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ACUTE RENAL INSUFFICIENCY IN PREGNANCY

WITH SPECIAL REFERENCE TO

BILATERAL CORTICAL NECROSIS OF THE KIDNEYS

Rodney Miller Thompson

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Introduction

Only recently has acute renal insufficiency
received serious consideration as a major compli-
cation in pregnancy. Although brief mention will
be made as to the etiologies of this complication,
the major portion of this paper is devoted to a
discussion of a unique lesion of the kidneys, bilateral
cortical necrosis. This disease occurs most frequently
following abruptio placenta and runs a course which
may be clinically undistinguishable from that of acute
renal failure. It has been only recently,,following
the isolation in 1948 of 5-hydroxytryptamine, that
an attempt has been made to explain its more frequent
occurrance in pregnancy complicated by premature
separation of the placenta.

Etiologies of

Acute Renal Insufficiency in Pregnancy

The etiologies of acute renal failure in pregnancy
are diverse. The classification of Slowinski (1)
presents the etiologies in a concise but complete form.

Group I - This group is dominated by peripheral
circulatory failure, "shock", or shock-like
states. Premature separation of the normally
implanted placenta, postpartum hemorrhage,
and ectopic pregnancy are a few examples. The
pregnant woman with her increased blood supply
would seem to be protected from shocking
circumstances leading to acute renal insuffic-
iency, but this is only partially true. Some
organs are more susceptible than others to an
abrupt deprivation of their blood supply.
These are the kidney and the pituitary and
adrenal glands because of their hypertrophy
and concomitant increased blood supply.
Group II - The excretion of pigment breakdown
products resulting from tissue destruction
such as hemolysis following incompatible blood
transfusions comprises this group.

Group III - Sensitizing agents or substances
which are directly nephrotoxic are included
here.

It will be observed in the following discussion
that all etiologies mentioned will not directly coincide
with specific examples given previously but will fall
into one or more of the three groups.

Swann and Merrill (2) reported several cases of
renal insufficiency in pregnancy in a review of eighty-
five cases of acute renal failure from all causes.
They noted that in a great percentage of their obstetric
cases renal failure resulted from some accidents or
incidents of pregnancy, such as premature separation of
the placenta, mismatched transfusions, hemorrhagic shock
including postpartum hemorrhage, and the use of aborte-
facients. All of these patients recovered except one,
and the cause of her death was not attributed directly
to renal disease.

Russell, Maharry, and Stehly (3) grouped their series
of thirty-four patients according to the period of
gestation in which renal failure became manifest.
During the first trimester septic induced abortions were
the major cause. Circulatory factors such as shock and
vasospasm were not major considerations. Twenty-seven

or approximately eighty percent of their cases were in this group. During the second trimester,,two of the three cases were septic induced abortions and the other was a case of abruptio placenta. All four cases of acute renal failure during the third trimester were attributed to premature separation of the placenta. In those cases circulatory disturbances were a major factor, and the degree of damage to the kidneys was directly proportional to the duration of the circulatory disturbance.

It was Russell's belief that some degree of utero-placental damage was common to renal failure in those patients and that even when other conditions were present, the underlying abnormal condition resided in the disturbed uterine and placental or decidual vascular relationships.

Schreiner and Berman (4) thought that shock was probably a common denominator in many of their eighteen obstetric patients with acute renal failure. Other factors which they considered to be primary were abortion, eclampsia, Clostridial infection, intravascular hemolysis, and abruptio placenta. The cases in their group were selected in part on the basis of "a twenty-four hour urine volume of less than four hundred milliliters in the absence of hypertension or mechanical obstruction".

Spickman (5) formed the opinion that the oliguria of eclamptic patients was especially common after delivery and was usually accompanied by a lowered blood pressure. In 1945, Adam (6) thought it significant that death in many cases of eclampsia was not due to uremia but to symmetrical cortical necrosis of the kidneys. This renal lesion was first described by Juhel-Renoy (7) in 1886.

