood gas analysis is the gold standard for studying the changes in the internal environment that occur in critically ill patients [7,8]. In preeclampsia, Lactate and base deficit have been used to evaluate maternal-fetal status and perinatal prognosis [9-11]. Lactate concentrations > 2 mmol/L and base deficit value greater than -8 mmol/L have been related to fetal deterioration at birth. It has also been documented that both parameters reflect the biochemical alterations of the cells that increase the chances of maternal organ dysfunction and failure occurring during the prepartum, intrapartum, or postpartum period in patients with SP [9-11].

LDH enzyme and arterial blood gas parameters can be modified to a greater or lesser extent when aerobic tissue metabolism or glucose availability is compromised. LDH enzyme is part of the routine clinical laboratory studies performed on SP patients when they are admitted to hospitals, and arterial blood gas analysis is carried out when patients are admitted to the Intensive Care Unit (ICU). LDH enzyme and arterial blood gases can be complementary studies to explore the metabolic conditions of pregnant patients with SP.

On the other hand, obesity, defined as Body Mass Index (BMI) ≥ 30 calculated with the formula described by Quetelet in 1832 (BMI = weight in K / (height in m)^2, is a common disease of the modern life widely related to the risk of preeclampsia [1,3,12]. In maternal obesity, a systemic pro-inflammatory state, increased oxidative stress, accelerated atherosclerosis and extensive endothelial damage have been documented due to the release of cytotoxic lipokines into the bloodstream when aerobic tissue metabolism or glucose availability is impaired in the placenta, fetal, or maternal territory. Obesity and preeclampsia have similar pathophysiological characteristics (hyperinsulinism, insulin resistance, elevated concentrations of Leptin, Adiponectin, Tumor Necrosis Factor α, Interleukin 6, High-sensitivity C-Reactive Protein, alterations in lipid metabolism, and reduced flow-mediated by vasodilation) [13].
Theoretically, obesity can exacerbate the severity of preeclampsia with a further increase in the blood concentration of total LDH enzyme and adverse changes in the blood gas pattern. The **objective** of the present investigation was to compare and correlate the blood concentration of LDH enzyme and the parameters of arterial blood gas analysis of pregnant patients with SP with and without obesity.

2. MATERIAL AND METHODS

This was a case-control study carried out on 73 pregnant patients with SP admitted to the ICU of the High Specialty Medical Unit, Gynecology and Obstetrics Hospital No. 3 of the National Medical Center of Mexican Institute of Social Security, Mexico City, in the year 2022. All patients began their clinical manifestations at least 48 hours prior to their admission and went to the Family Medicine Unit or the Zone General Hospital corresponding to their home for medical care. Before the next 8 hours had passed, they were sent to the tertiary center that hosted this investigation where they were evaluated in the Emergency Department and then admitted to the ICU.

The study included patients over 18 years of age, of any parity, any type of comorbidities and with a pregnancy greater than 20 weeks. Patients who were smokers, malnourished, patients with recurrent preeclampsia, recurrent HELLP syndrome, recurrent eclampsia, history of transfusion in the last 6 months, chronic liver disease, any type of cancer, or local or systemic infections were not included. Because the clinical records of all the selected patients were available, there were no cases of elimination. The diagnosis of SP was established according to the recommendations of the American College of Obstetrics and Gynecologists (ACOG) of the United States of America published in 2020 [14].

To form the groups, the pregestational Body Mass Index (BMI) was calculated with the Quetelet formula BMI = weight in K / (height in m)^2 using the data contained in the clinical records. Case group included 34 patients with pregestational BMI with obesity (BMI >30) and who remained obese during pregnancy, the data was corroborated with the measurement of the BMI upon admission to the ICU. Control group consisted of 39 women matched by weeks of pregnancy with normal pregestational BMI (BMI <25) and who maintained their normal BMI during pregnancy until admission to the ICU. Overweight patients were not studied to establish a clear difference in BMI between the groups.

