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Relationship between Clinical Test Serological Indexes and Preeclampsia and Prediction in Preeclampsia

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Abstract: Pre-eclampsia is extremely harmful to mothers and infants. It often leads to a series of poor prognosis such as fetal distress, fetal growth restriction, placental abruption, premature delivery, acute renal failure and so on. At present, there is no effective method for predicting preeclampsia at home and abroad, so further research is needed. The purpose of this article is to analyze the relationship between serological indicators and preeclampsia by observing and analyzing the serological indicators of clinical testing, to understand the role of clinical testing serological indicators in the prediction of preeclampsia, and to the pre-eclampsia clinical testing serological indicators before and after In contrast, study the relationship between clinical test serological indicators and preeclampsia. Using the method of this article, through the analysis of experimental data, we understand the role of clinical test serological indicators in the prediction of preeclampsia, and find that clinical test serological indicators play an important role. Through the combination of theory and experimental data, we analyze the clinical test serology the predictive effect of the indicator in preeclampsia reached 84.6%. The research results show that HCG value can be used as one of the factors for predicting preeclampsia. According to these indicators, effective prediction of preeclampsia is provided, which provides a real basis for clinical prediction of preeclampsia, and finds a breakthrough from the prediction of PE to reduce the incidence of PE and improve the prognosis of pregnant women and fetuses of PE.

1. Introduction

Preeclampsia is a special eclampsia disease during pregnancy and one of the serious clinical complications during pregnancy [1]. Relevant foreign scholars still have a large research difference in the analysis of the incidence of preeclampsia tumors in younger sons [2]. Most maternal

eclampsia deaths may be due to pre-eclampsia complications. The main clinical and pathological features of threatened uterine epilepsy are the appearance of hypertension and protein polyuria after 20 weeks of gestation [3]. Preeclampsia may also directly cause severe perinatal fetal complications, such as early fetal distress, intrauterine growth and development restriction, and may even directly lead to early premature fetal sudden death and pregnant women premature delivery.

Preeclampsia can also cause serious perinatal complications, such as fetal distress, intrauterine growth restriction, and even lead to sudden fetal death and premature delivery. Preeclampsia is not only a serious threat to maternal and child health, but also one of the main causes of maternal and perinatal illness and death [4-5].

Treatment of threatened uterine epilepsy the clinical diagnosis and treatment can be used in a variety of ways, including antispasmodic, antihypertensive, convulsive control, prevention and timely treatment of pregnancy complications and timely decision to terminate routine pregnancy [6]. Adverse pregnancy outcomes where pregnancy has ended may be another important cause of death in premature and perinatal infants. Continuous changes in blood morphological statistical parameters, including neutrophil white blood cell ratio and lymphocyte number ratio, platelet neutral lymphocyte number ratio, red blood cell ratio distribution volume width, average neutral platelet distribution volume, platelet specific volume for fetal eclampsia the occurrence of early disease has unpredictable value [7-8].

This article uses the method of experimental research to understand the role of clinical test serological indicators in predicting preeclampsia, and comparative exploration before and after use; through theoretical analysis and experimental exploration, find out the relationship between clinical test serological indicators and preeclampsia ; Process the data through data recording, sorting, calculation, drawing, and analysis; simulate through the statistical data set of the relationship between clinical testing serological indicators and preeclampsia; combine the data to empirically analyze the clinical testing serological indicators against The role of the prediction of pre-eclampsia, combined with effective data, summarize and analyze the relationship between clinical testing serological indicators and pre-eclampsia. The results show that with this method, the recognition rate reaches 50%, which is faster.

2. Proposed Method

2.1. Pre-Eclampsia

Preeclampsia can have functional damage to any organ or system, including the heart, liver, brain, kidney, lung, and other important organs, including abnormal changes in circulation, blood, digestion, nerves, urinary system, including fetal placenta participation [9] . The course and condition are gradual and continuous. The occurrence of PE is not based on the increase of blood pressure in the second and third trimesters of pregnancy. Its pathophysiological changes have already occurred before the increase of blood pressure. Starting from the etiology of PE, study its pathophysiological changes and find effective serological markers to make early prediction of preeclampsia possible [10].

At present, the etiology and pathogenesis of PE are not fully understood. In the past, some gynecologists have called it a disease located in the placenta. Maternal, placental, fetal and other environmental factors directly cause disease outcome factors include insufficient reconstruction of endometrial spirochetes and small aneurysms, inflammation leads to activation of the immune system, damage to endothelium and leukocytes in blood vessels, genetic disease factors Wait. But the relatively unified point of view is that PE is a pregnancy-specific disease caused by multiple factors and multiple pathways [11]. Its occurrence has epidemiological and individual

characteristics. Risk factors for preeclampsia are first pregnancy, interval between pregnancies ⩾ 10 years, systolic blood pressure ⩾130 mmHg or diastolic blood pressure ⩾80 mm Hg (in the early stages of pregnancy or the first prenatal examination), 24 Hourly urine protein volume ⩾ 0.3 g or continuous appearance of urine protein (random urine protein ⩾ ++ once or more) and so on [12].