It is interesting to note that abruptio placenta is usually accompanied by pre-eclampsia or eclampsia and that symmetrical cortical necrosis of the kidneys in pregnancy is limited to cases of premature placental separation.

Symmetrical Cortical Necrosis of the Kidneys

In 1933, Ash (8) made a complete survey of pertinent literature up to that time and was able to find forty-two cases which he considered to be authentic. Forty-five new cases were reported in the seven years following his report. This increase in the number of reported cases does not necessarily indicate an increasing incidence of the disease but rather points to an increasing interest in and recognition of this peculiar

lesion of the kidneys.

Duff and More (9), in reporting their cases in
1941, analyzed only those in which the diagnosis was
established beyond doubt by postmortem examination and
adequate description of the pathological changes. The
renal lesions themselves were of identical character in
all cases as were the clinical signs and symptoms
referable to these lesions. Forty-eight of the seventy-
one cases analyzed occurred at the termination of
pregnancy and were associated with pre-eclampsia or
eclampsia. They believed that even if toxic substances
in the blood were capable, under certain conditions, of
causing bilateral cortical necrosis of the kidneys, it
was also possible that the disease could occur without
any evidence that toxemia had played an etiologic role.
Seventeen of the thirty-three reported cases between
1933 and 1941 occurred in males or non-pregnant females.
This suggested that some individuals are predisposed to
develop bilateral cortical necrosis when exposed to the
influence of any one of a number of definite toxic
substances, or to the influence of unknown and in some
cases undetected general diturbances of metabolism or
vasomotor function.

According to Oertel (10) the whole vascular system
in pregnancy shows decided disturbances of its general

irritability, and such disturbances may affect the
vasculature of individual organs, such as the kidneys,
leading to long continued local or general vasocon-
striction which may give rise to vasoparalysis.

Although most authors agree that necrosis of the
renal parenchyma is ischemic in origin, there is much
divergence of opinion as to the cause of the ischemia.
Juhel-Renoy (7) expressed the opinion that the renal
lesions were caused by multiple emboli. Most writers
now reject the embolic theory because of the absence
of a source for the emboli, the lack of evidence of
embolism in other organs, and the uniformity of
distribution of the cortical necrosis throughout both
kidneys in almost all the reported cases up to 1941
including that of Juhel-Renoy.

A great number of authors have attributed the
necrosis of the renal cortex to ischemia caused by
thrombosis of the small cortical arteries. Bradford
and Lawrence (11), Carson and Rockwood (12), Dalrymple
(13), Evans and Gilbert (14), and others agree in
suggesting that some form of toxic damage to the
endothelium of the cortical arteries is of prime
importance in the cause of thrombosis in those vessels.

Ischemia leading to necrosis of a peripheral layer

of cortical tissue of varying thickness must be
attributed to an organic or a functional occlusion
of large numbers of the minute "end arteries which
supply that zone of the cortex". The areas of
cortical necrosis in any given case all seem to be
about the same age leading one to believe that vascu-
lar occlusion must become effective almost simultan-
eously in all parts of the cortex in both kidneys.

Richards and Schmidt (15) showed that the injection
of adrenalin caused arteriolar constriction which was
most pronounced in the glomerular arterioles at the
point of division into glomerular capillaries. Spon-
taneous contractility was also observed at this point.
De Novasquez (16) and others interpreted those experi-
mental observations as indicating a special suscepti-
bility of the small cortical arteries to various
stimuli.

By the daily intrapleural or intraperitoneal admini-
stration of massive doses of epinephrine into dogs for
periods of six to thirty days, Penner and Bernheim (17)
produced hemorrhagic necrosis of individual glomeruli
or of small patches of the renal cortical tissue.
Although the extent of the necrosis scarcely approximated
that observed in the kidneys of man, those experiments

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provided strong evidence for support of the view that
intense and prolonged vasospasm may be responsible for
the development of bilateral cortical necrosis in man.
Oertel (10) also thought this lesion was dependent upon
the ischemia produced by vasomotor disturbances, but he
constructed a rather complicated explanation including
various picture of the kidneys representing "the results
of irritations of the terminal arterial segments of
different intensities".