The records, clinical laboratory report, and arterial blood gas analysis of admission to the ICU were consulted to document general data, blood concentration of total LDH enzyme values, and blood gas parameters. Puncture of the radial artery to obtain the blood sample necessary for arterial blood gas titrations is a routine procedure carried out when patients are admitted to the ICU. For research purposes, blood concentrations of total LDH enzyme and arterial blood gas parameters, clinical characteristics, and maternal outcome were compared. The correlation of total LDH enzyme values with systolic and diastolic blood pressure and with each of the arterial blood gas parameters was calculated.

2.1 Data Analysis

For statistical analysis, descriptive statistics, the paired Student's t test and the Pearson correlation coefficient (r) were used with the SPSS™ version 24 program. The *P* value < .05 was taken as significant.

3. RESULTS

The general data are shown in **Table 1**. As can be seen, there were no differences in age, parity and weeks of pregnancy. As expected, the mean pregestational weight of both groups was different (*P*= .01) as was the weight upon admission to the ICU (*P*= .02). The patients in the case group increased their weight during pregnancy, the weight upon admission to the ICU was greater compared to the pregestational weight (*P*= .02). The patients in the control

group maintained their pregestational weight practically without modifications (*P*= .19). Consequently, the pregestational BMI of both groups was different (*P*= .007) as was the mean BMI upon admission to the ICU (*P*= .04). The BMI of the case group increased significantly during pregnancy (*P*= .01) and the BMI of the control group did not change (*P*= .44).

The percentage of prematurity in the case group was found to be 39.73% (29/73 cases) and 41.09% (30/73 cases) in the control group (*P*= .80). Morbidities such as chronic arterial hypertension, diabetic ketoacidosis, and complications of SP such as HELLP syndrome and acute kidney injury predominated in the case group. There were no patients with eclampsia in either group. In the case group, higher measurements of systolic blood pressure (*P*= .001) and diastolic blood pressure (*P*= .002) were found than the control group. When the mean uresis of both groups was compared, a significant difference was found in favor of the control group (case group 0.96 ± 0.67 vs. control group 1.25 ± 0.86 ml/hour, *P*= .01).
The time from admission to the ICU to termination of pregnancy was significantly longer in the case group (case group 17 ± 6.73 vs. control group 8.99 ± 6.95 hours, *P*= .01). To terminate the pregnancy, cesarean section was performed more frequently in both groups, while patients with vaginal delivery represented a low percentage. There was no difference regarding the estimated amount of intrapartum bleeding (*P*= .89). The length of stay in the ICU was similar in both groups (*P*= .45). No cases of maternal death were recorded.

Table 1.General data

Parameters	All patients n=73	Case group BMI >30 n=34	Control group BMI <25 n=39	*P* value
Age years	31 ± 7	33 ± 5	29.64 ± 7.94	.04
Parity median	2	2	2	-----
Weight K				
pregestational	67.47 ± 14.3	86.47 ± 12.04	55.99 ± 6.12	.01
admission to ICU	78.82 ± 13.78	94.20 ± 11.14	68.09 ± 7.40	.02
P value	.42	.02	.19	-----
Height m	1.57 ± 0.06	1.58 ± 0.05	1.57 ± 0.06	.34
BMI				
pregestational	27.26 ± 5.18	34.51 ± 4.20	22.75 ± 1.73	.007
admission to ICU	31.89 ± 4.93	37.64 ± 3.92	27.69 ± 2.44	.04
P value	.07	.01	.44	-----
Weeks of pregnancy	32.60 ± 4.33	32.81 ± 4.17	32.93 ± 4.19	.91
Prematurity (<37 weeks) % (n)	80.82 (59)	39.73 (29)	41.09 (30)	.80
Chronic arterial hypertension % (n)	1.36 (1)	1.36 (1)	0	-----
Diabetic cetoacidosis % (n)	1.36 (1)	1.36 (1)	0	-----
HELLP syndrome % (n)	24.65 (18)	17.81 (13)	6.84 (5)	rf 2.6
Eclampsia % (n)	0	0	0	-----
Acute kidney injury % (n)	10.95 (8)	9.59 (7)	1.36 (1)	rf 7
Blood pressure mmHg				
systolic	164.26 ± 17.44	172.92 ± 17.59	158.51 ± 16.19	.001
diastolic	101.50 ± 14.42	106.54 ± 9.89	97.87 ± 8.48	.002
Uresis ml/hour	1.17 ± 0.77	0.96 ± 0.67	1.25 ± 0.86	.01
Admission to ICU-delivery hours	9.10 ± 2.62	17 ± 6.73	8.99 ± 6.95	.01
Termination of pregnancy % (n)				
cesarean section	94.52 (69)	46.37 (32)	48.15 (37)	-----
vaginal delivery	5.48 (4)	2.74 (2)	2.74 (2)	-----

