Hemodynamic profiles in severe preeclamptic and superimposed preeclamptic pregnancies

G. UNCU (*) - C. CENGIZ (**) - S. SAHIN (***)

Summary: In this study, we investigated the hemodynamic profiles of pregnancy-induced hypertension cases. Hemodynamic measurements were performed in five severe preeclamptic and three superimposed preeclamptic cases. We applied Swan-Ganz catheters and measured hemodynamic parameters. According to the results, we suggest that pulmonary edema occurs more easily in severe preeclampsia than in superimposed preeclampsia.

Key words: Pulmonary capillary pressure; Cardiac output; Pulmonary edema; Preeclampsia; Superimposed preeclampsia.

INTRODUCTION

Investigations in hemodynamic profiles of pregnancy-induced hypertension have not obtained standard results regarding intravascular volume status and cardiac function in patients ([1-5]). We aimed to obtain basic information about cardiopulmonary hemodynamics in severe preeclamptic and superimposed preeclamptic pregnancies.

Received 5-8-1995 from the
Faculty of Medicine, Uludağ University, Turkey
(*) Assist. Professor, Dept. of Obstetrics and Gynecology
(**) Professor,
Dept. of Obstetrics and Gynecology
(***) Professor, Dept. of Anesthesiology
Revised manuscript accepted for publication September 20, 1995.

MATERIALS AND METHODS

We performed hemodynamic measurements in five severe preeclamptic and three superimposed preeclamptic cases who were treated in our clinic. None of the patients were controlled in our clinic nor having antenatal care before they were transferred to our hospital after becoming clinically worse. Severe preeclampsia was diagnosed when blood pressure was higher than 160/110 mmHg, or there was more proteinuria than 5 gr/24 hours, generalized edema, epigastric pain, oliguria and visual defects. The patients were accepted as superimposed preeclampsia because of a history of chronic hypertension, proteinuria and other symptoms of preeclampsia.

Following routine admission procedures a thermodilution type Swan-Ganz catheter was applied to the subclavian or jugular vein. After placing the catheter we confirmed its position by chest x-ray. Cardiac output was measured by a thermodilution technique and other hemodynamic measurements were obtained. Following a 4.5 gr $MgSO_4$ loading dose intravenously, we started to give 2 gr/h $MgSO_4$ infusion to five patients who were in the severe preeclampsia group and to one of three patients in the other group. The patients in the superimposed group used 4 × 250 mg p.o. alfa methyldopa and only one patient used nifedipin for an acute hypertensive attack.

Table 1. — *The characteristics of the pregnants.*

	PATIENTS							
	Severe Preeclampsia					Superimposed Preeclampsia		
	1	2	3	4	5	1	2	3
Age	27	26	25	38	33	25	28	33
Gravida	2	1	1	5	5	4	1	7
Para	0	0	0	2	1	3	0	3
Weight (kg)	59	70	100	104	66	85	63	63
Height (cm)	161	151	165	156	160	165	155	152
Gestational age (weeks)	34	32	32	35	26	34	33	25

RESULTS

The obstetrical characteristics of both groups are shown in table 1 and the hemodynamic findings are in table 2. The findings were not evaluated statistically because of the small numbers.

Mean arterial pressure (MAP) was higher than normal in all patients and central venous pressure was within normal ranges. Cardiac output (CO) was high in four patients in the severe preeclamptic group and was normal in one. In the superimposed group, CO was normal. Pulmonary capillary wedge pressure (PCWP) was high in three of the preeclamptic group, normal in two of them and normal in the superimposed preeclamptic group. Left ventricule stroke volume (LVSV) was high in all the patients in the two groups. In Starling plot graphics created by using PCWP and LVSV, three of the severe preeclamptic group were normal and two of them were found in depressed states. On the other hand, in the superimposed preeclamptic group all of them were in a hyperdynamic state.

Pulmonary vascular resistance (PVR) was low in all cases and systemic vascular resistance (SVR) was high. In hemodynamic measurembents, the intrapulmonary shunt (Qsp/Qt) was higher in the severe preeclamptic group than the superimposed preeclamptic group which is an important point.

