Plasma albumin level as an indicator of severity of preeclampsia

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Summary

We propose that hypoalbuminemia in preeclampsia is the result of reduced hepatic blood flow which is secondary to hypovolemia created by higher filtration pressure in the capillaries. Thus, hypoalbuminemia can be identified as an early sign in developing preeclampsia. We reviewed the medical records of 60 patients, aged 22-28, admitted with a diagnosis of preeclampsia during the third trimester of pregnancy. Normotensive patients served as a control group. Albumin levels were correlated with severity of the disorder. Levels between 3.0-3.5 g/dl were seen in both groups. All patients with severe preeclampsia had values below 3.0 gm/dl. Serum albumin levels may serve as an indicator of the severity of preeclampsia.

Key words: Albumin; Preeclampsia; Predicting.

Introduction

Published sources state that the multiple organ hypoperfusion seen in preeclampsia is the result of endothelial damage precipitated by placental hypoxia [1]. It has been proposed that the hypoalbuminemia seen in these patients is the result of reduced hepatic synthesis of albumin secondary to a significantly reduced hepatic blood flow [2]. This leads to hypoalbuminemia which lowers the plasma oncotic pressure, thus worsening the process even further. Hypovolemia is created by higher than normal filtration pressures in the capillaries extruding water out into the extravascular space.

Intravascular albumin supplementation has been the mainstay of treatment at our institution for patients with preeclampsia [3]. We propose that the degree of hypoalbuminemia at the time of admission may be an indicator of the potential severity of the disorder and may serve as a prognostic sign of this condition.

This study was designed to correlate severity of preeclampsia with albumin serum levels obtained at the time of admission.

Materials and Methods

All pregnant patients with a diagnosis of hypertension admitted to our institution during 2003 were included in the analysis. A total of 60 patients suitable for this study were identified. None of these patients had received treatment prior to admission to our institution. Patients who had received magnesium, antihypertensive medications or volume expansion prior to admission were excluded from the analysis. Patients with a history of chronic hypertension were also excluded. An additional group of 20 non-hypertensive controls matched for gestational age were used for comparison. Depending on the findings, the patients were divided into one of four groups:

– Group A (n = 20) consisted of gestational age-matched normotensive controls.

– Group B (n = 20) patients with gestational hypertension.

– Group C (n = 20) patients with mild preeclampsia.

– Group D (n = 20) patients with severe preeclampsia, HELLP syndrome or eclampsia.

Serum albumin levels, creatinine and blood urea nitrogen were evaluated on admission and compared using statistical analysis.

Results

There were no statistically significant differences identified in the serum creatinine or blood urea levels among the different groups of hypertensive patients and controls.

Analysis of serum albumin levels for each group were as follows: normotensive controls, 3.68 ± 0.20 gm/dl; patients with gestational hypertension, 3.46 ± 0.16 gm/dl; mild preeclamptic, 3.36 ± 0.18 gm/dl and severe preeclamptic, 2.9 ± 0.26 gm/dl. The only group that showed a statistically significant difference in serum albumin levels as compared to controls was the severe preeclamptic group ($p < .03$). Table 1 shows the results of the means and standard deviations for serum albumin levels for each group.

Although differences were not statistically significant between the other two groups, comparison of the median serum albumin levels for each group showed an almost linear correlation with the severity of hypertension.

Discussion

We have proposed in the past that preeclampsia is the clinical manifestation of the release of vasoactive substances by hypoxic trophoblasts. These substances act by stimulating the heart and blood vessels. The increase in cardiac inotropism is not completely compensated by the increase in peripheral resistance. The increase in perfusion pressure forces fluid out into the extravascular space

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Table 1. — *Mean serum albumin levels in controls and patients with hypertensive disorders of pregnancy.*

Group	A	B	C	D
Age	24.5 ± 7.2	24.4 ± 5.5	27.5 ± 6.57	25.7 ± 8.17
Parity	1 ± 1	0.5 ± 0.6	1.2 ± 0.2	0.6 ± 0.75
Gestational age	35.6 ± 3.0	35.0 ± 2.7	33.1 ± 2.3	31.7 ± 2.9
Serum albumin	3.66 ± 0.21	3.46 ± 0.16	3.33 ± 0.16	2.9 ± 0.26*

*This value is statistically significant at $p < .03$ when compared with controls.

producing edema and promoting the development of hypovolemia. The decrease in intravascular volume reduces perfusion to multiple organs. The body's response to hypovolemia increases catecholamine production worsening even further the circulation to "non-essential" organs such as the liver and kidneys. As liver perfusion is compromised, the production of albumin is decreased. This situation leads to the gradual development of hypoalbuminemia. The reduction in serum albumin levels causes a reduction in oncotic pressure which further facilitates the passage of fluid into the extravascular space and worsens the condition even more. Thus, volume expansion in the form of serum albumin administration has been one of the mainstays of therapy at our institution [3]. The finding of low serum albumin among untreated preeclamptic patients further supports our hypothesis and argues in favor of volume expansion as an important part of the treatment of severe preeclampsia.

Conclusion

Our data show that the lowest serum albumin levels were significantly associated to the more severe forms of preeclampsia. An abnormally low serum albumin level may serve as a prognostic indicator of severity and should be considered as part of the evaluation of every preeclamptic patient.

References

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