In 1944 Zucker (18) reported a study of the sub-
stances in blood serum and platelets which stimulated
smooth muscle. In her experiments injections of cat,
dog, human, or rabbit serum produced smooth muscle
contractions. In nine out of eleven instances fresh
citrated cat plasma had no action on the smooth muscle
being tested. Addition of citrate to serum did not
modify the effect on the smooth muscle, and the vaso-
constriction produced by plasma was approximately one-
fourth that produced by an equal volume of serum.

The origin of the active substance was studied by
testing the activity of lysed cat blood platelets, white
cells, and red cells on the smooth muscle and the
activity of similar fractions of rabbit blood cells on
the intestines of rats and on the vessels of the rabbit

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ear. A sample of the platelet fraction caused a
greater contraction of all three indicators than did
an equal sample of the red cell fraction. The concen-
tration of the active substance in the platelets was
at least eighty times that in the red cells, and the
extracts of red cells prepared from defibrinated blood
had less effect on the smooth muscle than did similar
fractions prepared from citrated blood. Therefore,
although the coagulation released the active constituent
from the platelets, it did not cause the production of
any additional smooth muscle stimulants.

        This substance, 5-hydroxytryptamine, was isolated
in 1948 by Rapport, Green, and Page (19) and was named
Serotonin. By comparing the quantity of iodine required
for inactivation of pure Serotonin and the concentrate
from serum, Page (20) obtained evidence favoring the
view that Serotonin is solely responsible for the vaso-
constricting activity of serum. He also noted that the
vascular response to Serotonin is highly variable and is
best described by the term "Amphibaric" since it is
usually both pressor and depressor but may be only one
or the other. The blood vessels of the perfused
innervated dogs' kidneys are very sensitive to Serotonin.
In contrast with the partial or occasionally entirely

depressor responses to Serotonin in intact animals, responses when injected into the renal circulation were uniformly vasoconstrictive, but the doses were usually large. The minimum antidiuretic dose of synthetic Serotonin was found to be about four micrograms per kilogram of body weight. This is believed to be one-fiftieth to one one-hundredth of the amount which would alter arterial pressure.

From the various lines of investigation which, up to now, have been unrelated, a better understanding has been obtained concerning the systemic changes which occur in the severe grades of abruptio placenta. Among the important effects upon the mother are a degree of shock out of proportion to the hypotension, a disseminated fibrin embolism, an in-vivo defibrination sometimes resulting in incoaguable blood, and ischemia of the renal cortex which leads to varying degrees of necrosis. Page, King, and Merrill (21) believed that an infusion of biologically active materials from the separation site into the maternal circulation is responsible for all events.

In severe cases of abruptio DeLee (22) in 1901 noted that there was hemophilia-like state, and Dieckmann (23) reported finding very low levels of fibrinogen. In 1948 Kellogg (24) postulated that

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defibrinogenation was due to the entrance of thrombo-
plastin into the maternal circulation. This was based
on Schneider's (25) demonstration that the lethal effects
of placental extracts were due to their thromboplastic
activity. The entrance of tissue extracts into the
maternal circulation results in, what he has termed, a
disseminated fibrin embolism which has been associated
with some cases of eclampsia and renal cortical and
pituitary necroses.

The classic monograph on renal cortical necrosis by
Sheehan and Moore (26) in 1953 presented evidence that
the lesion results from a spasm of the intra-lobular
arterial tree, but that the cause of the spasm was
unknown. That this lesion occurs more frequently with
abruptio placenta than with any other clinical entity
and is not necessarily associated with hypotension has
become a well documented fact.. Approximately one-third
of the deaths associated with severe abruptio placenta
result from renal cortical necrosis and conversely the
great majority of deaths from renal cortical necrosis
follow abruptio placenta.