Intrapartum bleeding ml	470 ± 326.75	418.75 ± 158	424.36 ± 154.69	.89
ICU stay days	2.04 ± 1.64	1.76 ± 1.09	2.01 ± 1.44	.45
Maternal mortality % (n)	0	0	0	0

BMI = Body Mass Index (weight K / (height m)^2
ICU = Intensive Care Unit
HELLP = hemolysis, elevated liver enzymes, low platelet count
rf = relative frequency

Table 2 shows the clinical laboratory results. As can be seen, only a significant difference was found regarding the platelet count in favor of the control group (case group 134,510 ± 83,450, control group 177,460 ± 79,500 platelets / µL, *P* = .04). The rest of the results were similar. When the means of blood concentration of the total LDH enzyme were compared, it was found that the difference was not significant (*P*= .21). Total LDH values > 600 U/L were found in 15.06% (11 patients) of the case group and 12.33% (9 patients) in the control group (*P*= .93).

Table 2. Clinical laboratory results

Parameters	All patients n=73	Case group BMI >30 n=34	Control group BMI <25 n=39	*P* value
Hemoglobin g/dL	12 ± 1.6	12.52 ± 1.51	12.39 ± 1.6	.75
Platelets /µL	157,340 ± 82,220	134,510 ± 83,450	177,460 ± 79,500	.04
Fibrinogen mg/dL	551.71 ± 143.07	629.81 ± 121.33	522.28 ± 140.05	.20
Uric acid mg/dL	5.52 ± 1.61	5.61 ± 1.58	5.59 ± 1.79	.96
Glucose mg/dL	100.49 ± 49.57	92.39 ± 28.58	108.21 ± 45.02	.12
Creatinine mg/dL	0.79 ± 0.24	0.76 ± 0.19	0.83 ± 0.27	.30
Albumin g/dL	3.01 ± 2.17	2.87 ± 0.35	2.74 ± 0.48	.25
Globulins g/dL	2.19 ± 0.43	2.23 ± 0.49	2.14 ± 0.37	.42
PCOP mmHg	19.8 ± 11.94	19.10 ± 2.08	18.26 ± 2.83	.25
AST U/L	86.71 ± 178.44	60.17 ± 88.32	107.97 ± 149.08	.16
ALT U/L	76.26 ± 136.32	64.76 ± 95.94	89.85 ± 111.78	.36
LDH U/L value >600 U/L % (n)	555.94 ± 53.57 27.39 (20)	669.54 ± 71.79 15.06 (11)	474.75 ± 30.33 12.33 (9)	.21 .93

BMI = Body mass index (weight in K / (height in m)^2
PCOP = Plasma colloid osmotic pressure
AST = Aspartate aminotransferase enzyme

ALT = Alanine aminotransferase enzyme
LDH = Lactic dehydrogenase enzyme

The results of arterial blood gases are shown in **Table 3**. The data from both groups were compatible with compensated metabolic acidosis. There were no significant intergroup differences. Due to the relevance that has been given to them in previous reports on the subject, it was interesting to review the behavior of Lactate and ecf base deficit (ecf = extra cellular fluid, for its acronym in English). As can be seen, a non-significant difference in Lactate concentration was found in favor of the case group (case group 1.46 ± 1.01, control group 1.05 ± 0.39 mmol/L, *P*= .06). The Lactate value >2 mmol/L was found in 6.85% (5 patients) of the case group and 0% in the control group. Regarding ecf base deficit, no intergroup differences were found (case group **-**8.4 ± 3.46, control group **-**8.47 ± 3.53 mmol/L, *P*= .93). The percentage of patients with a value >**-**8 mmol/L was similar (case group 30.13% (22 patients), control group 28.77% (21 patients), *P*= .98). **Table 3**