Although the research on the pathogenesis of preeclampsia is the most in-depth and extensive, the pathogenesis of PE is still not completely clear. There are many different clinical theories about the specific etiology and mechanism of hormone PE, such as oppression of the uterus, ischemia of the placenta endometrium, immunity, oxidative stress, metabolic disorders, genetics, and regulation of humoral metabolism in the central nervous system [13]. At the same time, the continuous development of molecular biology and clinical pathological cell physiology has also promoted its in-depth scientific analysis. At present, the main clinical theoretical basis for the etiology and mechanism of uterine PE tumors are: insufficient remodeling ability of benign endometrial spirochetes and arterial cells; capillary uterine endothelium and leukocytes are vulnerable to damage; abnormal body immune response regulation mechanism theory; excessive immune activation of the body Systemic inflammatory immune response; lack of related genetic disease factors; the body's insulin cannot be resisted; the body's lack of nutrition [14-15]. In the first stage of pregnancy, the normal developmental function of the placenta is greatly disturbed, and the placental remodeling function of the internal spirochete aorta of the mother is greatly damaged, resulting in a decrease in the normal blood and supply of the placenta at this time, and the placenta ischemic or hypoxic, Called acute placental dysfunction. In the second stage, the placenta is in a state of persistent ischemia and hypoxia, and various chemicals are secreted into the maternal blood circulation. After the placenta enters the maternal blood circulation, the chemicals secreted destroy the vascular endothelial cells on the one hand, and cause abnormal immune regulation and systemic inflammation on the other hand. This series of factors led to the occurrence of PE. A large number of studies have shown that the occurrence of PE is often accompanied by insufficient placental blood flow, and the most common cause of insufficient placental blood flow is the reconstruction of the uterine spiral artery. Although hypertension and proteinuria are the clinical diagnostic criteria for preeclampsia, neither is the main cause of multiple maternal organ damage. Vascular endothelial cell injury plays an important role in the pathophysiology of preeclampsia. Pregnancy is considered a controlled inflammation state [16]. Allogeneic fetuses can induce maternal immune and inflammatory responses. The inflammatory responses are present throughout the pregnancy and are conducive to the establishment and maintenance of normal pregnancy. Highly activated inflammatory cells, immune neutrophils and lymphocytes cause endothelial cell dysfunction by releasing inflammatory cytokines [17]. Excessive systemic inflammatory response is the physiological basis of clinical manifestations of PE [18]. The leukocytes in the maternal circulation are activated during pregnancy, while the leukocytes of pregnant women with PE will be further activated [19]. These activated white blood cells can reenter the maternal circulation and trigger endothelial dysfunction associated with preeclampsia. Neutrophils are often considered the first line of defense for wound infection. In pregnant women with preeclampsia, neutrophils can penetrate vascular tissues throughout the body, causing inflammation of vascular diseases.

2.2. Serological Indicators

Using various serological indicators to predict preeclampsia. HCG, AFP and uE3 are widely used to predict preeclampsia. The regular testing of serum prenatal screening for various indicators is mainly suitable for prenatal screening of 21-trisomy development syndrome and fetal neural tube function defects [20]. The abnormal index of prenatal eclampsia screening is related to the

abnormalities of fetal eclampsia and other adverse effects on the outcome of pregnancy, and to the development of preeclampsia. Compared with the normal high pregnant women in the early stage, the HCG hormone levels in the serum of the pregnant women with high PE were significantly increased in the early stage. In the early pregnancy of pregnant women with PE hormones, due to insufficient fetal endometrial spiral ligament small artery placenta remodeling ability, severe damage to the blood vessels inside the placenta, hypoxia of the placental endometrium, insufficient supply of blood to the uterine spiral placenta, compensated placental hyperplasia, fetus Excessive secretion of trophoblastic leukocyte hormones in the body leads to a significant increase in fetal serum HCG hormone levels. The HCG enzyme produced in the trophoblastic leukocytes in the placenta can directly reflect the developmental function of the infant placenta, which is directly related to the function formation of various early obstetric complications such as preeclampsia, small gestational age, and placental abruption [21-22]. PE is characterized by an increase in systemic inflammation. CRP is a sign of systemic inflammation. The serum CRP level of preeclampsia pregnant women is higher than that of normal pregnant women. The tumor necrosis factor-α general family contains 19 family members, including tumor necrosis factor-α and Fas ligand (FasL), which can induce apoptosis and regulate immune cells. Tumor necrosis factor-α is a multifunctional cytokine, mainly composed of macrophages, lymphocytes and trophoblast cells. It is a key participant in the initiation of the apoptosis cascade and may be related to the placental dysfunction of PE. There are many macrophages and lymphocytes in decidual and placental-like fibrous tissue [23]. Once they are stimulated, they produce TNF-α, and tumor necrosis factor-α binds to the corresponding receptor to play a pathological role in these tissues. Plasma and tumor necrosis factor-α were significantly higher in pregnant women with preeclampsia than healthy pregnant women. The rise of tumor necrosis factor-α is an important factor leading to injury, lack and coagulation of abnormal cell tubes, which played a key role in the development of preeclampsia [24]. Tumor necrosis factor-α in plasma and preeclampsia pregnant women is higher than normal pregnant women in late pregnancy. The difference between the two groups indicates that tumor necrosis factor-α is closely related to the onset and development of preeclampsia in late pregnancy. Tumor necrosis factor-α in plasma is an effective index to predict the onset and development of preeclampsia, especially in late pregnancy, the expression of tumor necrosis factor-α in the placenta of preeclampsia pregnant women is found in normal pregnant women [25]. Abnormal expression of tumor necrosis factor-α in the preeclampsia placental tube will damage the cells, cause cell dysfunction in the tube, increase cell adhesion, increase the permeability of the tube wall, resulting in contraction and pressure rise in the tube, the tube permeability increases, At the same time, the coagulation system is activated, which leads to abnormal coagulation function and eventually leads to the onset of preeclampsia. SFlt-1 is a soluble FMS, similar to tyrosine kinase-1 (SFlt-1), an anti-fallopian component, derived from extracellular endogenous receptor-1 (VEGFR-1). VEGF and PlGF are isolated and antagonized. This leads to a reduction in VEGFR signaling, destruction of renal tubular homeostasis, and internal dysfunction under pathological conditions. Preeclampsia is a common severe disease of pregnancy pressure, characterized by placental perfusion, placental hypoxia, and elevated serum sFlt-1. Although other factors may be involved in the pathogenesis of preeclampsia, there is no doubt that sFlt-1 is the driving factor. The fact manifested in sFlt-1 is that a membrane-bound β-co-receptor is responsible for the expansion of the tube before most eclampsia patients, tube formation and normal development of the tube are necessary, and negatively regulates invasive trophoblast differentiation Phenotype. Microtubule cells isolated from fat tissue during cesarean section in preeclampsia. In addition, in the fluid circulation of patients with preeclampsia, soluble endothelin (SG) egg cells increased significantly within a few weeks before clinical symptoms appeared. S-eng is produced by the division of transmembrane glycoprotein receptors.

