

Table 4 shows the neonatal outcome in group A and group B. The incidence of preterm delivery and intrauterine growth restriction were higher in severe preeclampsia (50%, 47.5%) as compared

to healthy pregnant. Furthermore, about 2/3 of preeclamptic pregnant have low birth weight infants in comparison to 12.5% of the control group. The fetal death rate in preeclamptic was 7.5%.

Table 4: The neonatal outcome in group A and group B

Neonatal outcome	Group A n (%)	Group B n (%)
Preterm delivery<37week	20(50%)	3(7.5%)
Intrauterine growth restriction	19(47.5%)	2(5%)
Low birth weight<2500gm	29(72.5%)	5(12.5%)
Fetal death	3(7.5%)	0(0%)

Discussion

In this study, we found that serum β -hCG levels were significantly elevated in severe preeclampsia, compared with the controls. The placenta seems to play a fundamental role in preeclampsia, as the condition improves rapidly after its removal. Examination of the placenta in pregnancies complicated by preeclampsia has revealed focal cellular necrosis in the syncytiotrophoblast and increased mitotic activity with cellular proliferation in the cytotrophoblast⁽¹³⁾. The proliferating syncytiotrophoblast in severe preeclampsia is rapidly transformed into syncytiotrophoblast within 72 hours⁽¹⁴⁾.

The normal placenta differentiates during pregnancy with the cytotrophoblast (undifferentiated stem cell) dominant in early gestation and the syncytiotrophoblast (differentiated trophoblast) dominant in late pregnancy⁽¹⁵⁾. Although the mechanism of regulation of gestational hCG remains largely unknown, it is generally accepted that hCG is only secreted by syncytiotrophoblast⁽¹⁰⁾. Barros et al.

found that the microdensitometric analysis of the section from normotensive and preeclamptic placenta indicated that there is statistically significant preeclampsia induced increased in immunohistochemical reaction intensity for hCG, which demonstrate that increase production of hCG by preeclamptic placenta is associated with strong hCG immunostaining of the syncytiotrophoblast⁽¹⁶⁾. Preeclampsia results at least in part from poor trophoblast invasion, thus Bahado et al.⁽¹⁷⁾ found that hCG may play a role in trophoblast invasion and measurement of this identifies women at high risk for developing preeclampsia. In preeclampsia early placental vascular damage leading to decreased oxygen supply might result in an increased hCG production by hyperplastic cytotrophoblast cells⁽¹⁸⁾. Also hCG productions has been shown to increase when normal placental villi in organ cultures were maintained under hypoxic conditions⁽¹⁹⁾.