Table 3. Arterial blood gas results

Parameters	All patients n=73	Case group BMI >30 n=34	Control group BMI <25 n=39	*P* value
pH	7.36 ± 0.6	7.42 ± 0.05	7.27 ± 0.95	.42
Partial pressure of carbon dioxide mmHg	24.6 ± 4.68	24.25 ± 5.07	24.92 ± 4.3	.57
Partial pressure of oxygen mmHg	85.94 ± 50.43	88.46 ± 27.06	90.56 ± 71.5	.89
Lactate mmol/L value >2 mmol/L % (n)	1.29 ± 0.87 6.85 (5)	1.46 ± 1.01 6.85 (5)	1.05 ± 0.39 0	.06 -----
Bicarbonate mmol/L	16.09 ± 3.06	15.84 ± 3	15.95 ± 3.09	.89
std Bicarbonate mmol/L	19.31 ± 2.45	19.28 ± 2.3	19.07 ± 2.43	.74
Total carbon dioxide mmol/L	16.69 ± 3.31	16.9 ± 3.51	16.44 ± 3.16	.59
ecf Base deficit mmol/L value >**-**8 mmol/L % (n)	-8.38 ± 3.5 58.90 (43)	-8.4 ± 3.46 30.13 (22)	-8.47 ± 3.53 28.77(21)	.93 .98
Base deficit mmol/L	-6.79 ± 3.71	-7.15 ± 2.92	-6.83 ± 3.91	.73
Capillary oxygen saturation %	94.16 ± 5.62	96 ± 4	93.49 ± 6.23	.24
Total capillary hemoglobin g/dL	9.99 ± 2.89	10 ± 3.23	9.92 ± 3	.92
Alveolar-arterial oxygen difference mmHg	18.84 ± 68.21	10.13 ± 21.69	14.15 ± 70.52	.78
Alveolar oxygen pressure mmHg	60.80± 104.06	62.29 ± 58.49	46.54 ± 89.61	.44

BMI = Body mass index (weight in K / (height in m)^2
std = standard
ecf = extracellular fluid

Table 4 shows the correlations of total LDH enzyme with each of the arterial blood gas parameters by group. In the case group, no correlation was found with systolic blood pressure (r= **-**.08) and diastolic blood pressure (r= **-**.15), a minimal negative correlation was

documented with pH (r= -.28), bicarbonate (r= -.39), std bicarbonate (r= -.43), ecf base deficit (r= -.42) and total base deficit (r= -.44). Specifically, Lactate had no correlation (r= -.003). In the control group, no correlation was found with systolic blood pressure (r= -.05), diastolic blood pressure (r= -.14) and pH (r= -.009), a minimal negative correlation was documented with bicarbonate (r= -.224), standard bicarbonate (r=-0.292), ecf base deficit (r=-0.354) and total base deficit (r=-0.215). Specifically, with Lactate the correlation was minimal positive (r= .32).

Table 4. Correlations of total LDH enzyme with arterial blood gas parameters.

Case group n=34		
	Parameters	Correlations
Lactic dehydrogenase enzyme U/L	Systolic blood pressure mmHg	-.08
	Diastolic blood pressure mmHg	-.15
	pH	-.28
	Partial pressure of carbon dioxide mmHg	-.23
	Partial pressure of oxygen mmHg	.10
	Lactate mmol/L	.003
	Bicarbonate mmol/L	-.39
	std Bicarbonate mmol/L	-.43
	Total carbon dioxide mmol/L	-.40
	ecf Base deficit mmol/l	-.42
	Base deficit mmol/L	-.44
	Capillary oxygen saturation %	-.005
	Total capillary hemoglobin g/dL	-.01
	Alveolar-arterial oxygen difference mmHg	.26
	Alveolar oxygen pressure mmHg	.35
Control group n=39		
Lactic dehydrogenase enzyme U/L	Systolic blood pressure mmHg	-.05
	Diastolic blood pressure mmHg	-.14
	pH	-.009
	Partial pressure of carbon dioxide mmHg	.03
	Partial pressure of oxygen mmHg	.21
	Lactate mmol/L	.32
	Bicarbonate mmol/L	-.22
	std Bicarbonate mmol/L	-.29
	Total carbon dioxide mmol/L	-.20
	ecf Base deficit mmol/L	-.35
	Base deficit mmol/L	-.21
	Capillary oxygen saturation %	.09
	Total capillary hemoglobin g/dL	.41
	Alveolar-arterial oxygen difference mmHg	.80
	Alveolar oxygen pressure mmHg	.72

