Original article:

Study of gross morophological changes in 50 cases of pregnancy induced hypertension

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Abstract:

Pregnancy induced hypertension is a common syndrome involving 7-10% of pregnancies labelled by clinical criteria BP>140/90, edema and proteinuria .This study is done to analyse PIH changes in gross morphology of 50 placentae examined in ESI-PGIMSR Hospital Basaidarapur delhiI. Gross morphological changes were studied and abnormalities foundwere retroplacental clots , infarcts, edema where p value was found significant while other features like partial membranes ,abnormal weight and abnormal vessels in descending order were not significant.

Keywords: Placenta, Gross, Pregnancy induced hypertension.

Introduction

Pregnancy induced hypertension (PIH) is an important cause of maternal morbidity and mortality throughout the world and its incidence is particularly high among underprivileged women of developing countries (Dawn 1982). Almost 13% of maternal deaths in India have been attributed to PIH and its complications (Park 2001). PIH is defined clinically as hypertension with $BP \ge 140/90$ mmHg at two occasions 6 hrs apart that develops as a consequence of pregnancy and regresses post partum (Cunningham et al 1997). It has been divided into three broad categories: hypertension alone, precelampsia (hypertension, proteinuria and edema) and eclampsia (convulsions precipitated by rise in blood pressure). Out of these three categories, eclampsia is the most dreaded one associated with significant foeto maternal mortality.

Preeclampsia is a common syndrome involving 7-10% of all primigravida, characterized by increased blood pressure, edema and proteinuria. Unpredictable in its onset and progression, clinical manifestations may come about slowly or occasionally, explosively. If untreated, preeclampsia may result in eclampsia, that is convulsions, which is most dreaded of all manifestations and is associated with very high foetomaternal mortality. The disease is much more common in first pregnancy, in very young gravida and in multipara. While the clinical features of PIH and its complications are well known, its exact etiology still remains an enigma (Menon et al 1994). The various theories that have been formulated for its genesis include immunological basis, genetic predisposition, dietary deficiencies, vasoactive factors and endothelial dysfunction (Cunningham et al 1997). There is no specific diagnostic test. It is recognized by pregnancy induced changes that regress afterdelivery.

As PIH has high maternofoetal mortality, most maternal deaths due to PIH are preventable, provided an early diagnosis is made and the treatment constituted at the earliest (Cunningham et al 1997).

The foetus is commonly growth retarded because of fairly characteristic alterations that result from a disturbed interaction between trophoblast and maternal uterine blood vessels. Placental delivery nearly always terminates the illness abruptly. However the preeclamptic syndrome is more polymorphic than the conventional definition, involving maternal liver, nervous system, coagulation (Redman 1990) and is associated with many serious complications such as placental infarction, abruptio placenta and foetal demise.

The clinical signs and symptoms of PIH are however notoriously subtle in the early disease and thus there is need to develop screening tests and criteria for early detection of PIH in antenatal period.

The placenta (term designed from Latin that translates as "flat cake"), an organ of metabolic interchange between foetus and mother, provides oxygen, nourishment and protection to foetus, has excretory and endocrinal functions too. It has been described as the mirror of the maternal and perinatal mortality.

Histopathological examination of placenta as an organ has been difficult as there is an overlap between the findings in normal and abnormal placenta and quantitative studies applied in practice are generally lacking. There are also the questions of who bears the cost of placental examination and of reducing the cost of medical care.

However, the results of placental examination in certain cases do explain perinatal morbidity or mortality and have an impact on management of mother and foetus. In 1989, The College of American Pathologists convened a conference entitled "Examination of placenta, patient care and risk management". Pathologists with expertise in placental pathology, obstetricians, neonatologists and attorneys were invited to participate. The proceedings were published in July 1991 issue of Archives of Pathology and Laboratory Medicine.

PIH is associated with variable degree of placental morphological and histopathological changes like infarcts, retroplacental bleeds, decidual vasculopathies, villous syncytial knotting and rarely molar changes (Lewis et al 1990). The study of these changes can provide an insight into the etiology of PIH

AIMS AND OBJECTIVES- study of gross morphological changes in 50 cases of pregnancy induced hypertension.

Materials and methods

This is a prospective study of fifty patients conducted in Department of Pathology, ESI Hosiptal, Basaidarapur who attended antenatal clinic and were admitted further in the labour room in the Department of Gynae & Obstetrics. A detailed clinical history was obtained including headache, epigastric pain, nausea, vomiting, visual

symptoms, respiratory symptoms, past history and other relevant history. General examination was done including Pulse, B.P., Pallor, cyanosis, pedal edema. Systemic examination, antenatal and fundus examination were done in all patients as relevant to PIH.

Laboratory investigations were performed including Hb, complete haemogram and blood group, urine (albumin), blood urea, serum creatinine, uric acid, LFT, Platelet count.

Criteria for selection of the patients in our study was:

- 1. Pregnant females followed up from the first trimester and who developed B.P. more than 140/90mmHg at two occasions six hours apart (with previous normal B.P.) after 20 weeks.
- 2. No previous or any present kidney disease
- 3. No H/o primary or previous hypertension in the past.
- 4. With normal functioning of liver, cardiovascular system and other systems.

The selected females were followed up in antenatal clinic and delivery was performed vaginally or by caesarean section. Placentae were collected and sent to histopathology lab, examined fresh and fixation was done to evaluate further. Gross abnormality was observed, photographed and noted. Weight was taken in the fresh specimen. Gross features were noted like cord length measurement, membranes knots, retroplacental clot, any infarct in the fresh specimen itself.

Examination of foetal placenta was done for any abnormality. Maternal surface was examined for completeness, tears, plaques, adherent clot for any missing cotyledon, membrane.