Theoretically, when PE deformity occurs, the large and small arteries of the whole body are

slightly spastic, and the placenta is in a mild ischemic contraction state. On the premise of ensuring the normal secretion of fetal milk, with the continuous increase of blood flow in the placental cavity of the baby, the amount of fetal cells transferred to the mother decreased, and the level of serum fetal AFP in the mother decreased significantly. Finally, the condition of severe acute eclampsia develops. The normal serum blood and AFP of pregnant women are usually significantly higher than those of normal fetal pregnant women.This is likely to be mainly caused by severe damage to the uterine barrier of normal pregnant women. The embolism, hemorrhage and decidual uterus with necrosis of malignant spirochetes and aneurysms are in severe preeclampsia, so the possibility of increased serum blood and AFP in some normal women with severe preeclampsia is increased. In the process of further improvement of the placenta in the second trimester, patients with PE developed placental dysfunction, but the structural changes of the placenta had not been fully formed, and the blood supply to the placenta was not significantly reduced. Alpha-fetoprotein can be combined with essential fatty acids to promote fetal growth and development. The change of AFP value can reflect the severity of placental dysfunction and pathological changes of placenta, thus predicting poor perinatal outcomes.

uE3 is a steroid hormone related to fetal metabolism, derived from the adrenal gland and liver of the fetus. The precursor of uE3 placenta synthesis is dehydroepiandrosterone sulfate (DHEAS). Under the action of placenta aromatase, dehydroepiandrosterone is converted into estrogen. uE3 cannot be used alone to predict PE in the second trimester. In order to meet the needs of fetal growth and development and the improvement of metabolic levels during pregnancy, normal pregnant women can have a certain degree of insulin resistance. The reason may be the change of insulin reserve function of pregnant women and the relative change of the number of insulin receptors on the cell membrane. At the same time, the placenta secretes a series of hormones that can antagonize insulin, such as adiponectin (AP), retinol binding protein (RBP), homocysteine, etc., is a physiologically adaptive response. For women with normal pregnancy, the IR will also increase from early pregnancy to late pregnancy. Insulin resistance during pregnancy is a physiologically adaptive change, but it is also an important cause of pregnancy metabolic syndrome. Compared with healthy pregnant women, there are hypertension, hyperinsulinemia, obesity and dyslipidemia. Other accompanying changes may include the rise of leptin, tumor necrosis factor alpha and testosterone. The characteristics of preeclampsia (including hypertension, endothelial dysfunction and lipid changes) are also characteristics of metabolic syndrome, so IR may play an important role in the pathogenesis of preeclampsia. The gold standard for judging insulin resistance is the high insulin clamp experiment, but the operation is complicated and clinical application is difficult. In recent years, more and more scholars have begun to pay attention to the prediction of preeclampsia. With the in-depth study of the etiology and pathogenesis of preeclampsia, it is suggested that single index cannot successfully predict preeclampsia. At present, the prediction models of preeclampsia are gradually diversified, and combined with various indicators to improve the sensitivity and specificity of prediction.

