Abdominal Pregnancy Associated with Toxemia of Pregnancy*

Report of Two Cases and Review of the Literature

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THE proposed etiologies for toxemia of pregnancy found in the literature are legion. From Greenhill's latest edition of "Obstetrics," there are approximately 19 different etiologies listed, ranging from specific placental toxins to dietary deficiencies. Each of these theories explains this condition in its own way.

Greenhill[1] states that the etiology of pre-eclampsia and eclampsia is unknown, but the text goes on to state further that the hypothesis currently favored is that of uterine ischemia, with increased myometrial tension as a suggested mechanical cause for this uterine ischemia.

Sophian[2] in 1953, published an article, "Myometrial Resistance to Stretch, the Cause of Pre-Eclampsia." His study showed that tonus of the uterus was increased by mineral corticoids and sodium during pregnancy. Eighty-eight per cent of the toxemics had increased uterine tonus when the blood pressure was greater than 140 mm./Hg systolic, and he also noted that blood pressures increased during labor due to the increased uterine tonus.

He proposed that toxemia of pregnancy was due to overdistention of the uterus producing a "utero-renal reflex." His study showed that increased resistance to stretching of the uterine muscle produced a reflex inhibition of the renal blood flow, mediated by nerve impulses from the uterus through the pre-sacral plexus, leading to various degrees of renal cortical ischemia. Renal ischemia can produce hypertension through the release of renin; albuminuria by impairing the circulation of the afferent glomerular vessels and its capillary tuft, and edema by lowered glomerular filtration and subsequent sodium retention.

Sophian's work laid the foundation for the theory that overdistention of the uterus produces toxemia in pregnancy.

The rationale today for the higher incidence of toxemia in the primigravidas, the multiple pregnancies, hydatidiform moles, and hydramnios is predicated on this theory of overdistention of the uterus.

Recent observations in our hospital, and a review of the literature, prompted the writing of this paper to present two cases of abdominal pregnancy associated with toxemia of pregnancy and discussions from the literature with similar observations.

Barnes,[3] of Baltimore, in a discussion of Beacham's 1962 paper on "Abdominal Pregnancy at Charity Hospital in New Orleans," made the following statement: "On one of Dr. Beacham's slides there is a patient listed as having abdominal pregnancy with toxemia. This becomes of more than average interest. One would like to know whether the placental implantation was on the back side of the uterus or on the mesentary, and whether the diagnosis of toxemia was carefully established, or whether this was essential hypertension, so much of the current thinking about toxemia revolves around ischemia of the placental uterine interface with the production of an unknown substance at that area. If we have a good clear-cut eclamptogenic toxemia in a patient who did not have such an interface relationship, we would have a finding of significance."

Beacham,[4] in his answer to this discussion by Dr. Barnes, confirmed the diagnosis of toxemia. He further states, "She had the usual criteria that goes with such a diagnosis (toxemia), and we were impressed in this regard, too."

Barnett,[5] in a paper on abdominal pregnancy, noted an interesting case which on the third postoperative day became frankly eclamptic following mild pre-eclampsia present before surgery. He could not be sure that an interface between placenta and uterus did not exist, but it was not apparent at the time of surgery.

Recently, from England, Benjamin[6] reported two cases of abdominal pregnancy associated with

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toxemia of pregnancy. In his discussion he states, "The hypothesis of Sophian (myometrial stretch as the etiology of toxemia) is in many ways plausible and has received much publicity in recent years. However, we have recently encountered two cases of severe pre-eclamptic toxemia where uterine distention was non-existent and where this theory is completely untenable."

Benjamin feels the occurrence of toxemia in cases of extra-uterine pregnancy would seem to show conclusively that intra-uterine distention is by no means the cause of toxemia. At the most, it may simply be a factor in aggravating toxemia or producing it in a few cases.

We now present two cases of abdominal pregnancy associated with toxemia of pregnancy, seen at our institution.

CASE REPORTS

Case No. 1: July 1948—C.W., a 35-year old Gravida 2, Para 1, was approximately 36 weeks gestation at the time of admission. Her chief complaint was swelling of the hands and feet with headache.

Past History: The patient had no past history of hypertension or kidney disease.

Physical Examination: Her blood pressure was 160/100. Her face, hands and feet were edematous. There was 3+ pitting edema of the lower extremities. Normal fetal heart tones were heard. The fundus was the size expected at that gestational age.

Laboratory Studies: Albuminuria was present.

Hospital Course: The patient was placed on ammonium chloride, a salt-free diet and sedation. Weight loss of 10 lbs. was produced after a week of hospitalization. The patient then signed herself out of the hospital.

Readmission: The patient returned 3 weeks later, approximately 38-39 weeks gestation. No fetal heart tones were heard and blood pressure was normal. A flat plate of the abdomen revealed findings suggestive of fetal death in utero. Stimulation of labor was attempted with IM Pitocin with no contractions. A rectal tube was then inserted into the uterus with no effect. A review of the x-rays with the rectal tube in place gave the impression of an abdominal pregnancy. A hysterosalpingogram was confirmatory. At surgery a dead fetus was found and the placenta was located on the anterior surface of the uterus involving the omentum and abdominal viscera. There was partial separation of the placenta at the time of surgery.

Case 2: September-October 1965—M.H., a 33-year-old Gravida 4, Para 3, was approximately 36-38 weeks gestation. The patient had a history of headaches for the 3 days prior to admission. She was seen in the clinic on her first visit and was admitted to the hospital.

Past History: The patient denied elevated blood pressure between pregnancies, in fact, she stated she had "low blood pressure."