Observations and results

In the present study, gross examination of placentae of PIH cases were carried out .In our study, maximum cases (46%) were seen in age group 29-33 yrs followed by 34% in 19-23 yrs, 16% in 24-28 yrs, and 4% in 34-38 yrs group with mean age 26.66 and standard

deviation 4.885,

Parity ranged from P1 to P6 in our study. Maximum percentage of cases were primipara (P1) i.e. 32% with mean 2.22 and SD 1.130 .Most prominent symptom in our study was nausea and vomiting (60%) followed by headache (58%) epigastric pain (54%)and visual symptoms (28%) in descending order 6% cases had positive family history while 94% were found negative B.P. was most important criteria for selection of cases. BP >140/90mmHg was considered as PIH group, so this was 100% followed by pedal edema 62% and fundus changes in 14% The cases with raised BP were further subdivided into mild and severe (>160 /110mmHg) categories. 40 cases were in mild PIH group (BP more than 140/90mmHg but less than 160/110mmHg) while 10 cases were in severe PIH group (BP more than 160/110mmHg)

In our study percentage of cases showing positive urine albumin was 74%, raised LFT was 50%, raised KFT was 20% and abnormal platelet count was 22% Gross findings-In the present study, gross examination of placenta was done. Weight between 345-617gm was taken as normal. 6% placentae had weight less than 345gm. 34% cases showed retroplacental clot. Infarct involving >5% area was taken as abnormal and 32% cases had significant placental infarct. Partial membranes were seen in 14%.

Umbilical cord examination was done and features observed were cord vessels; one case (2%) had 1A/V rest had 2 A/V, 6% cases showed umbilical cord knots while cord edema was seen in 11% cases.

Table

Gross Pathology	No. of cases	Percentage
Weight	3	6%
Retroplacental clot	17	34%
Infarcts	16	32%
Membranes (partial)	7	14%
Umbilical cord		
Vessels	1	2%
Knots	3	6%
Edema	11	22%

Discussion

Pregnancy induced hypertension is an important cause of maternal morbidity and is a disease of particularly underprivileged women of developing countries.

Incidence of pre-eclampsia was reported 10% in rural India, Mudaliar and Menon 1972 and 3-10% by Lewis in 1965. High incidence in developing countries with low socioeconomic status is attributed to the lack of antenatal care. This study was conducted in Departments of Pathology, Gynae and Biochemistry, ESI (Employees State Insurance) hospital in Basaidarapur, New Delhi. ESI corporation is a social security scheme which provides comprehensive medical care as one of its benefits to the factory workers; this hospital mostly caters to many factory workers who have low socioeconomic status.

The present study was a prospective study of pregnant females attending antenatal clinics in OPD, followed up in antenatal clinics and those who developed hypertension (PIH) were selected for the study; their 2nd and 3rd trimester hormonal assays were done. The number was confined to 50 and 50 cases were taken as control group. Age ranged from 19 years to 38 years in this study with mean age 26.66, SD 4.886. Maximum number of cases were found in 29 to 33 years group. Cuesley 1985 studied PIH and concluded that it more frequently affects teenagers or those older than 35 years, and pre-eclampsia in older women is more likely pregnancy aggravated hypertension. Parity ranged from P1 to P6 in our study. Maximum number of cases were primipara (32%) with mean parity 2.22±1.130.Lewis 1965, Mudaliar and Menon 1972, Dawn 1974 have also concluded that pre-eclampsia is more common in primigravidae (70%). In our study the most prominent symptoms were nausea, vomiting 60% followed by headache 58%, pain abdomen 54% and least were visual symptoms 4%. Family history of high BP in parents was found in 6% cases only.

50 cases were taken in the study which showed high BP >140/90 after 20 weeks of gestation on two occasions 6 hrs apart with or without edema and proteinuria. 40 were labelled as mild PIH 140/90mmHg but <160/100mmHg and 10 cases as severe PIH (BP >160/110mmHg). Cases with eclampsia were not included in the study. Pedal edema was seen in 62% followed by fundus changes in 14% cases. Biochemical parameters found abnormal in the study were albuminuria in 74%, impaired LFT in 50% cases, impaired KFT in 20% and low platelet count in 22% cases. Test of significance was calculated for cases with high blood urea, S. creatinine, high serum uric acid and low platelet count in severe PIH group, was <0.05, which is in conformity with other workers (Simon Shehav et al 2002, Dunlop and Donaldson 1977, Radman et al 1976).

Gross examination of placenta was done. 6% cases had weight less than normal (345-617gms) in severe PIH group. 34% cases showed retroplacental clot. Infarct area involving >5% was taken abnormal and 32% cases had significant placental infarct. Partial membranes were seen in 14% cases only.

Test of significance was calculated and P value was <0.05 for retroplacental clot, cord edema and infarct which is consistent with other workers who studied placental morphology in PIH (Mathews et al 1973, Maqueo et al 1964, Ker AU het al 1981

Summary and conclusions

This is a prospective study of 50 pregnant women with PIH, attending antenatal clinics in ESI Hospital, Basaidarapur. The control group consisted of 50 pregnant females who had normal B.P. through out their preganency. Clinical history, general physical examination, systemic and antenatal examination were conducted on all these cases.

Placentae of the 50 study and 50 control cases were collected at the time of delivery. Gross and microscopic examination of the placentae were carried out. The data was compiled and results of investigations were compared in study and control group Gross morophological changes were studied and abnormalities found were retroplacental clots, infarcts, edema where p value was found significant while other features like partial membranes, abnormal weight and abnormal vessels in descending order were not significant.



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