2.3. Pre-Eclampsia Prediction

Research on the risk factors of preeclampsia helps to take targeted preventive measures. The so-called high risk factors for preeclampsia so far are: primipara, chronic hypertension, gestational diabetes and so on. High BMI before pregnancy or excessive weight gain during pregnancy are also risk factors for PE. The reason may be that obesity will bring insulin resistance and some kind of inflammatory response, and insulin resistance will increase the release of endothelin-1, which will eventually lead to damage to endothelial cell function. Vitamin D deficiency during pregnancy may be an independent risk factor for PE, and smoking is considered to be another independent factor

for preeclampsia. Supplementing folic acid and calcium can reduce the risk of PE. High-risk pregnant women can supplement folic acid and calcium to prevent PE. Adequate sleep and proper exercise can reduce the incidence of preeclampsia. Neutrophils are important cells in the inflammatory response; lymphocytes are the most important immune cells in the body's immune response. During normal pregnancy, platelets may be severely damaged and lifespan may be shortened. At the same time, platelet aggregation increased with the progress of pregnancy, and the number of circulating platelets decreased with the increase of pregnancy. During normal pregnancy, especially 28-31 weeks of pregnancy, the average platelet count decreases and MPV increases. Platelet volume is a sign of platelet age and may be indirectly related to platelet aggregation efficiency. Platelet count and MPV have a non-linear relationship. In fact, elevated MPV is a sign of increased megakaryocyte count in bone marrow, such as stress thrombocytopenia or changes in the number of megakaryocytes after platelet reduction. Red blood cell distribution width (RDW) is a sign of polycythemia, which reflects the change of red blood cell volume. RDW, as part of the complete blood count, can be evaluated by a fully automated hematology analyzer. In addition, RDW can also be used to detect iron deficiency anemia. RDW has significant correlation with cardiovascular disease (CVD) and coagulopathy. It can be used as an inflammatory marker of hypertension and cardiovascular disease. More importantly, in the general population, high RDW is considered to be a powerful and independent risk factor for death. RDW has a certain relationship with preeclampsia. RDW can be used as an important diagnostic and prognostic indicator of preeclampsia. Hematocrit (HCT): refers to the percentage of cells to total cells. Its low level is mainly related to the number of red blood cells and plasma volume. The pathological basis of preeclampsia is to damage the cells in the arteries and tubules of the system, resulting in increased permeability of the tubules, leading to fluid concentration, protein deficiency and swelling. Therefore, an increase in the number of red blood cells or a decrease in the volume of plasma will cause an increase in hematocrit.

Pregnancy associated plasma cell a (Pregnancyassociated plasmaaegga, PAPP-A) is a kind of sugar egg, mainly synthesized by placental trophoblast cells. It and β-subunit chorionic gonadotropin (β-HCG) are secreted by the placenta. PAPP-A is considered to be an insulin-like factor that binds to egg enzymes and plays an important role in the invasion of decidual trophoblast cells. The content of PAPP-A can reflect the hypoxia and deficiency of the placenta and is closely related to pregnancy. It combines with egg cutting to regulate the activity of insulin-like growth factor (IGF) and plays an important role in the invasion of trophoblast. In early pregnancy, PAPP-A changes the differentiation and invasion of trophoblast cells by regulating IGF. PAPP-A can be detected in the serum at the 5th week of pregnancy. The PAPP-A level of pregnant women with preeclampsia during pregnancy is lower than that of normal pregnant women. The predictive value of PAPP-A is better than that of late-onset PAPP-A. Chorionic gonadotropin (β-HCG) is a syncytiotrophoblast secreted by placental hormone. HCG levels change with increasing gestational age. The human chorionic gonadotropin is composed of α and β subunits. The alpha subunit is homologous to follicle stimulating hormone (FSH), luteinizing hormone (LH), and thyroid stimulating hormone (TSH), which is common in many egg hormones. The β subunit is specific to HCG. Because of its specificity, it is often used as a target for clinical testing. The main factors for the increase of HCG are: excessive growth of trophoblast cells, lack of placental villi, hypoxic environment, placental blood flow perfusion failure, placental villi damage and tissue hypoxia. The increase of HCG in maternal serum has become a research hotspot of preeclampsia risk factors. Researchers speculate that changes in maternal serum HCG may indicate the presence of PE risks. The β-HCG level found in the preeclampsia group was higher than that in the normal pregnancy group. HCG levels in early pregnancy were lower than those in hyperthyroidism, and HCG levels in early pregnancy were lower than those in hyperthyroidism. Therefore, additional measurement of

HCG may be helpful in distinguishing pregnant women at risk of preeclampsia. The sensitivity of PAPP-A, β-HCG and uterine artery index predicts that preeclampsia is lower than a single uterine artery, so it can be used as an index to predict preeclampsia, but the prediction model combined with other indicators may have higher predictive value.