As has been previously mentioned, the shock which
accompanies abruptio placenta is out of proportion to
the mild or even absent hypotension. It was felt that
this can be explained by the frequent association of

toxemia with abruptio. In light of this, Page and
Glending (27) considered the possibility that a vaso-
constricting substance formed or liberated during the
episode of abruptio placenta might be responsible for
the renal cortical ischemia which results in necrosis.
First consideration, therefore, was given to the serum
vasoconstricting substance, 5-hydroxytryptamine or
Serotonin.

In normal blood, all the Serotonin is presumed to
be in the platelets although it is formed elsewhere in
the body by Chromaffin staining cells of the intestine
and central nervous system. There has been a paucity
of reports of platelet counts in humans during the acute
episodes of abruptio, but if man behaves like the dog,
one could expect a marked reduction or virtual disappear-
ance of platelets from the circulation at this time.
Erspamer (28) stated that in the rat Serotonin was anti-
diuretic due to "an elective action of the substance on
the contractile structure of the afferent arterioles of
the glomeruli". Page and Glending (27) and Corcoran,,
Masson, Del Greco, and Page (29) did not support this
view because small doses given intravenously to dogs
did not reduce renal blood flow regularly, and daily
subcutaneous doses in rats produced no renal lesions..
Page and Glending, however, did produce renal cortical

necrosis in rats following a continuous intravenous
infusion of Serotonin. In numerous fields the micro-
scopic picture of the rat kidneys was morphologically
almost identical to that so characteristically found
in fatal cases of human renal cortical necrosis.

In view of their findings, they postulated a
sequence of events which is stated as follows. "The
area of concealed retroplacental hemorrhage contains
native extracts of placental and decidual tissue high
in thromboplastic potency. The hydrostatic pressure in
this area is sufficiently great to cause infiltration of
blood through the myometrium, force these tissue extracts
into the venous sinuses, and then into the maternal
circulation. These extracts, adsorbed partially to
fibrin, are removed rapidly from the maternal circulation
by the liver, lungs, and other organs but serve to
initiate the coagulation process. Prothrombin is con-
verted to thrombin which in turn converts the fibrinogen
to fibrin. The same tissue extracts probably convert
plasminogen to plasmin resulting, in most cases, in
active fibrinolytic activity in the maternal blood. The
thrombin also causes platelet lysis, and a large mass of
platelets disappear from the circulation releasing
Serotonin into the maternal circulation. The Serotonin

causes intense vasoconstriction in the renal cortex
which, if persistant for several hours, leads to
varying degrees of renal cortical necrosis."

Clinical observation has shown that anuria or
extreme oliguria mark the onset of the disease. Tran-
sient hematuria may also be present in the early stages.
Ricker (30) outlined three stages in the pathogenesis of
cortical necrosis and correlated them with the clinical
course of the disease. The first stage consisted of
renal cortical hyperemia due to vasodilitation with an
increased blood supply in the kidney but a decreased
supply through the glomeruli, the latter explaining the
oliguria and the former explaining the transient hema-
turia. Stage two, renal cortical ischemia, is due to
vasoconstriction and is clinically manifested by a
severe degree of urinary suppression often amounting to
an anuria, a disappearance of blood from the urine, and
a rising Blood Urea Nitrogen. In the third stage the
renal cortical necrosis following the formation of
thrombi is characterized clinically by an almost complete
anuria leading to azotoxemia, terminal uremic manifesta-
tions, and death towards the end of the second week.
The theoretically irreversible changes of stage three
could be prevented if arteriolar vasoconstriction could

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be relieved at least temporarily and if some diuretic
substance could increase the excretory capacity of such
cortical tissues.