Physical Examination: Her blood pressure was 160/110; pulse 80; respiration 16/min. and temperature 98.6°. Her general physical examination was within normal limits except for obesity. The patient weighed 222 lbs. upon admission. The fundus was approximately 26 cm. above the symphysis. Fetal heart tones were normal. The cervix was long and closed. There was 3+ pitting edema.

Admitting Diagnosis: Intra-uterine pregnancy, approximately 36-38, with pre-eclampsia.

Laboratory Studies: Hct. 28; Hgb. 9.5; WBC 11,100; BUN 12, Albumin trace.

Hospital Course: The patient was placed on a low salt diet, Phenobarbital, Esidrix K, and bed rest. She had a weight loss of 19 lbs. after 11 days of hospitalization, and was discharged after the 12th day with blood pressure 130/90. During hospital stay, patient complained intermittently of vague lower abdominal pain. She was discharged with medication and a low calorie, salt-free diet.

Readmission: The patient was readmitted 4 days after discharge. At that time she complained of intermittent lower abdominal pain, crampy in nature, lasting approximately 20 seconds, with passage of dark red blood, approximately half a cup. The blood pressure at that time was 120/78, Pulse 96. The abdomen was described as being tender in the lower left quadrant. Moderate enlargement of the abdomen was present. The uterus was described as not definable, however, there was a mass, mostly on the right side, about 4 finger breadths above the umbilicus. On vaginal examination the cervix was long and closed. Tissue described as placental was found in the vagina and the uterus was enlarged to about 4-5 months gestation.

The admitting diagnosis was incomplete abortion, but after examination of a flat plate of the abdomen and a term size fetus with a collapsed skull in a transverse lie high in the abdomen was observed, a diagnosis of abdominal pregnancy was made. At surgery the placenta was found to be attached to the posterior surface of the uterus, broad ligament, adnexa and infundibulo-pelvic ligament on the right and the omentum.

A further observation was made concerning these cases at our institution. We have had 28 cases of abdominal pregnancy during the period from 1946 to 1966. These 2 cases presented constitute an incidence of seven per cent of toxemia in the cases of abdominal pregnancies. The incidence of toxemia at our hospital recently has ranged between three and six per cent; the incidence in the United States in the recent texts places the figure at 6 per cent. Because of the small number of cases (28), this is not statistically significant but is an interesting finding.

In Eastman's[7] "Obstetrics," it was pointed out by Zweifel that so many theories have been advanced concerning the etiology of eclampsia that he referred to it as "the disease of theories."

We do not propose to add to this list of theories but have presented what we feel are

significant findings from our institution and from the literature which refute one of the theories—that of overdistention of the uterus which, at present, is currently favored as the etiology of toxemia of pregnancy.

Sophian has stated that the elucidation of the etiology of toxemia of pregnancy ranks as the outstanding need in obstetrics, for upon it depends the rational treatment and the eradication of this complication.

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INFANT MORTALITY

The *infant mortality* rate in the United States in 1963 was the lowest ever recorded—25.2 per 1,000 live births. However, since about 1950, there has been a slowing down in the rate of decrease of infant mortality, from about 4.3 percent annually during the period 1933-49 to 1.0 percent annually between 1950 and 1963. In addition, certain foreign countries are experiencing lower infant mortality rates than the United States.

Sex and color differentials in infant mortality for 1963 show little change over recent years. Mortality rates for male infants were consistently higher than those for female, with an excess of about 30 percent in deaths among male infants. Nonwhite infants had a mortality rate twice that for the white group.

Diseases of the respiratory system and congenital malformations led the list among specific causes of infant deaths, but half of the deaths were attributed to general conditions peculiar to infants. The picture changed, however, when age of the infant was considered. Causes of neonatal deaths (under 28 days) followed the pattern of total infant deaths, with rates about 1.5 time greater for nonwhites than for whites for all causes except congenital malformations. During the postneonatal period (28 days to 11 months), pneumonia, except pneumonia of the newborn, was the principal cause of mortality.

Infant mortality rates varied by geographic area, ranging from a low of 18.6 per 1,000 live births in Utah to 41.3 in Mississippi. Rates for nonwhite infants were higher than those for white infants in nearly all States.

For white infants, the mortality rate was lowest for rural areas in metropolitan counties; nonwhite infants had lower mortality rates in the urban parts of metropolitan counties. Mortality rates were higher in the South region for both white and nonwhite infants.

Fetal mortality has declined steadily over the past twenty years. There is little doubt, however, that only a fraction of the actual number of fetal deaths is registered, mainly because of the variation in reporting requirements among registration areas. The total number of fetal deaths reported in 1963 was 94,194, a ratio of 15.8 fetal deaths per 1,000 live births. The nonwhite ratio was twice as high as the white ratio, but figures for both groups were the lowest ever recorded.

Fetal-death ratios for married women were lower in almost every age category than those for unwed mothers among both white and nonwhite women.

The fetal-death rate among twins was almost three times that for single births and even higher for other plural deliveries. Fetal loss was lowest among second pregnancies.

The *maternal mortality* rate has declined considerably since this statistic was first reported in 1915. There were 1,466 maternal deaths in 1963, a rate of 35.8 per 100,000 live births. Toxemia accounted for a third of the maternal deaths. Maternal death rates were about four times greater for nonwhite women than for white, a differential considerably more substantial than for either infant or fetal mortality. Maternal loss increased with age for both white and nonwhite women.

The number of maternal deaths for individual States was too small to provide statistically reliable rates. Combined data for 1961-63, however, show that the level of maternal mortality varied extensively among the 50 States and the District of Columbia, from a low of 11.2 maternal deaths per 100,000 live births in Minnesota to 84.1 in Mississippi.

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