Placental growth factor (PlGF) is a member of intravascular factor and has the function of regulating placental trophoblast cells and internal cells. Its main receptor is FMS, such as tyrosine kinase-1 (Flt-1). The main function of the placenta is to promote the proliferation and invasion of trophoblast cells, promote the development of the placental tube, and provide normal fluid circulation for the placenta during pregnancy. In early pregnancy, the invasion and secretion of pro-inflammatory cells due to the formation of tubules is not a possible cause of PE. These changes lead to internal dysfunction, cell activation, and elevated markers of inflammation. Placental lipid secretion activates all major cells, including neutrophils, lymphocytes and monocytes. Preeclampsia is closely related to inflammatory immune dysfunction. This is because the activated cells are deposited in the lumen of the body tube tissue, which leads to inflammation of the fallopian tube smooth muscle of the pregnant woman with pre-eclampsia, which eventually leads to fallopian tube contraction and dysfunction. The clinical manifestations of PE are related to general internal dysfunction, leading to tube contraction and lack of terminal organs. Neutrophils, lymphocytes and plate cells participate in the immune response of inflammatory cells by releasing inflammatory cytokines and body antibodies. NLR, PLR, RDW, MPV, PCT and other Sir markers obtained from peripheral blood complete cell count have prognostic and prognostic effects on various benign and malignant diseases, including coronary artery disease, inflammatory disease, PE, gynecology and gastrointestinal tumors, etc. Predicted value. The circulating cells of pregnant women are activated during pregnancy and are gradually activated in PE. These activated cells may cause PE-related tube dysfunction. NLR and PLR are considered to be signs of predicting the existence and severity of PE. Compared with healthy pregnant women, the NLR of women with severe preeclampsia is significantly increased, while the NLR of women with normal pregnancy is not significantly increased. The NLR of preeclampsia women is significantly higher than that of normal pregnancy women. NLR can predict the severity of the disease. NLR can be used to predict PE and its severity in early pregnancy. As the severity of PE increases, the increase in NLR may be the only predictor of the severity of preeclampsia. c-reactive protein (CRP) c-reactive protein is a clear β-spheroid. Its formation is regulated by IL-6, TNF-or and IL-1. Preeclampsia is a disease characterized by anti-angiogenesis and inflammation. Although the onset of this disease obviously originates from the placenta, it will release anti-angiogenic substances. The endoplasmic receptor-1 soluble receptor and the tumor factor β company activate the endoplasmic glycoprotein to participate in the anti-tube reaction, but the inflammatory response involves a variety of cytokines, including IL-6 levels and the cause of tumor death α in preeclampsia Incidence gradually increased. The expression of c-reactive protein (CRP) in these cells is maintained. The main feature of CRP is to activate the complement system, release inflammatory mediators, and promote intercellular adhesion and phagocytosis. The CRP content of the normal healthy group is extremely low. When the body is in an acute inflammation, trauma or stress state, CRP is rapidly synthesized under the induction of IL-6, TNF and other cytokines. As the condition improves, CRP will decline rapidly. CRP is not affected by the state of the body or the interference of external drugs, and is a sensitive indicator that reflects the body's inflammatory state.

3. Experiments

3.1. Experimental Materials

From December 2014 to June 2019, we collected clinical data of pregnant women in our hospital,

excluding high-risk factors such as old age, primary hypertension, kidney disease, and medical diseases related to the onset of preeclampsia, as well as 2018-2019 Examination of normal pregnant women. We collected data on 2,376 pregnant women. 803 cases of preeclampsia pregnant women were included in the preeclampsia group. Normal pregnant women were randomly matched at a 1: 1 ratio as a control group.

Definition of diagnostic criteria for preeclampsia: after 20 weeks of pregnancy, systolic blood pressure ⩾140 mm Hg and / or diastolic blood pressure ⩾90 mm Hg, accompanied by any of the following: urine protein ⩾0.3 g / 24 hours, or urine protein / Creatinine ratio ⩾0.3, or random urine protein ⩾ (+); no proteinuria but any of the following organs or systems involved: heart, lung, liver, kidney and other important organs, or blood system, digestive system, nervous system, placenta, etc. Abnormal changes affect the fetus, etc.

Inclusion criteria: regular inspections, fluid analysis results twice before 20 weeks and 28-41 + 6 weeks of pregnancy, and new outcome data were found when the pregnancy was terminated. The normal pregnancy group is pregnant women with complications. Exclusion criteria: twins and multiple births; traffic assisted reproductive technology; previous multiple abortions, multiple abortions, intrauterine fetal death; other complications of obstetrics during pregnancy; previous diabetes, hypertension, visceral diseases, abnormal thyroid function, liver Abnormal kidney function, poverty, history of thrombosis or exogenous disease; body immune disease; history of local or systemic infection, history of infectious disease, drinking, smoking and other bad habits, taking steroid-related drugs during pregnancy, fluid analysis indicators and clinical data are incomplete.

3.2. Experimental Method

Blood sample collection: sterilize hands, blood collector sets his body position, blood collector sterilizes skin from inside to outside and iodine carrier elbow vein puncture point, disinfection area 6cmx6cm, blood collected by blood sampling needle enters the corresponding color blood collection blood vessel, blood After collection, the blood collector is required to press the 3-4min blood collection website. Avoid strong oscillations of blood vessels. Place the blood vessels evenly in a centrifuge and centrifuge at 4000 rpm for 11 minutes. After centrifugation, test on the machine. The serum detected by InhA was collected and stored in an ultra-low temperature refrigerator at 80 ° C. Use Sysmex Company's whole cell analyzer, model: sysmexxn-1000, for blood analysis and detection. Reagents are provided by the company, and quality control is performed by the laboratory of Henan Hospital. The predictive ability of different liquid analysis indexes on PE was analyzed, and the unique predictive factors of PE were evaluated by using logistic regression analysis method, and a prediction model was established. All pregnant women participating in the study were registered with basic data before pregnancy. Time-resolved immunofluorescence method was used to detect HCG, AFP and uE3 values.

