

INTRODUCTION

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Preeclampsia remains one of the major causes of maternal and foetal morbidity and mortality. The etiology of preeclampsia remains elusive and its treatment is still symptomatic and empirical.

Many theories and etiological factors had been studied to elucidate the cause of preeclampsia. Recently, evidence suggested that a circulating digoxin-like immunoreactive substance (DLIS) may be an important factor in the pathophysiology of preeclampsia (Buckalew and Gruber, 1984).

This substance has been identified in foetal blood, maternal blood and in amniotic fluid of preeclamptic pregnant patient and there was an inverse correlation between this DLIS and both the birth weight and the gestational age of the neonate (Steven and Gordon, 1984; John et al., 1984).

This endogenous digoxin-like immunoreactive substance has been also measured in pediatric population (Phelps et al., 1987) and during different disease state (Greenway and Nanji, 1985).

Several investigators have proposed that digoxin like immunoreactive substance may have an aetiologic role in the development of preeclampsia, since the physiologic activities of this substance include natriuresis (De Wardener, 1982), Na/K adenosine triphosphatase inhibition (Battorff et al., 1986), and peripheral vasoconstriction (Kramer, 1981). However, no data have been reported that clearly established a causal relationship between digoxin-like immunoreactive substance and preeclampsia.