In Duff and More's (9) series of cases the ages of
the patients who had the disease at the termination of
pregnancy were distributed throughout the entire child-
bearing period. Almost two thirds of the cases occurred
in women from age thirty upward with the youngest being
nineteen and the oldest being forty-eight. Exactly one
third of the ages were between thirty and thirty-five
inclusive. The number of previous pregnancies had no
relationship to the occurrance of renal necrosis. Pre-
mature delivery occurred with striking frequency and
resulted in the birth of a dead fetus in all cases
except one. The month of gestation was recorded for
thirty-six of the forty-eight cases with prematurity
in thirty-three cases occurring in the fifth to eighth
month. It is interesting to note that twenty-one cases
of premature delivery were explained by premature
separation of the placenta and eight cases by the
occurrance of pre-eclampsia. Convulsions predated the
anuria in twelve cases, and headaches or disturbances
of vision or both were mentioned as occurring in one
half the cases, frequently beginning a week or so before
delivery.

The time of appearance of the anuria was noted
in forty-five of the forty-eight cases. In thirty-
six, or seventy-five per cent of them, the anuria began
on the day of delivery. Five occurred within three days
after delivery, and the anuria preceded delivery in
four cases. All of the latter had shown signs of some
form of toxemia before the anuria commenced. Several
of the patients had complained of pain or tenderness
in either the epigastrium or loins or of pain beginning
in the epigastrium and radiating to the loins. Complete
anuria persisted in some cases from the first day until
death. In those patients who survived the longest, a
period of complete anuria was followed by the excretion
of small quantities of urine. Mortality was as high as
forty-four per cent in the eclamptic patient if oliguria
lasted more than twenty-four hours and as high as nineteen
per cent if it lasted in spite of treatment.

Summary

The importance of acute renal insufficiency as
an obstetric complication has been recognized only
within recent years. There have been many factors
responsible for this complication; some of which have
been mentioned in this paper. Symmetrical cortical
necrosis of the kidneys in cases of abruptio placenta
is unique among the other factors. It was Adam in
1945 who noted its significance in relation to acute
renal failure in abruptio placenta and eclampsia.
Oertel pointed out that the whole vascular system in
pregnancy shows a decided irritability and that such
disturbances could affect the vasculature of individual
organs such as the kidneys leading to a long continued
vasoconstriction. Most authors have agreed that the
necrosis of renal parenchyma is ischemic in origin.
The cause of the ischemia, however, has caused some
divergence of opinion. Juhel-Renoy believed it was
caused by multiple emboli; others believed it was the
result of small cortical arterial thrombosis, while
still others suggested that some form of toxic
damage to the endothelium of the cortical arteries was
a prime factor leading to thrombosis. Penner and
Bernheim produced hemorrhagic necrosis of individual

glomeruli or of small patches of renal cortical
tissue in dogs by daily intrapleural or intraperi-
toneal administration of massive doses of epinephrine.
Although the extent of the necrosis was not as great
as that observed in man, these experiments provided
evidence in support of the view that intense and
prolonged vasospasm of the small cortical arteries
could be responsible for the development of bilateral
cortical necrosis in man.

In 1948 Rapport et al isolated 5-hydroxytrypta-
mine, the vasoconstricting substance of serum. In
normal blood it is presumed that all this substance
is in the platelets. It was found that the blood
vessels of dogs' kidneys are very sensitive to
Serotonin, 5-hydroxytryptamine, and that the response
was uniformly vasoconstrictive. A marked reduction
or virtual disappearance of platelets from the circu-
lation was also noted, and if man behaves like the
dog one could expect the same to be true during the
acute episode of abruptio when complicated by acute
renal failure due to bilateral cortical necrosis of
the kidneys.

Page and Glending have postulated that extracts
of placental and decidual tissue high in thrombo-

plastic potency are forced into the maternal circu-
lation.   These extracts, partially adsorbed to fibrin,
are removed from the maternal circulation by the
liver, lungs, and other organs but serve to initiate
the coagulation process.   The same tissue extracts
probably convert plasminogen to plasmin resulting in
active fibrinolytic activity in the maternal blood.
Thrombin also causes platelet lysis which releases
large amounts of Serotonin into the maternal blood
stream and thus to the kidneys where it causes intense
vasoconstriction in the renal cortex.   The length of
this intense vasoconstriction is directly proportional
to the degree of renal cortical necrosis.