After finishing all the data, send it to an excel table, and the data analysis is performed using statistical software SPSS17.0. When the measurement data is normally distributed, the mean ± standard deviation is used to indicate that the difference between the groups is greater than the one-sample t test; the t test is used to verify the difference between the preeclampsia group and the control group. The independent sample t test was used in the preeclampsia group and normal pregnancy group. The diagnostic value of each index for preeclampsia was evaluated through diagnostic tests: the ROC curve of each test index against PE was drawn, the area under the curve (AUC) of each index was calculated, and the accuracy and authenticity of each index were predicted. The area under the ROC curve reflects the diagnostic value of various indicators, and the AUC value is between 1.0 and 0.5. When the AUC is closer to 1, the diagnostic value is higher.

According to the correlation between each index and PE and the predicted value, the index with the highest diagnostic value is selected for joint prediction, the ROC curve of the joint prediction is obtained, and the value of the joint prediction is evaluated.

4. Discussion

4.1. Predictive Analysis

(1) Comparison of two sets of values

Table 1. Comparison of two sets of values

Classification	Preeclampsia group	Control group	P
Year	27.71±5.54	27.58±6.28	0.739
HCG(mIU/ml)	29990.62±14309.76	26596.83±12245.00	0.016
AFP(IU/ML)	38.15±14.31	41.23±14.13	0.035
uE3(ng/ml)	0.60±0.24	0.80±0.30	0.008
0h-Glu	4.34±0.55	4.01±0.45	0
1h-Glu	8.00±0.91	8.54±0.816	0
2h-Glu	7.77±0.71	7.25±0.77	0

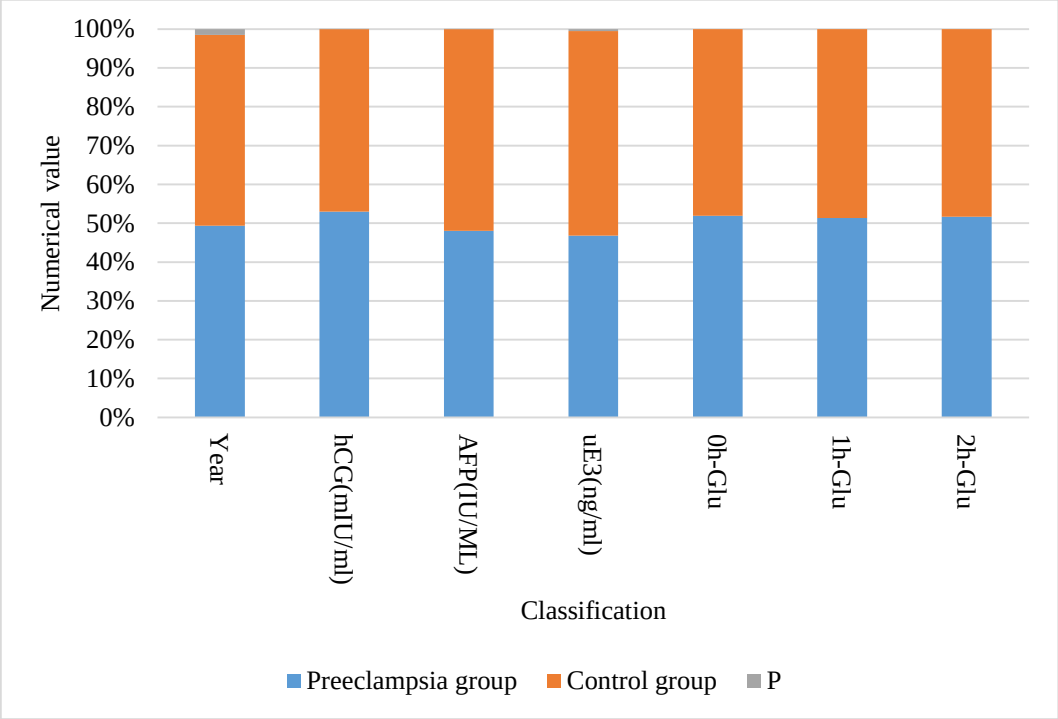

Figure 1. Comparison of two sets of values

According to the statistical analysis of data, as shown in Figure 1 and Table 1, the differences in serum HCG, AFP, uE3, 0h-Glu, 1h-Glu, 2h-Glu and other data are statistically significant. Alpha-fetoprotein is an important serum protein during fetal period. The yolk sac is produced before 12 weeks of gestation, and then the fetal liver gradually matures, replacing the yolk sac to secrete AFP. AFP plays an important role in fetal growth and development, especially in the nervous system. It can combine with essential fatty acids of the human body to play a biological role. HCG is a glycoprotein secreted by syncytiotrophoblasts. Mainly used for early pregnancy diagnosis,

mid-pregnancy serological screening, ectopic pregnancy and trophoblastic disease. uE3 is a related metabolite of estrogen, which combines with globulin to perform biological functions. The level of free uE3 in fetal serum reflects the growth and metabolism of the fetus.