BMI = Body mass index (weight in K / (height in m)^2
std = standard
ecf = extracellular fluid

4. DISCUSSION

Patients with obesity (case group) had serious morbidities (chronic arterial hypertension, diabetic ketoacidosis), weight gain during pregnancy (*P*= .01), higher systolic (*P*= .001) and diastolic (*P*= .002) blood pressure values, higher percentage of organic complications of preeclampsia (HELLP syndrome 17.81%, acute kidney injury 9.59%) with lower volumes of hourly uresis (*P*= .01), low platelet count (*P*= .04) and delay in time to end the pregnancy (*P*= .01). **Tables 1 and 2** However, no differences were found in the blood concentration of total LDH enzyme (*P*= .21) with the control group, unlike Fazal et al. [15] who in 2020 reported their results about the evaluation of total LDH enzyme and its isoenzymes as markers in preeclampsia. The authors found a significant elevation of total LDH enzyme in patients with mild preeclampsia and SP. Total LDH enzyme showed a moderate positive correlation with systolic and diastolic blood pressure with sensitivity 50% and specificity 80%. Furthermore, the pattern of LDH isoenzymes showed a decreasing distribution of aerobic forms in patients with preeclampsia-eclampsia. In the present investigation, we only had access to the measurement of total LDH enzyme and not the isoenzymes.

When the data were analyzed, no differences were found in the arterial blood gas parameters that are related to the acid-base state of the internal environment (pH *P*= .42, lactate P= .06, bicarbonate *P*= .89, standard bicarbonate *P*= .74, ecf base deficit *P*= .93, total base deficit *P*= .73). **Table 3** Furthermore, the correlation of total LDH enzyme with each of the blood gas parameters did not show significant values. **Table 4**

The secondary data deserve particular comments. The patients in the case group had a significant weight increase during pregnancy (*P*= .010), which has been reported as a permanent risk for the mother and fetus [16]. Furthermore, the systolic and diastolic blood pressure measurements were higher, but without a statistical relationship with the blood concentration of total LDH enzyme. In this scenario, it is possible that total LDH enzyme and its immediate product Lactate have a closer relationship with the deterioration of maternal water and energy status and its effect on tissue metabolism (pH values, bicarbonate and ecf base deficit), but not with the organic severity of preeclampsia manifested by arterial hypertension.

The arterial blood gas pattern was compatible with compensated metabolic acidosis in both groups; the data suggest that obesity did not make a difference. As in all types of metabolic acidosis, the first corrective therapeutic measures are the cautious administration of crystalloid fluids and the correction of the identified cause or the most probable cause [7,8]. In preeclampsia, the issue of parenteral fluid replacement has been a source of controversy [17]. The finding of compensated metabolic acidosis in patients in both groups and the data provided by Wheeler et al. [11] and Peguero et al. [9] are similar. They are support points to consider fluid replacement as an important part of pharmacological management of SP. In this context, the proposal is that cautious fluid replacement can be carried out simultaneously or immediately after the administration of antihypertensive drugs. Thus, the control of arterial hypertension and water management would be the priority of the medical protocol for patients with SP regardless of obesity. This proposal needs further supporting evidence.