Anuria or extreme oliguria marks the onset of
the disease and may begin prior to, at the time of,
or following delivery.   Mortality is as high as
forty-four per cent in the eclamptic patient if the
oliguria lasts more than twenty-four hours and as
high as nineteen per cent if it persists in spite
of treatment.

Conclusions

1. Acute renal insufficiency may occur at any
    time during pregnancy from any of a variety of
    causes.

2. Although it is a rather rare disease,
    symmetrical cortical necrosis of the kidneys should
    always be considered as a cause for acute renal in-
    sufficiency occurring during eclampsia and following
    abruptio placenta. This is especially true when
    there seems to be no other explanation such as shock,
    infection, or transfusion reaction.

3. Low platelet counts during the acute stage of
    abruptio may give indication of the possibility of
    development of bilateral cortical necrosis.

4. Evacuation of the uterus and the relief of
    hydrostatic pressure within the uterus as soon as
    possible following abruptio greatly reduces the
    danger of the development of symmetrical cortical
    necrosis of the kidneys.

Case Report I

This thirty-seven year old white female, Gravida
six, Para four, Ab. two, entered the University of
Nebraska College of Medicine Hospital on February 12,
1955. Her last menstrual period had been on July 15,
1954. No fetal movements were felt after January 1,
1955, and she noted a gradually increasing ankle
edema and periorbital edema which did not disappear
with rest. At her next prenatal check one month
later, she was told that the fetus was dead. Her
blood pressure and urine at this time were normal,
but during the following week she noted a definite
decrease in the amount of urine output per day, esti-
mating the twenty-four hour output at four hundred
fifty to five hundred cubic centimeters. She also
developed intermittent frontal headaches, anorexia,
and malaise. On the sixth of February she noted
vaginal bleeding and had mild pains across the lower
part of her back.

This patient was next seen by her physician on
February 9, at which time she was in shock due to the
loss of a considerable amount of blood. The placenta
was delivered upon hospitalization where she received

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four tenths of a liter of whole blood and developed
a chill followed by a rise in temperature. During her
three day hospital stay before referral, she was anuric,
the highest twenty-four hour volume being one hundred
and sixty cubic centimeters.

At the time of admission to the University of
Nebraska Hospital the patient was extremely weak and
lethargic. Her blood pressure, pulse, temperature,
and respiration were all within normal limits; the
physical examination findings and her general appear-
ance were compatible with marked anemia and dehydration.
The abdomen was distended and somewhat tympanetic with
bowel sound being present. The fundus of the uterus
was palpated ten centimeters above the symphysis and
was doughy and tender to palpation. Tenderness to
palpation was also present in the right flank, and
serosanguinous lochia was noted on the perineum. The
only edema evident was a 1 + pitting edema over both
lateral malleoli.

During the early part of her hospital stay she
was given nothing orally but was given parenteral
fluid consisting of thirty per cent glucose solution
with added regular insulin I.V. The twenty-four hour
urine output rose gradually from a low of thirty-seven

cubic centimeters on the day of admission to one thousand seventy-five cubic centimeters five days later. The diuretic phase, defined as a twenty-four hour urine volume of at least one liter, began on the ninth day. After the beginning of the diuretic stage, the daily fluid intake was equal to the calculated sum of the insensible loss (approximately eight tenths of a liter) and the urine output of the previous day. The daily electrolyte intake was calculated to match the daily urine electrolyte loss. The N P N on the day of admission had been eighty-five mg. per cent and rose gradually to a high of two hundred thirty-five mg. per cent on the second day of the diuretic phase, after which there was a gradual decline to normal levels.