(2) The predictive value of various indicators to PE

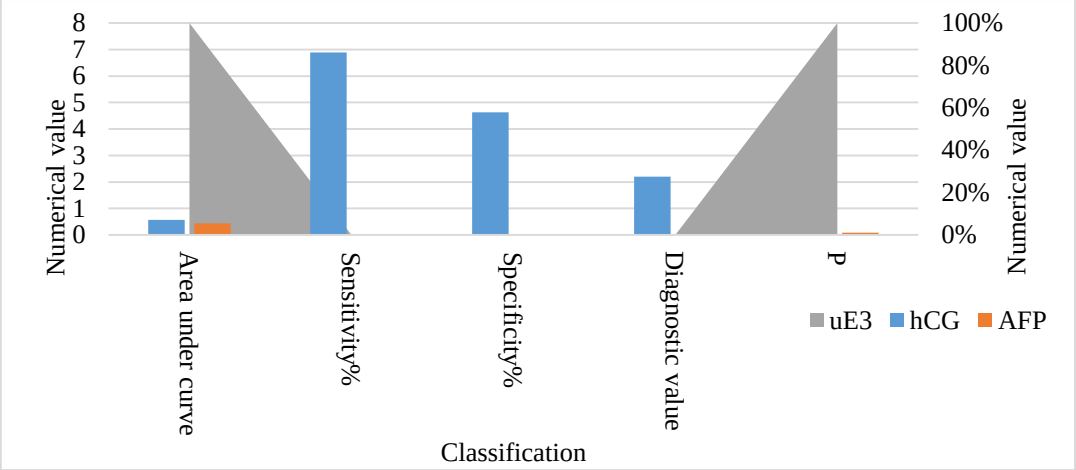

Figure 2. Forecast value of PE

According to statistical analysis of the data, as shown in Figure 2, the area under the ROC curve of AFP is 0.432, the area under the ROC curve of HCG is 0.57, and the area under the ROC curve of uE3 is 0.429. AFP is the most common fetal protein in fetal serum. During normal pregnancy, AFP can enter the mother through the placenta, so that the mother's serum AFP value gradually increases. Therefore, the serum AFP value of pregnant women is not only related to the absolute value of the fetus, but also to the increase in placental blood flow and membrane permeability. After embryo implantation, the serum HCG level of pregnant women gradually increased, reached a peak around 8 weeks of gestation, lasted for about 10 days, and then quickly dropped to about 10% of the peak period, and gradually dropped to normal levels during 18-20 weeks. A decrease in uE3 levels during the second trimester is associated with adverse pregnancy outcomes. The possible reasons are impaired placental function, damaged vascular endothelial cells, placental ischemia and hypoxia, and a large number of trophoblast apoptosis, resulting in a decrease in serum uE3 levels.

4.2. Correlation Analysis

(1) Blood index analysis

Table 2. Blood index analysis

Group	Normal group	Preeclampsia group	Mild preeclampsia group	Severe preeclampsia group
WBC	8.76±2.23	9.12±2.48	8.49±1.63	9.40±2.75
RBC	4.10±0.30	4.12±0.51	4.20±0.53	4.09±0.51
HCT	38.05±2.92	37.15±3.86	37.50±2.91	37.01±4.22
MCH	31.59±8.60	30.29±2.73	30.27±2.28	30.30±2.92
PLT	172.72±39.44	167.17±53.19	186.13±48.12	158.61±53.42
MONO	5.81±1.31	5.18±1.79	5.46±1.62	5.06±1.86
MCV	92.67±5.86	90.82±6.45	91.07±5.10	90.71±7.00
MPV	11.37±1.50	11.79±1.38	11.43±1.31	11.98±1.38
P-LCR	36.01±10.51	39.03±9.76	36.67±9.38	40.27±9.78