Table 2. — *Hemodynamic measurements of the patients.*

Hemodynamic findings	Severe Preeclamptic					Superimposed Preeclampsia		
	1	2	3	4	5	6	7	8
HR	78	111	97	80	64	75	62	80
MAP	115	134	150	140	147	125	145	126
CVP	4	11	3	7	8	5	6	5
MPAP	10	18	26	22	19	6	14	12
PCWP	6	16	19	17	16	2	10	7
CO	4.9	6.4	8.4	7.7	6.5	5.1	3.9	4.5
PVR	70	25	67	63	72	66	82	90
SVR	1950	1543	1410	1692	1805	1999	2850	2184
Qsp/Qt	13	23	15	19	11	8	7	4
LWSV	85	92	153	131	132	107	115	92

DISCUSSION

In this study, cardiac output was found to be high in the severe preeclamptic group. In severe preeclampsia different results for cardiac output were reported. In three different studies, Littler, Werko and Smith have reported that CO was below normal values ([1-3]). In contrast, Benedetti found CO to be high in all severe preeclamptic patients. In our study, CO was lower in the superimposed preeclamptic group than in the severe preeclamptic group ([4]).

PCWP, MPAP, CVP and PVR have been reported differently in various investigations. In the study reported by Mabie, PCWP was high in severe preeclampsia but PVR was low ([5]). In a study by Cotton, pulmonary edema was seen in two patients, and PCWP was high ([6]). In another study by Henderson, PCWP raised to 13 Torr from 9 Torr when pulmonary edema developed ([7]). In our study, there was no increase in PCWP in chronic hypertensive pregnancies, who became preeclamptic later. On the other hand, in three of the five severe preeclamptic patients, PCWP was high. Left ventricle function was reported in the hyperdynamic position in severe preeclamsia. In Benedetti's study there was a shift to the left (hyperdynamic state) in eight of the ten severe preeclamptic pregnancies ([4]). Henderson also reported similar findings ([7]). Mabie found that the shift was to the left in 73% of the severely preeclamptic pregnancies and found severe preeclampsia in 80% ([5]). In our study, the results did not agree with the results in the literature. In two of the five severe preeclamptic group, left ventricle functions were depressed. In these patients, PCWP and MPAP was also high. In three other cases, it was in a normal state. But in all of the superimposed patients, left ventricle function was found in a hyperdynamic state. In previous studies, the reason for a hyperdynamic state in severe preeclampsia could not be explained but in our study it might be explained by an adaptation mechanism formed by chronic hypertension. In severe preeclampsia, there is no adaptation period. So, an acute rise in blood pressure causes decompensation and even pulmonary edema.

Another important point is that the intrapulmonary shunt (Qsp/Qt) was high in preeclamptic patients. When the pulmonary shunt (Qsp/Qt), which represents the unoxygenated blood in the pulmonary veins, is lower than ten, it is accepted as normal. In pulmonary edema, pneumonia, pulmonary fibrosis, atelectasia, pulmonary embolism and adult respiratory distress syndrome, it rises. In our study, it was higher than ten in all severe preeclamptic pregnancies and lower in chronic hypertensive patients. The low intrapulmonary shunt in superimposed preeclampsia can be explained by an adaptation mechanism.

According to our study, we accept that CO, PCWP and MPAP rises more in severe preeclampsia than superimposed preeclampsia. The left ventricle function is hyperdynamic in chronic hypertensives and normal or depressed in severe preeclampsia. The intrapulmonary shunt is low in chronic hypertensives and high in severe preeclampsia.

Pulmonary edema occurs more easily in severe preeclampsia than in superimposed preeclampsia.

REFERENCES

1) Littler W. A., Bonnar C. W., Beilin L. J.: "Reduced pulmonary arterial compliance in hypertensive pregnancy". *Lancet*, 1973, *2*, 1274.

2) Werko L., Langerlot H., Hucht H.: "Circulatory changes in pregnancy". *Acta Med. Scand.*, 1950, *239*, 263.

3) Smith L.: "Cardiovascular alterations in toxemia". *Am. J. Obstet. Gynecol.*, 1970, *107*, 979.
4) Benedetti T. J., Cotton D. B., Read J. C.: "Hemodynamic observations in severe pre-eclampsia with a flow-directed pulmonary artery catheter". *Am. J. Obstet. Gynecol.*, 1980, *136*, 465-470.
5) Mabie W. C., Ratts T. E., Sibai B. M.: "The central hemodynamics of severe preeclampsia". *Am. J. Obstet. Gynecol.*, 1989, *161*, 1443-1448.
6) Cotton D. B., Lee I. N., Huhta I. C.: "Hemodynamic profile of severe pregnancy-induced hypertension". *Am. J. Obstet. Gynecol.*, 1988, *158*, 523-529.
7) Henderson D. W., Vilos G. A., Milne K J.: "The role of Swan-Ganz catheterization in severe pregnancy-induced hypertension". *Am. J. Obstet. Gynecol.*, 1984, *148*, 570-574.

Address reprint requests to:
Yrd. Doc. Dr. GÜRKAN UNCU
Uludağ Üniversitesi Tip Fakültesi
Kadin Hastaliklari ve Doğum A.B.D.
16069 Görükle Bursa (Turkey)