After oral feedings were instituted and tolerated on February 28, 1955, part of the calculated fluid intake was given orally and the remainder I.V. During the last two weeks of the hospitalization no I.V. fluids were necessary.

The patient continued to improve and was discharged on March 19, 1955, following recovery from an episode of acute renal failure. This case is particularly interesting because there appear to be several different possible etiologies for the renal failure in

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this patient. Bilateral cortical necrosis in its earliest stage could very well have been present. Unfortunately, this disease at this time cannot be diagnosed indisputably without kidney biopsy or post mortem examination.

The second case was, without question, one of bilateral cortical necrosis of the kidneys following premature separation of the placenta.

Case Report II

This patient was a twenty year old white female, Gravida one, Para 0,L.N.M.P. June 24, 1943, with calculated E.D.C. May 31, 1944. There was no history of previous pelvic, heart, lung, or kidney diseases. This patient was first seen on November 21, 1943, at which time active fetal movements were present, and the fundus was palpated twenty-two centimeters above the symphysis pubis. The blood pressure on November 27, 1943, was normal and showed no significant change by January 8, 1944, but there had been a seven pound weight gain in the previous two weeks. At this time the patient complained of sore breasts, frequency of urination, tiredness, and an irritating maculo-papular rash on the skin of her arms and legs.

On the morning of January 31, 1944, the patient was hanging curtains at home when she experienced a severe sharp pain in her abdomen. She was admitted to Methodist Hospital about five hours later in a wheel chair complaining of great pain in both flanks. At this time examination revealed the uterus to be en-larged to the size of seven months pregnancy, tightly contracted and tender to palpation. No fetal move-ment or heart tones were detected. Rectal examination revealed a long undilated cervix, and the diagnosis at that time was premature separation of the placenta and dead fetus.

A classical low cervical cesarean section was done approximately eight to ten hours after the initial onset of the symptoms. The placenta was completely separated and the entire maternal surface was covered with an organized clot of one to two centimeters thickness, totaling about one half a liter of clotted blood in the uterus. An indwelling catheter was put in place upon the patient's return to her room as she had not voided since admission to the hospital. Only fifteen cubic centimeters were obtained by the catheterization. On February 1, 1944, a C.B.C. revealed a marked anemia with a leucocytosis.

The following day the patient received one liter of ten per cent glucose in normal saline. There was tenderness to pressure in the lumbar region; the blood pressure was only slightly elevated, and an N P N of eighty-eight mg. per cent was recorded. No edema was detected, and no urine was obtained per catheter. On the third day of the anuria a flat film of the abdomen revealed the bowel to be grossly distended with gas, and the right renal shadow measured five by eleven centimeters while the left shadow measured six by twelve centimeters. There was no evidence of renal or ureteral calculi. The patient was listless, showed marked tenderness of the flanks, and had a temperature of one hundred and four tenths degrees.

On the fourth day of the anuric period, the patient showed labored respiration, a rapidly rising blood pressure, and an N P N of one hundred twenty-one mg. per cent. By the seventh anuric day the N P N had reached a high of one hundred eighty-two mg. per cent. An observation cystoscopy and ureteral catheter-ization showed the bladder and ureteral orifices to be essentially normal. Although the catheters passed to each renal pelvis readily, there was no flow from

either side. The patient remained anuric until death
on February 9, 1944, the ninth anuric day.

Autopsy findings revealed a right kidney that
weighed one hundred forty-five grams. The capsule
stripped with ease leaving a yellowish-gray smooth
surface. The cut surface of the kidney entire cortex,
three to five millimeters in depth, was grayish-
white in color and had the appearance of necrotic
infarcted tissue. There was congestion around the
discolored area, but the renal artery, veins, and
tributaries were devoid of any evidence of occlusion.
The left kidney also was completely necrotic.

Hemorrhagic areas measuring up to three centi-
meters across were found in the wall of the uterus
which was thickened in cut section and which con-
tained large blood spaces.

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