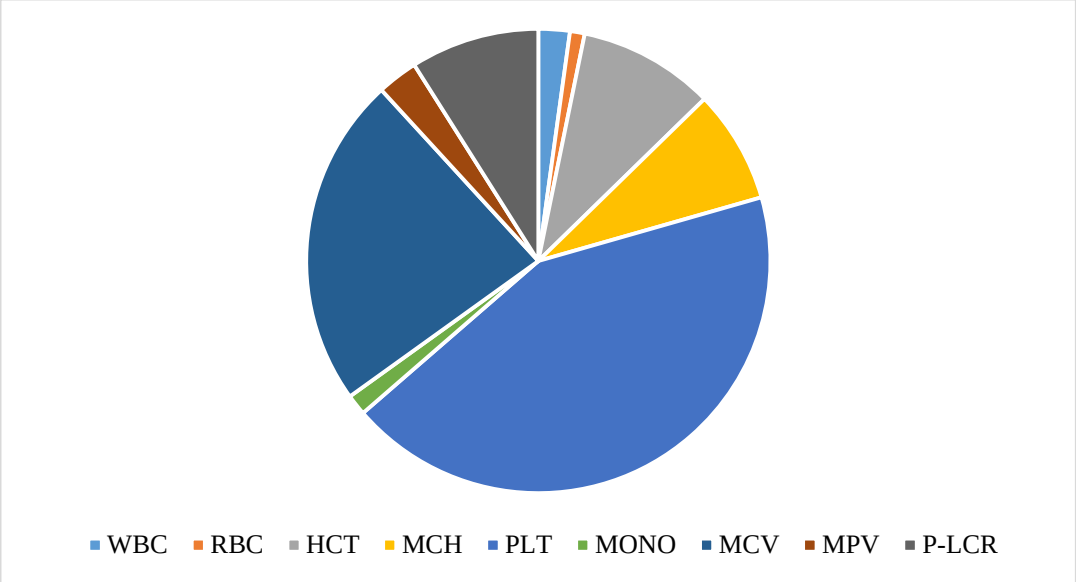

Figure 3. Blood index analysis

According to the statistical analysis of data, as shown in Figure 3 and Table 2, compared with the normal group, there are MONO% (5.18 ±1.79vs5.81 ±1.31%), MCV (90.82 ±6.45vs92.67 ±5.86), P-LCR (39.03 ± 9.76vs36.01 ± 10.51) and MPV (11.79 ± 1.38vs11.37 ± 1.50) were statistically significant (P <0.05). MPV in preeclampsia is higher than normal pregnancy. Diabetes, hypercholesterolemia, acute myocardial infarction, acute ischemic stroke, and elevated MPV in preeclampsia.

(2)Regression analysis

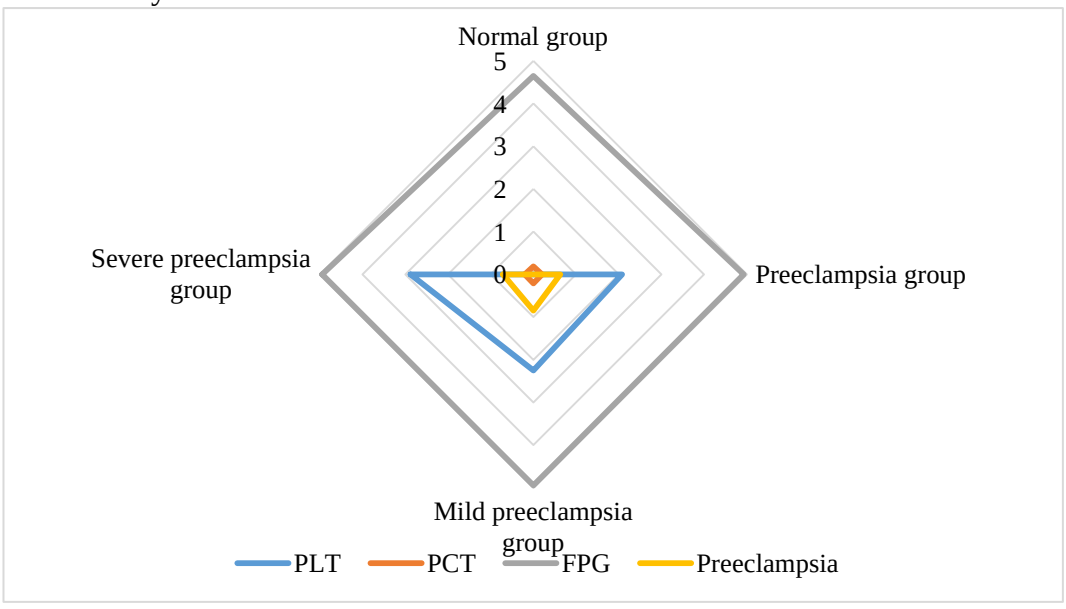

Figure 4. Regression analysis

According to the statistical analysis of the data, as shown in Figure 4, the levels of PLT, PCT and FPG in the preeclampsia group changed significantly. In preeclampsia group, PLT level was higher than normal group, PCT level was higher than normal group, FPG level was higher than normal

group. According to logistic regression analysis, FPG is a risk factor that can choose to build a model of preeclampsia. Establish a probabilistic prediction model for preeclampsia. Pre-eclampsia probabilistic prediction model: pre-eclampsia probabilistic prediction model Y = 0.624 (5'.NT) -1.785, its accuracy rate is 63.4%; probabilistic prediction model of pre-eclampsia Y = I.164 (INHA) +1.804 (basic DBP) + I.695 (pre-pregnancy BMI) -12.967, the accuracy rate is 84.6%; the probability prediction model of late preeclampsia Y = 0.375 (FPG) + 0.739 (basic SBP)-2.676, their accuracy the accuracy rate is 71.1%.

5. Conclusion

(1) Serological studies are closely related to the occurrence of pre-eclampsia, and provide new methods and ideas for the successful prediction of pre-eclampsia, such as high-lead environments, maternal smoking, maternal birth weight abnormalities, etc.; others are associated with pre-eclampsia related serological indicators, genetic polymorphisms, and immune factors are also considered to be useful in the prediction of preeclampsia. With the development of the research on the etiology and pathophysiology of pregnant women with preeclampsia, a noninvasive, simple and accurate method for predicting preeclampsia will be developed in the near future.

(2) The three serological screening indexes in the second trimester are related to the onset of preeclampsia, but only HCG can be used as one of the predictors of preeclampsia.

(3) Establish a predictive model of hypertension in pregnancy. We hope that the predictive model can be effectively and widely used in the early prediction of pregnancy-induced hypertension in pregnant women, so as to achieve early detection, early prevention, early monitoring, and early treatment of high-risk hypertension. The population has complicated pregnancy, and eventually reduces the morbidity and mortality of hypertension.

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Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

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