Lactate, the metabolite resulting from the action of LDH enzyme on pyruvate under conditions of anaerobiosis or low glucose availability, was found to be slightly elevated in the case group, but the group mean did not reach a significant difference compared to the control group (*P*= .06) and its correlation with total LDH enzyme was minimal or practically null (r= .003). **Tables 3 and 4** Lactate is an essential biomarker to evaluate the acid-base status of the internal environment of patients in critical condition and patients with SP seem

to be no exception [6-11]. However, only 5 patients with Lactate values >2 mmol/L were identified who corresponded to the case group, but not the control group. This data can be taken as a trigger for further research because it is interesting to determine if the elevated concentration of Lactate (specifically the concentration >2 mmol/L) significantly reflects the condition of the acid-base state of the maternal internal environment or if it can only function as a biomarker of the placental deterioration (trophoblastic cells) during pregnancy, the suffering of the myometrium during the contractions of a vaginal birth or as a clue to make the decision to start antibiotics due to suspicion of cervico-vaginal infection during labor and the puerperium as has been reported in previous research [10,18-20].

In 1996 Wheeler et al. [11] investigated the issue of base deficit and oxygen transport in SP. With the aim of evaluating the anaerobic metabolism reflected by the calculated base deficit and its relationship with oxygen transport and left ventricular function in women with SP, the authors studied forty women with singleton pregnancies and SP. They recorded data from invasive hemodynamic monitoring after placement of a pulmonary artery catheter and arterial blood gas parameters. Linear regression analysis demonstrated a negative correlation between calculated base deficit and oxygen delivery rate (r= **−**.64), cardiac index (r= **−**.62), and left ventricular stroke work rate (r= **−**.58). They concluded that initial maternal baseline deficit with values greater than **-**8.0 mmol/L consistently predicted fetal acidosis, fetal death, and maternal left ventricular ischemic injury.

In our research, a total of 58.90% (43/73 patients) were found with base deficit that exceeded the cut-off point **-**8 mmol/L described by Wheeler et al. [11]. **Table 3** The distribution was similar for both groups. This data leads to the suspicion that left ventricular dysfunction can occur in patients with any BMI. In clinical practice, an echocardiographic study with special measurements of the left ventricle is not performed in all patients with SP because it is a restricted resource only for patients with high suspicion or clinical evidence of left ventricular dysfunction or heart failure. Women without clinical data are excluded from all cardiological investigations. Considering our data, we can add to the observations of Wheeler et al. [11] that all patients with a base deficit greater than **-**8 mmol/L with any BMI should be considered candidates for an intentional echocardiogram to determine the characteristics of left ventricular function and potential abnormalities early. This is particularly important because a series of structural and functional changes of the left ventricle related to preeclampsia have been described, the alterations mark the beginning of permanent adverse modifications that do not reverse in the postpartum period and that can progress in the long term [21-24].

Blood concentrations of total LDH enzyme, arterial blood gas parameters, clinical course, and maternal outcome of patients in the case group and the control group were similar. This situation could have occurred because the patients were identified early as having a high-risk pregnancy at the hospital of origin where they were treated with antihypertensive drugs and parenteral fluids, thereby interrupting the progressive nature of SP. There was no access to information on the hydration status and the provision of intravenous fluids from the first contact in the hospital of origin and the condition of the circulatory volume upon admission to the ICU, these became two important disadvantages for the results analysis. Finally, in this research, women residing in the central zone of our country were studied, whose epidemiological and somatometric characteristics are not necessarily the same as those of patients residing in other geographic areas of Mexico and the world, so the results may be different.

The main strength of the present investigation lies in the fact that it was documented that maternal obesity does not make a difference in the blood concentrations of total LDH enzyme, the acid-base status, the clinical course and the outcome of pregnant patients with

SP. Weaknesses include the study design, the small sample size of the groups and the non-inclusion of patients with extreme obesity (BMI >40), a select group of patients in whom clearly adverse maternal and perinatal outcomes have been reported [25]. The fact that we did not have access to data on parenteral fluid intake in the hospital of origin and the hemodynamic status when the patients were admitted to the ICU are also limitations of the research.

5. CONCLUSION

Blood concentrations of total LDH enzyme, arterial blood gas parameters, clinical course, and maternal outcome of patients in the case group and the control group were similar. No significant correlations were found.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

To carry out the study, the authorization of the Local Health Research Committee and the Local Health Research Ethics Committee of the host hospital was obtained (Registration: r-2023-1905-34).

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